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Technical Memorandum

To: Project File and **Appendix K** to Remedial Investigation Report
From: Sheila Ugargol Keefe and Eric Dott
Subject: Screening Level Human Health Risk Evaluation
Date: March 27, 2013
Project: U.S. Steel Spirit Lake Sediment Investigation

This technical memorandum consists of the following sections:

1.0 Introduction

This Appendix contains the results of a Screening Level Human Health Risk Evaluation (HHRE) for the Spirit Lake Sediment Site (Site) in the St. Louis River, Minnesota. This evaluation was based on data provided in the *Draft Sediment Remedial Investigation (RI) Report*, prepared on behalf of U. S. Steel and the U.S. EPA Great Lakes National Program Office (GLNPO), under the Great Lakes Legacy Act (GLLA) Project at the Site in the St. Louis River, Duluth, Minnesota (Barr, 2012) as well as historical data from the Site, as referenced in this evaluation. This screening HHRE was requested by the Minnesota Pollution Control Agency (MPCA) in its response to the Draft RI. In a letter dated June 11, 2012, the

MPCA specifically requested a discussion of the potential human health exposure pathways identified in the conceptual site model and a comparison of Site data to draft Minnesota Department of Health (MDH) draft Sediment Screening Values (SSVs) for human health. Constituents of interest (COI) were identified for this comparison and include individual PAHs, carcinogenic PAHs as B[a]P equivalents, metals, and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) Equivalents. The exposure pathways identified for this evaluation included:

- incidental ingestion of sediments
- dermal exposure to sediments
- inhalation exposure to sediments
- incidental ingestion of surface water originating from sediments
- dermal exposure to surface water originating from sediments
- ingestion of fish exposed to sediments

The MPCA also requested the following:

- An inclusion-exclusion analysis of exposure pathways
- Determination of extent and likelihood of exposure to contaminants
- In-depth environmental fate and transport analyses for completed exposure pathways
- Assessment of exposure to sediment, to include the swimmer-wader exposure pathway
- Assessment of exposure via the fish consumption pathway
- A qualitative discussion of acute effects of dermal exposure to PAHs (to include rash, skin irritation, and the possible magnification of these effects following exposure to sunlight)
- Assessment of potential health risks via relevant exposure pathways
- Calculation of B[a]P equivalents for carcinogenic PAHs as prescribed by MDH
- Calculation of 2,3,7,8-TCDD Equivalents as prescribed by MDH
- Discussion of the potential for additive health effects
- Discussion of applicable sediment exposure pathways (incidental ingestion of sediment, dermal exposure to sediment, inhalation exposure to contaminants in sediment, incidental ingestion of contaminants in surface water originating from sediments, dermal exposure to contaminants in surface water originating from sediments, ingestion of contaminants via the fish consumption pathway).

This HHRE was a screening level evaluation. The procedures used in this screening HHRE were based on state-of-the-art, applicable science, policy, and procedures, including methodologies from MPCA guidelines, MDH Guidelines, U.S. EPA exposure and risk assessment guidelines, and recommendations of expert Federal panels. However, because of the limitations inherent in the risk assessment process, it is important to recognize that the risk characterization described in this screening HHRE or any health risk evaluation cannot predict actual health outcomes, such as cancer; in other words it estimates the potential human health risks, but does not provide an estimate of actual risk to an actual person.

In general, this screening HHRE was conducted based on the risk assessment guidelines listed below:

- Guidelines for the Health Risk Assessment of Chemical Mixtures (U.S. EPA, 1986).
- Risk Assessment Guidance for Superfund Volume 1 - Human Health Evaluation Manual Part A (U.S. EPA, 1989).
- Risk Assessment Guidance for Superfund Volume 1 - Human Health Evaluation Manual Part E (U.S. EPA, 2004).
- Guidelines for Developmental Toxicity Risk Assessment (U.S. EPA, 1991).
- Guidance for Data Usability in Risk Assessment (U.S. EPA, 1992).
- Guidelines for Exposure Assessment (U.S. EPA, 1992a).
- Guidelines for Carcinogenic Risk Assessment (U.S. EPA, 2005).
- Exposure Factors Handbook (U.S. EPA, 2011).
- Child-Specific Exposure Factors Handbook (EPA, 2008).
- Residual Risk Report to Congress (U.S. EPA, 1999).

This screening HHRE was conducted in five main steps, which included:

- Step 1: Identifying and eliminating potential COIs
- Step 2: Calculating mean sediment concentrations for remaining COIs for comparison to draft MDH SSVs (MDH, 2005)
- Step 3: Exposure assessment:
  - Identifying potentially complete exposure pathways requested by MPCA
  - Evaluating and calculating site-specific potential daily intake
- Step 4: Dose-Response/Toxicity Assessment: Compiling toxicity values (e.g. reference doses and dermal and/or oral slope factors) to estimate potential human health risks for the site
- Step 5: Risk Characterization: Combining exposure and toxicity information to calculate hazard indices (HIs) to assess potential noncancer effects and cancer risk estimates to assess potential cancer health risk using site-specific reasonable maximum exposure (RME) assumptions.

Each of these steps is described in detail in the following sections.

2.0 Step 1: Identifying and Eliminating Constituents of Interest (COIs)

The first step was to identify and then refine the list of COIs. COIs were selected from the constituents that were quantified during the RI or in previous site activities and based on discussions with GLNPO and MPCA (see Section 1.2 of the Draft RI, Barr 2012). The constituents that were analyzed in sediment included the metals arsenic, cadmium, chromium, copper, lead, mercury, nickel and zinc, PCBs, PAHs, total petroleum hydrocarbons (TPHs), dioxins/furans, organic carbon (total organic carbon (TOC) and black carbon), and cyanide. COIs were selected for further evaluation in terms of potential human health risks primarily if there was an existing draft MDH SSV for human health (MDH, 2005). Additionally, the list of 17 PAHs from the Draft RI (Table 4, Barr, 2012) were considered as COIs with the addition of perylene. The mean concentrations of perylene and phenanthrene were compared to the draft MDH SSVs. Draft MDH SSVs identify concentrations below which noncancer and/or cancer health effects are not expected based on long-term chronic exposure and MDH-developed criteria. COIs were evaluated either qualitatively or quantitatively, depending on the type of available data.

Of the potential COIs, acenaphthylene, benzo(g,h,i)perylene (a PAH),cadmium, carbazole, chromium, cyanide, dibenzofuran, methylmercury, perylene, phenanthrene, polychlorinated biphenyls (PCBs), TOC, black carbon and TPHs were eliminated from further qualitative or quantitative analyses for the reasons summarized in Table K-1.

Table K-1 COIs Excluded from the Screening Human Health Risk Assessment at the Spirit Lake Sediment Site, Duluth, Minnesota

Constituent Excluded from Quantitative Analyses	Reason for Exclusion
Acenaphthylene	No toxicity values were found to quantify potential health risks.
Benzo(g,h,i)perylene (a PAH)	No toxicity values were found to quantify potential health risks.
Cadmium	Cadmium concentrations in sediment samples collected from the Spirit Lake Sediment site are similar to background (or reference) concentrations. Therefore cadmium was not identified as a COI. Mean sediment concentrations were below the draft MDH SSV.
Carbazole	Measured concentrations were below the draft MDH SSVs and carbazole was infrequently detected.
Chromium	Draft MDH SSVs were speciated as Chromium (VI) and Chromium (III). Only total chromium was measured at this site. However, sediment conditions were determined to be

Constituent Excluded from Quantitative Analyses	Reason for Exclusion
	not conducive to the presence of chromium (VI) species because of predominantly reducing redox conditions.
Cyanide	Measured concentrations were below the draft MDH SSVs and measured sediment pH conditions were found to be acidic, which is not conducive to potential risk from the "free" form of this compound.
Dibenzofuran	Measured concentrations were below the draft MDH SSVs and dibenzofuran was infrequently detected.
Mercury/Methylmercury	Mean total mercury concentrations were below the draft MDH SSV for methyl mercury. Total mercury concentrations in sediment samples collected from Spirit Lake are similar to reference concentrations. Therefore, mercury (total and methyl) was not identified as a constituent of interest (COI).
Perylene	No toxicity values were found to quantify potential health risks.
Phenanthrene	No toxicity values were found to quantify potential health risks.
PCBs	-Barr detected PCBs in only four of thirty six samples at concentrations less than the limit of quantitation. The 4 samples were from two sampling locations, -SOMAT detected PCBs at only 1 sampling location. -The detection limits in all PCB samples were higher than the draft MDH SSVs. -The frequency and concentrations of PCBs detected at the Spirit Lake Sediment Site are similar to upriver reference concentrations.
Total Organic Carbon and Black Carbon	Total organic carbon and black carbon were evaluated to help determine bioavailability.
Total Petroleum Hydrocarbons	TPHs were mainly measured to determine if oily residues were present in both the Unnamed Creek and Wire Mill deltas. There is no clear methodology available for assessing potential human health risks from TPH contamination. Note: MPCA did not request TPHs be included in this screening HHRE.

The COIs retained for the exposure evaluation and for comparison to their respective draft MDH SSVs are shown in Table K-2.

Table K-2 Retained COIs and Draft MDH SSVs at the Spirit Lake Sediment Site, Duluth, Minnesota

COIs: Measured as part of the Remedial Investigation	Draft MDH SSVs from Spring 2012		Toxicity Endpoints Assessed in Screening HHRE Calculations/Additional Information [1]
	Draft Noncancer SSV mg/kg	Draft Cancer SSV mg/kg	
Metals			
Arsenic	32	30	Cancer and noncancer effects
Copper	5,400	None	Noncancer effects
Lead	300	None	Cancer effects
Nickel	2,900	None	Noncancer effects
Zinc	43,000	None	Noncancer effects
PAHs[2]			
2-Methylnaphthalene	18	None	Noncancer effects
Acenaphthene	54	None	Noncancer effects
Acenaphthylene	54	None	Only compared to draft MDH SSV
Anthracene	870	None	Noncancer effects
Benzo(a)anthracene	None	None	As benzo[a]pyrene equivalents, cancer effects
Benzo(a)pyrene	None	0.22	Cancer and noncancer effects
Benzo(a)pyrene equivalents	None	0.22	Cancer and noncancer effects
Benzo(b)fluoranthene	None	None	As benzo[a]pyrene equivalents, cancer effects
Benzo(k)fluoranthene	None	None	As benzo[a]pyrene equivalents, cancer effects
Chrysene	None	None	As benzo[a]pyrene equivalents, cancer effects
Dibenzo[a,h]anthracene	None	None	As benzo[a]pyrene equivalents, cancer effects
Fluoranthene	120	None	Noncancer effects
Fluorene	39	None	Noncancer effects

COIs: Measured as part of the Remedial Investigation	Draft MDH SSVs from Spring 2012		Toxicity Endpoints Assessed in Screening HHRE Calculations/Additional Information [(1)]
Indeno (1,2,3-cd)pyrene	None	None	As benzo[a]pyrene equivalents, cancer effects
Naphthalene	13	None	Cancer and noncancer effects
Pyrene	470	None	Noncancer effects
Dioxins and Furans[3]			
2,3,7,8-TCDD Equivalents	1.6E-06	2.5E-08	
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin (OCDD)	None	None	As TCDD Equivalents, cancer and noncancer effects
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	None	None	as TCDD Equivalents, cancer and noncancer effects
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	None	None	As TCDD Equivalents, cancer and noncancer

COIs: Measured as part of the Remedial Investigation	Draft MDH SSVs from Spring 2012		Toxicity Endpoints Assessed in Screening HHRE Calculations/Additional Information (1)
			effects
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects
1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)	None	None	As TCDD Equivalents, cancer and noncancer effects

(1) See Table K-9 for additional information about toxicity endpoints which were assessed in the screening HHRE.
(2) Only PAHs which were part of the Human Health Risk Evaluation are listed here.
(3) Doxin and furan congeners were included in the analysis as 2,3,7,8-TCDD equivalents as part of the MPCA-requested human health risk evaluation.

3.0 Step 2: Comparing Site Specific Mean COI Concentrations in Sediments to Draft MDH SSVs

The second step in this screening HHRE was to calculate mean sediment concentrations for the COIs and compare them to the draft MDH SSVs. Draft MDH SSVs identify concentrations below which noncancer and/or cancer health effects are not expected from long-term (chronic) exposure based on MDH criteria. MDH states that draft SSVs represent concentrations of chemicals which are *"...*protective of human health*"* (MDH, 2005). MDH goes on to state:

> Chemical concentrations in water-covered sediments at or below the human health-based Sediment Screening Values (SSVs) developed in this report are considered safe for the general public. Alternatively however, sediment concentrations greater than the screening values should not be considered unsafe, because the values were developed from conservative measures of 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.

For this screening HHRE, sediments are defined as "...loose particles of sand, clay, silt, and other substances that settle at the bottom of a body of water. Sediments may originate from the erosion of soil or from other decomposition of plants and animals. Wind, water, and ice often carry these particles great distances" (U.S. EPA, web link #1). Sediment does not include sand on the shoreline.

If mean concentrations of all COIs had been below the draft MDH SSVs, no further analyses would have been needed. However, mean constituent concentrations for some PAHs and TCDD Equivalents were above draft MDH SSVs, which warranted additional separate health risk calculations outside of comparison to draft MDH SSVs. The further evaluation was undertaken, which included additional health risk calculations for COIs for which concentration and toxicity values were available.

To develop relevant, but conservative exposure values, the human exposure point concentrations were calculated by taking the mean concentration of each COI from sediment core samples to a core depth of 2 feet. This value is conservative because exposure to sediments is expected to occur through contact with the upper few inches of the sediment during a wading event, when an individual would have contact with sediments over a relatively limited area. However, COI concentration data for sediments were not available for only the upper few inches in every sample location. Thus, concentration data from the upper 2 feet were used. Details on the calculation of the concentrations used for each COI are described below.

3.1 Calculating Mean Metal Concentrations

The mean metal concentrations in sediments from the Unnamed Creek delta and Wire Mill delta areas were each calculated using the U.S. EPA Software ProUCL. ProUCL is a statistical software package that is widely used to analyze environmental data sets with and without non-detect (ND) observations. This software was used to calculate the 95 percent upper confidence limit (UCL) of mean sediment concentrations of metals to a depth of two feet. The 95 percent UCL values presented were those recommended by ProUCL. The 95 percent UCL mean values of all metals analyzed at this site were below the draft MDH SSVs as summarized in Table K-3.

Table K-3 ProUCL-Generated 95%UCL of Mean Sediment Concentrations of Metals in Unnamed Creek and Wire Mill Delta Areas as Compared to Draft Minnesota Department of Health (MDH) Sediment Screening Values (SSVs) at Spirit Lake Sediment Site, Duluth, Minnesota

Metal	95 UCL Mean Metal Concentrations of core samples to a depth of 2 feet with non-detects counted at detection limit as recommended by ProUCL		Draft MDH SSVs	
	UC mg/kg	WM mg/kg	Noncancer SSV mg/kg	Cancer SSV mg/kg
Arsenic	7.02	5.66	32	30
Cadmium	1.12	0.79	97	none
Copper	33.2	175	5,400	none
Lead	104	116	300	none
Total mercury	0.271	0.186	43	none
Nickel	19.4	40.5	2,900	none
Zinc	341	445	43,000	none

UC = Unnamed Creek Delta area, WM = Wire Mill Delta area

3.2 Calculating Mean PAH Concentrations

PAHs were detected in most samples that were tested. Therefore, instead of using the ProUCL generated 95 percent UCL, the arithmetic mean concentration of all detected values was used to calculate the mean PAH concentrations. As noted above, mean PAH concentrations were calculated to a sediment depth of 2 feet. With the exception of naphthalene at an Unnamed Creek delta location, the concentrations of all PAH compounds that are classified as semi-volatile organic compounds (SVOCs) were below the draft MDH SSVs. The concentrations of eight other PAHs exceeded the draft MDH SSV for B[a]P equivalents. Of the nine PAHs exceeding the draft MDH SSVs, these PAHs were present in both the Wire Mill delta and Unnamed Creek delta with the exception of naphthalene, which was only present in the Unnamed Creek delta. Table K-4 lists the comparison of individual mean PAH site concentrations to the draft MDH SSVs.

