HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Volume IV of IV
Appendix E
Supporting Information and Calculations for the ERA (including CD)

Prepared for

International Paper

Pursuant to

Unilateral Administrative Order Docket No. V-W-04-C-796

Prepared by

integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80210

September 28, 2007

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Appendix E
Supporting Information and Calculations for the ERA (including CD)

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integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80021

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APPENDIX E1

SCREENING VALUES FOR THE ECOLOGICAL RISK ASSESSMENT

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

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Appendix E1
Screening Values for the Ecological Risk Assessment

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12303 Airport Way
Suite 370
Broomfield, CO 80021

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ARCS	Assessment and Remediation of Contaminated Sediments
CCC	criterion continuous concentration
CCME	Canadian Council of Ministers of the Environment
COPEC	chemicals of potential ecological concern
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ESL	ecological screening level
ESV	ecological screening value
ISQG	interim sediment quality guidelines
LAET	lowest apparent effects threshold
LEL	lowest effect level
MTCA	Model Toxics Control Act
OSWER	Office of Solid Waste and Emergency Response
PCP	pentachlorophenol
RAWP	risk assessment work plan
SCV	secondary chronic values
SSL	soil screening levels
SQB	sediment quality benchmark
SQC	sediment quality criteria
SQG	sediment quality guideline
TEC	threshold effect concentration
TEL	threshold effect level
TRV	toxicity reference value
WSDE	Washington State Department of Ecology

1 INTRODUCTION

In response to a letter from EPA to International Paper dated March 11, 2005, indicating the agency's requirement for advance review of toxicity reference values to be used in the ecological risk assessment (ERA), Integral Consulting Inc. prepared a technical memorandum (Integral 2005). The purpose of the technical memorandum was to identify toxicity reference values (TRVs) for use in the ERA. The technical memorandum was submitted to EPA on July 7, 2005.

On August 11, 2005, EPA provided written comments on the technical memorandum. In response, International Paper made several changes to the screening benchmarks presented in Integral (2005) prior to completing the screening and risk assessment. Text describing methods for selecting screening values was developed, also in response to EPA comments on the technical memorandum. EPA submitted additional comments on September 30, 2005, April 2006, and December 2006, which were also considered in development of this final document. This appendix provides a complete listing of final screening values used in the ERA and clarifies methods used to identify and select COPECs. This appendix is organized as follows:

- Sediment screening benchmarks
- Soil screening benchmarks
- Surface water screening benchmarks.

The tables in this appendix are intended to provide complete sets of screening benchmarks for all parameter groups that have been evaluated in the ERA process, including those that were screened out in the risk assessment work plan (RAWP) (USEPA 2004c). Chemicals that were not carried forward into the 2004 sampling program to address data gaps were not rescreened and are not discussed further in the risk assessment report.

2 SCREENING BENCHMARKS

The hierarchy for selection of screening values for sediment, soil, and water established in the RAWP (USEPA 2004c) was followed in selection of final screening values. For sediment, soil, and water, the hierarchy for selection of benchmarks (as described in the RAWP) is presented. Tables listing all screening values for sediment, soil, and water are also included.

2.1 SEDIMENT SCREENING BENCHMARKS

In general, SQGs help analysts characterize the potential for sediment toxicity to benthic organisms on the basis of sediment chemistry. Literature-based SQGs are typically derived from regional or national data sets originating from co-located samples for which both sediment chemistry and biological data are available. Examples of possible biological data include toxicity, benthic macroinvertebrate community analysis, and fish histopathology. SQGs derived from such data sets are appropriate for screening evaluations and have already been used at the St. Regis Site. Specifically, they were used in initial problem formulation, development of the RAWP, the screening level ecological risk assessment presented in the RAWP (USEPA 2004c), and in the interpretation of site-specific toxicity tests (Section 5.4; Appendix E6).

Sediment screening values were developed from the hierarchy of literature-based screening values identified in the RAWP (USEPA 2004c). For a given chemical, a screening value was selected from the first source in which it appears among the sources listed below. If there was no value for the chemical in that source, the next source was consulted. Sources of SQGs are listed below in priority order:

- **Consensus-Based Threshold Effect Concentration (TEC) Sediment Quality Guidelines (MacDonald et al. 2000):** Consensus-based TEC.
- **EPA Assessment and Remediation of Contaminated Sediments (ARCS) (Ingersoll et al. 1996):** Threshold effect level (TEL).
- **Ontario Ministry of Environment (Persaud et al. 1993):** Lowest effect level (LEL).
- **Washington State (Cubbage et al. 1997):** Lowest apparent effects threshold (LAET).
- **EPA Office of Solid Waste and Emergency Response (OSWER) (USEPA 1996):** Sediment quality criteria (SQC) based on equilibrium partitioning using EPA's ambient water quality criteria, or sediment quality benchmark (SQB) by equilibrium partitioning (based on Tier II values). Values provided assume 1 percent TOC; a formula is provided to calculate a screening value based on varying TOC as follows:

  SQC= Foc *Koc*FCV

Where:

Foc = mass fraction of organic carbon of the sediment (kg organic carbon/kg sediment)

Koc = organic carbon partition coefficient (L/kg organic carbon)

FCV= final chronic value, as provided in USEPA (1996) (mg/L)

The same equation can be used for SQB, substituting Tier II values for the SCV. Organic carbon partition coefficients were obtained at the Risk Assessment Information System web site (RAIS 2005).

- **Canadian Environmental Quality Guidelines, Canadian Council of Ministers of the Environment (CCME 2003)**: Freshwater interim sediment quality guidelines (ISQG).

If there was no screening value among these sources, sediment screening values provided by Tetra Tech EM (2002) were used, consistent with the RAWP (USEPA 2004c).

Because an SQG for pentachlorophenol (PCP) could not be found among these sources, a value provided by the Wisconsin Department of Natural Resources was used. WDNR (2003) uses a water quality criterion and the organic carbon-water partition coefficient (K_{oc}) for PCP and assumes 1 percent organic carbon to derive TEC of 150 µg/kg. Because this value is consistent with a bulk sediment LAET concentration of 360 µg/kg proposed by Jones et al. (1997) and USEPA (1996), but is somewhat more conservative, it is applied to this screening process.

Combination of the above SQGs according to the hierarchy established in the RAWP (USEPA 2004c) formed the full list of final sediment screening benchmarks, provided in Table E1-1.

Table E1-1. Screening Values for Analytes in Sediments

Analyte	Units	Ecological Screening Benchmark		
		Concentration	Type	Source
Dioxins/Furans				
Total PCDD (ND = 1/2 DL)	mg/kg dw	NA	NA	--
Total PCDF (ND = 1/2 DL)	mg/kg dw	NA	NA	--
Total PCDF/PCDD (ND = 1/2 DL)	mg/kg dw	NA	NA	--
Total PCDD (ND = 1/2 DL)	mg/kg dw	NA	NA	--
Metals				
Aluminum	mg/kg dw	26,000	TEL	(Ingersoll et al. 1996)
Antimony	mg/kg dw	3	LAET	(Cubbage et al 1997)
Arsenic	mg/kg dw	9.79	TEC	(MacDonald et al. 2000)
Barium[a]	mg/kg dw	20	TEC	(MacDonald et al. 2000)

Table E1-1. Screening Values for Analytes in Sediments

Analyte	Units	Ecological Screening Benchmark		
		Concentration	Type	Source
Beryllium	mg/kg dw	NA	NA	(MacDonald et al. 2000)
Cadmium[b]	mg/kg dw	0.99	TEC	(MacDonald et al. 2000)
Chromium	mg/kg dw	43 4	TEC	(MacDonald et al. 2000)
Cobalt	mg/kg dw	50	LEL	(Persaud et al. 1993)
Copper	mg/kg dw	31.6	TEL	(Ingersoll et al. 1996)
Iron[b]	mg/kg dw	190,000	TEL	(Ingersoll et al 1996)
Lead	mg/kg dw	35.8	TEC	(MacDonald et al. 2000)
Manganese	mg/kg dw	630	TEL	(Ingersoll et al. 1996)
Mercury	mg/kg dw	0.18	TEC	(MacDonald et al. 2000)
Molybdenum	mg/kg dw	NA	NA	--
Nickel	mg/kg dw	22.7	TEC	(MacDonald et al. 2000)
Selenium	mg/kg dw	0 1	--	(Tetra Tech EM 2002)
Silver	mg/kg dw	2	--	(Tetra Tech EM 2002)
Thallium	mg/kg dw	NA	NA	--
Vanadium	mg/kg dw	57	--	(Tetra Tech EM 2002)
Zinc	mg/kg dw	121	TEC	(MacDonald et al. 2000)
PAHs				
2-Methylnaphthalene	mg/kg dw	NA	NA	(Integral 2005)
Acenaphthene	mg/kg dw	4.1	LAET	(Cubbage et al. 1997)
Anthracene	mg/kg dw	0.57	TEC	(MacDonald et al. 2000)
Acenaphthylene	mg/kg dw	2.2	LAET	(Cubbage et al. 1997)
Benz[a]anthracene	mg/kg dw	0.108	TEC	(MacDonald et al. 2000)
Benzo[b]fluoranthene	mg/kg dw	0.037	--	(Tetra Tech EM 2002)
Benzo[k]fluoranthene	mg/kg dw	0.037	--	(Tetra Tech EM 2002)
Benzofluoranthenes, total	mg/kg dw	16	LAET	(Cubbage et al. 1997)
Benzo[ghi]perylene	mg/kg dw	0.016	TEL	(Ingersoll et al. 1996)
Benzo[a]pyrene	mg/kg dw	0 15	TEC	(MacDonald et al. 2000)
Chrysene	mg/kg dw	0.166	TEC	(MacDonald et al. 2000)
Dibenz[a,h]anthracene	mg/kg dw	0.033	TEC	(MacDonald et al. 2000)
Fluoranthene	mg/kg dw	0 423	TEC	(MacDonald et al. 2000)
Fluorene	mg/kg dw	0 01	TEL	(Ingersoll et al. 1996)
Indeno[1,2,3-cd]pyrene	mg/kg dw	0.017	TEL	(Ingersoll et al. 1996)

Table E1-1 Screening Values for Analytes in Sediments

Analyte	Units	Ecological Screening Benchmark		
		Concentration	Type	Source
Naphthalene	mg/kg dw	0 176	TEC	(MacDonald et al 2000)
Phenanthrene	mg/kg dw	0.867	--	(CCME 2002)
Pyrene	mg/kg dw	0.195	TEC	(MacDonald et al. 2000)
LPAHs	mg/kg dw	0 076	TEL	(Ingersoll et al. 1996)
HPAHs	mg/kg dw	0.19	TEL	(Ingersoll et al. 1996)
PAHs, total	mg/kg dw	1 61	TEC	(MacDonald et al. 2000)
Pesticides				
4,4'-DDD	mg/kg dw	0 00488	TEC	(MacDonald et al. 2000)
4,4'-DDE	mg/kg dw	0.00316	TEC	(MacDonald et al. 2000)
4,4'-DDT	mg/kg dw	0 00416	TEC	(MacDonald et al. 2000)
Aldrin	mg/kg dw	0.002	LEL	(Persaud et al. 1993)
α-BHC	mg/kg dw	0.006	LEL	(Persaud et al 1993)
β-BHC	mg/kg dw	0 005	LEL	(Persaud et al. 1993)
γ-BHC (Lindane)	mg/kg dw	0 00237	TEC	(MacDonald et al 2000)
Chlordane	mg/kg dw	0 00324	TEC	(MacDonald et al. 2000)
Dieldrin	mg/kg dw	0 0019	TEC	(MacDonald et al 2000)
Endosulfan II[c]	mg/kg TOC	$Foc*22000*(5.1*10^{-5})$	SQB	(USEPA 1996)
Endrin	mg/kg dw	0.00222	TEC	(MacDonald et al. 2000)
Heptachlor	mg/kg dw	0.0006	ISQG	(CCME 2003)
Heptachlor Epoxide	mg/kg dw	0.00247	TEC	(MacDonald et al. 2000)
Methoxychlor[c]	mg/kg TOC	$Foc*42600*(1.9*10^{-5})$	SQB	(USEPA 1996)
Toxaphene[c]	mg/kg TOC	$Foc*99300*(1.1*10^{-5})$	SQB	(USEPA 1996)
Pentachlorophenol				
Pentachlorophenol		0.15	TEC	(WDNR 2003)
PCB and PCB Congeners				
Aroclor-1248	mg/kg dw	0.03	LEL	(Persaud et al. 1993)
Dichloro Biphenyls, total	mg/kg dw	--	--	(Integral 2005)
Polychlorinated biphenyls	mg/kg dw	0.0230	ERL	(USEPA 1996)
PCB Aroclors, total[b]	mg/kg dw	0.0598	TEC	(MacDonald et al. 2000)
VOC/SVOC				
1,1,2,2-Tetrachloroethane[c]	mg/kg TOC	Foc*106.8*0 420	SQB	(USEPA 1996)
1,2,4-Trichlorobenzene[c]	mg/kg TOC	Foc*717.6*0.110	SQB	(USEPA 1996)

Table E1-1. Screening Values for Analytes in Sediments

Analyte	Units	Ecological Screening Benchmark		
		Concentration	Type	Source
1,2-Dichlorobenzene[c]	mg/kg TOC	Foc*443.1*0.014	SQB	(USEPA 1996)
1,3-Dichlorobenzene[c]	mg/kg TOC	Foc*434*0 071	SQB	(USEPA 1996)
1,4-Dichlorobenzene[c]	mg/kg TOC	Foc*434*0.015	SQB	(USEPA 1996)
4-Bromophenyl phenyl ether[c]	mg/kg TOC	Foc*4160*0.0015	SQB	(USEPA 1996)
Bis(2-ethylhexyl)phthalate	mg/kg dw	0.75	LAET	(Cubbage et al. 1997)
Carbazole	mg/kg dw	0.14	LAET	(Cubbage et al. 1997)
Chlorobenzene[c]	mg/kg TOC	Foc*268*0.130	SQB	(USEPA 1996)
Dibenzofuran	mg/kg dw	32	LAET	(Cubbage et al. 1997)
Diethyl phthalate[c]	mg/kg TOC	Foc*126.2*0 220	SQB	(USEPA 1996)
Di-n-butyl phthalate	mg/kg dw	0.043	LAET	(Cubbage et al. 1997)
Hexachloroethane[c]	mg/kg TOC	Foc*0 012*224.7	SQB	(USEPA 1996)
Phenol	mg/kg dw	0.048	LAET	(Cubbage et al. 1997)
Toluene[c]	mg/kg TOC	Foc*268*0.130	SQB	(USEPA 1996)
Trichloroethylene[c]	mg/kg TOC	Foc*67 7*0.35	SQB	(USEPA 1996)

Notes:
dw = dry weight
Foc = mass fraction of organic carbon in sediment
ISQG = interim sediment quality guidelines
LAET = lowest apparent effect threshold
NA = Not available
SQB = sediment quality benchmark
SQG = sediment quality guideline
TEC = threshold effect concentration
TEL = threshold effect level
TOC = total organic carbon
[a] Value has been added since publication of Integral (2005).
[b] Value has been corrected from previous table (Integral 2005)
[c] Equation provided to calculate value based on site-specific TOC. Equations for some SQBs were not provided in previous version of this table (Integral 2005, Table 4), those omissions have been corrected here.

2.2 SOIL SCREENING BENCHMARKS

The hierarchy for soil screening benchmarks is established in the RAWP (USEPA 2004c) and is applied as follows (in order of priority):

- **The ecological soil screening levels (SSLs) developed by USEPA (updates available online at http://www.epa.gov/ecotox/ecossl/).** Draft final and interim final SSLs were issued for several chemicals in 2005 (USEPA 2003a–e and USEPA 2005a–g). The level protective of the most sensitive receptor group was selected as the soil screening benchmark.

- **EPA Region 4 ecological screening values for soil (USEPA 2004a).** EPA Region 4 values were developed for use at the U.S. Department of Energy Savannah River Site in South Carolina (Friday 1998). Friday (1998) compiled a comprehensive list of ecological screening values for soil from five different sources and selected a conservative set of final screening values for soil from five different sources and developed recommended screening values.

- **EPA Region 5 RCRA ecological screening levels for soil (USEPA 2004b).**

- **The Washington State Model Toxics Control Act (MTCA) (WSDE 2001).** The dioxin value was derived using food chain models for birds and mammals that forage on vegetation, and for soil invertebrates that bioaccumulate chemicals from the soil.

The final soil screening values from this hierarchy are provided in Table E1-2.

Table E1-2. Screening Values for Analytes in Soils

	Ecological Screening Benchmark		
Analyte	Concentration (mg/kg)	Type	Source
Dioxins/Furans			
PCDD, total	1.99E-07	ESL	(USEPA 2004b)
PCDF, total	3.86E-05	ESL	(USEPA 2004b)
TEQDF-WHO98 (ND = 1/2 DL)	2.E-06	--	(WSDE 2001)
Metals			
Aluminum	50	ESV	(USEPA 2004a)
Antimony	0 3	Eco-SSL	(USEPA 2003a)
Arsenic	18	Eco-SSL	(USEPA 2005a)
Barium	330	Eco-SSL	(USEPA 2003b)
Beryllium	21	Eco-SSL	(USEPA 2005b)
Cadmium	0 36	Eco-SSL	(USEPA 2005c)
Chromium(III)[a]	26	Eco-SSL	(USEPA 2005d)
Chromium(VI)	81	Eco-SSL	(USEPA 2005d)
Cobalt	13	Eco-SSL	(USEPA 2005e)
Copper	40	ESV	(USEPA 2004a)
Iron	200	ESV	(USEPA 2003c)
Lead	11	Eco-SSL	(USEPA 2003d)
Manganese	100	ESV	(USEPA 2004a)
Mercury	0.1	ESV	(USEPA 2004a)
Molybdenum	2 0	ESV	(USEPA 2004a)
Nickel	30	ESV	(USEPA 2004a)
Selenium	0.81	ESV	(USEPA 2004a)
Silver	2.0	ESV	(USEPA 2004a)
Thallium	1.0	ESV	(USEPA 2004a)
Vanadium	7 8	Eco-SSL	(USEPA 2005g)
Zinc	50	ESV	(USEPA 2004a)
PAHs			
2-Chloronaphthalene	0.0122	ESL	(USEPA 2004b)

Table E1-2. Screening Values for Analytes in Soils

Analyte	Ecological Screening Benchmark Concentration (mg/kg)	Type	Source
2-Methylnaphthalene[b]	0.1	ESV	(USEPA 2004a)
Acenaphthene	20	ESL	(USEPA 2004a)
Acenaphthylene[b]	0.1	ESV	(USEPA 2004a)
Anthracene	0 1	ESV	(USEPA 2004a)
Benzo[b]fluoranthene[b]	0.1	ESV	(USEPA 2004a)
Benzo[k]fluoranthene[b]	0.1	ESV	(USEPA 2004a)
Benzo[ghi]perylene[b]	0 1	ESV	(USEPA 2004a)
Chrysene	0.1	ESV	(USEPA 2004a)
Dibenz[a,h]anthracene	18.400	ESL	(USEPA 2004b)
Fluoranthene	0.1	ESV	(USEPA 2004a)
Fluorene[b]	0.1	ESV	(USEPA 2004a)
Indeno[1,2,3-cd]pyrene[b]	0.1	ESV	(USEPA 2004a)
Naphthalene	0.1	ESV	(USEPA 2004a)
Phenanthrene	0.1	ESV	(USEPA 2004a)
Pyrene	0.1	ESV	(USEPA 2004a)
PAH, total	1.0	ESV	(USEPA 2004a)
Pesticides			
4,4'-DDD	NA		(USEPA 2004a)
4,4'-DDE	NA		(USEPA 2004a)
4,4'-DDT	NA		(USEPA 2004a)
Total DDx	0.0025	ESV	(USEPA 2004a)
α-BHC	0.0025	ESV	(USEPA 2004a)
β-BHC	0.001	ESV	(USEPA 2004a)
γ-BHC (Lindane)	5.0E-05	ESV	(USEPA 2004a)
δ-BHC	9.940	ESL	(USEPA 2004b)
Aldrin	0.0025	ESV	(USEPA 2004a)
Atrazine	5.E-05	ESV	(USEPA 2004a)
Chlordane	0.224	ESL	(USEPA 2004b)
Dieldrin	0.000032	ESL	(USEPA 2004b)
Endosulfan I	0.119	ESL	(USEPA 2004b)
Endosulfan II	0.119	ESL	(USEPA 2004b)
Endosulfan Sulfate	0 0358	ESL	(USEPA 2004b)
Endrin	0.001	ESV	(USEPA 2004a)
Endrin Aldehyde	0.0105	ESL	(USEPA 2004b)
Heptachlor	0.00598	ESL	(USEPA 2004b)
Heptachlor Epoxide	0 152	ESL	(USEPA 2004b)
Hexachlorobenzene	0.0025	ESV	(USEPA 2004a)
Methoxychlor	0 0199	ESL	(USEPA 2004b)
Toxaphene	0.119	ESL	(USEPA 2004b)
PCB and PCB Congeners			
Sum of total PCBs	0.02	ESV	(USEPA 2004a)

Table E1-2. Screening Values for Analytes in Soils

Analyte	Ecological Screening Benchmark		
	Concentration (mg/kg)	Type	Source
PCP			
Pentachlorophenol	0.0018	Eco-SSL	(USEPA 2005f)
VOC/SVOC			
1,1,1,2-Tetrachloroethane	225	ESL	(USEPA 2004b)
1,1,1-Trichloroethane	29 8	ESL	(USEPA 2004b)
1,1,2,2-Tetrachloroethane	0 127	ESL	(USEPA 2004b)
1,2,4,5-Tetrachlorobenzene	2.020	ESL	(USEPA 2004b)
1,2,4-Trichlorobenzene	11.1	ESL	(USEPA 2004b)
1,2-Dibromo-3-chloropropane	0.0352	ESL	(USEPA 2004b)
1,2-Dibromoethane	1 230	ESL	(USEPA 2004b)
1,2-Dichloroethane	21.2	ESL	(USEPA 2004b)
1,2-Dichloroethylene, *cis-* [c]	0.784	ESL	(USEPA 2004b)
1,2-Dichloroethylene, *trans-*	0.784	ESL	(USEPA 2004b)
1,2-Dichloropropane	700	ESV	(USEPA 2004a)
1,3-Dichloro-1-propene, *trans-*	0.398	ESL	(USEPA 2004b)
2,4,5-Trichlorophenol	4.0	ESV	(USEPA 2004a)
2,4,6-Trichlorophenol	10	ESV	(USEPA 2004a)
2,4-Dichlorophenol	87.5	ESL	(USEPA 2004b)
2,4-Dimethylphenol	0.01	ESL	(USEPA 2004b)
2,4-Dinitrophenol	20.0	ESV	(USEPA 2004a)
2,4-Dinitrotoluene	1.28	ESV	(USEPA 2004b)
2,6-Dinitrotoluene	0.0328	ESL	(USEPA 2004b)
2-Chlorophenol	0.243	ESL	(USEPA 2004b)
2-Hexanone	12 6	ESL	(USEPA 2004b)
2- Methylphenol	0.5	ESV	(USEPA 2004a)
2-Nitroaniline	74.1	ESL	(USEPA 2004b)
2-Nitrophenol	1.60	ESL	(USEPA 2004b)
3,3'-Dichlorobenzidine	0.646	ESL	(USEPA 2004b)
3- & 4- Methylphenol	0.5	ESV	(USEPA 2004a)
4-Methylphenol	0.5	ESV	(USEPA 2004a)
4-Nitroaniline	21.9	ESL	(USEPA 2004b)
4-Nitrophenol	7	ESV	(USEPA 2004a)
Acetone	2.50	ESL	(USEPA 2004b)
Biphenyl	60	ESV	(USEPA 2004a)
Bis(2-chloroethoxy)methane	0.302	ESL	(USEPA 2004b)
Bis(2-ethylhexyl)phthalate[d]	100	ESV	(USEPA 2004a)
Bromodichloromethane	0 54	ESL	(USEPA 2004b)
Butyl benzyl phthalate[c]	100	ESV	(USEPA 2004a)
Carbon disulfide	0.0941	ESL	(USEPA 2004b)
Carbon tetrachloride	1000	ESV	(USEPA 2004a)
Cyclohexane	0.1	ESV	(USEPA 2004a)
Dimethyl phthalate	200	ESV	(USEPA 2004a)

Table E1-2. Screening Values for Analytes in Soils

Analyte	Ecological Screening Benchmark		
	Concentration (mg/kg)	Type	Source
Di-n-octyl phthalate[d]	100	ESV	(USEPA 2004a)
Hexachlorobutadiene	0.0398	ESL	(USEPA 2004b)
Hexachlorocyclopentadiene	10	ESV	(USEPA 2004a)
Hexachloroethane	0.596	ESL	(USEPA 2004b)
Isophorone	139	ESL	(USEPA 2004b)
Methylene chloride	4.05	ESL	(USEPA 2004b)
Nitrobenzene	40	ESV	(USEPA 2004a)
N-Nitrosodiphenylamine	20	ESV	(USEPA 2004a)
Styrene	0.1	ESV	(USEPA 2004a)
Tetrachloroethylene	9.92	ESL	(USEPA 2004b)
Tetrachlorophenols, total	0.001	ESV	(USEPA 2004a)
Toluene	0.05	ESV	(USEPA 2004a)
Trichloroethylene[e]	0.001	ESV	(USEPA 2004a)
Trichlorofluoromethane[e]	16.4	ESL	(USEPA 2004b)
Vinyl chloride	0.01	ESV	(USEPA 2004a)

Notes:
Eco-SSL – ecological soil screening level
ESL – ecological screening level
ESV – ecological screening value
WSDE – Washington State Department of Ecology
[a] Value added since previous table (Integral 2005)
[b] Benzo[a]pyrene used as surrogate.
[c] *trans*-1,2-Dichloroethylene used as surrogate.
[d] Diethylphthalate used as surrogate.
[e] Value corrected from previous table (Integral 2005).

2.3 SURFACE WATER SCREENING BENCHMARKS

Surface water screening levels were selected from the following hierarchy:

- **Chronic Toxicity-based Water Quality Standards for Minnesota Class 2 Waters (MPCA 2004):** Value for subclass 2B chronic standard (the Minnesota regulatory subclass 2B chronic standard). For hardness-dependent values (cadmium, chromium (III), copper, lead, nickel, silver, and zinc), an equation is provided to calculate the screening value dependent on total hardness. Minnesota Rule 7050:0222 (State of Minnesota 2005), on which these values are based, was also consulted if an analyte was not found in MPCA (2004).
- **EPA National Recommended Water Quality Criteria (NRWQC) (USEPA 2002):** Freshwater criterion continuous concentration (CCC)

- **ORNL Tier II Benchmark Values (Suter and Tsao 1996):** Tier II secondary chronic values (SCVs)
- **EPA Region 5 RCRA Screening Levels (USEPA 2004b):** Ecological screening values (ESLs).

Final screening values for surface waters were selected from this hierarchy and are summarized in Table E1-3.

Table E1-3. Screening Values for Analytes in Surface Water

	Ecological Screening Benchmark		
Analyte	Concentration (mg/L)	Type	Source
Metals			
Aluminum, dissolved	0 125	2B CS	(MPCA 2004)
Aluminum, total[a]	0.125	2B CS	(MPCA 2004)
Antimony[b]	0.031	2B CS	(State of Minnesota 2005)
Arsenic, dissolved	0.15	CCC	(USEPA 2002)
Arsenic, total[a]	0.15	CCC	(USEPA 2002)
Barium, total	0.004	Tier II SCV	(Suter and Tsao 1996)
Beryllium, dissolved	0.00066	Tier II SCV	(Suter and Tsao 1996)
Cadmium, dissolved[c]	0.0009999; (Exp(0.7852(ln(TH)-3 490))*0.909	2B CS	(MPCA 2004)
Cadmium, total[c]	0.0011; Exp(0 7852(ln(TH)-3 490)	2B CS	(MPCA 2004)
Chromium(III), dissolved[c]	0.1782; (Exp(0.819(lnTH)+1.561))*0.860	2B CS	(MPCA 2004)
Chromium(III), total[a,c]	0 1782; (Exp(0.819(lnTH)+1.561))	2B CS	(MPCA 2004)
Chromium(VI), dissolved	0.010582	2B CS	(MPCA 2004)
Chromium(VI), total[a]	0.011	2B CS	(MPCA 2004)
Cobalt[b]	0.005	2B CS	(State of Minnesota 2005)
Copper, dissolved[c]	0.009408; (Exp(0.620(lnTH)-0.57))*0.960	2B CS	(MPCA 2004)
Copper, total[c]	0.0098; Exp(0.620(lnTH)-0 570)	2B CS	(MPCA 2004)
Iron, dissolved	1.000	CCC	(USEPA 2002)
Lead, dissolved[c]	0 0025,(Exp(1.273(lnTH)-4.705))*0.791	2B CS	(MPCA 2004)
Lead, total[a,c]	0.00253;Exp(1 273(lnTH)-4 705)	2B CS	(MPCA 2004)
Manganese[a]	0 120	Tier II SCV	(Suter and Tsao 1996)
Mercury, dissolved	0.00077	CCC	(USEPA 2002)
Mercury, total[a]	0.000855	CCC	(USEPA 2002)
Molybdenum	0.370	Tier II SCV	(Suter and Tsao 1996)
Nickel, dissolved[b,c]	0 158; (Exp(0.846(ln TH)+1.1645)*0.997	2B CS	(MPCA 2004)
Nickel, total[c]	0.158; Exp(0.846(ln TH)+1.1645	2B CS	(MPCA 2004)
Selenium, dissolved	0.0050	2B CS	(MPCA 2004)

Table E1-3. Screening Values for Analytes in Surface Water

	Ecological Screening Benchmark		
Analyte	Concentration (mg/L)	Type	Source
Selenium, total[a]	0.0050	2B CS	(MPCA 2004)
Silver, dissolved	0.0009	2B CS	(MPCA 2004)
Silver, total[a]	0.001	2B CS	(MPCA 2004)
Thallium, total	0.012	Tier II SCV	(Suter and Tsao 1996)
Vanadium	0.02	Tier II SCV	(Suter and Tsao 1996)
Zinc, dissolved[c]	0.10453; (Exp(0.8473(lnTH)+0.7615))*0.986	2B CS	(MPCA 2004)
Zinc, total[c]	0.106; Exp(0.8473(ln TH)+0.7615)	2B CS	(MPCA 2004)
PAHs			
1-Methylnaphthalene	0.0021	Tier II SCV	(Suter and Tsao 1996)
2-Methylnaphthalene	0.33	ESL	(USEPA 2004b)
Acenaphthene	0.038	ESL	(USEPA 2004b)
Acenaphthylene	4.84	ESL	(USEPA 2004b)
Anthracene	0.000035	2B CS	(MPCA 2004)
Benz[a]anthracene	0.000027	Tier II SCV	(Suter and Tsao 1996)
Benzo[a]pyrene	0.000014	Tier II SCV	(Suter and Tsao 1996)
Benzo[b]fluoranthene	0.00907	ESL	(USEPA 2004b)
Benzo[g h i]perylene	0.00764	ESL	(USEPA 2004b)
Fluoranthene	0.0019	2B CS	(MPCA 2004)
Fluorene	0.0039	Tier II SCV	(Suter and Tsao 1996)
Naphthalene	0.081	2B CS	(MPCA 2004)
Phenanthrene	0.0036	2B CS	(MPCA 2004)
Pyrene	0.0003	ESL	(USEPA 2004b)
PCP			
Pentachlorophenol[b]	0.0055(pH>6.95); Exp(1.005*[pH]-5.29) (pH<6.95)	2B CS	(State of Minnesota 2005)
VOC/SVOC			
1,1,2,2-Tetrachloroethane	0.61	Tier II SCV	(Suter and Tsao 1996)
1,1,2-Trichloro-1,2,2-trifluoroethane	0.098	Tier II SCV	(Suter and Tsao 1996)
1,2,4,5-Tetrachlorobenzene	0.003000	CSV	(USEPA 2004b)
1,2-Dichloroethylene, *trans*-[d]	0.5900	Tier II SCV	(Suter and Tsao 1996)
1,2-Dichloropropane	0.36	ESL	(USEPA 2004b)
2,4,6-Trichlorophenol	0.0049	ESL	(USEPA 2004b)
2,4-Dichlorophenol	0.011	ESL	(USEPA 2004b)
2,4-Dinitrophenol	0.0190	ESL	(USEPA 2004b)
2,6-Dinitrotoluene	0.0810	ESL	(USEPA 2004b)
2-Chloronaphthalene	0.000396	ESL	(USEPA 2004b)
2-Chlorophenol	0.024	ESL	(USEPA 2004b)

Table E1-3. Screening Values for Analytes in Surface Water

	Ecological Screening Benchmark		
Analyte	Concentration (mg/L)	Type	Source
2-Hexanone	0 099	Tier II SCV	(Suter and Tsao 1996)
Benzene[b]	0 114	2B CS	(State of Minnesota 2005)
Benzoic Acid	0 0420	Tier II SCV	(Suter and Tsao 1996)
Bis(2-ethylhexyl)phthalate	0.003	Tier II SCV	(Suter and Tsao 1996)
Carbon disulfide	0.015	ESL	(USEPA 2004b)
Carbon tetrachloride	0 0098	Tier II SCV	(Suter and Tsao 1996)
Chloroform	0.0280	2B CS	(Suter and Tsao 1996)
Dibenzofuran	0.0037	Tier II SCV	(Suter and Tsao 1996)
Ethylbenzene	0.068	2B CS	(MPCA 2004)
Isophorone	0.920	ESL	(USEPA 2004b)
Methylene chloride	2.2000	Tier II SCV	(Suter and Tsao 1996)
Nitrobenzene	0.2200	ESL	(USEPA 2004b)
N-Nitrosodiphenylamine	0.2100	Tier II SCV	(Suter and Tsao 1996)
o-Cresol	0 0670	SL	(USEPA 2004b)
p-Cresol	0 0250	ESL	(USEPA 2004b)
Phenol	0.123	2B CS	(MPCA 2004)
Styrene	0 0320	ESL	(USEPA 2004b)
Toluene	0.253	2B CS	(State of Minnesota 2005)
Trichloroethylene	0.0470	ESL	(USEPA 2004b)
Vinyl chloride	0.9300	ESL	(USEPA 2004b)
Xylenes, total	0 166	2B CS	(MPCA 2004)

Notes:
2B CS – refers to the Minnesota designated regulatory subclass 2B chronic standard for protection of water quality
CCC – criterion continuous concentration
ESL – ecological screening level
SCV – secondary chronic value
TH – total hardness

[a] Value added since previous table (Integral 2005)
[b] Value corrected since previous table (Integral 2005).
[c] Value computed assuming hardness is 100 mg/L, followed by equation for use with station-specific hardness value See Table E1-4 for location-specific screening values for these metals.
[d] 1,2-Dichloroethylene value (not trans-1,2-Dichloroethylene).

Table E1-4. Location-Specific Screening Level Values for Metals Based on Calculated Hardness (see Table E1-3 for Screening value equations used to derive these values)

		Ecological Screening Benchmark	
Analyte	Station	Calculated Hardness (mg/L)	Calculated Location-Specific Screening Level Value
Cadmium	CLDH-016566	156	0.0016
Cadmium	CLDH-028283	140	0.0015
Cadmium	FCCD-0102	197	0 0019
Cadmium	FCD-0102	170	0.0017
Cadmium	FCSW-0102	196	0.0019
Cadmium	HWY-0102	143	0.0015
Cadmium	PB-0102	143	0.0015
Cadmium	PBDH-018081	141	0.0015
Cadmium	PBDH-028081	131	0.0014
Cadmium	RR-0102	150	0.0016
Cadmium	WL-0102	152	0.0016
Cadmium, dissolved	FCCD-0102	197	0.0018
Cadmium, dissolved	FCD-0102	170	0.0016
Cadmium, dissolved	FCSW-0102	196	0.0017
Cadmium, dissolved	PB-0102	143	0.0014
Chromium	CLDH-016566	156	0.2976
Chromium	CLDH-028283	140	0.2727
Chromium	FCCD-0102	197	0.3614
Chromium	FCD-0102	170	0.3197
Chromium	FCSW-0102	196	0.3591
Chromium	HWY-0102	143	0.2772
Chromium	PB-0102	143	0.2768
Chromium	PBDH-018081	141	0.2739
Chromium	PBDH-028081	131	0.2579
Chromium	RR-0102	150	0.2884
Chromium	WL-0102	152	0.2923
Chromium, dissolved	FCCD-0102	197	0.3108
Chromium, dissolved	FCD-0102	170	0.2749
Chromium, dissolved	FCSW-0102	196	0.3089
Chromium, dissolved	PB-0102	143	0.2380
Copper	CLDH-016566	156	0.0129
Copper	CLDH-028283	140	0.0121
Copper	FCCD-0102	197	0.0150

Table E1-4. Location-Specific Screening Level Values for Metals Based on Calculated Hardness
(see Table E1-3 for Screening value equations used to derive these values)

		Ecological Screening Benchmark	
Analyte	Station	Calculated Hardness (mg/L)	Calculated Location-Specific Screening Level Value
Copper	FCD-0102	170	0.0137
Copper	FCSW-0102	196	0.0149
Copper	HWY-0102	143	0.0123
Copper	PB-0102	143	0.0122
Copper	PBDH-018081	141	0.0122
Copper	PBDH-028081	131	0.0116
Copper	RR-0102	150	0.0126
Copper	WL-0102	152	0 0128
Copper, dissolved	FCCD-0102	197	0.0144
Copper, dissolved	FCD-0102	170	0.0131
Copper, dissolved	FCSW-0102	196	0.0143
Copper, dissolved	PB-0102	143	0.0118
Lead	CLDH-016566	156	0.0056
Lead	CLDH-028283	140	0.0049
Lead	FCCD-0102	197	0.0076
Lead	FCD-0102	170	0.0063
Lead	FCSW-0102	196	0.0075
Lead	HWY-0102	143	0.0050
Lead	PB-0102	143	0.0050
Lead	PBDH-018081	141	0.0049
Lead	PBDH-028081	131	0.0045
Lead	RR-0102	150	0.0053
Lead	WL-0102	152	0.0054
Lead, dissolved	FCCD-0102	197	0.0060
Lead, dissolved	FCD-0102	170	0.0049
Lead, dissolved	FCSW-0102	196	0.0059
Lead, dissolved	PB-0102	143	0.0040
Nickel	CLDH-016566	156	0 2294
Nickel	CLDH-028283	140	0.2097
Nickel	FCCD-0102	197	0 2804
Nickel	FCD-0102	170	0.2470
Nickel	FCSW-0102	196	0.2786
Nickel	HWY-0102	143	0 2132
Nickel	PB-0102	143	0.2129
Nickel	PBDH-018081	141	0 2106
Nickel	PBDH-028081	131	0 1979
Nickel	RR-0102	150	0 2221
Nickel	WL-0102	152	0.2252

Table E1-4. Location-Specific Screening Level Values for Metals Based on Calculated Hardness (see Table E1-3 for Screening value equations used to derive these values)

		Ecological Screening Benchmark	
Analyte	Station	Calculated Hardness (mg/L)	Calculated Location-Specific Screening Level Value
Nickel, dissolved	FCCD-0102	197	0 2796
Nickel, dissolved	FCD-0102	170	0.2463
Nickel, dissolved	FCSW-0102	196	0.2777
Nickel, dissolved	PB-0102	143	0.2122
Zinc	CLDH-016566	156	0.1543
Zinc	CLDH-028283	140	0.1410
Zinc	FCCD-0102	197	0 1887
Zinc	FCD-0102	170	0.1662
Zinc	FCSW-0102	196	0.1875
Zinc	HWY-0102	143	0.1434
Zinc	PB-0102	143	0.1432
Zinc	PBDH-018081	141	0.1417
Zinc	PBDH-028081	131	0.1331
Zinc	RR-0102	150	0.1494
Zinc	WL-0102	152	0.1515
Zinc, dissolved	FCCD-0102	197	0 1860
Zinc, dissolved	FCD-0102	170	0 1639
Zinc, dissolved	FCSW-0102	196	0.1848
Zinc, dissolved	PB-0102	143	0.1412

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APPENDIX E2

ECOTOXICITY PROFILES

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Appendix E2
Ecotoxicity Profiles

Prepared for
International Paper
pursuant to
Unilateral Administrative Order Docket No. V-W-04-C-796

Prepared by

integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80021

September 28, 2007

CONTENTS

LIST OF ACRONYMS

AChE	acetylcholinesterase
Ah-R	aryl hydrocarbon receptor
ALAD	aminolevulinic acid dehydratase
AVS	acid-volatile sulfide
BAF	bioaccumulation factor
BCF	bioconcentration factor
BHC	benzene hexachloride
bw	body weight
COPEC	chemicals of potential ecological concern
CTR	critical tissue residue
DDT	dichlorodiphenyl-trichloroethane
DMBA	dimethyl benz[a]anthracene
DNA	deoxyribonucleic acid
DQO	data quality objective
dw	dry weight
Eco-SSL	ecological soil screening level
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
fw	fresh weight
HPAH	high molecular weight PAH
LD50	median lethal dose
LOAEC	lowest-observed-adverse-effects concentration
LOAEL	lowest-observed-adverse-effects level
LPAH	low molecular weight PAH
MATC	maximum acceptable toxicant concentrations
NOAEC	no-observed-adverse-effects concentration
NOAEL	no-observed-adverse-effects level
PAH	polycyclic aromatic hydrocarbon

PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-*p*-dioxins
PCDF	polychlorinated dibenzofurans
PCH	polychlorinated hydrocarbon
PCP	pentachlorophenol
ppb	parts per billion
ppm	parts per million
RAWP	risk assessment work plan
SSD	species sensitivity distribution
TCDD	tetrachlorodibenzo-*p*-dioxins
TCDF	tetrachlorodibenzofurans
TEQ	toxic equivalent
TRV	toxicity reference value
USACE	U.S. Army Corps of Engineers
ww	wet weight

1 INTRODUCTION

This appendix provides ecotoxicity profiles for all chemicals of potential ecological concern (COPECs) identified in soil and sediment for this baseline ecological risk assessment (ERA). Chemicals that are only COPECs because they exceed water quality criteria (WQC) in the forested wetland are not discussed in this appendix because the WQCs themselves provide the best community-level no effects threshold. The individual WQC are sufficient to address the assessment endpoint (survival, growth and reproduction of at least 95 percent of aquatic species). The profiles presented in this appendix reflect the ecological effects evaluation performed for this ERA consistent with U.S. Environmental Protection Agency guidance (USEPA 1997). Each profile briefly describes environmental chemistry, fate and transport, bioconcentration and bioaccumulation, and general toxicity for each chemical or group of chemicals. Each profile also identifies potential toxic effects associated with exposure of terrestrial and aquatic biota to these chemicals. Where applicable, toxicity reference values (TRVs) are identified for species considered representative of site-specific receptors. Site-specific receptors were selected to represent the following:

- Terrestrial plant communities
- Terrestrial invertebrate communities
- Terrestrial birds (American robin)
- Terrestrial mammals (short tailed shrew, meadow vole)
- Aquatic benthic invertebrate communities
- Fish (walleye, perch, white sucker)
- Semi-aquatic birds (kingfisher, great blue heron, mallard duck)
- Semi-aquatic mammals (muskrat, mink, raccoon)
- Snapping turtle.

Literature searches were performed to identify studies addressing toxicity of COPECs to receptors at the Site. For bird and mammal TRVs, this ERA draws heavily from two widely accepted reviews of wildlife TRVs: Sample et al. (1996) and the compilations associated with ecological soil screening values developed by EPA (Eco-SSL values, USEPA 2005g). Reliance on these sources is consistent with the risk assessment work plan (RAWP). Literature cited by EPA in support of the Eco-SSL values received careful and systematic scrutiny with oversight by a large panel of scientists. Literature was reviewed for development of Eco-SSLs and rated for acceptability using criteria similar to the data quality objectives (DQOs) for toxicity studies identified by Integral (2005). Therefore, when relevant to site-specific receptors and endpoints, documents supporting the Eco-SSL values were considered appropriate for developing TRVs for this ERA. Literature not identified or applied by Sample et al. (1996) or (USEPA 2005g) was also used to develop bird and mammal TRVs.

Consistent with Integral (2005), toxicity studies that evaluated survival, growth, or reproduction effects on birds and mammals following dietary exposure and that reported both a no-observed-adverse-effect level (NOAEL) and a lowest-observed-adverse-effect level (LOAEL) were preferentially selected as the basis of TRVs. Wherever possible, TRVs were selected from chronic tests. In general, an uncertainty factor of 10 was applied to convert from an acute or subchronic study exposure period to a chronic exposure period for bird and mammal TRVs. An uncertainty factor of 10 was also applied to convert from an unbounded LOAEL[1] to a NOAEL value, or to convert between major taxa where a TRV was lacking for a major taxonomic group.

All TRV values for birds and mammals are provided as doses. Where original toxicity studies reported effect levels as concentrations in food of test animals, and body weight and/or consumption rate were not reported, values used by Sample et al. (1996) for body weight and consumption rate provided the basis for conversions to dose values. Table E2-1 lists test species on which TRVs for birds and mammals were based and the default parameters for body weight and consumption rate for each. Dietary concentrations are presented in this appendix as dry weight (dw), unless otherwise noted.

Table E2-1. Default Body Weight and Consumption Rate for Test Species of Birds and Mammals.

BIRDS			MAMMALS		
Test Species	Body Weight (kg)	Consumption Rate (kg dw/day)	Test Species	Body Weight (kg)	Consumption Rate (kg/day)
American kestrel	NA	NA	Mink	1.0 [a]	0.137 [a]
Chicken	NA	NA	Mouse	0.03 [a]	NA
Heron	NA	NA	Rat	0.35 [a]	0.028 [a]
Japanese Quail	0.15 [a]	0.0169 [a]			
Mallard duck	1.13 [b]	NA			
Starling	NA	NA			
Turkey	NA	0.174 [a]			
NA – Not available					
[a] Sample et al. (1996)					
[b] USEPA (1993)					

TRVs for other species are expressed as concentrations in media, or as critical tissue residues (CTRs) associated with no effects on the study animal. No conversions were required for TRVs for fish, aquatic invertebrates, terrestrial vegetation, and terrestrial soil invertebrates.

[1] Usually the lowest dose administered in the toxicity study, a LOAEL is unbounded when a NOAEL cannot be identified.

2 METALS

2.1 ANTIMONY

Antimony is a naturally-occurring semi-metallic element. It exists in valences of 1, -3, +3, and +5. The tri- and pentavalent forms are the most stable and are of the most interest in biological systems (USEPA 2005g). Antimony trioxide is the most common industrial form of antimony, used in the production of flame retardant materials, and is the primary form found in the atmosphere (ATSDR 1992). Little is known about the occurrence of various forms of antimony in soil and water.

Ingested antimony is absorbed slowly and many antimony compounds are reported to be gastrointestinal irritants (USEPA 2005g). The toxic effects of antimony in mammals involve cardiovascular changes, including degeneration of the myocardium, arterial hypotension, heart dysfunction, arrhythmia, and altered electrocardiogram patterns (Rossi et al. 1987). The mode of action for antimony-induced cardiotoxicity is unknown (USEPA 2005g).

Vegetation and Soil Invertebrates

A search conducted by USEPA for literature describing effects of antimony on terrestrial plants did not identify any studies that met the criteria for derivation of a TRV (USEPA 2005g). Kabata-Pendias and Pendias (1984) provide a lowest-observed-adverse-effect concentration (LOAEC) of 5 mg/kg dw soil. This benchmark is cited by Efroymson et al. (1997b) for plants, with a low level of confidence for lack of detail.

EPA identified three studies appropriate for derivation of an Eco-SSL for soil invertebrates (USEPA 2005g). Toxicity studies on three soil invertebrates (*Eisenia fetida, Folsomia candida,* and *Enchytraeus crypticus*) identified EC20 values for antimony ranging from 30 to 194 milligram per kilogram (mg/kg) dw soil for reproductive effects (Kuperman et al. 2002; Phillips et al. 2002; Simini et al. 2002). The EC20 is the concentration at which 20 percent of the test population exhibits effects. The geometric mean of these concentrations is 78 mg/kg dw, which was applied in the ERA.

Birds

There is very little information regarding toxic effects of antimony on birds. A literature search conducted by EPA for studies concerning antimony and birds did not find any studies that met the criteria for derivation of a TRV (USEPA 2005g). No studies that met the DQOs for this ERA were identified for antimony toxicity to birds. The NOAEL for mammals, a conservative value based on a study in which dissolved antimony was administered to test animals in drinking water (discussed below) was multiplied by 0.1 to derive a NOAEL for antimony of 0.0059 mg/kg-day.

Mammals

The EPA identified 11 papers appropriate for evaluating toxic effects of antimony in mammals in development of the Eco-SSL values (USEPA 2005g). Of these, four key studies evaluating effects of oral exposure of rats to antimony were reviewed for this ERA. Three studies (Poon et al. 1998; Rossi et al. 1987; Schroeder et al. 1968) evaluated potential toxic effects of antimony in drinking water. Poon et al. (1998) found a NOAEL of 5.58 milligrams per kilogram body weight (bw) per day (mg/kg-day) for males and 6.13 mg/kg-day for females for growth effects following a 90-day drinking water exposure to potassium antimony tartrate. The LOAEL in the study was 42.17 mg/kg-day for males and 45.69 mg/kg-day for females. Schroeder et al. (1968) identified an unbounded LOAEL of 5 parts per million (ppm) or milligram per liter (mg/L) in drinking water administered over the natural lifetime for effects on longevity. Using average body weight and consumption data for rates, Sample et al. (1996) reported that the5 mg/L potassium antimony tartrate in drinking water is equivalent to an antimony dosage of 1.25 mg/kg-day (Sample et al. 1996). In a subchronic study, Rossi et al. (1987) reported a NOAEL of 0.1 mg antimony trichloride per deciliter (mg/dL) in drinking water for growth effects on pups. This is equivalent to a chronic NOAEL of 0.059 mg/kg-day (USEPA 2005g).

In the only study evaluated by EPA in which antimony was administered by food, Hext et al. (1999) reported an unbounded NOAEL of 1686 mg/kg-day in male rats and 1879 mg/kg-day in female rats (average of 1783 mg/kg-day) for growth effects and gross toxicity. Antimony in this study was administered in the diet as antimony trioxide.

The NOAEL values associated with antimony ingestion in drinking water and food differ by more than four orders of magnitude. One explanation for this difference may be that the drinking water studies were based upon soluble forms of antimony that may be more bioavailable than antimony in a food matrix. The dietary study was based on a diet containing antimony trioxide. There is insufficient data in the literature to quantify the potentially different bioavailability and toxicokinetics of these different forms of antimony. Therefore, the most conservative NOAEL for oral exposure via drinking water (0.059 mg/kg-day) and LOAEL (0.59 mg/kg-day) were selected as the TRVs for mammals with the understanding that they may be extremely conservative numbers for bird and mammal receptors.

Aquatic Biota

Insufficient data are available to develop an ambient water quality criterion for the protection of aquatic life, but a search on EPA's ECOTOX database results in nine studies on the toxicity of antimony to aquatic life, including four LC50 values for freshwater species, one EC50, and one NOEC. LC50s for antimony range from 0.3 mg/L for a toad to 15 mg/L for rainbow trout after 28 days of exposure. The NOEC for the survival endpoint in sheepshead minnow was 6.2 mg/L after 4 days of exposure. Because of the short-term exposure, this value was multiplied by 0.1 to provide a subchronic survival no-observed-adverse-effects concentration (NOAEC) for fish. Jarvinen and Ankley (1999) provide a survival NOAEC for juvenile trout of 5 mg/kg wet weight (ww) following exposure for 4 days.

2.2 BARIUM

Barium is a naturally occurring metal used in the manufacture of ceramics, pyrotechnics, paints, enamels, and television tubes, and can be released to the environment through related industrial processes and through coal and oil combustion. Barium is more soluble in sandy soils with low pH and low organic carbon content. In biota, the properties of barium allow it to replace calcium, particularly in the release of neurotransmitters and adrenal catecholamines (USEPA 2005a).

Vegetation and Soil Invertebrates

Barium is not a COPEC in terrestrial systems at the Site; therefore, TRVs were not identified for plants or soil invertebrate receptors.

Birds

There is very little data available to describe the toxicity of barium to birds. Only one study was identified by USEPA (2005a) and it did not meet quality criteria for development of a TRV. Therefore, the NOAEL for mammals (below) was multiplied by an uncertainty factor of 0.1 to derive a general avian TRV of 6.15 mg/kg-day, assumed to represent a no effects level in birds.

Mammals

USEPA (2005a) identified 27 studies of sufficiently high quality for use in developing a TRV, more than half of which are for endpoints relating to biochemical responses, behavior, physiology, or pathology. Four studies provide TRVs relating to survival, growth, or reproduction; of these, two provide both a NOAEL and a LOAEL and are therefore preferred. The lowest NOAEL/LOAEL pair was provided by Dietz et al (1992), and was derived from exposure of juvenile rats to barium in drinking water for 92 days. There was no significant difference from controls in body weight or in mortality at 61.5 mg/kg-day; the LOAEL for these endpoints was 121 mg/kg-day.

Aquatic Biota

Data on the toxicity of barium to aquatic life are insufficient to develop an ambient water quality criterion for the protection of aquatic life. However, some information is available on EPA's ECOTOX database. Two studies provide data for vascular plants, indicating that EC50s for growth in vascular plants (*Myriophyllum spicatum, Lemna minor*) range from 26 to 83.8 mg/L of barium, and population level EC50s for biomass production are from 41.2 to 103 mg/L. One study provides an LC50 for fish (sheepshead minnow) of 500 mg/L; LC50s for zooplankton and algae range from 320 to 530 mg/L. CTRs for barium in aquatic life are not provided by Jarvinen and Ankley (1999), and were not found in the literature.

2.3 BERYLLIUM

Beryllium is a naturally-occurring metal that may be associated with various manufacturing waste streams. Beryllium enters the environment principally from coal combustion. The most prevalent chemical form in the environment is beryllium oxide (USEPA 2005b). Within typical environmental soil pH ranges (i.e., pH 6–8), beryllium oxide is highly insoluble, and thus highly immobile in soil. If beryllium is bioavailable in soil, it can be assimilated by plants. Some plant species may act as accumulators of beryllium, however, it is not believed to biomagnify within food chains (USEPA 2005b).

Vegetation and Soil Invertebrates

Beryllium is not a COPEC in terrestrial systems at the site; therefore, TRVs were not identified for plants or soil invertebrate receptors.

Birds

A literature search conducted by EPA for studies concerning toxicity of beryllium and birds did not identify any studies that met the criteria for derivation of a TRV (USEPA 2005b). No studies that met the DQOs for this ERA were identified for beryllium toxicity to birds. Therefore, a TRV for beryllium for bird receptors was estimated by multiplying the mammalian TRV by an uncertainty factor of 0.1 to get 0.0523 mg/kg-day. This is considered a NOAEL for this project.

Mammals

Four studies evaluating toxic effects of beryllium to mammals were identified by USEPA (2005b) sufficient for derivation of a TRV (Freundt and Ibrahim 1990; Goel et al. 1980; Schroeder and Mitchener 1975a; 1975b). Of these, the study by Schroeder and Mitchener (Schroeder and Mitchener 1975a) resulted in the highest NOAEL (0.532 mg/kg-day) less than the lowest LOAEL for growth and reproductive effects. This study evaluated lethality of beryllium administered in drinking water on rats. None of the studies evaluated identified both a NOAEL and a LOAEL for beryllium. The NOAEL of 0.532 mg/kg-day based on the Schroeder and Mitchener (1975a) study was selected as the TRV for mammals.

Aquatic Biota

CTRs for beryllium in aquatic life are not provided by Jarvinen and Ankley (1999), and were not found in the literature. Insufficient data are available to develop a water quality criterion. The only available aquatic benchmark for beryllium is the surface water screening value cited in Appendix E1: 0.0006 mg/L.

2.4 CADMIUM

There is no evidence that cadmium, a relatively rare metal, is biologically essential or beneficial. On the contrary, cadmium has been implicated as a cause of human mortality and various deleterious effects in fish and wildlife. Freshwater biota are the most sensitive organisms to cadmium exposure, with toxicity inversely proportional to water hardness.

Cadmium bioconcentrates in aquatic organisms, primarily in the liver and kidney (USEPA 1999). Cadmium accumulated from water is slowly excreted, while cadmium accumulated from food is eliminated more rapidly. Metal-binding, proteinaceous metallothioneins appear to protect vertebrates from deleterious effects of high metal body burdens (Eisler 1985).

Vegetation and Soil Invertebrates

Cadmium is not essential for plant growth. If present in an available form, it is readily taken up by the roots, translocated through the plant and accumulated (Efroymson et al. 1997b). Cadmium depresses uptake of essential minerals and is toxic at low concentrations. Mechanisms of cadmium toxicity in plants include reduced photosynthetic rate, poor root system development, reduced conductivity of stems, and ion interactions in the plant (Efroymson et al. 1997b).

The toxic effects of cadmium on plants have been extensively studied. USEPA (2005c) reviewed 62 acceptable studies, and used 14 of these to derive an Eco-SSL value protective of plants. The geometric mean of the EC10 and maximum acceptable toxicant concentrations reported (MATC) is 32 mg/kg soil.

USEPA (2005c) identified 32 acceptable studies of the effects of cadmium on soil invertebrates. Of these, 10 were used for derivation of the Eco-SSL value, representing three species. The value applied in this ERA is 140 mg/kg, and is the geometric mean of the EC10 and MATC values reported by the acceptable studies.

Birds

Birds are comparatively resistant to the toxicity of cadmium, and mallards and chickens have been reported to tolerate 200 mg/kg of cadmium in diets for protracted periods. When present at sufficiently high doses, sublethal effects of cadmium in birds are similar to those in other animals and include growth retardation, anemia, and testicular damage.

EPA reviewed 35 papers that evaluated toxicity of cadmium to birds (USEPA 2005c). Based on the various NOAEL values for growth and reproduction, EPA calculated a TRV of 1.47 mg/kg-day (USEPA 2005c). This value was selected as the NOAEL for this ERA. The NOAEL is lower than the lowest bounded LOAEL of 2.37 mg/kg-day for reproductive effects in a 12-month dietary study of chicken (USEPA 2005c), which was selected as the LOAEL for this ERA.

Mammals

Mammals are relatively resistant to the toxicity of cadmium. The lowest oral doses producing death in rats and guinea pigs range from 150 to 250 mg/kg. Absorption and retention of cadmium decrease with prolonged exposure (Eisler 1985). Cadmium absorption through ingestion is inversely proportional to intake of other metals, especially iron and calcium.

EPA identified several papers containing data for cadmium toxicity to mammals (USEPA 2005c). From these, the most relevant and appropriate study on reproductive effects was selected as the basis of the TRV for mammals. Sutou et al. (1980) administered cadmium to male and female rats for six weeks over the mating and gestation period. A NOAEL of 1.0 mg/kg-day and a LOAEL of 10 mg/kg-day were reported for decreased implantation and live fetuses (Sutou et al. 1980). These were used as the cadmium TRVs for mammals.

Aquatic Biota

Exposure routes for aquatic organisms include ingestion and respiration. Cadmium concentrations of 0.8 to 9.9 μg/L in water were lethal to several species of aquatic insects, crustaceans, and teleosts; and concentrations of 0.7 to 570 μg/L were associated with sublethal effects such as decreased growth, inhibited reproduction, and population alterations (Eisler 1985). These effects were most pronounced in waters of comparatively low alkalinity. The CTR of 0.27 mg/kg ww whole body for *Daphnia magna* identified by Jarvinen and Ankley (1999) was selected for this ERA as a TRV protective of aquatic invertebrates.

Many studies evaluate the toxicity of cadmium in water to fish and invertebrates as critical body residues, but most of these are for the survival endpoint and are not applied in this ERA because of unacceptable uncertainties associated with critical body residues for the mortality endpoint (Integral 2005). Studies of sublethal endpoints for fish exposed to cadmium include Kumada et al. (1973), which found no effect on growth in rainbow trout (*Oncorhynchus mykiss*) with whole body residues of 0.54 mg/kg wet weight (ww). Because salmonid fishes are often the most sensitive to toxicants, other studies evaluating non-salmonid species were considered in evaluation of risks to fish at the site:

- Cope et al. (1994) reported no effect on growth or survival in bluegill (*Lepomis macrochirus*) with whole body residues of 1.33 mg/kg after 28 days exposure.
- Kumada et al. (1973) reported no effect on survival or growth in dace (*Triborodon hakonensis*) after 112 days exposure in water and with whole body residues of cadmium at 0.69 mg/kg ww.

Results of the Kumada et al. (1973) study were applied for this risk assessment.

2.5 COPPER

Copper occurs naturally in many animals and plants and is an essential micronutrient that animals incorporate into several essential enzymes. Copper may exist in two oxidation states: +1 or +2. Copper (+1) is unstable and, in aerated water over the pH range of most natural waters (6 to 8), oxidizes to the +2 state (USEPA 1999). Copper is not biodegraded or transformed and does not bioaccumulate (USEPA 1999). Adverse effects from copper exposure include hematological, hepatic, developmental, immunological, and renal impairment in vertebrates.

Copper may be toxic in aquatic environments and affects fish, invertebrates, and amphibians (Eisler 1998). Toxic effects in birds include reduced growth rates, lowered egg production, and developmental abnormalities. Toxicity in mammals occurs in a wide range of animals and includes effects such as liver cirrhosis, necrosis in kidneys and the brain, gastrointestinal distress, lesions, and low blood pressure.

Vegetation and Soil Invertebrates

Copper is a micronutrient essential for plant nutrition (Efroymson et al. 1997b). It is required as a co-factor for many enzymes and is an essential part of a copper protein involved in photosynthesis. Copper occurs as part of enzymes and enzyme systems in plants. Copper can be transported in the xylem and phloem of plants complexed with amino acids (Efroymson et al. 1997b). The basic deleterious effect of copper at phytotoxic levels is related to the root system where it interferes with enzyme functioning. It also strongly interferes with photosynthesis and fatty acid synthesis. The most common symptoms of copper toxicity in plants are reduced growth, poorly developed root system, and leaf chlorosis. Phytotoxicity appears at concentrations of copper in soil as low as 100 mg/kg dw (Efroymson et al. 1997b; Kabata-Pendias and Pendias 1984).

Beyer and Cromartie (1987) and Kabata-Pendias and Pendias (1984) showed that copper has a moderate potential to bioaccumulate in earthworms (bioaccumulation factors [BAFs] ranging from 0.01 to 2.5 depending upon soil conditions). Soil invertebrates show adverse effects at copper concentrations as low as 60 mg/kg in soil (Efroymson et al. 1997a). This value was applied as the CTR in this ERA.

Birds

Experiments with domestic poultry show that copper accumulates in livers of mallard ducklings at dietary concentrations as low as 15 mg/kg dw ration. Mehring et al. (1960) reported a NOAEL of 570 mg/kg copper and a LOAEL of 749 mg/kg dw for dietary copper exposure of chicks over a period of 10 weeks. Using standard assumptions regarding body weight (0.534 kg) and food consumption (0.044 kg/day), Sample et al. (1996) derived a NOAEL of 47 mg/kg-day and a LOAEL of 62 mg/kg-day. An uncertainty factor of 10 was applied for conversion from subchronic to chronic exposure. Therefore, a NOAEL TRV of 4.7 mg/kg-day and a LOAEL TRV of 6.2 mg/kg-day for copper were selected for birds.

Mammals

Copper can be lethal to mammals at high doses (Eisler 1998). Copper is lethal when eaten for extended periods at more than 80 mg/kg diet in sheep (equivalent to 5.1–10.7 mg/kg-day), more than 238 mg/kg diet in pigs, and more than 4,000 mg/kg diet in rats (equivalent to more than 133 mg/kg-day). Adverse sublethal effects of copper to sensitive mammals occur at dietary levels ranging from 7.9 mg/kg-day in food to 400 mg/L in drinking water.

Chronic toxicity of copper sulfate on the reproduction of mink was evaluated by Aulerich et al. (1982). Data from this study were used by Sample et al. (1996) to support development of a NOAEL of 11.7 mg/kg-day and a LOAEL of 15.1 mg/kg-day for kit mortality. Based on this study, a NOAEL TRV of 11.7 mg/kg-day and a LOAEL TRV of 15.1 mg/kg-day was selected for mammals.

Aquatic Biota

Copper exerts toxic effects by binding to deoxyribonucleic acid (DNA) or by generating free radicals (USEPA 1999). Aqueous copper speciation and toxicity depend on the ionic strength of the water. Primarily it is the dissolved cupric ion (Cu+2) and possibly hydroxyl complexes that are toxic to aquatic biota; copper complexes consisting of carbonates, phosphates, nitrates, ammonia, and sulfates are weakly toxic or nontoxic (USEPA 2000a). In hard waters, 43– 88 percent of the copper is associated with suspended solids and not available to biota (Eisler 1998). In general, mortality of tested aquatic species is greatest under conditions of low water hardness (as measured by $CaCO_3$), starvation, elevated water temperatures, and early developmental stages.

Many aquatic species are sensitive to dissolved concentrations of copper in the range of 1 to 20 µg/L (USEPA 2000a). Sensitive species of representative freshwater plants and animals die within 96 hours at waterborne copper concentrations of 5.0– 9.8 µg/L (Eisler 1998). The most sensitive freshwater species have LC50 (96 hour) values between 0.23 and 0.91 µg/L and include daphnids (*Daphnia* spp.), amphipods (*Gammarus pseudolimnaeus*), snails (*Physa* spp.), and chinook salmon (*Oncorhynchus tshawytscha*). The LC50 value indicated the lethal concentration for 50 percent of the exposed population. In aquatic invertebrates, copper causes gill damage at high concentrations, and in fishes it interferes with osmoregulation (Eisler 1998).

Most studies reporting CTRs of copper for aquatic invertebrates examined lethal endpoints, involve marine species, or do not provide NOAECs and lowest-adverse-effect concentrations (LOAECs). Several NOAECs are available, and illustrate the interspecies variability in response to copper:

- Stebbing and Pomroy (1978) exposed hydras (*Hydra littoralis*) to copper in water for 11 days. The reproductive NOAEC from this study is 3.4 mg/kg ww, and the LOAEC is 4.4 mg/kg ww.

- Borgmann and Norwood (1997) exposed the amphipod *Hyalella azteca* to copper in sediment for 28 days, and found no effect on growth in *Hyalella* specimens with 30.4 mg/kg ww.

One study was found reporting whole body concentrations of copper in fish associated with sublethal endpoints.

2.6 LEAD

Lead has no nutritional or biochemical function (NAS 1980). The mechanism by which lead acts is believed to be indirect interference in normal metal-dependent enzyme functions at specific cellular sites. Predicting the accumulation and toxicity of lead is difficult since its effects are influenced to a large degree by interactions among physical, chemical, and biological variables. Under controlled conditions, lead adversely affects survival, growth, reproduction, development, and metabolism of most species (Eisler 1998). In general, organolead compounds are more toxic than inorganic lead compounds; and young, immature organisms are more susceptible to lead's effects (Eisler 1998).

The disposition of lead in the body is dependent on diet, growth rate, and physiological stress. Of the lead that is available, approximately 90 percent accumulates in bones (NAS 1980). Lead absorption is 10 percent or less in adult mammals; young mammals have been shown to absorb lead at a much higher rate that adults. In addition to storage of lead in bone, lead also accumulates in the kidney.

In vertebrates, lead modifies the structure and function of the kidney, bone, central nervous system, and the hematopoietic system. It produces adverse biochemical, histopathological, neuropsychological, ferotoxic, teratogenic, and reproductive effects. Birds and mammals exhibit effects from lead poisoning such as damage to the nervous system, kidneys, liver, sterility, growth inhibition, developmental retardation, and detrimental effects in blood (Eisler 1988). Irreversible central nervous system damage and decreased intelligence at extremely low doses of lead have been observed in mammals (ATSDR 1997).

Inhibition of blood δ-aminolevulinic acid dehydratase (ALAD), an enzyme critical in heme formation, has been observed as a result of exposure to lead in a variety of fish, invertebrates, and birds (USEPA 2000a).

Vegetation and Soil Invertebrates

Lead does not play an essential role in plant metabolism (Kabata-Pendias and Pendias 1984). Lead forms stable complexes with organic matter in soils. Bioavailability of soil lead is highly influenced by its mineral state. Inorganic lead is the predominant form in soils, where it can exist in more than 200 mineral forms. When lead is dissolved, it is readily taken up by plants. While lead in plant tissue may be positively correlated with lead concentrations in soils, lead

generally remains in root tissue (Kabata-Pendias and Pendias 1992). Lead binds to the outside of roots, in the apoplast, and in cell walls and organelles of absorbing roots.

The phytotoxicity of lead is relatively low compared with other trace elements. It affects mitochondrial respiration and photosynthesis by disturbing electron transfer reactions. The primary symptom of lead toxicosis is reduction of root and shoot growth. Miller et al. (1977) exposed corn seeds to loamy sand spiked with lead for 31 days. At a soil lead concentration of 250 mg/kg, a 42 percent reduction in plant weight was observed; at a soil lead concentration of 125 mg/kg, no difference in growth was observed relative to controls. Other studies of lead toxicity to plants were not selected because they did not provide a NOAEC, they were tested in unusually low or high pH soils, or they were tested in soils unlike soils at the site. The results of Miller et al. (1977) were applied for this ERA.

Beyer and Cromartie (1987) and Kabata-Pendias and Pendias (1984) showed that lead has potential to bioaccumulate in earthworms (BAFs ranging from 0.01 to 228 depending upon soil conditions). USEPA (USEPA 2005d) used four acceptable studies of toxicity of lead to soil invertebrates to derive the Eco-SSL value of 1,700 mg/kg dw soil. This value is the geometric mean of the results of these four studies, all of them using one species of Collembola, *Folsomia candida.*

The endpoint was reproduction. Organic matter of soils was 10 percent in all of these studies, substantially higher than at the Site.

Birds

Among sensitive species of birds, survival was reduced at doses of 75 to 150 mg lead 2+ /kg bw or 28 mg alkyl lead/kg bw, reproduction was impaired at dietary levels of 50 mg lead 2+ /kg, and signs of poisoning were evident at doses as low as 2.8 mg alkyl lead/kg bw (Eisler 1988). EPA identified 54 papers containing relevant toxicity data for birds (USEPA 2005d). The TRV developed by EPA is equal to the highest bounded NOAEL of 1.63 mg/kg-day based on a dietary study of reproductive effects in chicken (*Gallus domesticus*) (Edens and Garlich 1983). EPA also reports a LOAEL of 3.26 mg/kg-day for egg production in chickens based upon the same study. Therefore, the NOAEL of 1.63 mg/kg-day and the LOAEL of 3.26 mg/kg-day were selected as the TRVs for birds for this ERA.

Mammals

Among sensitive species of mammals, survival was reduced at acute oral doses as low as 5 mg/kg bw in rats, at chronic oral doses of 0.3 mg/kg bw in dogs, and at dietary levels of 1.7 mg/kg bw in horses. EPA identified 219 papers containing relevant toxicity data for mammals (USEPA 2005d). The TRV developed by EPA is equal to the highest bounded NOAEL of 4.7 mg/kg-day based upon a drinking water study of effects on growth of rats (Kimmel et al. 1980). EPA also identified a LOAEL of 8.9 mg/kg-day based upon the same study. Therefore, the NOAEL of 4.7 mg/kg-day and the LOAEL of 8.9 mg/kg-day were selected as the TRVs for mammals for this ERA.

Aquatic Biota

In aquatic environments, dissolved lead is the most toxic form; organolead compounds are much more toxic to aquatic organisms than are inorganic lead compounds (Eisler 1988; USEPA 2000a). The common forms of dissolved lead are lead sulfate, lead chloride, lead hydroxide, and lead carbonate, but the distribution of salts is highly dependent on the pH of the water. Most lead entering surface waters precipitates in sediment as carbonates or hydroxides. Bioavailability from sediment is controlled by the sediment organic content and acid-volatile sulfide (AVS) concentration (USEPA 2000a).

Lead is accumulated by aquatic organisms equally from water and through food (USEPA 2000a). Although methylated lead is rapidly bioaccumulated from the water by trout, there is no evidence that lead biomagnifies in the aquatic environment.

Holcombe et al. (1976) provide a growth NOAEC in whole bodies of third generation juvenile brook trout (*Salvelinus fontinalis*) ranging from 2.5 to 5.1 mg/kg ww, and LOAECs ranging from 4.0 to 8.8 mg/kg ww. Early life stages of brook trout are more sensitive to lead exposures than adults. Other available studies report concentrations in individual tissues, not whole body concentrations. Therefore, Holcombe et al. (1976) was used for this ERA.

Few studies are available documenting lead CTRs for freshwater invertebrates. No effect on growth of the zebra mussel (*Dreissena polymorpha*) was observed at whole body concentrations of 36 mg/kg bw. This was the lowest CTR for lead in aquatic invertebrates cited by Jarvinen and Ankley (1999) and was used in this study.

2.7 MERCURY

Mercury is a highly toxic, non-essential element (NAS 1980; USEPA 1999). Mercury exists in three valence states: mercuric (Hg^{2+}), mercurous (Hg^{1+}), and elemental (Hg^{0}) mercury. Common bacteria convert inorganic forms of mercury to organic forms (Matilainen et al. 1991). Inorganic mercury compounds are less toxic than organomercury compounds, with methylmercury being of greatest concern for potential to cause toxicity. Methylmercury is highly stable and bioaccumulates and biomagnifies in food chains (USEPA 1999). The majority of mercury detected in biological tissues is present in the form of methylmercury.

The mechanism of mercury toxicity in animals is interference with metabolism and cell division. Mercury binds strongly with sulfhydryl groups causing inhibition or inactivation of proteins containing thiol ligands and ultimately leading to meiotic disturbances (USEPA 1999). In all vertebrate receptors, the target organs are the kidney and central nervous system.

At low doses to birds and mammals, mercury adversely affects reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, hearing, histology, and metabolism. In mammals, methylmercury irreversibly damages the central nervous system and can also be teratogenic and mutagenic. For all organisms tested, early

developmental stages were the most sensitive to mercury. Numerous biological and abiotic factors modify the toxicity of mercury compounds, sometimes by an order of magnitude or more, but the mechanisms are not clear (Eisler 1987a)

Vegetation and Soil Invertebrates

Mercury is not an essential plant nutrient (Kabata-Pendias and Pendias 1984). Although mercury in solution is readily taken up by plants, the correlation between levels in soils and plants is generally weak because mercury in soils is not bioavailable to plants. Organic forms of mercury may be translocated to a greater degree than inorganic forms in some plants (Efroymson et al. 1997a). Mercury appears to interfere with sulfur-containing enzymes, disrupt the metabolic processes of plants (Kabata-Pendias and Pendias 1984), and inhibit potassium uptake. Symptoms of mercury toxicity include stunting of seedling and root growth, and the inhibition of photosynthesis that leads to reduced growth and yield. Suszcynsky and Shann (1995) grew tobacco seedlings in solutions including mercury and found that dry weights of roots and shoots were reduced by half in seedlings grown in 1 mg/L mercury as $HgCl_2$. A solution with 0.1 mg/L mercury had no effect on these endpoints. The use of a soluble form of mercury in this study probably overestimates toxicity to plants. This was the only study available to provide a TRV for plants; concentration of mercury in water was used to estimate an equivalent concentration in soil using the sediment-water partitioning coefficient (K_d) for mercury, in Appendix E4. The NOAEC was derived as 5.2 mg/kg soil, and the LOAEC is 52 mg/kg dw.

Kabata-Pendias and Pendias (1984) report that mercury has a low potential to accumulate in earthworms (BAFs ranging from 0.33 to 0.40 depending on test conditions). Soil invertebrates show reduced survival and reduced cocoon production at a mercury concentration of 0.5 mg/kg (Efroymson et al. 1997a).

Birds

Hill and Schaffner (1976) found a NOAEL of 4 mg/kg diet for reproductive effects in Japanese quail. The LOAEL was 8 mg/kg diet for decreased fertility and hatchability of eggs. Using the data reported in this study, Sample et al. (1996) developed a NOAEL intake of 0.45 mg/kg-day and a LOAEL intake of 0.9 mg/kg-day. Heinz (1979) administered methyl mercury dicyandiamide in the diet to 3 generations of mallard duck. This study reported a chronic LOAEL of 0.5 mg/kg diet for decreased production of eggs and ducklings in the third and second generation, respectively. However, 0.5 mg/kg diet did not result in toxic effects in the first generation. Exposure to the same concentration at the same location is not likely to occur over three consecutive generations of birds at the Site. Therefore, for the purposes of this ERA, 0.5 mg/kg diet was considered to represent a NOAEL for mercury in ducks. A dose value of 0.069 mg/kg-day was developed by Integral using an assumed body weight of 1.13 kg and consumption rate of 0.156 kg/day from the study. The NOAEL of 0.069 mg/kg-day was selected as a NOAEL TRV for birds. The LOAEL of 0.9 mg/kg-day for reproductive effects from the Hill and Schaffner (1976) study was selected as the LOAEL TRV.

Mammals

Lethal concentrations of total mercury to mammals varied from 0.1 to 0.5 mg/kg-day and 1.0 to 5.0 mg/kg in the diet for mammals (Heinz 1979). For sensitive mammals, these levels were 250 µg/kg-day, or 1,100 µg/kg diet (Heinz 1979).

Adverse effects from methyl mercury occur in mammals at oral doses ranging from 0.045 to 0.418 mg/kg-day depending on body size and species according to a study of methyl mercury chloride toxicity to rats and mink (Sample et al. 1996).

Wobeser et al. (1976) administered methyl mercury chloride in the diet to mink over a period of 93 days. They found a NOAEL of 1.1 mg/kg diet and a LOAEL of 1.8 mg/kg ww diet for mortality, weight loss and behavioral abnormalities. Sample et al. (1996) used the data from this study to calculate a NOAEL of 0.015 mg/kg-day and a LOAEL of 0.025 mg/kg-day, which were selected as the TRVs for mammals for this ERA.

Aquatic Biota

There is a high potential for bioaccumulation and biomagnification of mercury as methylmercury in aquatic food webs, with concentrations reported in fish up to 100,000 times the ambient water concentrations (Eisler 1987a). Methylmercury is highly water soluble and has an octanol-water partition coefficient (K_{ow}) that varies dependent upon the pH and ionic strength of water (Major et al. 1991). Mercury adversely affects reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange in marine and freshwater organisms. Lethal concentrations of total mercury to sensitive, representative organisms varied from 0.1 to 2.0 µg/L for aquatic fauna. Reproduction was inhibited among sensitive species of aquatic organisms at water concentrations of 0.03 to 0.1 µg/L. Many studies with invertebrates do not report concentrations in whole bodies. Biesinger et al. (1982) reported a reproduction NOAEC in tissue of cladocerans (*Daphnia magna*) of 3.05 mg/kg ww and a LOAEC of 4.66 mg/kg ww.

Studies of mercury suggest that fish are not as sensitive to mercury as they are to other toxicants. In evaluating effects of exposures to mercury to multiple generations of brook trout, McKim et al. (1976) reported no effect on the survival, growth, or reproduction in juveniles with body residues of 3.4 mg/kg ww. This study provides a LOAEC for these endpoints of 9.4 mg/kg ww (McKim et al. 1976).

2.8 MOLYBDENUM

Molybdenum is a beneficial or essential micronutrient found in all living organisms (Eisler 1989a). It occurs in five oxidation states, with Mo(IV) and Mo(VI) the most common. In soil and natural waters, the dominant form is molybdate anion. Molybdenum deficiency and toxicity often result from interactions with other metals, particularly copper. Generally, signs of toxicity in humans include anemia and elevated uric acid in the blood, while in animals signs additionally include a copper deficiency, joint abnormalities, gastrointestinal irritation, reduced skeletal ossification, and hair discoloration. There is no evidence that molybdenum causes cancer.

Vegetation and Soil Invertebrates

Molybdenum is not a terrestrial COPEC for the Site, so no soil TRVs for molybdenum were derived.

Birds

A TRV calculated by Sample et al (1996) was based on a study in which chickens were fed three dose levels of molybdenum in food for 21 days, including during reproduction. In the lowest dose, there was failure of all eggs to survive to hatching. The LOAEL calculated by Sample et al (1996) for molybdenum was 35.3 mg/kg bw-day. The NOAEL was derived by multiplying this value by 0.1, resulting in 3.53 mg/kg bw-day. These values were used in risk calculations for birds exposed to molybdenum.

Mammals

For mammals, a TRV was developed by Sample et al. (1996) based on a study in which mice were administered molybdenum in both food and drinking water for three generations. Only one dose level was tested, and the effect documented was reduced body weight of juveniles at birth. Sample et al (1996) applied general assumptions about the body weight and food and water consumption rates of mice to derive a LOAEL for reproduction from this study of 2.6 mg/kg-day. By multiplying this value by 0.1, a NOAEL of 0.26 mg/kg-day is derived..

Aquatic Biota

Tissue-based TRVs as CTRs for fish and aquatic invertebrates for molybdenum were not available in the literature (Jarvinen and Ankley 1999). Eisler (1989a) provides several benchmarks for molybdenum, including 60 mg/L as a no-effects level in amphipod after 96 hours, and 18.5 mg/L as a no-effects level on survival, growth and blood hematocrit in juvenile trout. The lowest LC50 for aquatic species reported in Eisler's (1989a) report is 70 mg/L for fathead minnow after 96 hours. These are considerably higher than the screening value provided by Suter and Tsao (1996) for water of 0.370 mg/L, which provides the primary aquatic benchmark for molybdenum in this risk assessment.

2.9 SELENIUM

Selenium is a beneficial or essential element for some plants and animals at low ppb concentrations. It occurs naturally in the environment, especially with sulfide minerals of iron, lead and copper. In nature, selenium exists in five valence states (2–, hydrogen selenide; 0, elemental selenium; 2+, selenium dioxide; 4+, selenite; and 6+, selenate).

Selenium may favorably or adversely affect growth, survival and reproduction of plants, invertebrates, fish, birds and mammals. Sensitivity to selenium is extremely variable in all classes of organisms (Eisler 1985). In some plants and animals, selenium constitutes part of the enzyme glutathione peroxidase and may have a role in the enzyme formic dehydrogenase and Vitamin E. Some animals require selenium-containing amino acids. Bioavailability of selenium is greater in plant foods than in foods of animal origin (Eisler 1985).

Acute selenium poisoning associated with ingestion of seleniferous plants containing 400–800 mg/kg selenium has been extensively documented in domestic livestock. Chronic selenosis in mammals may be induced by dietary exposures to selenite, selenate, or seleniferous plants at dietary concentrations between 1 mg/kg (rat) and 44 mg/kg (horse) (Eisler 1985). Chronic selenosis is characterized by skin lesions, lymph-channel inflammation, loss of hair/nails, anemia, enlarged organs, fatigue, lassitude, and dizziness. Elevated levels of selenium in the diet are also associated with reproductive abnormalities, including congenital malformation, selective bioaccumulation and growth retardation (Eisler 1985).

Vegetation and Soil Invertebrates

Selenium is not proven to be essential for plant growth. Active uptake of selenium most likely occurs as selenate and selenium is translocated to all parts of the plant (Efroymson et al. 1997b). Toxicity symptoms include chlorosis, stunting and yellowing of leaves. Efroymson et al. (1997b) identified a toxicity benchmark of 1 mg/kg selenium in soil for plants.

Selenium concentrations in soil as low as 70 ppm selenium in soil adversely affect growth and reproduction in earthworms (Efroymson et al. 1997a).

Birds

Domestic chickens (*Gallus domesticus*) are extremely sensitive to selenium; reduced hatching of eggs was observed at 6–9 mg/kg selenium in feed (Ort and Latshaw 1978). In Japanese quail, reduced hatching of eggs was observed at 6–12 mg/kg dietary selenite (El-Begearmi et al. 1977). There is evidence that selenium may have severe reproductive effects in aquatic wild birds exposed to very high levels of selenium in surface water and aquatic biota (Ohlendorf et al. 1986).

A study by Heinz et al. (1989) evaluated effects on reproduction of mallard duck exposed to selenomethionine in the diet and reported a NOAEL of 4 ppm and a LOAEL of 8 ppm. A body weight of 1 kg and food consumption of 100 g/day from the study supported development of a

NOAEL of 0.4 mg/kg-day and a LOAEL of 0.8 mg/kg-day for reduced duckling survival (Sample et al. 1996). The NOAEL of 0.4 mg/kg-day and the LOAEL of 0.8 mg/kg-day were selected as TRVs for mallard duck and other omnivorous birds.

A study by Smith et al. (1988) evaluated effects on reproduction of black-crowned night heron to selenomethionine in the diet. They reported a NOAEL of 10 mg/kg diet ww; data were insufficient to report a LOAEL. The NOAEL of 10 mg/kg diet ww was converted by Integral to a value of 9.1 mg/kg diet dw, using moisture content values reported by Smith et al. (1988). An assumed body weight of 0.883 kg and food consumption of 160.6 g/day were used to develop a NOAEL intake of 1.66 mg/kg-day (Sample et al. 1996). The NOAEL of 1.66 mg/kg-day was selected as a TRV for herons and kingfishers.

Mammals

In mammals, there is a relatively narrow range separating selenium deficiency from selenium poisoning. In rats, the nutritional requirement for selenium is about 0.08 mg/kg-diet. However, diets containing 0.8 mg/kg over lifetime resulted in intestinal lesions (Eisler 1985). A study by Rosenfeld and Beath (Rosenfield and Beath 1954) evaluated reproductive effects on rats administered potassium selenate in drinking water. The 1-year reproductive study reported a NOAEL of 1.5 mg/L and a LOAEL of 2.5 mg/L (Rosenfield and Beath 1954). An assumed body weight of 0.35 kg and water consumption of 0.046 L/day was used to develop a NOAEL of 0.2 mg/kg-day and a LOAEL of 0.33 mg/kg-day (Sample et al. 1996). These values were selected as TRVs for selenium in mammals.

Aquatic Biota

Selenium occurs in a variety of forms in water: as selenates, selenic acid, selenites, selenous acid, elemental selenium, hydrogen selenide, and organic selenides, with only elemental selenium, selenites, and selenates occurring in ambient waters within a normal physiological pH range and reduction potential permitted by water. The toxicity of selenium has been tested in a wide range of aquatic organisms, with *Hyalella azteca* being the most sensitive in acute toxicity studies, and the range of acute values spanning a factor of 440. The LC50 for *H. azteca* is 0.57 mg/L. (USEPA 2004a) considers the whole body tissue concentration to be the best basis for determination of chronic toxicity. The lowest tissue-based mean chronic value provided in EPA's recent review of the aquatic toxicity of selenium is 9.5 mg/kg dw tissue, and this was derived for *Lepomis macrochirus*, the bluegill. Assuming 75 percent moisture in fish tissue, this equates to 7.13 mg/kg ww, and is assumed to represent a LOAEC for fish.

2.10 SILVER

Silver has no known biological function, but it is a normal trace constituent of many organisms (Irwin et al. 1997; NAS 1980). In terrestrial environments, silver occurs primarily as sulfides in association with other minerals. In surface water, silver can occur as a monovalent ion (sulphide, bicarbonate, or sulphate salts), as a more complex ion (chlorides or sulphates), or adsorbed to particulate matter (ATSDR 1990). Silver, as ionic Ag+, is one of the most toxic metals to aquatic organisms in laboratory tests (Irwin et al. 1997). Therefore, most toxicity information available for silver focuses on its aquatic toxicity. Less is known on the toxicity of silver to wildlife.

The most likely route of exposure to silver by wildlife is ingestion of food and water (Irwin et al. 1997). Signs of chronic silver intoxication in tested birds and mammals included cardiac enlargement, vascular hypertension, hepatic necrosis, anemia, lowered immunological activity, altered membrane permeability, kidney pathology, enzyme inhibition, growth retardation, and a shortened life span (Irwin et al. 1997). Silver was not mutagenic, carcinogenic, or teratogenic to tested animals by normal routs of exposure (Eisler 1996).

Vegetation and Soil Invertebrates

Silver is not a COPEC in terrestrial systems at the site; therefore, TRVs were not identified for plant and soil invertebrate receptors.

Birds

Exposure of birds to silver may result in selenium and/or copper deficiency. Toxic effects of silver are increased in animals deficient in Vitamin E and selenium (Jensen et al. 1974). Most toxicity studies in birds have focused on interactions of silver with selenium, vitamin E, and copper. Adverse effects of silver on poultry occur at 10 mg/kg in copper-deficient diets (reduced hemoglobin), and 200 mg/kg in copper-adequate diets (growth suppression), or when the birds are given drinking water containing 100 mg Ag/L (liver necrosis) (WHO 2002). In chicks (*Gallus serregineus*) with normal diets, Sharma et al. (2004) found an unbounded NOAEL of 15 mg/kg-day for growth and gross toxicity when silver foil was administered for 10 days. This study does not meet the DQOs for this ERA.

Jensen et al. (1974) administered silver acetate in the diet to turkey poults for 4 weeks and reported a NOAEL of 300 mg/kg and a LOAEL of 900 mg/kg diet for growth rate effects. Using body weight of 4.5 kg from the study and an assumed food consumption of 0.174 kg/day (Sample et al. 1996), a NOAEL of 11.6 mg/kg-day and LOAEL of 34.8 mg/kg-day were derived. In accordance with USEPA (1997) guidance, an uncertainty factor of 10 was applied to derive a NOAEL of 1.16 mg/kg-day and a LOAEL of 3.5 mg/kg-day for chronic exposure. These chronic values were selected as TRVs for birds.

Mammals

Toxic effects resulting from silver ingestion by mammals are not well characterized. Effects of silver on sensitive species of mammals include death at 13.9-20.0 mg/kg bw by intraperitoneal injection; histopathology of kidney and brain at 250-450 mg/L drinking water; and liver necrosis when animals consumed diets with more than 130 mg/kg (WHO 2002). Rungby and Danscher (1984) administered silver nitrate in drinking water to mice for 125 days, and identified an unbounded NOAEL of 0.9 mg/kg-day for effects on growth and gross toxicity. Assuming an average body weight of 0.03 kg (Sample et al. 1996) provides a NOAEL of 30 mg/kg-day for chronic exposure. The NOAEL TRV of 30 mg/kg-day was selected for mammals. A LOAEL TRV was not identified.

Aquatic Biota

Silver does not appear to be a highly mobile element under typical conditions in most aquatic habitats (USEPA 2000a). Free silver ion is lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at water concentrations of 1.2–4.9 µg/L (Eisler 1996). Adverse effects on development of trout occur at concentrations as low as 0.17 µg/L and on phytoplankton species composition and succession at 0.3–0.6 µg/L. Silver uptake by aquatic organisms appears to be almost entirely from the dissolved form, and little evidence exists to indicate biomagnification of silver within marine or freshwater food webs. Critical tissue residues of silver for fish and invertebrates were not identified in the literature.

No CTR for aquatic invertebrates exposed to silver was identified for this ERA. For fish, a CTR of 0.06 mg/kg ww was used.

2.11 THALLIUM

Thallium has applications in rodenticides and insecticides (banned in the U.S. since 1975), treatment of skin infections, manufacture of glass and semiconductors, and infrared detectors. It is considered highly toxic and has been used historically in human poisonings.

Vegetation and Soil Invertebrates

Thallium is not a COPEC in terrestrial systems at the Site; therefore terrestrial TRVs were not identified for thallium.

Birds

USEPA (1999) compiled TRVs for its guidance for conducting screening level risk assessment at combustion facilities, and identified an LC50 of 35 mg/kg-day for starlings exposed to thallium. They applied an uncertainty factor of 0.01 to derive a TRV for the basis of their screening risk assessment; this value is considered a NOAEL for this risk assessment. No thallium LOAEL for birds is available.

Mammals

Formigli et al. (1986) exposed male rats to thallium in drinking water for 60 days and measured a range of reproductive endpoints, including histopathological, biochemical, and functional endpoints, including the reduction of sperm motility. Using data provided by the study, the mean LOAEL for this endpoint was calculated as 0.71 mg/kg-day. No NOAEL was provided by this study. A conservative uncertainty factor of 0.1 was used to derive a NOAEL of 0.071 mg/kg-day for this COPEC.

Aquatic Biota

Relatively little information on the toxicity of thallium to aquatic life is available. According to USEPA (1986), acute and chronic thallium toxicity to aquatic life occurs at 1.4 and 0.04 mg/L, respectively. According to a review by Peter and Viraraghavan (2005), toxicity to algae depends strongly on the valence state, with Tl(3+) being more toxic to *Chlorella* than Tl(1+) by a factor of 50,000. These authors reviewed several studies and report that thallium kills insects at 2 mg/L, tadpoles at 0.4 mg/L, and fish at 1 mg/L. This lethal concentration was multiplied by 0.1 to provide a general TRV for thallium in water for fish of 0.1 mg/L, considered an LOAEC for this risk assessment. CTRs for thallium in aquatic life were not available in Jarvinen and Ankley (1999) and were not identified in the literature.

2.12 VANADIUM

Vanadium can exist in many valence states (most often 5+) and is common in the earth's crust. It is used in ferrous metallurgy in the manufacture of special steels. Alloys of vanadium with non-ferrous metals are used in aircraft and space technology. Sources to the environment include combustion of fossil fuels and disposal of coal wastes and flyash.

Vegetation and Soil Invertebrates

USEPA (2005f) identified only one paper describing the toxicity of vanadium to plants that met their acceptability criteria. According to USEPA (2005f), this paper reports a NOAEL of 100 mg/kg dw in soils as a NOAEL for plants, based on a test of effects on growth with broccoli. No studies of toxicity to soil invertebrates were considered acceptable for use as TRVs (USEPA 2005f).

Birds

Numerous studies report on the toxicity of vanadium to birds, many of which address survival, growth, and reproductive endpoints. However, the majority of data are for the chicken, with only two studies reporting toxicity to ducks, and one to Japanese quail. To derive a TRV for birds, USEPA takes the geometric mean of TRVs for all endpoints, and for birds, that value is 0.344 mg/kg-day. Because this value is generally consistent with the lower NOAELs for survival, growth, and reproductive endpoints, it was used as the NOAEL for birds. The lowest LOAEL

among this collection of studies that exceeded this NOAEL was 0.413 mg/kg-day. Both of these values are for the growth endpoint.

Mammals

There were 101 studies considered acceptable for derivation of a TRV for mammals by USEPA (2005f). These studies used a broader range of species than the available literature for birds, with rats, sheep, pigs, and mice represented in papers with survival, growth, or reproduction endpoints. USEPA's (2005f) TRV for mammals is 4.16 mg/kg-day, the geometric mean of all acceptable TRVs for all endpoints, and this was applied as the NOAEL for mammals in this risk assessment (this value is also reported as a reproductive and survival NOAEL for mice). The lowest LOAEL among this collection of studies that exceeded this NOAEL was 5.11 mg/kg-day, and was a LOAEL for growth in rats exposed via drinking water for 10 weeks.

Aquatic Biota

Vanadium is not a COPEC in aquatic systems at the Site; therefore, aquatic life TRVs were not identified for vanadium.

2.13 ZINC

Zinc is required for normal growth, development, and function in all animal species that have been studied (NAS 1980). Zinc attaches to organic molecules such as amino acids, proteins, and nucleic acids, directly binding to sulfhydryl, amino, imidazole, and phosphate groups (NAS 1980). Zinc has low toxicity to birds and mammals. Exposures to high concentrations of zinc may result in reduced growth, anemia, reduced bone ash, decreased tissue concentrations of iron, copper, and manganese, and decreased use of calcium and phosphorus (NAS 1980).

Vegetation and Soil Invertebrates

Zinc is an essential element for plant growth (Efroymson et al. 1997b). It has a part in many enzymes and is involved in disease protection and metabolism of carbohydrates and proteins. Zinc is actively taken up by roots in ionic form and, to a lesser extent, in organically chelated form. It is fairly uniformly distributed between roots and shoots being transported in the xylem in ionic form. Transport in the phloem appears to be as an anionic complex. Symptoms of toxicity include chlorosis and depressed plant growth.

The number of seeds produced by soybean plants grown in ordinary garden soils containing 25 mg/kg zinc was reduced by 25 percent, with no effect on seed production in plants grown in 10 mg/kg zinc (Aery and Sakar 1991). The leaf weights and root weights were reduced 33 percent in soybeans grown in sandy loam soils with pH of 6.5 and containing 393 mg/kg zinc (White et al. 1979). At pH 5.5, no effect was observed in soil containing 115 mg/kg zinc, while leaf weights in this species were reduced at 131 mg/kg dw.

Beyer and Cromartie (1987) and Kabata-Pendias and Pendias (Kabata-Pendias and Pendias 1984) report that zinc has a high potential to accumulate in earthworms (e.g., reported BAFs range

from 0.1 to 26 depending upon soil conditions). Soil invertebrates show no adverse effects at zinc concentration of 100 mg/kg (Efroymson et al. 1997a), which was the TRV applied in this ERA. Representative soil invertebrates showed reduced growth at 300–1,000 mg/kg diet and reduced survival at 470–6,400 mg/kg soil (Eisler 1993), suggesting the selected TRV is conservative.

Birds

Growth of domestic poultry and wild birds was reduced at concentrations in the diet >2,000 mg/kg, and survival was reduced at concentrations >3,000 mg/kg in diet, or at a single oral dose >742 mg/kg bw. Younger stages (i.e., chicks, ducklings) were least resistant (Eisler 1993). A study of dietary exposure of white Leghorn hens to zinc sulfate for 44 weeks found a NOAEL of 228 mg/kg diet and a LOAEL of 2028 mg/kg diet for decreased egg hatchability. Sample et al. (1996) used data from this study to develop a NOAEL intake of 14.5 mg/kg-day and a LOAEL intake of 131 mg/kg-day. These values were selected as TRVs for birds.

Mammals

Sensitive species of livestock and small laboratory animals are adversely affected at 90–300 mg/kg diet, >90 mg/kg-day repeated oral does, >300 mg/L drinking water, and >350 mg/kg bw single oral dose. A study of dietary exposure of rats to zinc oxide during gestation reported a NOAEL of 2000 mg/kg diet and a LOAEL of 4,000 mg/kg diet for increased rates of fetal resorption and reduced fetal growth rates (Schlicker and Cox 1968). Sample et al. (1996) used data from this study to develop a NOAEL intake of 160 mg/kg-day and a LOAEL intake of 320 mg/kg-day; these were selected as TRVs for mammals.

Aquatic Biota

Zinc in the water column can partition to dissolved and particulate organic carbon. Bioavailability of zinc in sediments is controlled by the AVS concentration. Water hardness (i.e., calcium concentration), pH, and metal speciation are important factors in controlling the water column concentrations of zinc since the divalent zinc ion is believed to be responsible for observed biological effects (USEPA 2000a). Significant adverse effects of zinc on growth, survival, and reproduction occur in sensitive species of aquatic plants, protozoans, sponges, molluscs, crustaceans, echinoderms, fish, and amphibians at nominal water concentrations between 10 and 25 μg/L (Eisler 1993).

Acute LC50 (96 h) values for freshwater invertebrates were between 32 and 40,930 μg/L; in fish, this range was 66 to 40,900 μg/L. Daphnids and trout have been identified as some of the most sensitive species with adverse effects occurring at concentrations between 5 and 19 μg/L (USEPA 2000a). In general, zinc is more toxic to embryos and juveniles than to adults. Zinc is not a highly mobile element in aquatic food webs and there appears to be little evidence to support the general occurrence of biomagnification of zinc within marine or freshwater food webs (USEPA 2000a).

A CTR for fish exposed to zinc was identified by Jarvinen and Ankley (1999), from a study performed with female flagfish (*Jordinella floridae*) for 100 days. Flagfish with 34 mg/kg ww in whole bodies experienced no effects on growth during development from larvae to adulthood. Those with 40 mg/kg ww showed reduced growth after 100 days. This CTR was applied in this ERA.

The lowest CTR identified for aquatic invertebrates by Jarvinen and Ankley (1999) was 12.7 mg/kg ww, a NOAEC for survival of crayfish.

3 ORGANIC COMPOUNDS

3.1 BENZENE HEXACHLORIDE

Benzene hexachloride (BHC) also known as 1,2,3,4,5,6-hexachlorocyclohexane, is an organochlorine pesticide with eight isomers (ATSDR 2003), of which the alpha (α), beta (β), gamma (γ), and delta (δ) isomers are of commercial significance. The isomer γ-BHC is a commercial pesticide also known as lindane (ATSDR 2003). γ-BHC generally has low mobility in soils due to adsorption of γ-BHC to soil particulates (ATSDR 2003). γ-BHC in soil or sediment is degraded primarily by biodegradation, although hydrolysis may occur in moist soils under alkaline conditions (ATSDR 2003). Various studies of BHC in soil indicate that β-BHC is the most persistent isomer, followed by γ-BHC, α-BHC and δ-BHC (ATSDR 2003).

γ-BHC is bioconcentrated to high levels following uptake from surface water by aquatic organisms. However, uptake from soils and bioconcentration by plants and terrestrial organisms appear to be limited. BHCs do not appear to undergo biomagnification in terrestrial food chains to a great extent, although there is a moderate potential for transfer of γ-BHC to animal tissue as a result of soil ingestion or ingestion of contaminated foliage (ATSDR 2003). Clark et al. (1974) found that γ-BHC levels in the adipose tissue of cattle were 10 times higher than in the feed (0.002 mg/kg). Szokolay et al. (1977) examining relative accumulation of BHC isomers (including γ-BHC and various components) in the food chain in Czechoslovakia found that γ-BHC residues were lower in tissues of animals (chickens, sheep, pigeons) feeding on plant material than in carnivores.

At sufficiently high dosages, BHC is neurotoxic, cytotoxic, and hepatotoxic in mammals (ATSDR 2003). The mechanism of toxicity of BHC on the nervous system is similar to that of other neurotoxic organochlorine pesticides. The toxicity of BHC in mammals varies. γ-BHC is the most acutely toxic, followed by α, δ, and β-BHC. Following chronic exposure, however, β-BHC is the most toxic followed by α, γ, and δ-BHC. The greater chronic toxicity of β-BHC is probably due to its longer biological half-life in the body and its accumulation in the body over time (ATSDR 2003).

Vegetation and Soil Invertebrates

Lindane (γ-BHC) at concentrations less than 100 mg/kg in soil may inhibit seed germination of various plants (Bidlan et al. 2004). An uncertainty factor of 10 was applied to derive a NOAEC in soils for BHC of 10 mg/kg dw. This value was applied for this ERA.

BHC has also been found to be toxic to various soil invertebrate species. γ-BHC was found to have a LC50 of 3.57 mg/kg soil for isopod *Porcellionides pruinosus* (Santos et al. 2003). An uncertainty factor of 10 was applied to derive a LOAEC in soils of 0.36 mg/kg dw, which was applied in this ERA. This value is considered to be the lowest concentration in soils at which mortality in soil invertebrates may be observed.

Birds

BHC has been identified in tissues of wild birds. The acute toxicity of BHC in birds is low, the oral lethal dose of a 15 percent suspension of BHC in female Japanese quail was found to be greater than 1 g/kg (Vos et al. 1971). A 90-day study of adult Japanese quail (*Coturnix coturnix japonica*) fed mixed isomers of BHC found a NOAEL of 5 mg/kg diet and a LOAEL of 20 mg/kg diet for reproductive effects (egg volume) (Vos et al. 1971). Using an average body weight for quails of 0.15 kg and a food consumption rate of 0.0169 kg/day from other sources, a NOAEL intake of 0.563 mg/kg-day and a LOAEL intake of 2.25 mg/kg-day were derived (Sample et al. 1996). No other studies evaluating BHC toxicity for birds were identified that met the DQOs. Therefore, a NOAEL TRV of 0.563 mg/kg-day and a LOAEL TRV of 2.25 mg/kg-day were selected for birds.

Mammals

There is limited information on the chronic effects of BHC on reproduction, growth, and survival in mammals. Grant et al. (1977) evaluated the reproductive effects following dietary exposure of rats to several concentrations of BHC. A dietary concentration of 40 mg/kg was identified as a NOAEL and a dietary concentration of 840 mg/kg was identified as a LOAEL for reproductive (low birth weight) effects (Grant et al. 1977). Using an average body weight of 0.35 kg and a food consumption rate of 0.028 kg/day (Sample et al. 1996). Integral developed a NOAEL of 3.2 and a LOAEL of 6.4 mg/kg-day based on this study. It was unclear whether the methods for this study met the DQO for randomization of treatment, but all other DQOs were met.

Two studies evaluating BHC toxicity to mink were evaluated. Rush et al. (1983) found an effect of increased kit mortality in mink exposed to dietary concentrations of 1 mg/kg. Bleavins et al. (1984) administered BHC in the diet to mink for 331 days through mating. They found a LOAEL for reproductive effects (smaller litter size and increased kit mortality) to occur at a dietary concentration of 25 mg/kg and a LOAEL for decreased birth weight effects of a dietary concentration of 1 mg/kg. Using an average body weight of 1.0 kg and a food consumption rate of 0.137 kg/day, the dietary concentration of 1 mg/kg was converted to an intake of 0.14 mg/kg-day by Sample et al. (1996). Because it is an unbounded LOAEL, an uncertainty factor of 10 was applied to derive a potential NOAEL of 0.014 mg/kg-day.

A NOAEL of 0.014 mg/kg-day was selected for mink. Mink are known to be particularly sensitive to organochlorine contaminants. Therefore, a NOAEL of 3.2 mg/kg-day, and a LOAEL of 6.4 mg/kg-day, based upon reproductive effects for rats, were selected as TRVs for all other mammals.

Aquatic Biota

BHC is not a COPEC in aquatic systems at the site, so ecotoxicity for aquatic receptors is not addressed in this ERA.

3.2 DICHLORODIPHENYL-TRICHLOROETHANE

The term dichlorodiphenyl-trichloroethane (DDT) is the name that is commonly applied to 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane. DDT has several isomeric forms, including o,p'-DDT (2,4-DDT), but the collective ecological toxicity data on DDT has focused on p,p'-DDT (4,4-DDT) as being the most toxicologically significant. For the purposes of this risk assessment, the term DDT refers to the p,p'-isomer, unless otherwise noted. In the environment, DDT is metabolized to DDE (1,1-dichloro-2,2-bis[*p*-chlorophenyl]ethylene) and DDD (1,1-dichloro-2,2,bis[*p*-chlorophenyl]ethane).

DDT and its primary metabolites, DDE and DDD are very persistent in the environment (ATSDR 2002) and bioaccumulate. Dominant fate processes in aquatic environments are volatilization and adsorption to biota, suspended particulate matter, and sediments. Transformation includes biotransformation and photolysis in surface waters (ATSDR 2002). DDT in surface water degrades to DDD. In sediment, degradation of DDT is altered by invertebrates, with the conversion of DDT to DDMU (1-chloro-2,2-bis[*p*-chlorophenyl]ethane) (ATSDR 2002). DDT has been found to accumulate to greater concentrations in fattier fish and higher trophic levels than in leaner fish and lower trophic levels. Accumulation of DDT has also been found to be significantly greater in the pelagic food web than the benthic food web (ATSDR 2002).

Chronic effects of DDT and its metabolites on ecological receptors include changes in enzyme production, hormonal balance, and calcium metabolism, which may cause changes in behavior and reproduction. The most well-documented response is eggshell thinning in birds which results in embryo mortality, and decreased hatchling survival. Because of the tendency of DDT to magnify in food chains, higher trophic level birds appear to be at greater risk for egg loss due to shell thinning. Eggshell thinning of greater than 20 percent has been associated with decreased nesting success due to eggshell breakage (Anderson and Hickey 1972; Dilworth et al. 1972).

The effects of DDT on other receptor groups are not as well-studied. Invertebrate species are generally more susceptible than fish species to effects associated with exposure to DDT in the water column (USEPA 2000a). Sediments contaminated with pesticides, including DDT, have been implicated as the cause of benthic community alteration at sediment concentrations exceeding 2 µg/kg (USEPA 2000a).

Vegetation and Soil Invertebrates

Soil TRVs found by USEPA (2007) to specifically address risks to plants range from 7.1–50.0 mg/kg (dw) (the lowest of these values was used as the TRV for plants) also provides several LC50 values for DDT in soils ranging from 0.08 to 77.2 mg/L. A value for this ERA was derived using 34 studies of aquatic invertebrates and is at the low end of this range (1.2 mg/kg [dw]). Derivation of this value is described in Section 5.4.3.1.

Birds

DDT at sufficiently high doses can induce death in birds by disrupting central nervous system function. Single median lethal doses (LD50s) of DDT in birds range from 595 mg/kg bw for California quail (*Callipepla californica*) to greater than 2,240 mg/kg bw for mallard duck (*Anas platyrynchos*) (Caux and Roe 2000). At lower doses, DDT and its metabolites DDE and DDD (DDX) have been shown to affect reproductive success in birds. Chronic exposures to sufficiently high levels of DDX in the diet of birds have been linked to eggshell thinning, egg breakage, embryo mortality, and decreased hatchling survival. The mode of action of DDX is to affect the activity of Ca^{2+} ATP-ase systems in the shell gland, thereby interfering with the deposition of calcium in the shell (Lundholm 1987).

Evidence strongly indicates that DDE is the metabolite most responsible for reproductive toxicity in birds (USEPA 2000a). Dietary concentrations of approximately 10 mg/kg dw DDE in the diet of mallard (Heath et al. 1969), black duck (Longcore and Samson 1973), American kestrel (Lincer 1975; Wiemeyer and Porter 1970), screech owl (McLane and Hall 1972), and barn owl (Mendenhall et al. 1983) resulted in statistically significant shell thinning, which has been correlated with decreased reproductive success in wild or laboratory bird populations. In all these studies, birds were exposed for a minimum of one breeding season and for as much as 2 years. DDT and DDD can induce shell thinning, but at higher dietary concentrations than DDE. Heath et al. (Heath et al. 1969) studied shell thinning and reproductive success in mallards exposed for two breeding seasons to DDE, DDT, or DDD in the diet. They observed a statistically significant decrease in shell thickness in mallards exposed to 25 mg/kg dw DDT, but not significant shell thinning at 10 mg/kg dw. No effects on shell thinning were reported in mallards exposed to 10 or 40 mg/kg dw DDD in the diet.

There is a large amount of variability in sensitivity to DDT and its metabolites among bird species, with waterfowl and raptor species showing the greatest sensitivities. The brown pelican is most susceptible to adverse effects, with eggshell thinning and depressed productivity occurring at 3.0 μg/g of DDE in the egg and total reproductive failure when residues exceed 3.7 μg/g (USEPA 2000a).

Measurements of residues in eggs of birds are a reliable indicator of adverse effects. At least two studies provide both NOAEL and LOAEL values associated with dietary exposures:

- Lincer (1975) evaluated eggshell thinning in American kestrels exposed to DDE during the breeding season for 2 years, and derived a NOAEL of 0.023 mg/kg-day, and a LOAEL value of 0.23 mg/kg-day.
- Heath et al. (1969) evaluated eggshell thinning and percent of cracked eggs in mallards exposed to 4,4'-DDT over 2 years and derived a NOAEL of 0.30 mg/kg-day, and a LOAEL of 0.75 mg/kg-day. The same study provides a NOAEL of 1 mg/kg-day for mallards exposed to 4, 4'-DDD.

Other literature has reported NOAEL values for similar reproductive endpoints. Longcore and Stendell (1983) reported a NOAEL for eggshell thickness of 0.090 mg 4,4'-DDE/kg-day. Other LOAEL values for reproductive endpoints in birds in the literature are consistent with those provided by Heath et al. (1969) and Lincer (1975), ranging from 0.30 mg/kg-day to 3.3 mg/kg-day (e.g., Jefferies 1971; Keith and Mitchell 1993; Longcore and Samson 1973; Mendenhall et al. 1983) for 4,4'-DDE, and 2.4 mg/kg-day for 4,4-DDT (Jefferies 1971).

For DDE, a NOAEL of 0.023 mg/kg-day and a LOAEL of 0.23 were selected as TRVs for bird receptors. For DDT a NOAEL of 0.3 mg/kg-day and a LOAEL of 0.75 mg/kg-day were selected as TRVs for bird receptors. For DDD, a NOAEL of 1.0 mg/kg-day was selected as the TRV for bird receptors. A LOAEL was not identified for DDD. Because there was only one earthworm sample from the Site for which DDT and metabolite concentrations were reported, the EcoSSL for total DDx was applied to evaluate risks to terrestrial birds. The most conservative avian EcoSSL value for total DDx is 0.093 mg/kg (dw).

Mammals

Several toxicity studies have been conducted for DDX in various mammals. The bulk of the studies have been conducted on rodents, although toxic effects on dogs and monkeys and some wildlife species have also been evaluated (ATSDR 2002). Many of the rodent studies have focused on endpoints of interest to human health, not growth, reproduction, and survival which are of interest to the ERA. Five dietary studies on rodents that reported toxicity for developmental effects were reviewed for this ERA (Clement and Okey 1974; Fitzhugh 1948; Ottoboni 1969; Tomatis et al. 1972; Turusov et al. 1973). Clement and Okey (1974) identified a NOAEL of 20 mg/kg diet and a LOAEL of 200 mg/kg diet for growth effects in rats. These results are supported by the findings of Ottoboni (1969) who identified 200 mg/kg diet as a NOAEL for reproductive effects in rats. These results are further supported by Tomatis et al. (1972) and Turusov et al.(1973), which identified a LOAEL of 250 mg/kg diet for pre-weaning mortality in mice. Fitzhugh (1948) reported a NOAEL of 10 mg/kg diet DDT administered to rats over 2 years and a LOAEL of 50 mg/kg was identified for a decline of the percent of young weaned. Of the studies reviewed, only the study by Clement and Okey (1974) met the DQOs for this ERA. Clement and Okey administered p,p'-DDT in diet to male and female rats for a six month period through weaning of pups at doses of 0, 20, 200 or 500 mg/kg. They reported a NOAEL of 20 mg/kg diet and a LOAEL of 200 mg/kg diet for growth effects on rat pups. Using an average body weight of 0.35 kg and a consumption rate of 0.028 kg/day, a NOAEL intake of 1.6 mg/kg-day was calculated (Sample et al. 1996).

Several additional studies have been conducted on organochlorine toxicants using mink (Aulerich and Ringer 1970; Duby et al. 1971; Frank and Holdrinet 1975; Giesy et al. 1994; Jensen et al. 1977; Proulx et al. 1987). None of these studies meet the DQOs for establishing TRVs for this ERA.

Insufficient data were identified for this ERA to evaluate DDE and DDD separately in mammals. Therefore, a NOAEL of 1.6 mg/kg-day and a LOAEL of 16 mg/kg-day were selected as TRVs for

DDX. This value was applied to assessment of risk to semiaquatic birds and mammals. Because only one earthworm sample was analyzed for DDT and metabolites, the EcoSSL value for mammals (USEPA 2007) was applied to assess risks associated with exposure of terrestrial mammals to these compounds in soil. The most conservative mammalian EcoSSL for total DDx is 0.021 mg/kg.

Aquatic Biota

For fish, the primary route of uptake is ingestion of prey items, but both DDT and its metabolites can be accumulated through the skin or gills upon exposure to DDX in water. DDX can affect survival and reproduction in fish. Short-term exposure to DDT concentrations of less than 1 µg/L in water and 1.1 to 2.4 mg/kg in fish embryos have been reported to elicit toxic responses in fish (USEPA 2000a).

The collective data suggest a number of general relationships characterizing DDX toxicity in fish. First, toxicity is related to uptake and uptake is dose- and time-dependent. Jarvinen et al. (1976; 1977) reported that 2 ppb DDT in water decreased the probability of survival in juvenile and adult fathead minnows (*Pimephales promelas*), but that exposure to 0.5 ppb in water did not. Mortality occurred rapidly in the fish exposed to the higher dose, with approximately 40 percent mortality observed within the first 28 days of the 266-day exposure. Accumulation and mortality increased gradually throughout the 266-day exposure period in fish exposed to the lower dose. Warlen et al. (1977) also observed dose- and time-dependent accumulation of DDT in Atlantic menhaden.

Second, DDT administered via the diet appears to be less toxic than DDT dissolved in water, though ingestion contributes to overall accumulation and toxicity, probably because of diminished bioavailability from the diet. In fathead minnows, dietary DDT at 46 mg/kg decreased the probability of survival by approximately 20 percent after 266 days of exposure, whereas, 2 ppb DDT in water decreased the probability of survival by approximately 50 percent in minnows (Jarvinen et al. 1976; 1977). Warlen et al. (1977) reported no effects on growth, reproduction or survival in menhaden exposed to 0.093 mg/kg DDT in the diet. Macek et al. (1970) reported no effects in rainbow trout (*Salmo gairdneri*) fed 1 mg/kg DDT for 140 days.

Third, toxicity is life-stage dependent. Juveniles and young appear more sensitive than adults, and spawning adults appear more sensitive than non-spawning adults (Jarvinen et al. 1976; 1977).

DDT in fish is metabolized to a variety of compounds including DDE and DDD, with the majority of residues in some species typically present as DDE, depending on the route of administration (Jarvinen et al. 1976; Rhead and Perkins 1984). Rhead and Perkins (1984) reported that dietary DDT was converted to DDD to a greater extent (16 percent) than waterborne DDT (3 percent) in goldfish (*Carasius auratus*). Jarvinen et al. (1977) found similar results in fathead minnow.

USACE (2001) and Jarvinen and Ankley (1999) have compiled residue databases that relate DDX body burdens to specific effects in fish. However, the great majority of these data are for tissues other than whole body or for life stages (e.g., embryo, egg). The limits to applicability of critical body residues to risk assessment were discussed in the RAWP (USEPA 2004c) and by Integral (Integral 2005).

Because DDX CTRs for fish are not available for nonlethal endpoints, a concentration in food was used in this ERA. Buhler et al. (1969) reported on several toxicity tests using chinook and coho salmon as test animals. Chinook was the more sensitive species, and showed no effect on survival when ingesting food with 6.25 mg/kg ww. A weakness of Buhler's study was that no statistical analyses were performed to determine significance of responses. Nevertheless, this TRV is conservative because it is based on a chronic exposure to a highly sensitive species.

There was no effect on the survival of mayflies (*Ephemera danica*) exposed to DDT in water for 9 days (Sodergren and Svensson 1973). Concentration in the whole body of the test species at the end of the test was 3.1 mg/kg ww. This CTR was applied in this ERA.

3.3 DIOXINS/FURANS

Polychlorinated dibenzo-*p*-dioxins (PCDD), as a group, represent 75 different positional isomers, and polychlorinated dibenzofurans (PCDF) consist of more than 135 compounds (ATSDR 1998). These two chemical classes are generally referred to as dioxins and furans. Tetrachlorodibenzodioxins (TCDD) and tetrachlorodibenzofurans (TCDF) are a subset of these groups.

Dioxins and furans are introduced into the environment primarily as a result of anthropogenic combustion processes, although natural combustion sources (e.g., forest fires) also contribute. Dioxins are lipophilic compounds, and persist in environmental media. Volatilization and particulate deposition are the primary transport mechanisms responsible for transfer of these lipophilic compounds from environmental media into the food chain.

Dioxin and furan toxicity is mediated intracellularly by binding with the aryl hydrocarbon receptor (Ah-R). The resulting Ah-R complex moves into the cell nucleus, where it binds to DNA, and alter the expression of a number of gene sequences. Many of the observed toxic effects of dioxins (and the coplanar polychlorinated biphenyls [PCBs]) are attributable to specific alterations in gene expression (USEPA 2000b). Unlike most toxic chemicals, the lethality of TCDD is delayed and species specific (USEPA 2000b). The characteristic signs and symptoms of severe toxicity by TCDD are drastic weight loss and thymic atrophy (USEPA 2000b). Other toxic effects include hyperplasia or atrophy of the spleen, testes, or ovaries, bone marrow depletion, and systemic hemorrhage (USEPA 2000b). Dioxins are believed to cause alterations to developmental endocrine (thyroid and steroid hormones) and immune functions, as well as interference in vitamin production, which results in disruption of patterns of embryonic development at critical stages (USEPA 2000b).

Information is relatively scarce on the biological effects of PCDD isomers, except 2,3,7,8-TCDD (Eisler 1986a). The latter has been associated with lethal, carcinogenic, teratogenic, reproductive, mutagenic, histopathologic, and immunotoxic effects. There are substantial inter- and intraspecies differences in sensitivity and toxic responses to 2,3,7,8-TCDD; toxicological mechanisms are imperfectly understood. In general, dioxins are not acutely toxic to adult organisms, but their long-term accumulation is thought to be expressed chronically, and may ultimately result in death.

Because PCDDs, PCDFs, and certain PCBs cause toxicity in wildlife via a common mechanism, toxic equivalency factors (TEFs) have been derived for these chemicals (Van den Berg et al. 1998). The TEF indicates an order of magnitude estimate of the toxicity of a compound relative to 2,3,7,8-TCDD. Chemical-specific TEF values can be used to calculate toxic equivalent (TEQ) concentrations or doses. Therefore, for the purposes of this ERA, TRVs were identified for 2,3,7,8-TCDD.

Vegetation and Soil Invertebrates

Dioxins are highly lipophilic compounds that tend to bind to organic matter in the soil. They may accumulate on the root surfaces of plants. However, since dioxins are high molecular weight compounds, they have a negligible potential to translocate from roots into plants via the xylem (Bacci et al. 1992; McCrady and Maggard 1993; McCrady et al. 1990). Vapor-phase dioxins can sorb to the leaf cuticle (ATSDR 1998). Wet and dry deposition of airborne dioxin-containing particles onto plant surfaces can also occur. Dioxins sorbed onto plant surface have a negligible potential to enter the tissues (McCrady et al. 1990). Since dioxins are not taken up into plants, exposure is incomplete and toxicity has not been reported. No soil benchmark is available or needed.

Reinecke and Nash (Reinecke and Nash 1984) show that dioxin has a moderate to high potential to bioaccumulate in earthworms (BAFs range from 0.17 to 9.4 depending on soil conditions). Aquatic invertebrates have been found to be insensitive to dioxin toxicity. The insensitivity of invertebrates to dioxin-like toxicity is consistent with the recent finding that they lack the protein necessary to mediate the toxic effects of dioxin (USEPA 2003). It is expected that terrestrial invertebrates will likewise be insensitive to dioxin toxicity; there was no effect on survival of two species of earthworms at 5 ppm in soils, but both species died at 10 ppm (Eisler 1986a).

Birds

LD50 values computed 37 days after a single oral dose of 2,3,7,8-TCDD varied from 15 μg/kg bw in Northern bobwhite (*Colinus virginianus*), with 95 percent confidence limits of 9.2 and 24.5 μg/kg, to more than 810 μg/kg bw for the ringed turtle-dove (*Streptopelia risoria*) (Eisler 1986a). Mallards (*Anas platyrhynchos*) were intermediate in sensitivity with an acute oral LD50 value of more than 108 μg/kg bw. For all 3 species, death occurred 13–37 days after treatment; remission in survivors had apparently occurred by day 30 post-treatment. Domestic chickens were relatively sensitive to PCDDs, especially 2,3,7,8-TCDD, with an estimated 2,3,7,8-TCDD oral LD50 range of 25 to 50 μg/kg bw (Eisler 1986a). Chickens fed 1 or 10 μg of 2,3,7,8-TCDD,

1,2,3,7,8,9-hexa CDD, or hepta-CDDs per kg bw daily for 21 days showed signs of chick edema disease: pericardial, subcutaneous, and peritoneal edema, liver enlargement and necrosis with fatty degeneration; frequently resulting in death.

EPA currently recommends evaluation of risk to birds from exposures to dioxin-like compounds on the basis of concentrations in bird eggs (Suter 2003). Concentrations of 2,3,7,8-TCDD toxic equivalents (TEQs) in eggs are not available for the Site. Toxicity studies used for this ERA were those describing risk thresholds as ingested doses. Schwetz et al. (1973) fed chickens 2,3,7,8-TCDD for 21 days, providing a LOAEL for reduced survival of 1 μg/kg -day, with a NOAEL from the same study of 0.1 μg/kg-day. Hoffman et al. (1996) fed American kestrels (*Falco sparverius*) PCB 126 for 10 days, providing an oral LOAEL for bone growth of 25 μg TEQ/kg-day, and a NOAEL for bone growth of 5 μg TEQ/kg-day. The NOAEL of 0.0001 mg/kg-day and the LOAEL of 0.001 mg/kg-day from Schwetz et al. (1973) were selected as TRVs for birds.

Mammals

The sensitivity of mammals to 2,3,7,8-TCDD toxicity is highly variable. Acute toxicity studies with 2,3,7,8-TCDD have shown marked differences—up to a factor of 8,400—between the single oral LD50 dose for the guinea pig and the hamster (*Cricetus* sp.) (Eisler 1986a). The acute oral LD50 value of 0.6 μg/kg bw for guinea pigs, suggests that 2,3,7,8-TCDD may be the most toxic compound ever tested on small laboratory animals. The unusual resistance of hamsters may be associated with its enhanced rate of metabolism and excretion of 2,3,7,8-TCDD relative to other PCDD isomers examined. Poisoning in mammals by 2,3,7,8-TCDD is typically characterized by loss of body weight and delayed lethality. Atrophy of the thymus is a consistent finding in mammals poisoned by 2,3,7,8-TCDD, and suppression of thymus-dependent cellular immunity, particularly in young animals, may contribute to their death. Developing mammalian fetuses are especially sensitive to 2,3,7,8-TCDD, and maternal exposure results in increased frequencies of stillbirths. Several studies with mammals are available for interpreting exposures to dioxin-like compounds at the site:

- Khera and Ruddick (1973) measured litter size and pup weight in rats exposed to 2,3,7,8-TCDD for 10 days during the period of gestation, reporting a LOAEL of 0.25 μg TEQ/kg-day and a NOAEL of 0.125μg TEQ/kg-day.
- Kociba et al.(1978) exposed rats to 2,3,7,8-TCDD for 2 years, observing increased mortality in females dosed with 0.1 μg TEQ/kg-day, and no effect on female mortality at 0.01 μg TEQ/kg-day.
- Murray et al. (1979) exposed three generations of rats to 2,3,7,8-TCDD. There were no effects on the first generation at the LOAEL. Second and third generations showed reductions in fertility, litter size, and size and survival of pups at the LOAEL.

For mink, only studies reporting the dose which was lethal to 50 percent of specimens (LD50) values or feeding studies with fish contaminated with chemicals other than the target dioxin-like compounds were found, but the data suggest mink are also highly sensitive to toxicity of dioxin-like compounds.

The lowest NOAEL of 0.001 μg TEQ/kg-day of the acceptable rat studies and the associated LOAEL of 0.001 μg/TEQ/kg-day from Murray et al. (1979) were selected as TRVs for mammals.

Aquatic Biota

Dioxins and furans bioaccumulate in aquatic systems. In outdoor pond studies, a major portion of the added 2,3,7,8-TCDD concentrated in aquatic plants and at the sediment-water interface; however, most (85-99 percent) of the 2,3,7,8-TCDD originally added to the ecosystem remained in the sediments at the end of the study. Among bony fish, body burdens of 2,3,7,8-TCDD increased with increasing concentration in the water column and with increasing duration of exposure; on removal to uncontaminated water, less than 50 percent was lost in 109 days (Eisler 1986a).

Aquatic invertebrates are presumed to lack the Ah receptor, and, as such, are thought to be relatively insensitive to dioxins. *Daphnia magna* exposed to nominal concentrations of TCDD in water were not affected at concentrations as high as 1,030 ppb. Similarly, dioxins have been reported to bioaccumulate in benthic invertebrates that also lack the Ah receptor to significant concentrations without adverse effects. West et al. (1997) exposed *Chironomus tentans* and *Lumbriculus variegatus* to TCDD in food and no toxic effects were observed in full life-cycle tests with either species at tissue residue concentrations up to 9,533 μg/kg-lipid of TCDD. The lowest NOAEC reported in a literature review by Jarvinen and Ankley (1999) was for snails (*Physa* spp.) following 32 days exposure to 2,3,7,8-TCDD in water (0.0097 mg/kg ww). The studies cited by Jarvinen and Ankley (1999) also reported no-effects levels of 2,3,7,8-TCCD for *Daphnia magna* of 0.017 mg/kg ww and for *Chironomus tentans* of 0.138 mg/kg ww. The value for *Daphnia magna* was used in this ERA.

The chronic toxicity of dioxin-like compounds to the salmonid fishes has been well studied, with most investigations focusing on the concentration of 2,3,7,8-TCDD in eggs that results in yolk sac edema, developmental effects, or other failure of embryos to survive through the larval stage. Using acceptability criteria very similar to those presented by Integral (2005) and Steevens et al. (2005) compiled toxicity literature and identified thresholds for 2,3,7,8-TCDD in eggs from 10 studies. They generated a species sensitivity distribution (SSD) based on geometric means of no-observed-effect and lowest-observed-effect residue levels in fish eggs. The SSD is a statistical distribution that captures the variation in sensitivity among species represented by the database, and is expressed as a cumulative distribution function. SSDs can be developed on the basis of any type of benchmark available; Steevens et al. (2005) compiled SSDs using the geometric mean of the LOAEC and NOAEC for all 10 studies, and a second one using LC50 values (i.e., concentrations lethal to half the test organisms). Residue data were fitted to a logistic distribution.

Using the SSD based on the geometric mean of LOAEC and NOAEC values, Steevens et al. (2005) generated residue-based TRVs for dioxin-like compounds in fish tissue that are protective of 90, 95, 97.5, and 99 percent of species, with confidence limits. For this risk assessment, the mean tissue level protective of 95 percent of species will be applied: 0.321 ng 2,3,7,8-TCDD/g

lipid, consistent with EPA's methods for derivation of water quality criteria (USEPA 1985). The range of values provided by their SSD (including lower and upper confidence limits and levels protective of different fractions of fish species) will be also considered in the risk assessment.