Table K-4 Arithmetic Mean Sediment Concentrations of PAHs in Unnamed Creek and the Wire Mill Delta Areas as Compared to Draft Minnesota Department of Health (MDH) Sediment Screening Values (SSVs) at the Spirit Lake Sediment Site, Duluth, Minnesota

PAH	Mean PAH Concentrations of core samples to a depth of 2 feet for all detected values		Draft MDH SSVs	
	UC mg/kg	WM mg/kg	Noncancer SSV mg/kg	Cancer SSV mg/kg
2-Methylnaphthalene (SVOC)	5.72	0.539	18	None
Acenaphthene (SVOC)	0.855	2.16	54	None
Acenaphthylene (SVOC)	16.3	0.315	54	None
Anthracene (SVOC)	29.7	3.53	870	None
Benzo(a)anthracene(1)	**21.7**	**3.02**	None	0.22[1]
Benzo(a)pyrene(1)	**20.6**	**2.44**	None	0.22
Total Benzo(a)pyrene equivalents(2)	**33.8**	**3.33**	None	0.22
Benzo(b)fluoranthene(1)	**17.1**	**1.86**	None	0.22[1]
Benzo(k)fluoranthene(1)	**16.1**	**2.46**	None	0.22[1]
Chrysene(1)	**26.4**	**2.45**	None	0.22[1]
Dibenzo[a,h]anthracene(1)	**9.21**	**0.669**	None	0.22[1]
Fluoranthene (SVOC)	72.7	7.29	120	None
Fluorene (SVOC)	18.1	2.59	39	None
Indeno (1,2,3-cd)pyrene(1)	**21.1**	**1.81**	None	0.22[1]
Naphthalene (SVOC)	**19.1**	1.69	13	None
Phenanthrene (SVOC)	73.7	4.52	890	None
Pyrene (SVOC)	39.3	4.34	470	None
Perylene (SVOC)	6.81	1.91	490	None

(1) Used to calculate B[a]P Equivalents
(2) Reported as Total of B[a]P equivalents based on the non-detected PAHs used as part of the B(a)P equivalent concentration being counted at the full detection limit
Values in enlarged **bold** text exceed draft MDH SSV
UC = Unnamed Creek Delta area, WM = Wire Mill Delta area

3.3 Calculating Mean Dioxin/Furan 2,3,7,8-TCDD Equivalent Concentrations

Reference (or background) concentrations of 2,3,7,8-TCDD Equivalents likely already exceed the draft MDH SSVs and are estimated to range from 9.3E-07 mg/kg to 1.2E-05 mg/kg (MDH, 2005). When reference concentrations at a site are expected to exceed the draft MDH SSVs, MDH recommends using reference concentrations as sediment screening values. Moreover, sediment samples taken upriver of

Unnamed Creek and Wire Mill deltas show similar concentration and frequency distributions of 2,3,7,8-TCDD Equivalent concentrations as compared to the Spirit Lake Sediment site (see Appendix L). The data in Appendix L suggests that upriver sediment concentrations are an appropriate reference data set for comparison and that there are likely no significant concentration differences between upriver and Unnamed Creek and Wire Mill delta area sediment concentrations of 2,3,7,8-TCDD Equivalents.

The MPCA requested that 2,3,7,8-TCDD Equivalents be calculated for the risk screening evaluation. Some dioxin/furan congeners were detected in approximately two thirds of the samples, although a number of dioxin/furan congeners had non-detect values. ProUCL, therefore, used one-half the detection limit for samples with non-detects to generate the 95 percent UCL mean concentration of 2,3,7,8-TCDD Equivalents. The 95 percent UCL mean values presented were those recommended by ProUCL. Mean concentrations of 2,3,7,8-TCDD Equivalents exceeded the draft MDH SSVs at both Unnamed Creek and Wire Mill delta areas. Table K-5 compares the mean 2,3,7,8-TCDD Equivalent concentrations to the draft MDH SSVs for the two delta areas. However, if the estimated upper value of the range (1.2E-05 mg/kg) of the reference concentration of 2,3,7,8-TCDD Equivalents is used as an SSV, which MDH recommends when the draft SSVs are lower than reference concentrations, the estimated mean concentration at Unnamed Creek only slightly exceeds the reference concentration. The mean sediment concentrations of 2,3,7,8-TCDD Equivalents as compared to the estimated reference concentrations can be found in Table K-6. The draft MDH SSV for cancer effects is lower than the detection limits for dioxins/furans, and, as illustrated by Table K-5 as compared to Table K-6, is also two to three orders of magnitude lower than reference concentrations.

Table K-5 ProUCL Generated 95%UCL of Mean Sediment Concentration of 2,3,7,8-TCDD Equivalents in Unnamed Creek and the Wire Mill Delta Areas as Compared to Draft Minnesota Department of Health (MDH) Sediment Screening Values (SSVs) at the Spirit Lake Sediment Site, Duluth, Minnesota

Name	Mean 2,3,7,8-TCDD Equivalents Concentrations of core samples to a depth of 2 feet for detected for all detected values		Draft MDH SSVs	
	UC mg/kg	WM mg/kg	Noncancer SSV mg/kg	Cancer SSV mg/kg
2,3,7,8-TCDD Equivalents	**1.6E-05**	**1.1E-04**	1.6E-06	2.5E-08

Values in enlarged **bold** text exceed the draft MDH SSV
UC = Unnamed Creek Delta area, WM = Wire Mill Delta area

Table K-6 Estimated Concentrations of 2,3,7,8-TCDD Equivalents at the Spirit Lake Sediment Site as Compared to Estimated Reference Concentrations

Name	Mean 2,3,7,8-TCDD Equivalents Concentrations of core samples to a depth of 2 feet for detected for all detected values		Estimated Reference Concentrations of 2,3,7,8-TCDD Equivalents (MDH, 2005)
	UC mg/kg	WM mg/kg	mg/kg
2,3,7,8-TCDD Equivalents	1.6E-05	1.1E-04	9.3E-07-1.2E-05

3.4 Site-Specific Concentrations of COIs in Surface Waters

Existing surface water data were reviewed to assess potential exposure to humans from incidental ingestion of water while wading and/or swimming.

Three sets of surface water data were available for analysis:

1. Letter Report from U. S. Steel to the MPCA dated August 31, 2005 (Subject: Chemicals of Interest in Sediments and Surface Water USS- Former Duluth Works).
2. Data from URS dating from 1993-May 2012.
3. Data from the United States Geological Survey (USGS) dating from 1994-May 2012.

After review of available recent surface water data from URS and USGS, it was determined that many of the analytical results were reported as at or below the detection limit, qualified as "estimated" values, or flagged as blank contamination. Additionally, the numbers of surface water samples collected at each location were unknown. These data limitations, combined with the likelihood that the concentrations of

metals, dioxins, and PAHs in sediment are higher than in the water column due to their physical-chemical properties, warranted using risk estimates from the sediment exposure pathway as a qualitative surrogate of potential risks from exposure to surface water. Direct exposure to surface water through incidental ingestion of surface water, or dermal contact with surface water while swimming or wading could not be quantitatively evaluated. Thus, evaluation of the sediment pathway served as an indicator for both pathways. Potential health risks from dermal contact with sediment or incidental ingestion of sediment at the Site are expected to be higher than potential health risks from dermal contact with or incidental ingestion of surface water.

4.0 Step 3: Exposure Assessment

The third step of this screening HHRE was to evaluate how people might be exposed to COIs at this site and potential exposure concentrations. The MPCA requested that this screening HHRE include a discussion of complete and potential exposure pathways. In this screening HHRE, exposures are stated in terms of the external dose or intake and the toxicity values (cancer slope factors, Reference Doses) are also expressed as intakes.

4.1 Evaluation of Exposure Pathways

A "complete" exposure pathway means that evidence exists that a COI may be released from a source and may be transported into and through the environment to an exposure point where a human receptor is assumed to be present. Only “complete” exposure pathways were quantitatively evaluated. A complete exposure pathway does not indicate that adverse effects will occur, only that the effort to quantify exposures is warranted from the standpoint of assessing potential effects on human health. COIs which were detected in sediment at concentrations which were likely above reference concentrations were evaluated quantitatively as requested by the MPCA. Additionally, some COIs that were below the draft MDH SSVs were included in the screening HHRE to address the potential for additive health effects as requested by the MPCA (see Table K-1 for a discussion of excluded COIs). COIs detected in surface water were evaluated qualitatively. Of the six potential pathways requested by the MPCA, only four were considered potentially complete. A summary of the exposure pathway completeness analysis is provided in Table K-7.

Table K-7 Exposure Pathway Completeness Assessment at the Spirit Lake Sediment Site, Duluth, Minnesota

Migration Pathway	Potential Exposure Route	Exposure Potential	Pathway Included/ Excluded
COIs in sediments	Incidental ingestion of sediments	Possible incidental sediment ingestion sediment while wading or swimming.	Included
	Dermal exposure to sediments	COIs in sediments may result in human exposure through dermal contact with impacted sediments while wading or swimming. Although metals do not generally penetrate the skin barrier and enter the body to such a degree that they would pose a human health risk, to address the potential for additive health effects, metals were included in the screening HHRE.	Included
	Inhalation of volatile/semi-volatile organic compounds originating in sediments and volatilizing to air and dermal contact with volatile/semi volatile organic compounds originating in sediments and volatilizing to air	It is possible that volatile/semi-volatile organic compounds in sediments could volatilize and result in human exposure through inhalation and dermal contact. However, Dioxins, Furans and PAHs are not likely to volatilize to such an extent that they would pose a human health risk. In addition metals are not volatile or likely to volatilize and subject to inhalation or dermal contact via the volatilization pathway. Therefore, the volatilization pathway is considered de minimis and uptake of COIs through dermal contact in air is also considered de minimis.	Excluded
COIs in surface water	Incidental ingestion of surface water impacted by COIs originating from sediments	Incidental ingestion of impacted surface water during swimming or wading may occur.	Included
	Intentional ingestion of impacted surface water	It is possible that surface water may be used on occasion by campers as a source of drinking water. However because camping occurs only sporadically, the frequency of exposure is limited. This pathway is considered de minimis	Excluded
	Dermal contact with surface water impacted by COIs originating from sediments	Dermal contact with surface water during swimming or wading may occur. The concentration of metals, dioxins, and PAHs in sediment is higher in sediment than in water due to the physical and chemical properties of these compounds. In addition, metals do not generally penetrate the skin barrier and enter the body to such a degree that they would pose a human health risk. This pathway is considered de minimis.	Excluded

Migration Pathway	Potential Exposure Route	Exposure Potential	Pathway Included/ Excluded
	Inhalation of volatile/semi-volatile organic compounds originating in sediments now in surface water and volatilizing to air and dermal contact with volatile/semi volatile organic compounds originating in sediments now in surface water and volatilizing to air	It is possible that volatile/semi-volatile organic compounds in sediments could volatilize and result in human exposure through inhalation and dermal contact. However, Dioxins, Furans and PAHs are not likely to volatilize to such an extent that they would pose a human health risk. In addition metals are not volatile or likely to volatilize and subject to inhalation or dermal contact via the volatilization pathway Therefore, the volatilization pathway is considered de minimis and uptake of chemicals through dermal contact in air is also considered de minimis.	Excluded
	Fish consumption	Uptake by fish through direct contact with sediments and ingestion of surface water and sediments is possible. Sport fishing occurs in impacted surface waters. However, fish consumption advisories have been published by the MDH and signs are posted in the area to advise against fish consumption.	Included

Although a person may have dermal contact with sediment or incidentally ingest some sediment, the concentration in the sediment is typically not the concentration that will reach the blood. Bioaccessibility and bioavailability both play important roles in determining how much of a COI reaches the blood and potentially a target organ. Bioaccessibility is the fraction of a COI in an environmental medium that is available for absorption based on data from *in vitro* extraction but not necessarily absorbed into the body. Bioaccessibility depends on the relation between *in vitro* chemical extraction systems and what is measured *in vivo* (in animals or humans). *In vitro* extraction methods were developed as an inexpensive alternative to more expensive *in vivo* experiments (Ruby et al., 1999). The bioavailability ("absolute bioavailability") of a compound can be defined as the fraction of an administered dose that reaches the central (blood) compartment, whether through the gastrointestinal tract, skin, or lungs (NEPI, 2000). Bioavailability plays an important role in determining whether exposure to a specific COI will result in adverse effects and the severity of such effects.

4.2 Describing and Quantifying Exposure

The MPCA requested consideration of the potential for additive health effects from exposure to chemical mixtures. Therefore in addition to comparison of mean sediment concentrations to the draft MDH SSVs, potential health risks were calculated using the methods described below. The third step was to calculate potential noncancer and cancer health risks based on mean sediment concentrations using a site-specific reasonable maximum exposure (RME) scenario. Mean surface water concentrations were not calculated for reasons outlined in Section 3.4.

Table K-8 shows a list of the COIs that were assessed for potential noncancer and cancer health risks and their potentially complete exposure pathways.

Table K-8 List of Constituents of Interest Considered in Human Health Risk Calculations and the Completed Routes of Exposure at the Spirit Lake Sediment Site, Duluth, Minnesota

Chemical for Evaluation	Exposure via Sediment		Exposure via surface water	
	Dermal Contact with Sediment	Incidental Ingestion of Sediment	Incidental Ingestion While Wading or Swimming	Ingestion of Fish
Metals				
Arsenic	x	x	x	x
Copper	x	x	x	x
Lead	x	x	x	x
Nickel	x	x	x	x
Zinc	x	x	x	x
PAHs				
2-Methylnaphthalene	x	x	x	x
Acenaphthene	x	x	x	x
Anthracene	x	x	x	x
Benzo(a)anthracene	x	x	x	x
Benzo(a) pyrene	x	x	x	x
Benzo(a)pyrene equivalents	x	x	x	x
Benzo(b)fluoranthene	x	x	x	x
Benzo(k)fluoranthene	x	x	x	x
Chrysene	x	x	x	x
Dibenz[a,h]anthracene	x	x	x	x
Fluoranthene	x	x	x	x
Fluorene	x	x	x	x
Indeno(1,2,3-cd)pyrene	x	x	x	x

Chemical for Evaluation	Exposure via Sediment		Exposure via surface water	
	Dermal Contact with Sediment	Incidental Ingestion of Sediment	Incidental Ingestion While Wading or Swimming	Ingestion of Fish
Naphthalene	x	x	x	x
Pyrene	x	x	x	x
Dioxins/Furans	x	x	x	x
2,3,7,8-TCDD Equivalents	x	x	x	x

In order to estimate potential health risks it was necessary to find toxicity values to compare to the estimated sediment concentrations. Table K-9 shows the COIs that were evaluated as part of the screening HHRE and their specific toxicity endpoints. The following paragraphs describe the site-specific calculation of potential exposure amounts.