Steevens et al. (2005) remark that because so many of the species represented in the SDD are salmonids, which are generally very sensitive to many toxicants, the resulting toxicity residue benchmarks derived from the SSD are conservative for many non-salmonid fish species. Only one of the receptor fish species is a salmonid, the whitefish (*Coregonus clupeaformis*), so the SSD derived by Steevens et al. (2005) is considered to be conservative for application at Cass Lake and for aquatic habitats at the site. The individual studies from which Steevens et al. (2005) extracted data will also be considered (Elonen et al. 1998; Guiney et al. 1996; Henry et al. 1997; Johnson et al. 1998; Walker et al. 1991a; Walker et al. 1991b; Walker et al. 1996; Walker and Peterson 1994; Zabel et al. 1995).

3.4 PENTACHLOROPHENOL

Pentachlorophenol (PCP) exists in soil as either a nonionic species or as an organic anion; in the pH range relevant to most environmental scenarios, the majority exists as the organic anion (USEPA 2005e). Soil pH modifies the solubility, sorption, transport and bioavailability of PCP. PCP is expected to have slight to no mobility in more acidic soils (USEPA 2005e). The half-life of PCP in soil may range from weeks to months. Fate processes for PCP in soil are primarily biodegradation and photolysis (USEPA 2005e).

PCP is rapidly absorbed, rapidly excreted, and has little tendency to persist in living organisms. PCP binds extensively to plasma proteins in mammalian tissues. It is not completely metabolized in mammals and PCP and its conjugate are readily excreted in urine (ATSDR 2000). PCP causes acute toxicity by uncoupling oxidative phosphorylation. It affects energy metabolism by increasing oxygen consumption and altering the activities of several glycolytic and citric acid cycle enzymes and by increasing the consumption rate of stored lipid (USEPA 2000a). PCP is fetotoxic and teratogenic during early gestation, however, evidence of its mutagenic effects is incomplete (Eisler 1989b).

Vegetation and Soil Invertebrates

Terrestrial plants were adversely affected when grown in PCP solution at 0.3 mg/L (root growth) and soil invertebrates were adversely affected at 1 to 5 g/m^2 soil (reduction in soil biota populations) (Eisler 1989b). PCP may be phytotoxic at soil concentrations as low as 3 mg/kg (Efroymson et al. 1997b) and may be toxic to soil biota at soil concentrations as low as 30 mg/kg (Efroymson et al. 1997a). Hulzebos et al. (1993) tested the toxicity of PCP on lettuce seedlings. Lettuce seedlings were exposed to PCP for 14 days in two loamy soils (one with 12 percent clay, the other with 24 percent clay), and in solutions for up to 21 days. Hulzebos et al. (1993) only provide EC50 values, that is, concentrations which affect 50 percent of exposed specimens. Soils with higher clay content were less toxic, even with the same concentration of PCP. The EC50 for 12 percent clay was 3.2 mg/kg and the EC50 for 24 percent clay was 8 mg/kg. Because the clay

content for soils at the site is generally less than 4 percent, the lower (i.e., more conservative) EC50 of 3.2 mg/kg was selected to represent a LOAEC for plants.

Birds

Ingestion of PCP by birds at the site may occur through consumption of flora or fauna or by incidental ingestion of soil. Fatal PCP doses for birds ranged from 380 to 504 mg/kg bw (acute oral) (Eisler 1989b). Residues (mg/kg, fresh weight [fw]) in birds found dead from PCP poisoning were 11 in brain, 20 in kidney, and 46 in liver, and ranged from 50 to 100 in egg (Eisler 1989b).

For birds, USEPA (USEPA 2005e) identified three studies suitable for evaluating the toxicity of ingested PCP on growth and survival endpoints: Nebeker et al. (1994); Stedman et al. (1980); and Prescott et al. (1982). EPA calculated NOAELs and LOAELs from these studies as follows:

- Data reported by Nebeker et al. (1994) support a NOAEL for growth in mallard ducks of 40.9 mg/kg bw and a corresponding LOAEL of 92.9 mg/kg bw. This study supports a NOAEL for mortality in ducks of 111 mg/kg-day.
- Data reported by Stedman et al. (1980) support a NOAEL for growth in chickens of 6.73 mg/kg-day, and a corresponding LOAEL of 67.3 mg/kg-day.
- Data reported by Prescott et al. (1982) support only a LOAEL for growth in chickens of 22.5 mg/kg-day. This study supports a NOAEL for mortality of 90.0 mg/kg-day.

The most conservative NOAEL of 6.73 mg/kg-day from the available acceptable studies (Stedman, Jr. et al. 1980) and its associated LOAEL of 67.3 mg/kg-day were selected as TRVs for birds.

Mammals

Data are scarce on the toxicokinetics of PCP in mammals, but studies with livestock and small laboratory animals show that the chemical is rapidly excreted (Eisler 1989b)). However, there is great variability between species in their ability to depurate PCP, as well as in their overall sensitivity. Acute oral LD50's observed in laboratory animals ranged from 27 to 300 mg/kg bw. Tissue residues were elevated at dietary levels as low as 0.05 mg/kg feed and at air levels greater than 0.1 mg/m3. Histopathology, reproductive impairment, and retarded growth were evident at doses of 0.2 to 1.25 mg/kg bw, and when diets contained greater than 30 mg PCP/kg (Eisler 1989b).

USEPA (2005e) identified and reviewed 16 studies evaluating effects of ingested PCP on survival, growth, and reproduction of mammals. Of these, studies focusing on reproduction and growth and that reported both NOAEL and LOAEL values were reviewed for this ERA.

Schwetz et al. (1974) evaluated the effect of PCP administered to rats by oral gavage on days 6 through 15 of gestation. Using data from this study, EPA calculated a NOAEL of 14.7 mg/kg-day and a LOAEL of 29.4 mg/kg-day, for both reproduction and growth (USEPA 2005g).

Welsh et al. (1987) exposed rats to dietary levels of PCP for 181 days and reported a NOAEL for rats of 3.96 mg/kg-day and LOAEL of 12.9 mg/kg-day for embryo lethality.

One dietary study on the survival of mink exposed to PCP for 162 days supported the development of an unbounded NOAEL of 0.0753 mg/kg-day for reproductive effects (Beard and Rawlings 1998). However, this study did not identify a LOAEL.

The NOAEL of 3.96 mg/kg-day and LOAEL of 12.9 mg/kg-day based upon reproductive effects on rats were selected as the TRVs for mammals.

Aquatic Biota

PCP is not a COPEC in aquatic systems at the site. Therefore, TRVs for aquatic receptors were not developed for PCP.

3.5 POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic aromatic hydrocarbons (PAHs) occur in the environment as complex mixtures associated with creosote, crude oil and other hydrocarbon products. The toxicities of individual PAHs are additive and increase with increasing K_{ow}s, but bioavailability of PAH compounds in sediment and soil decreases with increasing K_{ow} values (USEPA 2000a). Low molecular weight PAHs (LPAHs) are those with 2 to 4 hydrocarbon rings and high molecular weight PAHs (HPAHs) are those with greater than 4 hydrocarbon rings.

Environmental fate and transport and bioavailability of PAHs may be predictable on the basis of molecular weight. LPAHs are more likely to be bioavailable in sediment pore water, while HPAH are more likely to be adsorbed to sediment particles (USEPA 2000a). In water, there are several processes that affect the environmental fate of PAHs: photooxidation, chemical oxidation, and biodegradation. In general, there are no clear correlations between molecular weight and the rate of breakdown of individual PAHs in water (ATSDR 1995). However, for crude oil in aquatic systems, the PAHs that remain in the mixture after weathering are predominantly LPAHs (Stubblefield et al. 1995a). In soil, the primary process affecting the fate of PAHs is biodegradation. For PAHs in soil, there is a clear correlation between molecular weight and rate of biodegradation, with HPAH biodegrading more slowly in soils than LPAHs (ATSDR 1995).

A variety of adverse biological effects resulting from exposures to PAHs have been reported in numerous species under laboratory conditions, including effects on survival, growth, metabolism, and tumor formation (Eisler 1987b). For exposure to most PAHs, toxic effects not associated with tumor formation are not well understood (Eisler 1987b).

Like many nonionic organic chemicals, PAHs may act through a non-specific narcotic mode of action to cause aquatic toxicity (Di Toro et al. 2000). The narcosis mode of action by individual PAHs is additive. Individual PAHs may also act through other specific modes of action,

resulting in more specific effects, such as adverse reproductive effects (MacKenzie and Angevine 1981; Rigdon and Neal 1965). In mammals, toxic and carcinogenic effects of PAHs are believed to be mediated by reactive diol-epoxide intermediates that interact directly with DNA and ribonucleic acid (RNA), producing adducts (ATSDR 1995).

Vegetation and Soil Invertebrates

Plants can take up dissolved PAHs from soil pore water through their roots, and translocate them to other plant parts such as developing shoots or fruits (Eisler 1987b). Uptake rates are governed in part by PAH concentration, PAH water solubility, soil type, and PAH physicochemical state (vapor or particulate). LPAHs are taken up by plants more readily than HPAHs (Eisler 1987b). Under laboratory conditions, some plants concentrated selected PAHs above that of their immediate geophysical surroundings, but bioaccumulation of PAHs by plants has not been conclusively demonstrated. PAH-induced phytotoxic effects have rarely been observed; few studies have been published on the topic. Most higher plants catabolize benzo[a]pyrene, and possibly other PAHs, but metabolic pathways are not well described (Eisler 1987b). Individual PAH compounds were tested for toxicity to plants by Sverdrup et al. (2003) and Hulzebos et al. (1993). These two papers establish EC20 and EC50 values for growth for several PAH compounds, ranging from 37 to >100 mg/kg. These were applied in this ERA and are listed in Section 5.4.3.1.

An NOAEC for growth of isopods in a chronic feeding study by van Straalen and Verweij (1991) of 25 mg/kg soil was used as the soil TRV for invertebrates.

Terrestrial Biota

For the purposes of evaluating risks resulting from exposure of birds and mammals to PAHs, three options were considered: 1) evaluate each individual PAH separately, 2) evaluate total PAHs as a mixture, 3) divide PAHs into categories of similar compounds and evaluate as mixtures. The first option is impractical because available toxicity studies for individual PAHs in birds and mammals address only a few individual compounds sufficiently for derivation of TRVs (Kaputska 2004).[2]

For the second option, exposures to TPAH are compared to a TRV for a surrogate PAH compound with high potency, such as benzo[a]pyrene, to evaluate risks. The surrogate approach assumes that the mixture of PAHs in the environment is as toxic as the equivalent concentration of a single potent PAH, and has been used at other sites. Of all the PAHs studied in mammals, benzo[a]pyrene has the lowest NOAEL for developmental effects (ATSDR 1995), and as a result, it is often selected as the conservative surrogate compound for PAH toxicity. The assumption that all PAHs may pose toxicity as great as that of benzo[a]pyrene is clearly conservative for mammals and possibly for birds, resulting in TRVs that are unnecessarily low because benzo[a]pyrene makes up a small proportion of environmental mixtures historically found at wood treatment sites (ATSDR 1995).

[2] Benzo[a]pyrene, naphthalene, 7,12-dimethyl benz[a]anthracene (DMBA), and 3-methylcholanthrene

The third option of dividing PAHs into LPAH and HPAH, and identifying a surrogate PAH for each group was selected for this risk assessment. This method has the advantage of consolidating compounds for which there are no toxicity data individually without being overly conservative in the use of the most potent PAH to interpret exposures to all PAHs together. The method is appropriate due to differences between LPAH and HPAH for both toxicokinetics and toxicity. ATSDR (1995) uses this approach for evaluating environmental health effects. Evidence supporting this framework for evaluating exposures to PAHs is provided below.

Molecular weight of PAHs appears to be correlated to toxicokinetics in birds and mammals. For birds, there is evidence that HPAHs such as benz[a]anthracene, chrysene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene are significantly more toxic in embryos than low-molecular weight PAHs (Brunstrom 1990; Brunstrom et al. 1991). HPAH compounds are ligands of the Ah receptor (Brunstrom et al. 1991). Brunstrom et al. (1990) and Brunstrom (1991) have speculated that the embryotoxicity shown by several HPAHs is associated with EROD induction. HPAHs also bind to rat or mouse Ah receptors. Investigators agree that unsubstituted aromatic PAHs with less than four condensed rings have not shown tumorigenic activity in mammals; and that many, but not all 4-, 5-, and 6-ring PAH compounds are carcinogenic; and that only a few with 7-rings or greater are carcinogenic (Eisler 1987b).

There is evidence that the HPAHs that exhibit high embryotoxicity in birds (benz[a]anthracene, chrysene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene) also bind to the rat or mouse Ah receptor. A similarity may exist between the mechanism for the toxicity of the most potent PAHs and that of coplanar chlorobiphenyls in avian embryos (Brunstrom 1990). It has been suggested that a common mode of action exists for these high-molecular-weight PAHs in both avian and mammalian receptors, although evidence for this theory is incomplete at this time.

One study of starling nestlings found a NOAEL for dimethyl benz[a]anthracene (DMBA) (an HPAH) administered via gavage of 2 mg/kg-day and a LOAEL of 20 mg/kg-day for growth and blood chemistry effects (Trust et al. 1994). A study of birds (mallards) exposed to weathered crude oil (consisting almost entirely of LPAHs) provides evidence that LPAH mixtures are substantially less toxic; the NOAEL for mortality, reproductive effects and gross toxicity of approximately 213 mg weathered crude oil/kg-day was found for the mallard (Stubblefield et al. 1995b).

There is a similar disparity between toxicity of the most studied HPAH, benzo[a]pyrene, and the most studied LPAH, naphthalene in mammals. According to a recent literature review by Kapustka (2004), studies on benzo[a]pyrene identified a mammalian TRV as low as 1.25 mg/kg-day for developmental effects. For naphthalene, a chronic NOAEL of 100 mg/kg bw and a LOAEL of 133 mg/kg bw for gastric and liver lesions are supported by studies on mice (Borzelleca 1983; Poole and Buckley 1989).

The toxicological literature for birds and mammals consistently shows greater toxicity associated with HPAHs. In addition, current evidence suggests that the genotoxic and carcinogenic

potential of PAHs is associated with structural features of the molecule (ATSDR 1995). PAH molecules with a strong and reactive bay region structure are most likely to be genotoxic and carcinogenic (e.g., benzo[a]pyrene, benz[a]anthracene, chrysene, dibenz[a,h]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-c,d]pyrene) (ATSDR 1995). In general, HPAHs are genotoxic and carcinogenic and LPAHs are not. Due to these differences, PAHs are grouped as LPAH and HPAH for this risk assessment. TRVs based on surrogate PAHs were selected for LPAHs and HPAHs for bird and mammal receptors as described in the paragraphs below.

Birds

Few studies on the toxic effect of PAHs to birds are available (Eisler 1987b; Kaputska 2004). PAHs have been associated with impaired reproduction, growth retardation, morphological abnormalities, and metabolic and behavioral alterations in birds (Eisler 2000). Various studies have reported adverse reproductive effects on birds resulting from dietary exposure to unweathered crude oils and petroleum-derived products containing different mixtures of PAHs and other compounds (Stubblefield et al. 1995a). However, more recent studies on weathered crude oil (containing mostly low-molecular-weight PAH mixtures) showed no adverse reproductive effects at similar dietary doses (Stubblefield et al. 1995a). The literature review identified acceptable avian toxicity studies for only six individual PAHs: DMBA, benz[a]anthracene, benzo[k]fluoranthene, dibenz[a,h]anthracene, benzo[b]naphtha[2,3-d]thiophene, and naphthalene. These studies address only three bird species: European starling, domestic chicken, and bobwhite.

HPAHs—The most pertinent study of the responses of birds to acute exposures of HPAHs evaluated oral exposure of nestling starlings (*Sturnus vulgaris*) to two doses of DMBA via gavage for 5 days (Trust et al. 1994). Results indicated a NOAEL of 2 mg/kg-day and a LOAEL of 20 mg/kg-day for gross toxicological effects of decrease in body mass and decrease in blood hemoglobin concentration (Trust et al. 1994). The nestlings may have been exposed to other contaminants during the experiment.

Other studies available for birds have evaluated effects of PAHs administered via egg injection. Although investigators suggest that exposure during a 2-week toxicity test, where the embryos were exposed via yolk-sac uptake, is similar to the way avian embryos in the wild are exposed to lipophilic environmental contaminants (such as PAHs) (Brunstrom et al. 1991), resulting TRVs are difficult to interpret because egg concentrations of PAHs were not measured at the site, and reliable methods to predict PAH compounds in eggs from an oral doses are not available. Results of studies using egg injection were not used for this risk assessment. Instead, they provide perspective on the relative sensitivity of different species.

One egg injection study illustrated the difference in sensitivity among bird species to embryo mortality resulting from exposure to PAHs. Among four species of birds (chicken, turkey, domestic duck and common eider) exposed to benzo[k]fluoranthene via egg injection, the chicken was the least sensitive of the tested species at a dose of 0.2 mg/kg egg. The domestic

duck was the most sensitive, followed by the turkey, the common eider, and the chicken, with percent mortality ranging from 100 to 25 percent (Brunstrom 1990).

Because Trust et al. (1994) provide the only oral TRV for HPAH, the NOAEL of 2 mg/kg-day and the LOAEL of 20 mg/kg-day for DMBA were selected as the TRVs for HPAHs for birds. These reported values were multiplied by a factor of 0.1 to account for the uncertainty in extrapolating an acute exposure duration to a chronic exposure duration (USEPA 1997), resulting in a NOAEL TRV of 0.2 mg/kg-day and a LOAEL TRV of 2.0 mg/kg-day.

LPAH—An acute study of dietary naphthalene in 13-day-old bobwhite administered for 5 days reported a NOEC of 1000 mg/kg for reduction in body weight gain (Wildlife International 1985). Using average body weights and food consumption reported in the study, a NOAEL of approximately 364 mg/kg-day was derived. However, this study did report sufficient information on significance of difference between treatment and control groups to meet the DQOs for this ERA.

Other than the study of naphthalene (Wildlife International 1985), acceptable toxicity studies for birds exposed to individual LPAHs were not identified. Several studies have been performed on unweathered crude oil and a few studies address weathered crude oil. The weathered crude oil toxicity tests are appropriate for evaluation of LPAH mixtures because weathered crude consists largely of LPAHs. These studies evaluate the same LPAHs that are associated with wood treatment sites (Gile et al. 1982).

In multiple studies of mallards exposed to weathered Alaskan North Slope crude oil, Stubblefield et al. (1995a) found that the weathered crude oil (consisting primarily of naphthalenes, fluorenes, phenanthrene, anthracene and dibenzothiophene) presented little potential for acute toxicity to birds from oral ingestion. Acute and subacute oral exposures of mallards to weathered crude in the diet resulted in no mortalities, no effects on body weight or growth, no effects on feed consumption, and no treatment-related abnormalities. No significant effects were found on measured parameters for blood chemistry, clinical chemistry, organ weights or histopathology. As a result, Stubblefield et al. (1995a) concluded that LD50 and NOAEL values were greater than the maximum tested doses (5,000 mg/kg-day in the oral study and 50,000 mg/kg diet in the subacute dietary study).

In a follow-up 22-week study on reproductive effects of weathered crude oil on mallards, the reported NOAEL was 2,000 mg/kg diet (approximately 213 mg/kg-day) and the LOAEL was 20,000 mg/kg diet (approximately 2,120 mg/kg-day) for effects on clinical-chemistry parameters, reductions in eggshell thickness and strength, and liver and spleen weight changes (Stubblefield et al. 1995b). The results suggest that LPAHs are not highly toxic to mallards.

An acute toxicity study on a wild population of pigeon guillemots supports Stubblefield et al.'s (1995a; 1995b) findings that the potency of weathered crude oil to cause adverse effects to growth, reproduction and survival is low when birds are exposed orally (Prichard et al. 1997). In the pigeon guillemot study, wild nestlings were dosed with either 0, 0.05, or 0.2 mL of

weathered Prudhoe Bay crude oil (containing mostly LPAHs) twice at day 20 and 25 post-hatching. No significant adverse effects on growth or serum levels of sodium were observed (Prichard et al. 1997).

Based on these studies, a NOAEL of 213 mg/kg-day and a LOAEL of 2,120 mg/kg-day. LPAHs for reproductive effects were selected as the TRVs for low-molecular-weight PAHs for birds.

Mammals

Several studies have evaluated the potential adverse effects of individual PAHs on rats and mice (ATSDR 1995). Most were designed to examine specific modes of action or toxicokinetics of PAHs of concern to endpoints relevant to human health risk assessment, such as cancer (Kaputska 2004). For the purposes of ecotoxicology, three acceptable studies were identified for benzo[a]pyrene: Mackenzie and Angevine (1981); Hakura et al. (1998); Rigdon and Neal (1965).

Mackenzie and Angevine (1981) evaluated reproductive effects of benzo[a]pyrene on mice and found a LOAEL of 10 mg/kg-day administered on days 7–16 of gestation, resulting in significantly reduced fertility relative to controls. A NOAEL was not identified. Rigdon and Neal found a NOAEL for reproductive effects of 1,000 mg/kg diet (approximately 3571 mg/kg-day) administered for 10 days prior to mating (Rigdon and Neal 1965). The study by Hakura et al. (1998) evaluated tumor induction in male mice treated for 5 consecutive days and found a LOAEL of 75 mg/kg-day. No NOAEL was identified. Based upon these studies, the TRV for HPAHs in mammals was based upon the unbounded LOAEL of 10 mg/kg-day for reproductive effects adjusted by an uncertainty factor of 10 in accordance with USEPA guidance (USEPA 1997), resulting in a TRV of 1.0 mg/kg-day.

Five potentially acceptable studies evaluating the effects of naphthalene exposure in mammals were identified and reviewed (Borzelleca 1983; Kawai and Maruta 1977; Plasterer et al. 1985; Poole and Buckley 1989; Shopp et al. 1984). These studies reported a range of NOAELs from 53 mg/kg-day for gross toxic effects and liver lesions to 192 mg/kg-day for reproductive effects. Reported LOAELs ranged from 133 mg/kg-day for liver lesions to 200 mg/kg-day for gastric epithelial erosions. The only study to meet the DQOs for this ERA evaluated naphthalene toxicity in mice for 90-days by oral gavage (Shopp et al. 1984). Shopp et al. (1984) administered naphthalene at doses of 5.3, 53, and 133 mg/kg-day to male and female CD-1 mice over 90 days and evaluated effects on survival, body weight, organ weight, hematological parameters, serum chemistry data, hepatic and immunological parameters. For the endpoints relevant to this ERA, Shopp et al. (1984) reported a NOAEL of 133 mg/kg-day for mortality. Application of an uncertainty factor of 0.1 for conversion from subchronic to chronic effects, results in a NOAEL TRV value of 13.3 mg/kg-day for HPAHs in mammals. A LOAEL TRV was not identified.

The TRV based on mice is supported by a study of ferrets exposed to weathered crude oil (containing predominantly LPAHs) in a 5-day acute toxicity study (Stubblefield et al. 1995a). The ferret study found a NOAEL of 500 mg/kg-day for endpoints of mortality, gross toxicity, organ weight, and clinical chemistry parameters.

Aquatic Biota

The toxicity of PAHs to aquatic organisms is variable. In general, toxicity increases as molecular weight increases (although HPAHs have low acute toxicity, possibly due to their low solubility in water) and with increasing alkyl substitution on the aromatic ring. Toxicity is most pronounced among crustaceans and least among teleost (boney) fishes (Eisler 2000). In all but a few cases, PAH concentrations acutely toxic to aquatic organisms are several orders of magnitude higher than concentrations found in even the most heavily polluted waters (Eisler 2000). Sediments from polluted regions, however, may contain PAH concentrations similar to those which are acutely toxic, but limits on the bioavailability may of PAHs in sediment render them substantially less toxic than PAHs in solution (Eisler 2000).

Although fish efficiently metabolize PAHs, elevated concentrations in sediments have been linked to hepatic disorders, such as adenomas and carcinomas, and external lesions in common carp (*Cyprinus carpio*) and brown bullhead (*Ameirurus nebulosus*). Rice (2000) fed a marine flatfish, English sole, polychaete worms containing B(a)P, HPAH, and LPAH. The study had a fairly small samples size (N= 6 fish), endpoints were growth, CYP1A, and DNA adducts. Although this study had potential problems (test animals were collected from the field), it provides useful TRVs for HPAH and LPAH of 10.6 mg/kg dw and 0.7 mg/kg dw in the food of fish. These concentrations in food of fish caused no effect on growth after 28 days of exposure. No other PAH TRVs for fish were found.

Bioaccumulation of low-molecular-weight PAHs from sediments by *Rhepoxynius abronius* (amphipod) and *Armandia brevis* (polychaete) was similar; however, a large difference in tissue concentration between these two species was measured for high-molecular-weight PAHs (USEPA 2000a). Conclusions drawn from this study were: 1) low-molecular-weight PAHs were available to both species from interstitial water; 2) sediment ingestion was a much more important uptake route for the high-molecular-weight PAHs; and 3) bioavailability of the high-molecular weight-PAHs to amphipods was significantly reduced due to their partitioning to dissolved organic carbon. CTRs for aquatic invertebrates were difficult to find. Landrum et al. (2003) exposed amphipods to naphthalene, phenanthrene, and pyrene and found EC50s in tissue of 2.7, 1.7, and 3.5 μmol/g. The effect was mobility. These TRVs were applied in this ERA. Landrum et al. (2003) also cite a TRV for fluorine of 12.3 μmol/g, which was also applied.

3.6 POLYCHLORINATED BIPHENYLS

PCBs can produce a wide variety of responses in organisms and act as neurotoxicants, hepatotoxicants, immunotoxicants, and carcinogens (Safe 1991; Shain et al. 1991). While sensitivity and responses tend to be species-specific, general responses include lethality, reproductive and/or developmental toxicity, hepatic lesions, tumor promotion, suppression of the immune system, and induction of drug-metabolizing enzymes (Eisler and Belisle 1996; McFarland and Clarke 1989; Safe and Phil 1990).

For vertebrates, PCBs induce metabolic breakdown in the liver through enzyme induction within the cytochrome P450 system (Eisler and Belisle 1996). The degree of metabolic breakdown is primarily dependent on the degree of chlorination and the spatial arrangement of chlorine atoms. As the number of chlorine atoms in the PCB molecule increases and the number of unsubstituted adjacent carbon atoms decrease, metabolic transformation decreases. PCB elimination is limited by the highly lipophilic nature of these compounds. This causes PCBs to bioaccumulate in organisms and biomagnify up the food chain.

Of the 209 possible PCB congeners, research has indicated that as much as 75 percent of tissue burdens of PCBs in invertebrates, fish, birds, and mammals consist of only 25 congeners (McFarland and Clarke 1989). These congeners with the greatest likelihood for bioaccumulation and toxicity are the planar non-, ortho-, or mono-ortho substituted PCBs, which chemically resemble and toxicologically behave similarly to the 2,3,7,8- substituted PCDFs and PCDDs (Walker and Peterson 1991). Specifically, several lines of testing have implicated the planar PCB congeners 77, 81, 126, and 169 as major contributors to the toxicity of PCB mixtures (Ankley et al. 1991).

Examination of field and laboratory data suggests that many of the toxic effects caused by planar PCBs are mediated subcellularly by the Ah-R, the same receptor responsible for mediating dioxin toxicity. This receptor is involved in the translocation of PCBs into the nucleus and their subsequent binding to the polychlorinated hydrocarbon (PCH)–Ah receptor complex on the DNA (Safe 1991). The signs of PCB 126 toxicity in lake trout early life stages are similar to those shown by TCDD, and include yolk-sac edema, multifocal hemorrhages, craniofacial malformation, in addition to mortality (Zabel et al. 1995).

However, recent work has suggested that while the TCDD-like congeners act by a common mechanism (i.e., the Ah receptor), the combined effects of TCDD with the coplanar PCB congeners may not be strictly additive (Walker et al. 1996). Despite this uncertainty, the additive model continues to be acceptable for assessing risk because deviation from additivity has been estimated to be within an accepted tenfold range (Walker et al. 1996).

The toxicity of PCBs to mammals and birds is relatively well studied. TRVs for exposure of birds and mammals to PCBs are available as effects levels for three forms: 1) Aroclors (USEPA 2004b); 2) total PCBs; and 3) 2,3,7,8-TCDD TEQs. For the TEQ form, different sets of TEFs are available for birds and mammals, which capture (to some degree) differences in the sensitivities of birds and mammals to both PCB mixtures and to dioxins and furans. Each of these TRV forms has advantages and disadvantages. For example, while use of the TEQ approach allows risk analysts to evaluate cumulative exposures to multiple compounds, any effects not mediated by the aryl-hydrocarbon receptor biochemical pathway are not accounted for by this method.

The choice of TRV for PCBs to apply to the ERA for the St. Regis Paper Company Site was driven by the results of site-specific chemistry. Tissue data for aquatic species in Fox Creek (where PCBs are a COPEC) that are available include total PCBs data for *Lumbriculus* tissue samples, *Corbicula* tissue sample, and crayfish sample collected in 2004. Other than

Aroclor 1254, Aroclors were generally not detected in tissue of *Lumbriculus* and *Corbicula*. Individual congeners of PCBs were not measured in site-specific invertebrate tissue, but were measured and detected in earthworm tissue. Tissue concentrations of PCBs in food of bird and mammal receptors associated with aquatic habitats were expressed as Aroclor 1254. A site-specific model for estimating PCBs in aquatic invertebrate tissue is available for only Aroclor 1254 (Integral 2005) and literature-based BSAFs will be used to estimate PCB concentrations in fish available to mink, raccoon, and kingfisher. Therefore, TRVs identified for PCBs at this site to assess risk to birds and mammals were primarily effects levels for Aroclor 1254. These were used to interpret exposures either to Aroclor 1254 or to total PCBs based on the sum of Aroclors 1254 and 1260.

Vegetation and Soil Invertebrates

Efroymson et al. (1997b) reviewed three studies in which plants were exposed to total PCBs in soils. No-effects levels in all three studies were consistently at or below 20 ppm for growth endpoints, and Efroymson et al. (1997b) derive a benchmark of 40 m/kg. These were applied as the NOAEC and LOAEC, respectively, for plants.

Birds

PCB-induced reproductive impairment has been demonstrated for a number of insectivorous and piscivorous birds (Gilbertson et al. 1991; Kubiak et al. 1989; Tillitt et al. 1992). Bird embryos are the most sensitive life stage for assessing the effects of contaminants (Elliott et al. 1996; Kubiak and Best 1991). A study by Kubiak et al. (1989) showed that concentrations of 22 mg/kg in the piscivorous Forster stern (*Sterna fosteri*) eggs were associated with significantly reduced hatching success.

The basis for Aroclor-based TRVs for birds includes studies of chickens (*Gallus domesticus*) indicating LOAEL values of 0.3 to 1.4 mg/kg-day of various Aroclors for reproductive effects (Lillie et al. 1975; 1974; Platonow and Reinhart 1973). Chickens and other gallinaceous birds (e.g., pheasant) are among the most sensitive species tested for effects of PCBs and dioxins. Studies in which mallard ducks were exposed to Aroclor 1254 show no effect on reproductive success at 7 mg/kg-day (Custer and Heinz 1980) and no effect on egg production at 6.5 mg/kg-day (Risebrough and Anderson 1975). Among studies with other non-gallinaceous birds, the passerine northern bobwhite (*Colinus virginianus*) also appear to be less sensitive to effects of ingested Aroclors, with these species' NOAELs at 2.3 mg/kg-day (reproductive success) and 4.7 mg/kg-day (egg production) (Heath et al. 1969; Scott 1977). The NOAEL of 6.5 mg/kg-day was selected as most relevant NOAEL for reproductive effects on non-gallinaceous birds.

Mammals

Toxic responses in mammals to PCBs are highly species-specific, and younger mammals appear to be more susceptible to PCB poisoning than adults (Eisler 1986b). PCB-induced reproductive impairment has been demonstrated for mink (Bleavins et al. 1980; Heaton et al. 1995b; Heaton et al. 1995a; Tillitt et al. 1996; Wren 1991), and mink are the most sensitive mammal to ingested

PCBs. In a multigenerational study of mink fed the same Saginaw Bay PCB-contaminated carp, Restum et al. (Restum et al. 1998) determined that after 18 months of exposure to PCBs, mink with a dietary intake of 0.5 mg/kg PCB had significantly decreased kit survival. This study is confounded by the possibility that other chemicals were present in the fish used to dose the test animals.

Other studies show an enormous range of sensitivity in mammals. A compilation of TRVs produced by EPA for the Hudson River ecological risk assessment (USEPA 2000a) shows mammalian LOAELs ranging from 0.04 to 72.4 mg/kg-day for ingested PCBs. Two relevant chronic studies are available for mammals other than mink:

- USEPA (2000a) cites a study (Villeneuve et al. 1971) in which rabbits were fed Aroclor 1254 via gavage for 28 days and an increase in the incidence of fetal death was observed at a LOAEL of 12.5 mg/kg-day. The NOAEL from this study was 10 mg/kg-day.
- Sager and Girard (1994) fed Aroclor 1254 to female rats on alternate days during lactation and observed reduced growth in offspring at a dose of 32 mg/kg-day, with an NOAEL observed at 8 mg/kg-day.

Aulerich and Ringer (1977) observed fewer kits born alive to female mink fed Aroclor 1254 for four months, providing a NOAEL of 0.2 mg/kg-day and a LOAEL of 0.8 mg/kg-day. Mink exhibited reduced reproduction in this study at 0.7 mg/kg-day. Based on this study, a NOAEL TRV of 0.2 mg/kg-day and a LOAEL TRV of 0.8 mg/kg-day were selected for mammals.

Aquatic Biota

Invertebrates do not have an Ah receptor and are, therefore, not impacted by PCB toxicity mediated by the Ah receptor. Also, invertebrates have a limited cytochrome P450 detoxification system, so there is limited metabolic breakdown of these compounds by invertebrate metabolism. As a result, PCB toxicity to invertebrates may be less than that experienced by vertebrate species. A review of PCB toxicity by Niimi (1996) suggests that PCB concentrations of greater than 10 μg/L cause zooplankton death within a few days, and concentrations of 1 to 10 μg/L cause death over longer periods of exposure.

No studies were found in which reported concentrations of PCBs in whole bodies of freshwater aquatic invertebrates were associated with observations of survival, growth, or reproduction. Risks to aquatic invertebrates from PCBs are not addressed using tissue residues, but are addressed on the basis of sediment chemistry.

The effects of PCBs on Great Lakes fish and wildlife have been extensively studied. PCB-induced reproductive impairment has been demonstrated for several fish species (Ankley et al. 1991; Mac 1988; Walker et al. 1991a; Walker et al. 1991b; Walker and Peterson 1991; Williams and Giesy 1992). Generally, the most sensitive endpoints for effects of PCBs in fish are early life-stage survival and recruitment where exposure has resulted from transfer of PCBs from maternal tissue to eggs (Eisler and Belisle 1996; Walker et al. 1996). Whole body concentrations of PCBs in adult fish that are commonly found in the environment do not

generally result in death (Eisler and Belisle 1996). This is consistent with numerous field studies evaluating PCB fish tissue concentrations and adverse effects summarized by Niimi (1996). Based on several field studies, lethal body burden concentrations have been estimated at greater than 100 mg/kg for young fish and greater than 250 mg/kg for older fish (Niimi 1996).

Numerous studies report residues in tissue of fish affected by PCBs administered through water only, food only, or water and food combined. Acceptable NOAEC values for studies evaluating effects on growth following chronic exposures to Aroclors range from whole body concentrations of 1.9 mg/kg ww Aroclor 1254 in channel catfish *(Ictalurus punctatus;* Mayer et al. 1977)(*Ictalurus punctatus;* Mayer et al. 1977) to 645 mg/kg ww Aroclor 1254 in adult coho salmon *(Oncorhynchus kisutch;* Mayer et al. 1977) (*Oncorhynchus kisutch;* Mayer et al. 1977). Available LOAECs for fish range from 40 mg/kg ww Aroclor 1248 in fathead minnow (20 percent reduction in body weight of progeny) to 360 mg/kg ww Aroclor 1248 in fathead minnow (egg production and hatching success; DeFoe et al. 1978). DeFoe et al.(1978) reported a NOAEC of 25 mg/kg ww and the LOAEC of 40 mg/kg ww for a sensitive reproductive endpoint. Although these values from DeFoe are applied in this risk assessment, variation in critical residue effect levels for PCBs suggests that this type of TRV is not a reliable metric for evaluation of risk to fish.

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APPENDIX E3

RECEPTOR PROFILES

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Appendix E3
Receptor Profiles

Prepared for
International Paper
pursuant to
Unilateral Administrative Order Docket No. V-W-04-C-796

Prepared by

integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80021

September 28, 2007

CONTENTS

LIST OF ACRONYMS

DNR	Department of Natural Resources
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ha	hectare

1 INTRODUCTION

Receptors were selected for the ecological risk assessment (ERA) to represent species of potential concern and the range of feeding guilds expected to inhabit terrestrial and/or aquatic habitat types at the Site. Patterns of behavior, life history, and habitat use that affect the frequency and magnitude of exposure of each receptor to hazardous substances at the Site are quantified to estimate exposures for the ERA. Quantitative estimates of parameters describing rates of ingestion of water, soil, and food; information on life history (e.g., timing of migration and breeding); and habitat areas for each receptor are used to build exposure models. Specific information about how receptors use habitats at the Site is also used to interpret the ecological significance of estimated exposures relative to effects thresholds.

Not all receptor groups are addressed in this appendix; aquatic and terrestrial invertebrate and plant species are not discussed. Only fish, reptiles, birds and mammals are discussed below because specific life history or biological factors affect exposures in a manner relevant to risk estimation for these receptors. Quantitative biological variables required for modeling the exposures of fish, snapping turtle, birds, and mammals included:

- Body weight (kg)
- Average home range (km)
- Rates of ingestion of food, water, and soil or sediment (g/kg bw-day)
- Composition of the diet.

Other variables that can be important to make risk models more realistic or that support qualitative interpretation of estimated exposures relative to effects levels include:

- Numbers of breeding cycles per year
- Numbers of young per brood and period of gestation
- Seasonal patterns of migration and months expected on site
- Seasonal changes in diet
- Preferences for certain habitat types or physical conditions
- Density and territoriality.

To compile the necessary information, estimates of relevant descriptors of each receptor were taken from primary scientific publications and from synthetic reviews *(e.g., Wildlife Exposure Factors Handbook;* USEPA 1993). Available data include a range of studies from across North America and Europe conducted in a variety of climates and habitat types. In many cases, only one or two studies were available to describe a species of interest. When multiple values for a

relevant exposure parameter were found in the literature, the following were considered in the final selection of a quantitative value:

- Geographic proximity to the Site
- Ecological similarity to the Site (e.g., mixed deciduous riparian zones for semi-aquatic species, bluegrass/shortgrass prairie or mixed coniferous forest for terrestrial species, freshwater wetlands for species using Fox Creek, etc.)
- Climatic similarity to the Site (e.g., northern temperate ecosystems).

The following sections provide the basis for specific exposure assumptions used in the exposure modeling. Detailed exposure assumptions are presented in Section 5.3 of the main text of the report. Receptor profiles also provide context for interpretation of risk models.

2 FISH

Four fish species found in Cass Lake are targeted for ecological risk evaluation: white sucker (*Catostomus commersoni*), lake whitefish (*Coregonus clupeaformis*), yellow perch (*Perca flavescens*), and walleye (*Sander vitreus*). These species were selected because they are found in different types of aquatic habitat and have different feeding behaviors and life histories. They may also be important prey items for upper trophic levels as well as valuable fish for human harvest. Relevant information on each species is summarized below.

2.1 WHITE SUCKER

The white sucker is one of Minnesota's most common fish (Minnesota DNR 2005). Adults are most often found in the warm shallows of streams and lakes. In Area B aquatic habitats, adult white suckers are likely to be found along the shoreline of Pike Bay, and may be present in Fox Creek and the Channel. Spawning may occur along the shallow shoreline of Pike Bay and the channel. Juveniles may also be present along the shoreline of Pike Bay and in the Channel.

White suckers are bottom-dwelling omnivores and feed on plants, benthic invertebrates (e.g., clams, molluscs, worms), and other organic material or detritus from the benthic environment of lakes and streams. The average life span for white suckers is 10–12 years; the maximum life span is as long as 17 years (Fishbase 2005; Minnesota DNR 2005). They typically grow to about 30 cm long and weigh from 0.9 to 1.4 kg (Minnesota DNR 2005). No information on home range or optimal population densities of white sucker was found.

Adult white suckers spawn in April and May when water temperatures reach about 10°C (McCormick et al. 1977; Roberge et al. 2002). White suckers spawn in shallow water (0.6–1.2 m) of lakes and streams with gravel substrate (Roberge et al. 2002). The time to hatching varies with temperature, but is typically 5–10 days (Minnesota DNR 2005). White sucker fry remain in or near the spawning areas for one to two weeks after hatching, and then leave the gravel and move downstream. Juveniles form schools and remain in shallow water along the shore during the day and move to deeper water at night. Unlike the adults that feed solely on the bottom, juveniles feed on plankton near the water surface. By late summer, or when temperatures reach about 30°C, the young suckers move to the bottom.

White suckers are important prey species for several sport fish, including walleye, trout, northern pike, and bass. Small suckers may also be eaten by birds. Small suckers are commercially harvested for use as bait (Minnesota DNR 2005).

2.2 LAKE WHITEFISH

Lake whitefish adults are found in cool, deep (up to 60 m) waters of lakes. In Area B, juvenile lake whitefish are likely to occur on the shoreline of Pike Bay and possibly in the channel. They would not be expected in Fox Creek. Adult lake whitefish dwell in the deeper oxygenated waters bottom of deep lakes like Cass Lake and are less likely to be found in Area B aquatic habitats. The exception is during spawning where the shallow lakeshore areas of Pike Bay include suitable spawning habitat.

Adult lake whitefish feed on or near the bottom, ingesting crayfish, zooplankton, freshwater shrimp, small fish and fish eggs, and other bottom organisms. The typical lake whitefish life span is 10 years, but individuals as old as 25 years have been reported (Roberge et al. 2002). Typical adult size is 1.8–2.7 kg in weight and 40–45 cm in length (Minnesota DNR 2005). No information on home range was located. While lake fish are reported to form schools, no information on typical population densities was located.

Adult lake whitefish spawn in early winter (usually mid-October to early December) when water temperatures fall below 7°C (Roberge et al. 2002). Spawning is in shallow (less than 7 m deep) rocky- or sandy-bottomed lake waters (Lasenby et al. 2001; Michigan DNR 2005). Mud is avoided for spawning (Roberge et al. 2002). Young lake whitefish hatch in spring and begin swimming and feeding within a few days. Juveniles remain in shallow lake waters with sand or rock substrate until moving to deep water in early summer. The juveniles feed on planktonic crustaceans and insect larvae (Minnesota DNR 2005).

Small lake whitefish are prey for trout, northern pike and burbot; adult lake whitefish are prey for the sea lamprey. In Minnesota, there is a small sport fishery for this species, and it is a popular food fish for human consumption (Minnesota DNR 2005).

2.3 YELLOW PERCH

Yellow perch occur in all major drainages in Minnesota and are generally more tolerant of warmer, low-oxygen waters than the other fish receptor species (Minnesota DNR 2005). They are found in a variety of nearshore, shallow (less than 9 m deep) water habitats, including lakes, slow-moving rivers, and ponds (Michigan DNR 2005). Preferred temperatures for adult yellow perch are 19–23°C (Minnesota DNR 2005). They are associated with various substrates including muck, sand or gravel, and vegetation. Adult yellow perch could be expected in shoreline of Pike Bay, Fox Creek, and the channel. The shore of Pike Bay and the channel are the most likely spawning habitat, and juveniles are likely to be present in these areas. Spawning by yellow perch in Fox Creek is unlikely because of the soft bottom substrates.

Adult yellow perch generally feed in the water column on zooplankton, minnows, and other small fish and in littoral vegetation beds on epiphytic or benthic insects. The average life span

is 7–10 years, but may extend up to 13 or more years. Typical adult size is 20–29 cm and 170–300 g (Minnesota DNR 2005).

The yellow perch is a schooling fish. They can overpopulate in the absence of predators, and productivity appears to be cyclical, with one year-class very abundant and another year-class nearly absent (Minnesota DNR 2005). No specific information on density or home range was located.

Adults spawn after ice melt between April and mid-May when water temperature reach about 7°C. Spawning takes place in sheltered, shallow (0.6–3 m deep) waters with sand, gravel, or vegetated bottoms (Minnesota DNR 2005). The time to hatching depends on temperature, but is generally about 2 weeks. The fry move downstream to pools, backwaters, or lakes. Juvenile yellow perch often form schools and remain in shallow water with some vegetation or other cover (Roberge et al. 2002). They feed on plankton and algae.

Yellow perch are a common prey for many piscivorous fishes and in many lakes may be the most important prey species for bass, pike, and walleye. Piscivorous birds, such as herons, gulls, mergansers, and loons, also feed on yellow perch. Yellow perch are one of the most commonly caught fish in Minnesota (Minnesota DNR 2005).

2.4 WALLEYE

Walleye occur in all major drainages in Minnesota and are most commonly found in clear, deep lakes (Minnesota DNR 2005). Walleyes may migrate up to 100 miles or more in large river systems to find suitable spawning habitat, but self-sustaining populations have also developed after stocking land-locked lakes (Wisconsin DNR 2005). They are most common in lakes over 500 acres, and are less common in lakes under 100 to 200 acres. Their preferred water temperature is 20–22°C (Michigan DNR 2005; Minnesota DNR 2005). The typical life span is approximately seven years, but can be as long as 20 years. Adult size commonly ranges from 38 to 48 cm and from 2.5 to 4 kg, but lengths up to 66 cm have been reported (Minnesota DNR 2005).

Walleye are most often found in deep lakes with clear, cold water. Preferring to avoid bright light, they are found in shadows or deep in the water column during the day but will move to shallow waters to feed at dusk and dawn. In Area B, adult walleye are likely to be present for limited periods (i.e., while feeding at dusk and dawn and during spawning in early spring) along the lakeshore of Pike Bay. Juvenile walleye are also likely to be found along the lakeshore and Pike Bay, and possibly in the channel. Walleye are not expected to occur in Fox Creek.

Adult walleye spawn in early spring soon after ice melt when water temperatures reach 4–7°C. Spawning occurs on rock or gravel in tributary streams, in lakes if good inlet streams are unavailable, or in shallow (0.3–1.8 m) nearshore waters (Minnesota DNR 2005; Roberge et al. 2002). The spawning area must have enough current to clear away fine sediment and aerate the

eggs. Because there are no clean gravels in the shoreline of Pike Bay, Fox Creek or the channel, walleye would not be expected to spawn in these areas. The embryos hatch in about 1–3 weeks, depending on water temperature (Roberge et al. 2002). Larval walleye are generally found along shore during the night and may seek cover in aquatic plants during the day, but they soon move into deeper, open water (Michigan DNR 2005). Year classes are highly variable, and only one year in four or five may contribute significantly to the adult population (Wisconsin DNR 2005).

Adult walleye are strictly carnivores, feeding primarily on fish (including yellow perch) and secondarily on insects and other aquatic animals (e.g., crayfish, mudpuppies). Young walleye are carnivorous, ingesting zooplankton and small insect larvae in the water column (Roberge et al. 2002).

Young walleye are eaten by northern pike, tiger muskellunge, largemouth bass, and older walleye. Humans are the primary predator of adult walleye. Walleye are Minnesota's most popular sport fish and highly desirable for human consumption (Minnesota DNR 2005).

3 REPTILES

The snapping turtle (*Chelydra serpentina*) is the only reptile selected as a receptor for the ERA. This species was selected because snapping turtles spend most of their lives in water and are omnivorous, consuming plants, small fish, insects, snakes, and carrion. Additional information on their life history and feeding behavior is provided below.

3.1 SNAPPING TURTLE

The snapping turtle is among the largest of freshwater turtles, and is found in permanent ponds, lakes, marshes, and larger rivers, though they will occasionally travel over land. Adult snapping turtles reach 37 cm in length; average weight of a Michigan population of snapping turtles was 5.5 kg for males and 5.0 kg for females (USEPA 1993). Males generally attain larger sizes and can be up to twice as large as females (USEPA 1993). Snapping turtles are found throughout southern Canada and the continental United States east of the Rocky Mountain range (Dillon 1998), and are common, year-round residents in the Cass Lake region (Nelson et al. 2004). Snapping turtles are vulnerable as young hatchlings, but after their first few years of life they have few natural predators. This species is estimated to live up to 30 years in the wild (UMMZ 2005).

Snapping turtles are omnivores; the diet may include insects, crustaceans, bivalves, earthworms, fish (adult, fry and eggs), amphibians, snakes, birds, small mammals, plants, and algae. A dietary shift may occur in the early spring when aquatic vegetation is limited, forcing them to focus primarily on animal matter during this time. Young snapping turtles are primarily carnivorous and prefer smaller streams where aquatic vegetation is less abundant (USEPA 1993). Predators of the eggs and hatchlings of snapping turtles include other turtles, great blue herons, crows, raccoons, and large predatory fish. However, once snapping turtles become larger, there are few animals that prey on them (UMMZ 2005). Snapping turtles are active mainly at dawn and dusk and nocturnal, and may bask in the sun or move into deeper waters during the day to regulate their temperature.

Nesting occurs between late spring and fall, peaking in June, with older females nesting earlier in the season than younger ones (USEPA 1993). Females do not begin laying eggs until they reach an appropriate size–approximately 200 mm carapace width, but may be larger at higher latitudes (Dillon 1998), which usually occurs sometime between age 6 to 19 years (USEPA 1993). Females will often move some distance outside of their normal foraging range to lay eggs; one study found that females tagged at their nesting site moved an average of 5.5 (±1.8 SD) km from the nest site afterwards (USEPA 1993). Eggs take 9 to 18 weeks to hatch, dependant on temperature (UMMZ 2005). In northern populations, hatchlings may over-winter in the nest before emerging the following spring.

Snapping turtles are not social animals, and interactions outside of breeding are limited to aggressive behavior mainly between males. Most turtles stay within the same marsh or aquatic system from year to year, except when breeding. Reported home range sizes for adult snapping turtles range from 0.007 to 0.089 km^2; home ranges can overlap both between and within sexes (USEPA 1993). Young turtles tend to remain in small streams until shortly before sexual maturity, when they migrate to habitats preferred by adults (ponds, marshes, lakes). Snapping turtle density appears to be positively correlated with the productivity of the surface water (e.g., higher densities in eutrophic than oligotrophic systems) (USEPA 1993).

4 MAMMALS

The following mammals are selected as receptors for the ERA: raccoon (*Procyon lotor*), muskrat (*Ondatra zibethicus*), mink (*Mustela vison*), meadow vole (*Microtus pennsylvanicus*), and short-tailed shrew (*Blarina brevicauda*). These species were selected because they occupy different habitats and have different feeding behaviors and life histories. Relevant information on each of these species is provided in the following sections

4.1 RACCOON

The raccoon is the most abundant and widespread medium-sized, omnivorous mammal in North America (USEPA 1993). Raccoons exploit a wide variety of habitats; areas associated with water are particularly important, as raccoons use water for both drinking and foraging. Habitats include floodplain forests, swamps and marshes. Because of this close association with water, raccoons were selected as a receptor in Area B, which includes aquatic and the near-shore terrestrial portions of the Site. Raccoons are extremely adaptable to human environments, and can be found in abundance in suburban residential areas and farmlands. High-quality habitat for raccoons includes sites that have access to fresh water, trees or other structures for nesting, and high food availability including fruits, grains, invertebrates and other animals.

Raccoons are permanent, year-round residents in the Cass Lake region (Nelson et al. 2004). Adult male raccoons collected in an Illinois study averaged 7.6 kg; parous females (having produced at least one litter) averaged 6.4 kg; and nulliparous females averaged 6.0 kg (Sanderson 1987). Mortality is high in young-of the year raccoons; average lifespan in the wild is 5 years, with a maximum recorded age of 16 years (UMMZ 2005).

Raccoons are highly opportunistic feeders and omnivorous, with a diet that may include carrion, garbage, birds, mammals, fish, amphibians, reptiles, grains, fruits, most food prepared for human or domestic animal consumption, agricultural crops, and invertebrates including insects, crayfish and mussels. Proportions of different foods in the diet depend on location and season. Plant foods dominate raccoon diets for most of the year except during spring and early summer, concurrent with the breeding season, when animal matter may be consumed more frequently (USEPA 1993). Food ingestion rates for raccoons were not found in the literature.

Raccoons escape many predators by remaining active during the day in a den, and are alert and can be aggressive when active at night. Large predators may prey on raccoons, including coyotes, wolves, and owls, and their young may be taken by snakes (UMMZ 2005).

Throughout most of North America, raccoons mate during February and March. Most females will produce one litter per year, and many raccoons produce litters within their first year of life. Gestation averages 63 days (Sanderson 1987), and most litters consist of three to four young. Nesting sites are primarily in hollow trees, but raccoons will also use ground dens, brush piles,

and abandoned human structures for nesting, usually within a few to a few hundred meters of surface water.

Population densities are strongly dependent on habitat quality, including food availability and abundance of potential nest sites, with suburban areas generally having higher densities than rural/wild areas. Home range areas range from less than 0.05 km^2 in suburban neighborhoods to more than 5 km^2 in the wild, though values of one to a few km^2 are most commonly reported (USEPA 1993). Juvenile and adult males tend to have larger home ranges than do females (Sanderson 1987).

4.2 MUSKRAT

The muskrat is a semi-aquatic, primarily herbivorous rodent that is common and widely distributed throughout North America. The muskrat is a permanent, year-round resident in the Cass Lake region (Nelson et al. 2004). Muskrats' anatomical adaptations to an aquatic environment include a thick, waterproof underfur overlaid by long guard hairs and proximally webbed hind feet. High-quality muskrat habitat includes lentic or slow-flowing, low-gradient, permanent waters with depths of 0.5–1.2 meters, including marshes, lakes, ponds, sloughs and streams. Waterways supporting abundant aquatic vegetation bordered by dense herbaceous fringe and upland vegetation, and abundant retreats including debris, pools, undercut banks, and backwaters provide preferred habitat. Predators of muskrat include mink, raccoon, owls, snapping turtles, large fish such as bass and bowfin, and house cats.

Reported weights of adult males range from 0.7 to 1.6 kg, and reported weights of females range from 0.8 to 1.5 kg (USEPA 1993). Expected average lifespan is 3 years; the longest lifespan recorded is 10 years for a muskrat in captivity (UMMZ 2005).

Muskrats feed on the roots and basal portion of aquatic vegetation. Important plants include cattail (*Typha* spp.), which provides food and the preferred building material for muskrat houses (Erb and H.R.Perry Jr. 1982). Other important plant foods include sedges (*Carex* spp.), sweetflag (*Acorus*), and wild rice (*Zizania*). Shoots, bulbs, and plant leaves may also be consumed. Carnivory has been suggested to result from either a shortage of preferred vegetation or an effort to supplement the diet with high-nitrogen food sources (Erb and H.R.Perry Jr. 1982). Vegetation makes up more than 97 percent of the muskrat diet (USEPA 1993).

The muskrat breeding period in the northern U.S. is restricted to between mid-June and August, with one to three litters produced per female each season. Where ice cover is present in winter, breeding is initiated after waterways become ice-free (Erb and H.R.Perry Jr. 1982). Age at first breeding is approximately 12 months in the northern range of muskrat distribution; the gestation period is typically 30 days, and nine to 10 young are typically produced per litter in the northern U.S. (Erb and H.R.Perry Jr. 1982). Young are usually weaned in the fourth week, and by the time young are independent, the mother is often ready to give birth again.

Muskrat dwellings take two forms: burrows excavated in shoreline banks, or lodges constructed of emergent vegetation. Although muskrats may use a variety of sites for burrows, they prefer sites with low water velocity, high banks with steep slopes, clay-rich soils for dens, and abundant shoreline/aquatic vegetation. In one study, 96 percent of lodges were found to be constructed within 1 m of water (Allen and Hoffman 1984). Moderate depths of 0.5–1.2 m are preferred (Allen and Hoffman 1984).

Muskrat populations are cyclical as a result of changes in food availability, development of disease resistance, habitat quality and quantity of suitable nesting sites, and socially induced changes in reproduction. Muskrats are territorial, especially during the breeding season, and dispersal in the fall is common. A home range value of 0.002 km^2 reported for two North American regions (Ontario and Iowa; USEPA 1993) was assumed to be the home range of muskrats using aquatic habitats at the Site. Population density may vary widely; reported densities include 23 muskrats per km of riverbank along a Pennsylvania river to 48 muskrats per km of riverbank in a Massachusetts wetland/river habitat (USEPA 1993).

4.3 MINK

Mink are a carnivorous, predominantly nocturnal, semi-aquatic mammal widespread through most of North America. This species is associated with streams, rivers, lake shores, and marshes. Wetlands with irregular, diverse shorelines and dense vegetation provide the most suitable mink habitat (Allen 1984). The mink is a common permanent, year-round resident in the Cass Lake region (Nelson et al. 2004). Adult males average 1.2 kg, and females 0.55 kg in a Montana population sampled in fall (USEPA 1993). The mink's maximum lifespan is approximately 10 years (UMMZ 2005).

Shoreline and emergent vegetation are primary hunting areas of the mink. Its diet varies depending on location and season. Mammals are the most important prey for many mink populations, but mink also consume fish, crustaceans, and terrestrial insects. In marsh habitats in summer, muskrats can be an important food source depending on their population density and vulnerability (USEPA 1993). Diet may vary with water level; one study found a shift in diet from voles and crayfish during periods with high water levels to a diet dominated by birds and muskrats from deeper in the marsh during periods of low water levels (USEPA 1993). Mink are aggressive animals and do not hesitate to defend themselves, even against larger animals, and so have few natural enemies (besides humans). They are occasionally prey to coyotes, bobcats, and other carnivores (UMMZ 2005).

The availability of den sites may limit the number of mink a wetland area can support. The home ranges of mink follow the shape of the water body on which they live. The size of the home range is directly affected by the amount and density of vegetative cover, so when vegetation is sparse, the home range is greatly expanded (Allen 1984). Females tend to use a more restricted area than males, whose foraging areas may overlap except during the breeding

season from late February to early April (USEPA 1993). Population densities generally fall in the range of 1 to 10 minks per km^2 (USEPA 1993). A linear home range of 3.7 km shoreline was used for this assessment (Linn and Birks 1980).

4.4 MEADOW VOLE

The meadow vole is a small, primarily herbivorous rodent that is distributed throughout Canada and the northern U.S., from southern Alaska to northern Georgia at its southernmost range. This species is a permanent, year-round resident in the Cass Lake region (Nelson et al. 2004). Meadow voles are found primarily in areas with dense herbaceous vegetation such as prairies and agricultural fields, and along fencerows, rights-of-way and railroads, and in riparian short- and tall-grass habitat. All of Area A and all terrestrial portions of Area B (excepting roads and buildings) were therefore considered suitable habitat for this species for this assessment. This species forms a network of well-kept runways through vegetation and litter that is used as protection against predation, to maintain body temperature, and for access to food.

Voles are primarily herbivorous, with less than 10 percent of the diet from animal sources, including insects and other animal matter (Pugh et al. 1982). Meadow voles feed primarily on the leaves and shoots of succulent green vegetation; roots, bark, seeds, and fungi may also be eaten. Unlike many rodents, voles do not hibernate or exhibit torpor, and must find food to meet their metabolic needs year-round. Reported weights of adult voles range from 17 to 52 g (USEPA 1993), with an average of 37 g. Average life span is less than 1 year, but may be as long as three years in captivity (UMMZ 2005). Predators of voles include crows, herons, hawks, owls, short-tailed shrews, coyotes, trout, bullfrogs, and snakes.

Microtus species have high reproductive potential during their short life span. Females may reach reproductive maturity as early as 3 weeks of age. Reproductive activity occurs throughout the year, but peaks from May to October, coincidental with the period of highest moisture availability (USEPA 1993). Nests generally lie about 12 cm below the soil surface and are approximately 10–15 cm in diameter (Pugh et al. 1982). The gestation period is 20–23 days, and weaning occurs between days 8 and 17 (Pugh et al. 1982). As evidence of their reproductive potential, a theoretical calculation showed that beginning with 100 pairs of voles in April, a population of 8,900 voles could be produced by September (Pugh et al. 1982).

Many populations of voles, including *M. pennsylvanicus*, undergo multiannual population cycles. Peaks occur at intervals of 4–5 years, and may be related to food supply, predation, or an interaction of these two factors. Male and female prairie voles defend shared territories, and offspring tend to remain in the natal territory.

4.5 SHORT-TAILED SHREW

The short-tailed shrew is a small, primarily carnivorous rodent that is more active at night than in the day and is widely distributed throughout temperate North America. This species is a permanent, year-round resident in the Cass Lake region. Short-tailed shrews are common in areas with abundant, dense vegetation, and need cool, moist habitats because of their high metabolic rate and rapid rate of water loss (USEPA 1993). All of Area A (except for roads and buildings) and all terrestrial portions of Area B (except for roads and buildings) were assumed to be capable of supporting short-tailed shrew for the purpose of this assessment.

The small body size of the shrew results in a very high metabolic rate that requires the shrew to ingest food at a much greater rate per kg body weight than most other mammals. Body weights of shrews range from 12.5 to 22.5 g (USEPA 1993). Northern short-tailed shrews have been known to live as long as 3 years, but most die within their first year of life (UMMZ 2005). Short-tailed shrews consume a variety of animal matter, primarily invertebrates as earthworms, snails, and beetle larvae, and may also eat small mammals and amphibians. Shrews also consume fungi and other vegetation. A summer survey of 220 short-tailed shrew stomach contents found earthworms, slugs, snails, fungi, and beetles to be among the most common taxa present (Whitaker and Ferraro 1963).

Predators of short-tailed shrew may include owls, snakes, hawks, weasels, foxes, and coyotes. However, short-tailed shrew can exude a musky substance from glands on their belly and sides, and many mammal predators, such as weasels and foxes, may refuse to eat them because of their foul taste (UMMZ 2005).

The breeding period of the short-tailed shrew generally extends from early spring through early fall (March–September), though scattered reproductive activity may occur throughout the year (UMMZ 2005). Nests are built out of shredded leaves or grass placed underground in burrows or under logs or rocks. Burrows are generally near the surface but may be as deep as 20 inches below surface (GMNH 2005). Females reach sexual maturity at six weeks, and males at 12 weeks. Two to three litters of five to seven pups are produced per year; gestation is 21–23 days, and young leave the nest at 18–20 days of age and are weaned several days later (UMMZ 2005).

The short-tailed shrew is not gregarious or sociable. Territories generally do not overlap, and are related to prey density. Home range areas are related to prey density; home ranges have been reported as small as 0.0003 km^2 in areas with high prey densities, to 0.02 km^2 at low prey densities (USEPA 1993). Densities are seasonally variable; for example, densities of short-tailed shrew in an Illinois alfalfa field averaged 2.3/ha in winter to 11.3/ha in summer (USEPA 1993).

5 BIRDS

Birds selected as receptors for the ERA include the belted kingfisher *(Ceryle alcyon)* for the shoreline of Pike Bay and the channel, great blue heron *(Ardea herodias)* for Fox Creek, American robin *(Turdus migratorius)* for terrestrial portions of the Site, and mallard duck *(Anas platyrhynchos)* for Pike Bay, the channel, and Fox Creek. These species were selected because they are found in different habitats and have unique life histories and feeding behaviors. Relevant information on each of these species is provided in the following sections.

5.1 BELTED KINGFISHER

The belted kingfisher is a piscivorous, medium-sized bird (33 cm bill tip to tail), weighs approximately 158 g (USEPA 1993), and is commonly found along watercourses. The kingfisher was selected to represent other piscivorous birds in the ERA because its diet is dominated by fish, it migrates from the Site during winter months, and it can be found associated with any of the aquatic habitats at the Site. Other piscivorous birds in the area include loons, mergansers, herons, bald eagles, and ospreys. Kingfishers were observed using the Pike Bay shoreline in July 2005, and likely use the channel connecting Pike Bay to Cass Lake; both areas provide shallow water habitat with perches for foraging. However, upstream of the beaver pond at its mouth, Fox Creek is unlikely to be used by kingfisher because it has poor visibility and few overhanging perches.

This belted kingfisher is a summer breeding resident in the Cass Lake region (Nelson et al. 2004), but likely migrates south in winter to find ice-free water for fishing. Kingfishers are thought to be relatively long-lived; the oldest birds recorded in the kingfisher family were greater than 15 years old (UMMZ 2005).

Kingfishers feed mostly on fish that swim near the surface or in shallow water (<60 cm depth). Ideal foraging conditions for the kingfisher include water with low wave action, an average transparency greater than 40 cm depth, and relatively little cover by floating and/or emergent aquatic vegetation, which might obscure prey. Kingfishers are virtually absent around turbid waters such as at river deltas or during periods of high runoff that increase turbidity. Kingfishers prefer a bare branch or snag at the water's edge as an observation perch for fishing; telephone wires, stakes, or piers may also be used. Sizes of fish consumed by kingfisher averaged less than 7.6 cm with a maximum size of 17.8 cm in a Michigan study (Prose 1985). Kingfishers are opportunistic in terms of species of fish consumed, taking species in proportion to their abundance. Crayfish can also be important to the diet of kingfishers. A variety of other items may be included as alternate foods when fish are scarce, including mussels, frogs, snakes, turtles, insects, salamanders, newts, young birds, mice, and berries.