Table K-9 List of Constituents of Interest Considered in Human Health Risk Calculations and Evaluated Toxicity Endpoints at the Spirit Lake Sediment Site, Duluth, Minnesota

Chemical for Evaluation	Dermal Exposure via Sediment		Ingestion Exposure via Sediment	
	Potential Cancer Effects	Potential Noncancer Effects	Potential Cancer Effects	Potential Noncancer effects
Metals				
Arsenic	x	x	x	x
Copper		x		x
Lead	x		x	
Nickel	x	x	x	x
Zinc		x		x
PAHs				
2-Methylnaphthalene		x		x
Acenaphthene		x		x
Anthracene		x		x
Benzo(a)anthracene	x		x	
Benzo(a) pyrene	x	x	x	x
Benzo(a)pyrene equivalents	x		x	
Benzo(b)fluoranthene	x		x	
Benzo(k)fluoranthene	x		x	
Chrysene	x		x	
Dibenz[a,h]anthracene	x		x	
Fluoranthene		x		x

Chemical for Evaluation	Dermal Exposure via Sediment		Ingestion Exposure via Sediment	
Fluorene		x		x
Indeno(1,2,3-cd)pyrene	x		x	
Naphthalene	x	x	x	x
Pyrene		x		x
Dioxins/Furans				
2,3,7,8-TCDD Equivalents	x	x	x	x

4.3 Additional Environmental Fate and Transport Information for PAHs

PAHs consist of hydrogen and carbon arranged in the form of two or more fused benzene rings. There are thousands of PAH compounds, each differing in the number and position of aromatic rings, and in the position of substituents on the basic ring system. Environmental concern has focused on PAHs that range in molecular weight from 128.16 (naphthalene, 2-ring structure) to 300.36 (coronene, 7-ring structure). In general, PAHs show little tendency to biomagnify in food chains, despite their high lipid solubility, probably because most PAHs are rapidly metabolized. Inter- and intraspecies responses to individual PAHs are quite variable, and are significantly modified by many inorganic and organic compounds, including other PAHs. Until these interaction effects are clarified, the results of single substance laboratory tests may be extremely difficult to apply to field situations of suspected PAH contamination.

In water, PAHs may either evaporate, disperse into the water column, become incorporated into bottom sediments, concentrate in aquatic biota, or experience chemical oxidation and biodegradation. The most important degradative processes for PAHs in aquatic systems are photooxidation, chemical oxidation, and biological transformation by bacteria and animals. Most PAHs in aquatic environments are associated with particulate materials; only about 33% are present in dissolved form. PAHs dissolved in the water column will probably degrade rapidly through photooxidation, and degrade most rapidly at higher concentrations, at elevated temperatures, at elevated oxygen levels, and at higher incidences of solar radiation. PAHs that accumulate in aquatic sediments can undergo biotransformation and biodegradation by benthic organisms (Eisler, 1987) and microorganisms (Lu et al., 2011). Biodegradation of PAHs by microorganisms in sediment appears to be a site-specific phenomenon which depends on varying physical-chemical parameters. PAHs in aquatic sediments degrade very slowly in the absence of penetrating radiation and oxygen (Eisler, 1987). Benthic organisms and microorganisms can metabolize PAHs to products that may ultimately be completely degraded.

4.4 Institutional Controls to Limit Exposure at the Spirit Lake Sediment Site

Institutional controls are in place to reduce exposure to COIs at this site. Typically, institutional controls such as fish consumption advisories or signs prohibiting certain activities are not 100% effective.

4.4.1 Federal and State Restrictions

Recreational activities such as boating, swimming, wading, and fishing are prohibited at the Spirit Lake Site (33 CFR Section §165.905 (1830) (b)) and have been since October 1995.

Signs are posted on the site warning against using the area for boating, swimming, wading and fishing. The posted signs also inform people that there are MDH fish advisories for Spirit Lake. Although signs are posted warning people to avoid exposure to sediments at this site, human health risks arising from direct contact with sediment may potentially still occur as a result of recreational activities along shorelines or in small creeks. However, these direct exposures to sediments are generally limited to older children and adult populations *via* incidental ingestion and dermal contact. Exposure for very young children would be minimal for these types of activities, as adult supervision is likely to limit their contact which is why potential health risks were calculated for children ages 6-16, 16-18, and adults from 18-33 years old.

Given these site-specific limitations to direct exposure, a likely exposure scenario could be occasional older children and adult trespassers wading in sediments adjacent to the Site. MDH assessed this site when they derived their first human health sediment screening values (SSVs) in 2005 (MDH, 2005). Based on conversations with local residents, MDH estimated that local residents wade along the shore of the St. Louis River from May through October (MDH, 2005) but there was no mention of people specifically wading at the Spirit Lake Sediment Site. Sediment at Unnamed Creek is firm which will likely further limit dermal contact. Swimming in the river was assumed to occur during the summer months of June, July, and August (MDH, 2005). In the MDH assessment, the lifetime yearly average exposures were assumed to be 17.2 events/year for wading and 56.6 events/year for swimming. On a site-specific basis, however, during sediment data collection activities in 2011(including the months of February, May, June, October and November) and 2012 (including the months of May, July, August, September, October, and November), no swimming or wading was observed in the Unnamed Creek or Wire Mill delta areas. There were no reports in personnel field notes of seeing swimmers or waders at the Spirit Lake Sediment Site. Based on these site-specific observations by personnel performing Site related investigation activities, the exposure frequency (events or days per year, assuming 1 event per day) for

older children and adults wading, swimming, or engaged in other aquatic activities was assumed to occur once every two weeks during the warmer months of the year, for a total of eight exposure events or days per year.

4.4.2 Minnesota Department of Health (MDH) Fish Consumption Advisories for the St. Louis River from Fond du Lac to Lake Superior

MDH has established fish consumption advisories (MDH, Web link #2) for the stretch of the St. Louis River starting from Fond du Lac to Lake Superior as a result of mercury and PCB concentrations found in fish. Compliance with the fish consumption advisories would limit exposure to persistent and bioaccumulative chemicals from fish consumption. The MDH fish consumption advisories are shown in Table K-10.

Table K-10 Minnesota Department of Health (MDH) Fish Consumption Advisories for the St. Louis River from Fond du Lac Dam to Lake Superior

Fish	For women who are or may become pregnant and children under 15				For men and women not planning on becoming pregnant			
	1 meal per week	1 meal per month	DO NOT EAT	Contaminants	Un-restricted	1 meal per week	1 meal per month	Contaminants
Carp		All sizes		Mercury and PCBs			All sizes	Mercury and PCBs
Channel Catfish		All sizes		Mercury and PCBs		All sizes		Mercury and PCBs
Crappies	All sizes			Mercury	All sizes			
Northern Pike		All sizes		Mercury		All sizes		Mercury
Redhorse Sucker	NA	NA	NA	NA		All sizes		Mercury
Smallmouth bass		All sizes		Mercury and PCBs		All sizes		Mercury and PCBs
Sunfish	All sizes			Mercury	All sizes			
Walleye		Shorter than 20 inches	Longer than 20 inches	Mercury and PCBs		Shorter than 20 inches	Longer than 20 inches	Mercury and PCBs
White Sucker	All sizes			Mercury		All sizes		Mercury
Yellow Perch	All sizes			Mercury		All sizes		Mercury

NA=No advisory

4.5 Estimating Daily Intake from Incidental Ingestion of Sediments

Incidental ingestion of impacted sediments was included as a completed pathway. U.S EPA recommends using equations developed for soil exposure as a surrogate for sediment exposure (U.S. EPA, 1989-Chapter 6). All exposure assumptions used from MDH were based on reasonable maximum exposure (RME) assumptions (MDH, 2005). The MDH values used in this screening HHRE can be found in Attachment 1. RME exposure is defined as "the highest exposure that is reasonably expected to occur at a site, and is intended to estimate a conservative case (i.e. well above the average case) that is still within the range of possible exposures" (U.S. EPA, 1989). RME refers to people at the high end of the exposure distribution, approximately the 95th percentile. Within a potentially exposed population, the magnitude of potential exposure is variable and associated with differences in individual characteristics and recreational activity patterns. Therefore, a distribution of exposure across the population exists where the difference in exposure between the low end, average and high end is considerable. Factors such as age, sex, and activity patterns affect the amount of COIs to which an individual is exposed over time. All exposure assumption used in this report represent high end exposure assumptions.

Human exposure to impacted sediments can occur through incidental ingestion of sediment in surface water while wading/swimming or other recreational activities. In this screening HHRE, ingestion rates were conservatively estimated based on the assumption that a person could swallow sediment in surface water while swimming. An exposure duration of 33 years of residing in one place was chosen by MDH based on the 95^{th} percentile value for residential occupancy period to derive the draft SSVs (U.S. EPA, 2011). It was assumed in this screening HHRE that children ages 1-6 years old would not be wading or swimming at the Site. Therefore potential health risks were calculated for people ages 6-16, 16-18, and 18-33 years old. In most cases, contact and ingestion of sediment at locations associated with industrial/commercial land use are not expected to be relevant exposure pathways (USEPA, 1989). The potential health risks of exposure to incidental ingestion of sediments can be assessed by calculating a potential daily intake (DI) (U.S. EPA, 1989-Chapter 6, Exhibit 6-14). When the medium of exposure in a site assessment differs from the medium of exposure used to derive the toxicity value U.S. EPA allows for an absorption adjustment. An oral absorption factor (OAF) was included in the daily intake calculation to account for adjustment from the medium used in RfD derivation to sediment ingestion (U.S. EPA, 1989-Appendix A). Chemical specific oral absorption factors (OAFs) can be found in Attachment 2. Daily intake from potential incidental ingestion of sediments was calculated using the following equation:

$$DI = (C \times CF \times OAF \times FI \times IR \times EF \times ED)/ BW \times AT$$

Where:

DI= daily intake in mg/kg-day

C=Concentration of COI in sediment in mg/kg at Unnamed Creek or Wire Mill

CF=Conversion Factor of 10^{-6} kg/mg

OAF=Oral absorption factor: COI specific, unitless, see Attachment 2 for COI specific values

FI=Fraction of sediment impacted: 1 maximum value, unitless, (MDH 2005)

IR= Sediment ingestion rate in mg/day: assumes 95th percentile value of ingestion rate of surface water containing the maximum detected amount in surface water samples from Unnamed Creek (see age specific values in Attachment 1)

EF=Exposure frequency in events (or days)/year: 8 days/year (site specific value based on observation by on-site personnel)

ED=Exposure duration in years: 6-16 year olds =10 years, 16-18 year olds = 2 years, 18-33 year olds = 15 years (see age specific values in Attachment 1)

BW= Body weight in kg: see age specific values in Attachment 1 (MDH 2005)

AT=Averaging time in years: 25550 days (70 years) for carcinogens; age specific equivalent to exposure duration in Attachment 1 for noncarcinogens

Note: These calculations assume one event/day for wading or swimming

4.5.1 Values Used to Estimate Daily Intake by Incidental Ingestion of Sediment Using Site-Specific Reasonable Maximum Exposure (RME) Assumptions

Some of the values used in the screening HHRE were values used by MDH (MDH, 2005) to derive the draft SSVs. MDH assumed that a person would be exposed to the Spirit Lake Sediment site for 33 years of a lifetime as 1-6 year olds, 6 -16 year olds, 16-18 year olds, and 18-33 year olds. This screening HHRE assumed that young children would not be wading or swimming at the site and evaluated potential risks for 6-16 year olds, 16-18 year old and 8-33 year olds. Conservatively, ingestion rates for sediment were based on 97th percentile values for ingestion of surface water (containing sediment) while swimming to estimate potential risk (USEPA, 2011). The sediment ingestion rates (mg/day) used in the calculations were based on the assumptions for an RME as outlined below:

- Ingestion of 0.1 L/event for 6-18 year olds (U.S. EPA, exposure factors, 2011, also MDH, 2005)
- 1 event/day for wading and swimming for ages 16-18 years (site specific assumption)
- Ingestion of 0.0071 L/event and 1 event/day for ages > 18 years (USEPA, 2011)

The resulting daily sediment ingestion rates can be found in Attachment 1.

Site-specific information was developed based on observations at the Spirit Lake Sediment Site. The maximum total suspended solid concentration for samples taken in the Unnamed Creek delta area was 427 mg/L (Barr, 2012)

A sediment ingestion rate for children was estimated using the following equation:

For 6-18 year olds: 0.1 L/hour x 1 hour/event x 1 event/day x 427 mg/L= 51.2 mg/day
For > 18 year olds: 0.071 L/hour x 1 hour/event x 1 event/day x 427 mg/L=30.3 mg/day

Site specific observations were used to adjust the exposure frequency (EF). On-site personnel have never reported seeing waders or swimmers at this site. Based on site specific observations, an event frequency of 8 days (or events)/year was used to calculate potential health risks for the site-specific exposure scenario. The factors that could limit the exposure frequency include the:

- cold, northern climate with limited summer months
- lack of good sandy beaches to swim from near the site
- availability of alternative recreational facilities within the Duluth area
- limited direct access to the Spirit Lake shoreline (other than from a boat)
- existence of warning signs advising no swimming or recreation

4.6 Estimating Dermal Exposure to Sediments

Similar to the discussion of exposure factors associated with incidental ingestion, significant uncertainties are associated with estimating the potential factors that could be applied to evaluate direct exposure to sediments via dermal contact. As noted above, it was assumed in this screening HHRE that children ages 1-6 years old would not be exposed to sediment via wading or swimming. Therefore, potential health risks were calculated for people ages 6-16, 16-18, and 18-33 years old. The potential health risk of dermal contact with sediments can be assessed by calculating a potential dermally absorbed dose (DAD), using formulas 3.11 and 3.12 from U.S. EPA Risk Assessment Guidance for Superfund (USEPA, 2004). The dermally absorbed dose is calculated as follows:

$$DAD = (C \times CF \times DAF \times F \times SA \times AF \times EF \times ED)/ BW \times AT$$

Where:

DAD = dermally absorbed dose in mg/kg-day

C = concentration of COI in sediment in mg/kg at Unnamed Creek or Wire Mill delta areas

CF = conversion factor of 10^{-6} kg/mg

DAF = dermal absorption fraction: COI specific, unitless (see values in Attachment 1)

F = Fraction of sediment impacted: 1 maximum value, consistent with MDH, 2005

SA = Surface area of skin exposed: see age specific values in Attachment 1

AF = Sediment/skin adherence factor: see age specific values in Attachment 1, (consistent with MDH, 2005)

EF = Exposure frequency in events (or days)/year: 8 events/year (assumed site specific value)

ED = Exposure duration: 6-16 year olds=10 years, 16-18 year olds = 2 years, 18-33 year olds = 15 years (see age specific values in Attachment 1, MDH 2005)

BW = Body weight: see age specific values in Attachment 1 (MDH, 2005)

AT =Averaging time: Carcinogen 25550 days (70 years); age specific value equivalent to exposure duration for noncarcinogens (see age specific values in Attachment 1)

Note: These calculations assume 1 event/day for swimming or wading

4.6.1 Values Used to Estimate Daily Intake from Dermal Contact for Using Site-Specific Reasonable Maximum Exposure (RME) Assumptions

Default values used in the screening HHRE to estimate potential health risks from dermal contact with sediment were divided into specific age ranges: 6 -16 year olds, 16-18 year olds, and 18-33 year olds. The skin surface area available for contact was based on the assumption that a person would go wading and have direct contact with sediment on their feet.

On-site personnel have reported firm sediments in the Unnamed Creek delta area and somewhat softer sediments in the Wire Mill delta area. Therefore exposure to sediments, especially in the Unnamed Creek delta area is expected to be limited.

With respect to potential dermal contact with sediment while swimming, USEPA reports (USEPA, 2004) that "Sediments which are consistently covered with water are likely to wash off before the individual reaches the shore." This would limit dermal exposure to sediment while swimming. Therefore the assumptions regarding wading were used in this screening HHRE.

The surface area for contact during wading for 6-16 year olds was estimated by using the 95th percentile value of surface area for feet of 1360 cm^2 (USEPA, 2011-Table 7-2). These data from USEPA are for 11-16 year olds as this was the closest age group for which data was summarized.

The surface area for contact during wading for 16-18 year olds was estimated by using the 95th percentile value of surface area for the feet of 1420 cm^2 (USEPA, 2011-Table 7-2). These data from USEPA are for 16-21 year olds as this was the closest age group for which data was summarized.

The surface area for contact during wading for 18-33 year olds was estimated by using the 95th percentile value of the surface area for feet 1610 cm^2 (values are from Table 7-12-USEPA, 2011). These data from USEPA are for adult males 21 years and older as this was the closest age group for which data was summarized.

The values to estimate the surface area of feet exposed to sediment represent reasonable maximum exposure assumptions (at least the 95th percentile values) and are therefore a conservative estimate of skin surface area potentially exposed to sediment.

4.6.2 General Conservatism in Estimating Dermally Absorbed Dose

In addition to the potential exposure frequency of eight days/year, as described in Section 4.3.2 , the next most significant uncertainty associated with the dermal exposure assessment is estimating COI specific dermal absorption factors based on estimating the amount of sediment on the skin and the length of exposure (U.S. EPA, 2007). Dermal exposure to COIs in the sediment is a function of dermal adherence and dermal absorption. A study by Kissel, *et al.* (1996) showed that the highest adherence corresponded to contact with wet sediments, such as might occur during wading or other shoreline activities. PAHs have a strong affinity for organic carbon in particulate matter leading to higher PAH concentrations in sediment than in surface water. U.S.EPA notes that if contact with sediment occurs in areas that are covered with considerable amounts of water the sediment is likely to be washed off before the individual reaches the shore (USEPA, 2004). COI specific dermal absorption factors (DAFs) can be found in Attachment 2. Additional discussion regarding dermal absorption and permeability can be found in Attachment 3.

4.6.3 Dermal Absorption of Metals

Many COIs, including metals, have little to no dermal absorption and therefore pose limited dermal contact risk. However metals were included in the screening HHRE calculations as the MPCA requested

discussion of the potential for additive health effects. The MDH has studied this site on the St. Louis River and concluded the following about metals (MDH, Web link #1):

> High levels of zinc and chromium (as well as elevated levels of lead, cadmium, nickel, and copper) have been found in river sediments near the site. Because human exposure to Saint Louis River sediments is limited, human uptake of these metals is not expected to be above levels of concern for human health.

4.6.4 Dermal Absorption of PAHs

In the U.S., most people are exposed to PAHs primarily from inhalation of PAHs in tobacco smoke, wood smoke, and ambient air or from ingestion of foods containing PAHs (ATSDR, 1995). Although there are signs prohibiting swimming and wading at this site, incidental dermal contact with contaminated sediments may occur during swimming or wading.

There are limited data available addressing absorption of PAHs via dermal contact with PAH impacted sediment. It appears that PAHs dermally applied are generally not well absorbed through the skin. In a study conducted to determine differences in absorption of polycyclic aromatic hydrocarbons (PAH), coal-tar ointment was applied to the skin of volunteers. This study indicates, based on the urinary excretion of 1-OH-pyrene, that after 6 h of exposure, 20–56% of a low dermal dose of PAH will be absorbed implying that in general, PAHs are not well absorbed through the skin (VanRooij, et al., 1993).

Dermal contact with mixtures of carcinogenic PAHs has been shown to cause skin disorders in humans and animals; however, specific effects in humans have only been reported for dermal contact with benzo[a]pyrene (ATSDR, 1995). Additionally, these skin effects in humans were noted in only one study where exposures occurred over four months in patients with pre-existing skin conditions using a 1% solution containing benzo(a)pyrene (ATSDR, 1995).

Skin contact with anthracene and naphthalene has been shown to cause mild irritation in animal studies (Integrated Criteria Document PAH, 1989). Additionally, anthracene is a photosensitizer in animal studies meaning skin contact with anthracene combined with exposure to sunlight produced increased skin allergic reactions (Integrated Criteria Document PAH, 1989). For example, in one animal study, hairless mice were exposed to dermal application of anthracene over a 96 hour period followed by exposure to 40 minutes of ultraviolet radiation. This combination produced enhanced skin inflammation

as compared to mice exposed to ultraviolet radiation alone. However, the skin inflammation was reversible within 48 hours (ATSDR, 1995).

4.7 Estimating Incidental Ingestion of Surface Water

People may incidentally swallow small amounts of surface water that may have been impacted by COIs partially derived from sediments while wading, swimming or participating in recreational activities. However, because of the limited surface water data that were available (see Section 3.4), potential health risks from incidental ingestion of surface water were not calculated. Instead, the potential health risks from ingestion of sediments were qualitatively considered a surrogate for potential surface water exposure. Potential health risks from incidental ingestion of surface water are expected to be minimal as concentrations of metals, PAHs and dioxin/furans are expected to be higher in sediment than surface water, based on low solubilities and high affinity to adsorb to sediment particles (U.S. Fish and Wildlife Service, 2013, U.S. EPA-Technical Factsheet on Dioxin, and WATERSHEDDS-Heavy Metals). Therefore no further analysis of this exposure pathway was conducted.

4.8 Estimating Exposure from Ingestion of Fish

Humans can be indirectly exposed to COIs present in surface water and sediments when they eat fish from the same water body. Some COIs which are persistent can bioaccumulate in fish. Depending upon the solubility of a COI and other factors, uptake of COIs into fish may occur directly from the water, or indirectly through sediments. The methodology for estimating chemical concentrations in fish is related to the chemical specific octanol-water partition coefficient (K_{ow}). For chemicals with a log K_{ow} less than 4.0, concentrations in fish tissue are estimated using bioconcentration factors (BCFs). For chemicals with a log K_{ow} greater than 4.0, bioaccumulation factors (BAFs) are used. For chemicals with a high affinity for bed sediments, biota-sediment accumulation factors (BSAFs) are used. Surface water concentrations are also required for this calculation.

COIs at this site that are potentially relevant are PAHs, mercury, PCBs, and dioxins/furans (U.S. EPA, Web link #2). The MDH has studied this site and concluded that "...some exposures could occur from eating PAH contaminated fish. Without information about the future use of PAH contaminated areas, MDH cannot determine whether exposures will reach levels of health concern…" (MDH, 2005). Daily intake of COIs via the fish consumption pathway and potential health risks were not calculated for fish consumption due to the lack of data on the concentrations of COIs in surface water. It should also be noted that target fish species for potential fisher persons are mobile and are likely to bioaccumulate

chemicals from varying locations. As noted previously, signs warning against swimming and fishing are posted at the Site and MDH has issued a fish consumption advisories that encompass the entire lower St. Louis estuary.

5.0 Step 4: Dose-Response/Toxicity Assessment

The fourth step in this screening HHRE was to assess the toxicity of the COIs. The toxicity assessment is used to identify and evaluate available evidence regarding the potential for particular COIs to cause adverse effects in exposed individuals and provides, where possible, a method to estimate the relationship between the extent of exposure to a chemical and the likelihood and/or severity of potentially adverse effects. This toxicity assessment is comprised of two components: hazard identification and dose-response assessment. Hazard identification is the process of determining whether exposure to a substance can cause a statistically significant increase in the incidence of a particular health effect. Health effects are divided into two categories: cancer (carcinogenic) and noncancer (noncarcinogenic) effects. Only potential chronic health effects were considered in this screening HHRE. Health effects for some COIs associated with the Site fall within both the carcinogenic and noncarcinogenic categories. The dose response relationships considered in this evaluation for cancer and noncancer effects are described in the following paragraphs.

Human health risk evaluations usually have a conceptual gap between the exposure assessment for COIs in the various media of interest (e.g., sediments) and the toxicity assessment for the COIs in those same media. The exposure assessment yields quantitative estimates of dose for each COI based on bulk concentrations in environmental media such as sediments. The toxicity assessment uses values derived from a dose-response assessment using data from studies of the COI administered to laboratory animals in drinking water or food. Toxicity values can also be based on epidemiological studies of human populations exposed to specific COIs under certain exposure conditions (e.g. in workplace). Toxicity values based on epidemiology studies of human populations also are not based on exposure to the COI in sediments. Therefore, direct application of these toxicity values to doses of a COI from sediments can be inaccurate if the COI behaves differently in sediments, for example, if it is less bioavailable.

5.1 Describing Cancer (or carcinogenic) Effects

The toxicity value that represents the dose-response relationship for cancer effects from exposure to a chemical is the slope factor. The slope factor represents the plausible upper bound estimation of the probability of a carcinogenic response per unit intake of a chemical over a lifetime. Cancer, unlike many

noncancer health effects, is generally thought by the U.S. EPA to be a phenomenon for which risk evaluation based on presumption of a threshold is inappropriate (U.S. EPA, 1989). For carcinogens, U.S. EPA conservatively assumes that a small number of molecular events can cause changes in a single cell that can lead to uncontrolled cellular proliferation and eventually lead to a clinical manifestation of disease. This hypothesized phenomenon is referred to as "non-threshold" because there is believed to be essentially no level of exposure that does not pose a finite probability, however small, of generating a carcinogenic response (U.S. EPA, 1989).

Evidence of the cancer and/or noncancer health effects comes from the following major sources:

- Human epidemiological studies (e.g. data from occupational exposures, poisoning cases, etc.)
- Data from animal studies
- **A** variety of genotoxic, cytogenetic, pharmacokinetic and metabolic studies
- Physical/chemical properties and structural relationships

Additional information about USEPA's classification of carcinogens can be found in Attachment 4.

5.2 Describing Noncancer (or noncarcinogenic) Effects.

The toxicity value that represents the dose-response relationship for chronic, noncancer effects for ingestion or dermal contact is the reference dose (RfD). The RfD is defined as "… an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. …" (U.S. EPA, 1998). The RfD is generally expressed in units of milligrams per kilogram of body weight per day (mg/kg-day).

Noncarcinogenic health effects include a wide range of toxic effects on body organs or systems. Well-conducted epidemiological studies are given first priority in the dose-response assessment and animal studies are used as supportive evidence. When human data are lacking, U.S. EPA infers the potential for the substance to cause an adverse effect in humans from toxicity information derived from studies with laboratory animals. There are occasions in which observations in animals may be of uncertain relevance to humans. U.S. EPA considers the likelihood that the substance will have adverse effects in humans to increase, as similar results are observed across sexes, strains, species, and routes of exposure in animal studies (U.S. EPA, 1989). Adverse effects may range from effects that can cause incapacitation or death to subtle effects at the cellular level. The distinction between adverse and non-adverse effects is not

always clear-cut, and considerable professional judgment is required in applying criteria to identify adverse effects (U.S. EPA, Web link #4).

For many noncancer effects to occur, it is believed that the body's protective mechanism must be overcome before an adverse effect is manifested. When these protective mechanisms, or thresholds, are exceeded, adverse health effects may occur. For this evaluation, based on characteristics of the COIs and their concentrations in surface water and sediment, only potential health effects from chronic exposures were analyzed. Acute exposures to surface water or sediment at this site are unlikely to pose health risks. Additional information about RfDs can be found in Attachment 5.

5.3 Toxicity Values Used in this Screening Health Risk Evaluation

The exposure point concentrations were compared to the draft MDH SSVs in Section 3. Draft MDH SSVs define concentrations of chemicals in sediment below which no adverse health effect are expected based on acceptable MDH criteria (MDH, 2005). The potential health effects considered were cancer and noncancer health effects. Since the mean concentrations of some of the COIs in sediment were above the draft MDH SSVs, potential health risks were calculated. The dermal and oral cancer slope factors used in this screening HHRE can be found in Attachment 6. The dermal and oral RfDs used in this screening HHRE can be found in Attachment 7. The mean concentrations in sediment were compared to other available toxicity values in the following order of priority in order to calculate potential health risk:

1. Toxicity values from Minnesota Department of Health (MDH) promulgated Health Risk Values (HRVs) or from MDH chemical specific guidance.

2. Toxicity values published by U.S. EPA and available from an on-line database called the Integrated Risk Information System (IRIS). IRIS is a U.S. EPA database containing current toxicity values that have been verified by EPA work groups. IRIS is considered by EPA to be the preferred source of toxicity information.

3. Toxicity values developed by the State of California Environmental Protection Agency (Cal EPA), Office of Environmental Health Hazard Assessment (OEHHA). External experts have reviewed most, but not all, toxicity reference values published by the OEHHA.

4. Toxicity values published by U.S. EPA in the Health Effects Summary Tables (HEAST) document. U.S.EPA recently withdrew the HEAST values and their current status is not known. No HEAST values were used in this toxicity assessment.

5. Minimum Risk Levels (MRLs) developed by the Agency for Toxic Substances and Disease Registry (ATSDR). No ATSDR values were used in this toxicity assessment.

5.4 Potency Equivalency Factors Used to Calculate Benzo(a)pyrene Equivalents and Additional Toxicity Information about PAHs

The MPCA requested in its comments, that benzo(a)pyrene equivalents be calculated as per MDH recommendations (MDH, Web link #3). However, MDH is currently in the process of revising this guidance. The potency factors currently used by MDH and in this screening HHRE are in Table K-11.

Table K-11 Minnesota Department of Health (MDH) PAH Potency Equivalency Factors used in this Screening Human Health Risk Evaluation (HHRE)

PAHs	MDH Health Endpoint	MDH Recommended Potency Equivalency Factors
Benzo(a)anthracene	Cancer- as benzo[a]pyrene equivalents	0.1
Benzo(a)pyrene	Cancer- as benzo[a]pyrene equivalents	1 (index compound)
Benzo(b)fluoranthene	Cancer- as benzo[a]pyrene equivalents	0.1
Benzo(k)fluoranthene[1]	Cancer- as benzo[a]pyrene equivalents	0.1
Chrysene	Cancer- as benzo[a]pyrene equivalents	0.01
Dibenz[a,h]anthracene	Cancer- as benzo[a]pyrene equivalents	0.56 (From MDH, 2004)
Indeno (1,2,3-cd)pyrene	Cancer- as benzo[a]pyrene equivalents	0.1

5.5 Toxic Equivalency Factors Used to Calculate 2,3,7,8-TCDD Toxic Equivalents (Equivalents)

The terms “dioxins” or “furans” usually refer to a family of chlorinated compounds that are similar in structure. These chemicals, with varying locations of chlorine substitution, are referred to as congeners. These congeners are also similar in terms of biological activity. Some of these congeners interfere with

the Aryl hydroxylase (Ah) receptor in cells resulting in disruption of regulation of enzymes and other proteins. However, there were not data available on the toxicity of each of these congeners. Thus far, the most toxic and studied congener is 2,3,7,8-TCDD. Therefore the toxicity of the other congeners is compared to the toxicity of 2,3,7,8-TCDD by use of toxic equivalence (Equivalents). The MPCA requested in its comments, that 2,3,7,8-TCDD Equivalents be calculated as per MDH recommendations (MDH, Web link #4). The 2,3,7,8-TCDD equivalence factors currently used by MDH and in this screening HHRE are in Table K-12.

Table K-12 Minnesota Department of Health (MDH) Recommended 2,3,7,8-TCDD Toxic Equivalent Factors Used in the Human Health Risk Evaluation for the Spirit Lake Sediment Site, Duluth, Minnesota

Dioxin/Furans	MDH Health Endpoint	MDH Recommended Toxic Equivalency Factors
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	Cancer and noncancer	1
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	Cancer and noncancer	1
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	Cancer and noncancer	0.1
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	Cancer and noncancer	0.1
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	Cancer and noncancer	0.1
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	Cancer and noncancer	0.01
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin (OCDD)	Cancer and noncancer	0.0003
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	Cancer and noncancer	0.1
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	Cancer and noncancer	0.03
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	Cancer and noncancer	0.3
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	Cancer and noncancer	0.1
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	Cancer and noncancer	0.1
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	Cancer and noncancer	0.1
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	Cancer and noncancer	0.1
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	Cancer and noncancer	0.01
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	Cancer and noncancer	0.01
1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)	Cancer and noncancer	0.0003

5.6 Potential Health Effects Caused by the Constituents of Interest (COIs)

A summary of the potential health effects associated with COIs evaluated at this site are listed in Table K-13.