Belted kingfishers will mob predators and be quite aggressive. In addition, predation on nestlings is low because they are raised in protected nest cavities. Fledgling kingfishers may fall

prey to hawks, and additional known predators of this species include mink, raccoons, and snakes (UMMZ 2005).

Belted kingfishers excavate nesting burrows within their breeding territories. Burrows are built into the side of a vertical, bare bank or cliff in sandy soil at least 1.5 m from the cliff base whenever possible. Sites can include man-made banks such as railroad banks, roadside cuts or gravel pits. Where suitable nest sites are in short supply, kingfishers may resort to unusual nest sites, including very low soil banks, soil among the roots of a fallen tree, decaying tree stumps, and holes in dead trees. Nest sites are as close to water as possible, within 1.6 km of fishing sites (Prose 1985). Burrows may be used for more than one season (USEPA 1993). Six to eight eggs are laid per pair; both parents incubate eggs for a period of 22-24 days and feed the young, who remain with the parents for 10 to 15 days after fledging (UMMZ 2005; USEPA 1993).

In a study in Michigan, stream territories tended to be longer than those on lake shorelines (Salyer and Legler 1946, as cited in Prose 1985). While non-breeding territory size appears to be inversely related to food abundance, breeding territory size appears to be more strongly related to the distribution of food sources, with the smallest territories containing the richest food sources near the nest.

5.2 GREAT BLUE HERON

The great blue heron is the largest member of the heron family in North America, with body weight of males averaging slightly greater than body weight of females. A mean value of 2.2 kg for both sexes was assumed for this ERA (USEPA 1993). The great blue heron is found in freshwater and nearshore marine habitats throughout North and Central America. Habitats for great blue herons include streams, creeks, lake margins, and estuaries, with shallow water (<0.5 m) and a firm substrate on which to wade. Nearby wooded cover (within a few kilometers) is important for nesting. The great blue heron is a seasonal breeding resident of the Cass Lake region (Nelson et al. 2004); the breeding season in the northern part of its range is typically March–May.

The preferred prey of great blue herons is fish; great blue herons will also eat amphibians, reptiles, crustaceans, insects, birds, and mammals (Alexander 1997; USEPA 1993). When fishing, great blue herons use either a sit-and-wait strategy, waiting for prey to move within striking distance, or slow wading to catch more sedentary prey such as sculpin. They require shallow waters (to 0.5 m) with a firm substrate for fishing. Great blue herons consume relatively small fish that can be swallowed whole; 95 percent of fish consumed by a Wisconsin population of great blue herons were less than 25 cm in length (USEPA 1993).

In some areas, herons defend feeding territories, but in other areas they are opportunistic and lack fidelity to a particular feeding site (USEPA 1993). Adult herons tend to feed the same type and size of food to their nestlings as they consume themselves. Predation on herons is mainly on eggs and young. Predators of young great blue heron eggs include crows and ravens.

Eagles, raccoons, bear, and hawks prey on the young birds and occasionally even adults (UMMZ 2005).

Great blue heron nests generally consist of a stick platform over 1 m in diameter; the nests may be re-used and expanded for multiple years. Only one brood, with an average clutch size of 3 to 5 eggs, is raised per year, although if the clutch is destroyed the parents may lay a replacement clutch. Both parents incubate and feed the young. Chicks fledge at approximately 2 months (UMMZ 2005). During the breeding season, great blue herons are monogamous and colonial. Breeding colonies are generally close to foraging grounds; a study of great blue herons in Minnesota lakes found the distance between nesting colonies and feeding sites to range from 0 to 4.2 km, averaging 1.8 km (USEPA 1993).

The green-backed, or little green heron is a smaller member of the heron family that was observed in Fox Creek in July 2005, and has similar habitat requirements, preferring waterways with adjacent wooded cover. The great blue heron is used to represent heron species found along Fox Creek where use of the kingfisher as a receptor is inappropriate.

5.3 AMERICAN ROBIN

The American robin is a medium-sized bird, with an average adult body weight of 80 g (Wheelright 1986). The American robin occurs throughout most of North America including northern Minnesota during the breeding season and winters in the southern half of the U.S. and in Central America. The robin is a summer breeding resident in the Cass Lake region (Nelson et al. 2004). Robins inhabit woodlands, swamps, suburbs, fields, and parks. Robins forage on the ground in open areas, along edges of streams or fields, as well as above ground in shrubs and the lower branches of trees. High-quality habitat for robins includes sites that have access to fresh water, protected nesting sites, and productive foraging areas high in fruit and invertebrate resources. Forest edges, high invertebrate abundance, and fruit-bearing trees and shrubs indicate suitable habitat for this species. The species has an average life span of 2 years in the wild and a 25 percent survival rate past the first summer (UMMZ 2005).

The robin's diet consists of fruit and insects or other invertebrates. This species' dietary composition shifts seasonally. In the months preceding and during the breeding season (April-July, UMMZ 2005), robins feed mainly on invertebrates, primarily earthworms but also spiders, beetles, larvae of butterflies and moths, and ants (USEPA 1993). During the non-breeding season, robins forage primarily on fruits, including dogwood, sumac, blackberries, grapes, and plums. During periods of increased frugivory, robins may need to consume quantities of fruit daily in excess of their body weight to meet their metabolic needs. Free-living adult birds in summer, feeding on a mix of invertebrates and fruit, have been estimated to consume 0.75 kg fresh weight per kg body weight each day (Hazelton et al. 1984); adult robins subsisting on a strictly fruit diet consume as much as 1.5 kg fresh weight per kg body weight per day (Skorupa and Hothem 1985).

Eggs and young robins are often eaten by squirrels, snakes, and birds including grackles, crows, and common ravens. Predators of adult robins include hawks, cats, and larger snakes. Robins may mob small predators, produce warning calls when predators are near and may forage in loose flocks as protection against predators (UMMZ 2005).

Nesting areas tend to have higher densities of vegetation than foraging areas. Nests are built out of mud and dried vegetation in trees on horizontal limbs or at the joining of stem and trunk. A study in Wisconsin found that most preferred nesting sites were approximately 2 m above ground (Young 1955). First clutches generally contain three or four eggs; subsequent clutches tend to contain fewer eggs (USEPA 1993). Nestling and fledgling robins are primarily fed invertebrates. After reaching independence, juveniles often form foraging flocks in areas of high food availability.

5.4 MALLARD

The mallard is a dabbling duck that feeds on aquatic plants, seeds, and aquatic invertebrates. Mallards are found throughout North America, breeding in the northern U.S. and Canada, migrating into the central and southern U.S. during the winter. Mallards are seasonal breeding residents in the Cass Lake region (Nelson et al. 2004). Mallard ducks are relatively accepting of human disturbance. As a result, while several other waterfowl species' distributions have declined in the U.S., mallard distribution has expanded over the past century as they have taken advantage of suburban and urban edge habitats.

Mallard adults average 58 cm in length and 1.1 kg in weight–average of adult males and females from a North American Survey (USEPA 1993). The average mallard lifespan is 7–9 years, with survival in the first two years often less than 50 percent (PGC 2005).

In fall and winter, mallards feed primarily on seeds of plants, including wild rice, grass seeds, and agricultural grains, along with some shoots and tubers. In spring and throughout the summer breeding season, females shift from the herbivorous winter diet to one of mainly invertebrates. Laying females consume a higher proportion of invertebrates on the breeding grounds than males or non-laying females (USEPA 1993). Ducklings consume aquatic invertebrates almost exclusively (USEPA 1993). Invertebrates consumed include snails, insects, crustaceans, and worms. In wintering areas, average water depths of 20 to 40 cm have been suggested to be ideal for mallard foraging (Allen 1987).

Mallard eggs are eaten by several predators including crows, snakes, and raccoons. Until they can fly, at about 2 months, ducklings are vulnerable to a variety of predators including foxes, raccoons, snapping turtles, and large fish. As adults, one of the mallard's greatest predators is humans, who take mallards in the millions per year by hunting (UMMZ 2005).

Mallard breeding territories combine shallow aquatic habitats for foraging and thickly vegetated sites for nesting. For nesting, mallards prefer dense, grassy vegetation at least 50 cm

high and within 2 km of water; nests are often placed within 100 m of water. Adequate vegetative cover is important to protect nest sites from predators including foxes, skunks and crows. First clutches are usually hatched by late April to May in the northern U.S (USEPA 1993), with an incubation period of 26–28 days (UMMZ 2005); mallards may re-nest after the first clutch has fledged and produce a second clutch, which is usually smaller in size than the first (USEPA 1993).

Mallard densities during the breeding season are positively correlated with availability of terrestrial cover for nesting and with availability of ponds and wetlands that provide food. The size of breeding territories is dependent on the type and distribution of available aquatic habitats. An average home range for both sexes of 5.8 km^2, based on a study of wetland and river habitat in Minnesota (USEPA 1993), was chosen for this assessment.

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APPENDIX E4

METHODS FOR ESTIMATING TISSUE CONCENTRATIONS

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Appendix E4
Methods for Estimating Tissue Concentrations

Prepared for
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Prepared by

integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80021

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BCF	bioconcentration factor
BEF	bioconcentration equivalency factor
BSAF	biota-sediment accumulation factor
COPEC	chemicals of potential ecological concern
DQO	data quality objective
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentrations
ERA	ecological risk assessment
HPAH	high molecular weight polycyclic aromatic hydrocarbon
LPAH	low molecular weight polycyclic aromatic hydrocarbon
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCP	pentachlorophenol
PSDC	porewater-surface water disequilibrium coefficient
RAWP	risk assessment work plan
RME	reasonable maximum exposure
TEQ	toxic equivalent
TPAH	total polycyclic aromatic hydrocarbon
USACE	U.S. Army Corps of Engineers

1 INTRODUCTION

This appendix describes methods and provides algorithms that are used to predict concentrations of chemicals of potential ecological concern (COPECs) in aquatic plant, earthworms, and fish tissue for the purposes of exposure assessment in the ERA. Because the data describing environmental chemistry at the Site are extensive, the approach to modeling COPECs in tissue makes use of these data to the greatest extent possible. The approach emphasizes site-specific data and provides specific equations, biota sediment accumulation factors (BSAFs), and bioconcentration factors (BCFs) used.

The general technical approach for estimating tissue concentrations was initially documented in a technical memorandum (Integral 2005b) submitted to EPA on June 30, 2005. Integral (2005b) described an approach for estimating tissue concentrations needed for modeling exposure to fish, birds, and mammals that emphasized site-specific data and provided specific equations, bioaccumulation factors (BAFs), BSAFs and BCFs to be used. This appendix, which provides the final set of equations and assumptions used to predict COPEC concentrations in tissue, documents how the approach has been refined since Integral (2005b) was published, provides clarifications to Integral (2005b), and incorporates responses to EPA comments on Integral (2005b) and Integral (2005a). Most of the equations and factors provided in this appendix are the same as those provided by Integral (2005b); the general technical approach is unchanged. Any additions or corrections to specific equations provided in Integral (2005b) are clearly noted. Several equations provided by Integral (2005b) were not used and are not reported here; several additional COPECs identified in the final screening process are addressed. Methodological details are included here for completeness.

2 METHODS FOR PREDICTING COPEC CONCENTRATIONS IN TISSUE

The final list of tissue types and COPECs for which predictions in tissue are necessary is provided in Table E4-1. The rationale for selecting a tissue type and COPEC for modeling is described in Section 5.3 of this report. The following media required no modeling of tissue concentrations (see Section 5.3):

- Terrestrial plant tissue including grasses and fruits
- Benthic invertebrates (*Lumbriculus* and *Corbicula*)
- Crayfish.

For some tissue types, exposure point concentrations (EPCs) for exposure modeling were calculated using site-specific sampling results for some COPECs and modeled values for others. Those tissue types for which modeling was applied include:

- Wild rice (using site-specific BSAFs)
- Fish in Fox Creek (using literature based BSAFs and BCFs)
- Terrestrial invertebrates (earthworms using Site-specific regression models)

This section describes methods, algorithms, and assumptions used in each model that was applied to estimate COPECs in these tissue types.

2.1 WILD RICE

Available data for metals in wild rice tissue and co-located sediments were used to calculate site-specific biota-sediment accumulation factors (BSAFs) for plants, as:

$$BSAF = C_T / C_s$$

Where:

BSAF = biota-sediment accumulation factor (kg sediment dry weight/kg tissue dry weight)
C_T = concentration in plant tissue (mg/kg dry weight)
C_S = metal concentration in sediment (mg/kg dry weight).

Non-detects for either plant tissue or sediments were set to one half the detection limit. These BSAFs were used to predict tissue concentrations of metals in wild rice plants that were used in exposure calculations for higher trophic levels. The calculated BSAFs are in Table E4-2.

Table E4-1. Summary of Tissue Types for Which Modeling of COPEC Concentrations Was Required

Medium	Area A		Area B			
			Terrestrial		Aquatic	
	Predicted	Site-specific	Predicted	Site-specific	Predicted	Site-specific
Grass (terrestrial plants)	None	All COPECs	None	All COPECs	NA	NA
Earthworms[a]	Antimony, Cadmium[b], Lead, Dioxins/Furans,[c] HPAHs	Selenium, Vanadium, Zinc,[b] PCP, LPAH	Antimony, Cadmium[d], Lead, Dioxins/Furans[c], HPAHs	Copper,[d] Mercury,[d] Zinc, BHC,[d] PCP, LPAH	NA	NA
Fruit (sumac berries)	None	All COPECs	None	All COPECs	NA	NA
Fish: Central Mudminnow, Yellow Perch and White Sucker[e]	NA	NA	NA	NA	All metals except molybdenum PCBs[f], DDTs and isomers[g], and Dioxins/Furans[g]	Molybdenum, PAHs
Corbicula	NA	NA	NA	NA	None	All COPECs
Lumbriculus	NA	NA	NA	NA	None	All COPECs
Wild Rice[h]	NA	NA	NA	NA	All metals except beryllium	Beryllium, All organics

Notes:
NA = not applicable
BHC = benzene hexachloride
COPEC = chemical of potential ecological concern
HPAH = high molecular weight polycyclic aromatic hydrocarbon
LPAH = low molecular weight polycyclic aromatic hydrocarbon
PCB = polychlorinated biphenyl
PCP = pentachlorophenol
TPAH = total polycyclic aromatic hydrocarbon

[a] Earthworm was the only tissue for which some chemicals were estimated using Site-specific regression models.
[b]COPEC in soils for the forested wetland only in Area A.
[c]Modeling only for congeners 1,2,3,7,8-PeCDD; 1,2,3,4,7,8-HxCDD; 1,2,3,4,6,7,8-HpCDD; and 1,2,3,4,6,7,8-HpCDF Remaining congeners are from site-specific data.
[d]COPEC for the city dump only.
[e] Fish tissue concentrations were estimated for fish in Fox Creek only, using literature-derived BCFs and BSAFs
[f]PCBs and all metals except antimony, barium, lead, and thallium are aquatic COPECs only for Fox Creek and are modeled only for Fox Creek fish tissue.
[g]DDT and metabolites, and dioxins/furans in fish tissue are only modeled for Fox Creek. For Pike Bay, the channel, and overall aquatic areas, site-specific white sucker data are used to estimate EPCs for these chemicals
[h] Wild rice tissue concentrations were estimated using Site-specific BSAFs.

Table E4-2 Sediment-Plant BSAF for Metals in Wild Rice

COPEC	N	BSAF[a]
Antimony	3	0.05
Barium	3	0.06
Cadmium	3	0.015
Copper	3	0.08
Lead	3	0.003
Molybdenum	3	0.22
Mercury	3	0.41
Silver	3	0.036
Selenium	3	0.04
Thallium	3	0.04
Zinc	3	0.14

[a] These values are revised relative to earlier drafts due to inclusion of all data, with non-detects set to 1/2DL. This change is in response to EPA comments of April 6, 2006.

These site-specific BSAFs were used to estimate concentrations in plant tissue directly from sediment concentrations, as follows:

$$C_T = BSAF * C_s$$

The mean and reasonable maximum exposure (RME) for C_T (mg/kg, dry weight) were calculated using the mean and RME for sediment from the area of interest.

There are no data for beryllium in wild rice tissue samples. Therefore, EPCs for this COPEC in plants is represented in exposure models by data for cattail root (the arithmetic mean and RME were calculated treating non-detects according to the procedures defined in Section 2.2.2. of the main risk assessment report.

2.2 FISH

Tissue concentrations of both metals and organic COPECs were required for all aquatic habitats at the Site. To evaluate exposures and risks to fish in Cass Lake and Pike Bay, site-specific tissue concentrations of COPECs from fish samples collected from Cass Lake and Pike Bay (Section 2 of the risk assessment report) were used. To evaluate exposures to consumers of fish in Fox Creek and to evaluate risks to fish in Fox Creek, it was necessary to estimate tissue concentrations of metals (beryllium, cadmium, copper, lead, mercury, silver, selenium and zinc) and organic compounds (total PCBs, dioxins/furans, DDT and metabolites). Metals concentrations in fish were estimated using BCFs used by EPA in deriving water quality criteria

and an estimated concentration in porewater, as described in detail in Section 2.2.1 below. Organic COPECs in fish tissue, except for polycyclic aromatic hydrocarbons (PAHs), were estimated using BSAFs obtained from the literature.

PAH compounds were not estimated in tissue of fish. BSAFs for PAHs were not found in the literature, and PAHs are expected to be rapidly metabolized by fish and not accumulated. Exposures to PAHs by consumers of fish are based on site-specific tissue concentrations because there are no reliable methods to predict PAHs in fish tissue. Exposure models for birds and mammals use PAH data for sucker only. Exposures of fish to PAHs are evaluated by considering concentrations in food of fish; risks to fish are interpreted by comparing the PAH concentrations in fish food to PAH concentrations in food that are known to have no effects on fish.

To evaluate uncertainties with the approach to modeling COPECs in fish tissue, COPEC concentrations were estimated in fish tissue for Pike Bay, and were compared with measured concentrations in fish tissue from Pike Bay. The discussion of uncertainty is provided in Section 5.5.

2.2.1 Metals

Integral (2005b) proposed that trace metals would be predicted in fish tissue by first estimating the metal concentration in sediment porewater (using the sediment-water partition coefficient, Table E4-3) from metals in sediment, and then applying a BCF to predict tissue metals using the estimated porewater concentration in sediment. The concentration in porewater was estimated from the sediment-water partition coefficient as follows:

$$C_{PW} = C_S/K_d$$

Where:

C_{PW} = the concentration in porewater (mg/L_{PW})
C_S = the concentration in sediment (mg/kg dry weight)
K_d = sediment-water partitioning coefficient (L_{PW}/kg dry weight).

The following expression was used to estimate trace metal concentrations in fish tissues from sediment concentrations:

$$C_T = BCF \times C_S/K_d$$

Where:

C_T = concentration in tissue (mg/kg wet weight)
BCF = bioconcentration factor (L/kg wet weight)

BCFs and sediment-water K_d values to predict metals concentrations in fish tissue are presented in Table E4-3.

Table E4-3. Bioconcentration Factors, Sediment-Water Partition Coefficients, and Porewater-Surface Water Disequilibrium Coefficients for Inorganic COPECS at the St Regis Site

COPEC[a]	BCF[b]	K_d[c]	PSDC
Beryllium	62	790	0.056
Cadmium	907	75	0.00064
Copper	710	35	0.0032
Lead	0.09	900	0.041
Mercury	11,168	52	0.005
Selenium	126	5	0.000285
Silver	87.7	8.3	0.000003
Zinc	2,059	62	0.00027

[a]No BCF was available for molybdenum. Site specific molybdenum concentrations in sucker from Pike Bay were used to represent concentrations of molybdenum in fish of Fox Creek.
[b]Source: USEPA (1999)
[c]Source: Risk Assessment Information System (http://risk.lsd.ornl.gov/index.shtml)

This method includes many conservative assumptions. As a check on these predictions, tissue concentrations of metals were also predicted using measured concentrations of the metals in surface water and the BCF, as described by Integral (2005b). Results of both methods were compared to concentrations measured in fish from the Site. This comparison was performed for Pike Bay, because sediment, water, and fish were all available from this location. Concentrations in fish tissue predicted using both methods are presented in Table E4-4, and compared to measured concentrations of metals in fish tissue from Pike Bay.

Table E4-4. Measured vs. Predicted Concentrations of Metals (mg/kg ww) in Pike Bay Fish

	Measured Concentrations in Pike Bay "Sucker"		Concentration in Fish Predicted Directly from Estimated Sediment Porewater		Concentrations in Fish Predicted Directly from Measured Surface Water		Concentrations in Fish Predicted Using Estimated Porewater and the PSDC	
COPEC[a]	Mean	Max	Mean	Max	Mean	Max	Mean	95%UCL
Beryllium	na	0 0050 U	0 00480	**0.0118**	0.000620	NA	*0.000940*	*0 001283*
Cadmium	na	0.013	0.270	**0.665**	0 0907	NA	0 0009	*0.0015*
Copper	1 7	2 4	**13.5**	**26.4**	3 44	NA	*0.14*	*0 23*
Lead	0.1	0.2	*0.000110*	*0 000300*	*0.0000775*	NA	*0 0002697*	*0 000463*
Mercury	Na	0.03	2.97	**7.52**	0.826	NA	0 079	**0.142**
Selenium	0 23	0.30	**5.2**	**17.50**	*0.038*	NA	*0 0002*	*0 0002*
Silver	Na	0 0050	0.680	**2.54**	0 000439	NA	0 000002	*0 000004*
Zinc	Na	13.005	139	**315**	15 3	NA	0 2	0 4

Notes:
Bold values indicate overprediction of measured concentration, *italics* indicate underprediction

Means calculated using 1/2 DL.

Undetected values which were > the largest detected concentration for each analyte were excluded.

na = Not applicable. Only one sample collected and analyzed, or exclusion of undetected values > highest detected value resulted in only one value.

[a] Molybdenum concentrations in fish could not be estimated because no BCF was found. Empirical values of molybdenum in fish tissue from the Site was used in bird and mammal exposure calculations for all exposure units.

Table E4-4 illustrates that estimated porewater concentrations multiplied by BCFs substantially overpredicts most metals in fish tissue; lead is underpredicted by all available methods. As a result of this comparison, a site-specific correction factor, the porewater-surface water disequilibrium coefficient (PSDC), was calculated to account for the dilution of porewater concentrations in metals by surface waters. The site-specific PSCD was calculated using samples of water and sediment collected in 2001 in close proximity to each other, as follows:

1. Surface water stations from 2001 for Fox Creek were identified
2. Sediment stations from the same year nearest these three stations were identified
3. The K_d value in Table E4-3 was used to predict porewater concentration at each sediment sample location from concentration of each metal in sediment
4. The measured concentration of the dissolved metal in surface water (mg /L_{SW}) was divided by the predicted concentration of the metal in porewater (mg /L_{PW}) to calculate the ratio of surface water concentration to porewater concentration. This ratio is the PSDC (L_{SW}/L_{PW}).
5. Using the K_d and the overall average and RME for each metal in sediment for Fox Creek, the average and reasonable maximum metal concentration in porewater in Fox Creek were predicted.
6. Predicted porewater concentrations were multiplied by the PSDC to estimate surface water concentrations.
7. The estimated surface water concentrations were used to estimate the fish tissue concentration, as

$$C_T = BCF \times C_{SW}$$

Where:

C_{SW} = estimated dissolved metal concentration in surface water (mg/L_{SW}).

Values for the estimated PSDC are listed in Table E4-4. This approach to estimating tissue concentrations associated with inorganic chemicals in sediments mitigates some of the conservatism of predicting fish tissue directly from estimated concentrations of metals in porewater, while recognizing that sediment metals in Fox Creek are higher than elsewhere, so therefore fish tissue metals may be higher. The advantage, that the method ties concentrations of inorganic COPECs in fish directly to sediments (the chemistry of which may have been influenced by historical or ongoing chemical releases) rather than ambient surface water, is retained.

2.2.2 Organic Compounds

Organic COPECs that were estimated in fish tissues include PCBs, DDT and metabolites, and dioxins and furans (Table E4-1) following EPA's methods for estimating concentrations of these COPECs in fish tissue from sediment concentrations using BSAFs from the literature (USEPA 1993; 1995). Concentrations of PCBs were estimated in fish tissue for Fox Creek only, DDT and metabolites were estimated for fish in Fox Creek and the channel. Dioxins and furans were estimated to calculate exposures to piscivorous receptors in Fox Creek. Site-specific fish tissue data for all COPECs (except DDTs) were used to calculate EPCs for piscivorous wildlife foraging in Pike Bay and the channel.

The BSAF is calculated as the ratio of the lipid-normalized concentration of a COPEC in the tissue of an aquatic organism to the organic carbon-normalized concentration in surface sediment (in kg of lipid/ kg of organic carbon). It is assumed that the ratio does not change substantially over time or across the range of sediment concentrations (i.e., that lipid-normalized concentration in tissue increases in a linear fashion with increases organic carbon-normalized concentrations in sediment), both the organism and its food are exposed within the same environment, and the surface sediment on which the value is based is representative of average surface sediment in the vicinity of the organism. It is also assumes that the concentrations of the COPEC in sediment, water, and foods of the modeled organism and the modeled organism itself are in equilibrium.

Several sources of BSAFs were reviewed for relevant and appropriate values. The following hierarchy was used to obtain literature reporting BSAFs for dioxins, furans, PCBs, and DDT and DDE:

- The U.S. Army Corps of Engineers (USACE) BSAF database was reviewed for relevant values for dioxins, furans, PCBs, and DDT and DDE (USACE 2005). This database provides BSAFs obtained primarily from peer-reviewed articles for several organic chemicals and aquatic species.
- TCDD bioconcentration equivalency factors (BEFs) from the Great Lakes Water Quality Initiative (USEPA 1995) were used to generate BSAFs for dioxin and furan congeners where a BSAF for only 2,3,7,8-TCDD was available, as described below. This approach was used when no value for a given dioxin/furan congener was available in the USACE database.
- If values were not attainable through sources 1 or 2, Lake Ontario trout BSAFs were used (USEPA 1995). This data set is reported in USEPA (1995), and is based on a sampling of fish and sediments in 1978 for EPA's Lake Ontario TCDD bioaccumulation study for the purpose of determining BSAFs.

- If databases or summary literature were used, the original article describing the derivation of the BSAF was reviewed for relevance and appropriateness of the studies. The following criteria were applied for selection of studies to review:
  - The data were for freshwater ecosystems
  - The data were organic carbon- and lipid-normalized
  - Whenever possible, BSAFs based on whole-body data were used
  - Sediments were sampled at the surface (top 5 cm) in depositional environments
  - The size of the fish or the age of fish was comparable to sizes of fish that could be eaten by receptors at the St. Regis Site. If length- or age-specific data were available, BSAFs for fish 5–20 cm long were preferentially used because this size range overlaps with the size range of fish likely to be consumed by the aquatic receptors.
- BSAFs were obtained for the same fish species that are known to inhabit Fox Creek and that would be considered prey for piscivorous receptors whenever possible. These species include the following:
  - Central mud-minnow (Umbra limi), evaluated as prey because it has been observed in Fox Creek
  - Yellow perch (Perca flavescens)
  - White sucker (Catostomus commersoni).

Although BSAFs for whitefish and walleye were available, these fish are not expected in Fox Creek. No modeling was conducted for concentrations of COPECs in the tissue of whitefish and walleye for the ERA.

If BSAF values were not available for a given species, a species with a similar trophic ecology for which a BSAF value was available was selected as representative of the species of interest. The bottom-feeding black bullhead (*Ameiurus melas*) was substituted for white sucker if values for the former but not the latter could be found. For central mud-minnow, insectivorous mid- and bottom-feeding small fish including the bluntnose guppy, *Poecilia reticulata*, the bluntnose minnow, *Pimephales promelas*, or sculpin, *Cottus* spp. were used. For yellow perch, lake trout, *Salvelinus namaycush*–most values based on 2-year old lake trout sampled in Lake Michigan by Burkhard et al. (2004), were used because both species are mid-water feeders on invertebrates and small fish.

For some PCB congeners, BSAFs were not available for all three species. If two BSAFs were available, the BSAF of the species most similar to the species lacking a BSAF was used. Only one BSAF was found for DDD (white sucker) and for PCB congeners 156, 157, 169, and 189 (lake trout), so in those cases, the single available BSAF was used for all three species. For two PCB

congeners (123 and 157), no BSAF was available; in these two cases, the highest available BSAF from the list of detected congeners was used as a conservative estimate.

If BSAFs were not available for dioxin congeners for a given species, then the BSAF was estimated by modifying the BSAF for 2,3,7,8-TCDD by the BEF, as reported in Great Lakes Water Quality Initiative (USEPA 1995). The TCDD BEF is the ratio between each PCDD and PCDF congener's BSAF to that of TCDD, such that:

$$BSAF_{conj} = BEF_{conj} * BSAF_{TCDD}$$

The resulting list of BSAFs is provided in Table E4-5 for dioxins and furans and in Table E4-6 for PCBs and DDT and metabolites.

Table E4-5. BSAFs for Dioxins and Furans

	Central Mudminnow (*Umbra limi*)		Yellow Perch (*Perca flavescens*)		White Sucker (*Catostomus commersoni*)	
	BSAF	Reference	BSAF	Reference	BSAF	Reference
Dioxins						
2,3,7,8-TCDD	0.155	Loonen et al. (1994) [a]	0 229	Burkhard et al. (2004) [b]	0 063	Schell et al. (1993) [c]
1,2,3,7,8-PCDD	0.08	Loonen et al. (1994) [a]	0.198	Burkhard et al. (2004) [b]	0 05796	USEPA (1995) [d]
1,2,3,4,7,8-HxCDD	0.024	Loonen et al. (1994) [a]	0.00982	Burkhard et al (2004) [b]	0.01953	USEPA (1995) [d]
1,2,3,6,7,8-HxCDD	0.024	Loonen et al. (1994) [a]	0.0368	Burkhard et al. (2004) [b]	0.00756	USEPA (1995) [d]
1,2,3,7,8,9-HxCDD	0 008	Loonen et al. (1994) [a]	0.00399	Burkhard et al (2004) [b]	0.00882	USEPA (1995) [d]
1,2,3,4,6,7,8-HpCDD	0.014	Loonen et al (1994) [a]	0.00137	Burkhard et al (2004) [b]	0.00321	USEPA (1995) [d]
OCDD	0 003	Loonen et al. (1994) [a]	0 002748	USEPA (1995) [b]	0 00075	USEPA (1995) [d]
Furans						
2,3,7,8-TCDF	0.014	Loonen et al (1994) [a]	0 209	Burkhard et al. (2004) [b]	0 0504	USEPA (1995) [d]
2,3,4,7,8-PCDF	0.088	Loonen et al. (1994) [a]	0.212	Burkhard et al (2004) [b]	0 1008	USEPA (1995) [d]
1,2,3,7,8-PCDF	0 002	Loonen et al. (1994) [a]	0 0715	Burkhard et al. (2004) [b]	0 01386	USEPA (1995) [d]
1,2,3,4,7,8-HxCDF	0.031	Loonen et al (1994) [a]	0.017404	USEPA (1995) [d]	0 00478	USEPA (1995) [d]
1,2,3,6,7,8-HxCDF	0 021	Loonen et al. (1994) [a]	0.0263	Burkhard et al (2004) [b]	0.01197	USEPA (1995) [d]
1,2,3,7,8,9-HxCDF	0.09765	USEPA (1995) [d]	0 114	Burkhard et al. (2004) [b]	0 03969	USEPA (1995) [d]
2,3,4,6,7,8-HxCDF	0.10385	USEPA (1995) [d]	0 0173	Burkhard et al (2004) [b]	0 04221	USEPA (1995) [d]
1,2,3,4,6,7,8-HpCDF	0.016	Loonen et al. (1994) [a]	0.00367	Burkhard et al (2004) [b]	0.00069	USEPA (1995) [d]
1,2,3,4,7,8,9-HpCDF	0 06045	USEPA (1995) [d]	0 00709	Burkhard et al. (2004) [b]	0 02457	USEPA (1995) [d]
OCDF	0.00248	USEPA (1995) [d]	0 00048	USEPA (1995) [d]	0 00100	USEPA (1995) [d]

Notes:

ND = no data

[a] *Poecilia reticulata* used as substitute

[b] *Salvelinus namaycush* used as substitute

[c] *Ameiurus melas* used as substitute

[d] congener BSAF = congener BEF x TCDD BSAF; BEF from USEPA (1995)

Table E4-6. BSAFs for PCBs and DDT and Metabolites

	Central Mudminnow *(Umbra limi)*		Yellow Perch *(Perca flavescens)*		White Sucker (Catostomus commersoni)	
	BSAF	Reference	BSAF	Reference	BSAF	Reference
PCBs						
PCB-105	ND		3.77	Cook et al. (2003) [a]	ND	
PCB-114	ND		4.96	Burkhard et al. (2004) [a]	ND	
PCB-118	ND		4.36	Burkhard et al. (2004) [a]	ND	
PCB-123	ND		ND		ND	
PCB-126	ND		6.83	Cook et al. (2003) [a]	ND	
PCB-156	ND		ND		3	Lutz et al. (1994) [b]
PCB-157	ND		ND		ND	
PCB-167	ND		6.22	Burkhard et al. (2004) [a]	ND	
PCB-169	ND		12 9	Cook et al. (2003) [a]	ND	
PCB-189	ND		0.71	USEPA (1995) [a]	ND	
PCB-77	ND		0.413	Cook et al. (2003) [a]	1.73	Lutz et al (1994) [b]
PCB-81	ND		1.47	Cook et al. (2003) [a]	ND	
Total PCBs	0.31	Ankley et al. (1992) [c]	4.94	Clarke et al. (1988); [d] MacDonald et al. (1993)	1.91	Ankley et al. (1992) [b]
Pesticides						
DDT	2.15	Wong et al. (2001) [e]	1.67	USEPA (1995) [a]	1.047	Wong et al. (2001)
DDE	5.23	Wong et al. (2001) [e]	7.7	USEPA (1995) [a]	10.385	Wong et al. (2001)
DDD	ND		ND		2.8	Wong et al. (2001) [f]

Notes:

ND = no data

[a] *Salvelinus namaycush* used as substitute

[b] *Ameiurus melas* used as substitute

[c] *Pimephales promelas* used as substitute

[d] Grand mean of three reported values for *P. flavescens*

[e] *Cottus* sp. used as substitute

[f] o'p'DDD value for white sucker

2.3 TERRESTRIAL INVERTEBRATES

Statistical correlations between chemical concentrations in soil and earthworm tissue were modeled using regression analysis of site-specific data. Correlations between soil and earthworm tissue for COPECs with detected values at all or most paired soil and earthworm tissue stations were evaluated using least squares simple linear regression (data for the regressions used are provided in Attachment A to this Appendix). Three COPECs had non-detects in either soil or earthworm tissue: LPAHs and PCP had non-detects in the earthworm tissue, and the dioxin congener 1,2,3,7,8-PCDD had one non-detect in soil. For these COPECs, correlations between soil and earthworm tissue were evaluated using a combination of censored regression and graphical evaluation. No COPECs were undetected in both earthworm tissue and soil among the synoptic data.

For both the least squares linear and censored regressions, a minimum sample size of four paired data points was required to establish a relationship between soil and earthworm tissue concentrations. Because organic carbon data were not available for most soil samples evaluated in these regressions, concentrations of organic chemicals were not normalized to organic carbon, and tissue concentrations were not normalized to lipid content.

2.3.1 Regression on Samples with No Non-Detects

For linear regression of paired soil-tissue stations for COPECs with no non-detects, COPEC concentrations in soil and tissue were evaluated both untransformed and $\log_{10}$-transformed to determine whether statistical models could be improved by transforming the data. Soil and tissue values were both multiplied by 1,000 prior to $\log_{10}$-transformation. The model with the highest R^2 value (i.e., amount of variance explained by the regression model) and lowest p value (i.e., percent significance level for the regression) was chosen to predict tissue concentrations.[1]

2.3.2 Regression on Samples with Non-Detects

The statistical relationship between soil and earthworm tissue for the COPECs with non-detects in tissue (LPAHs and PCP) was evaluated using parametric and nonparametric methods outlined in Helsel (2005). Parametric censored regression was conducted using maximum likelihood estimation (MLE) to estimate the best fit-line for the data, incorporating the detection limit of non-detect data. Non-parametric censored regression was performed using a Theil-Sen

[1] R^2 expresses the percentage of variation in the y variable that can be explained by the x variable. For example, if the R^2 is 0.81, then 81 percent of the variation in y is explained by x. Larger values of R^2 indicate a stronger relationship between the variables. The p-value represents the probability of accepting the observed relationship as statistically significant when it is actually not statistically significant.

line to estimate slope of the regression line. Theil-Sen lines estimate slope as the median of all slopes between paired data (Helsel 2005).

The dioxin congener 1,2,3,7,8-PCDD had one non-detect in the soil data. The detection limit of this non-detect was evaluated graphically relative to the range of the detected soil data and was found to lie within the range of the detected data. This soil-tissue pair was therefore excluded and a simple linear regression was conducted on the remaining data (Section 2.3.5). Results of these analyses are discussed in Section 2.3.4, below.

2.3.3 Metals

Significant correlations for antimony, cadmium and lead were derived for soil - earthworm tissue relationships. Correlations were absent or weak and non-significant for copper, mercury, selenium, vanadium and zinc. Log-transformation of the data did not substantially improve statistical correlations for any of these COPECs. For those metals for which statistically significant relationships with soil were not found, EPCs in the ERA were estimated from empirical tissue concentration data as described in the RAWP and in Section 5.3 of the risk assessment report.

Equations relating concentrations of antimony, cadmium, and lead in soil to concentration in earthworm tissue are in Table E4-7.

Table E4-7. Linear Regression Models for Predicting Concentrations of Antimony, Cadmium, and Lead in Earthworm Tissue as a Function of Soil Concentration

COPEC	Regression Equation	R^2, n, p
Antimony	$Ct = 0.1097C_S + 0.0211$	R^2=0.98, n=5, p<0.01
Cadmium	$Ct = 3.3298Cs + 4.1192$	R^2=0.56, n=6, p=0.09
Lead[a]	$Ct = 0.215Cs + 5.5549$	R^2=0.98, n=5, p<0.01

Notes: C_S – concentration in soil (mg/kg dry weight)
C_T – concentration in earthworm tissue (mg/kg dry weight)

[a] This equation was not reported by Integral (2005b).

2.3.4 PAHs and Pentachlorophenol

Statistical correlations between concentrations in soil and in earthworm tissue were evaluated for PAH compounds (as total PAH [TPAH], low molecular weight PAH [LPAH], and high molecular weight PAH [HPAH]) and for pentachlorophenol (PCP). Integral (2005b) reported equations for individual PAH compounds. Because doses to bird and mammal consumers of terrestrial invertebrates are evaluated based on summed PAH concentrations for LPAH and

HPAH, correlations between earthworm tissue and soils for these PAH groups were evaluated for the ecological risk assessment, and are discussed below.

Simple linear regression was conducted on both untransformed and $\log_{10}$-transformed datasets for both HPAH and TPAH. (Only the HPAH model was actually used in the risk assessment; the TPAH model is included here as additional evidence in support of the relationship described for HPAH.) The equations with the untransformed data had higher R^2 values than equations with the log transformed data, so the equations generated using untransformed data were selected. Regression models that were applied to predict concentrations of HPAH and TPAH compounds in earthworm tissue as a function of soil concentration are summarized in Table E4-8. The data used to derive the regression equations for HPAH and TPAH all included one station with concentrations in soil and tissue substantially higher than the others (ECO07). The resulting regression model for each is strongly influenced by the station with these high values. The uncertainty associated with these equations is discussed in Section 5.5.

Regression analysis suitable for censored data was conducted using two additional methods, described in Section 2.3.2, for both PCP and LPAH because non-detects were present in the earthworm tissue for both chemicals. No difference was found between the statistical results of the two methods for both LPAHs and PCP. Integral (2005b) reported statistically significant correlations in soil and earthworm tissue for both of these COPECs. However, when the non-detect concentration was included and censored regression conducted, neither regression was statistically significant ($p < 0.01$) (Table E4-9). As a result, EPCs were calculated using available empirical data, with treatment of non-detects according to procedures described in Section 2.2.2.

Table E4-8. Linear Regression Models for Predicting Concentrations of HPAH and TPAH in Earthworm Tissue as a Function of Soil Concentration.

COPEC	Regression Equation	R^2, n, *p*
HPAH	C_T=0.0333Cs + 0.3012	R^2= 0.99, n=6, *p* <0.01
TPAH	C_T=0.031Cs + 0.3743	R^2= 0.99, n=6, *p* <0.01

Notes: C_S – concentration in soil (mg/kg dry weight)
C_T – concentration in earthworm tissue (mg/kg dry weight)

Table E4-9. P-values from Censored Regression for Predicting Concentrations of LPAH and PCP in Earthworm Tissue as a Function of Soil Concentration.

COPEC	Theil-Sen p-value	MLE p-value
PCP	0.85	0.72
LPAH	0.13	0.16

2.3.5 Dioxins and Furans

Because of differences in chemical properties among individual dioxin and furan congeners, statistical correlations between soil and earthworm tissue concentrations were evaluated for

individual congeners and for homolog groups (Table E4-10), consistent with EPA guidance (USEPA 1999). However, because only a subset of individual congeners was detected in both earthworms and soils, statistical regression models could not be developed in all cases. For congeners lacking an acceptable regression model, tissue EPCs (the mean and RME values) were estimated using site-specific concentrations of the congener measured in earthworms, with concentrations of undetected congeners assumed to equal one-half the detection limit. Measured or estimated concentrations of each congener multiplied by the appropriate TEF value, and adjusted congener concentrations were summed to calculate the TEQ.

The congener 1,2,3,7,8-PCDD was detected in the majority, but not all, of earthworm-soil tissue pairs, so a regression model was developed using the detected data only. To determine whether the omission of one sample would likely affect the predictive model, all detected and non-detected results for 1,2,3,7,8-PeCDD in soil used in the regression were evaluated graphically to determine if the range of detected soil concentrations is inclusive of the one non-detect at station ECO-09 (Figure E4-1). Figure E4-1 shows that the detected concentration at station ECO-10 is lower than the non-detect at ECO-09. Excluding this non detect from the regression model presented for 1,2,3,7,8-PeCDD (Table E4-10) is not expected to change the resulting model from one derived using only stations with detected values.

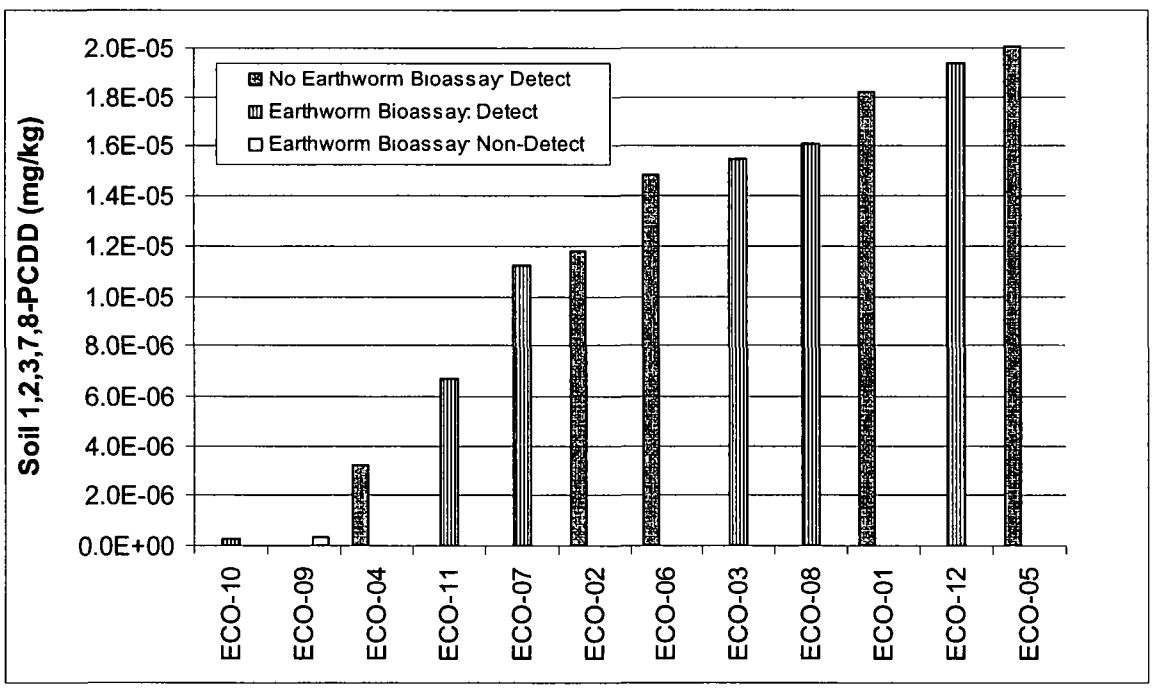

Figure E4-1. Detect and Non-Detect Concentrations of 1,2,3,7,8-PeCDD in Soil

Table E4-10. Statistically Significant Correlations between Soil and Earthworm Tissue Concentrations for Individual Dioxin and Furan Congeners.

Chemical	Regression Equation[a]	R^2, n, *p*
1,2,3,7,8-PeCDD[b]	Log C_T = 0 9339(Log C_S) - 0 8095	R^2 = 0 62, n=6 *p* = 0 06
1,2,3,6,7,8-HxCDD[c]	Log C_T = 0.7768(Log C_S) - 0.9192	R^2 = 0.62, n=7 *p* = 0 03
1,2,3,4,6,7,8-HpCDD	Log C_T = 0 6293(Log C_S) – 0.7518	R^2 = 0.59, n=7 *p* = 0.05
OCDD	Log C_T = 0.542(Log C_S) – 0 3799	R^2 = 0 54, n=7 *p* = 0.06
1,2,3,4,6,7,8-HpCDF	Log C_T = 0.7546(Log C_S) – 0.8785	R^2 = 0.70, n=7 *p* = 0.02
OCDF[b]	Log C_T = 0.5977(Log C_S) – 0.8572	R^2 = 0.51, n=7 *p* = 0.07

Notes· C_S – concentration in soil (mg/kg dry weight)
C_T – concentration in earthworm tissue (mg/kg dry weight)

[a] Soil and earthworm tissue dioxin concentrations were multiplied by 1,000 for ease of analysis and are therefore expressed as ug/kg prior to log-transformation; tissue estimates resulting from the regression must be divided by 1,000 to provide concentrations in mg/kg.
[b] The equation was reported incorrectly by Integral (2005b).
[c] This congener was incorrectly reported as not having a significant correlation by Integral (2005b).

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APPENDIX E5

EXPOSURE POINT CONCENTRATIONS FOR THE ECOLOGICAL RISK ASSESSMENT

CONTENTS

Table E5-1. Mean Concentrations of COPECs in Abiotic Exposure Media of Terrestrial Receptors

Area	Area A				Area B				Area A		Area B			
Medium	Soil				Soil				Water		Water			
Units	mg/kg dw				mg/kg dw				mg/L		mg/L			
Subarea	All		Forested Wetland		All		City Dump		All[a]		All[b]		City Dump[c]	
EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
Antimony	1.0E+00	1.1E+00	NA	NA	3.5E-01	5.5E-01	NA	NA	2.9E-04	8 4E-04	2 1E-04	5.0E-04	NA	NA
Cadmium	NA	NA	1.1E+00	1.7E+00	NA	NA	3.5E-01	6.8E-01	3.0E-04	1.1E-03	NA	NA	1 2E-04	2.5E-04
Copper	NA	NA	NA	NA	NA	NA	1.1E+01	1.9E+01	NA	NA	NA	NA	9.6E-03	1.7E-02
Lead	1.3E+01	1 8E+01	3.1E+01	5.0E+01	4.3E+01	8 6E+01	NA	NA	1.8E-03	5.7E-03	2 6E-03	4.5E-03	NA	NA
Mercury	NA	NA	NA	NA	NA	NA	7 9E-02	1.8E-01	NA	NA	NA	NA	ND	ND
Selenium	3.0E-01	4.3E-01	NA	NA	NA	NA	NA	NA	3.5E-04	5 0E-04	NA	NA	NA	NA
Vanadium	8.8E+00	9 4E+00	NA	NA	NA	NA	NA	NA	9.7E-04	2.0E-03	NA	NA	NA	NA
Zinc	NA	NA	1.5E+02	2.3E+02	6 2E+01	1.1E+02	NA	NA	5 7E-02	1 4E-01	1 4E-02	1 9E-02	NA	NA
tBHC	NA	NA	NA	NA	NA	NA	5.8E-04	1.6E-03	NA	NA	NA	NA	ND	ND
LPAH	6.2E-01	8.1E-01	NA	NA	1.5E+00	3.6E+00	NA	NA	4.2E-05	7.4E-05	8 9E-05	1.7E-04	NA	NA
HPAH	5 1E+00	8.5E+00	NA	NA	1.1E+01	2.3E+01	NA	NA	3.4E-05	4.8E-05	5.6E-05	1 3E-04	NA	NA
DDD	1 4E-03	1.7E-03	NA	NA	7.3E-04	2 0E-03	NA	NA	NA	NA	ND	ND	NA	NA
DDE	1 6E-03	2.3E-03	NA	NA	7.8E-03	2 2E-02	NA	NA	NA	NA	ND	ND	NA	NA
DDT	5.0E-03	1.1E-02	NA	NA	5.5E-03	1.3E-02	NA	NA	ND	ND	ND	ND	NA	NA
Total DDX	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PCP	1.2E+00	1.9E+00	NA	NA	7 4E-01	1.8E+00	NA	NA	4.8E-05	9 5E-05	2 8E-04	4.3E-04	NA	NA
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	6.1E-04	8.3E-04	4.2E-05	1.3E-04	2.5E-04	4.1E-04	NA	NA	ND	ND	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	3.6E-04	5.0E-04	2.2E-05	6.8E-05	1.3E-04	2 0E-04	NA	NA	ND	ND	ND	ND	ND	ND
tPCBs	NA	NA	NA	NA	NA	NA	6.3E-02	1 3E-01	NA	NA	NA	NA	ND	ND

Notes:

NA= not applicable, because analyte is not a COPEC for the given subarea

ND= no data available for COPEC

[a]Concentrations of COPECs in water in the Forested Wetland are used for estimating exposure to terrestrial receptors in Area A and in the Forested Wetland

[b]Concentrations of COPECs in water from Overall Aquatic Area B are used for estimating exposure to terrestrial receptors in Area B

[c]Concentrations of COPECs in water in Fox Creek are used for estimating exposure to terrestrial receptors in the City Dump

Table E5-2 Mean Concentrations of COPECs in Biotic Exposure Media of Terrestrial Receptors

Area	Area A		Area B		Area B		Area A		Area B		Area B		Area A	
Medium	Grasses		Grasses		Grasses		Earthworms		Earthworms		Earthworms		Grubs	
Units	mg/kg dw		mg/kg dw		mg/kg dw		mg/kg dw		mg/kg dw		mg/kg dw		mg/kg dw	
Subarea	All[a]		All		City Dump		All[b]		All		City Dump		All[c]	
EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
Antimony	1.7E-01	3.5E-01	2.9E-01	4 9E-01	NA	NA	NA[d]	NA[d]	NA[d]	NA[d]	NA	NA	1 3E-01	1.3E-01
Cadmium	NA	NA	NA	NA	1 0E-01	1.5E-01	NA[d]	NA[d]	NA	NA	NA[d]	NA[d]	3.3E-01	3.3E-01
Copper	NA	NA	NA	NA	6 8E+00	1 3E+01	NA	NA	NA	NA	2 6E+01	4 3E+01	NA	NA
Lead	2 0E-01	3.6E-01	3.5E-01	8.1E-01	NA	NA	NA[d]	NA[d]	NA[d]	NA[d]	NA	NA	7 3E+00	7.3E+00
Mercury	NA	NA	NA	NA	2.9E-02	5.2E-02	NA	NA	NA	NA	2 9E-01	4 7E-01	NA	NA
Selenium	7 5E-02	2 0E-01	NA	NA	NA	NA	3.1E+00	3.1E+00	NA	NA	NA	NA	2 0E-01	2.0E-01
Vanadium	1 4E-01	1.8E-01	NA	NA	NA	NA	9 5E+00	9.5E+00	NA	NA	NA	NA	4.0E+00	4.0E+00
Zinc	NA	NA	5.0E+01	1.2E+02	NA	NA	2 3E+02	2.3E+02	4.8E+02	1 1E+03	NA	NA	1 1E+02	1.1E+02
tBHC	NA	NA	NA	NA	ND	ND	NA	NA	NA	NA	1.3E-03	2 5E-03	ND	ND
LPAH	1 6E-02	3 6E-02	4 9E-03	1.3E-02	NA	NA	1 0E-01	1 0E-01	1.5E-01	3.9E-01	NA	NA	6.4E-02	6.4E-02
HPAH	2.6E-02	5.6E-02	9.0E-03	2.4E-02	NA	NA	NA[d]	NA[d]	NA[d]	NA[d]	NA	NA	1.2E+00	1.2E+00
DDD	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
DDE	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
DDT	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total DDX	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
PCP	6.1E-03	4.0E-02	1.8E-02	5.2E-02	NA	NA	2.3E+00	2.3E+00	9.2E+00	4.4E+01	NA	NA	1.6E-01	1.6E-01
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	3.0E-07	4 5E-07	1.3E-07	1 7E-07	NA	NA	8 3E-05	8.3E-05	1.7E-05	4.0E-05	NA	NA	1.0E-04	1.0E-04
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	1.6E-07	2 4E-07	9 7E-08	1 4E-07	NA	NA	1.1E-04	1.1E-04	1.3E-05	2.9E-05	NA	NA	8.4E-05	8.4E-05
tPCBs	NA	NA	NA	NA	ND	ND	NA	NA	NA	NA	1.3E-02	2 3E-02	NA	NA

Notes:

NA= not applicable, because analyte is not a COPEC for the given subarea or because empirical value not used (see footnote d)

ND= no data available for COPEC

[a]Concentrations of COPECs in grass in Area A are used for estimating exposure to terrestrial receptors in the Forested Wetland

[b]Concentrations of COPECs in earthworms in Area A are used for estimating exposure to terrestrial receptors in the Forested Wetland

[c]Concentrations of COPECs in grubs in Area A are also used for estimating exposure to terrestrial receptors in the Forested Wetland

[d]Modeled value, empirical data not used See Appendix E4

Table E5-3. Mean Concentrations of COPECs in abiotic Exposure Media of Aquatic Receptors (Area B only), Except Fish

Medium	Sediment								Water			
Units	mg/kg dw								mg/L			
Subarea	All		Fox Creek		Pike Bay		Channel		All		Fox Creek	
EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
Antimony	NA	NA	NA	NA	2 6E+00	7 9E+00	2 4E+00	3.6E+00	NA	NA	NA	NA
Barium	NA	NA	NA	NA	1 1E+01	1.3E+01	1.2E+02	1.4E+02	NA	NA	NA	NA
Beryllium	NA	NA	2.1E-01	2.9E-01	NA	NA	NA	NA	NA	NA	1 0E-04	2 0E-05
Cadmium	NA	NA	1.3E+00	2.1E+00	NA	NA	NA	NA	NA	NA	1.2E-04	2 5E-04
Copper	NA	NA	4 8E+01	8.0E+01	NA	NA	NA	NA	NA	NA	9.6E-03	1 7E-02
Lead	3.9E+01	6.1E+01	5.8E+01	9.9E+01	1 1E+00	1.7E+00	2 4E+01	3 1E+01	2.6E-03	4 5E-03	4 2E-03	8.4E-03
Mercury	NA	NA	1 1E+00	2.0E+00	NA	NA	NA	NA	NA	NA	NA	NA
Molybdenum[b]	NA	NA	1 1E+00	1.5E+00	NA	NA	3.9E-01	8.8E-01	NA	NA	6 1E-05	1.9E-04
Selenium	NA	NA	3 7E+00	4.9E+00	NA	NA	NA	NA	NA	NA	3.0E-04	6.0E-04
Silver	NA	NA	7.4E+00	1.3E+01	NA	NA	NA	NA	NA	NA	3.2E-04	8.5E-04
Thallium	NA	NA	NA	NA	NA	NA	3.1E-02	6.5E-02	NA	NA	NA	NA
Zinc	NA	NA	3.1E+02	5.4E+02	NA	NA	NA	NA	NA	NA	1.3E-02	4 8E-02
LPAH	1 2E+00	2.2E+00	1.3E+00	2 7E+00	1.2E-02	3.0E-02	1.5E+00	2.2E+00	8.9E-05	1.7E-04	8 7E-05	2.3E-04
HPAH	3.8E+00	5.9E+00	3.4E+00	6.0E+00	6.2E-02	1.9E-01	6.1E+00	1.1E+01	5.6E-05	1.3E-04	6.8E-05	2 2E-04
DDD	NA	NA	3.1E-03	5.4E-03	NA	NA	2.3E-03	9.7E-03	NA	NA	ND	ND
DDE	NA	NA	6.9E-03	1.9E-02	NA	NA	1 2E-03	4 9E-03	NA	NA	ND	ND
DDT	NA	NA	4.5E-03	1.0E-02	NA	NA	2.5E-04	1.1E-03	NA	NA	ND	ND
Total DDX	NA	NA	0 013437	0 031924	NA	NA	0.003859	0.01515	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	5.6E-07	9 5E-07	3.9E-06	9.5E-06	1.7E-07	2 9E-05	8.8E-07	1 5E-06	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	2 8E-06	6 9E-06	4 7E-06	1 2E-05	2 3E-07	4 4E-07	5 6E-07	9.2E-07	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	1.6E-05	2 6E-05	2 2E-05	3.8E-05	4 3E-07	4.5E-07	9 9E-06	1.9E-05	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	1 8E-05	3 1E-05	2.7E-05	4.7E-05	6 2E-07	7 1E-07	6 7E-06	1 3E-05	ND	ND	ND	ND
tPCBs	NA	NA	3 0E-01	6 4E-01	NA	NA	NA	NA	NA	NA	ND	ND

Notes:
NA= not applicable, because analyte is not a COPEC for the given subarea or because empirical data not used (see footnote b)
ND= no data available for COPEC
[a]Use of TOC-normalized sediment data for exposure models limited to PCBs, DDT and isomers for modeling concentrations in fish tissue in Fox Creek. See Appendix E4.
[b]Surface water data for Fox Creek and the Channel not available; data from surface water in Forested Wetland used

Table E5-3. Mean Concentrations of COPECs in abiotic Exposure Media of Aquatic Receptors (Area B only), Except Fish

Medium	Water				TOC-norm sediment[a]	
Units	mg/L				mg/kg dw TOC	
Subarea	Pike Bay		Channel		Fox Creek	
EPC type	Mean	RME	Mean	RME	Mean	RME
Antimony	3 1E-04	4 4E-04	2.5E-04	5 0E-04	NA	NA
Barium	4 6E-02	4.7E-02	5.0E-02	6.0E-02	NA	NA
Beryllium	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA
Copper	NA	NA	NA	NA	NA	NA
Lead	NA	NA	2 0E-03	3.9E-03	NA	NA
Mercury	NA	NA	NA	NA	NA	NA
Molybdenum[b]	NA	NA	6.1E-05	1.9E-04	NA	NA
Selenium	NA	NA	NA	NA	NA	NA
Silver	NA	NA	NA	NA	NA	NA
Thallium	NA	NA	NA	NA	NA	NA
Zinc	NA	NA	NA	NA	NA	NA
LPAH	1.3E-04	1.2E-03	1.5E-05	1.5E-05	NA	NA
HPAH	2.0E-05	2.0E-05	ND	ND	NA	NA
DDD	NA	NA	ND	ND	2.5E-02	4 7E-02
DDE	NA	NA	ND	ND	2.5E-02	6.3E-02
DDT	NA	NA	ND	ND	4.8E-02	3.5E-01
Total DDX	NA	NA	ND	ND	NA	NA
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	ND	ND	ND	ND	NA	NA
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	ND	ND	ND	ND	NA	NA
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	ND	ND	ND	ND	NA	NA
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	ND	ND	ND	ND	NA	NA
tPCBs	NA	NA	NA	NA	3 0E-01	6.4E-01

Notes:
NA= not applicable, because analyte is not a COPEC for the given subarea or because empirical data not used (see footnote b)
ND= no data available for COPEC
[a]Use of TOC-normalized sediment data for exposure models limited to PCBs, DDT and isomers for modeling concentrations in fish tissue in Fox Creek See Appendix E4
[b]Surface water data for Fox Creek and the Channel not available; data from surface water in Forested Wetland used

Table E5-4. RME Concentrations of COPECs in biotic Exposure Media of Aquatic Receptors (Area B only), Except Fish

	Medium	*Lumbriculus*						*Corbicula*			
	Units	mg/kg dw						mg/kg dw			
	Subarea	All		Fox Creek		Channel		All		Fox Creek	
	EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
Antimony		NA	NA	NA	NA	5 1E-02	9 2E-02	NA	NA	NA	NA
Barium		NA	NA	NA	NA	1 7E+02	1.8E+02	NA	NA	NA	NA
Beryllium[a]		NA	NA	3 9E-03	1.1E-02	NA	NA	NA	NA	3 5E-03	7 0E-03
Cadmium		NA	NA	1.9E-01	2 5E-01	NA	NA	NA	NA	6 0E-01	6.3E-01
Copper		NA	NA	1.4E+01	1.8E+01	NA	NA	NA	NA	4.7E+01	4.7E+01
Lead		1 8E+00	2.7E+00	1.1E+00	2.1E+00	3.1E+00	6 6E+00	5 7E-01	2 9E+00	2 7E-01	2.9E-01
Mercury		NA	NA	1.7E-02	3.5E-02	NA	NA	NA	NA	1.2E-01	1 3E-01
Molybdenum		NA	NA	4 8E-01	8.0E-01	3.9E-01	5.0E-01	NA	NA	7 1E-01	7 5E-01
Selenium		NA	NA	1 4E+00	2.5E+00	NA	NA	NA	NA	3 4E+00	3 4E+00
Silver		NA	NA	5 3E-02	1.3E-01	NA	NA	NA	NA	9 6E-02	9.8E-02
Thallium		NA	NA	NA	NA	1 7E-03	5 0E-03	NA	NA	NA	NA
Zinc		NA	NA	1.5E+02	1.8E+02	NA	NA	NA	NA	9 6E+01	1 0E+02
LPAH		5.6E-02	1 1E-01	2 2E-02	3.7E-02	1.2E-01	2.8E-01	8.7E-02	2 0E-01	8.3E-02	1.4E-01
HPAH		2 0E-01	4 1E-01	5 1E-02	9.7E-02	4.6E-01	1.1E+00	6.9E-02	1.4E-01	2.0E-01	3.2E-01
DDD		NA	NA	1.8E-03	8.2E-03	7 4E-04	1.7E-03	NA	NA	5 9E-03	6.2E-03
DDE		NA	NA	3.2E-03	6.1E-03	3.0E-03	3.5E-03	NA	NA	8.5E-02	8.7E-02
DDT		NA	NA	6.0E-04	1.9E-03	4.2E-04	1 1E-03	NA	NA	ND	ND
Total DDX		NA	NA	5 5E-03	9.0E-03	4.1E-03	5.8E-03	NA	NA	9.0E-02	9.3E-02
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)		2 4E-07	3.3E-07	2 4E-07	3.8E-07	2.4E-07	3.3 E-07	2.6E-07	4 6E-07	2 2E-07	4.0E-07
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)		6.6E-07	9.3E-07	3 9E-07	6.3E-07	1.2E-06	1.3E-06	3.8E-07	6 6E-07	3 5E-07	3.9E-06
tPCBs		NA	NA	NA	NA	NA	NA	NA	NA	4.7E-02	4 9E-02

Notes:

NA= not applicable, because analyte is not a COPEC for the given subarea or because empirical value not used (see footnote c)

ND= no data available for COPEC

[a]No data for beryllium in wild rice was available; cattail data used for this COPEC

[b]Concentrations of COPECs in grass from Area B are used, as substitute for fruit, for estimating exposure to frugivorous aquatic receptors in area B

[c]Modeled value; empirical data not used See Appendix E4.