Table K-13 Summary of Potential Health Effects Caused by the Constituents of Interest based on Information Mainly from the U.S. EPA Integrated Risk Information System (IRIS) for the Spirit Lake Sediment Site, Duluth, Minnesota

COIs Measured as part of the Remedial Investigation	MDH Classification of draft SSVs based on cancer or noncancer toxicity endpoints	Toxicity Endpoints Assessed as per U.S. EPA IRIS
Metals		
Arsenic	Cancer and noncancer	Cancer (skin cancer) and noncancer (hyperpigmentation, keratosis and possible vascular complications) effects.
Copper	Noncancer	Noncancer effects (irritation of GI tract).
Lead	Noncancer (no RfD-U.S. EPA determined an RfD is not appropriate for lead)	Assessed for cancer effects, causes noncancer neurological effects.
Nickel	Noncancer	Noncancer and cancer effects.
Zinc	Noncancer	Noncancer effects (anemia, decreasing erythrocyte count).
PAHs		
2-Methylnaphthalene	Noncancer	Noncancer effects (pulmonary alveolar proteinosis).
Acenaphthene	Noncancer	Noncancer effects (toxicity to the liver).
Anthracene	Noncancer	Noncancer effects.
Benz(a)anthracene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (increase in tumors).
Benzo(a)pyrene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (increase in tumors of the fore-stomach) and noncancer effects.
Benzo(b)fluoranthene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (skin cancer, pleomorphic sarcomas).
Benzo(k)fluoranthene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (increase in tumors).
Chrysene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (increase in tumors).
Dibenz[a,h]anthracene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (lungs, respiratory, and mammary tumors).
Fluoranthene	Noncancer	Noncancer effects (circulatory effects, kidney, liver and blood effects).
Fluorene	Noncancer	Noncancer effects (circulatory effects, decreased red blood cell count and hemoglobin).
Indeno (1,2,3-cd)pyrene	Cancer- as benzo[a]pyrene equivalents	Cancer effects (bronchilar hyperplasia, metaplasia).
Naphthalene	Noncancer	Cancer and noncancer effects (oral exposure, decreased body weight in males).
Pyrene	Noncancer	Noncancer effects (mouse oral exposure-kidney effects).

COIs Measured as part of the Remedial Investigation	MDH Classification of draft SSVs based on cancer or noncancer toxicity endpoints	Toxicity Endpoints Assessed as per U.S. EPA IRIS
Dioxins/Furans		
2,3,7,8-TCDD	Cancer and noncancer	Cancer (lung and others) and noncancer effects (diabetes, cardiovascular, respiratory, neurological, immune system, reproductive, developmental, liver and endocrine effects and decreased sperm count and motility in men exposed to TCDD as boys). (1)

(1) Data is from the U.S. EPA IRIS database, ATSDR Toxicological Profile for Chlorinated Dibenzo-p-dioxins (CDDs), Addendum to the ATSDR Toxicological Profile, and the IARC Monograph for 2,3,7,8-Tetrachlorodibenzo-para-dioxin, 2,3,4,7,8-pentachlorodibenzofuran, and 3,3',4,4',5-Pentachlorobiphenyl.

6.0 Step 5: Risk Characterization-Estimating Potential Health Risks

The fifth and final step in this screening HHRE was combining information from the exposure assessment (calculated daily intakes) with the toxicity information (RfDs or slope factors) to calculate potential health risks. Risk analysis is a comparison of the toxicity of a chemical with the likely exposure to that chemical. Regardless of how risks are expressed, risks remain dependent on toxicity and exposure and to alter either alters the risk. Therefore, an accurate assessment of risk requires thorough knowledge of the existing information concerning the toxicity of the chemical associated with the specific route of exposure, predicted intake, absorption, metabolism, excretion, tissue accumulation and species variation. Because of the limitations inherent in the risk assessment process it is important to recognize that the risk characterization described in this or any assessment cannot predict actual risk to a real person.

As defined by the MPCA, the term "risk" generally refers to estimated cancer risks (cancer risk estimate) and the potential for noncancer health effects. Noncancer health effects were assessed by calculating a Hazard Quotient (HQ) (for a single chemical) or a Hazard Index (HI) as the sum of HQs. Potential cancer risks are expressed as a cancer risk estimate or the probability of developing cancer if exposed to a specific potential carcinogen and specific exposure conditions over a lifetime (e.g. 1 excess cancer case in a million people or 1E-06 or 1 $x10^{-6}$).

6.1 Estimating Potential Noncancer Health Risks using the Hazard Index

In this screening HHRE, for each retained COI, a noncancer risk is calculated by taking the ratio of the estimated dose to the RfD for each chemical for evaluation. The resulting value is called the Hazard Quotient (HQ). The HQs for each chemical are then summed for all chemicals to calculate a Hazard Index

(HI). The MDH and U.S. EPA guideline value for comparison to estimated noncancer risks (HQ or HI) is one (1).

HQ = Dose / RfD

Where: Dose = the estimated amount in the body in mg/kg-day

RfD = the amount of a chemical that a person can consume over a lifetime without expected adverse health effects in mg/kg-day

$$HI = HQ_{\text{chemical 1}} + HQ_{\text{chemical 2}} + HQ_{\text{chemical n}}$$

To estimate potential health risk from exposure to all of the COIs (e.g. additive health effects), the hazard quotients for noncarcinogens were summed regardless of toxic endpoint, with the resulting Hazard Index (HI) reported. A hazard quotient of less than one indicates that there is no appreciable risk that noncancer health effects will occur. If the HQ exceeds 1, then there is some possibility that noncancer effects may occur. The likelihood of adverse effects is increased and more refined analyses are needed (although the magnitude of the uncertainty factors used to derive the RfD should also be considered). Because of the uncertainties involved with these estimates, values between 1 and 10 may be of concern, particularly when additional significant risk factors are present. However, because RfDs do not have equal accuracy or precision and they are not based on the same severity of toxic effects, evaluation of hazard indices (the sum of two or more hazard quotient values for multiple substances and/or multiple exposure pathways) should take into account the uncertainties associated with specific RfDs.

6.2 Estimating Potential Cancer Health Risks Using the Cancer Risk Estimate

Carcinogenic risk is expressed as a probability of developing cancer as a result of exposure to a specific potential carcinogen and specific exposure conditions. For example, a risk of one in 100,000 (1E-05 or $1x10^{-5}$) represents an upper-bound probability that an individual has a chance of one in one hundred thousand of developing cancer as a result of exposure to the specific chemical over an approximately 70-year lifetime and under specific exposure conditions.

Estimated sediment concentrations were used to estimate the daily intake averaged over 70 years (chronic daily intake) for each COI. The chronic daily intake was multiplied by the slope factor to estimate potential cancer risks to an individual. The MDH guideline for acceptable cancer risks is a risk level of 1 in 100,000 (1E-5).

Risk = Chronic Daily Intake x Slope factor

Where:

CDI = Chronic daily intake averaged over 70 years (mg/kg-day)

SF = Slope Factor expressed in $(mg/kg\text{-}day)^{-1}$

For carcinogens, with the assumption of linear dose-response relationship, the estimated increase in risk at an estimated exposure level is summed across all chemicals as an initial screening. For estimating cancer risk from exposure to multiple potential carcinogens with linear dose-response relationship, the estimated cancer risks for each chemical are summed as follows:

$$\text{Total Risk} = \text{Risk}_1 + \text{Risk}_2 + \ldots + \text{Risk}_n$$

where:

Risk_1 = Risk chemical 1

Risk_n = Risk for the n^{th} chemical

Carcinogenic substances with nonlinear modes of action through unrelated mechanisms or in different tissues should not be combined (U.S. EPA, 2005). However, for this screening HHRE, in accordance with MDH and MPCA policy, carcinogenic risk was assumed to be additive and the conservative assumption of additivity was carried through the risk calculation regardless of carcinogenic of mode of action (e.g. linear or nonlinear).

6.3 Summary of Estimated Potential Health Risks for COIs Using Site-Specific Reasonable Maximum Exposure Assumptions

The estimated potential health risks from dermal contact with sediment and incidental ingestion of impacted sediments are shown below in Tables K-14a and 14b. The potential health risks were estimated for children and adults as follows:

- Children from 6-16 years old
- Children from >16-18 years old
- Adults from >18-33 years old
- summed for ages 6-33 years old

The results can be summarized as follows:

- Using conservative reasonable maximum exposure assumptions, the potential risks for each age group for dermal contact and incidental ingestion of sediment from both Unnamed Creek and Wire Mill delta areas were below the MDH guidelines of 1 for noncancer effects and 1E-5 for cancer effects.
- Skin contact with sediment was assumed to occur while wading with sediment contact on the feet for all age groups. Any sediment that might get on the body was assumed to be washed off while swimming in water which covers the sediment which is consistent with USEPA guidance (USEPA,1989).
- Ingestion of sediment was assumed to occur while swimming using the maximum concentration of total suspended solids measured in the Unnamed Creek delta area. The amount of surface water swallowed while swimming was assumed to be 97th percentile values of volume of water swallowed, leading to high end risk estimates.

Table K-14a. Summary of Estimated Potential Health Risks from Exposure to Metals, PAHs, and 2,3,7,8-TCDD Equivalents at Unnamed Creek and Wire Mill Deltas by Dermal Contact with Sediment Using Site-Specific Reasonable Maximum Exposure Assumptions at the Spirit Lake Sediment Site, Duluth

Constituents of Interest Measured as part of the Remedial Investigation (COIs)	Potential Chronic Health Risks from Dermal Contact with Sediment			
	Noncancer Hazard Indices (compare to guideline of 1)		Cancer Risk Estimate (compare to MDH guideline of 1E-05)	
	Unnamed Creek	Wire Mill	Unnamed Creek	Wire Mill
Metals				
Ages 6-16 years	0.0003	0.0003	3E-08	4E-08
Ages 16-18 years	0.0002	0.0002	5E-09	6E-09
Total ages 6-18 years	0.0005	0.0005	3E-08	4E-08
Ages 18-33 years	0.0003	0.0003	4E-08	6E-08
PAHs				
Ages 6-16 years	0.003	0.0003	9E-07	1E-07
Ages 16-18 years	0.002	0.0003	1E-07	2E-08
Total ages 6-18 years	0.005	0.0006	1E-06	1E-07
Ages 18-33 years	0.003	0.0003	1E-06	1E-07

Constituents of Interest Measured as part of the Remedial Investigation (COIs)	Potential Chronic Health Risks from Dermal Contact with Sediment			
	Noncancer Hazard Indices (compare to guideline of 1)		Cancer Risk Estimate (compare to MDH guideline of 1E-05)	
	Unnamed Creek	Wire Mill	Unnamed Creek	Wire Mill
2,3,7,8-TCDD Equivalents				
Ages 6-16 years	0.0003	0002	4E-08	3E-07
Ages 16-18 years	0.0002	0.002	6E-09	4E-08
Total ages 6-18 years	0.0005	0.003	5E-08	3E-07
Ages 18-33 years	0.0003	0.002	6E-08	4E-07
TOTAL Potential Health Risks				
Ages 6-18 years	0.006	0.005	1E-06	5E-07
Ages 18-33 years	0.004	0.003	1E-06	6E-07
Ages 6-33 years	0.01	0.008	2E-06	1E-06

Unnamed Creek = Unnamed Creek delta area,
Wire Mill= Wire Mill delta area.

Table K-14b. Summary of Estimated Potential Health Risks from Exposure to Metals, PAHs, and 2,3,7,8-TCDD Equivalents at Unnamed Creek and Wire Mill Deltas for Incidental Ingestion of Sediment While Swimming Using Site Specific Reasonable Maximum Exposure Assumptions at the Spirit Lake Sediment Site, Duluth, Minnesota

Constituents of Interest Measured as part of the Remedial Investigation (COIs)	Potential Chronic Health Risks from Ingestion of Sediment			
	Noncancer Hazard Indices (compare to guideline of 1)		Cancer Risk Estimate (compare to MDH guideline of 1E-05)	
	UC	WM	UC	WM
Metals				
Ages 6-16 years	0.0003	0.0003	4E-08	6E-08
Ages 16-18 years	0.0002	0.0002	5E-09	7E-09
Total ages 6-18 years	0.0005	0.0005	4E-08	6E-08
Ages 18-33 years	0.0001	0.00009	2E-08	3E-08

Constituents of Interest Measured as part of the Remedial Investigation (COIs)	Potential Chronic Health Risks from Ingestion of Sediment			
	Noncancer Hazard Indices (compare to guideline of 1)		Cancer Risk Estimate (compare to MDH guideline of 1E-05)	
	UC	WM	UC	WM
PAHs				
Ages 6-16 years	0.0006	0.00006	2E-07	2E-08
Ages 16-18 years	0.0004	0.00004	2E-08	2E-09
Total ages 6-18 years	0.0009	0.0001	2E-07	2E-08
Ages 18-33 years	0.0002	0.00002	7E-08	8E-09
2,3,7,8-TCDD Equivalents				
Ages 6-16 years	0.0003	0.002	5E-08	3E-07
Ages 16-18 years	0.0002	0.001	6E-09	4E-08
Total ages 6-18 years	0.0006	0.004	5E-08	4E-07
Ages 18-33 years	0.0001	0.0008	2E-08	2E-07
TOTAL Potential Health Risks				
Ages 6-18 years	0.002	0.004	3E-07	4E-07
Ages 18-33 years	0.0004	0.0009	1E-07	2E-07
Ages 6-33 years	0.002	0.005	4E-07	6E-07

UC = Unnamed Creek delta area, WM = Wire Mill delta area.

6.4 Summary of Additivity of Potential Health Risks

The HQs for noncancer effects for each chemical were summed to generate HIs to estimate the total potential for noncancer health effects. Cancer risk estimates were summed to assess total potential cancer health risk. Using conservative reasonable maximum exposure assumptions, the summed potential health risks, for all chemicals assessed, for dermal contact with sediment and incidental ingestion of sediment from both Unnamed Creek and the Wire Mill delta areas were below the MDH guidelines of 1 for noncancer effects and 1E-05 for cancer effects. The cancer risk estimates were summed regardless of the carcinogenic mode of action of each chemical or an assessment of the confidence in the cancer classification.

Results for dermal contact if wading 8 times a year from ages 6-33 years old:

- The summed HI for the Unnamed Creek delta for dermal contact with sediments was 0.01 which is well below the guideline value of 1.
- The summed HI for the Wire Mill delta for dermal contact with sediments was 0.008 which is well below the guideline value of 1.
- The summed potential cancer risk estimate for the Unnamed Creek delta area for dermal contact with sediments was 2E-06 which is well below the MDH guideline of 1E-05.
- The summed potential cancer risk estimate for the Wire Mill delta area for dermal contact with sediment was 1E-06 which is well below the MDH guideline of 1E-05.

Results for incidental ingestion of sediment in surface water based on amounts of water swallowed while swimming 8 times a year from ages 6-33 years old:

- The summed HI for the Unnamed Creek delta for incidental ingestion of sediments was 0.002 which is well below the guideline value of 1.
- The summed HI for the Wire Mill delta for incidental ingestion of sediments was 0.005 which is well below the guideline value of 1.
- The summed potential cancer risk estimate for the Unnamed Creek delta area for incidental ingestion of sediments was 4E-07 which is well below the MDH guideline of 1E-05.
- The summed potential cancer risk estimate for the Wire Mill delta area for incidental ingestion of sediment was 6E-07 which is well below the MDH guideline of 1E-05.

Results for summed potential health risks at Unnamed Creek from both dermal contact and incidental ingestion of sediments from ages 6-33 years old:

- The summed HIs for both dermal contact and incidental ingestion are 0.01 which is well below the MDH guideline of 1.
- The summed cancer risk estimates for both dermal contact and incidental ingestion are 2E-06 which is well below the MDH guideline of 1E-05.

Results for summed potential health risks at Wire Mill from both dermal contact and incidental ingestion of sediments from ages 6-33 years old:

- The summed HIs for both dermal contact and incidental ingestion are 0.01 which is well below the MDH guideline of 1.
- The summed cancer risk estimates for both dermal contact and incidental ingestion are 2E-06 which is well below the MDH guideline of 1E-05.

In summary, given the conservative exposure assumptions used in the screening HHRE, the conservative assumptions regarding the amount of surface water containing sediment swallowed while swimming, the surface area of skin in contact with sediment, and conservative assumptions in the derivation of the toxicity values, adverse health impacts from chronic dermal exposure and/or incidental ingestion of sediment at the Spirit Lake Sediment Site are not expected.