Table E5-4. RME Concentrations of COPECs in biotic Exposure Media of Aquatic Receptors (Area B only), Except Fish

Medium	*Corbicula*				Wild Rice		Grasses[b]	
Units	mg/kg dw				mg/kg dw		mg/kg dw	
Subarea	Pike Bay		Channel		Channel		All	
EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME
Antimony	7 7E-03	1.0E-02	1.6E-02	2.6E-02	NA[c]	NA[c]	2 9E-01	4 9E-01
Barium	4 3E+00	5.8E+00	1.6E+02	6 4E+02	NA[c]	NA[c]	4.1E+01	5 5E+01
Beryllium[a]	NA	NA	NA	NA	1.5E-03	3 0E-03	1.5E-03	3 0E-03
Cadmium	NA	NA	NA	NA	NA[c]	NA[c]	7.2E-02	1.5E-01
Copper	NA	NA	NA	NA	NA[c]	NA[c]	5 5E+00	1.3E+01
Lead	NA	NA	8 2E-01	2.9E+00	NA[c]	NA[c]	3 5E-01	8 1E-01
Mercury	NA	NA	NA	NA	NA[c]	NA[c]	3 0E-02	5 2E-02
Molybdenum	NA	NA	6.7E-01	1 1E+00	NA[c]	NA[c]	2.5E+00	6 4E+00
Selenium	NA	NA	NA	NA	NA[c]	NA[c]	2 0E-01	4 0E-01
Silver	NA	NA	NA	NA	NA[c]	NA[c]	2.0E-02	4 5E-02
Thallium	NA	NA	1.5E-03	4.0E-03	NA[c]	NA[c]	5 0E-03	8.0E-03
Zinc	NA	NA	NA	NA	NA[c]	NA[c]	5.0E+01	1 2E+02
LPAH	2 0E-02	2.2E-02	2.5E-02	6 4E-02	1.4E-03	1 5E-03	4.9E-03	1 3E-02
HPAH	2 0E-02	3 6E-02	4.9E-02	1 4E-01	2 4E-03	3.4E-03	9.0E-03	2.4E-02
DDD	NA	NA	4.1E-03	7.0E-03	ND	ND	ND	ND
DDE	NA	NA	6.0E-02	9.4E-02	ND	ND	ND	ND
DDT	NA	NA	5.3E-04	2.4E-03	ND	ND	ND	ND
Total DDX	NA	NA	6.4E-02	1.0E-01	ND	ND	ND	ND
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	1 4E-07	1.9E-07	3.3E-07	7.4E-07	1 1E-07	1.4E-07	1 3E-07	1 7E-07
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	2 2E-07	3.4E-07	4.8E-07	1.0E-06	9.6E-08	1.3E-07	9.7E-08	1 4E-07
tPCBs	NA	NA	NA	NA	ND	ND	ND	ND

Notes:

NA= not applicable, because analyte is not a COPEC for the given subarea or because empirical value not used (see footnote c)

ND= no data available for COPEC

[a]No data for beryllium in wild rice was available; cattail data used for this COPEC

[b]Concentrations of COPECs in grass from Area B are used, as substitute for fruit, for estimating exposure to frugivorous aquatic receptors in area B

[c]Modeled value; empirical data not used See Appendix E4.

Table E5-5. The Mean Concentration in Fish Tissue modeled for Fox Creek

Medium	Mudminnow[a]		Yellow perch[a]		Fish: modeled metals[a]	
Units	mg/kg dw		mg/kg dw		mg/kg dw	
Subarea	Fox Creek		Fox Creek		Fox Creek	
EPC type	Mean	RME	Mean	RME	Mean	RME
Beryllium	N/A	N/A	N/A	N/A	3.5E-03	4.8E-03
Cadmium	N/A	N/A	N/A	N/A	3.6E-02	5.9E-02
Copper	N/A	N/A	N/A	N/A	1.2E+01	1.9E+01
Lead	N/A	N/A	N/A	N/A	8.8E-04	1.5E-03
Mercury	N/A	N/A	N/A	N/A	4.8E-01	8.7E-01
Molybdenum	N/A	N/A	N/A	N/A	N/A[b]	N/A[b]
Selenium	N/A	N/A	N/A	N/A	1.0E-01	1.3E-01
Silver	N/A	N/A	N/A	N/A	9.6E-04	1.7E-03
Zinc	N/A	N/A	N/A	N/A	1.0E+01	1.8E+01
LPAH	N/A	N/A	N/A	N/A	N/A	N/A
HPAH	N/A	N/A	N/A	N/A	N/A	N/A
DDD	1.3E-02	2.4E-02	8.7E-03	1.6E-02	N/A	N/A
DDE	2.4E-02	6.1E-02	2.4E-02	6.0E-02	N/A	N/A
DDT	1.9E-02	1.4E-01	1.0E-02	7.2E-02	N/A	N/A
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	8.9E-07	1.5E-06	1.8E-06	2.9E-06	N/A	N/A
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	1.2E-06	2.1E-06	2.9E-06	4.9E-06	N/A	N/A
TEQ_{DFP} (ND = 1/2 DL, fish TEFs)	8.6E-07	1.4E-06	1.7E-06	2.9E-06	N/A	N/A
tPCBs	1.7E-02	3.7E-02	1.8E-01	3.9E-01	N/A	N/A

Notes:

NA= not applicable, because analyte is not a COPEC for the given subarea or because modeling was not possible for given COPEC (see footnote c)

[a]Metals are modeled using BCF values; organics modeled using species-specific BSAF values. See Appendix E4 for discussion of these modeling procedures. LPAH and HPAH are not modeled; site-specific sucker data is used (see Table 5-6).

[b]A BCF was not available for molybdenum; site-specific sucker data was used for estimating exposure and risk from this metal (See Table E5-6).

Table E5-6. The RME Concentration in Fish Tissue, Site Data for Whole-body White Sucker

Medium	White sucker	
Units	mg/kg dw	
Subarea	Cass Lake/Pike Bay	
EPC type	Mean	RME
Antimony	5.5E-03	2.0E-02
Barium	6 0E+00	7.4E+00
Beryllium	N/A[a]	N/A[a]
Cadmium	N/A[a]	N/A[a]
Copper	N/A[a]	N/A[a]
Lead	3.8E-01	5.6E-01
Mercury	N/A[a]	N/A[a]
Molybdenum	9 6E-02	1.4E-01
Selenium	N/A[a]	N/A[a]
Silver	N/A[a]	N/A[a]
Thallium	2.1E-03	1.0E-02
Zinc	N/A[a]	N/A[a]
LPAH	7.7E-02	9.9E-02
HPAH	1.4E-01	1.8E-01
DDD	3.2E-03	7.1E-03
DDE	3.4E-02	5.3E-02
DDT	4.3E-03	7.5E-03
TEQ_{DFP} (ND = 1/2 DL, mammal TEFs)	3.3E-06	4.2E-06
TEQ_{DFP} (ND = 1/2 DL, bird TEFs)	7.8E-06	1.0E-05
TEQ_{DFP} (ND = 1/2 DL, fish TEFs)	1.2E-06	1.5E-06
tPCBs	8.1E-02	1.2E-01

Note:
[a]N/A = not applicable because values modeled for this COPEC in fish (see table E5-5).

Table E5-7 Average and RME Daily Rates of Ingestion of Each COPEC: Terrestrial Receptors

Area	Area A						Forested Wetland					
Receptor	Robin		Shrew		Vole		Robin		Shrew		Vole	
EPC Type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs)	1 4E-05	1.6E-05	8.1E-06	8.5E-06	1 0E-06	1 4E-06	9 4E-06	1 0E-05	6 8E-06	7 0E-06	1.5E-07	3 0E-07
PCBs[c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
LPAH	1.7E-02	2 1E-02	7 6E-03	8.2E-03	2.1E-03	3 7E-03	N/A	N/A	N/A	N/A	N/A	N/A
HPAH	1 5E-01	2 1E-01	7 2E-02	8.2E-02	1 0E-02	1.8E-02	N/A	N/A	N/A	N/A	N/A	N/A
Antimony	3 3E-02	4.3E-02	1 4E-02	1.6E-02	1 3E-02	2.5E-02	N/A	N/A	N/A	N/A	N/A	N/A
Cadmium[b c]	N/A	N/A	N/A	N/A	N/A	N/A	4 1E-01	5 1E-01	3 0E-01	3 7E-01	7 2E-03	1 0E-02
Copper[c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Lead	9 4E-01	1.1E+00	6 2E-01	6 7E-01	3 9E-02	5.9E-02	1.4E+00	1 8E+00	7 7E-01	9 4E-01	6 9E-02	1 1E-01
Mercury[c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Selenium	1 6E-01	1 8E-01	1 3E-01	1,4E-01	6.5E-03	1 5E-02	N/A	N/A	N/A	N/A	N/A	N/A
Vanadium	7 7E-01	7 8E-01	5 2E-01	5 3E-01	2 8E-02	3 1E-02	N/A	N/A	N/A	N/A	N/A	N/A
Zinc[b]	N/A	N/A	N/A	N/A	N/A	N/A	1 9E+01	2 0E+01	1 3E+01	1 3E+01	1 7E+00	2.2E+00
PCP	1 3E-01	1 5E-01	9 5E-02	9 7E-02	3 1E-03	6 5E-03	N/A	N/A	N/A	N/A	N/A	N/A
BHC[c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

	Area B					
	Robin		Shrew		Vole	
	Mean	RME	Mean	RME	Mean	RME
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs)	2 6E-06	4 9E-06	8 7E-07	1 8E-06	4 0E-07	6 7E-07
PCBs[c]	2 6E-03	4 9E-03	1 2E-03	2.2E-03	N/A	N/A
LPAH	3.6E-02	8 8E-02	1 4E-02	3 6E-02	2 9E-03	6 8E-03
HPAH	2 1E-01	4.3E-01	6 8E-02	1 2E-01	1 8E-02	3.9E-02
Antimony	2 2E-02	3 5E-02	8 5E-03	1 3E-02	2 0E-02	3 3E-02
Cadmium[b,c]	5 2E-01	6.3E-01	4 0E-01	4 9E-01	1 1E-02	1 5E-02
Copper[c]	2 9E+00	4 8E+00	2 0E+00	3 4E+00	4 8E-01	8 8E-01
Lead	2 1E+00	3 6E+00	1 2E+00	2 0E+00	1 0E-01	2 1E-01
Mercury[c]	3 0E-02	5 0E-02	2 2E-02	3 7E-02	2 2E-03	4 0E-03
Zinc[b]	4 9E+01	1 1E+02	3 7E+01	8 3E+01	3 7E+00	8 6E+00
PCP	8 9E-01	4 2E+00	7 0E-01	3.3E+00	8 5E-03	3 5E-02
BHC[c]	1 8E-04	3 6E-04	1 1E-04	2 2E-04	1 7E-06	4 2E-06

Notes:
NA= not applicable, because analyte is not a COPEC for the given subarea
a=Bird TEFs were used for robin, mammal TEFs were used for shrew and vole
b=Forested Wetland COPEC only in Area A
c=City Dump COPEC only in Area B

Table E5-8 Average and RME Daily Rates of Ingestion of Each COPEC Aquatic Receptors, Fox Creek

Area	Fox Creek									
Receptor	Great blue heron		Mallard		Mink		Muskrat		Raccoon	
EPC type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) excluding EPA-A data[d]	1 8E-08	4 5E-08	6 7E-10	1 3E-09	1 2E-08	2 8E-08	1 4E-08	2 5E-08	2 5E-09	5 7E-09
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) including EPA-A data[d]	1.3E-07	2 2E-07	1 7E-09	3 0E-09	7 2E-08	1 2E-07	4 3E-08	7 2E-08	1 4E-08	2 3E-08
PCBs	4 54E 03	9 7E-03	3 3E-05	5 9E-05	2.3E-03	4 8E-03	N/A	N/A	4 5E-04	9 2E-04
LPAH	5 4E-03	8 3E-03	8 8E-05	1 7E-04	4 4E-03	7.7E-03	2 3E-03	4 5E-03	8 9E-04	1 6E-03
HPAH	1 1E-02	1 7E-02	2 2E-04	3 9E-04	1 0E-02	1 6E-02	5 7E-03	1 0E-02	2 1E-03	3 5E-03
Beryllium	4 7E-04	6 4E-04	1 5E-05	2 7E-05	5 2E-04	7 1E-04	4 6E-04	6 8E-04	1 1E-04	1 5E-04
Cadmium	3.7E-03	5 9E-03	2 7E-04	3 7E-04	4 4E-03	6 6E-03	3 3E-03	5 5E-03	1 4E-03	1 9E-03
Copper	6 0E-01	9 8E-01	1 9E-02	2 6E-02	4 1E-01	6 3E-01	3 4E-01	5 7E-01	1 1E-01	1 5E-01
Lead[c]	8 4E-02	1 5E-01	4 0E-03	7 1E-03	1 2E-01	2 0E-01	1 1E-01	1 8E-01	2 6E-02	4 5E-02
Mercury	2 3E-02	4 1E-02	2 4E-04	4 3E-04	1 2E-02	2 2E-02	3 3E-02	5 9E-02	1 9E-03	3 4E-03
Molybdenum	6 1E-03	8 6E-03	6 6E-04	1 1E-03	5 5E-03	7 3E-03	1 8E-02	2 5E-02	3 8E-03	7 9E-03
Selenium	1 1E-02	1 4E-02	1 4E-03	2 5E-03	1.6E-02	1 9E-02	1 6E-02	2 1E-02	5 7E-03	6 5E-03
Silver	1 1E-02	1 9E-02	5 1E-04	9 3E-04	1.5E-02	2 7E-02	3 0E-02	5 3E-02	3 7E-03	5 9E-03
Zinc	9 4E-01	1 6E+00	1 9E-01	2 3E-01	1 0E+00	1 7E+00	3 4E+00	6 0E+00	3 1E-01	5 0E-01
DDT[b,c]	6 5E-04	4.6E-03	1 1E-06	3 2E-06	N/A	N/A	N/A	N/A	N/A	N/A
DDE[b,c]	1.1E-03	2 7E-03	4 9E-06	9 7E-06	N/A	N/A	N/A	N/A	N/A	N/A
DDD[b,c]	4 8E-04	9 0E-04	2 7E-06	1 2E-05	N/A	N/A	N/A	N/A	N/A	N/A
Total DDX[b,c]	N/A	N/A	N/A	N/A	1 2E-03	3 9E-03	N/A	N/A	2 8E-04	7 8E-04

NA= not applicable, because analyte is not a COPEC for the given subarea

[a]Bird TEFs were used for great blue heron, mallard, kingfisher, and mammal TEFs were used for mink, muskrat, and raccoon

[b]Total DDX used for estimating exposure and risk to mammals, DDT and isomers used for estimating exposure and risk to birds

[c]COPEC for Fox Creek and Channel. Lead is additionally a COPEC in Overall Aquatic Area B.

Table E5-8 (continued) Average and RME Daily Rates of Ingestion of Each COPEC Aquatic Receptors, Pike Bay and the Channel

Area	Pike Bay										Channel									
Receptor	Kingfisher		Mallard		Mink		Muskrat		Raccoon		Kingfisher		Mallard		Mink		Muskrat		Raccoon	
EPC Type	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) excluding EPA-A data[f]	7 6E-07	1 0E-06	4 4E-10	7 3E-10	6 9E-08	8.7E-08	8 1E-09	1.0E-08	8.5E-09	1 1E-08	5 6E-07	7 4E-07	1 1E-09	1 2E-09	3 5E-08	4 6E-08	8 9E-09	1 2E-08	7 9E-09	1 1E-08
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) including EPA-A data[f]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	5 6E-07	7 4E-07	1 4E-09	1 9E-09	4 5E-08	6 4E-08	2 4E-08	4 1E-08	1 1E-08	1 7E-08
LPAH	7 9E-03	1 0E-02	2 0E-05	2 5E-05	1 7E-03	2 2E-03	1 3E-04	2 7E-04	2 2E-04	2 9E-04	5 8E-03	7 9E-03	1 6E-04	3 4E-04	2 4E-03	3 4E-03	2 5E-03	3 7E-03	7 2E-04	1 1E-03
HPAH	1 4E-02	1 8E-02	2 3E-05	4.4E-05	3 0E-03	4 1E-03	2 7E-04	5 4E-47	3 9E-04	5.6E-04	1 0E-02	1 5E-02	6 4E-04	1 4E-03	8 0E-03	1 4E-02	1 0E-02	1 8E-02	2.6E-03	4 4E-03
Antimony[d]	8 3E-04	2 4E-03	1 6E-04	4.9E-04	5 6E-03	1 7E-02	1 3E-02	4 0E-02	1 3E-03	3.7E-03	7 2E-04	2 0E-03	1.8E-04	2 7E-04	2.7E-03	4.05-03	1 2E-03	1 8E-02	1 1E-03	1 7E-03
Barium[d]	6 9E-01	8 6E-01	1 6E-01	6 3E-01	1 6E-01	1 9E-01	6 6E-02	8 0E-02	5 8E-02	7.7E-02	2 7E+00	9 9E+00	1 6E-01	1 7E-01	3 4E-01	8 5E-01	6 8E-01	8 3E-01	2 3E-01	6 8E-01
Lead	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	3 9E-02	8 2E-02	3 7E-03	7 1E-03	3 1E-02	4 2E-02	4 5E-02	5 8E-02	1 1E-02	1 6E-02
Molybdenum	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	1 7E-02	2 7E-02	3 8E-04	5 3E-04	2 1E-03	3 4E-03	6 5E-03	1 5E-02	2 9E-03	6 5E-03
Thallium[e]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	1 8E-04	7 7E-04	3 1E-06	7 7E-06	5 7E-05	1 8E-04	1 4E-04	2 8E-04	2 1E-05	5 5E-05
DDT[b,c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	3 2E-04	5 7E-04	5 0E-07	1 3E-06	N/A	N/A	N/A	N/A	N/A	N/A
DDE[b,c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	3 3E-03	5 2E-03	3 6E-06	4 3E-06	N/A	N/A	N/A	N/A	N/A	N/A
DDD[b,c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	2 9E-04	6 0E-04	9 7E-07	2 4E-06	N/A	N/A	N/A	N/A	N/A	N/A
Total DDX[b,c]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	4 9E-04	7 1E-04	N/A	N/A	1 8E-04	2 7E-04

NA= not applicable, because analyte is not a COPEC for the given subarea

[a]Bird TEFs were used for great blue heron, mallard, kingfisher, and mammal TEFs were used for mink, muskrat, and raccoon

[b]Total DDX used for estimating exposure to mammals, DDT and isomers used for estimating exposure to birds

[c] COPEC for Fox Creek and Channel Lead is additionally a COPEC in Overall Aquatic Area B

[d]COPEC for Pke Bay and Channel only

[e]COPEC for Channel only

Table E5-8 (continued) Average and RME Daily Rates of Ingestion of Each COPEC Aquatic Receptors, Overall Aquatic Area B

COPEC	Overall Aquatic Portion Area B									
	Great blue heron		Mallard		Mink		Muskrat		Raccoon	
	Mean	RME	Mean	RME	Mean	RME	Mean	RME	Mean	RME
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) excluding EPA-A data[d]	3 5E-07	4 6E-07	2 4E-09	3 8E-09	1 7E-07	2 2E-07	8 3E-09	1 1E-08	2 6E-08	3 4E-08
TEQ_{DFP} (ND = 1/2 DL, mammal/bird[a] TEFs) including EPA-A data[d]	3 7E-07	5 0E-07	4 4E-09	6 9E-09	2 5E-07	3 4E-07	3 2E-08	4 9E-08	4 3E-08	6 1E-08
LPAH	5.2E-03	7 3E-03	3 2E-04	5 8E-04	1 1E-02	1 7E-02	2 1E-03	3 4E-03	2 3E-03	3 8E-03
HPAH	1.2E-02	1 6E-02	1 1E-03	2 0E-03	2 7E-02	4 0E-02	6 4E-03	9 8E-03	5 8E-03	8 8E-03
Lead[c]	7.3E-02	1 2E-01	1.1E-02	1 6E-02	2 2E-01	3 6E-01	7 1E-02	1 1E-01	5 2E-02	8 9E-02

NA= not applicable, because analyte is not a COPEC for the given subarea

[a]Bird TEFs were used for great blue heron, mallard, kingfisher, and mammal TEFs were used for mink, muskrat, and raccoon

[b]Total DDX used for estimating exposure to mammals, DDT and isomers used for estimating exposure to birds. COPEC for Fox Creek and Channel only

[c]COPEC for Fox Creek and Channel Lead is additionally a COPEC in Overall Aquatic Area B

APPENDIX E6

ANALYSIS OF *CHIRONOMUS TENTANS* GROWTH IN SITE SEDIMENTS

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Appendix E6
Analysis of *Chironomus tentans* Growth in Site Sediments

Prepared for
International Paper
pursuant to
Unilateral Administrative Order Docket No. V-W-04-C-796

Prepared by

integral consulting inc

12303 Airport Way
Suite 370
Broomfield, CO 80021

September 28, 2007

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AET	apparent effects threshold
AFDW	ash-free dry weight
AIC	Akaike Information Criterion
ARCS	Assessment and Remediation of Contaminated Sediments
CCC	criterion continuous concentration
CCME	Canadian Council of Ministers of the Environment
COPEC	chemicals of potential ecological concern
DDx	DDT and metabolites
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ESL	ecological screening level
ESV	ecological screening value
HPAH	high-molecular-weight polycyclic aromatic hydrocarbon
ISQG	interim sediment quality guidelines
LAET	lowest apparent effects threshold
LEL	lowest effect level
LPAH	low-molecular-weight polycyclic aromatic hydrocarbon
MLR	multiple linear regression
MTCA	Model Toxics Control Act
NOM	natural organic matter
OSWER	Office of Solid Waste and Emergency Response
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-*p*-dioxin
PCDF	polychlorinated dibenzofuran
PCP	pentachlorophenol
RAWP	risk assessment work plan
ROS	regression on order statistics

RSS	residual sum of squares
SCV	secondary chronic values
SSL	soil screening levels
SQB	sediment quality benchmark
SQC	sediment quality criteria
SQG	sediment quality guideline
TEC	threshold effect concentration
TEL	threshold effect level
TRV	toxicity reference value
WSDE	Washington State Department of Ecology

1 INTRODUCTION AND BACKGROUND

Toxicity tests were conducted using 27 sediment samples, including 23 stations from the Site and 4 upstream, with 3 of the upstream samples used as reference stations. Sediment samples were collected in 2004 from the channel, Pike Bay, and Fox Creek. Three endpoints were tested in support of the ERA for the Site:

- *H. azteca* (amphipod) survival
- *C. tentans* (midge) survival
- *C. tentans* growth.

Statistical comparisons of effects in Site sediments relative to reference sediments were performed for each of these endpoints to determine whether the survival of amphipods and the survival and growth of midges in Site sediments were significantly different from those in reference sediments. Results from all three reference stations were pooled for the comparisons. All methods and results of those comparisons are discussed in Section 5.4.2 of the main text of this report.

Survival of *C. tentans* and *H. azteca* exposed to Site sediments was not significantly different from survival of the organisms exposed to reference sediments (Section 5.4.3), and is not addressed further in this appendix. However, in the *C. tentans* growth test, growth of midges in nine stations in Fox Creek, including one from upstream of the Site, was significantly different from that in the pooled reference samples (Figure E6-1).

This appendix provides a series of analyses that explore the possible causes of the observed differences in growth between Site stations and the pooled reference sample. The analysis employs information on sediment chemistry and physicochemical parameters including sediment grain size, total organic carbon (TOC), and concentrations of organic forms of nitrogen and phosphorus; sediment quality guidelines (SQGs) used in the screening analysis (Section 5.1 of the risk assessment report, Appendix E1); and the response data from the results of the 10-day growth bioassay, and the final individual midge ash-free dry weight (AFDW, mg). An overview of the approach taken to investigate causal factors is provided below, followed by the detailed documentation of each analytical step. Conclusions are summarized at the end of this appendix.

2 OVERVIEW OF CAUSALITY INVESTIGATION

The objectives of the analyses discussed below and a related analysis presented in Section 5.5.2.2 of the risk assessment report are 1) to identify the set of possible causes of *C. tentans* growth effects observed in the nine affected stations, and 2) to develop tools for understanding the potential for adverse effects on benthic invertebrates in sediments at which no toxicity tests have been performed, using only sediment TOC and chemistry data. The investigation described in this appendix is focused on the first objective, and has been conducted in an iterative fashion, such that the results of one analysis has led to a series of questions pursued in subsequent analyses, resulting in a complex set of interrelated results. The second objective is described in Section 5.5.2.2 of the main risk assessment report, and uses the multiple linear regression (MLR) analysis developed below, but is otherwise not addressed in this appendix.

This investigation of possible causes of the observed growth effects applied multivariate statistical techniques to quantify the relationships between sediment characteristics and toxicity test results. Analyses of relationships among sediment variables and organisms' responses using individual correlation statistics, MLR methods, and principal components analysis (PCA) can help refine the understanding of cause-effect relationships. Growth effects in the toxicity tests are presumed to be caused by systematic differences in sediment conditions in the test chambers. A correlation between the cause and effect is therefore to be expected. In this context, if a chemical is not correlated with growth, then it is unlikely to be the cause of any growth effect. Although a correlation between concentrations of a chemical and growth does not prove the existence of a cause-effect relationship, a chemical correlating with growth cannot be ruled out as the cause of the pattern. Conversely, the absence of correlation can be used to eliminate a chemical from among the list of candidate causes. When the list of correlates with a dependent variable is sufficiently narrowed and consistently observed, inferences about the cause of the effect can be made with greater confidence.

The procedure used to evaluate potential causes of the observed growth effects consisted of the following steps:

1. Identify chemicals that are negatively correlated with growth (i.e., where higher concentrations are associated with lower growth)
2. Identify chemicals that are statistically significant predictors of growth using multiple linear regression techniques
3. Identify chemicals among those directly correlated with growth or those that are good predictors of growth in multiple linear regression models that are present at concentrations that exceed conservative SQGs
4. Evaluate a mechanism by which high TOC may affect midge growth in the bioassay environment by quantifying the nutritional quality of midge diets

5. Evaluate whether the results of the previous steps are compatible with published data on *Chironomus* toxicity.

These steps were carried out independently, and provide several different ways of assessing the potential causes of growth effects.

3 DATA SETS AND DATA TREATMENT

Two data sets were used for this analysis:

- Bioassay results (AFDW), sediment chemistry, TOC, and grain size for 27 bioassay stations sampled in 2004 (bioassay stations)
- Chemistry, TOC, and grain size for 46 stations at which toxicity tests were not conducted, mostly sampled in 2001 (chemistry-only stations)

The bioassay data sets used are described in full detail by Barr (2005) and are discussed in Section 5.4 of the risk assessment report. Chemical and physical data sets are described in Section 2 of the risk assessment report. In addition to these data, eight sediments were analyzed for nutrients, as discussed in Section 7 of this appendix. Figure E6-1 provides the locations of all stations in data sets, stations at which nutrients were analyzed in sediments, and the results of the statistical comparisons of *C. tentans* growth in Site sediments relative to those in pooled reference sediments.

Chemical detection limits and detection frequencies vary in the two data sets (Table E6-1 for the bioassay data set Table E6-2 for the chemistry-only data set), with detection limits generally higher in the chemistry-only data set. Methods for addressing the uncertainties associated with these left censored data are outlined below. Also, PCA was used to reduce sets of correlated chemical parameters to a smaller number of parameters that aggregate information from the related variables, and to evaluate whether chemical patterns in the bioassay data set are similar to those in the chemistry-only data set. This latter analysis was performed to determine whether a predictive model developed with the bioassay data set could be used to make predictions using the chemistry-only data set. Results of PCA are briefly described in this section; details of PCA are described in Attachment E6A.

3.1 TREATMENT OF NONDETECTS

In the chemistry data set for all sediment stations, several chemicals were not detected in some samples (Table E6-1); pesticides including DDT and metabolites (DDx) had very low detection frequencies, as did several metals. In bioassay stations only, detection frequencies of some chemicals of potential ecological concern (COPECs) were 33 percent or lower (Table E6-2). Although regression on order statistics (ROS) can be used to impute information to substitute for nondetects when calculating means or related statistics in censored data sets (i.e., as for computation of exposure point concentrations), this method does not assign specific concentration estimates to specific samples (Helsel 2005). As a result, ROS cannot be used to estimate values for nondetects when performing analyses of correlations among variables, such as when generating a correlation matrix or conducting a regression analysis.

In the case of chemistry data sets, the ability to compute the traditional parametric measure of correlation, Pearson's r, is diminished with non-normally distributed, highly censored data sets. For analysis of simple two-way correlations, Helsel (2005) recommends that Kendall's tau-b is often the most appropriate statistic when the subject data set is heavily censored. Given low detection frequencies for several chemicals and variable, non-normal distributions for most chemicals, Kendall's tau-b was used to evaluate the correlations among the sediment parameters in the bioassay station data set including concentrations of COPECs, sediment grain size information, and percent TOC, and between these parameters and growth.

Robust statistical methods are not available to perform regression analysis and PCAs on data sets with extensively censored data. For MLR and PCA, non-detects were substituted at one-half the detection limit, and the analysis repeated with substitution at the full detection limit to determine if results were consistent. Some chemicals were excluded if they were too highly censored. Differences resulting from different substitution methods are highlighted and discussed.

3.2 PRINCIPAL COMPONENTS ANALYSIS WITH SEDIMENT CHEMISTRY

From the analysis of chemical patterns using PCA (Attachment E6A), it was concluded that sums of low-molecular-weight polycyclic aromatic hydrocarbons (LPAHs) and high-molecular-weight PAHs (HPAHs) adequately capture the variability of the list of PAH chemicals, so that aggregate values for these compound mixtures can be used in MLR of sediment characteristics against AFDW. These summary variables were used in the MLR because with individual concentrations of PAHs, the number of variables to be used in the MLR analysis was too large relative to the number of samples. These summary variables were also used in evaluation of the role of nutritional quality in the growth of *C. tentans*.

The analysis of whether chemical relationships among samples for which it may be necessary to predict growth are similar to the relationships among the synoptic chemistry/bioassay samples that will be used to derive the predictive model concluded that patterns are generally similar. No extreme outlier groups were identified. However, the presence of high detection limits in the chemistry-only data set (exceeding some detected values in the overall data set) will require that some predictions are based on extrapolation slightly beyond the range of the data used to fit the regression model. This could result in important uncertainties in predicting growth, if the predictor variables resulting from the MLR are among those that are highly censored in the chemistry-only data set. If predictor variables are among those with many nondetects, predictions should be bounded by different substitutions. Alternatively, MLR conducted without variables that are highly censored in the data for which growth predictions are needed (i.e., the chemistry-only stations) reduces the uncertainty created by high detection limits in the chemistry-only data set.

4 CORRELATION ANALYSES

Among the 27 stations tested for toxicity, 54 physical and chemical attributes were measured in sediment, any of which could potentially be the single cause of the growth effect, or could play a role in the reduced growth. Two exploratory steps were taken using these data to help narrow the range of parameters in this evaluation:

- Analysis of correlations among COPECs in bioassay stations
- Analysis of univariate correlations between growth and individual sediment parameters.

Because the objective of this analysis is to identify possible causes of the observed growth effect, the first correlation analysis was performed only on COPECs that emerged from the screening process (Section 5.1). COPECs are those chemicals that both exceed a conservative screening value and background, are higher in site sediments than in background (for chemicals without screening values), or have a significant toxicity or bioaccumulation potential (e.g., total polychlorinated dibenzo-*p*-dioxins [PCDDs] and total polychlorinated dibenzofurans [PCDFs]). Those chemicals present only at concentrations below SQGs are considered to be unlikely to be the cause of effects on growth. The set of SQGs used are those defined in Appendix E1, according to the hierarchy established by the risk assessment work plan (RAWP) (USEPA 2004), and represent concentrations at or below which effects on benthic invertebrates are unlikely.

4.1.1 Correlations Among Sediment Attributes

Values for Kendall's tau-b (Table E6-3) indicate that most correlations among COPECs, sediment grain size, and TOC are statistically significant, although the correlations are generally weak (tau-b < 0.5). Most chemicals are significantly correlated with TOC and grain size, and the physical associations between chemicals and particle surfaces may be the reason why many of the chemicals are significantly correlated with one another. Based on the relatively strong correlations (tau-b > 0.5), there appear to be two fairly distinct groups of correlated chemicals, one of them consisting of PAH compounds and the other consisting of the metals barium, beryllium, cadmium, copper, lead, mercury, silver, and zinc. The LPAH and HPAH concentrations are the most strongly correlated in the bioassay station data set. Zinc is not only correlated with other metals, but also with total PCDD, total PCDF and total DDx. This multiple covariance, especially that signaled by the relatively strong correlation of physical properties with most other parameters, complicates the process of identifying the parameter(s) that cause the observed growth effect. The additional analyses reported in subsequent sections help to distinguish between causes and covariates.

4.1.2 Correlations Between Sediment Attributes and AFDW

Kendall's tau-b was also used to evaluate the strength of correlations between all of the individual sediment physical and chemical parameters (not just COPECs) and AFDW (mg) (Table E6-4). The results are sorted both by the strength of the correlation (i.e., by tau-b) and by the chemical group. This analysis shows that the chemicals with the strongest, and statistically significant, negative correlations with growth are TOC, most of the metals, percent fines, and percent silt. Of the PAH compounds, only 2-methylnaphthalene is included in this group. Among the metals, molybdenum and chromium have the strongest negative correlations with growth, and also with TOC, having Kendall's tau-b values of 0.62 and 0.60, respectively.

Taken together, these correlations indicate that metals, TOC, and 2-methylnaphthalene are the parameters that are most strongly related to the effects on growth. These results do not allow a single chemical, or small set of chemicals, to be specifically identified as a likely cause of the observed growth effects. However, if TOC is influencing growth primarily through the association of chemicals with TOC, then only those chemicals that are more strongly correlated with AFDW than is TOC (i.e., molybdenum and chromium) are most likely to be causal agents. However, TOC may have effects on growth through mechanisms other than a physical association with metals and other chemicals (see Section 7), so it should be carried forward as a potential causative agent, or practical surrogate for a causal agent.

5 MULTIPLE LINEAR REGRESSION

To further refine the set of chemicals that may be responsible for growth effects, and to provide a means of predicting growth effects where they were not directly measured, an MLR analysis was carried out to identify the chemicals with the strongest ability to predict AFDW. Visual inspection of the relationship between growth (AFDW, mg) and sediment chemical concentrations (Figure E6-2) shows that for many sediment variables, there appears to be a threshold concentration associated with a decreasing growth response. Below the threshold concentration, growth responses were all high (>0.87 mg AFDW) and above the threshold concentration is an apparent change in the pattern of AFDW relative to the chemical concentration. Figure E6-2 illustrates that for both original and $\log_{10}$ scale axes for the sediment variables, TOC is strongly associated with a threshold response for both decreased growth and increased chemical concentrations. Given this threshold, there are two choices for the form of a regression model built from these data:

- A single multiple linear model
- A model with both a threshold and multiple linear model (a "threshold" model).

The form of the model, and the choice of transformation for the independent variables, will determine which variables are the best predictors of growth. The best model within each of the two model forms can be identified, and the goodness of fit (distribution of the residuals, adjusted R^2, and Akaike Information Criterion [AIC]) for the final model of each form can be used to compare them.

5.1.1 Data Sets and Data Treatment

The variables included in the regression models were TOC, percent fines, the metals, total LPAHs, total HPAHs, total PCDD, and total PCDF. Aggregated PAH groups (LPAH and HPAH) were considered representative of their constituent chemicals on the basis of the PCA performed on the chemistry data set for bioassay stations (Attachment E6A). Other variables were not included (i.e., total polychlorinated biphenyls [PCBs], pesticides, DDT and metabolites) because of very low detection frequencies (<45 percent) (Table E6-1).

For this analysis, normal (Gaussian) regression techniques were applied, and detection limits were used for the undetected observations. The effect of substituting detection limits for undetected values can be minimized by pre-selecting variables with minimal nondetects. The effect of the value chosen for substitution can be evaluated by bounding the substitution value, (i.e., using a substitution value near zero and another at the detection limit). For the chosen set of variables, cadmium, mercury, selenium, and silver had detection frequencies < 89 percent. Correlations between growth and each of these metals was affected very little by the substitution value used for the nondetects. The largest change in Pearson's r was from –0.77 (substitution at

the reported detection limits) to –0.82 (substitution at half the minimum detection limit) for cadmium.

Substitution at the reported detection limits will result in a steeper slope than would a lower substitution value. For this reason, the MLRs were run using two different sets of chemical data: first, with all available predictors in the data set for the bioassay stations, and second, with the same data set but with those chemicals with very low detection frequencies in the chemistry-only data set removed. It may be more appropriate to apply models developed with the latter data set when predicting stations at which there may be an effect on growth using the chemistry-only data set.

Thallium and molybdenum are excluded from the set of possible predictors because they are missing from too many stations in the data set to which the predictive model will be applied. These were briefly investigated to ensure that excluding them would not result in loss of important information (Figure E6-2).

- Thallium concentrations were not elevated above background in creek sediments where significant growth effects were observed. Thallium also did not have as strong a relationship with growth as many other metals (Table E6-4), although the relationship was significant.
- Molybdenum was the best individual predictor of growth; however, molybdenum ($\log_{10}$ scale) is highly correlated with TOC (Pearson's correlation coefficient of 0.93). Therefore, little information is lost by excluding molybdenum and retaining TOC in the set of predictor variables.

5.1.2 Methods

The forward stepwise method based on Efroymson (Miller 1990) was used for model building. At each step, the variable that provided the greatest reduction in residual sum of squares among available variables was added; partial correlations were also considered at each step to see if any of the variables already in the model should be dropped. The forms considered for the initial relationship between growth and the independent variables were:

- Model Type 1: strictly linear for $\log_{10}$ (concentration) data.
- Model Type 2: a threshold plus linear model—the threshold was determined by the $\log_{10}$ concentration data, based on visual inspection. The addition of each new variable was evaluated for both a linear and threshold contribution ($\log_{10}$ concentration data), and the best form was chosen based on the smallest residual sum of squares between the two choices.

For both model types, two models were generated. The first uses all available predictors in the bioassay data set, the second does not include arsenic, cobalt and silver as predictors because of the low detection frequency for these metals in the chemistry-only data set.

5.1.3 Results

The "stepwise" function in S-Plus (2000) was used to apply the forward stepwise method, with results for Model Type 1 presented in Table E6-5a and E6-5b. Results in Table E6-5b describe the model from which arsenic, cobalt, and silver were removed prior to the analysis. The final residual sum of squares (RSS) with all predictors was 0.049, and the adjusted R^2 was 0.96. The model without arsenic, cobalt, and silver included was slightly poorer (R^2 = 0.93). Residuals of the first model of Type 1 were not significantly different from normal (p=0.47 for correlation of the QQ-Plot shown in Figure E6-3) and variances appear homogeneous over the range of predicted values (Figure E6-3).

A forward stepwise method similar to Efroymson was manually employed in S-Plus (2000) to develop model type 2, both with and without arsenic, cobalt, and silver. For the first step, each variable was considered with either a linear or linear plus a threshold contribution. At this step, the best reduction in deviance, or RSS, was found using selenium, TOC, and chromium (in order of decreasing goodness of fit). The goodness of fit for the selenium and TOC models were comparable (the difference in AIC was less than 1). Selenium had a low detection frequency (67 percent) (Table E6-1), so this model was not considered optimal and TOC was left as the most desirable individual model for the first step (Tables E6-6a and E6-6b).

At each additional step, the best model from the previous step was used as the starting point and additional variables were added as either a strictly linear relationship or linear plus a threshold. The addition of variables followed a very similar order as the addition of variables in the MLR for Model Type 1 (Table E6-6a). An exhaustive search of the best additional variable was performed at each step to find the best combination of variables that resulted in the highest adjusted R^2 and normality of residuals.

The iterative search for the best parameter values tended to have difficulty converging when there were four or more linear variables in the threshold model. This was due to flexibility in the TOC threshold because of the data gap between 5 and 18 percent TOC. The complete stepwise process was attempted with several fixed thresholds; a higher threshold (TOC < 18 percent) gave a slightly better RSS, but did not always converge. Eighteen percent TOC may be too high a threshold, and there is no way to test it with the data. A TOC threshold of 10 percent is approximately the mid-point of the data gap and this fixed threshold gave comparable RSS results to those obtained for the 18 percent thresholds. The TOC threshold was fixed at 10 percent starting in Step 3 of the stepwise additions for Model Type 2.

The final RSS for the threshold model using all potential predictors was 0.041, and the adjusted R^2 was 0.96 (Table E6-6a). Residuals are not significantly different from normal (p=0.70 for correlation of the QQ-Plot shown in Figure E6-3), and variances appear homogeneous over the range of predicted values (Figure E6-3).

The predicted values for the two final models of types 1 and 2 derived using all predictor variables are shown in Figure E6-4. The observed vs. predicted results for AFDW indicate very good fits for both models over the entire range of growth. The bottom plot in Figure E6-4 compares the predicted values for the two models, and they are very similar. Both models have similar adjusted R^2 values and their AIC values differ by only 2 units. This indicates that while the threshold model has a slightly better fit than the MLR, the improvement is not substantial and either could be used for comparable and reliable predictions of AFDW of *C tentans*. The coefficients for the two final models based on the full set of predictor variables are shown in Tables E6-7a and E6-8a.

Both of these two final models include arsenic, cobalt, and silver which have low detection frequencies (≤ 65 percent) and/or high detection limits in the chemistry-only data set (for which growth will be predicted). Consequently, both model types were refit excluding these three variables from the potential list of predictor variables. These results are shown in Tables E6-5b and E6-6b for the MLR and threshold model, respectively. The R^2 for the MLR based on the short list of variables is 0.93 (Table E6-5b) and for the threshold model is 0.92 (Table E6-6b). The plots of residuals are shown in Figure E6-5 (both sets of residuals are not significantly different from normal, $p = 0.59$ and 0.54, for MLR and threshold model, respectively). The relationships among the observed and predicted values are shown in Figure E6-6. Once again, the fits are similar, though the adjusted R^2 and AIC show a slight preference for the MLR model. The coefficients for the two final models based on the short list of predictor variables are shown in Tables E6-7b and E6-8b.

5.1.4 Conclusions

Any of the four final models could be used for predictions of growth. For the full list of potential predictors, the threshold model (Table E6-8a) is slightly better than the MLR; for the short list of potential predictors, the MLR (Table E6-7b) is slightly better than the threshold model. The presence of nondetects and multicollinearity in the data set used warrant some special considerations when predictions are made:

- The use of one of the short list models (Tables E6-7b and E6-8b) would avoid uncertainties associated with using substitution values for metals with high degrees of censoring in the chemistry-only data set (i.e., arsenic, cobalt, and silver). Because none of these metals are chemicals of concern, use of one of the short list models will minimize uncertainty in predictions. Using the full list models (Tables E6-5a and E6-6a) to predict AFDW for stations in the chemistry-only data set should be bounded by making predictions using a substitution value for arsenic, silver, and cobalt at a low minimum value (e.g., half the minimum detection limit, or less) and at the reported detection limit. Results of the two runs would bound the growth predictions resulting from the uncertainty associated with the censored data.

- Multicollinearity is correlation among the independent variables, a condition that is present in this data set. Also, the accuracy of model predictions assumes that the same multicollinearity is present in the prediction data set as was present in the data set on which the model was based. The PCA summarized in section 3.2 of this appendix and discussed in the second half of Attachment E6A suggests that overall the patterns are similar between the chemistry-only and the model data set. However, predictions of growth for individual stations that deviate from the general chemical patterns will be more uncertain than those conforming to the general pattern.

Finally, because of the presence of multicollinearity in the data set, it is not possible to state the relative importance of the individual predictors in each model. Nevertheless, chemicals that were never included among predictors in this analysis can be considered to have a very low probability of being the cause of observed effects. These include total PCDD and total PCDF and several metals (aluminum, antimony, beryllium, cadmium, copper, iron, magnesium, mercury, selenium, vanadium and zinc). Conversely, this analysis indicates that arsenic, barium, chromium, cobalt, lead, manganese, nickel, silver, LPAH, HPAH, and TOC all are potential contributors to the observed growth effects. Negative coefficients for arsenic, barium, chromium, lead, LPAH, HPAH, and TOC indicate that these chemicals correlate negatively with growth.

6 COMPARISON OF MLR PREDICTOR VARIABLES AND VARIABLES CORRELATED WITH GROWTH TO SQGS

Literature-based sediment screening level guidelines (SQGs) are listed in Appendix E1 of the risk assessment report, and are applied to the initial evaluation of sediment chemistry to select COPECs for the ecological risk assessment. These were selected for application in the risk assessment according to a hierarchy of SQG sources established by the RAWP for the St. Regis Site (USEPA 2004). These values are generally conservative, denoting concentrations of chemicals at or below which adverse effects to benthic organisms are not expected to occur.

The sediment constituents that are effective predictors of growth in one or more of the regression models are a subset of the metals (arsenic, barium, chromium, cobalt, lead, manganese, nickel, silver), LPAH, and HPAH. TOC is also correlated with growth in three of the four models. Antimony, cadmium, copper, mercury, molybdenum, selenium, vanadium, thallium, zinc, and 2 methylnaphthalene are also significantly correlated with growth (Table E6-4). Those chemicals not correlated with growth are considered highly unlikely to be the cause of the observed pattern. The list of potential causes can be further reduced by comparing concentrations of each chemical predictor variable from the regression models and those individually correlated with growth to SQGs. Because SQGs are generally protective of benthic organisms, if a chemical constituent does not consistently exceed its SQG at stations with reduced growth, it is eliminated as a cause of the growth effect at that station.

Concentrations of these chemicals were compared to SQGs by computing a ratio of the chemical to the SQG, or hazard quotient. For chemicals that exceed SQGs at one or more tested stations, additional factors are considered to support decisions about whether to retain a chemical as a potential cause of the observed growth effect: consistent exceedances of SQGs among stations and the magnitude of exceedances (hazard quotients). Whether the constituent was determined to be within the range of concentrations in sediments from background reference areas (Appendix B) is also noted.

6.1.1 Results

Ratios for those chemicals that exceeded SQGs in one or more bioassay stations are listed in Table E6-9 (sorted by station) and Table E6-10 (sorted by chemical); chemicals not exceeding an SQG in any bioassay station are not included (i.e., chromium, cobalt, nickel, and vanadium). there are no SQGs for molybdenum, thallium, and 2-methylnaphthalene. For any given chemical, hazard quotients are sorted, and the result of the statistical comparison with reference (Section 5.4) is indicated.

Two metals, arsenic and barium, have several exceedances of SQGs among bioassay stations, but concentrations of these metals in Fox Creek are not different from background. Among the four stations with the highest hazard quotients for barium, two were associated with significantly

reduced growth, one was a reference station, and the station with the highest barium concentration had no growth effect. For arsenic, which was also not elevated above background, the maximum hazard quotient is 1.4, further suggesting that this metal is unlikely to be an important cause of the observed effects. Selenium also exceeded its SQG at 15 stations,[1] including 2 reference stations, with the one of the highest exceedance factors in a reference sediment (SDREF-0403). Manganese exceeded its SQG at three stations, and all other metals exceed at two or fewer stations. Manganese was below background in Fox Creek sediment. For all but selenium, the pattern of exceedances is not sufficiently strong or consistently associated with stations showing growth effects to suggest that any individual metal is consistently causing reduced growth. The highest selenium exceedances were consistently associated with reduced growth; lower SQG exceedances (HQ ≤ 10) were consistently associated with an absence of statistically significant reduced growth.

HPAH and LPAH exceed SQGs at 13 and 10 stations, respectively. Among the five stations with the highest hazard quotients for each, one reference station is represented, and only one of the remaining stations with an LPAH or HPAH exceedance was associated with a significantly reduced growth relative to reference (Table E6-10).

6.1.2 Conclusion

The comparison of concentrations of chemicals that are correlated with growth to SQGs showed that concentrations of individual chemicals other than selenium either did not exceed, or did not consistently exceed SQGs, at stations where significant growth effects were observed. Conversely, many of the individual SQG exceedances for chemicals other than selenium were at locations without significant growth effects. Therefore, among all the individual chemical parameters, only selenium was identified as a potential cause, in and of itself, of the observed growth effects based on comparisons to SQGs. Although selenium was relatively elevated in one reference station sediment, it was generally elevated in stations with growth effects; and exceedances of its SQG were relatively low at non-toxic stations.

Three chemicals that are correlated with growth on the basis of Kendall's tau-b (Table E6-4) were excluded from this analysis because they lack screening values: molybdenum, thallium, and 2-methylnaphthalene. Along with selenium and TOC, these chemicals cannot be eliminated as potential causes of the observed growth effect using the method described in this section.

[1] While selenium was retained as a COPEC for analysis of risks to birds and mammals, comparisons to background were somewhat equivocal Due to low detection frequencies, values had to be estimated to facilitate statistical comparisons with reference creek sediments. The quantile test detected no significant difference between selenium in Fox Creek and selenium in reference creek sediments, while the Mann-Whitney *U* tests did detect a difference. See Appendix B for detail.

7 NUTRITIONAL QUALITY OF ORGANIC MATTER

The simple correlation analyses using Kendall's tau-b, the multilinear regression analyses, and comparison of the best correlates from these analyses to SQGs do not identify a single sediment parameter, or consistent combination of parameters, as the cause of the observed differences in midge growth in Site sediments relative to that in reference sediment. TOC is elevated (> 18 percent) in all nine stations showing significant growth effects relative to reference, while it exceeds 18 percent in only three of the 18 stations without significant effects. If the cause of the poor growth relative to reference in bioassay chambers is attributable primarily to natural TOC, then this cause-effect relationship cannot be eliminated by risk management actions.

To help isolate the effect of the TOC itself from the effects of the remaining chemicals on midge growth, International Paper submitted 8 of the 27 sediment samples used in toxicity tests for analysis of nitrogen (N) compounds, phosphorus (P) compounds, and TOC, so that the concentrations of organic forms of N and P in natural sediments could be estimated. The food used in bioassays (Tetra Fin) was also analyzed for the same chemical parameters.

The following text describes a conceptual model to explain how very high TOC in sediment may affect the ability of midges in bioassay chambers to obtain high-quality diets, and the analysis of the abundances and nutritional quality of the natural and laboratory foods available to *C. tentans* while in the bioassay chambers. The quality of the midge diet relative to AFDW is analyzed. The results lead to a final refinement of the list of sediment constituents that may be the cause of the observed effect on growth.

7.1 CONCEPTUAL MODEL

A conceptual model to explain how high concentrations of natural TOC might result in impaired growth in bioassay organisms is as follows: Larval midges are thought to feed non-selectively, consuming organic matter in the immediate vicinity of their burrow. In the Cass Lake bioassays, midges in all bioassay chambers were administered a fixed amount of food (i.e., the fish food Tetra Fin) during the test. A midge living in a bioassay chamber with low (or very low) natural TOC is more likely to find and consume particles of Tetra Fin than other organic carbon sources and thus receives a consistently high-quality diet. Midges living in bioassay chambers with increasingly high concentrations of natural TOC are increasingly unlikely to find and consume the Tetra Fin in the mix of available organic matter, and as a result, a larger proportion of their diet consists of natural TOC. If natural TOC is a lower-quality food source, the dilution of high quality food (Tetra Fin) by increasing amounts of low-quality food (natural organic matter; NOM) would explain why reduction in growth of midges is strongly correlated to high TOC in sediments.

Based on data for C, N, and P for fish food and for natural sediments in the literature (Vos et al. 2000; Vos et al. 2002), Integral's hypothesis is that the natural TOC in Fox Creek sediments provides a substantially poorer food source for midges in bioassay chambers than does Tetra Fin. If it is demonstrated that growth is correlated with measures of the quality of the midge diet, then multivariate analysis can be used to evaluate the role, if any, that other chemical parameters may play in the pattern of midge growth.

7.1.1 Chemical Analyses

Eight sediment samples from Fox Creek that have been stored frozen since September 2004 were submitted to Columbia Analytical Services for analysis of nutrient parameters (Table E6-11). Samples were selected to represent the range of TOC concentrations and midge responses (as AFDW in mg at the end of the test). Among the selected stations were two reference stations and stations at which growth was significantly different and not different from growth in reference sediment. Not all of the Fox Creek sediment samples were included because it is likely that all of the sediments have similar C/N, N/P, and C/P ratios (Vos et al. 2002).Two of these samples were analyzed in triplicate to help characterize the intra-sample variability; one additional sample was analyzed for P twice for QA purposes.

Table E6-11. Sediment Samples Analyzed for Nutrients; Results of *C. tentans* Growth Test and Percent Organic Carbon Measured in 2004.

Sediment Sample ID	AFDW mg	%TOC (2004)
SR-SDFC-0403	0.43[b]	26.6
SR-SDFC-0404	1.03	0 74
SR-SDFC-0406[a]	0.5[b]	42.8
SR-SDFC-0408[a]	0.69[b]	18.3
SR-SDFC-0410	0.66[b]	37.6
SR-SDFC-0412	0.45[b]	31
SR-SDFC-REF-0401	0.99	1.62
SR-SDFC-REF-0403	0.86	21

[a]Sample analyzed in triplicate
[b]Growth is significantly different than reference

A sample of Tetra Fin was also analyzed. Analytes for all samples included:

- TOC
- Ammonia N
- Kjeldahl N
- Total P
- Hydrolyzable P

7.1.2 Data Analyses and Assumptions

Results of the analysis of nutrients in sediment were analyzed in several steps. To estimate the fractions of organic nitrogen (ON) and organic phosphorus (OP):

- ON (mg/kg) = Kjeldahl N (mg/kg) – ammonia N (mg/kg)
- OP (mg/kg) = total P (mg/kg) – hydrolyzable P (mg/kg)

Because each bioassay chamber contained a different amount of Tetra Fin relative to NOM, several steps were taken to normalize the metric of food quality to represent the diet of the bioassay organisms and not just the quality of the sediment alone. To characterize the quality of the diet (NOM + Tetra Fin):

- The dry mass of sediment in each chamber was estimated.
    - 100 mL of sediment and water was added directly from the sediment sample to each bioassay chamber. Dry mass of sediment (g) for each sample was calculated from the percent solids data for these samples reported by Barr (2004).[2]
- The mass of dry NOM was calculated: dry sediment (g) * (%TOC) = dry NOM (g)[3]
- The proportion of all organic matter (OM) consisting of Tetra Fin was estimated for each bioassay chamber, assuming the mass of food added to each bioassay chamber was 0.006 g/day (2004):
    - Percent of OM consisting of Tetra Fin = 0.006 g food/[(NOM (g)/100)+ 0.006 g food]
- Nutritional quality of the midge diets and nutrient ratios were calculated by weighting the values for ON and OP by the fraction of the food source from which the nutrient is derived. For example:
    - Dietary N/P = [(%Tetra Fin*N_{TF})+(%NOM*N_{NOM})]/[(%Tetra Fin*P_{TF}) +(%NOM*P_{NOM}]

This diet normalization of nutrients then represents the quality of the food in the gut of the individual midge in the bioassay chamber. The analysis requires the following assumptions:

- Midges feed randomly on the sediment surface
- Food is incompletely mixed in the bioassay chamber. Calculation of the percent of organic matter consisting of Tetra Fin divides NOM by 100 before adding NOM to Tetra Fin. This adjustment is arbitrary, but is used to reflect the assumption that Tetra Fin is

[2] Although total solids was also measured in 2007, the 2004 value was selected to better represent conditions in bioassay chambers, since some water may have evaporated during storage

[3] TOC used in this and subsequent calculations is the average of TOC measured in 2004 and 2007. The average was assumed to best represent the actual condition, i e., 2004 and 2007 were treated as replicates, because no TOC would have been lost or altered as a result of sample storage.

not mixed into sediment, but is deposited on the surface. The value of 100 means that 1/100 of the dry mass of sediment is available to the animal feeding on the surface, and that the remaining 99/100 of sediment is not "available" to a surface feeder.

- All Tetra Fin is consumed each day and has no nutrient value after it is digested and excreted.

7.1.3 Nutritional Quality of Sediment

Results of nutrient analyses for sediments at each station and for Tetra Fin are summarized in Table E6-12. Tetra Fin contained levels of TOC comparable to the highest levels in sediment stations (Figure E6-7), and contained higher concentrations of organic nitrogen and organic phosphorus than any sediment stations (Figure E6-7). Nutrient ratios demonstrate that Tetra Fin had the lowest (most favorable) C/N ratio (Table E6-12, Figure E6-7). Phosphorus ratios (C/P) were lowest (most favorable) at sediment stations SDFC-0404 and SDREF-0401, which also had the highest values for AFDW (Table E6-12; Figure E6-8). Tetra Fin exhibited the next most favorable phosphorus ratios. Figure E6-8 highlights the low phosphorus value of Station SDFC-0410 (5 mg/kg). Reasons for this very low OP value are unclear. Subsequent analyses of these ratios were run with and without Station SDFC-0410; although the magnitude of some results changed, the direction of the results never did. These results support the hypothesis that Tetra Fin is consistently of higher nutrient quality, as defined by low C/N, N/P, and C/P ratios, than Fox Creek sediment.

7.1.4 Nutritional Quality of Diet

In the conceptual model, the diet of midges in the bioassay chamber consists of Tetra Fin and NOM in proportion to their availability at the surface of the sediment in the bioassay chamber. Normalization of the nutrient data to the diet accounts for nutrient content of both Tetra Fin and NOM, and dietary nutrient ratios represent the quality of the gut contents of a test larva. The nutrient ratios of the midge diet are presented in Table E6-12.

Dietary nutrient ratios for the eight stations were plotted against the corresponding *C. tentans* growth results (Figure E6-9). Consistent with the hypothesis that nutrient quality affects *C. tentans* growth in Fox Creek sediments, all dietary nutrient ratios (C/N, N/P, and C/P) exhibited negative relationships with AFDW. The results indicate that larval growth is affected by diet quality.

The inverse of the carbon-related dietary ratios (i.e. C/N to N/C, and C/P to P/C) reflect the nutritional quality of the diet, but also control for organic carbon as a potential growth factor, which is useful because of the consistent correlations between OC and other chemical parameters. Plots of growth as a function of N/C and P/C indicate good positive correlations (R^2 = 0.69 and R^2 = 0.61, respectively; Figure E6-10), strongly suggesting that growth is linked to the

quality of the diet. The variables that describe the quality of the diet, N and P, explain 69 and 61 percent of the variation in growth, respectively, when the effect of organic carbon is controlled.

Subsequent analytical steps evaluate the possibility that chemical constituents of sediment explain the remaining fraction of the variation in growth among these eight samples.

7.1.5 Correlation Matrix and MLR Analysis

To investigate whether chemical constituents in sediment can help explain the rest of the variance in the growth data, correlations between each chemical or chemical group (see Attachment E6A) and the indicators of diet quality, dietary N/C and P/C, for the eight sediment samples were evaluated;

- A significant negative correlation of any individual chemical with N/C or P/C reflects a pattern in which decreases in the chemical with increases in diet quality act together to allow for better growth
- A significant positive correlation between diet quality (N/C or P/C) and any of the chemical constituents would suggest that the chemical constituent is acting antagonistically to the nutrient effect, potentially negatively affecting growth as nutritional quality increases, and thereby confounding the interpretation of nutrient effects on growth.

Analyses of correlations between each of the two ratios best correlated with growth, dietary N/C and dietary P/C, and sediment constituents (total PCDD, total PCDF, all the metals, HPAH, LPAH and total PCBs (sum of Aroclors) with Kendall's tau-b indicate that a significant and positive correlation occurred only between HPAH (and several HPAH compounds individually) and P/C (Table E6-13); no other chemicals were significantly positively correlated with either dietary N/C or P/C. Those that are negatively correlated include several metals. This negative correlation indicates that increases in nutritional quality and decreases in these metals are acting in the same way (i.e., that as nutritional quality of the diet increases, metals decrease). Because growth increases as P/C increases (Figure E6-10), a decrease in metals with increased diet quality (P/C) suggests that these metals are not acting in conflict with the benefit of a high quality diet. In contrast, the positive correlation of HPAH with P/C suggests that HPAH could work against increases in nutrient quality in affecting growth. Adding HPAH to the linear model with P/C represented in Figure E6-10 could improve growth predictions.

To test the hypothesis that HPAH, when combined with information on the quality of the diet, improves the ability to predict growth at the eight stations for which nutrient data are available, an MLR was conducted. This allows a determination of the degree to which accounting for HPAH improves a model that uses P/C to predict growth. HPAH data were $\log_{10}$- transformed to satisfy the assumptions of the regression analysis. Type III sum of squares calculations were used to assess model effects, because neither HPAH nor dietary P/C were presumed to have stronger effects at the outset.

The regression results demonstrated that dietary P/C and $\log_{10}$-transformed HPAH together had significant effects on growth (ANOVA: p=0.008,Table E6-14), and the resulting model had an R^2 = 0.86 and provides a better fit to the data than the model with only P/C (AIC of –8.8 vs. –2.9). Examination of the Type III sum of squares results (Table E6-14) shows that both dietary P/C and $\log_{10}$-transformed HPAH had significant individual effects on growth (p=0.004 and 0.03, respectively). Dietary P/C, or the quality of the diet, had a strong, positive effect on growth of *C. tentans* (β =1578), whereas $\log_{10}$-transformed HPAH had a much weaker, negative effect on growth (β = –0.379). The results of the regression model are summarized in Table E6-15.

7.1.6 Conclusions

Concentrations of ON and OP in Tetra Fin were higher than in natural sediment, indicating that Tetra Fin provides a higher quality diet to *C. tentans* than NOM. Assuming that *C. tentans* feeds randomly on the surface of the sediment within a bioassay chamber, the relative abundances of each of these two food sources in the diet of an individual midge can be estimated according to the proportions of each food type in the available organic matter, and midge growth can be compared to the overall quality of the midge diet. When the quality of the diet is standardized to organic carbon, which is itself correlated with several other chemicals, metrics of diet quality explain a large fraction of the variability in the growth data (61 to 69 percent) within the eight stations evaluated, but not all of it. The possibility that other chemical constituents in sediment could improve predictions of growth based on diet quality was evaluated by quantifying correlations of dietary N/C and P/C with chemical constituents. No chemicals were found to correlate positively and significantly with the best nutritional predictor (N/C); HPAH (and all the individual HPAH compounds) was the only constituent positively correlated with dietary P/C. Subsequent MLR resulted in a significant model with which AFDW is predicted by the combination of dietary P/C and HPAH concentration. The result shows that the quality of the diet represented by dietary P/C has a strong, positive influence on midge growth, and HPAH has a statistically significant but relatively minor antagonistic effect at the eight stations included in the nutrient analysis. Because diet quality depends on the amount of sediment TOC, TOC is negatively correlated with dietary P/C as a measure of diet quality (Table E6-13), and P/C has a greater influence on growth than any antagonistic chemical, this analysis indicates that the effect of TOC on midge growth is greater than that of COPECs.

8 LITERATURE REVIEW

In the absence of chemical stressors, several sediment characteristics alone or in combination, affect growth in *C tentans* and in the other midge commonly used for toxicity bioassays, *C. riparius*: food quality, sediment grain size, and TOC content of sediments (e.g., ASTM 2001; Lacey et al. 1999). No published studies have examined the effect of TOC at the levels observed in the Fox Creek and channel sediments while holding the other factors constant. Nevertheless, taken together, the literature supports the premise that the very high TOC in sediment from the St. Regis site is an important driver of reduced growth relative to growth in sediments from reference areas under laboratory test conditions.

Several authors have investigated the role of high organic carbon in reducing the growth of chironomids in laboratory bioassays (Lacey et al. 1999; Ristola et al. 1999). Ristola et al. (1999) examined *C. riparius* bioassay endpoints based on four clean sediments with varying physical properties under a range of feeding levels. TOC levels in the samples were 0.5, 2.3, 6.7, and 57.2 percent. Growth was generally depressed at low feeding levels, but when feeding levels were increased, differences in growth within a feeding regimen among samples could be detected, with the highest growth rates associated with the lowest TOC content at the second-highest feeding level (0.12 mg/larva-day), and the highest TOC sediment having the lowest growth at the two highest feeding levels. However, the lowest TOC sediment also had the lowest percent fine-grained sediments, and the highest C/N ratio, suggesting that both nutritional quality and sediment grain size could have played important roles in determining growth. Although they did not find that very high TOC had a negative effect on growth, Ristola et al. (1999) concluded that "effects of sediment physicochemical characteristics on larval growth and development cannot be totally compensated for by food addition... therefore, we suggest use of more than one reference sediment with physicochemical characteristics (e.g., particle size distribution, organic content) covering the range measured in test sediments so that the background variation in the response can be evaluated."

Lacey et al. (1999) reported relationships between TOC and *C. tentans* growth using four different types of TOC: peat moss, alpha-cellulose, leaves, and natural sediment. In this experiment, all treatments received laboratory food (Tetra Fin) each day. Leaves and alpha-cellulose, and to a lesser extent peat moss, show a bell curve of growth response as a function of TOC content, with higher growth rates towards the middle of the curve, at approximately 5 percent TOC, dropping off substantially at 10 percent TOC for leaves. Natural pond sediments with up to 25 percent TOC showed no significant difference in growth among different TOC treatments, up to 25 percent TOC.

Lacey et al (1999) provide a helpful examination of the factors affecting their results. In a simpler comparison of the growth of *C tentans* in different types of organic matter with all treatments at approximately 10 percent TOC, results described above for the first experiment were reversed: growth in sediment augmented with peat moss was less than growth with leaves

or natural sediments at the same TOC level. In the dilution series growth was highest in sediment augmented with peat moss at 10 percent TOC. Lacey et al. (1999) concludes that the quality of the organic matter is at least as significant as quantity in determining growth of *C. tentans*.

Another possible explanation for the effect seen in the various dilution experiments is the differences in the quality of food available, although Lacey et al. (1999) do not measure food quality. In a feeding experiment, Vos et al. (2000) fed *C. riparius* one of the following 10 food formulations: two treatments with laboratory fish food (including Tetra Fin), two treatments with animal based foods (ground *Gammarus* or *Chaoborus* tissue), three treatments with aquatic plant-based foods (ground *Ceratophyllum, Potamogeton,* or *Utricularia* tissue), one treatment with leaves of a terrestrial plant (*Populus*), one treatment with algae (*Scenedesmus*), and one with yeast. Growth was measured as length after 1 week. The four vascular plant foods (aquatic plants and *Populus* leaves) generated the worst growth in *C riparius*. *C riparius* grew best on foods high in carbon, nitrogen, phosphorus, and lipids. Vos et al. (2000) conclude that these results are consistent with the biological uses of food: carbohydrate rich foods provide energy, while foods high in protein and lipid supply materials essential to growth. They argue that, at high food levels, the quality of food is the limiting factor for growth.

9 SUMMARY

In site-specific sediment bioassays conducted with *C. tentans* in 2004, a reduction in growth relative to reference was observed in nine test stations in Fox Creek. Understanding the cause is important because, if the natural sediment physicochemistry is the cause of the observed effect in bioassay chambers then the effect cannot be changed or mitigated by remedial actions such as sediment capping or removal. The growth of midges (as AFDW) in the 27 sediment samples used for toxicity tests is strongly and inversely associated with the TOC content of sediment, and the TOC is also associated with several grain size parameters and numerous chemicals. These correlations confound efforts to understand the underlying causes of the observed pattern in midge growth.

A series of quantitative analyses was undertaken to evaluate the role of TOC and other sediment constituents in the growth pattern. Results are summarized in Table E6-16, which lists the sediment constituents identified by each of the lines of evidence as possible causes of the observed effect on growth. Correlations between chemicals and toxicity test results, and stepwise linear regression of growth against chemical constituents in sediment, refined the list of possible causes and predictive factors, primarily by eliminating from further consideration chemicals that did not help predict the growth effect.

Comparison of the chemicals that were among the good predictors of growth in the MLR models or were individually correlated with growth to conservative sediment screening values (SQGs) further narrowed the list of potential causes of toxicity (Table E6-16), leaving only selenium as a consistent potential cause, in and of itself, of the observed pattern in growth, based on the comparison to SQGs.

Because concentrations of many chemicals were closely tied to TOC in sediment, the quality of TOC as a food source for the midges was evaluated by measuring the organic forms of phosphorus and nitrogen in eight of the sediment samples tested for toxicity. This analysis provides an independent line of inquiry about the cause of the pattern in growth. The diet of a midge in a bioassay chamber was assumed to consist of both NOM and the food given to them in the laboratory (Tetra Fin), and the proportions of each food in the diets of midges was quantified. Patterns of growth were evaluated relative to the nutritional quality of the diet, and both N/C and P/C were found to be good, but not perfect, predictors of growth in the eight sediment samples. HPAH (and several HPAH compounds individually) was found to be the only COPEC with a significant positive correlation with a metric of the nutritional quality of the diet (dietary P/C), and, based on linear regression analysis, it appears that HPAH exerts a small effect antagonistic to nutrient quality in determining growth.

MLR confirmed that nutritional quality (as P/C) provides a strong positive influence on growth of midges, and HPAH has a weak antagonistic effect on growth. This line of evidence is independent of the stepwise linear regression modeling and comparison of resulting predictors to SQGs (Table E6-16). By comparing concentrations of chemicals at all bioassay stations to

SQGs, however, it is evident that growth at four of the five bioassay stations with the highest HPAH values was not impaired relative to reference, and several stations with normal growth have concentrations of HPAH in excess of SQGs. The lack of a consistent pattern of decreases in growth with increasing HPAH strongly suggests that HPAH is not a consistent causal factor in the observed growth effects.

10 CONCLUSIONS

The analyses described in this appendix demonstrate that the relatively elevated fractions of natural TOC in sediments of Fox Creek play a primary role in causing reductions in growth in bioassay chambers, and that selenium is the only chemical that exceeds SQGs in a pattern consistent with the observed growth effect. From the analysis of correlations among sediment chemistry and growth, it is clear that the very high fractions of TOC in the sediments from Fox Creek play a role in the reduced growth of *C. tentans* in nine bioassay stations. Determination of the quality of the midge diet and correlation of diet quality with growth at eight representative stations suggests a mechanism by which very high natural TOC in sediment may impair midge growth relative to reference: individual midges, when living in a bioassay chamber containing very high quantities of natural organic matter (which is a relatively poor food source) obtain a diet that is low in the nutrients required for growth. Although they are able to survive on the high-TOC diet, they do not obtain the higher quality nutrients needed to add biomass. This is consistent with studies by Vos et al. (2000; 2002) in which diets high in lipids (phosphorus-based compounds) and protein (nitrogen-rich compounds) support higher and faster growth in *C riparius*. This is also consistent with observations in the bioassays, which included no significant mortality among any bioassay organisms.

Chemical constituents also may play a role, but the extent to which each contributes to the effect on growth is not clear, and cannot be determined with the available data. Barium was consistently elevated above SQGs in stations with reduced midge growth, but was also elevated above SQGs in stations that were nontoxic and was not elevated in Fox Creek sediments relative to reference creek sediments. Selenium was also consistently elevated above SQGs in stations with growth effects, and comparisons of selenium to background were equivocal (e.g., SDREF-0403 has one of the higher selenium concentrations). Three chemicals cannot be ruled out using this line of inquiry because they lack SQGs: molybdenum (which is closely correlated with TOC as well as with growth), thallium, and 2-methylnaphthalene.

Evaluation of the quality of the diet relative to midge growth in a subset of the bioassay stations (N=8) indicates that P/C and N/C individually explain 61 and 69 percent of the variance in growth, respectively. A multiple linear regression model to predict growth based on nutritional quality (dietary P/C) and HPAH (which correlated positively and significantly with dietary P/C) suggests that HPAH plays a small role relative to nutrient quality in growth limitation. Exceedances of SQGs for HPAH were not consistently associated with stations at which growth was impaired, and four of the highest five exceedances were at stations with no growth impairment (Table E6-10). Therefore, although the MLR with dietary P/C and HPAH is significant, the lack of a consistent pattern of HPAH exceedance of SQGs supports the conclusion that HPAH is not a major causal agent in the growth effect.

Processes of elimination allow the majority of chemicals to be considered unlikely to be the cause observed effects on growth with a high degree of confidence (Table E6-16). Selenium is the only

chemical that exceeds SQGs in a pattern roughly consistent with observed growth effects. Three chemicals with no SQG values (molybdenum, thallium, and 2-methylnaphthalene) also cannot be dismissed from further consideration. Among all the analyses, the best individual predictors of growth were two metrics of the nutritional quality of midge diets, N/C and P/C, which explained 69 and 61 percent of the variability in growth, respectively, and chromium (Tables E6-5a and E6-5b), which did not exceed its SQG in any sediment sample. Neither selenium nor chromium is associated with wood processing activities formerly conducted at the Site. Because concentrations of many other chemical parameters in sediments correlate with TOC, it cannot be stated conclusively that any one sediment constituent is the individual cause of the observed pattern in growth. However, the weight of evidence strongly suggests that elevated TOC, because it provides a poor source of nutrients and reduces the overall quality of the diet to midges in bioassay chambers, is the strongest single factor in causing the observed growth pattern in midges.

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FIGURES

Legend

Sediment Sampling Locations

(by sample type)

- Chemistry Only
- Bioassay
- Bioassay - with Nutrient Analysis
- Bioassay - with Significant Growth Effect (SGE)
- Bioassay - with Nutrient Analysis and SGE

- Channel
- Fox Creek
- Limit of Former Operations
- Southwest Area
- City Dump Area
- 'Area A'
- 'Area B'

Area A

Area B

Pike Bay

HWY-01
HWY-02
WL-03
WL-01
SDWR-0402
SDWR-0403
HWY-03
SDCH-0401
WL-02
RR-01
SDCH-0402
RR-02
SDWR-0401
GP-10
RR-03
SDCH-0403
SDCH-0404
SDCH-0405
SDPB-0401
SDPB-0402
SDPB-0403
PB-A
PB-B
PB-C
PB-D
SDPB-0404
SDPB-0405
SDPB-0406
SDPB-0407
SDREF-0401
SDREF-0402
SDREF-0403
SDFC-0412
SDFC-0401
SDFC-0402
FCSW-01
FCSW-02
SDFC-0403
SDFC-0404
FCSW-03
GP-08
SDFC-0405
SDFC-0406
FCCD-01
SDFC-730002
FCCD-02
FCCD-03
SDFC-0407
SDFC-730003
FCCD-04
SDFC-730004
FCCD-05
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SDFC-730006
SDFC-0409
SDFC-0410
SDFC-730007
SDFC-730008
SDFC-730009
SDFC-730010
SDFC-0411
FCD-05
FCD-04
FCD-03
FCD-02
FCD-01
SDFC-730001

NOTE:
- the aerial imagery is from 2003, is courtesy of Barr Engineering, and is intended for reference purposes only

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Figure E6-1
Sediment Sampling Locations
St. Regis Paper Company Site, Cass Lake, MN

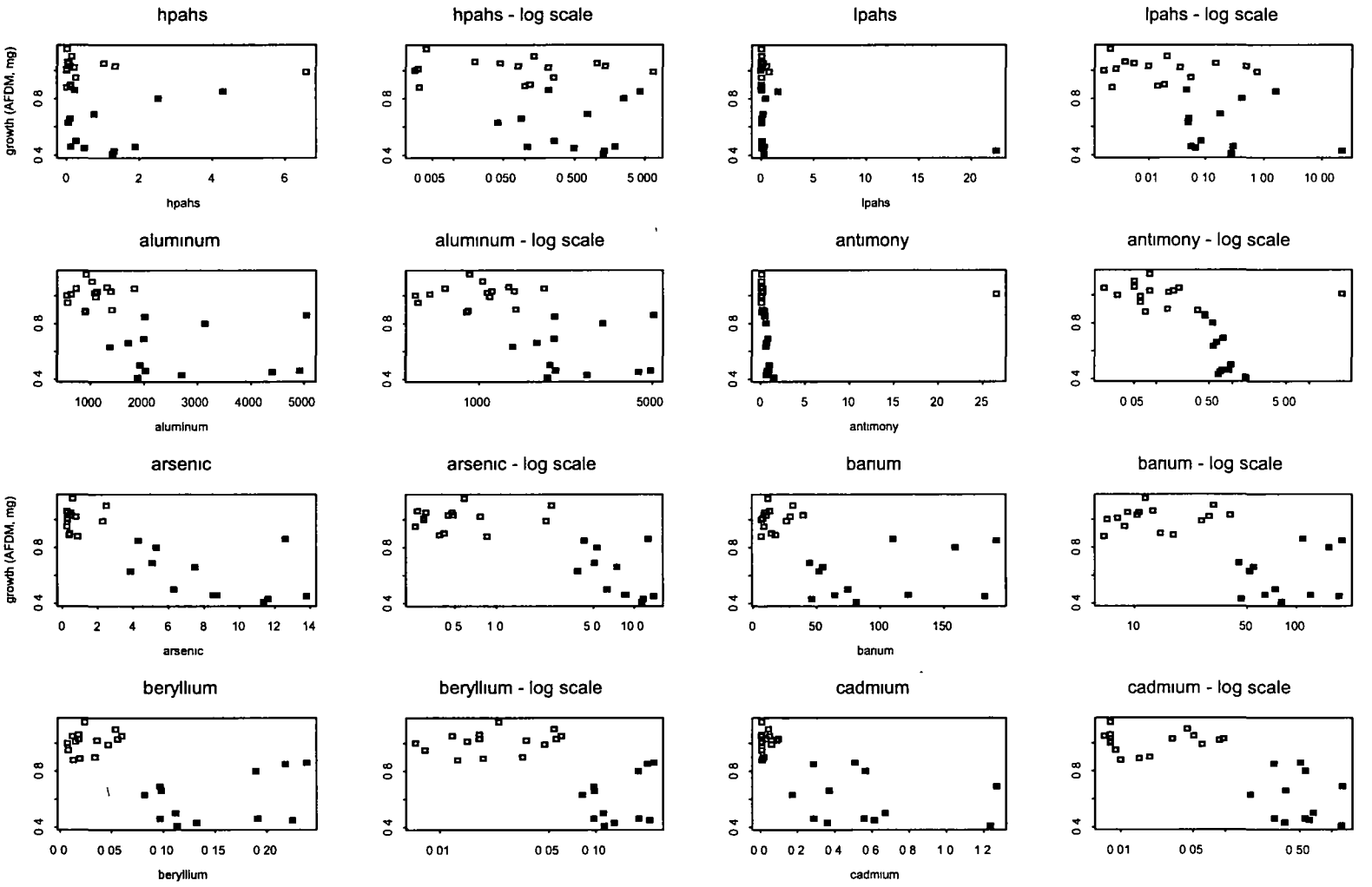


Figure E6-2. Growth (AFDM, mg) vs. Sediment Concentrations on Original Scale (columns 1 and 3) and log10 Scale (columns 2 and 4). Symbols on each plot indicate TOC level: open squares have TOC < 5%; filled squares have TOC > 18%

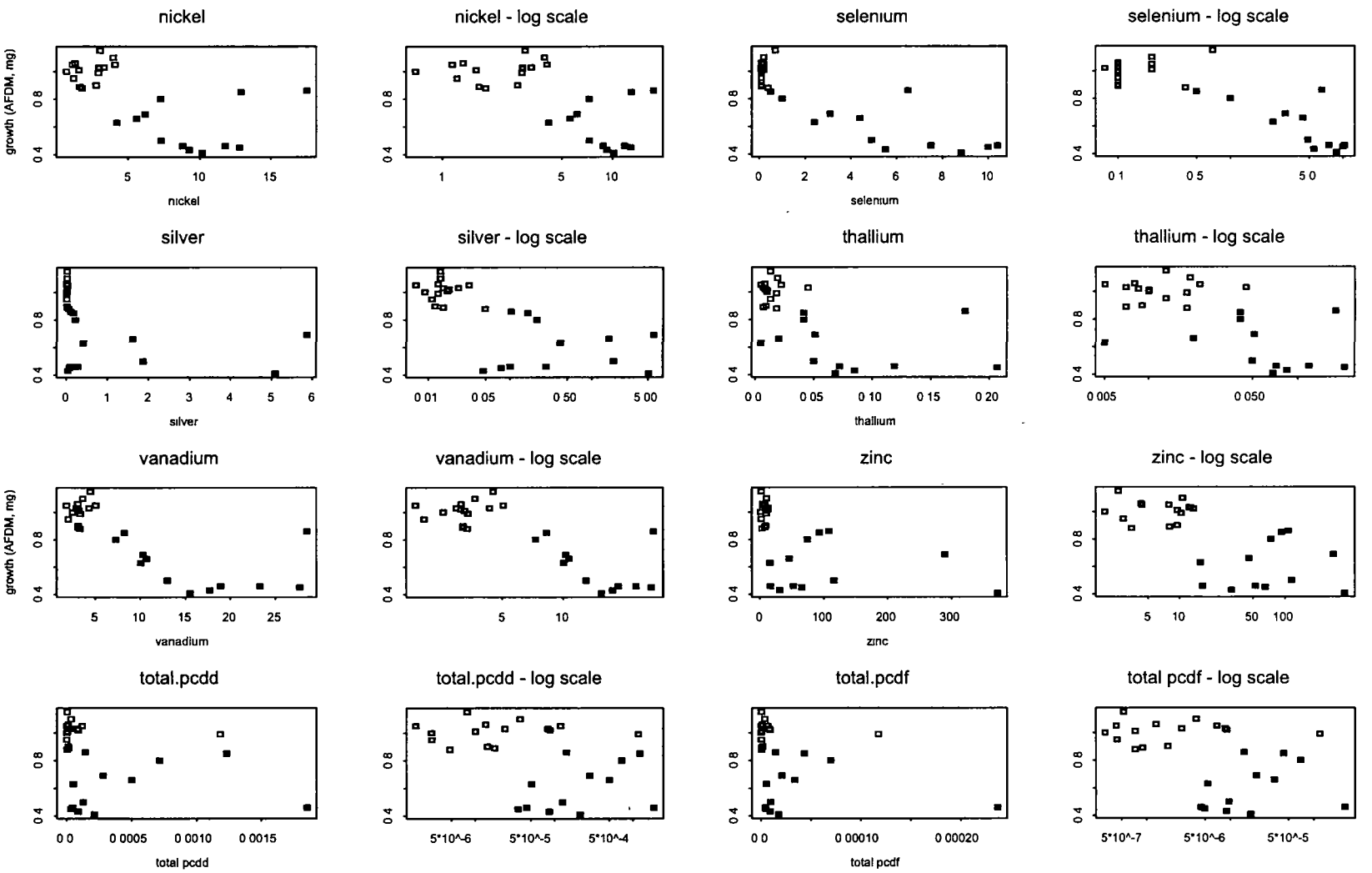


Figure E6-2 (continued). Growth (AFDM, mg) vs. Sediment Concentrations on Original Scale (columns 1 and 3) and log10 Scale (columns 2 and 4). Symbols on each plot indicate TOC level: open squares have TOC < 5%; filled squares have TOC > 18%

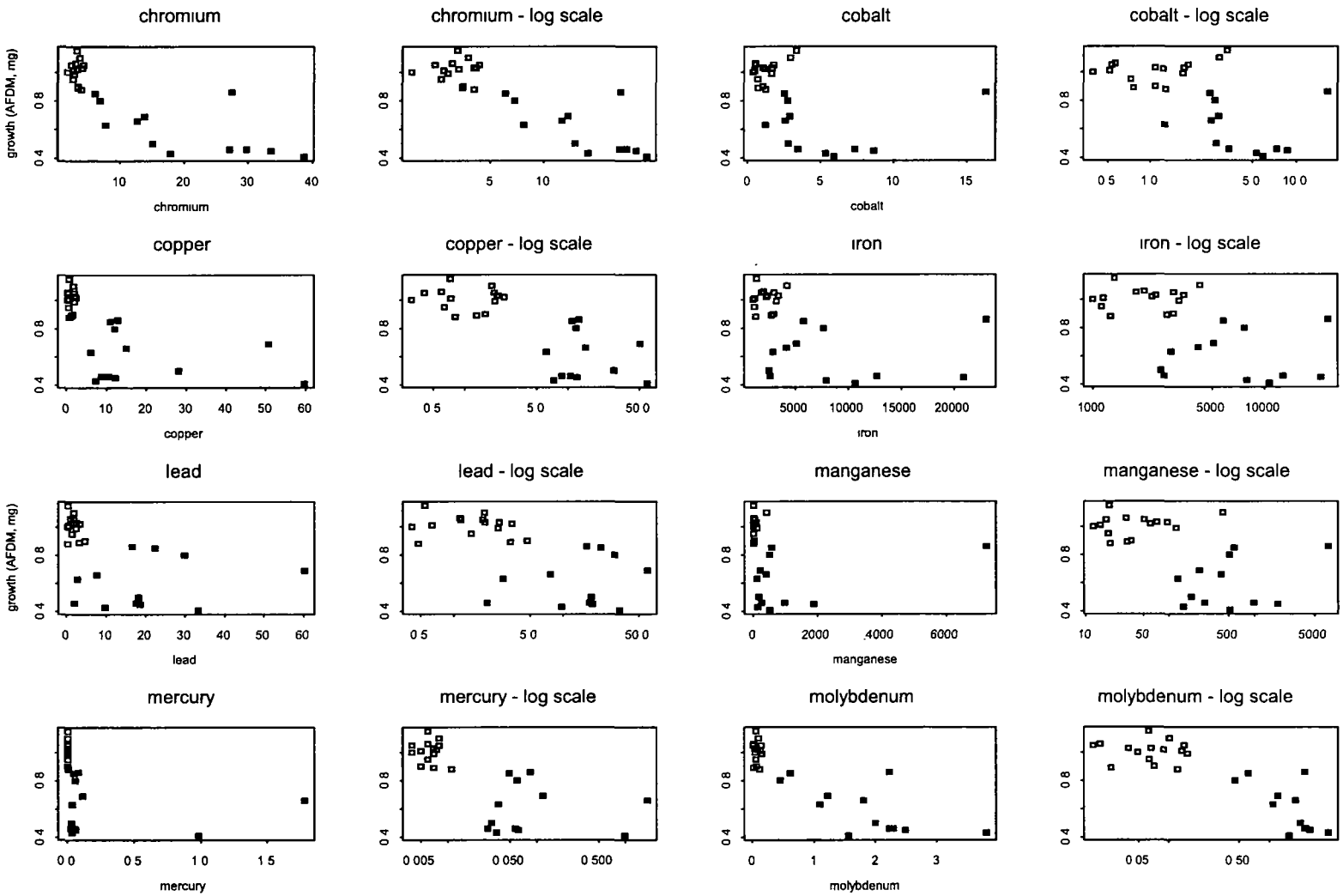


Figure E6-2 (continued). Growth (AFDM, mg) vs. Sediment Concentrations on Original Scale (columns 1 and 3) and log10 Scale (columns 2 and 4). Symbols on each plot indicate TOC level: open squares have TOC < 5%; filled squares have TOC > 18%

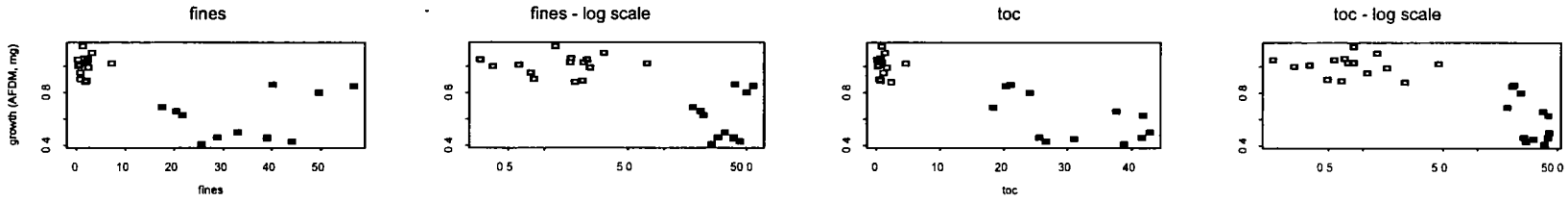


Figure E6-2 (continued). Growth (AFDM, mg) vs. Sediment Concentrations on Original Scale (columns 1 and 3) and log10 Scale (columns 2 and 4). Symbols on each plot indicate TOC level: open squares have TOC < 5%; filled squares have TOC > 18%.

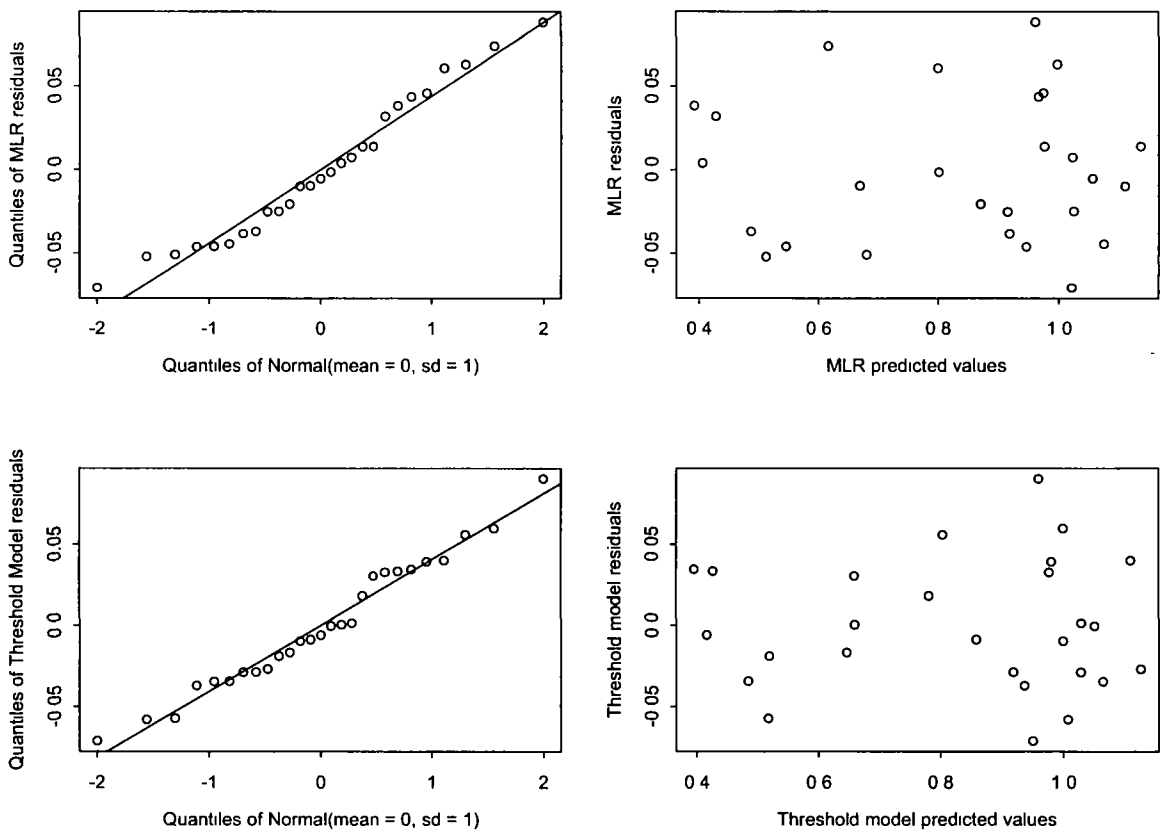


Figure E6-3. Residuals Plots for the Final Multiple Linear Regression (MLR) Model and Threshold Model.

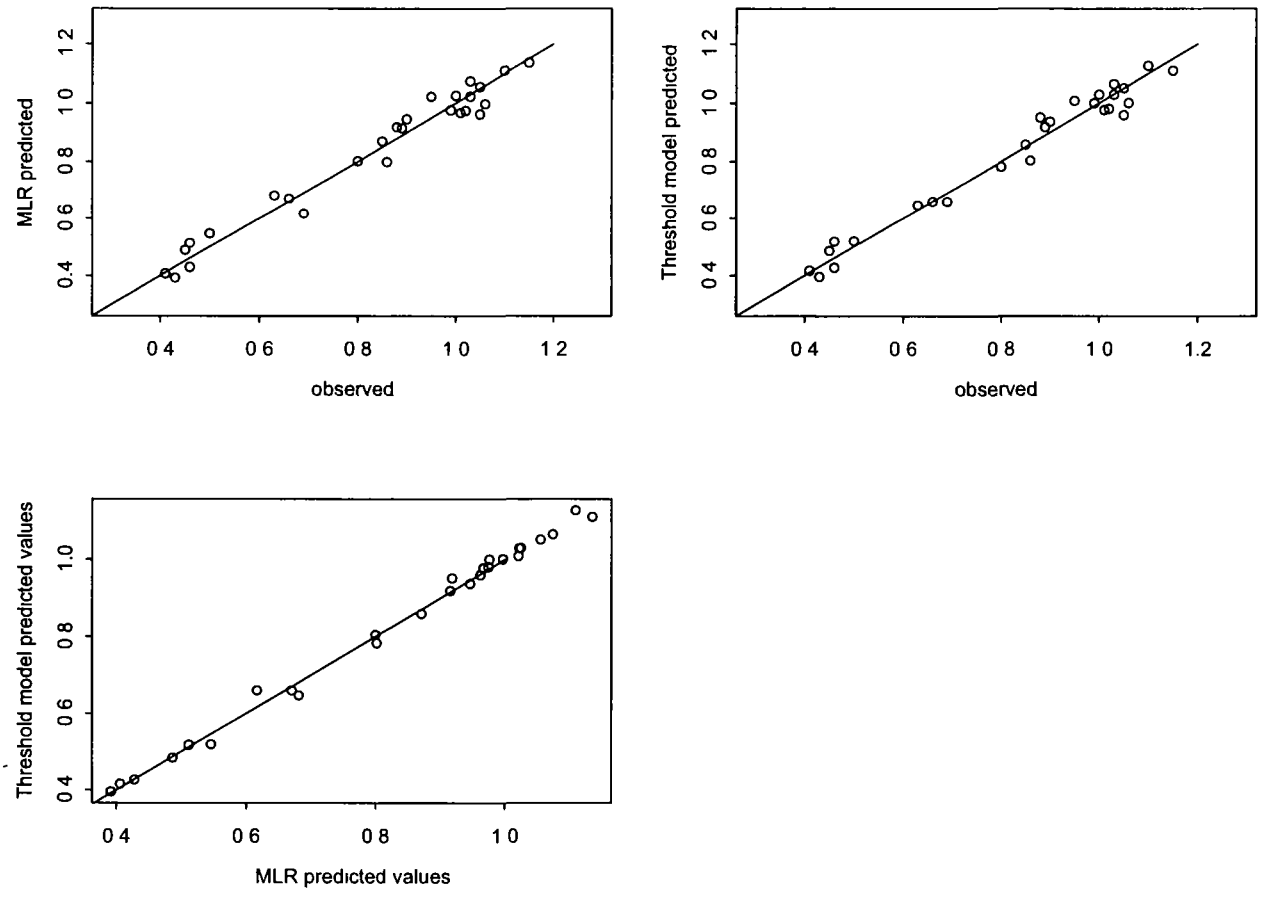


Figure E6-4. Relationship between Observed and Predicted Values for the Final Multiple Linear Regression (MLR) Model and Threshold Model and Comparison of Predicted Values from the Two Models.

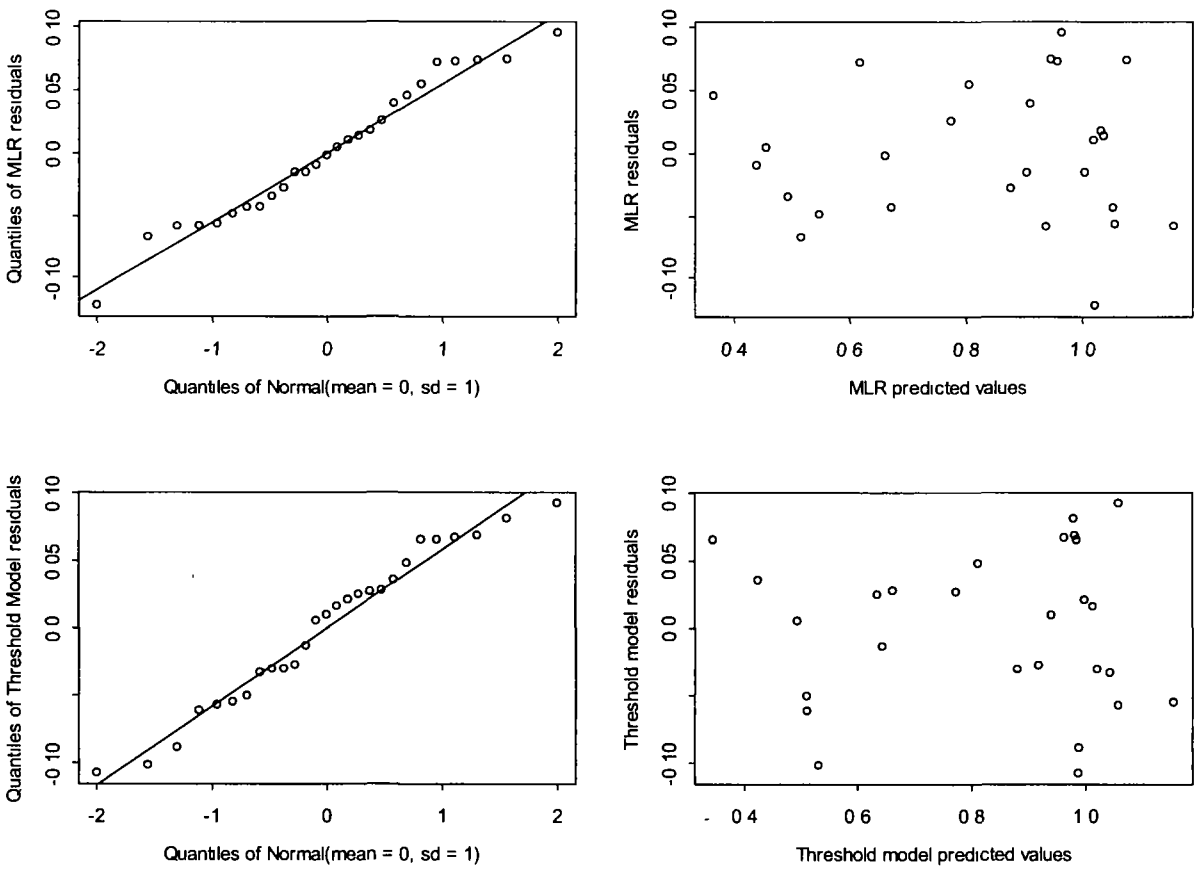


Figure E6-5. Residuals Plots for the Final Multiple Linear Regression (MLR) Model and Threshold Model, Excluding Arsenic, Cobalt, and Silver.

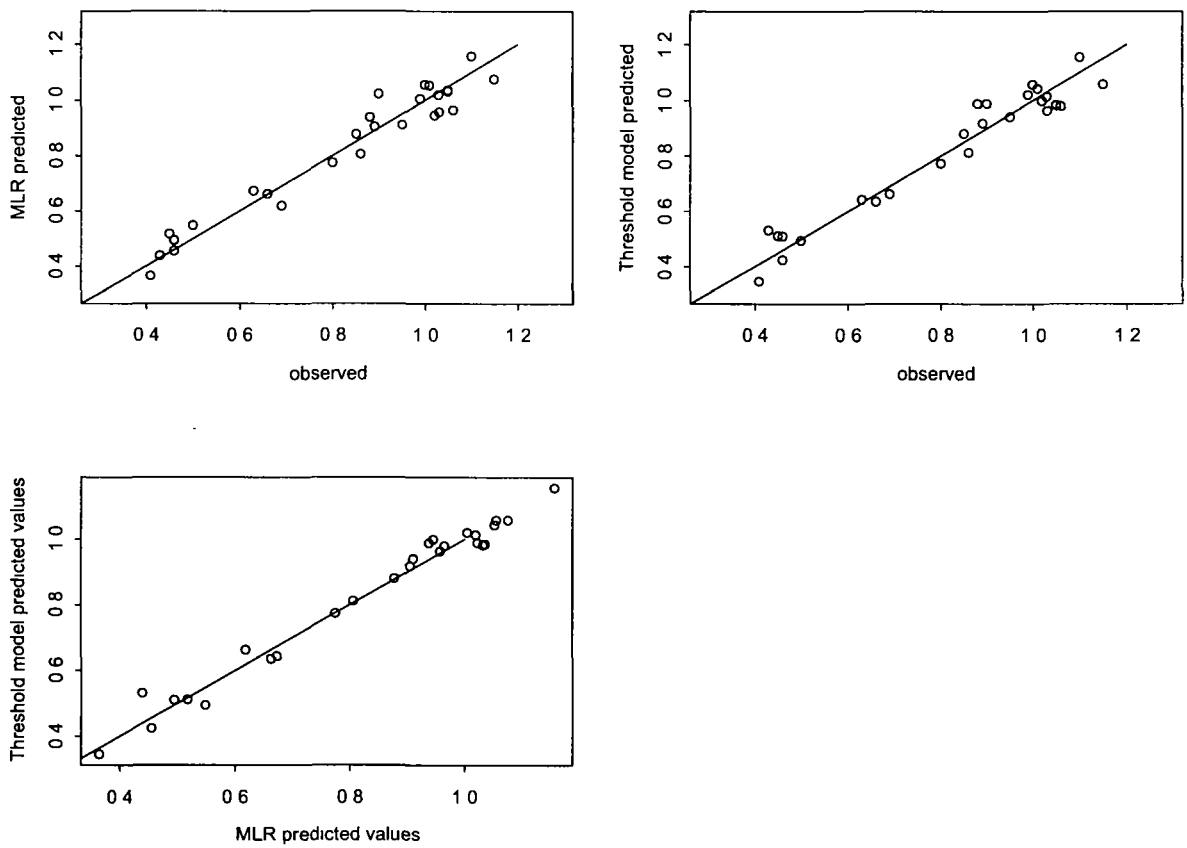
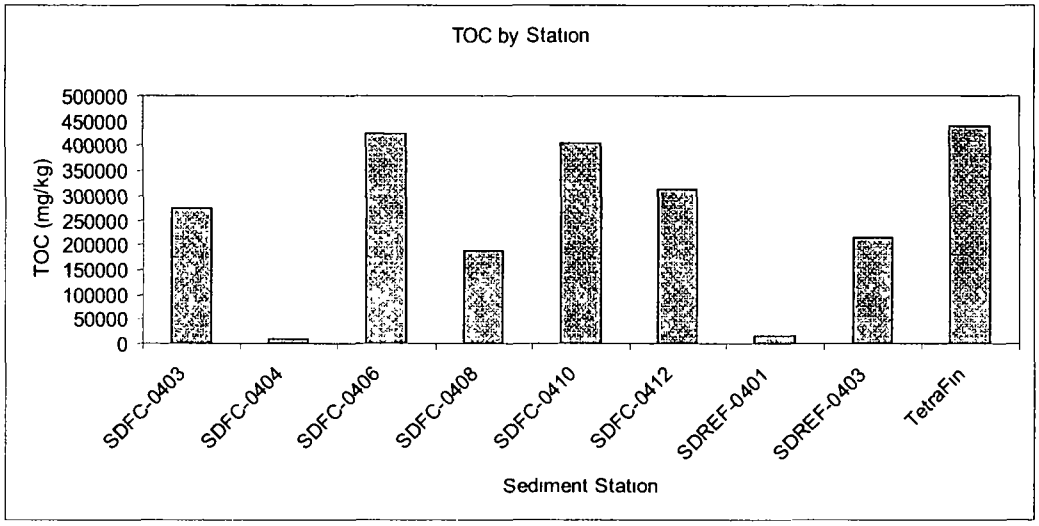
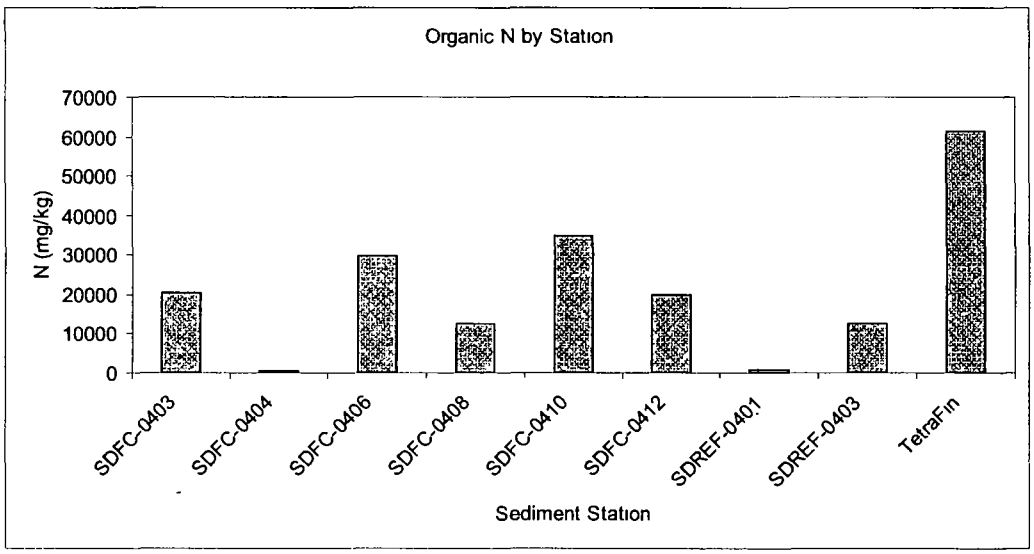


Figure E6-6. Relationship between Observed and Predicted Values for the Final Multiple Linear Regression (MLR) and Threshold Models, Excluding Arsenic, Cobalt, and Silver and Comparison of Predicted Values from These Two Models.





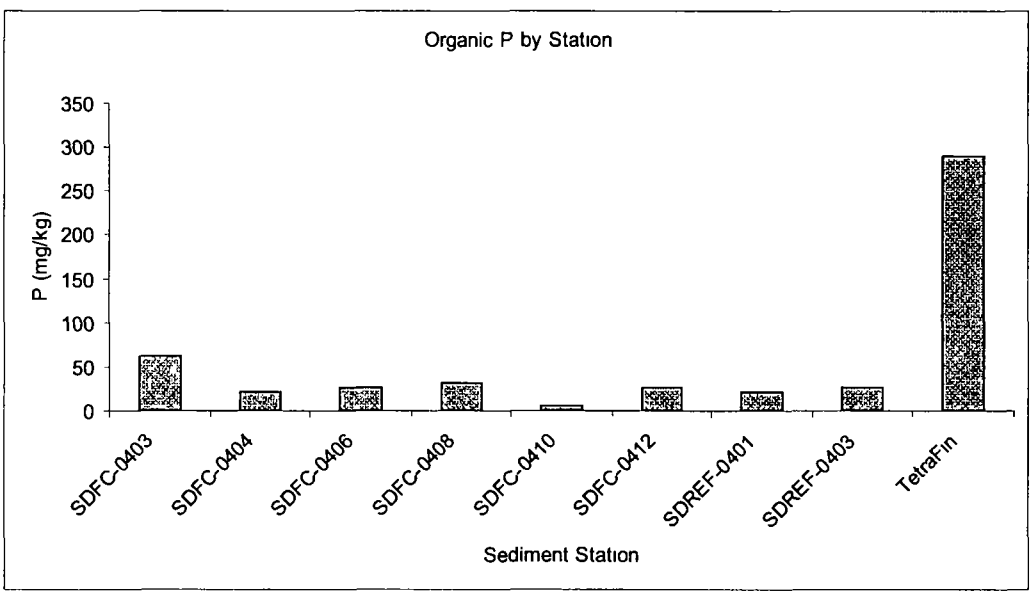
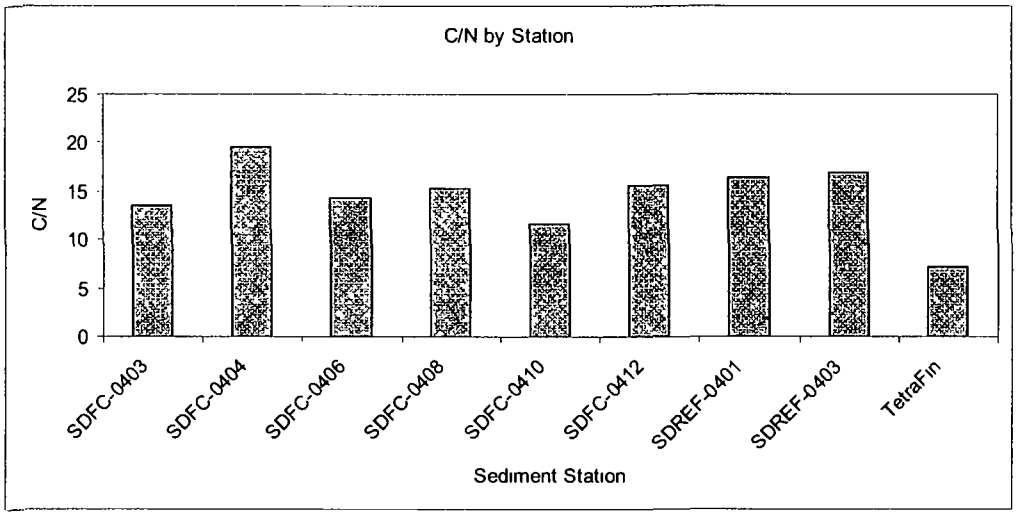
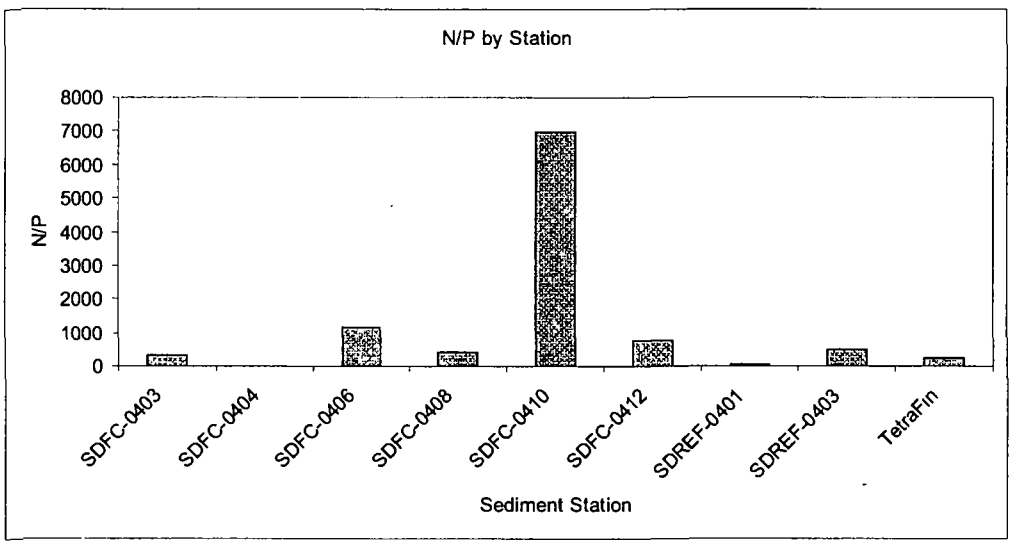


Figure E6-7. Concentrations of Organic Forms of Nitrogen, Phosphorus, and Carbon in Eight Sediment Samples from Fox Creek





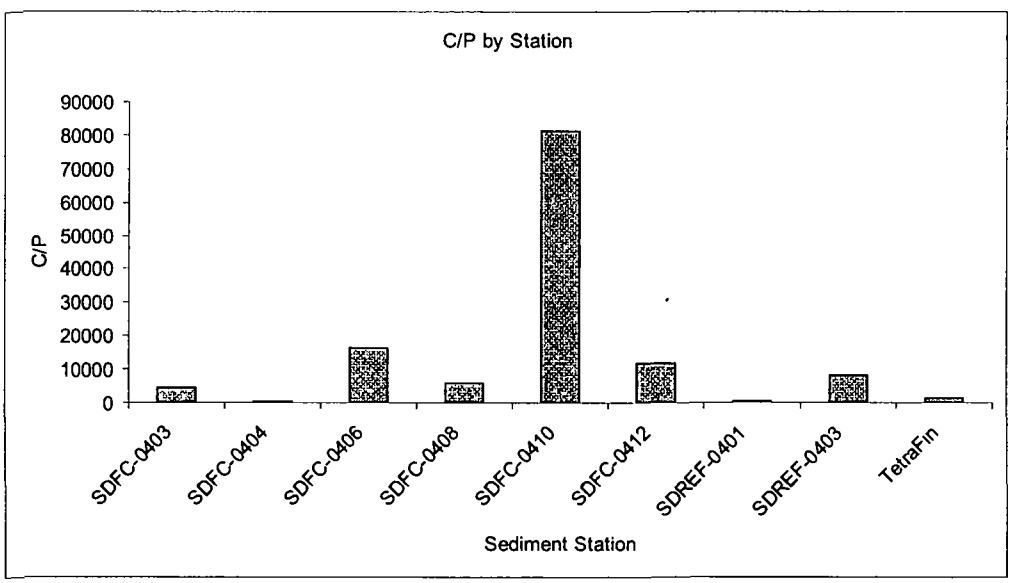
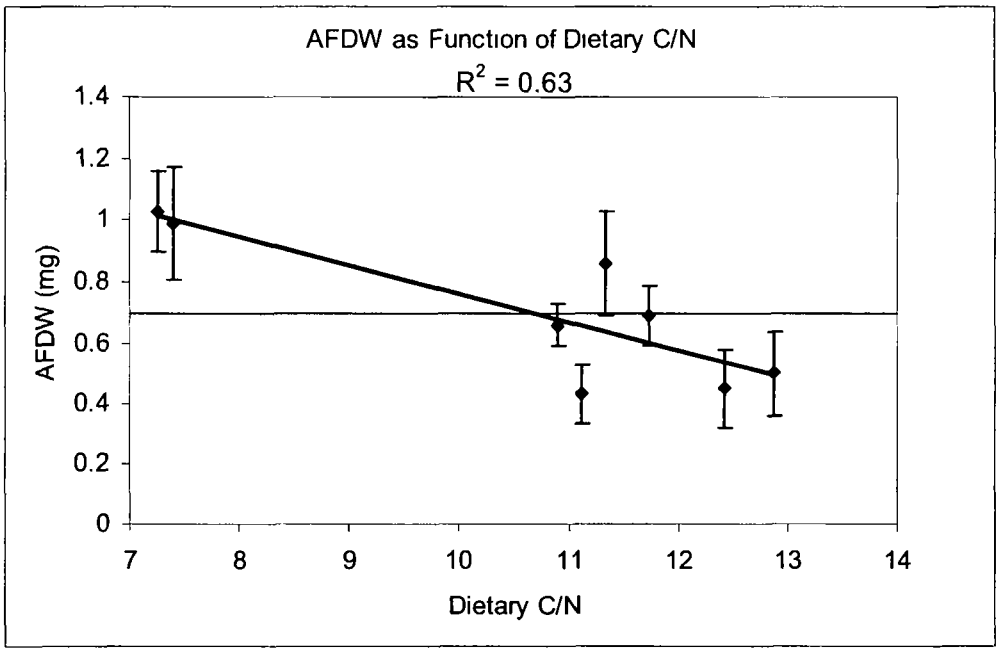


Figure E6-8. Nutrient Ratios in Eight Sediment Samples and in TetraFin



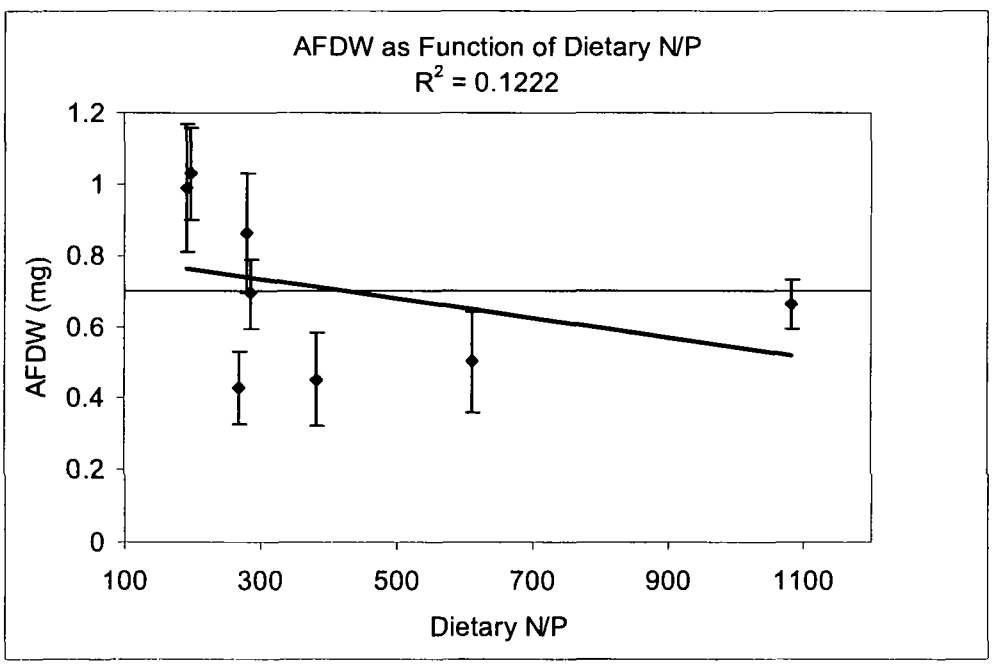
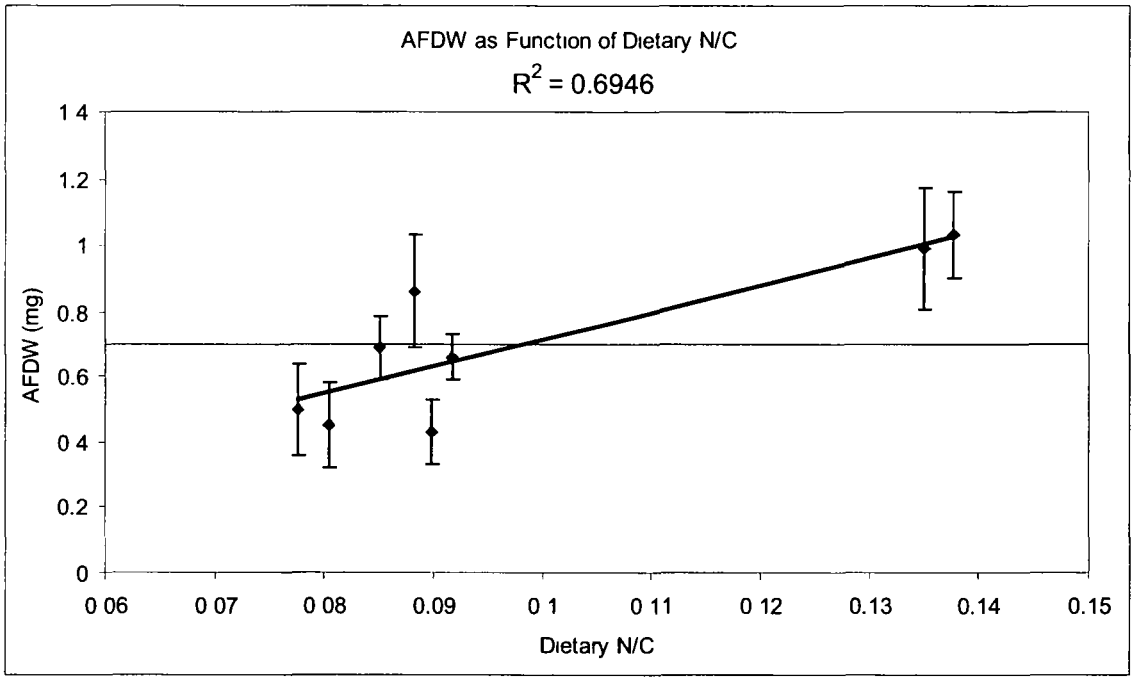


Figure E6-9. Dietary Nutrient Ratios Relative to Growth in Eight Bioassay Tests with Sediment from Fox Creek



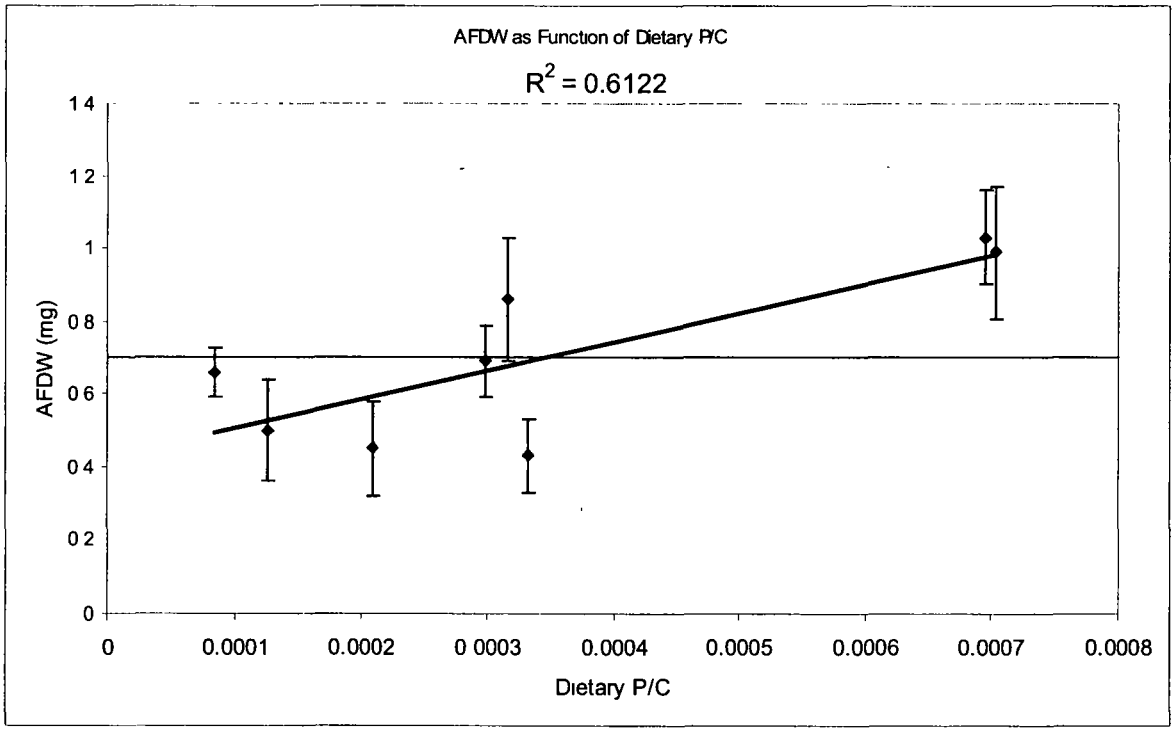


Figure E6-10. Dietary P/C and N/C Relative to Growth

TABLES

Table E6-1 Chemical Variables and Their Detection Frequency in the Bioassay Data Set

Chemical Code	Chemical Name	Units	Number of stations at which chemical was analyzed	Number of Non-detects	Detection Frequency	Group
pcbs	Total PCBs (Aroclors)	mg/kg	27	20	26%	PCBs
baa	Benzo(a)anthracene	mg/kg	27	2	93%	PAHs
bap	Benzo(a)pyrene	mg/kg	27	3	89%	PAHs
bbf	Benzo(b)fluoranthene	mg/kg	27	5	81%	PAHs
bjf1	Benzo(j)fluoranthene	mg/kg	7	2	71%	PAHs
bghip	Benzo(g,h,i)perylene	mg/kg	27	4	85%	PAHs
bkf	Benzo(k)fluoranthene	mg/kg	27	5	81%	PAHs
chrysene	Chrysene	mg/kg	27	4	85%	PAHs
dibenzaha	Dibenz(a,h)anthracene	mg/kg	27	9	67%	PAHs
fluoranthene	Fluoranthene	mg/kg	27	0	100%	PAHs
icdp	Indeno(1,2,3-cd)pyrene	mg/kg	27	4	85%	PAHs
pyrene	Pyrene	mg/kg	27	0	100%	PAHs
hpahs	HPAHs	mg/kg	27	0	100%	PAHs
2methnap	2-Methylnaphthalene	mg/kg	27	4	85%	PAHs
acenapthene	Acenaphthene	mg/kg	27	7	74%	PAHs
acenaptyle	Acenaphthylene	mg/kg	27	5	81%	PAHs
anthracene	Anthracene	mg/kg	27	4	85%	PAHs
fluorene	Fluorene	mg/kg	27	6	78%	PAHs
napthalene	Naphthalene	mg/kg	27	19	30%	PAHs
phenanthrene	Phenanthrene	mg/kg	27	0	100%	PAHs
lpahs	LPAHs	mg/kg	27	0	100%	PAHs
tpahs	TPAHs	mg/kg	27	0	100%	PAHs
aluminum	Aluminum	mg/kg	27	0	100%	Metals
antimony	Antimony	mg/kg	27	3	89%	Metals
arsenic	Arsenic	mg/kg	27	0	100%	Metals
barium	Barium	mg/kg	27	0	100%	Metals
beryllium	Beryllium	mg/kg	27	0	100%	Metals
cadmium	Cadmium	mg/kg	27	10	63%	Metals
chromium	Chromium	mg/kg	27	0	100%	Metals
cobalt	Cobalt	mg/kg	27	0	100%	Metals
copper	Copper	mg/kg	27	0	100%	Metals
iron	Iron	mg/kg	27	0	100%	Metals
lead	Lead	mg/kg	27	0	100%	Metals
manganese	Manganese	mg/kg	27	0	100%	Metals
mercury	Mercury	mg/kg	27	12	56%	Metals
molybdenum	Molybdenum	mg/kg	27	0	100%	Metals
nickel	Nickel	mg/kg	27	0	100%	Metals
selenium	Selenium	mg/kg	27	9	67%	Metals
silver	Silver	mg/kg	27	9	67%	Metals
thallium	Thallium	mg/kg	27	1	96%	Metals
vanadium	Vanadium	mg/kg	27	0	100%	Metals
zinc	Zinc	mg/kg	27	0	100%	Metals
pcp	Pentachlorophenol	mg/kg	27	27	0%	Pesticides
pp ddd	4,4'-DDD	mg/kg	27	18	33%	Pesticides
pp dde	4,4'-DDE	mg/kg	27	18	33%	Pesticides
pp.ddt	4,4'-DDT	mg/kg	27	24	11%	Pesticides
a.bhc	a-BHC	mg/kg	27	26	4%	Pesticides
a.chlordane	a-Chlordane	mg/kg	27	21	22%	Pesticides
b bhc	b-BHC	mg/kg	27	25	7%	Pesticides
d bhc	d-BHC	mg/kg	27	27	0%	Pesticides
dieldrin	Dieldrin	mg/kg	27	25	7%	Pesticides
endosulfan i	Endosulfan I	mg/kg	27	27	0%	Pesticides
endosulfan.ii	Endosulfan II	mg/kg	27	27	0%	Pesticides
endosulfan.sulfate	Endosulfan Sulfate	mg/kg	27	27	0%	Pesticides
endrin ald	Endrin Aldehyde	mg/kg	27	26	4%	Pesticides
endrin ket	Endrin Ketone	mg/kg	27	26	4%	Pesticides
endrin	Endrin	mg/kg	27	27	0%	Pesticides
g.bhc	g-BHC (Lindane)	mg/kg	27	26	4%	Pesticides

Table E6-1 Chemical Variables and Their Detection Frequency in the Bioassay Data Set

Chemical Code	Chemical Name	Units	Number of stations at which chemical was analyzed	Number of Non-detects	Detection Frequency	Group
g.chlordane	g-Chlordane	mg/kg	27	26	4%	Pesticides
heptachlor epoxide	Heptachlor Epoxide	mg/kg	27	27	0%	Pesticides
heptachlor	Heptachlor	mg/kg	27	26	4%	Pesticides
methoxychlor	Methoxychlor	mg/kg	27	26	4%	Pesticides
total.ddx	Total DDX	mg/kg	27	15	44%	Pesticides
toxaphene	Toxaphene	mg/kg	27	27	0%	Pesticides
total pcdd	Total PCDD (ND = 1/2 DL)	mg/kg	27	1	96%	Dioxins/Furans
total.pcdf	Total PCDF (ND = 1/2 DL)	mg/kg	27	1	96%	Dioxins/Furans
clay	Clay	pct	27	0	100%	Conventionals
gravel	Gravel	pct	27	0	100%	Conventionals
sand	Sand	pct	27	0	100%	Conventionals
silt	Silt	pct	27	0	100%	Conventionals
fines	the sum of clay + silt	pct	27	0	100%	Conventionals
toc	Total Organic Carbon	pct	27	0	100%	Conventionals

Table E6-2. Detection Frequencies for All Sediment Stations Combined

Chemical Name	Units	Number of stations at which chemical was analyzed	Number of Non-detects	Detection Frequency	Group
Clay	pct	53	0	100%	Grain Size
Gravel	pct	53	0	100%	Grain Size
Sand	pct	53	0	100%	Grain Size
Silt	pct	53	0	100%	Grain Size
Total Carbon	pct	9	0	100%	Matrix Properties
Aluminum	mg/kg	72	0	100%	Metals
Barium	mg/kg	72	0	100%	Metals
Copper	mg/kg	72	0	100%	Metals
Iron	mg/kg	72	0	100%	Metals
Magnesium	mg/kg	30	0	100%	Metals
Manganese	mg/kg	72	0	100%	Metals
Molybdenum	mg/kg	42	0	100%	Metals
Vanadium	mg/kg	72	0	100%	Metals
Zinc	mg/kg	72	0	100%	Metals
TPAHs	mg/kg	68	0	100%	PAHs
Lead	mg/kg	72	1	99%	Metals
LPAHs	mg/kg	68	1	99%	PAHs
Chromium	mg/kg	72	2	97%	Metals
HPAHs	mg/kg	68	2	97%	PAHs
Total PCDF (ND = 1/2 DL)	mg/kg	65	3	96%	Dioxins/Furans
Total PCDD (ND = 1/2 DL)	mg/kg	65	5	93%	Dioxins/Furans
Nickel	mg/kg	72	9	88%	Metals
Cobalt	mg/kg	72	14	81%	Metals
Beryllium	mg/kg	72	21	71%	Metals
Arsenic	mg/kg	72	22	69%	Metals
Silver	mg/kg	72	29	60%	Metals
Thallium	mg/kg	72	29	60%	Metals
Antimony	mg/kg	72	31	57%	Metals
Selenium	mg/kg	72	31	57%	Metals
Cadmium	mg/kg	72	34	53%	Metals
Mercury	mg/kg	72	36	50%	Metals
Total DDX	mg/kg	48	32	33%	Pesticides
4,4'-DDD	mg/kg	45	34	24%	Pesticides
4,4'-DDE	mg/kg	48	37	23%	Pesticides
Endosulfan Sulfate	mg/kg	45	36	20%	Pesticides
Total PCBs (Aroclors)	mg/kg	52	42	19%	PCBs (total)
a-Chlordane	mg/kg	45	37	18%	Pesticides
Dieldrin	mg/kg	45	38	16%	Pesticides
g-Chlordane	mg/kg	45	38	16%	Pesticides
4,4'-DDT	mg/kg	48	43	10%	Pesticides
Pentachlorophenol	mg/kg	68	67	8%	PCP
Endrin Aldehyde	mg/kg	45	42	7%	Pesticides
Heptachlor Epoxide	mg/kg	45	42	7%	Pesticides
Methoxychlor	mg/kg	45	42	7%	Pesticides
b-BHC	mg/kg	45	43	4%	Pesticides
Endosulfan I	mg/kg	45	43	4%	Pesticides
Heptachlor	mg/kg	45	43	4%	Pesticides
a-BHC	mg/kg	45	44	2%	Pesticides
Endosulfan II	mg/kg	45	44	2%	Pesticides
Endrin	mg/kg	45	44	2%	Pesticides
Endrin Ketone	mg/kg	45	44	2%	Pesticides
g-BHC (Lindane)	mg/kg	45	44	2%	Pesticides
Aldrin	mg/kg	45	45	0%	Pesticides
Atrazine	mg/kg	26	26	0%	Pesticides
d-BHC	mg/kg	45	45	0%	Pesticides
Toxaphene	mg/kg	45	45	0%	Pesticides