7.0 Discussion

Potential health risk at the Spirit Lake Sediment site was assessed in this screening HHRE by following the five steps below:

Step 1: Identifying and eliminating potential constituents of interest (COIs).

Step 2: Calculating mean sediment concentrations for remaining COIs for comparison to draft MDH SSVs (MDH, 2005).

Step 3: Identifying potentially complete exposure pathways and calculating site-specific potential daily intake.

Step 4: Compiling toxicity values (e.g. reference doses and/slope factors) to estimate potential human health risks.

Step 5: Combining exposure and toxicity information to calculate HIs to assess potential noncancer effects and cancer risk estimates to assess potential cancer health risk.

The summed potential health risks from incidental ingestion of sediment and/or dermal contact with sediment from both Unnamed Creek and Wire Mill delta areas were below the MDH guidelines of 1 for noncancer effects and 1E-5 for cancer effects for all age groups.

Estimation of potential health risks from exposure to surface water by incidental ingestion or dermal contact was not possible given the data limitations outlined in Section 3.4. Qualitatively, however, adverse health impacts from dermal contact with surface water or incidental ingestion of surface water are not expected based on the physical and chemical properties and solubility of metals, PAHs and dioxin/furans.

8.0 Summary and Conclusions

Based on reasonable maximum exposure assumptions, the summed potential health risks for dermal exposure and/or incidental ingestion of sediments for both potential noncancer and cancer effects at the Spirit Lake Sediment site were below the MDH guidelines. Therefore adverse health effects from exposure to sediment by incidental ingestion and dermal contact are not expected.

Institutional controls, such as restrictions on recreational activities (i.e., prohibition against swimming, wading, and fishing – 33 CFR §165.905) and MDH fish consumption advisories are in place. These use restrictions and notice of the fish consumption are posted around the site. Because of posted rules and guidelines that are in place, human exposures to COIs in sediment, surface water, and fish at this this Site are expected to be negligible.

While institutional controls limit the potential for human exposures, it is generally understood that not all potential exposures are eliminated. Anecdotally, people use Morgan Park, areas north of the Unnamed Creek and Wire Mill deltas, and Spirit Lake for wading, fishing and swimming. Although fishing has been occasionally observed by Barr staff at this site, swimming and wading have not been observed (observations of Barr staff, 2011-2012). However, to be conservative, the potential incremental health risks from these activities were evaluated and, as discussed, are not expected.

Exposure to COIs in surface water through incidental ingestion of surface water and dermal contact with surface water could not be quantitatively evaluated, but were evaluated qualitatively. The potential health risk from incidental ingestion of surface water and dermal contact with surface water were considered negligible for the following reasons:

- COI concentrations in surface water were not expected to be significant due to their low water solubility and the tendency of PAHs and dioxins to adhere to organic carbon particles and deposit in the sediment.
- Using sediment as a surrogate for surface water, potential health risks from ingestion of sediment for cancer and noncancer effects were below MDH guidelines. This qualitative extrapolation to

surface water is conservative because potential health risks from ingestion of sediment is likely to be higher than potential health risks from incidental ingestion of surface water based on the physical and chemical properties and solubilities of the COIs. Thus, since potential ingestion of sediment does not pose an unacceptable risk, neither does incidental ingestion of surface water.

- The potential health risk from dermal contact with surface water is likely to be negligible based on limited absorption of COIs through the skin.
- There would be greater absorption of chemicals from the gastrointestinal tract from ingestion than there would be from dermal absorption through the skin (Crane 1992; U.S. EPA, 1993). Because incidental ingestion of surface water does not pose an unacceptable risk, neither does dermal contact with surface water.

Uptake of COIs in fish could not be quantitatively evaluated. However, while recreational fishing in the areas considered in this screening HHRE may occasionally occur, exposure through this pathway is unlikely to be significant for the following reasons:

- Based on anecdotal information, recreational fishing occurs only sporadically at this site.
- Fish species of typical interest to fisher persons are mobile and no single fish is likely to reside for its entire life exclusively within the Unnamed Creek and Wire Mill delta areas.
- MDH has issued fish consumption advisories for Spirit Lake based on concentrations of mercury and PCBs, which should limit fish consumption. MDH concluded that "In the absence of site-specific information about PAHs in fish tissue, fish consumption advice for mercury and PCBs should be used" (MDH, 2005). Following the fish consumption advisories for PCBs and/or mercury also limits exposure to other COIs (e.g. TCDD Equivalents, PAHs).

When taking under consideration the conservative RME assumptions regarding incidental ingestion of sediment and dermal contact with sediment, the conservative assumptions in the derivation of the toxicity values, the institutional controls in place to limit exposure to COIs associated with the Spirit Lake Sediment Site, and that all of the individual and summed potential health risks for dermal contact and/or incidental ingestion at both Unnamed Creek and Wire Mill delta areas were below the MDH guideline values, exposure to COIs present in sediments by incidental ingestion or dermal contact are not expected to pose a risk to public health.

9.0 Uncertainty and Variability in the Human Health Risk Evaluation

The risk assessment process is subject to uncertainty and variability from a variety of sources. These are inherent in the risk assessment process and are not unique to this screening HHRE. Uncertainties represent incomplete knowledge about certain parameters such as environmental sample collection and analyses at the beginning of the process through exposure estimation and toxicity assessment through the final step of risk characterization. Variability, on the other hand, represents true heterogeneity and inherent differences within a population, across geographic regions, and throughout a given time period. It is important to emphasize that the estimated risks presented in this screening HHRE should not be interpreted as estimates of actual health risks to a specific person. The risk estimates presented in this screening HHRE are conditional estimates of risk that depend on the assumptions involved in the assessments of exposure to and toxicity of the constituents of interest.

9.1 Uncertainty in Exposure Point Concentrations

Estimates of exposure depend on the quality and quantity of the environmental data. The exposure point concentrations used in this screening HHRE were based on analytical data presented in Section 3.0. For both metals and 2,3,7,8-TCDD Equivalents, the 95UCL of the mean concentration as recommended by ProUCL was used in screening HHRE calculations to estimate exposure point concentrations. For PAHs, the arithmetic mean of the detected values was used to estimate exposure point concentrations.

9.2 Uncertainty in Exposure and Dose

This screening HHRE used generally accepted generic equations to calculate potential chemical exposure to a hypothetical receptor through incidental ingestion and dermal contact with sediments. Most of the exposure parameters chosen were based on RME (greater than 95th percentile value) exposure assumptions.

9.3 Uncertainty in Toxicity Values

A significant source of uncertainty is inherent in the derivation of U.S. EPA, MDH, ATSDR, and Cal PA-OEHHA toxicity values (i.e., reference doses and slope factors). Adequate data reflecting human exposure to low levels of environmental chemicals are generally not available. Data that are available for human exposures are usually based on exposures in the workplace, where concentrations are generally higher than those encountered in the environment. Because of the lack of human data, toxicity values are derived from studies with laboratory animals. To apply data derived from animal studies to humans, extrapolation factors are used. In developing these dose-response values, U.S. EPA currently uses

conservative assumptions to assure that the toxicity value is conservative and that the resultant risk estimate is more likely to overestimate risk than underestimate risk. Additional discussion of the uncertainty in the toxicity values can be found in Attachment 8.

9.4 Uncertainty in Risk Characterization

To develop a cancer risk estimate associated with exposure to multiple chemicals identified by U.S. EPA as carcinogens, the chemical specific cancer risk estimates were summed in accordance with MPCA and U.S. EPA guidance. U.S. EPA recognizes that there are several limitations associated with this approach. For chemicals where the unit risk is based on the upper 95th percentile of the probability distribution, addition of these percentiles may become progressively more conservative as risks from a number of carcinogens are summed (U.S. EPA, 1989). A description of the uncertainty associated with the screening HHRE is in Table K-15.

Table K-15. Summary of Sources of Uncertainty in the Screening Human Health Risk Evaluation (HHRE) for the Spirit Lake Sediment Site, Duluth, Minnesota

Assessment Component		Comment	Effect on Risk Estimate	Overall Impact
Basis of Constituent Selection	Constituents included in the screening HHRE	Many COIs were included in the screening HHRE calculations even though their estimated concentrations were below the draft SSVs.	Overestimates potential risk	Moderate
		Some of the COIs (e.g., acenaphthalene, benzo (g,h,i) perylene, perylene, and phenanthrene) were not included in the screening HHRE because they lacked toxicity values.	May underestimate potential risk	Low
Exposure Assessment	Exposure point concentrations in sediment	For metals and TCDD Equivalents, the 95 percent UCL as recommended by ProUCL was used to estimate exposure point concentrations.	May overestimate potential risk	Moderate
	Exposure point concentrations in sediment	For PAHs, the arithmetic mean of the detected values was used to estimate exposure point concentrations.	May underestimate potential risk	Low

Assessment Component		Comment	Effect on Risk Estimate	Overall Impact
	Exposure point concentrations in surface water	Inadequate surface water concentration data to assess ingestion and dermal exposure	May underestimate potential risk	Low (because a relevant surrogate was used to qualitatively evaluate this potential pathway)
	Exposure point concentrations via fish consumption	Unable to estimate potential exposure to COIs via fish consumption because of inadequate fish concentrations. Potential exposure of fish to Site sediments and/or contaminants is uncertain but likely low and infrequent because of the mobile nature of fish. Consequently, the delta areas are likely to be used periodically by fish but are unlikely to concentrate or hold fish for extended lengths of time.	May underestimate potential risk	Likely low
	Exposure parameters	Assumed that an individual would wade at the site yearly over a 27 year period. This assumed exposure duration is highly unlikely to occur at this Site.	Overestimates potential risk	High
	Exposure parameters	Assumed that a person would swallow some sediment in surface water while wading-using the 97th percentile value for swimming to be conservative (USEPA, 2004). Ingestion of sediments is generally not a relevant pathway for industrial/commercial land use (USEPA, 1989).	Overestimates potential health risk	Moderate
	Exposure parameters	Assumed that sediment that gets on skin would be washed off while swimming which is consistent with USEPA guidance (USEPA, 2004).	Likely no effect on risk estimate	Likely no effect on risk estimate
	Exposure parameters	The maximum detected total suspended sediment concentration in the water column at Unnamed Creek was used to calculated potential daily intake of sediment. Average concentrations of total suspended solids are significantly lower.	Overestimates potential risk	High

Assessment Component		Comment	Effect on Risk Estimate	Overall Impact
Toxicity Assessment	Chronic toxicity Values	Toxicity values primarily derived from animal studies. Use of data from most sensitive species/strain/sex. Use of data solely from positive studies.	May overestimate potential risk	Moderate
		Incorporation of uncertainty factors, modifying factors and safety factors.	May overestimate potential risk	Moderate
		Use of draft MDH Sediment Screening Values to compare concentrations	May overestimate potential risk	Low
		Toxicity values derived primarily from high doses while most exposures are at low doses.	May overestimate potential risk	Moderate
		The oral RfD used in the calculation of potential noncancer health effects for exposure to benzo(a)pyrene is based on the MDH value used to derive a Health Based Value, and hasn't been used in rulemaking.	May overestimate potential risk	Low
		Toxicity values may not incorporate all possible endpoints.	May underestimate potential risk	Moderate
	Cancer toxicity Values	Use of cancer unit risk/slope factors which are generally upper 95th percent confidence limits derived from the linearized model. General assumption of dose/effect linearity.	Overestimates potential risk	Moderate
		Cancer unit risks/slope factors primarily derived from animal studies. Use of data from most sensitive species/strain/sex. Use of data solely from positive studies.	May overestimate potential risk	Moderate
	Cancer toxicity values	Toxicity values were derived for individual PAHs by extrapolation and are highly uncertain	Overestimates potential risk	High
		The slope factor used for benzo(a)pyrene and benzo(a)pyrene equivalents was age adjusted. So the cancer risk estimates for the 18-33 year olds, along with the cancer risk estimates for the 6-16 year olds, were adjusted for childhood exposure. Potential childhood risk is accounted for in both child the adult risk calculations.	Overestimates potential risk	Moderate
Risk Characterization		Assumed that the chemicals are in the same form as the chemicals upon which the toxicity values are based.	May overestimate potential risk	Moderate
		Assumed that all chemicals have an additive	Overestimates	Moderate

Assessment Component		Comment	Effect on Risk Estimate	Overall Impact
		effect	potential risk	
		Assumed that all noncarcinogenic toxicity values have the same level of accuracy and precision and severity of toxic effects.	Likely overestimates potential risk	Moderate
		Assumed that all carcinogenic unit risks have the same weight of evidence for human carcinogenicity.	Overestimates potential risk	High
		Chemicals without toxicity values could not be directly evaluated.	Underestimates potential risk	Low
		Risks to especially sensitive receptors was not specifically evaluated for each COI.	May underestimate potential risk	Low
		Synergism/antagonism was not considered	May under- or overestimate potential risk	Moderate
		The oral slope factor for benzo(a)pyrene and equivalents was age adjusted to account for early life exposure – even for adults.	Overestimates potential risk	Moderate
		Potential ingestion risks were based on ingestion of surface water at exposure rates for the 97th percentile of the population for a person while swimming.	Overestimates potential risk	High

(1) Key for Effects Determination:

- ► Overestimates potential risk: A value or assumption intentionally chosen to provide high risk estimates
- ► Likely Overestimates potential risk: A value or assumption intentionally chosen that is expected to provide high risk estimates
- ► May overestimate potential risk: A value or assumption that has some level of scientific uncertainty which may lead to a high risk estimate
- ► Underestimates potential risk: A gap in information or an available value that is known to provide a low risk estimate
- ► Likely underestimates potential risk: A gap in information or an available value that may provide a low risk estimate
- ► May underestimate potential risk: A value or assumption that has some level of scientific uncertainty which may lead to a low risk estimate.
- ► Likely no effect on estimated risk: Value or assumption that is known or suspected to have very little, if any, effect on potential risk

9.5 Variability in the Human Health Risk Evaluation

Table K-16. Summary of Sources of Variability in the Parameters used for the Screening Human Health Risk Evaluation (HHRE) for the Spirit Lake Sediment Site, Duluth, Minnesota

Source of Variability	Comments	Impact of Risk Analysis
Differences in human susceptibility to actual COI exposure and actual exposure durations.	Toxicity values are developed to be conservative and protective of sensitive populations. Actual exposures are likely lower than the potential exposures evaluated in this screening HHRE and that is why these risk results from this screening HHRE, or any risk assessment, cannot be used as an indicator of actual risk to any receptor.	Likely small
Sediment concentrations may vary seasonally and from core location to core location.	The sediment concentrations may vary seasonally and with core location. However, the 95 UCL concentrations were used metals and TCDD Equivalents which may overestimate potential risks.	Likely small.
Sediment adherence can vary with moisture, particle size and activity.	Dermal adherence of sediments is correlated with sediment moisture and inversely correlated with particle size, as many COIs are concentrated within small sediment particles.	Likely small