Notes:

Biasing non-detects have been included

All toxicity stations have been evaluated, including toxicity reference stations

Reference stations where toxicity has not been conducted have been excluded

Table E6-3 Kendall's tau-b among Sediment COPECs and Grain Size in Toxicity-Tested Sediments

	pcbs	hpahs	lpahs	tpahs	Sb	Ba	Be	Cd	Cu	Pb	Hg	Mo	Se	Ag	Tl	Zn	pp ddd	pp dde	pp ddt	t ddx	t pcdd	t pcdf	clay	gravel	sand	silt	fines	toc
pcbs	**0.46**	0 13	0 20	0 15	0 28	0 26	0 23	**0.34**	**0.37**	**0.34**	**0.35**	0 21	0 23	**0.42**	0 10	**0.36**	**0.35**	**0.35**	0 18	0 33	0 28	0 27	0 28	0 33	-0 25	0.23	0 23	0.29
hpahs		**1.00**	**0.83**	**0.93**	0 15	**0.44**	**0.50**	0 32	**0.42**	**0.55**	0 24	0 31	0 17	0 27	**0.40**	**0.44**	0 26	0 23	0 05	0 34	**0.59**	**0.61**	**0.50**	0 29	**-0.50**	**0.41**	**0.49**	0 25
lpahs			**1.00**	**0.90**	0 27	**0.48**	**0.54**	**0.39**	**0.49**	**0.54**	0 30	**0.42**	0 23	**0.36**	**0.40**	**0.52**	0 29	0 27	0 07	**0.38**	**0.63**	**0.65**	**0.53**	**0.38**	**-0.56**	**0.49**	**0.54**	**0.36**
tpahs				**1.00**	0 21	**0.47**	**0.54**	**0.36**	**0.44**	**0.57**	0 26	**0.38**	0 21	0 29	**0.42**	**0.48**	0 26	0 26	0 06	**0.37**	**0.62**	**0.64**	**0.53**	0 30	**-0.53**	**0.46**	**0.53**	0 30
Sb					**0.96**	**0.44**	**0.42**	**0.60**	**0.56**	**0.46**	**0.44**	**0.52**	**0.57**	**0.53**	0 31	**0.51**	0 32	0 28	0 15	**0.35**	0 34	0 31	**0.36**	0 28	**-0.37**	**0.44**	**0.38**	**0.49**
Ba						**1.00**	**0.74**	**0.62**	**0.64**	**0.66**	**0.51**	**0.51**	**0.48**	**0.45**	**0.39**	**0.70**	0 26	0 34	0 11	**0.35**	**0.58**	**0.58**	**0.71**	**0.39**	**-0.68**	**0.73**	**0.75**	**0.56**
Be							**0.99**	**0.56**	**0.71**	**0.61**	**0.55**	**0.66**	**0.56**	**0.51**	**0.60**	**0.70**	0 30	**0.34**	0 11	**0.43**	**0.63**	**0.63**	**0.75**	**0.40**	**-0.77**	**0.73**	**0.78**	**0.49**
Cd								**0.84**	**0.76**	**0.66**	**0.60**	**0.50**	**0.50**	**0.59**	**0.40**	**0.74**	**0.36**	**0.42**	0 16	**0.46**	**0.48**	**0.50**	**0.55**	**0.38**	**-0.50**	**0.54**	**0.53**	**0.55**
Cu									**1.00**	**0.71**	**0.64**	**0.58**	**0.49**	**0.69**	**0.50**	**0.85**	**0.42**	**0.47**	0 18	**0.55**	**0.60**	**0.64**	**0.62**	**0.50**	**-0.61**	**0.62**	**0.64**	**0.61**
Pb										**1.00**	**0.52**	**0.43**	**0.35**	**0.53**	**0.40**	**0.77**	**0.38**	**0.44**	0 15	**0.48**	**0.59**	**0.62**	**0.64**	**0.50**	**-0.61**	**0.54**	**0.58**	**0.43**
Hg											**0.79**	**0.51**	**0.52**	**0.60**	**0.41**	**0.59**	0 36	0 42	0.19	**0.46**	**0.44**	**0.46**	**0.56**	**0.38**	**-0.51**	**0.52**	**0.53**	**0.53**
Mo												**1.00**	**0.71**	**0.54**	**0.67**	**0.53**	**0.27**	**0.29**	0 13	**0.38**	**0.43**	**0.42**	**0.54**	0 24	**-0.55**	**0.59**	**0.63**	**0.62**
Se													**0.86**	**0.54**	**0.58**	**0.44**	0 28	0 31	0 15	**0.37**	0 29	0 28	**0.46**	0 17	**-0.46**	**0.53**	**0.49**	**0.59**
Ag														**0.87**	**0.43**	**0.62**	**0.44**	**0.44**	0.18	**0.50**	**0.47**	**0.46**	**0.52**	**0.44**	**-0.48**	**0.53**	**0.52**	**0.62**
Tl															**0.99**	**0.46**	0 28	0 31	0 11	**0.42**	0 33	0 35	**0.52**	0 11	**-0.49**	**0.47**	**0.52**	**0.44**
Zn																**1.00**	**0.38**	**0.46**	0 16	**0.52**	**0.59**	**0.62**	**0.64**	**0.46**	**-0.56**	**0.63**	**0.64**	**0.51**
pp ddd																	**0.50**	**0.36**	0 19	**0.48**	**0.37**	**0.37**	0 31	0 30	-0 32	0 26	0.28	0 30
pp dde																		**0.54**	0 18	**0.51**	**0.38**	**0.38**	**0.37**	0 28	-0 32	**0.35**	0 34	0 33
pp ddt																			0 19	0 19	0 11	0 11	0 10	0 13	-0 09	0 09	0 09	0 13
total ddx																				**0.65**	**0.45**	**0.46**	**0.44**	0 29	**-0.42**	**0.38**	**0.42**	**0.39**
total pcdd																					**0.99**	**0.93**	**0.64**	**0.46**	**-0.58**	**0.55**	**0.61**	**0.41**
total pcdf																						**0.99**	**0.64**	**0.46**	**-0.61**	**0.54**	**0.60**	**0.42**
clay																							**0.99**	**0.38**	**-0.78**	**0.72**	**0.82**	**0.54**
gravel																								**1.00**	**-0.44**	**0.37**	**0.37**	0 34
sand																									**1.00**	**-0.68**	**-0.77**	**-0.53**
silt																										**1.00**	**0.90**	**0.60**
fines																											**1.00**	**0.60**
toc																												**1.00**

Notes:

* censored data treated as valid pairs when direction is clear (e g , 10 to <3 is a decrease), treated as a tie, and therefore does not contribute to the tau when direction is unclear (e g , <10 to <3)

Note that the correlations on the diagonal (a variable against itself) may not be 1 due to ties or censoring, the lower the censoring, the closer it is to 1

bold values indicate that the correlation is statistically significant (at p=0 01)

Table E6-4. Kendall's Correlation Results between mg AFDW (X) and Chemical Endpoint (Y)

Sorted by correlation coefficient (tau-b) *Sorted by chemical group and endpoint*

Chemical Code	DF[a]	tau-b	p-value[b]	Chemical Code	DF[1]	tau-b	p-value[b]
molybdenum	100%	-0.65	**0.0000**	pcbs	26%	-0 27	0.020
chromium	100%	-0.60	**0.0000**	baa	93%	-0 25	0 035
toc	100%	-0 60	**0.0000**	bap	89%	-0 23	0 047
antimony	89%	-0 59	**0.0000**	bbf	81%	-0 24	0 041
cadmium	63%	-0 57	**0.0000**	bghip	85%	-0 30	0 014
arsenic	100%	-0 57	**0.0000**	bkf	81%	-0 26	0 031
selenium	67%	-0 56	**0.0000**	chrysene	85%	-0 28	0 019
vanadium	100%	-0 53	**0.0001**	dibenzaha	67%	-0.26	0 030
mercury	56%	-0 52	**0.0001**	fluoranthene	100%	-0.26	0 030
lead	100%	-0 51	**0.0001**	icdp	85%	-0.28	0 021
copper	100%	-0 51	**0.0001**	pyrene	100%	-0 25	0.033
silver	67%	-0.50	**0.0001**	hpahs	100%	-0 26	0 032
fines	100%	-0 49	**0.0002**	2 methylnaphthalene	85%	-0 46	0 000
zinc	100%	-0.48	**0.0002**	acenapthene	74%	-0 37	0 003
silt	100%	-0 47	**0.0003**	acenaptyle	81%	-0.37	0 003
2 methylnaphthalene	85%	-0.46	**0.0003**	anthracene	85%	-0.31	0.011
thallium	96%	-0 45	**0.0006**	fluorene	78%	-0.38	0 003
nickel	100%	-0 43	**0.0008**	napthalene	30%	-0.15	0.125
barium	100%	-0.43	0 0009	phenanthrene	100%	-0 31	0.011
aluminum	100%	-0 42	0 0012	lpahs	100%	-0.38	0 003
beryllium	100%	-0.42	0 0012	tpahs	100%	-0 31	0 011
manganese	100%	-0 41	0.0014	aluminum	100%	-0 42	0 001
clay	100%	-0 41	0 0014	antimony	89%	-0.59	0 000
cobalt	100%	-0.40	0 0016	arsenic	100%	-0 57	0 000
fluorene	78%	-0 38	0 0025	barium	100%	-0.43	0 001
lpahs	100%	-0.38	0 0031	beryllium	100%	-0.42	0 001
acenapthene	74%	-0.37	0 0034	cadmium	63%	-0 57	0.000
acenaptyle	81%	-0.37	0 0035	chromium	100%	-0 60	0 000
iron	100%	-0 35	0 01	cobalt	100%	-0 40	0.002
total.ddx	44%	-0.33	0 01	copper	100%	-0.51	0 000
total.pcdf	96%	-0 32	0 01	iron	100%	-0.35	0.005
anthracene	85%	-0 31	0 01	lead	100%	-0.51	0 000
phenanthrene	100%	-0 31	0 01	manganese	100%	-0.41	0 001
tpahs	100%	-0 31	0.01	mercury	56%	-0.52	0 000
gravel	100%	-0.31	0.01	molybdenum	100%	-0.65	0.000
total.pcdd	96%	-0.30	0.01	nickel	100%	-0 43	0.001
bghip	85%	-0.30	0 01	selenium	67%	-0 56	0 000
pp ddd	33%	-0 29	0 01	silver	67%	-0.50	0 000
chrysene	85%	-0 28	0.02	thallium	96%	-0.45	0.001
pp.dde	33%	-0.28	0 02	vanadium	100%	-0.53	0.000
icdp	85%	-0.28	0 02	zinc	100%	-0.48	0 000
pcbs	26%	-0 27	0 02	pp ddd	33%	-0 29	0 015
fluoranthene	100%	-0 26	0 03	pp dde	33%	-0 28	0.016
bkf	81%	-0 26	0 03	pp ddt	11%	-0.15	0 126
dibenzaha	67%	-0 26	0 03	a chlordane	22%	-0 01	0.474
hpahs	100%	-0 26	0 03	total ddx	44%	-0 33	0.007
pyrene	100%	-0 25	0 03	total pcdd	96%	-0 30	0 013
baa	93%	-0 25	0 03	total pcdf	96%	-0.32	0 010
bbf	81%	-0 24	0 04	clay	100%	-0 41	0 001
bap	89%	-0 23	0 05	gravel	100%	-0 31	0 013
napthalene	30%	-0.15	0 12	sand	100%	0 43	
pp ddt	11%	-0 15	0 13	silt	100%	-0 47	0 000
a chlordane	22%	-0.01	0 47	fines	100%	-0 49	0 000
sand	100%	0 43	**0.00**	toc	100%	-0 60	0 000

Notes:

[a] DF = Detection frequency

critical p (alpha=0 05, Bonferroni correction) 0.00093

[b]The one-tail p-value of the regression is shown; it is one-tailed for the test of a significant *negative* correlation We are not interested in positive correlations, therefore a p-value is not shown if tau-b is greater than 0. The critical value of p, based on a Type 1 error rate (alpha) of 5 percent and a Bonferroni correction for multiple comparisons, is 0.000926. Results with a lower p value are shown in bold

Table E6-5a. Stepwise Results for Predicting Growth (AFDM, mg) from Multiple Linear Regression Model, Full List of Potential Predictors (MLR1)

Model	Adjusted R-square	t-statistic[a]	p	Model Change
With non-detects substituted at the DL				
1 chromium	0.763	-9.45	0.000	
2 chromium + cobalt	0.853	4.06	0.000	*add cobalt*
3 chromium + cobalt + arsenic	0.888	-2.93	0.008	*add arsenic*
4 chromium + cobalt + arsenic + manganese	0.928	3.73	0.001	*add manganese*
5 chromium + cobalt + arsenic + manganese + lpahs	0.940	-2.32	0 030	*add lpahs*
6 chromium + cobalt + arsenic + manganese + lpahs + silver	0.946	1 81	0.085	*add silver*
7 chromium + cobalt + arsenic + manganese + lpahs + silver + lead	0.954	-2.13	0.046	*add lead*
8 chromium + cobalt + arsenic + manganese + silver + lead	0.955	-0.62	0.542	*remove lpahs*
chromium + cobalt + arsenic + manganese + silver + lead	**0.955**	**AIC = -78**		**Best Model for Full List**

Note:

[a]The statistic to test the significance of the last parameter added (or dropped) in the current model. It describes the reduction in error due to this parameter, conditional on the other parameters already being in the model

Table E6-5b. Stepwise Results for Predicting Growth (AFDM, mg) from Multiple Linear Regression Model, Excluding Arsenic, Cobalt, and Silver from List of Potential Predictors [a]

Model	Adjusted R-square	t-statistic[b]	p	Model Change
With non-detects substituted at the DL				
1 chromium	0.763	-9.45	0.000	
2 chromium + manganese	0 824	3.13	0.005	*add manganese*
3 chromium + manganese + TOC	0.878	-3.41	0.002	*add TOC*
4 chromium + manganese + TOC + iron	0.886	-1.60	0.124	*add iron*
5 chromium + manganese + TOC + iron + nickel	0.895	1 74	0.097	*add nickel*
6 lpahs	0.907	-1.96	0.064	*add lpahs*
7 chromium + manganese + TOC + nickel + lpahs	0.909	-0.86	0.401	*remove iron*
8 barium	0.931	-2.80	0.011	*add barium*
chromium + manganese + TOC + nickel + lpahs + barium	**0.931**	**AIC = -66**		**Best Model for Short List**

Notes:

[a]Arsenic, cobalt, and silver were removed from the list of potential predictors because of low detection frequencies and/or high detection limits in the chemistry on'y dataset

[b]The statistic to test the significance of the last parameter added (or dropped) in the current model. It describes the reduction in error due to this parameter, conditional on the other

Table E6-6a. Stepwise Results for Predicting Growth (AFDM, mg) from Threshold and Multiple Linear Regression Model, Full List of Potential Predictors (MLR3)

Model	Multiple	Adjusted R-square	t-statistic[a]	p	Model Change
With non-detects substituted at the DL					
1 TOC with threshold	0.877	0.784	-3.073	0.005	
2 TOC + chromium	0.899	0.835	-2.914	0.008	*add chromium*
3 TOC + chromium + manganese	0.920	0.891	3.562	0.002	*fix TOC threshold at 10%; add manganese*
4 TOC + chromium + manganese + hpahs	0.938	0.909	-2.342	0.029	*add hpahs*
5 TOC + chromium + manganese + hpahs + nickel		0.915	1.671	0.110	*add nickel*
6 TOC + chromium + manganese + hpahs + nickel + arsenic		0.927	-2.067	0.052	*add arsenic*
7 TOC + chromium + manganese + hpahs + nickel + arsenic + cobalt		0 936	2.027	0.057	*add cobalt*
8 TOC + chromium + manganese + hpahs + nickel + arsenic + cobalt + silver		0.949	2.377	0.029	*add silver*
9 TOC + chromium + manganese + hpahs + nickel + arsenic + cobalt + silver + lead		0.958	-2.311	0 034	*add lead*
10 TOC + chromium + manganese + hpahs + nickel + arsenic + cobalt + silver + lead + zinc		0.959	1.144	0.269	*add zinc*
11 TOC + chromium + manganese + hpahs + arsenic + cobalt + silver + lead + zinc		0.962	0 122	0.905	*remove nickel*
12 TOC + chromium + manganese + arsenic + cobalt + silver + lead + zinc		0 963	-0 740	0.469	*remove hpahs*
13 TOC + chromium + manganese + arsenic + cobalt + silver + lead		0 960			*remove zinc to improve distribution of residuals*
TOC + chromium + manganese + arsenic + cobalt + silver + lead		**0.960**	**AIC = -80**		**Best Model for full list**

Note:

[a]The statistic to test the significance of the last parameter added (or dropped) in the current model. It describes the reduction in error due to this parameter, conditional on the other parameters already being in the model

Table E6-6b. Stepwise Results for Predicting Growth (AFDM, mg) from Threshold and Multiple Linear Regression Model, Excluding Arsenic, Cobalt, and Silver from List of Potential Predictors (MLR4)

Model	Multiple	Adjusted R-square	t-statistic[a]	p	Model Change
With non-detects substituted at the DL					
1 TOC with threshold	0.877	0.784	-3.073	0 005	
2 TOC + chromium	0 899	0.835	-2.914	0.008	*add chromium*
3 TOC + chromium + manganese	0 920	0.891	3.562	0.002	*fix TOC threshold at 10%; add manganese*
4 TOC + chromium + manganese + hpahs	0.938	0.909	-2.342	0.029	*add hpahs*
5 TOC + chromium + manganese + hpahs + nickel		0.915	1.671	0.110	*add nickel*
7 TOC + chromium + manganese + hpahs + nickel + barium		0.922	-1.714	0.102	*add barium*
TOC + chromium + manganese + hpahs + nickel + barium		**0.922**	**AIC = -63**		**Best Model for Short List**

Notes:

Arsenic, cobalt, and silver were removed from the list of potential predictors because of low detection frequencies and/or high detection limits in the dataset requiring predictions

[a]The statistic to test the significance of the last parameter added (or dropped) in the current model. It describes the reduction in error due to this parameter, conditional on the other parameters already being in the model.

Table E6-7a. Coefficients for the Final Multiple Linear Regression Model, Using the Full List of Potential Predictors (MLR1)

	Value	Std Error	t-value	p-value
Intercept	1.056	0.079	13.37	0
$\log_{10}$(chromium)	-0.678	0.064	-10.61	0
$\log_{10}$(cobalt)	0.360	0 063	5.75	0
$\log_{10}$(arsenic)	-0.330	0.048	-6.86	0
$\log_{10}$(manganese)	0.219	0.037	5.92	0
$\log_{10}$(silver)	0.077	0.022	3.49	0.002
$\log_{10}$(lead)	-0.100	0.031	-3.26	0.004

Table E6-7b Coefficients for the Final Multiple Linear Regression Model, Using the Short List of Potential Predictors (MLR2)

	Value	Std. Error	t-value	p-value
Intercept	1.077	0 080	13.42	0
$\log_{10}$(chromium)	-0.705	0 077	-9 22	0
$\log_{10}$(manganese)	0 178	0.048	3 74	0.001
$\log_{10}$(TOC)	-0.073	0.037	-1.95	0 066
$\log_{10}$(nickel)	0.409	0.113	3.62	0 002
$\log_{10}$(lpahs)	-0.059	0.017	-3.36	0 003
$\log_{10}$(barium)	-0.212	0.076	-2.80	0.011

Table E6-8a. Coefficients for the Final Threshold and Multiple Linear Regression Model, Using the Full List of Potential Predictors (MLR3)

	Value	Std. Error	t-value	p-value
Intercept	1.069	0.075	14.33	1.23E-11
$\log_{10}$(TOC)	-0 220	0.116	-1.89	0.073481
$\log_{10}$(chromium)	-0 597	0 073	-8.13	1 31E-07
$\log_{10}$(manganese)	0 216	0.035	6.23	5 56E-06
$\log_{10}$(arsenic)	-0.261	0.058	-4.49	0.000253
$\log_{10}$(cobalt)	0 284	0.071	3.97	0.001
$\log_{10}$(silver)	0.090	0.022	4.12	0.001
$\log_{10}$(lead)	-0.112	0.029	-3.80	0.001

Table E6-8b. Coefficients for the Final Threshold and Multiple Linear Regression Model, Using the Short List of Potential Predictors (MLR4)

	Value	Std. Error	t-value	p-value
Intercept	1.050	0.088	11.95	1.46E-10
$\log_{10}$(TOC)	-0.370	0.122	-3.04	0.006454
$\log_{10}$(chromium)	-0 594	0 093	-6 36	0 000
$\log_{10}$(manganese)	0.177	0.052	3.39	0.003
$\log_{10}$(hpahs)	-0 049	0.019	-2 65	0 015
$\log_{10}$(nickel)	0.250	0 113	2.21	0.039
$\log_{10}$(barium)	-0 142	0.083	-1.71	0.102

Table E6-9. Concentrations of Chemicals at Bioassay Stations that Exceed SQGs

Station ID	Analytical Group	Analyte	Sed. Conc mg/kg	Qual	Screening value (mg/kg)	Exceedance Factor	Growth Effect?	TOC
SDCH-0401	Metals	Barium	159		20	8.0	N	24
SDCH-0401	Metals	Selenium	1	j	0.1	10 0	N	24
SDCH-0401	PAHs	HPAHs	2.525		0.19	13 3	N	24
SDCH-0401	PAHs	LPAHs	0.422	J	0 076	5.6	N	24
SDCH-0401	PAHs	TPAHs	2.947	J	1 61	1 8	N	24
SDCH-0401	Pesticides	4,4'-DDD	0.0097		0.00488	2.0	N	24
SDCH-0401	Pesticides	4,4'-DDE	0 0049		0.00316	1.6	N	24
SDCH-0402	Metals	Barium	191		20	9.6	N	20.3
SDCH-0402	Metals	Selenium	0.5	j	0 1	5.0	N	20.3
SDCH-0402	PAHs	HPAHs	4.29		0.19	22.6	N	20.3
SDCH-0402	PAHs	LPAHs	1 617		0.076	21.3	N	20.3
SDCH-0402	PAHs	TPAHs	5.907		1.61	3 7	N	20.3
SDCH-0403	Metals	Barium	29.5		20	1.5	N	4.26
SDCH-0403	PAHs	HPAHs	0.21415	J	0.19	1.1	N	4.26
SDCH-0404	Metals	Barium	39.6		20	2.0	N	0.83
SDFC-0401	Metals	Selenium	0 2	j	0 1	2.0	N	0.56
SDFC-0401	PAHs	HPAHs	1 019		0.19	5.4	N	0.56
SDFC-0401	PAHs	LPAHs	0.1487	J	0.076	2.0	N	0.56
SDFC-0402	Metals	Barium	122		20	6 1	Y	20.3
SDFC-0402	Metals	Manganese	984		630	1 6	Y	20.3
SDFC-0402	Metals	Selenium	7 5		0 1	75.0	Y	20.3
SDFC-0402	PAHs	HPAHs	1.891	J	0.19	10.0	Y	20.3
SDFC-0402	PAHs	LPAHs	0.2995	J	0.076	3.9	Y	20 3
SDFC-0402	PAHs	TPAHs	2.1905	J	1.61	1.4	Y	20.3
SDFC-0403	Metals	Arsenic	11.6		9 79	1.2	Y	26.6
SDFC-0403	Metals	Barium	46.2		20	2 3	Y	26.6
SDFC-0403	Metals	Selenium	5.5		0.1	55.0	Y	26.6
SDFC-0403	PAHs	HPAHs	1.312	J	0.19	6.9	Y	26.6
SDFC-0403	PAHs	LPAHs	22 3425	J	0 076	294 0	Y	26.6
SDFC-0403	PAHs	TPAHs	23.6545	J	1 61	14 7	Y	26.6
SDFC-0403	Pesticides	Heptachlor	0.0015	U	0.0006	2.5	Y	26.6
SDFC-0404	PAHs	HPAHs	1 3442		0.19	7 1	N	0.74
SDFC-0404	PAHs	LPAHs	0.495		0.076	6 5	N	0.74
SDFC-0404	PAHs	TPAHs	1 8392		1 61	1.1	N	0.74
SDFC-0405	Metals	Barium	64.3		20	3 2	Y	41.6
SDFC-0405	Metals	Selenium	10.4		0 1	104.0	Y	41.6
SDFC-0406	Metals	Barium	74.5		20	3.7	Y	42.8
SDFC-0406	Metals	Selenium	4.9		0 1	49.0	Y	42.8
SDFC-0406	PAHs	HPAHs	0.2619	J	0.19	1.4	Y	42.8
SDFC-0406	PAHs	LPAHs	0.0842	J	0.076	1.1	Y	42.8

Table E6-9. Concentrations of Chemicals at Bioassay Stations that Exceed SQGs

Station ID	Analytical Group	Analyte	Sed. Conc. mg/kg	Qual	Screening value (mg/kg)	Exceedance Factor	Growth Effect?	TOC
SDFC-0407	Metals	Arsenic	11.35		9.79	1 2	Y	39
SDFC-0407	Metals	Barium	81.1		20	4.1	Y	39
SDFC-0407	Metals	Cadmium	1.235		0.99	1.2	Y	39
SDFC-0407	Metals	Copper	59 65		31.6	1.9	Y	39
SDFC-0407	Metals	Mercury	0.9835		0.18	5 5	Y	39
SDFC-0407	Metals	Selenium	8.8		0.1	88 0	Y	39
SDFC-0407	Metals	Silver	5.09		2	2 5	Y	39
SDFC-0407	Metals	Zinc	372.5		121	3 1	Y	39
SDFC-0407	PAHs	HPAHs	1.272	J	0.19	6 7	Y	39
SDFC-0407	PAHs	LPAHs	0.27655	J	0.076	3 6	Y	39
SDFC-0407	PAHs	Pyrene	0.2		0.195	1 0	Y	39
SDFC-0407	PCBs (total)	Total PCBs	0.1065		0.0598	1 8	Y	39
SDFC-0407	Pesticides	4,4'-DDD	0.0195	*	0.00488	4.0	Y	39
SDFC-0407	Pesticides	4,4'-DDE	0.00555	j	0.00316	1 8	Y	39
SDFC-0407	Pesticides	Heptachlor	0.001825	jp	0.0006	3.0	Y	39
SDFC-0408	Metals	Barium	44.6		20	2.2	Y	18.3
SDFC-0408	Metals	Cadmium	1.27		0.99	1.3	Y	18.3
SDFC-0408	Metals	Copper	50.8		31.6	1.6	Y	18.3
SDFC-0408	Metals	Lead	60 3		35.8	1.7	Y	18.3
SDFC-0408	Metals	Selenium	3.1		0.1	31.0	Y	18.3
SDFC-0408	Metals	Silver	5.86		2	2.9	Y	18.3
SDFC-0408	Metals	Zinc	290		121	2.4	Y	18.3
SDFC-0408	PAHs	HPAHs	0.757	J	0.19	4 0	Y	18.3
SDFC-0408	PAHs	LPAHs	0.1791	J	0.076	2 4	Y	18.3
SDFC-0408	PCBs (total)	Total PCBs	0.148	p	0.0598	2.5	Y	18.3
SDFC-0408	Pesticides	4,4'-DDD	0 016		0.00488	3.3	Y	18.3
SDFC-0408	Pesticides	4,4'-DDE	0.0082	p	0 00316	2.6	Y	18.3
SDFC-0408	Pesticides	Heptachlor	0 001	U	0.0006	1 7	Y	18.3
SDFC-0409	Metals	Barium	52		20	2.6	Y	41.8
SDFC-0409	Metals	Selenium	2.4	j	0.1	24.0	Y	41.8
SDFC-0409	PCBs (total)	Total PCBs	0.061	j	0.0598	1.0	Y	41.8
SDFC-0410	Metals	Barium	54.9		20	2 7	Y	37.6
SDFC-0410	Metals	Mercury	1.78		0.18	9.9	Y	37.6
SDFC-0410	Metals	Selenium	4.4		0.1	44.0	Y	37.6
SDFC-0410	PCBs (total)	Total PCBs	0.088	j	0.0598	1 5	Y	37.6
SDFC-0410	Pesticides	4,4'-DDD	0.017	p	0.00488	3 5	Y	37.6
SDFC-0410	Pesticides	4,4'-DDE	0 005		0.00316	1 6	Y	37.6
SDFC-0411	Metals	Selenium	0 4	j	0 1	4.0	N	2 32
SDFC-0412	Metals	Arsenic	13.8		9 79	1.41	Y	31
SDFC-0412	Metals	Barium	182		20	9.10	Y	31
SDFC-0412	Metals	Manganese	1880		630	2.98	Y	31
SDFC-0412	Metals	Selenium	10		0.1	100.00	Y	31
SDFC-0412	PAHs	HPAHs	0.4905	J	0.19	2.58	Y	31

Table E6-9. Concentrations of Chemicals at Bioassay Stations that Exceed SQGs

Station ID	Analytical Group	Analyte	Sed. Conc. mg/kg	Qual	Screening value (mg/kg)	Exceedance Factor	Growth Effect?	TOC
SDPB-0404	Metals	Selenium	0.7		0.1	7.00	N	0.83
SDPB-0407	Metals	Selenium	0.2		0.1	2.00	N	.0.34
SDREF-0401	Metals	Barium	26 5		20	1.33	R	1 62
SDREF-0401	PAHs	HPAHs	6.597		0.19	34.72	R	1 62
SDREF-0401	PAHs	LPAHs	0 7691		0.076	10.12	R	1 62
SDREF-0401	PAHs	Pyrene	1 4		0.195	7.18	R	1.62
SDREF-0401	PAHs	TPAHs	7.3661		1.61	4.58	R	1.62
SDREF-0402	Metals	Barium	31 4		20	1.57	R	1 32
SDREF-0402	Metals	Selenium	0.2	j	0.1	2.00	R	1.32
SDREF-0403	Metals	Arsenic	12.6		9.79	1.29	R	21
SDREF-0403	Metals	Barium	110		20	5.50	R	21
SDREF-0403	Metals	Manganese	7240		630	11.49	R	21
SDREF-0403	Metals	Selenium	6.5		0.1	65.00	R	21
SDREF-0403	PAHs	HPAHs	0.2193	J	0.19	1.15	R	21
SDREF-0403	Pesticides	4,4'-DDE	0.0032	j	0 00316	1.01	R	21

Notes: R = Reference station
ND=1/2DL

Table E6-10. Comparison of Concentrations of MLR Predictor Variables in Bioassay Sediments to SQGs

StationID	Analyte Group	Analyte	Concentration (mg/kg)	SQG (mg/kg)	HQ	Growth effect?	Exceeds background? Creek	Exceeds background? Lake	Is this chemical a cause of toxicity?
Chemicals that were predictor variables in one or more multiple linear regression models									
SDFC-0407	Metals	Arsenic	11 35	9.79	1.2	Y	N	N	Unlikely (low HQs), not consistently
SDFC-0403	Metals	Arsenic	11 6	9 79	1 2	Y	N	N	elevated at affected stations, below
SDREF-0403	Metals	Arsenic	12 6	9.79	1.3	R	N	N	background in Fox Creek
SDFC-0412	Metals	Arsenic	13.8	9 79	1 4	Y	N	N	
SDREF-0401	Metals	Barium	26.5	20	1.3	R	N	Y	unlikely, exceeds SQG in affected and
SDCH-0403	Metals	Barium	29 5	20	1 5	N	N	Y	unaffected stations
SDREF-0402	Metals	Barium	31.4	20	1 6	R	N	Y	
SDCH-0404	Metals	Barium	39 6	20	2 0	N	N	Y	
SDFC-0408	Metals	Barium	44 6	20	2 2	Y	N	Y	
SDFC-0403	Metals	Barium	46.2	20	2 3	Y	N	Y	
SDFC-0409	Metals	Barium	52	20	2.6	Y	N	Y	
SDFC-0410	Metals	Barium	54.9	20	2 7	Y	N	Y	
SDFC-0405	Metals	Barium	64.3	20	3 2	Y	N	Y	
SDFC-0406	Metals	Barium	74.5	20	3 7	Y	N	Y	
SDFC-0407	Metals	Barium	81 1	20	4 1	Y	N	Y	
SDREF-0403	Metals	Barium	110	20	5.5	R	N	Y	
SDFC-0402	Metals	Barium	122	20	6.1	Y	N	Y	
SDFC-0412	Metals	Barium	182	20	9 1	Y	N	Y	
SDCH-0402	Metals	Barium	191	20	9 6	N	N	Y	
SDCH-0403	PAHs	HPAHs	0 21415	0 19	1 1	N	NA	NA	Unlikely. Highest HQs are at non toxic
SDREF-0403	PAHs	HPAHs	0 2193	0 19	1 2	R	NA	NA	or reference stations
SDFC-0406	PAHs	HPAHs	0 2619	0 19	1.4	Y	NA	NA	
SDFC-0412	PAHs	HPAHs	0 4905	0.19	2 6	Y	NA	NA	
SDFC-0408	PAHs	HPAHs	0 757	0.19	4 0	Y	NA	NA	
SDFC-0401	PAHs	HPAHs	1 019	0.19	5 4	N	NA	NA	
SDFC-0407	PAHs	HPAHs	1.272	0 19	6 7	Y	NA	NA	
SDFC-0403	PAHs	HPAHs	1 312	0.19	6 9	Y	NA	NA	
SDFC-0404	PAHs	HPAHs	1 3442	0.19	7.1	N	NA	NA	
SDFC-0402	PAHs	HPAHs	1.891	0.19	10 0	Y	NA	NA	
SDCH-0401	PAHs	HPAHs	2.525	0.19	13.3	N	NA	NA	
SDCH-0402	PAHs	HPAHs	4.29	0 19	22.6	N	NA	NA	
SDREF-0401	PAHs	HPAHs	6 597	0.19	34 7	R	NA	NA	

Table E6-10 Comparison of Concentrations of MLR Predictor Variables in Bioassay Sediments to SQGs

StationID	Analyte Group	Analyte	Concentration (mg/kg)	SQG (mg/kg)	HQ	Growth effect?	Exceeds background? Creek	Exceeds background? Lake	Is this chemical a cause of toxicity?
Chemicals that were individually correlated with growth using Kendall's tau-b									
SDFC-0408	Metals	Lead	60.3	35.8	1 7	Y	E	NA	Unlikely· low HQ and not consistently high at toxic stations
SDFC-0406	PAHs	LPAHs	0 0842	0.076	1 1	Y	NA	NA	Unlikely highest exceedance at reference station and next four highest are non toxic Pattern not consistent with HPAH pattern of exceedances.
SDFC-0401	PAHs	LPAHs	0.1487	0 076	2 0	N	NA	NA	
SDFC-0408	PAHs	LPAHs	0 1791	0.076	2 4	Y	NA	NA	
SDFC-0407	PAHs	LPAHs	0.27655	0.076	3 6	Y	NA	NA	
SDFC-0402	PAHs	LPAHs	0 2995	0 076	3.9	Y	NA	NA	
SDCH-0401	PAHs	LPAHs	0 422	0.076	5 6	N	NA	NA	
SDFC-0404	PAHs	LPAHs	0 495	0.076	6 5	N	NA	NA	
SDREF-0401	PAHs	LPAHs	0.7691	0.076	10 1	R	NA	NA	
SDCH-0402	PAHs	LPAHs	1 617	0.076	21 3	N	NA	NA	
SDFC-0403	PAHs	LPAHs	22 3425	0.076	294.0	Y	NA	NA	
SDFC-0402	Metals	Manganese	984	630	1.6	Y	N	N	Unlikely (low HQs); below background in Fox Creek
SDFC-0412	Metals	Manganese	1880	630	3.0	Y	N	N	
SDREF-0403	Metals	Manganese	7240	630	11 5	R	N	N	
SDFC-0401	Metals	Selenium	0 2	0 1	2 0	N	E	N	Possibly Highest exceedances are at stations with growth effects; stations without effects show relatively small exceedances
SDREF-0402	Metals	Selenium	0 2	0 1	2 0	R	E	N	
SDFC-0411	Metals	Selenium	0 4	0.1	4 0	N	E	N	
SDCH-0402	Metals	Selenium	0.5	0.1	5 0	N	E	N	
SDCH-0401	Metals	Selenium	1	0 1	10.0	N	E	N	
SDFC-0409	Metals	Selenium	2.4	0.1	24.0	Y	E	N	
SDFC-0408	Metals	Selenium	3.1	0.1	31.0	Y	E	N	
SDFC-0410	Metals	Selenium	4 4	0.1	44.0	Y	E	N	
SDFC-0406	Metals	Selenium	4.9	0.1	49.0	Y	E	N	
SDFC-0403	Metals	Selenium	5.5	0.1	55.0	Y	E	N	
SDREF-0403	Metals	Selenium	6 5	0 1	65 0	R	E	N	
SDFC-0402	Metals	Selenium	7 5	0.1	75.0	Y	E	N	
SDFC-0407	Metals	Selenium	8.8	0.1	88 0	Y	E	N	
SDFC-0412	Metals	Selenium	10	0.1	100.0	Y	E	N	
SDFC-0405	Metals	Selenium	10 4	0.1	104.0	Y·	E	N	
SDFC-0407	Metals	Silver	5.09	2	2.5	Y	Y	NA	Unlikely low HQ and not consistently high at toxic stations.
SDFC-0408	Metals	Silver	5 86	2	2 9	Y	Y	NA	

Table E6-10. Comparison of Concentrations of MLR Predictor Variables in Bioassay Sediments to SQGs

StationID	Analyte Group	Analyte	Concentration (mg/kg)	SQG (mg/kg)	HQ	Growth effect?	Exceeds background? Creek	Exceeds background? Lake	Is this chemical a cause of toxicity?
Chemicals that were individually correlated with growth using Kendall's tau-b									
SR-SDPB-0407	Metals	Antimony	26 5	3	8.8	N	NA	Y	Unlikely not consistently elevated at toxic stations
SDFC-0407	Metals	Cadmium	1 235	0.99	1 2	Y	E	NA	Unlikely not consistently elevated at
SDFC-0408	Metals	Cadmium	1.27	0 99	1 3	Y	E	NA	toxic stations
SDFC-0408	Metals	Copper	50 8	31 6	1.6	Y	E	NA	Unlikely not consistently elevated at
SDFC-0407	Metals	Copper	59 65	31 6	1 9	Y	E	NA	toxic stations
SDFC-0407	Metals	Mercury	0 9835	0 18	5.5	Y	Y	NA	Unlikely not consistently elevated at
SDFC-0410	Metals	Mercury	1.78	0 18	9 9	Y	Y	NA	toxic stations
SDFC-0407	Metals	Zinc	372.5	121	3 1	Y	E	NA	Unlikely not consistently elevated at
SDFC-0408	Metals	Zinc	290	121	2 4	Y	E	NA	toxic stations

Note: E = Results of comparisons to background were equivocal (inconclusive)
NA = Not applicable

Table E6-12 Sediment and Dietary Nutrient Analysis in Selected Cass Lake Sediments

	Sediment and TetraFin Analysis								*Dietary Analysis*					*Growth Data*	
Station	Solids (%)	TOC (%)	Organic C (mg/kg)	Organic N[a] (mg/kg)	Organic P[b] (mg/kg)	C/N	N/P	C/P	Total Available Organic Matter (g dw/100ml)[c]	Proportion of Fish Food in Daily Diet[d]	Dietary C/N	Dietary N/P	Dietary C/P	Mean Individual AFDW	AFDW St. Dev
SDFC-0403	11	27	2 7E+05	2 0E+04	62	14	325	4403	0 030	0.17	11	270	3002	0 43	0.1
SDFC-0404	76	0 80	8 0E+03	4 1E+02	21	20	19	380	0 0061	0 50	7 3	198	1439	1 03	0 13
SDFC-0406	12	42	4.2E+05	3 0E+04	26	14	1142	16308	0 050	0 11	13	611	7875	0.5	0 14
SDFC-0408	21	19	1 9E+05	1 2E+04	32	15	387	5891	0 039	0 13	12	285	3344	0 69	0 1
SDFC-0410	13	41	4 1E+05	3 5E+04	5	12	6957	81200	0 052	0 10	11	1082	11800	0 66	0.07
SDFC-0412	10	31	3.1E+05	2 0E+04	26	16	768	12019	0 030	0 17	12	384	4768	0 45	0 13
SDREF-0401	68	1 5	1.5E+04	9.1E+02	22	16	42	678	0 010	0.37	7 4	192	1422	0.99	0.18
SDREF-0403	10	21	2 1E+05	1 3E+04	26	17	484	8173	0 022	0 22	11	279	3158	0.86	0.17
TetraFin	96	44	4 4E+05	6 1E+04	290	7 2	211	1517	N/A	N/A	N/A	N/A	N/A	N/A	N/A

Notes: NA = not applicable

[a]Calculated as Kjeldahl nitrogen minus amonia nitrogen

[b]Calculated as total phosphorus minus hydrolyzable phosphorus

[c]Calculated as the product of total solids and %TOC divided by 100 based on the assumption that 1/100th of organic solids are available as food, and the average density of sediments is 1g/ml

[d]Calculated as the mass of fish food per day (0 006g) divided by the summed masses of total available organic matter and fish food

Table E6-13. Kendall's tau-b Correlation Matrix for Selected Constituents in Eight Sediment Stations

Analyte	TOC	Dietary N/C	Dietary P/C
PCDD, Total	0 00	-0.36	-0 07
PCDF, Total	0.14	-0.21	-0 36
Aluminum	0 21	-0.43	-0 14
Antimony	0 50	**-0.71**	**-0.57**
Arsenic	0.43	-0.50	-0.36
Barium	**0.64**	**-0.57**	-0 43
Beryllium	0.36	-0.43	-0.29
Cadmium	0.43	**-0.79**	-0.50
Chromium	0 36	**-0.57**	-0.29
Cobalt	0 21	-0.43	-0 14
Copper	0 43	-0.50	**-0.64**
Iron	0.00	-0.21	-0 07
Lead	0.21	**-0.71**	-0 43
Manganese	0.36	-0.43	-0.43
Mercury	0.25	-0.18	-0.55
Molybdenum	0 50	-0.43	-0.14
Nickel	0.29	-0.50	-0.21
Selenium	0.47	-0.55	-0 33
Silver	0 43	-0.50	**-0.64**
Thallium	0.21	-0.43	-0.14
Vanadium	0.36	-0.43	-0 29
Zinc	0.29	**-0.64**	-0.50
Benzo(a)anthracene	**-0.57**	0.36	**0.79**
Benzo(a)pyrene	**-0.57**	0.36	**0.79**
Benzo(b)fluoranthene	-0 50	0.29	**0.71**
Benzo(g,h,i)perylene	-0 43	0.21	**0.64**
Benzo(k)fluoranthene	-0.50	0.29	**0.71**
Chrysene	-0.55	0.33	**0.76**
Dibenz(a,h)anthracene	-0.43	0.21	**0.64**
Fluoranthene	**-0.57**	0.36	**0.79**
Indeno(1,2,3-cd)pyrene	-0 43	0.21	**0.64**
Pyrene	**-0.57**	0.36	**0.79**
HPAHs	**-0.57**	0.36	**0.79**
LPAHs	-0.29	0.21	0 50
TPAHs	-0.43	0.21	**0.64**
Total PCBs (Aroclors)	**0.55**	-0.55	**-0.69**
Total Organic Carbon	1.00	-0.50	**-0.64**
Dietary N/C	-0.50	1.00	**0.57**
Dietary P/C	**-0.64**	**0.57**	1.00
N/C	0 57	-0.21	-0.50
P/C	**-0.79**	**0.57**	**0.86**

Note: Significant correlations (at alpha= 0.05) are in **bold**.

Table E6-14. ANOVA for Log_{10}-Transformed Total HPAH and Dietary P/C Predicting Growth

Source	DF	Sum of squares	Mean squares	F	p-value
Analysis of variance					
Model	2	0.34	0.17	15	0.008
Error	5	0.057	0.011		
Corrected Total	7	0.40			
Type III Sum of Squares analysis					
Log_{10}HPAH	1	0.096	0 096	8 4	0.03
dietary P/C 100	1	0.28	0 28	24	0 004

Table E6-15. Regression Analysis Model Parameters for Log_{10}-Transformed HPAH and Dietary P/C predicting Growth

Source	Value	Std. Error	t-value	p-value	Adjusted $R^2 = 0.86$	AIC
Model Parameters					0 798	-8.8
Intercept	0 077	0.14	0.550	0 61		
Log_{10}HPAH	-0.38	0.13	-2 9	0.034		
Dietary P/C 100	1578	321	4.9	0.0044		
Standardized coefficients						
Log_{10}HPAH	-0.92	0.32	-2 9	0.03		
dietary P/C 100	1 6	0.32	4.9	0.004		

Table E6-16. Candidate Causes of Growth Effect Resulting from Each Analytical Step

	Chemicals that individually correlate significantly with growth (Kendall's tau-b)	Predictor Variables in all possible MLR Models	Comparison of Correlates and Predictors to SQGs	Analysis of the Nutritional Quality of the Diet	SQGs not Available
TOC	X	X	na	X	X
aluminum					
antimony	X				
arsenic	X	X			
barium		X	X		
beryllium					
cadmium	X				
chromium	X	X			
cobalt		X			
copper	X				
iron					
lead	X	X			
manganese		X			
mercury	X				
molybdenum	X				X
nickel	X	X			
selenium	X		X		
silver	X	X			
thallium	X				X
vanadium	X				
zinc	X				
LPAH		X			
HPAH		X		X	
2 methylnaphthalene[a]	X				X
PCBs					
total PCDD					
Total PCDDF					
Total DDx		NA			
p,p' DDT		NA			
p,p'-DDE		NA			
p,p'-DDD		NA			

Notes: NA = Not applicable

[a]Correlations between individual PAH compounds and growth were evaluated, and none were significant other than 2-methylnaphthalene. Regression analyses and comparison to SQGs was performed using LPAH and HPAH as aggregates (Attachment A), so individual PAH compounds are not listed here.

ATTACHMENT E6A

PRINCIPAL COMPONENTS ANALYSIS WITH SEDIMENT CHEMISTRY DATA

HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT

St. Regis Paper Company Site
Cass Lake, MN

Attachment E6A
Principal Components Analysis with Sediment Chemistry Data

Prepared for
International Paper
pursuant to
Unilateral Administrative Order Docket No. V-W-04-C-796

Prepared by

integral consulting inc.

12303 Airport Way
Suite 370
Broomfield, CO 80021

September 28, 2007

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1 INTRODUCTION

Principal components analysis (PCA), a technique for reducing multiple sets of correlated parameters to a smaller number of uncorrelated parameters that aggregate information from the related variables, was employed to evaluate patterns of chemical co-occurrence in sediment. Results of two PCAs were employed to facilitate subsequent analyses, and are reported in this attachment:

- A PCA on chemical parameters for only bioassay stations. This PCA was performed to evaluate general patterns in sediment chemistry, and to determine whether aggregate expressions of sediment PAHs, rather than individual PAH compounds, could be used in the MLR.
- A PCA on the chemical parameters at all sediment stations to determine whether patterns in chemical concentrations at stations tested for toxicity are similar to patterns at stations that have not been tested for toxicity. Similarity among chemical patterns across all sediment stations would support application of an MLR model developed using only bioassay stations to predict toxicity at chemistry-only stations.

The results of the second PCA inform application of the multiple linear regression (MLR) models derived later in this appendix. The application of the MLR is discussed in Section 5 of the risk assessment report.

2 PCA FOR AGGREGATING PAH COMPOUNDS

The objectives of this analysis are to gain a general understanding of whether there are different suites of chemicals detected in sediments, and whether sums of low-molecular-weight polycyclic aromatic hydrocarbons (LPAH) and high-molecular-weight (HPAH) adequately capture the variability of the concentrations of PAHs in sediments. This analysis evaluates whether grouping PAH into aggregate values for the MLR is valid.

2.1 PCA METHODS

PCA is most useful for data that are approximately normally distributed; it can be strongly influenced by data with asymmetrical distributions. The distributions of chemical data in the bioassay data set are right-skewed. Thus, all chemical variables were $\log_{10}$-transformed prior to running PCAs to reduce the effects of outliers.

When the original variables have widely differing ranges or units of measure, the PCA should be based on the correlation matrix rather than the covariance matrix. If the covariance matrix were used on a data set with widely disparate units of measure, the variables with the widest range and therefore largest variability would drive the principal component results. The correlation matrix was used for PCAs described in this appendix.

The analysis was applied to the bioassay station data set, including chemistry, TOC and grain size (Table E6A-1). Chemicals other than PAH compounds with a detection frequency less than 50% were excluded from this analysis to avoid results strongly influenced by censored values; this resulted in elimination of the pesticides and PCBs (Table E6A-1). Benzo(j)fluoranthene was eliminated because there were only six records in the data set. Naphthalene was retained in some of the analyses because it is included in the PAH sums, and it may be an influential chemical. The analyses described below were run both including and excluding naphthalene.

Table E6A-1. Detection Frequencies of Chemicals Included in PCA to Determine Patterns in PAH Compounds in the Bioassay Dataset

Splus code	Original name	Units	Detection Frequency (n=27)	Comments
baa	Benzo(a)anthracene	mg/kg	93%	
bap	Benzo(a)pyrene	mg/kg	89%	
bbf	Benzo(b)fluoranthene	mg/kg	81%	
bjf	Benzo(j)fluoranthene	mg/kg	29%	omit; n=6
bghip	Benzo(g,h,i)perylene	mg/kg	85%	
bkf	Benzo(k)fluoranthene	mg/kg	81%	
chrysene	Chrysene	mg/kg	85%	
dibenzaha	Dibenz(a,h)anthracene	mg/kg	67%	
fluoranthene	Fluoranthene	mg/kg	100%	
icdp	Indeno(1,2,3-cd)pyrene	mg/kg	85%	
pyrene	Pyrene	mg/kg	100%	
2methnap	2-Methylnaphthalene	mg/kg	85%	
acenapthene	Acenaphthene	mg/kg	74%	
acenaptyle	Acenaphthylene	mg/kg	81%	
anthracene	Anthracene	mg/kg	85%	
fluorene	Fluorene	mg/kg	78%	
napthalene	Naphthalene	mg/kg	30%	Analyses run both with and without
phenanthrene	Phenanthrene	mg/kg	100%	
aluminum	Aluminum	mg/kg	100%	
antimony	Antimony	mg/kg	89%	
arsenic	Arsenic	mg/kg	100%	
barium	Barium	mg/kg	100%	
beryllium	Beryllium	mg/kg	100%	
cadmium	Cadmium	mg/kg	63%	
chromium	Chromium	mg/kg	100%	
cobalt	Cobalt	mg/kg	100%	
copper	Copper	mg/kg	100%	
iron	Iron	mg/kg	100%	
lead	Lead	mg/kg	100%	
manganese	Manganese	mg/kg	100%	
mercury	Mercury	mg/kg	56%	

Table E6A-1. Detection Frequencies of Chemicals Included in PCA to Determine Patterns in PAH Compounds in the Bioassay Dataset

Splus code	Original name	Units	Detection Frequency (n=27)	Comments
molybdenum	Molybdenum	mg/kg	100%	
nickel	Nickel	mg/kg	100%	
selenium	Selenium	mg/kg	67%	
silver	Silver	mg/kg	67%	
thallium	Thallium	mg/kg	96%	
vanadium	Vanadium	mg/kg	100%	
zinc	Zinc	mg/kg	100%	
total.pcdd	Total PCDD	mg/kg	96%	
total.pcdf	Total PCDF	mg/kg	96%	
clay	Clay	pct	100%	omit; included in fines
gravel	Gravel	pct	100%	
sand	Sand	pct	100%	omit
silt	Silt	pct	100%	omit; included in fines
fines	Sum of clay + silt	pct	100%	
toc	Total Organic Carbon	pct	100%	

PCA requires that all data points have a value (i.e., all cells in a matrix of chemicals by samples must be populated). Samples with concentrations below detection limits must be assigned a value. Three non-detect substitution methods were used:

- Substitution at the detection limit
- Substitution at half of the detection limit
- Substitution of all non-detects to a common value—This technique assigns a common concentration to all non-detected samples based on the set of detection limits. Using a value greater than the minimum detection limit results in assigning higher concentrations than we know to be true to some of the samples. Thus, the minimum detection limit or half of the minimum detection limit are appropriate values. Note that substitution at a low level will also overestimate variance among nondetects, as above.

Substitution of nondetects at one-half the detection limit is consistent with the treatment of nondetects in the calculation of PAH sums. Results using substitution at the full detection limit provide a means of evaluating the range of influence of nondetects on the PCA. Because both of these methods may inflate the variance of the low chemical concentrations, a method assigning one common value to all nondetect values (e.g., substituting detection limits with the minimum detection limit for the chemical) was included to provide additional insight. Thus, three methods of non-detect substitution were applied to all analyses: detection limit, half of the detection limit, and half of the minimum detection limit. If results from the three substitution methods show substantial difference, then the PCA would appear to be describing the variance of detection limits.

PCA should not be run on a data set that has more variables than samples because this does not provide enough degrees of freedom to describe the correlation or covariance matrix. Consequently, three sets of PCAs were performed. The first two sets, performed separately on PAHs and metals, were done to evaluate whether those variable sets could be reduced to a smaller set of composite variables that maintained most of the variability in the data set. The final PCA included representations from all chemical groups. Within each set of PCAs, multiple runs were performed to test the effect of the detection limit substitution method:

1) PAHs only
    a. No naphthalene, detection limit substitution
    b. No naphthalene, half detection limit substitution
    c. No naphthalene, half minimum detection limit substitution
    d. Naphthalene included, half minimum detection limit substitution

2) Metals only
    a. Detection limit substitution
    b. Half detection limit substitution
    c. Half minimum detection limit substitution

3) Selected principal components from #1 and #2 combined with remaining chemicals
    a. Half detection limit substitution
    b. Half minimum detection limit substitution

Each PCA analysis is summarized by:

1) The **screeplot**, which shows the relative contribution of each principal component to the reduction of overall variance;
2) The **loadings plot**, which shows the largest coefficients for each principal component (and therefore the strongest correlations)
3) **Biplots** which give a visual interpretation of the selected principal components. The vectors shown on the biplots illustrate the relative strength and direction of the dominant variables in relation to each principal component.

2.2 RESULTS: PAHS

Results of the first set of PCAs on PAH compounds are displayed in Figures E6A1- E6A4. Two principal components are adequate to explain 94-97% of the variance in the four PCA runs. In each case, the first component contains fairly equal amounts of all of the variables – representing the correlation that is present among all PAHs. The second component in each of the four runs represents the combination of PAHs for those samples that deviate from this generally correlative pattern. The PAH compounds that have the highest loading coefficients onto the 2nd PC (i.e., 2-methyl naphthalene, acenaphthene, and fluorene; also naphthalene in the fourth run; all of these are LPAHs) have more variability and are noisier in their correlations with the remaining compounds. These results indicate that little information will be lost by using the sums of HPAHs and LPAHs to summarize the set of PAHs.

2.3 RESULTS: METALS

The results for the set of PCAs on metals are displayed in Figures E6A5-7. The differences among the three runs are minimal, indicating that the substitution method does not overly influence the results. This is not surprising, because most of the metals were detected in all samples. In each run, three principal components are adequate to capture most (i.e., >90%) of the variability. The first component represents positive correlation among most metals. The second and third components represent the combination of metals for those samples that deviate from this generally correlative pattern. The second component identifies where antimony, silver, and mercury (all less than 100% detected) separate from the patterns of iron, cobalt, thallium, and aluminum. The third component identifies where lead and zinc separate from the patterns of primarily selenium, vanadium, molybdenum, antimony, chromium, and thallium. Reduction of the metals data is best summarized using the 1st three PCs.

2.4 RESULTS: ALL CHEMISTRY

This analysis combines data from all chemical groups including: the first two principal components from the PAH PCA, the first three principal components from the metals PCA, total PCDD, total PCDF, gravel, fines, and TOC. Two analyses were run, one with half-DL and one with the half-min DL. Substitution at the DL was not pursued because there was very little difference among substitution methods. Naphthalene is not included because of its very low detection. The results for these final two runs are displayed in Figures E6A8 and E6A9.

Comparison of the two runs using different substitution methods for DLs shows very little difference between them. Three principal components explain 86% of the variance. The first component is again an overall measure of positive correlation among all variables. The second component identifies where *PAH2* (individual LPAHs), *metal2* (antimony, silver, mercury), and *metal3* (selenium, vanadium) deviate from the general correlative pattern. The third component contrasts *metal3* (selenium, vanadium) and *metal1* (all metals) with *metal2* (antimony, silver, mercury) and gravel.

2.5 CONCLUSION

There is a fairly strong correlation among all the PAHs, although some individual LPAHs deviate from this pattern for some stations. The aggregate variables LPAH and HPAH should adequately capture the variation among the individual chemicals in each class, allowing the use of LPAH and HPAH as summary variables in fitting the MLR model to predict growth.

The PCA using all variables indicates that the pattern of increasing chemical contamination is generally correlated with increasing levels of fines and TOC. Several individual LPAH compounds (i.e., 2-methyl naphthalene, acenaphthene, and fluorene) show more variability among samples. The set of metals shows less inter-correlation.

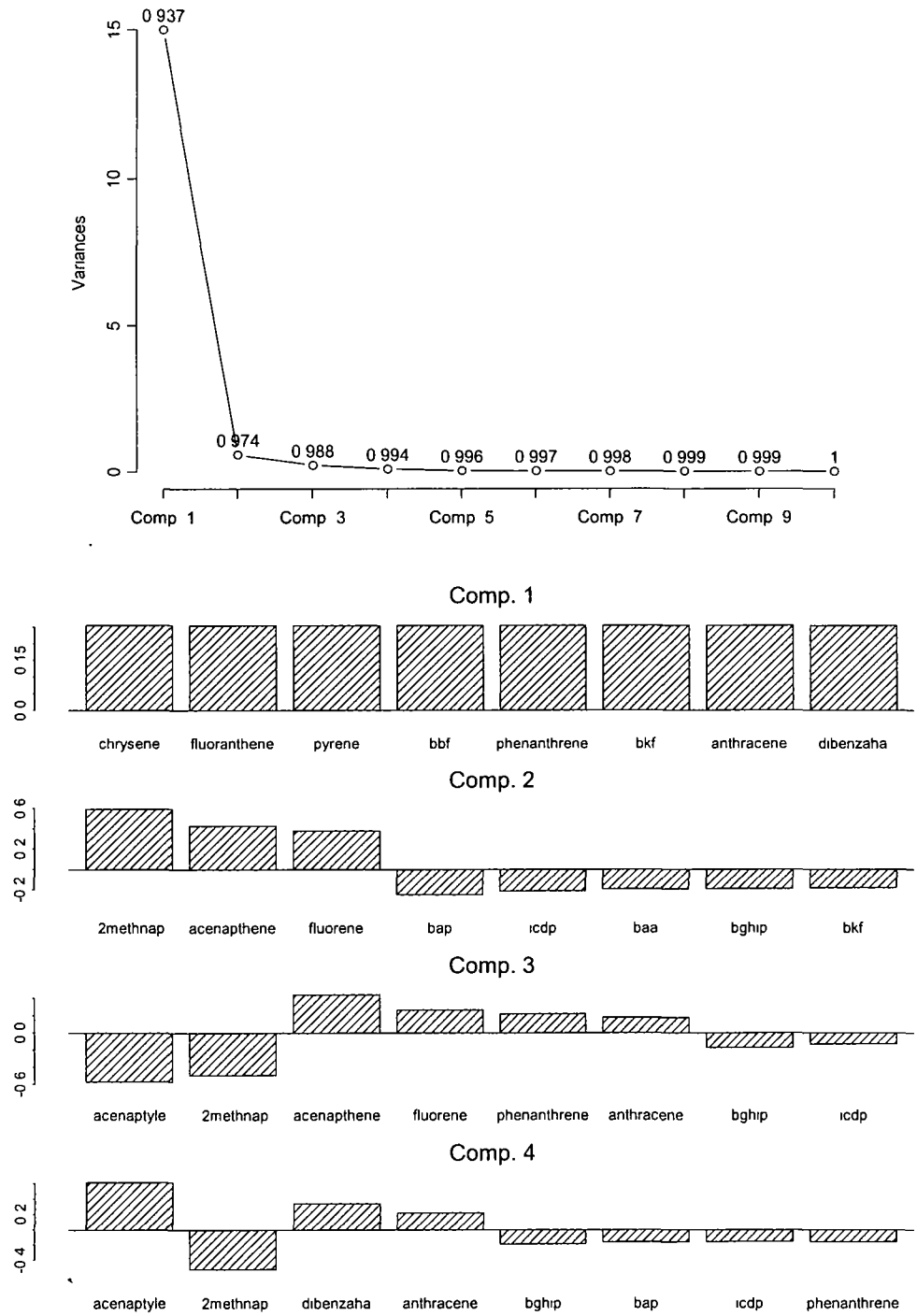

Figure E6A-1a. Screeplot (top) and loadings plot for PCA on PAHs with no naphthalene, DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

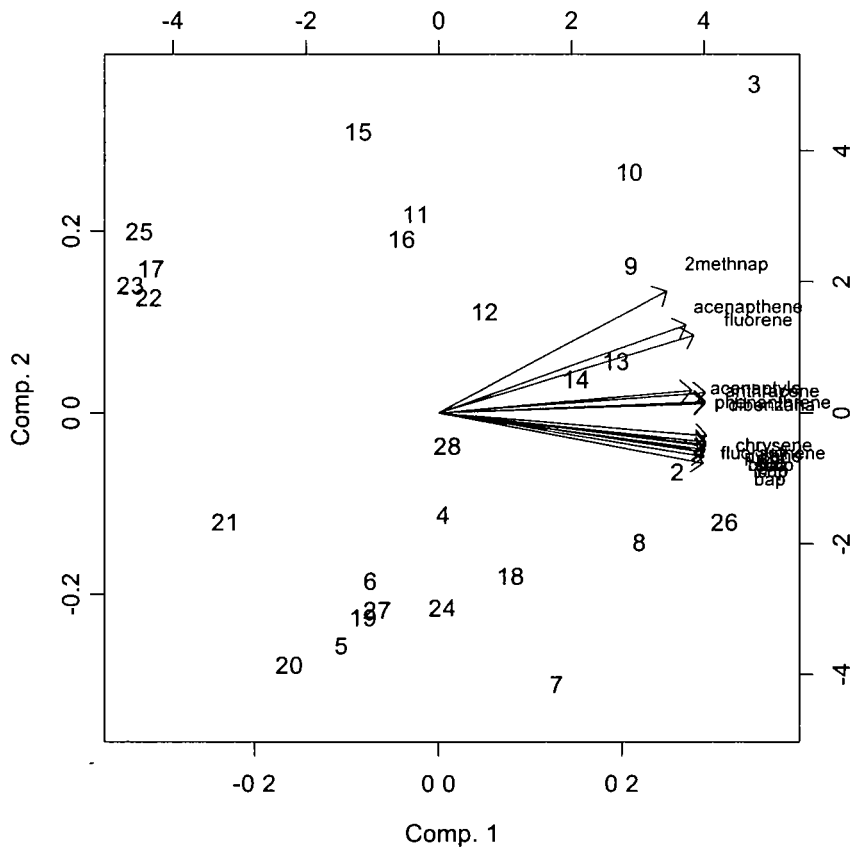

Figure E6A-1b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on PAH data with no napthalene and DL substitution.