10.0 Glossary of Terms Used in this Assessment

Term	Definition
Acute exposure	Single or multiple exposure occurring within 24 hours or less.
Acute toxicity	Adverse health effects that occur or develop rapidly after a single administration of a chemical.
Additivity	Refers to a situation where the combined effect of exposure to two or more chemicals is equal to the sum of the effect of each of those chemicals given alone.
Antagonism/Antagonistic	Description of two or more chemicals which when given together interfere with each other's actions.
Applied dose	Amount of chemical, which reaches an exposed individual. For purposes of this Assessment, it is equivalent to exposure and is a function of chemical concentration, frequency and duration of exposure.
Benchmark Dose Method	An alternative method to the NOAEL/LOAEL approach that has been used in dose response assessment. A benchmark dose is an adverse effect, used to define a benchmark dose from which and RfD (or RfC) can be developed.
Bioaccessible	A value representing the availability of a chemical for absorption based on studies outside of living systems (i.e. animal studies or observations in humans) when dissolved in *in vitro* surrogates of body fluids or juices (add ref.)
Bioavailable	The fraction of a dose that becomes available for distribution to internal target tissues and organs.
Bioaccumulation	A general term that describes the process by which chemicals are taken up by an organism either directly from exposure to a contaminated medium or by consumption of food containing the chemical (U.S. EPA 2012). Bioaccumulation includes both bioconcentration and biomagnification.
Bioconcentration	The accumulation of chemicals in the tissues of an organism resulting from exposure to the surrounding environment but excluding exposure from dietary intake.
Bioconcentration Factor (BCF)	The ratio of a contaminant concentration in biota to its concentration in the surrounding medium (e.g. water).
Biomagnification	An increase in concentration of a substance that occurs in a food change.
Biota-Sediment Accumulation Factors (BSAFs)	$C_O/f_l/C_S/f_{soc}$ C_O=concentration of a chemical in an organism (µg/kg wet weight) f_l = lipid fraction in the organism in g liquid/g wet weight C_s = concentration in surficial sediment (µg/kg dry weight) f_{soc} = fraction of the sediments as organic carbon (g organic carbon/g dry weight

Term	Definition
	http://nepis.epa.gov/Adobe/PDF/P100E2O5.pdf
Carcinogen	A chemical that is capable of causing cancer in mammals. For purposes of this screening HHRE a carcinogen is a chemical that is defined by the U.S. EPA as a carcinogen.
Chronic exposure	Prolonged or repeated exposure typically occurring over a period of several years. The COIs at this site were assessed mainly for chronic exposure.
Chronic toxicity	Adverse health effects that occur after a lapse of time between the initial exposure, or effects that persist over a long period of time whether or not they occurred immediately or are delayed.
Constituents of Interest (COI)	Chemicals measured at the site and analyzed for potential health risks either quantitatively or qualitatively based on sediment and surface water data from previous site activities.
Cytogenetic	Refers to effect of chemical on genetic material, studied on cellular level.
Dermal Absorption Factors	
Dosimetric methods	Corrections for differences in body weight, surface area and metabolic rate applied to dosage. Usually these corrections are made when comparing animal exposure data to human exposure data.
Epidemiological	Refers to the study of disease and its spread in people.
Genotoxic	Substance that can cause damage to cellular DNA.
Hazard Index (HI)	The sum of HQs for non-carcinogenic chemicals with similar modes of action and toxic endpoints. A HI of one or more indicates that there is a potential for adverse health effects.
Hazard Quotient (HQ)	The calculated or measured exposure to a given chemical divided by the RfD for that chemical. An HQ of one or greater indicates that there is a potential for adverse noncancer health effects.
Health Risk Value (HRV)	A Health Risk Value is the concentration of a chemical (or defined mixture of chemicals) defined by the Minnesota Department of Health (MDH) that is likely to pose little or no risk to human health. For carcinogens, MDH defines significant risk as a risk of 1 in 100,000. For noncarcinogens, MDH defines significant risk as a Hazard Index greater than 1 (for an individual chemical) or a Hazard Quotient greater than 1 (for a mixture of chemicals.
Human equivalent concentration (HEC)	Exposure concentration for humans that has been adjusted for dose related differences (such as metabolic rate and surface area to body weight ratio) between experimental animal species and humans to be equivalent to the exposure concentration associated with observed effects in the experimental animal species.
Internal dose	The dose of a chemical that reaches and is absorbed by internal organs.
In vitro	Observable in an artificial environment (e.g. in glass or a test tube)

Term	Definition
In vivo	Within a living body
Lowest observed adverse effect level (LOAEL)	The lowest exposure level at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its control group.
No observed adverse effect level (NOAEL)	The highest dose level for a given chemical that does not produce a significant elevated increase in adverse response. Significance is defined both biologically and statistically and depends in part on the number of doses tested, the number of animals tested, the background level of the adverse effect in the test population, and the sensitivity of the measurement methods. NOAELs should not be viewed as "no risk levels".
Noncarcinogen	For the purposes of this risk assessment, a noncarcinogen is a chemical, which is not included on the U.S. EPA list of carcinogens.
Octanol-water partition co-efficient (Kow)	"An organic compound's octanol-water partition coefficient, K_{OW}, is defined as the ratio of the compound's concentration in a known volume of n-octanol to its concentration in a known volume of water after the octanol and water have reached equilibrium ... Water solubility was found to be the major factor affecting the partition coefficient" (Smith, 1988).
One-hit model/equation	A dose-response model based upon the concept that a tumor can be induced after a single susceptible target or receptor has been exposed to a single effective dose unit of a substance.
Oral Absorption Fraction	See bioavailability
Pharmacokinetic	Refers to the modeling and mathematical description of the distribution of chemicals over time in a whole organism based upon the pharmacological (what is known about the chemical and biological behavior) characteristics of the chemical.
Reference dose (RfD)	An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous ingestion exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of adverse noncancer effects during a lifetime.
Reference Exposure Level (REL)	RELs are derived for the California Hot Spots program (by the Office of Environmental Health Hazard Assessment-OEHHA) in a manner similar to U.S. EPA values and have undergone internal and external review. An REL represents an airborne concentration of a chemical at or below which no adverse effects are anticipated in individuals exposed to that level. RELs can apply to exposures for 1 hour, 8 hours, or up to a lifetime. http://oehha.ca.gov/air/allrels.html
Reasonable Maximum Exposure (RME)	Reasonable maximum exposure (RME) assumptions - is defined as "the highest exposure that is reasonably expected to occur at a site, and is intended to estimate a conservative case (i.e. well above the average case) that is still within the range of possible exposures" (U.S. EPA, 1989).

Term	Definition
Semi-volatile organic compound (SVOC)	Organic compounds which may be present in both vapor and particulate phase within the atmosphere. These compounds tend to evaporate very slowly at normal temperatures and can be very persistent in the environment. SVOCs have vapor pressures ranging from 10^{-1} to 10^{-7} mmHg and boiling points that range from 240 to 400^{o}C (http://www.epa.gov/iaq/voc2.html). As used in this U.S. Steel Report, SVOCS refers to chemicals as analyzed by as defined EPA Method SW-846 Base, Neutral and Acid Analyses. Additionally, in terms of health risks, SVOCs were considered the noncarcinogenic PAHs as defined by the MPCA in their document "Remediation Division Policy on Analysis of Carcinogenic Polynuclear Aromatic Hydrocarbons (cPAH). http://www.pca.state.mn.us/index.php/view-document.html?gid=16052
Slope factor	An upper bound, approximating 95% confidence limit, on the increased cancer risk from a lifetime oral exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg-day, is generally reserved for use in the low-dose region of the dose-response relationship, that is for exposures corresponding to risks less than 1 in 100.
Synergistic	The combined effect of two or more chemicals given together is greater than the sum of the effects of those chemicals (e.g. 2+2=10).
Toxicity	Measure or degree of adverse effect of a given chemical on a living organism. In the case of this risk assessment – humans.
Toxicity factor	Can refer to a toxicity value used to calculate a risk estimate (e.g. slope factor, unit risk, RfC, RfD, etc.)
Unit risk (UR)	The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m^3 in air. The interpretation of unit risk would be as follows: if unit risk = 2 x10^{-6} per µg/L, 2 excess cancer cases (upper bound estimate) are expected to develop per 1,000,000 people if exposed daily for a lifetime to 1 µg/of the chemical per liter of drinking water.
Weight-of-evidence	Procedure for evaluating the toxicity, and in particular the carcinogenicity of a chemical using evidence from human (epidemiological) studies, and animal studies. Studies are weighted based upon their relevance to human exposure, and assessed quality of the study. Well-designed studies are given greater weight in the consideration of toxicity than poorly designed studies. Similarly human studies are given greater weight than animal studies.

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List of Tables

Attachments

Attachment 1: Exposure Values Used in this Screening HHRE Using Site-Specific Reasonable Maximum Exposure Assumptions

Parameter	Ages 6-16 years old	Ages 16-18 years old	Ages 18-33 years old
	Site-Specific Reasonable Maximum Exposure	Site-Specific Reasonable Maximum Exposure	Site-Specific Reasonable Maximum Exposure
Skin Surface area exposed (cm^2) (1)	1360	1420	1610
Skin adherence factor (unitless) (2)	0.52	0.62	0.80
Sediment ingestion rate (mg/day) (3)	51.2	51.2	30.2
Fraction Ingested (unitless) (4)	1	1	1
Exposure frequency (days or events/year) (5)	8	8	8
Exposure duration (years) (4)	10	2	15
Body Weight (kg) (4)	39	60	70
Averaging Time (days) (4)	3650	730	5475

(1)-Skin surface area (cm^2) was based on the 95th percentile values feet for all ages (USEPA, 2011).
(2) Skin adherence factors are from MDH, 2005.
(3) Sediment ingestion rate for 6-18 year olds was calculated taking 427 mg/L (maximum total suspended particulate concentration at Unnamed Creek) x 0.120 L/hour (97th percentile amount of water swallowed per hour as per USEPA,2011) 1 hour/event (site specific assumption) x 1 event/day (site specific assumption)=51.2 mg/day. Sediment ingestion rate for 18-33 year olds was calculated by taking 427 mg/L x 0.071 L/hour (97th percentile amount swallowed when swimming by an adult in a pool as per USEPA, 2011) x 1 hour/event x 1 event per day.
(4) Fraction ingestion, body weight and averaging times were from MDH, 2005.
(5) Exposure frequency was based on site-specific observations

Attachment 2: Oral Absorption Fractions and Dermal Absorption Factors used in this Screening HHRE

Oral Absorption Factors and Dermal Absorption Factors Used in the Spirit Lake Sediment Site Screening HHRE, Duluth, Minnesota

Chemical Name	Oral Absorption Factor	Source	Dermal Absorption Factor	Source
Metals				
Arsenic	0.41	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Arsenic_ragsa.html	0.03	U.S. EPA, 2004, exhibit 3-4 ;
Copper	0.3	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/copper_ragsa.html	0.01	Technical Guidance from Region 3: http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
Lead	0.15	Unknown	0.01	Technical Guidance from Region 3 ;http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
Nickel	0.27	Available: risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/nickel_and_nickel_compounds_ragsa.html	0.01	Technical Guidance from Region 3: http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
Zinc	0.2	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/zn_ragsa.html	0.01	Technical Guidance from Region 3 http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm

Chemical Name	Oral Absorption Factor	Source	Dermal Absorption Factor	Source
PAHs				
2-Methylnaphthalene (SVOC)	0.8		0.1	U.S. EPA, 2004, exhibit 3-4, and http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
Acenaphthene (SVOC)	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4, and http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
Anthracene (SVOC)	0.76	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Anthracene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4
Benzo(a)anthracene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4
Benzo(a)pyrene	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4;
Benzo(b)fluoranthene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4
Benzo(k)fluoranthene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4
Chrysene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4

Chemical Name	Oral Absorption Factor	Source	Dermal Absorption Factor	Source
Dibenz[a,h]anthracene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.13	U.S. EPA, 2004, exhibit 3-4
Fluoranthene (SVOC)	0.31	Risk Assessment Information System: http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4
Fluorene (SVOC)	0.5		0.1	U.S. EPA, 2004, exhibit 3-4;
Indeno (1,2,3-cd)pyrene[(2)]	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4
Naphthalene (SVOC)	0.8	Risk Assessment Information System (RAIS): ornl.gov/tox/profiles/naphthalene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4
Pyrene (SVOC)	0.31	Risk Assessment Information System (RAIS): http://rais.ornl.gov/tox/profiles/Benzoapyrene_ragsa.html	0.1	U.S. EPA, 2004, exhibit 3-4 ;, and http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm
TCDD Equivalents	0.5		0.03	U.S. EPA, 2004, exhibit 3-4 ;, and http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm

Attachment 3: Discussion of Dermal Absorption and Dermal Permeability

Dermal Absorption

There are significant uncertainties associated with estimating potential COI exposure via the dermal exposure pathway. The most significant of these uncertainties are associated with determining the impact of sediment characteristics and the extent of exposure (e.g., the amount of sediment on the skin and the length of exposure) on estimating compound-specific absorption fractions (ABS).

The human skin is the largest organ of the body. It consists of a thin (approximately 100 μm) epidermal layer superimposed on a thick dermal layer (approximately 300-400 μm). The epidermis consists of four layers, the outermost layer being the stratum corneum (SC) (approximately 10-40 μm), which overlays the strata lucidum, granulosum, and germinativum. The SC layer is composed of flat highly keratinized squamous cells that are nonviable. This layer is highly hydrophobic and provides the protective barrier function of skin. If the SC layer is removed the permeability of the skin to chemicals increases dramatically.

The uptake of chemicals through these two skin layers is controlled by diffusion. There are no active transport mechanisms. Chemicals deposited on the outside of the skin set up a concentration gradient between the outer skin concentration and the concentration within the dermis. This gradient produces a mass transfer that is dependent on the physical properties of the skin at that site and also the chemical properties of the substance. Diffusion across the complex membrane of the skin is thought to be regulated by Fick's law, which states that the rate of diffusion across a barrier will be directly proportional to the concentration gradient. The driving force for dermal uptake is similarly the concentration of the substance on the skin surface (mg/cm^2).

The passage of a chemical through the skin barrier is dependent on many factors. The skin is not uniform in terms of thickness, epidermis to dermis ratio, density of hair follicles, and many other parameters that will affect permeability. The amount of material that may be absorbed will, as a consequence, vary depending on the anatomical site of the exposure. For example, exposure to more permeable sites such as scrotal skin can result in uptake some 50 times greater than the same exposure applied to the thicker, less permeable skin of the legs and abdomen. Other factors that can play an important role in determining the degree of uptake include temperature and the presence of other materials on the skin.

Dermal Permeability Coefficient (K_p)

Many studies focus on the quantity of material deposited on the skin as the factor regulating dermal uptake. This measurement is a skin loading (mg/cm^2) and is not a concentration, and while it is true that dermal absorption cannot physically exceed the mass of material on the skin, it is the concentration of the substance that drives the diffusive process. The fact that the flux through the skin is determined not by the mass, but by the concentration of material on the skin, is described in work by Cherrie and Robertson (1995). The transfer of a chemical substance through the skin can therefore be defined by two measurements. The lag-time is the time taken from initial contact with the skin until the material enters the blood supply, while the flux is the steady state diffusion rate of the material when the lag-time is complete. The flux (J) is measured in units of mass per unit area per time period ($mg/cm^2/h$). The flux is directly proportional to the concentration gradient and the rate is regulated by the chemical specific permeability constant (K_p).

The skin's physico-chemical characteristics, as described above, determine the limits of percutaneous absorption of chemicals from impacted water. As a vehicle, water hydrates the skin, which may itself enhance absorption through the skin. Clearly, the aqueous solubility of a constituent sets the upper limit on the obtainable driving force for its diffusion, and thus sets the upper limits on both the absorption rate and dose. Relative solubility (partitioning) of the constituents between water and skin, and between the skin and the systemic circulation, also governs the overall absorption of the chemicals into the body, as partitioning sets the steepness of the concentration gradients across critical tissues. Other physico-chemical attributes of constituents define their interactions with the various skin components, thereby determining the ease of diffusion of solutes through the skin's barrier phases. Typically the rate of solute penetration from water through skin is proportional to the solute's concentration in water (C_w) as long as C_w is less than the solute's solubility in water (S_w). The steady-state flux across the skin from water ($J_{ss,w}$) is often described by the product of C_w and the permeability coefficient for dermal absorption from water ($K_{p,w}$). In other words the $K_{p/w}$ is a flux value, normalized for concentration, that represents the rate at which the constituent penetrates the skin in units of centimeter per hour (cm/hr). For a molecule to pass from one side of the membrane to the other it must partition into the membrane and then migrate across the full thickness of the membrane. Therefore the $K_{p/w}$ is a function of the path length of chemical diffusion, the membrane/vehicle (water) partition coefficient of the constituent and the diffusion coefficient of the constituent in the membrane.

The permeability coefficient of a constituent in water ($K_{p,w}$) is affected by both the lipophilic character and size of the penetrating solute, which are frequently approximated by the octanol-water partition coefficient (K_{ow}) and molecular weight (MW). Several structure-activity relationships for estimating $K_{p,w}$ have been derived using K_{ow} and MW. In their critical review of permeability coefficient data measured *in vitro* in human skin from water, Vecchia and Bunge (2002) concluded that the Potts and Guy (1992) equation provided reasonable estimates of the existing data. However, because there are almost no data for compounds with $\log K_{ow} < -1$ or $\log K_{ow} > 4$, using the Potts and Guy equation or any other structure-activity relationship beyond these limits is unsupported by data and may produce estimates that are incorrect.

There are many experimental challenges that make reliable and reproducible determination of $K_{p,w}$ difficult for compounds with $\log K_{ow} > 4$ (TCDD log K_{ow} = 6.8) . Basically, measuring steady-state flux of a lipophilic solute from water is difficult because its water solubility is low, its absorption rate into the lipophilic stratum corneum is relatively large, and its low solubility in the more hydrophilic viable epidermis limits mass transfer from the stratum corneum into these tissues. The difficulty in measuring $K_{p,w}$ increases as log K_{ow} increases and S_w (solubility in water) decreases (Romonchuck et al, 2005).

An alternative interpretation for the limiting permeability of the skin is that, for compounds of very high lipophilicity, the transport out of the stratum corneum (rather than transport through the stratum corneum) is the rate-determining step in the overall penetration process (Guy and Hadgraft, 1988). Therefore, the significant physical event is the interfacial transfer of the constituent from the lipophilic stratum corneum into the aqueous underlying viable tissue. For highly lipophilic constituents such as dioxins, the viable epidermis also serves as a significant resistance to penetration into the skin. The time for these compounds to reach steady-state may be on the order of hours (Dugard, 1986), and therefore can have a significant impact on the use of the simple steady-state Fick's first law in the evaluation of the dermally absorbed dose for constituent exposure in environmentally relevant scenarios. The resulting flux through the skin becomes a function of both the exposure period and the physico-chemical properties of the constituents as they influence the relative resistance of these two layers. This effect of the viable epidermis on the cumulative mass which enters the stratum corneum can be characterized by a parameter B, which describes the relative contribution of the permeability coefficients of the constituent in the stratum corneum and the viable epidermis. Consequently, increasing constituent lipophilicity causes B to become larger. As an initial estimate based on literature values of the magnitude of these variables, B can be approximated by the following equation (Cleek and Bunge, 1992):

$B = Kow/10,000$

As constituents increase in lipophilicity, the viable epidermis will restrict the flux of chemicals leaving the stratum corneum. If K_{ow} is large enough ($B > 100$ or log K_{ow} of approximately 5), the viable epidermis entirely controls the steady-state flux of the constituent.

Attachment 4: Information about EPA's Classification of Carcinogens

U.S. EPA's carcinogen system was used to determine if a chemical is considered a carcinogen in this Assessment. In 1986 (U.S. EPA, 1986), the U.S. EPA published a carcinogen classification scheme using a weight-of-evidence approach to classify the likelihood of a chemical to be a human carcinogen. Information considered in the classification included human studies of the association between cancer incidence and exposure as well as long-term animal studies under controlled laboratory conditions. U.S. EPA issued more current guidelines for the classification of carcinogens in 2005 (U.S. EPA, 2005). These new guidelines recommend expressing weight of evidence by narrative statements rather than by only hierarchical categories. Even though U.S. EPA has adopted these new guidelines, U.S. EPA's chemical database, the Integrated Risk Information System (IRIS) lists the old classification.

U.S. EPA 1986 Classification of Carcinogens	U.S. EPA 2005 Classification of Carcinogens
A – Human carcinogen	Carcinogenic to humans
B1 – Probable human carcinogen	Likely to be carcinogenic to humans
B2 – Probable human carcinogen	Suggestive evidence of carcinogenic potential
C – Possible human carcinogen	Suggestive evidence of carcinogenic potential
D – Not classifiable as to human carcinogenicity	Inadequate information to assess carcinogenic potential
E – Evidence of noncarcinogenicity for humans	Not likely to be carcinogenic in humans

Since risks at low levels of exposure cannot be quantified directly from either animal or epidemiological studies, mathematical models are used to extrapolate from relatively high experimental doses to low doses which are usually encountered in the environment. When adequate human epidemiological data are available, maximum likelihood estimates are used in the model. When only animal data are available, the cancer slope factors or unit risk values are typically derived from the upper 95% confidence limit of the largest linear slope that is consistent with the laboratory data. A number of mathematical models exist (i.e.,Weibull, Probit, Logit, Gamma multi-hit, Linearized multi-stage) that can be used for high to low dose extrapolation. Application of these models to a specific data set may provide a reasonable fit to the observed data.

Under the assumption of low dose linearity, the slope factor (SF) is a constant, and risk has a linear relationship to exposure, meaning that any increase in exposure results in a linear increase in risk. For ingestion, the SF represents the risk per mg/kg per day. Generally, the SF is defined by the U.S. EPA as "… a plausible upper-bound estimate of the probability of a response per unit intake of a substance over a

lifetime …," or in other words a plausible upper-bound estimate of an individual developing cancer as a result of exposure to a particular level of a carcinogenic substance. U.S. EPA cautions however that "It should be emphasized that the linear multistage procedure leads to a plausible upper limit to the risk that is consistent with some mechanism of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown and may be as low as zero." (U.S. EPA, 1989; 1999*)*.

Attachment 5: Information about Noncarcinogens

One important step in the process of deriving toxicity values for non-carcinogens is to identify the concentration of the chemical that presented either a "no observed adverse effect level" (NOAEL), or a "lowest observed adverse effect level" (LOAEL). NOAELs and LOAELs derived from animal studies are converted to "human equivalent concentrations" (HEC) using dosimetric methods to account for the differences in human and animal physiology (U.S. EPA, 1994).

More recently, the U.S. EPA in its review of noncarcinogenic values has been applying the benchmark dose (BMD) or benchmark concentration (BMC) approach. The BMD/BMC is an alternative to the NOAEL approach to identify the dose with a statistically significant effect (10% increase over background) based on the experimental data. The BMD/BMC considers the entire data set, and does not depend on a single data point as does the NOAEL and provides information on the overall dose-response relationship.

Attachment 6: Carcinogenic Toxicity Values

Chemical Name	CAS No.	U.S. EPA IRIS Cancer Class (1)	Dermal Slope Factor (SF)		Oral Slope Factor (SF)	
			(mg/kg-day)$^{-1}$	Source	(mg/kg-day)$^{-1}$	Source
Inorganic Arsenic	7440-38-2	A	1.5E+00	IRIS	1.5E+00	IRIS
Copper	7440-50-8	D	NA	NA	NA	NA
Lead	7439-92-1	B2	8.5E-03	Cal EPA	8.5E-03	Cal EPA
Nickel	7440-02-0	A	9.1E-01	Cal EPA	9.1E-01	Call EPA
Anthracene	120-12-7	D	NA	NA	NA	NA
Benzo(a)anthracene	56-55-3	B2	1.2E+00	Cal EPA	1.2E+00	Cal EPA
Benzo(a) pyrene	50-32-8	B2	2.8E+00	MDH Guidance, 2001, Age Adjusted	2.8E+00	MDH Guidance, 2001, Age Adjusted
Benzo(b)fluoranthene[1]	205-99-2	B2	2.8E-01	MDH Guidance, 2001, Age Adjusted	2.8E-01	MDH Guidance, 2001, Age Adjusted
Benzo(k)fluoranthene[1]	207-08-9	B2	2.8E-01	MDH Guidance, 2001, Age Adjusted	2.8E-01	MDH Guidance, 2001, Age Adjusted
Chrysene	218-01-9	B2	2.8E-03	MDH Guidance, 2001, Age Adjusted	2.8E-03	MDH Guidance, 2001, Age Adjusted
Dibenz[a,h]anthracene	53-70-3	B2	1.6 E+00	MN HRV	1.6E+00	MN HRV
Fluoranthene	206-44-0	D	NA	NA	NA	NA
Fluorene	86-73-7	D	NA	NA	NA	NA
Indeno (1,2,3-cd)pyrene	193-39-5	B2	2.8E-01	MDH Guidance,2001, Age Adjusted	2.8E-01	MDH Guidance, 2001, Age Adjusted
Naphthalene	91-20-3	C	1.2E-01	Cal EPA	1.2E-01	Cal EPA
Pyrene	129-00-0	D	NA	NA	NA	NA
Dioxins and Furans						
2,3,7,8-TCDD Equivalents	NA	B2	1.4E+06	MDH Guidance, 2009	1.4E+06	MDH Guidance, 2009

NA=Not Applicable
Derived = slope factor likely derived from a value for a different route of exposure
(1) Some chemicals are listed that only have an EPA IRIS classification, but no toxicity values. These chemicals are left in the table so that the reader can see the IRIS carcinogen classification.

Attachment 7: Noncarcinogenic Toxicity Values

Chemical	CAS No.	Dermal Reference Dose		Oral Reference Dose	
		mg/kg-day	Source	mg/kg-day	Source
Inorganic Arsenic	7440-38-2	3.0E- 04	IRIS	3.0E- 04	IRIS
Copper	7440-50-8	4.00E-02	Risk Assessment Information System, Toxicity Profile for Copper	4.00E-02	Risk Assessment Information System, Toxicity Profile for Copper
Nickel, soluble salts	7440-02-0	2.0E-02	IRIS	2.0E-02	IRIS
Zinc	7440-66-6	3.0E-01	IRIS	3.0E-01	IRIS
2-Methylnaphthalene	91-57-6	4.3E-03	IRIS	4.3E-03	IRIS
Acenaphthene	83-32-9	6.0E-02	IRIS	6.0E-02	IRIS
Acenaphthylene	208-96-8	NA	NA	NA	NA
Anthracene	120-12-7	3.0E-01	IRIS	3.0E-01	IRIS
Benzo(a)anthracene	56-55-3	NA	NA	NA	NA
Benzo(a) pyrene	50-32-8	4.0E-04	MDH, 2012, RfD used in the derivation of chronic HBV	4.0E-04	MDH, 2012, RfD used in the derivation of chronic HBV
Fluoranthene	206-44-0	4.0E-02	IRIS	4.0E-02	IRIS
Fluorene	86-73-7	4.0E-02	IRIS	4.0E-02	IRIS
Indeno (1,2,3-cd)pyrene	193-39-5	7.3E-01	MN HRV	NA	NA
Naphthalene	91-20-3	2.0E-02	IRIS	2.0E-02	IRIS
Pyrene	129-00-0	3.0E-02	IRIS	3.0E-02	IRIS
Dioxins and Furans					
2,3,7,8-TCDD Equivalents		7E-10	IRIS	7E-10	IRIS

NA=Not Applicable
Derived = reference dose likely derived from a value for a different route of exposure

Attachment 8: Uncertainty in Toxicity Values

Route to Route Extrapolation of Toxicity Values

Dermal contact with constituents can result in direct toxicity at the site of application and/or contribute to systemic toxicity via percutaneous absorption. The issue of direct toxicity (or portal-of entry effects) is addressed in Section 3.3.2 Ideally, a route-specific (i.e., dermal) toxicity factor would not only consider portal-of-entry effects (i.e., direct toxicity) but would also provide dosimetry information on the dose-response relationship for systemic effects via percutaneous absorption.

In the absence of dermal toxicity factors, U.S. EPA has devised a simplified method for making route-to-route (oral-to-dermal) extrapolations for systemic effects. This process is outlined in Appendix A of RAGS/HHEM (U.S. EPA, 1989). Primarily, it accounts for the fact that most RfDs and SFs are expressed as the amount of substance administered per unit time and body weight, whereas exposure estimates for the dermal pathway are expressed as absorbed dose. The process utilizes the dose-response relationship obtained from oral administration studies and makes an adjustment for absorption efficiency to represent the toxicity factor in terms of absorbed dose.

This approach is subject to a number of factors that might compromise the applicability of an oral toxicity factor for dermal exposure assessment. The estimation of oral absorption efficiency, to adjust the toxicity factor from administered to absorbed dose, introduces uncertainty. Part of this uncertainty relates to distinctions between the terms “absorption” and “bioavailability.” Typically, the term absorption refers to the “disappearance of chemical from the gastrointestinal lumen,” while oral bioavailability is defined as the “rate and amount of chemical that reaches the systemic circulation unchanged.” That is, bioavailability accounts for both absorption and pre-systemic metabolism. Although pre-systemic metabolism includes both gut wall and liver metabolism, for the most part it is liver metabolism or liver “first pass” effect that plays the major role.

In the absence of metabolic activation or detoxification, toxicity adjustment should be based on bioavailability rather than absorption because the dermal pathway purports to estimate the amount of parent compound entering the systemic circulation. Metabolism in the gut wall and skin can serve to complicate this otherwise simplified adjustment process. Simple adjustment of the oral toxicity factor, based on oral absorption efficiency, does not account for metabolic by-products that might occur in the gut wall but not the skin, or conversely in the skin, but not the gut wall.

More importantly the oral administered dose experiences the liver "first pass" effect. The efficiency of "first pass" metabolism, and whether this is an activating or detoxifying process determines the nature of the impact this effect has on route-to-route extrapolations. One example is a compound that exhibits poor oral systemic bioavailability due to a prominent "first pass" effect which creates a highly toxic metabolite. The adjusted dermal toxicity factor may overestimate the true dose-response relationship because it would be based upon the amount of parent compound in the systemic circulation rather than on the toxic metabolite. Additionally, percutaneous absorption may not generate the toxic metabolite to the same rate and extent as the gastrointestinal route.

Toxicity is a function of constituent concentration at critical sites-of-action. Absorption rate, as well as extent of absorption, determines constituent concentration at a site-of-action. Differences in the anatomic barriers of the gastrointestinal tract and the skin can affect rate as well as the extent of absorption; therefore, the route of exposure may have significant dose-rate effects at the site-of-action (U.S. EPA, 2004).

For the site-related COIs, the U.S. EPA does not recommend an adjustment to the oral SF or RfD and has, accordingly, when possible, not been performed for the Assessment. The only exception to this is nickel. The oral slope factor was derived directly from the inhalation slope factor based on inhalation studies. This introduces an unknown level of uncertainty that may have under- or overestimated risk.

Acknowledging Confidence and Precision in the Data

The toxicity values are derived from data sets with varying levels of confidence in the adequacy of the data. Depending on the type, quality, and quantity of the data used to develop toxicity values, U.S. EPA and the MDH use a combination of uncertainty (UF) and modifying factors (MF). The UFs are applied to account for recognized areas of uncertainty in the extrapolation from the experimental data and exposure conditions to the human lifetime exposure conditions. In general, UFs and/or MFs are applied as follows:

- A UF of 10 is used to account for variation in the general population.
- A UF of 10 is used when extrapolating from animals to humans.
- A UF of 10 is used when using a NOAEL based on a subchronic study.
- A UF of 10 is used when a LOAEL is used as a starting point.
- Additional modifying factors up to 10 may be applied to account for other uncertainties associated with the critical study upon which the NOAEL/LOAEL is based.

The toxicity values used in this Assessment have been derived using composite UFs that range from 10 (e.g., cadmium) to 3,000 (e.g., anthracene).

The relative precision and the magnitude of the composite UFs are important considerations in decisions involving comparisons of hazard quotients for different chemicals and in assessing the hazard index for a mixture of chemicals (U.S. EPA, 1989; 1999a).

It should be noted that exposures above a toxicity value do not necessarily imply unacceptable risk or that health effects are expected. Because of the inherent conservatism of the toxicity value methodology, the significance of exceedances must be evaluated on a case-by-case basis, considering such factors as the size of the UFs used, the slope of the dose-response curve and the magnitude of the exceedance (U.S. EPA, 1989, 1999a).