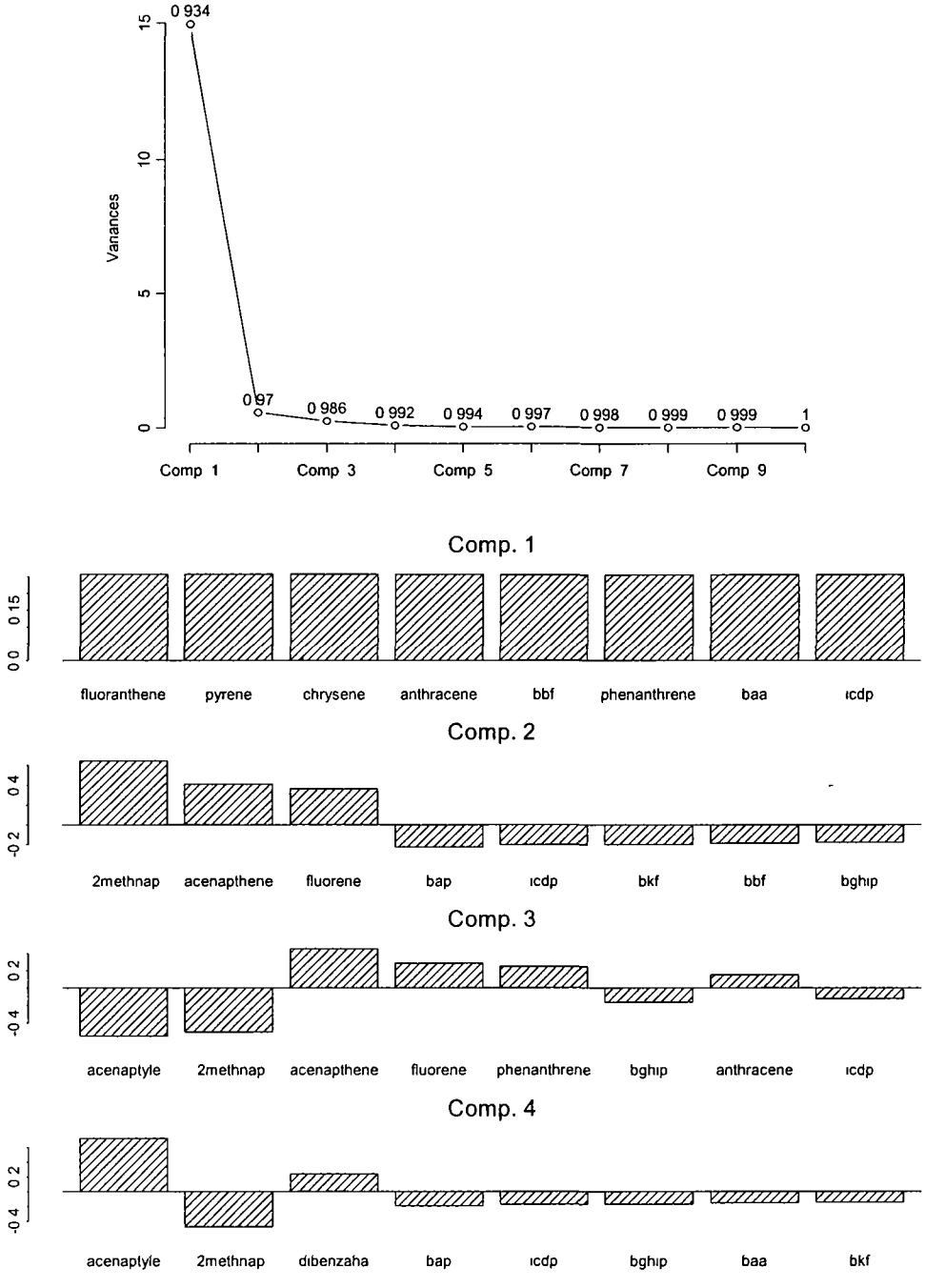

Figure E6A-2a. Screeplot (top) and loadings plot for PCA on PAHs with no naphthalene, ½ DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

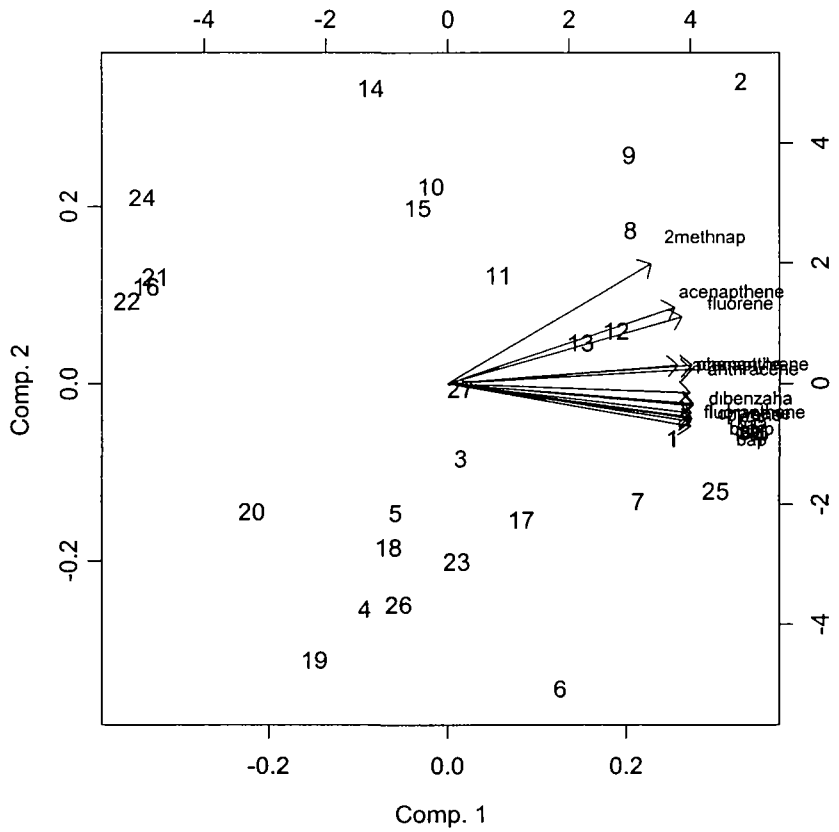

Figure E6A-2b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on PAH data with no napthalene and ½ DL substitution.

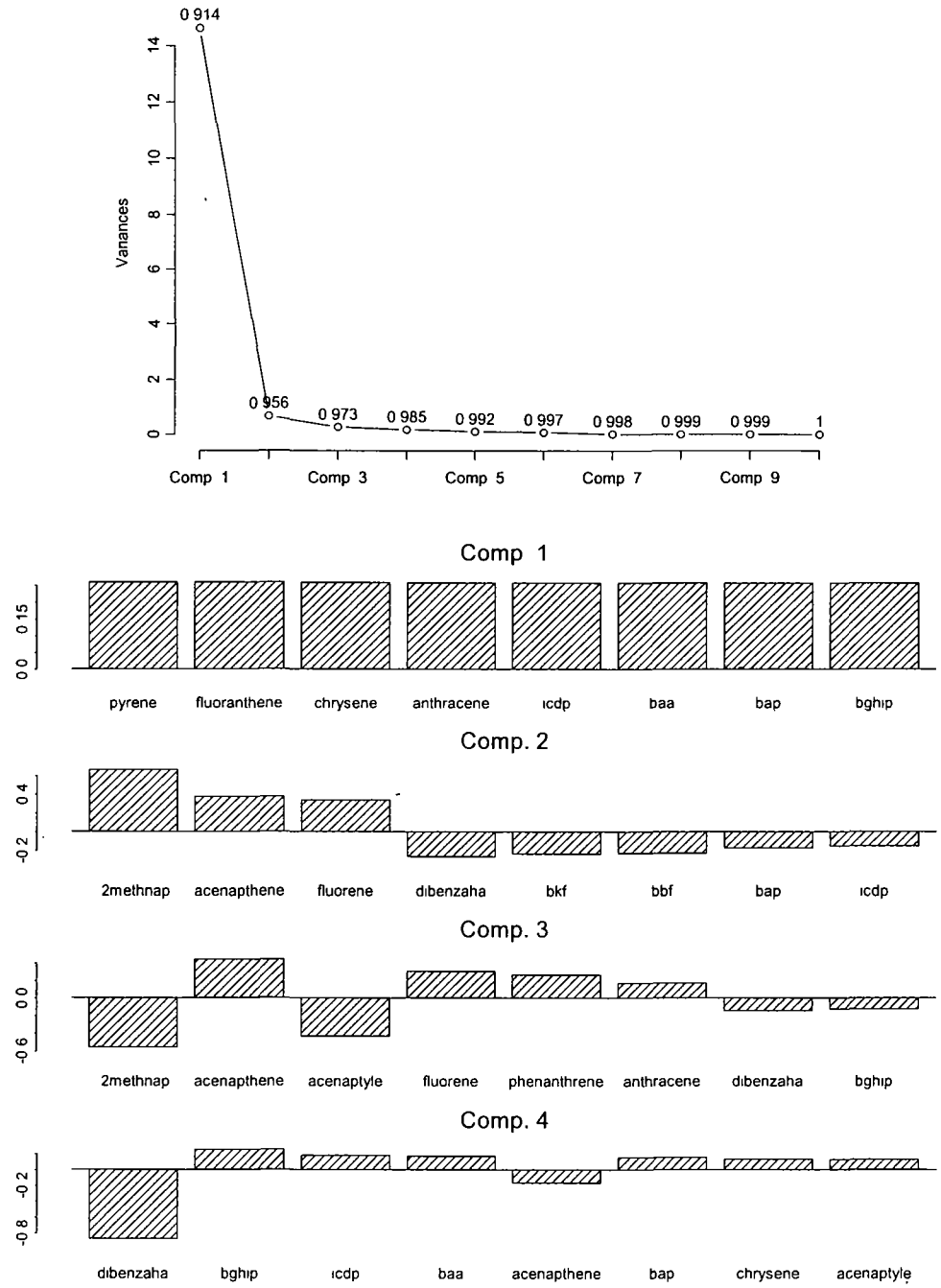

Figure E6A-3a. Screeplot (top) and loadings plot for PCA on PAHs with no naphthalene, half-min DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

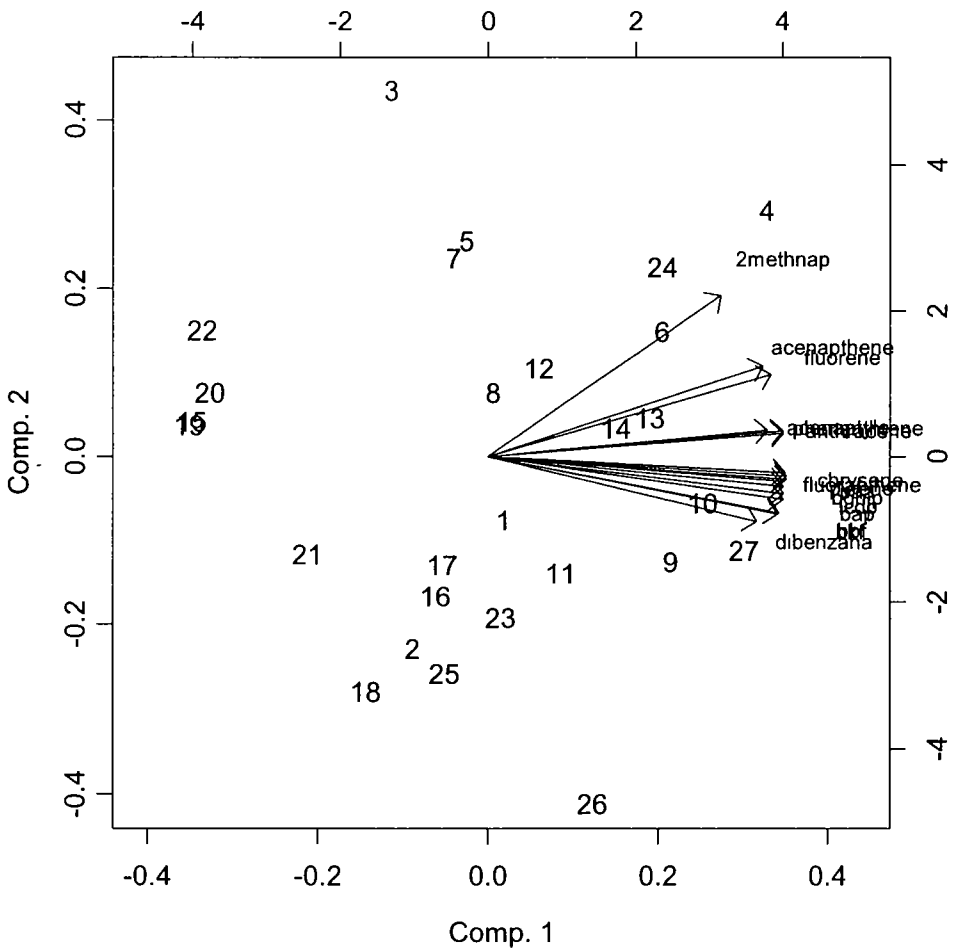

Figure E6A-3b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on PAH data with no napthalene and half-min DL substitution

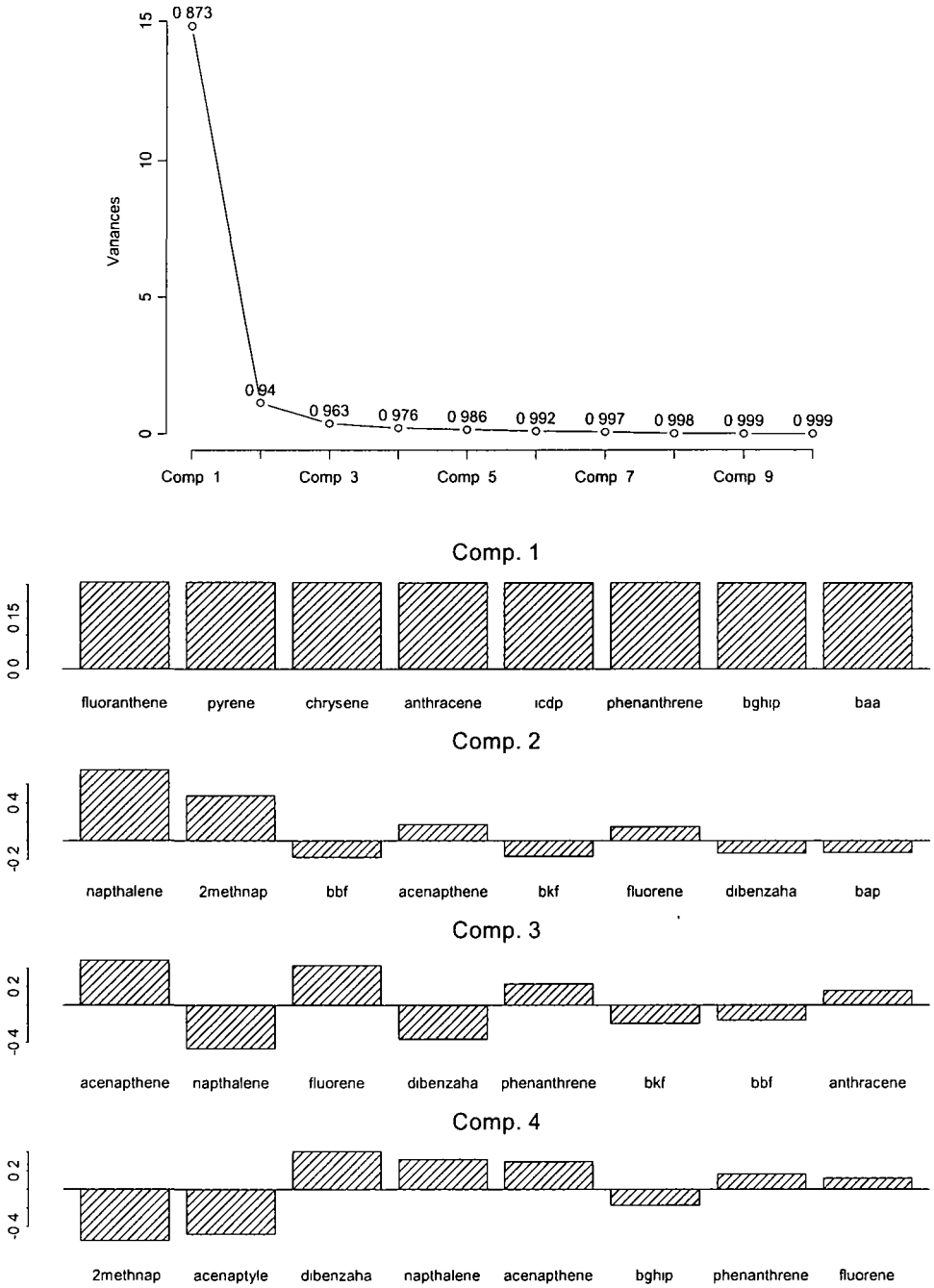

Figure E6A4a Screeplot (top) and loadings plot for PCA on PAHs with naphthalene, half-min DL substitution. The loadings plot includes the top eight variables for each of the first four principal components

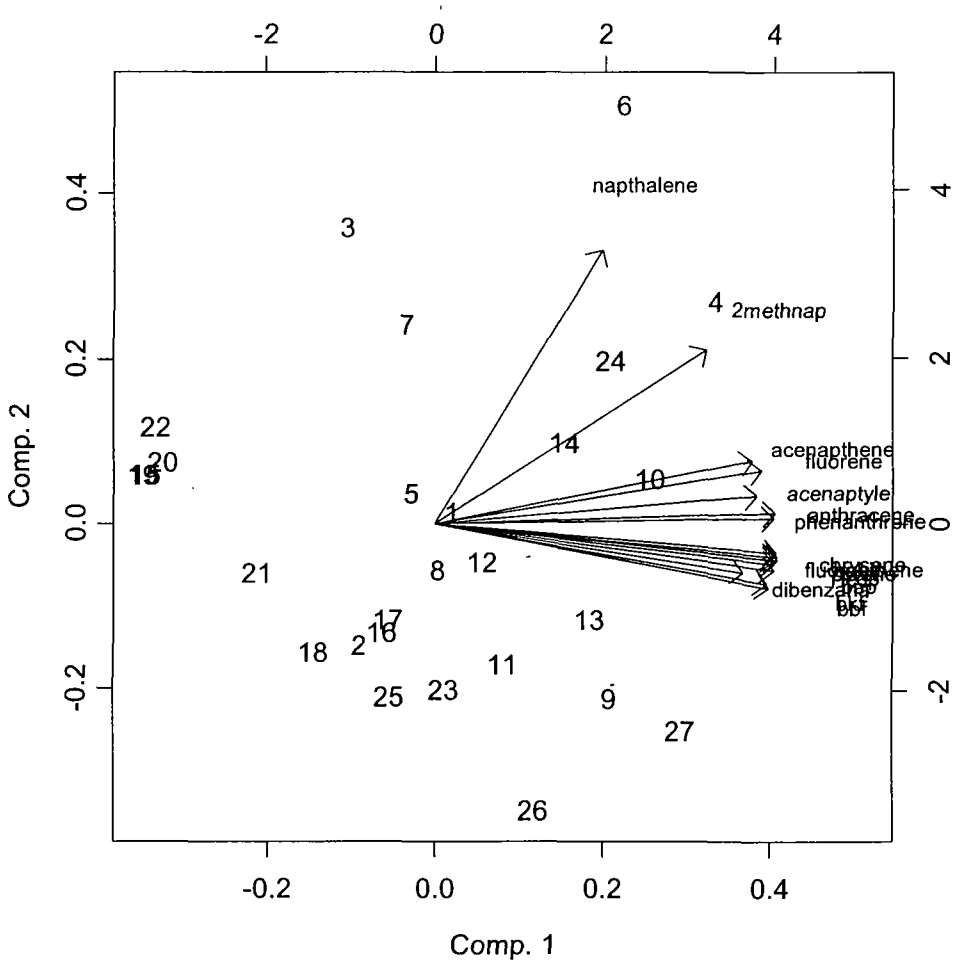

Figure E6A-4b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on PAH data with napthalene and half-min DL substitution.

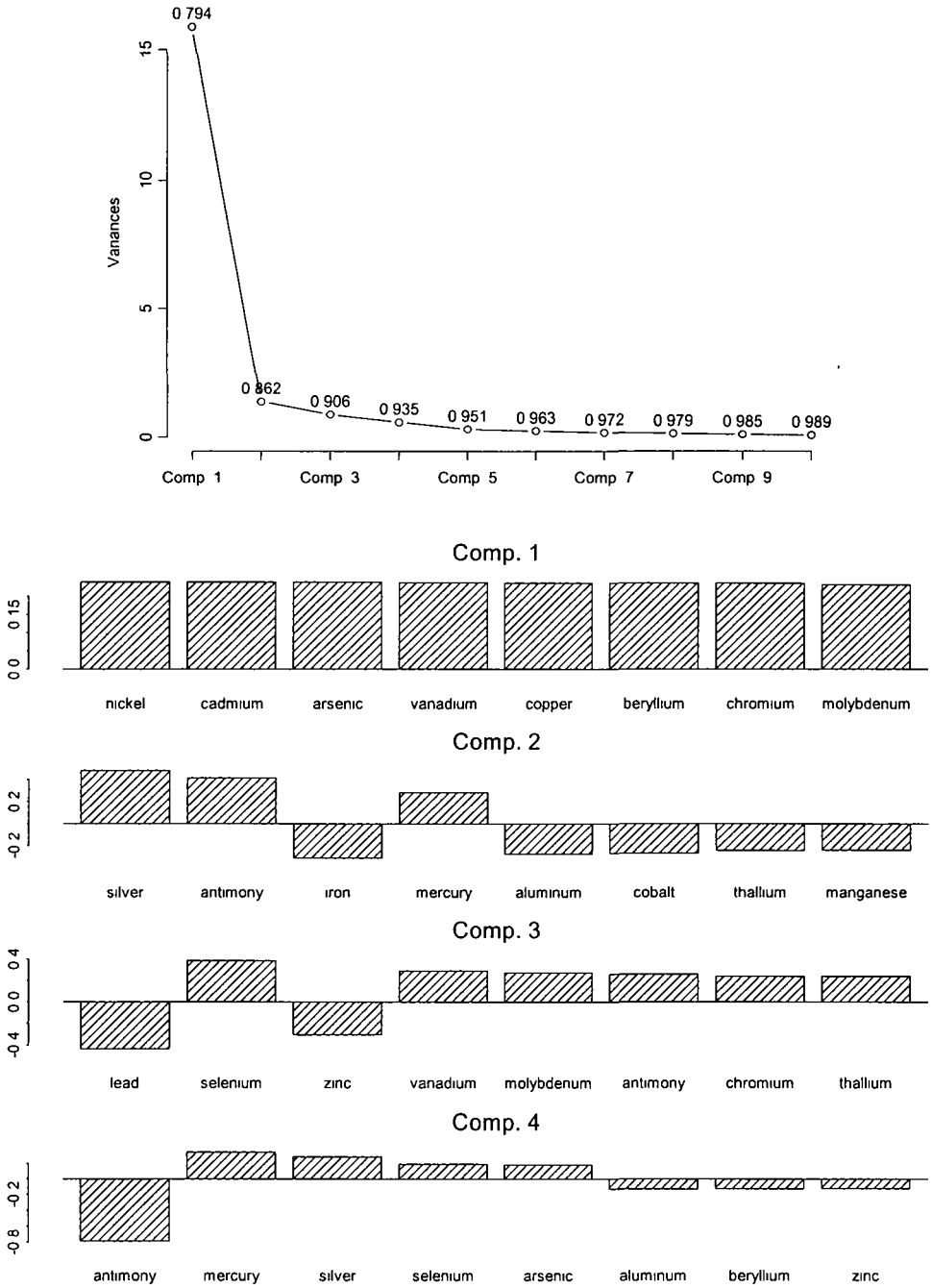

Figure E6A-5a Screeplot (top) and loadings plot for PCA on metals with DL substitution. The loadings plot includes the top eight variables for each of the first four principal components

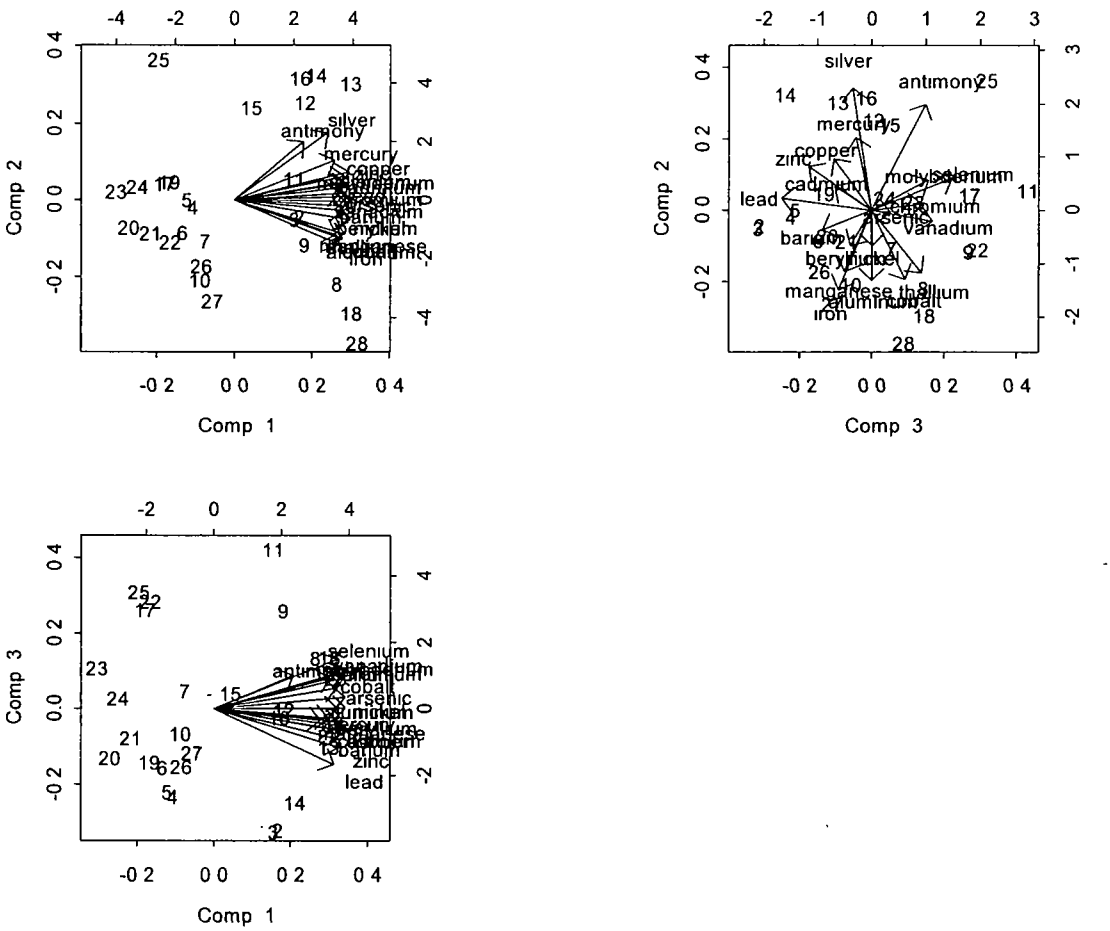

Figure E6A-5b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on metals with DL substitution.

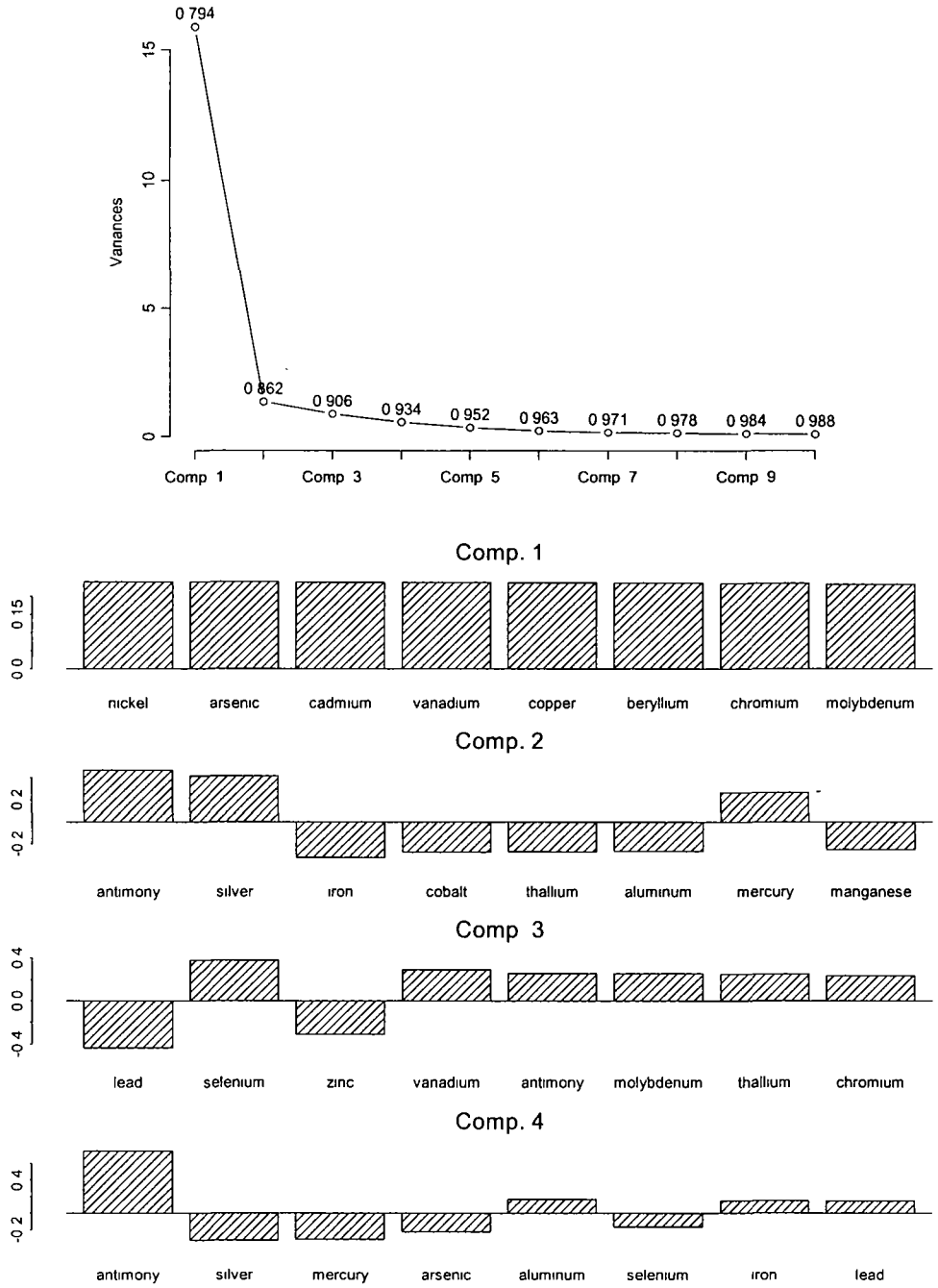

Figure E6A-6a. Screeplot (top) and loadings plot for PCA on metals with ½ DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

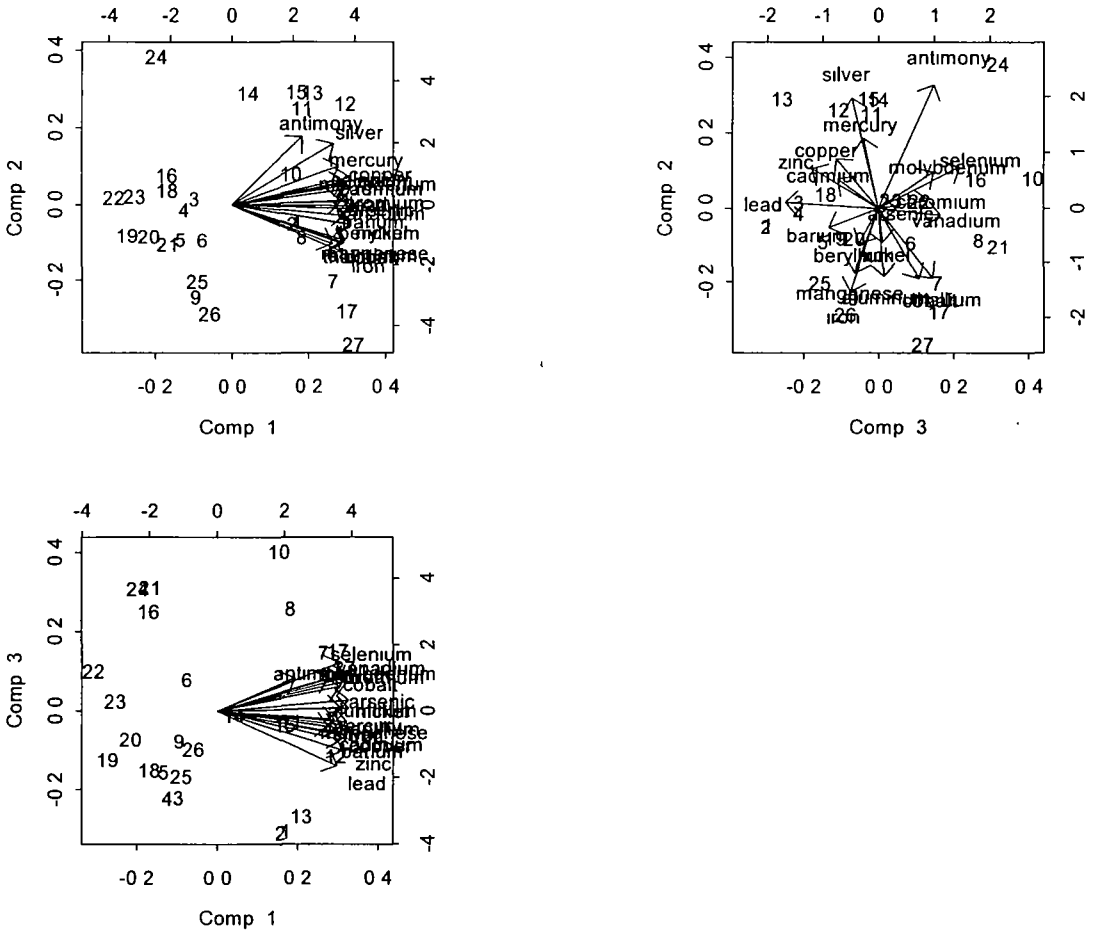

Figure E6A-6b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on metals with ½ DL substitution.

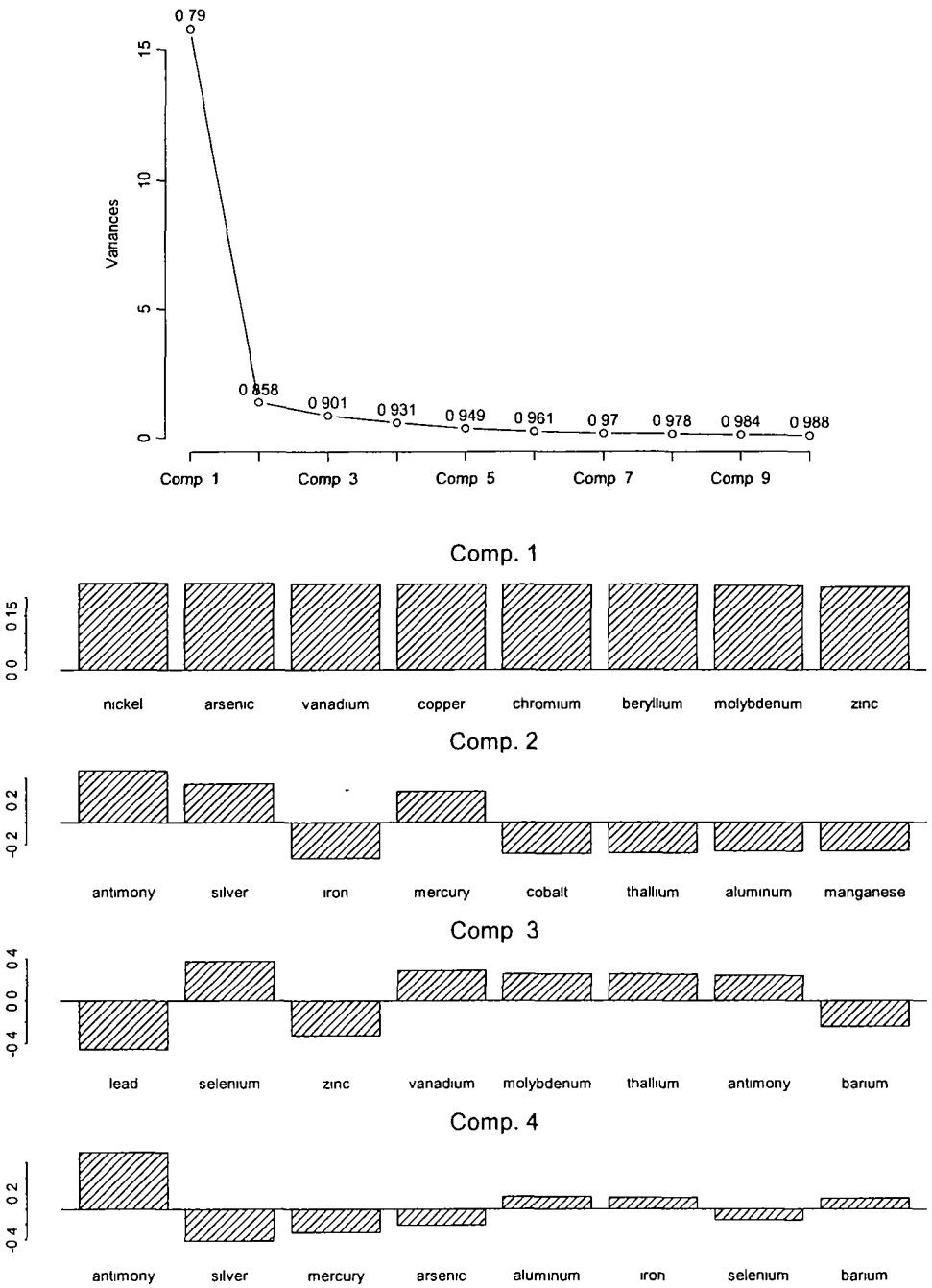

Figure E6A-7a. Screeplot (top) and loadings plot for PCA on metals with half-min DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

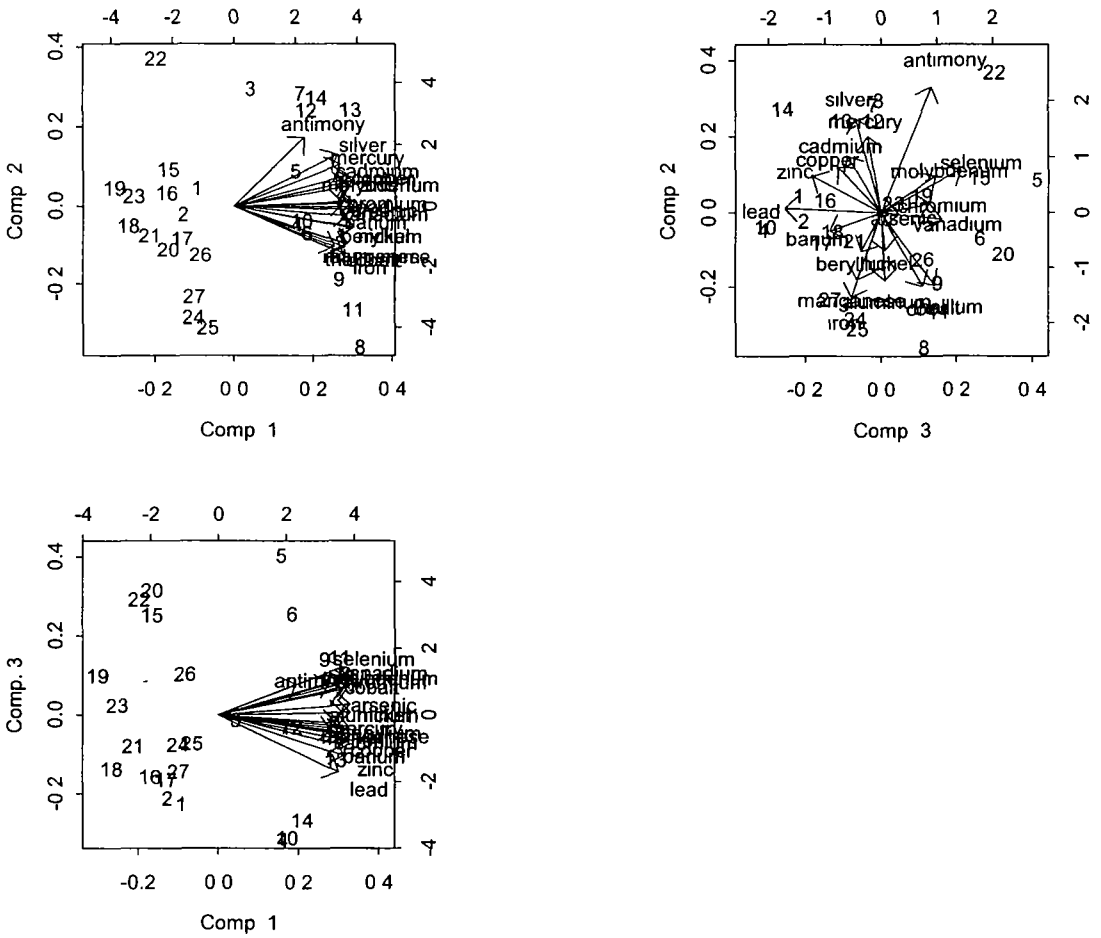

Figure E6A-7b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on metals with half-min DL substitution.

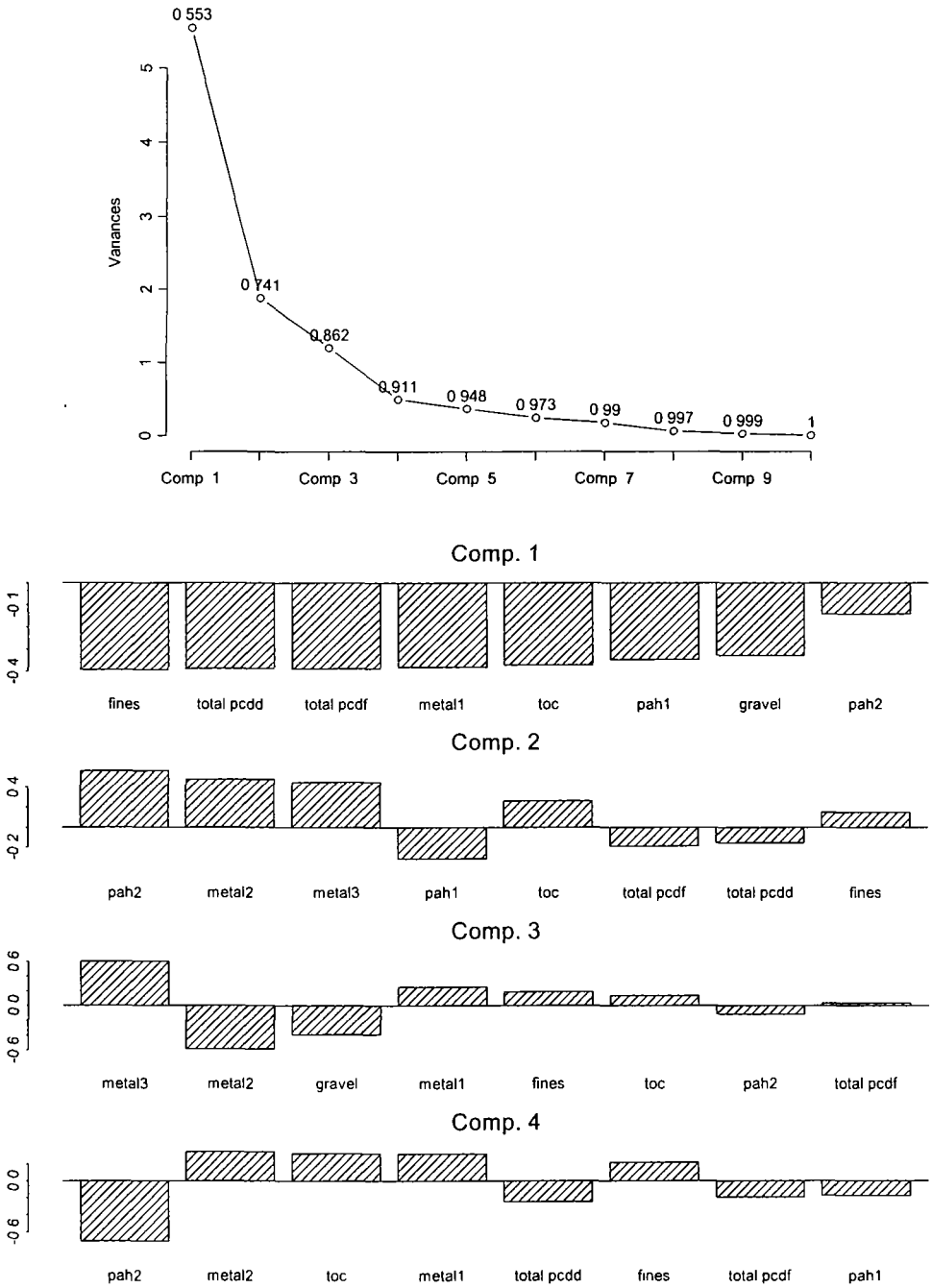

Figure E6A-8a. Screeplot (top) and loadings plot for PCA on all chemicals with ½ DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

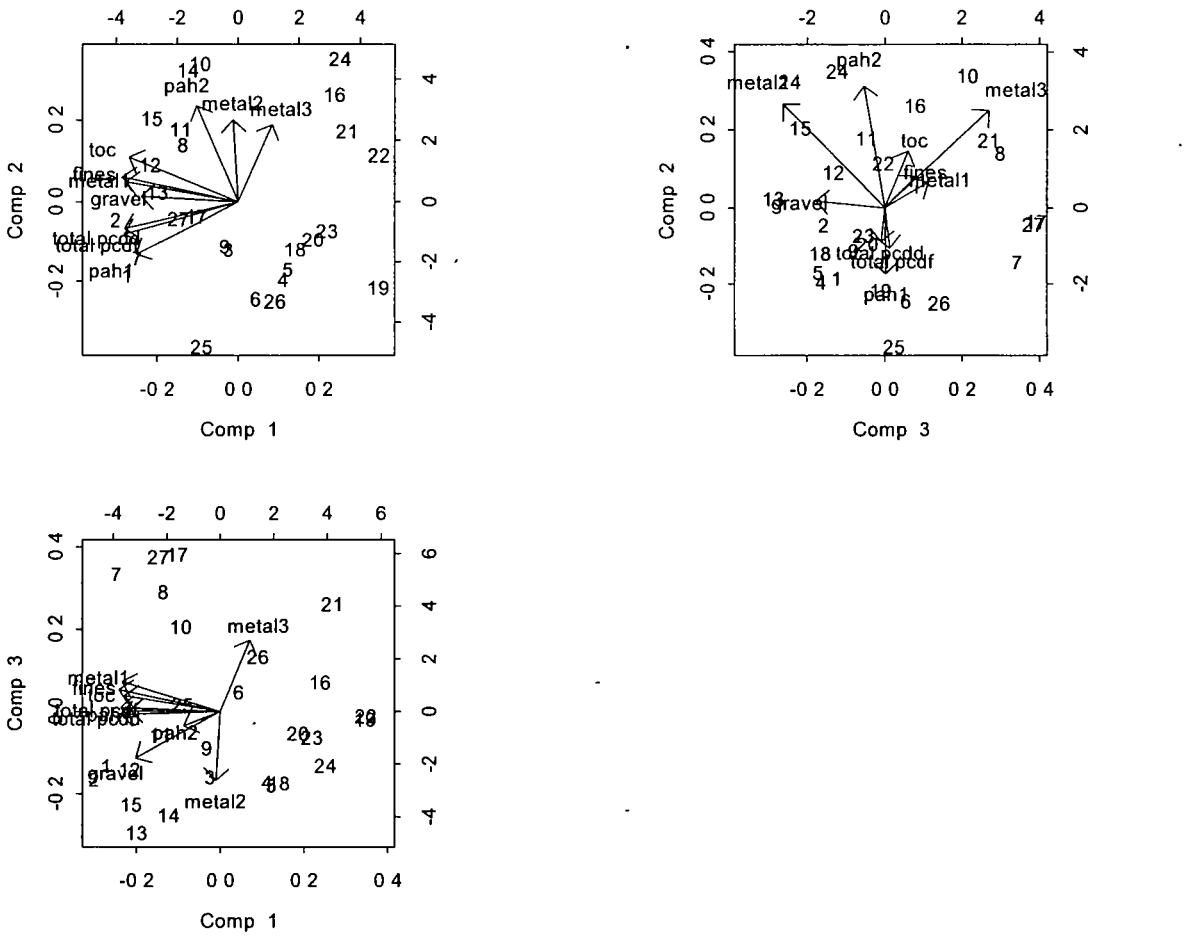

Figure E6A-8b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on all chemicals with ½ DL substitution

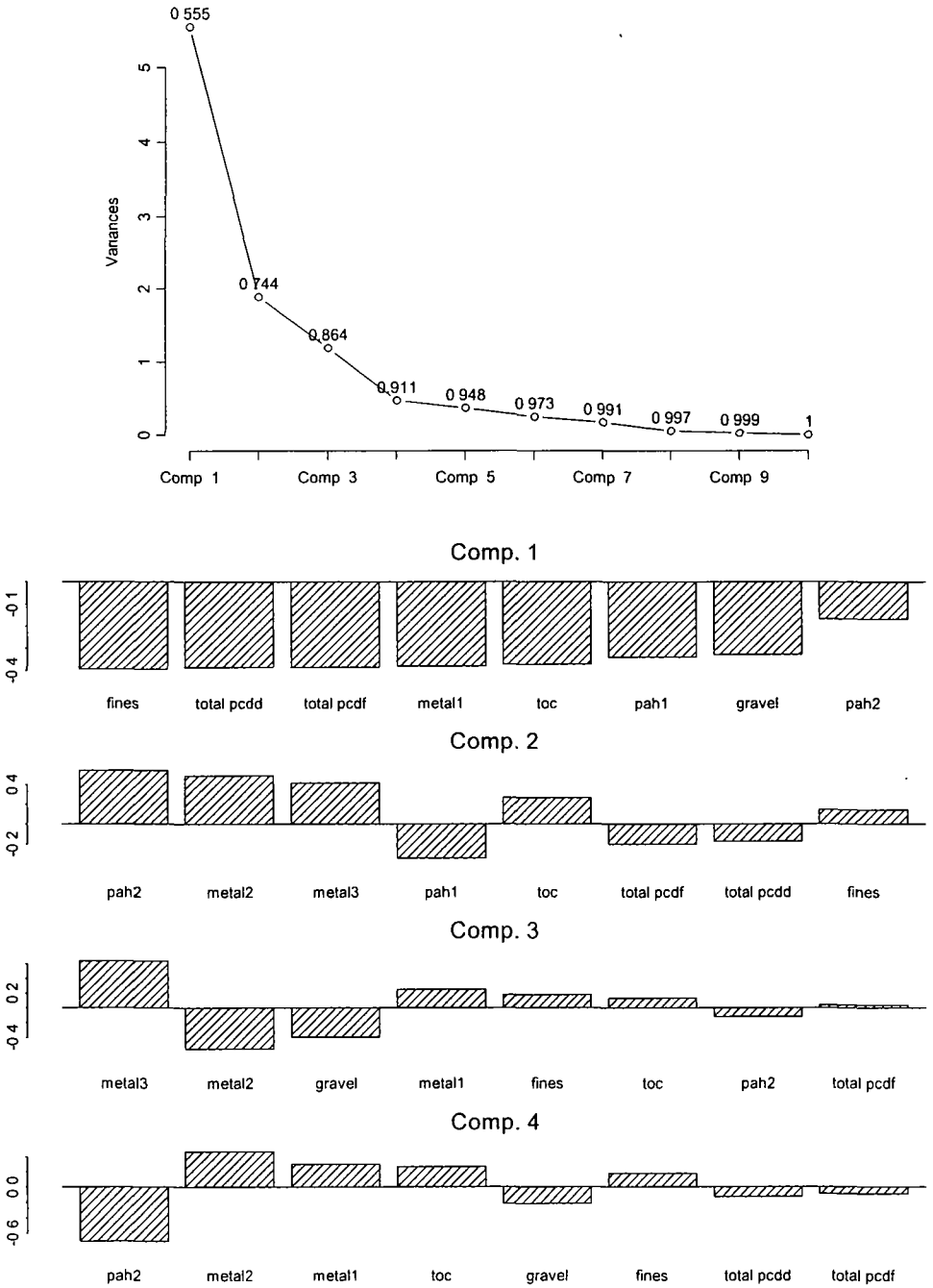

Figure E6A-9a. Screeplot (top) and loadings plot for PCA on all chemicals with half-min DL substitution. The loadings plot includes the top eight variables for each of the first four principal components.

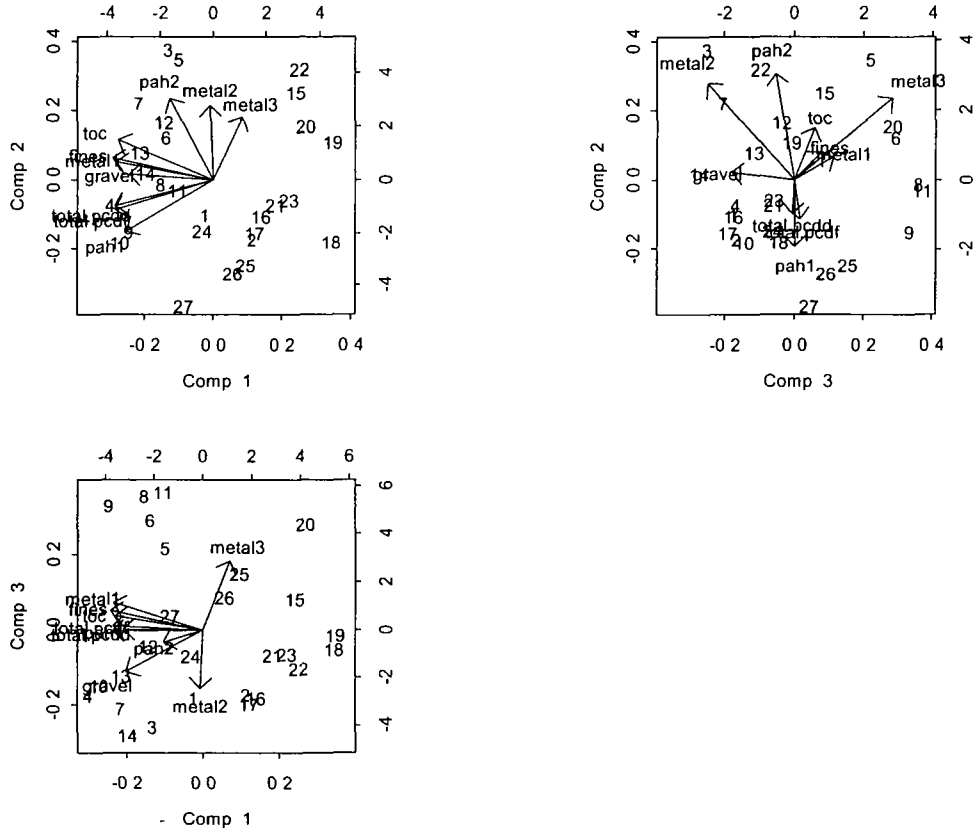

Figure E6A9b. Biplot showing the relationship of the original variables and data to the first two principal components for the PCA on all chemicals with half-min DL substitution

3 PCA FOR EXTRAPOLATING MLR TO LARGER DATA SET

There are two sets of samples: 1) the synoptic chemistry/bioassay samples used to derive the predictive multiple linear regression (MLR) model, and 2) the chemistry only samples for which it may be necessary to predict growth. This analysis investigates the similarity of chemical relationships among these two sets of samples.

3.1 PCA METHODS

The 27 synoptic chemistry/bioassay stations are used in the MLR; there are an additional 46 stations with chemistry only. Only 40 chemistry only stations were considered for this PCA[1] (Table E6A-2). Of the combined dataset with 67 stations, 55 stations were ultimately used; 12 stations were excluded because there were one or more missing values for variables. There were 23 and 25 missing values for thallium and molybdenum, respectively. In addition, 12 of the 40 stations had missing values for one or more of the following chemicals: selenium (4 missing values), TOC (2 missing values), total PCDD and total PCDF (7 missing values each). The PCA analysis included the following variables: TOC, the metals (excluding thallium and molybdenum), HPAH sum, LPAH sum, total.PCDD and total.PCDF. Variables were $\log_{10}$-transformed to reduce skewness and match the scale of the data used in the regression model.

Detection frequency was less than 80 percent for several metals (antimony, arsenic, beryllium, cadmium, cobalt, mercury, selenium, silver). Within this group, the DLs for the chemistry only data were often much higher than for the bioassay stations. The PCA was conducted using ½ DL and full DL for some evaluation of the effect of substitution value.

[1] Stations PB-A, PB-B, PB-C, PB-D, and SDCH-MI-01 had limited sets of analytes. Since all cells in the PCA matrix must be populated, data from these stations could not be used. Station FCD-04 was accidentally left out of the analysis

Table E6A-2. Station IDs and their Numeric Identifiers for Figures E6A-13 and E6A-14

Numeric ID	Data Type	Station ID	Numeric ID	Data Type	Station ID
1	chemonly	CLDH-01	41	bioassay	SDCH-0401
2	chemonly	CLDH-02	42	bioassay	SDCH-0402
3	chemonly	FCCD-01	43	bioassay	SDCH-0403
4	chemonly	FCCD-02	44	bioassay	SDCH-0404
5	chemonly	FCCD-03	45	bioassay	SDCH-0405
6	chemonly	FCCD-04	46	bioassay	SDFC-0401
7	chemonly	FCCD-05	47	bioassay	SDFC-0402
8	chemonly	FCD-01	48	bioassay	SDFC-0403
9	chemonly	FCD-02	49	bioassay	SDFC-0404
10	chemonly	FCD-03	50	bioassay	SDFC-0405
11	chemonly	FCD-05	51	bioassay	SDFC-0406
12	chemonly	FCSW-01	52	bioassay	SDFC-0407
13	chemonly	FCSW-02	53	bioassay	SDFC-0408
14	chemonly	FCSW-03	54	bioassay	SDFC-0409
15	chemonly	GP-08	55	bioassay	SDFC-0410
16	chemonly	GP-10	56	bioassay	SDFC-0411
17	chemonly	HWY-01	57	bioassay	SDFC-0412
18	chemonly	HWY-02	58	bioassay	SDPB-0401
19	chemonly	HWY-03	59	bioassay	SDPB-0402
20	chemonly	PBDH-01	60	bioassay	SDPB-0403
21	chemonly	PBDH-02	61	bioassay	SDPB-0404
22	chemonly	RR-01	62	bioassay	SDPB-0405
23	chemonly	RR-02	63	bioassay	SDPB-0406
24	chemonly	RR-03	64	bioassay	SDPB-0407
25	chemonly	SDFC-730001	65	bioassay	SDREF-0401
26	chemonly	SDFC-730002	66	bioassay	SDREF-0402
27	chemonly	SDFC-730003	67	bioassay	SDREF-0403
28	chemonly	SDFC-730004			
29	chemonly	SDFC-730005			
30	chemonly	SDFC-730006			
31	chemonly	SDFC-730007			
32	chemonly	SDFC-730008			
33	chemonly	SDFC-730009			
34	chemonly	SDFC-730010			
35	chemonly	SDWR-0401			
36	chemonly	SDWR-0402			
37	chemonly	SDWR-0403			
38	chemonly	WL-01			
39	chemonly	WL-02			
40	chemonly	WL-03			

3.2 RESULTS

1st four principal components cumulatively explained 73%, 82%, 87%, and 90%, respectively (Figures E6A10 – E6A14 illustrate the PCA results).

- Comp1 is an overall average, showing small positive contributions of every variable. A cluster of chemistry only stations are found at the extreme end of the Comp1 scale due to slightly higher concentrations of most of the metals and dioxin/furan sums among the chemistry only stations. In general, the chemistry only stations have higher values for Comp1 than the bioassay stations.
- Comp2 separates PAH sums and dioxin/furan sums, from vanadium and TOC. The distributions of this principal component are comparable for the two groups of stations.
- Comp3 separates antimony, selenium, and silver from cobalt, HPAHs, and iron. The chemistry only stations tend to have lower values than the bioassay stations, driven by the fact that many chemistry only stations had higher antimony and silver concentrations.
- Comp4 separates silver, zinc, and mercury from barium, LPAHs, and beryllium. Both station types span the range of this component, but the chemistry only stations tend to have just slightly lower Comp4 values than the bioassay stations due to the higher beryllium, barium and LPAH concentrations.

3.3 CONCLUSION

There is some correlation among the individual chemical variables as evidenced by the similar loading values for all variables onto Comp1 (the 8 variables with the highest loadings are shown in Figure E6A10). When a set of correlated independent variables are included in a regression model, this is referred to as multicollinearity. Multicolinearity affects interpretation of the multilinear regression model, and is discussed in Section 5.XX.

Chemical patterns in bioassay stations are similar to those among chemistry only stations (Figure E6A11). There are several chemistry only stations that have slightly higher concentrations of most variables. As a result, using the MLR derived from bioassay stations to predict effects in this group will require extrapolating somewhat beyond the range of the data used to fit the model.

Non-detects were included at half DL for this analysis. An identical PCA was conducted with non-detects set to the full DL. The patterns were identical, but chemistry only stations were a bit more separated from the bioassay stations. The substitution value has an effect due to the low detection frequency for some of the variables. As a result, the detection frequency of the predictor variables resulting from the model fitting process should be taken into account when

making predictions of toxicity (C tentans growth as AFDW) for chemistry only stations. If predictions are made using chemicals with a high rate of non-detects in the chemistry only dataset, the predictions should be bounded using different substitution methods.

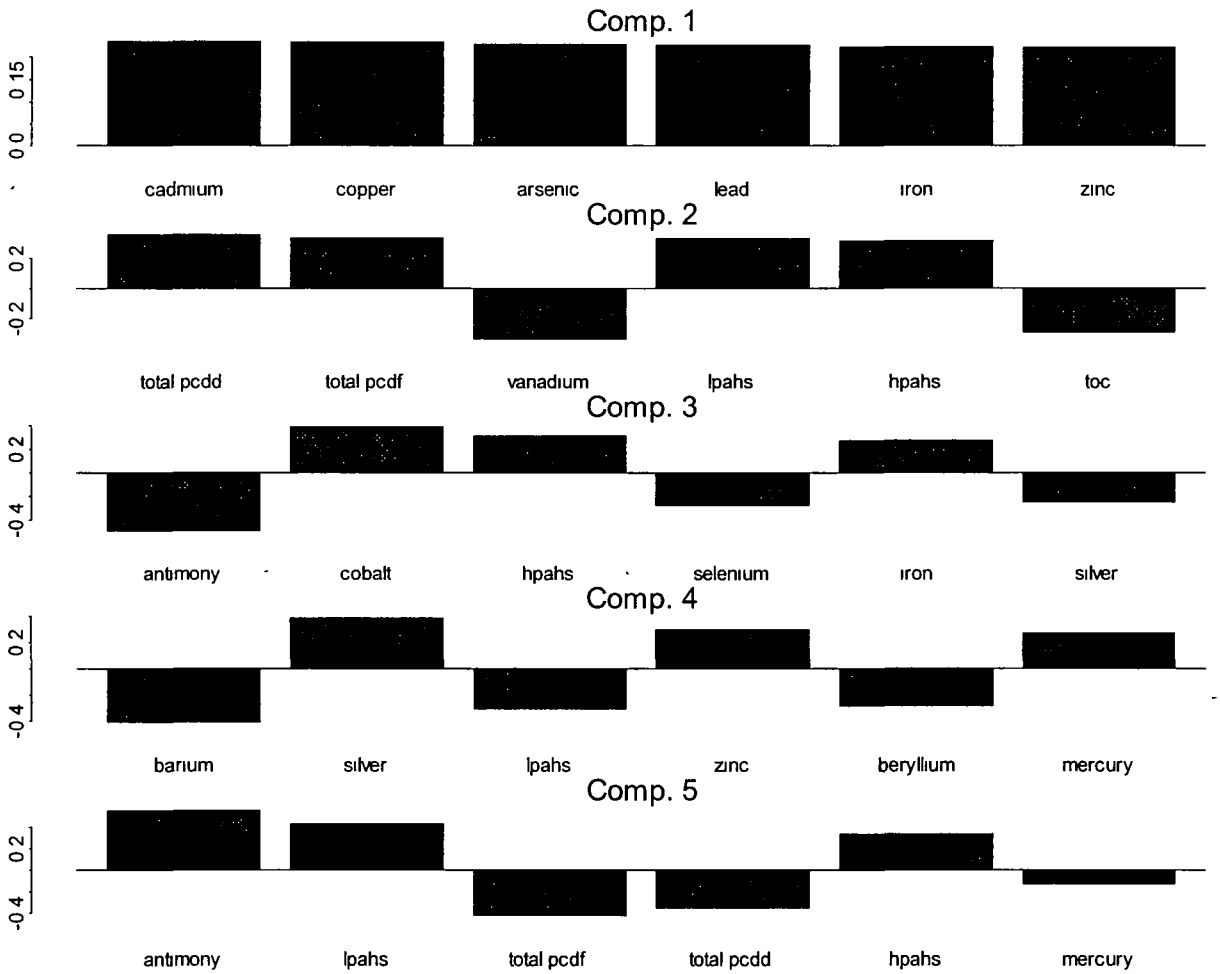

Figure E6A-10 Loadings plot for PCA

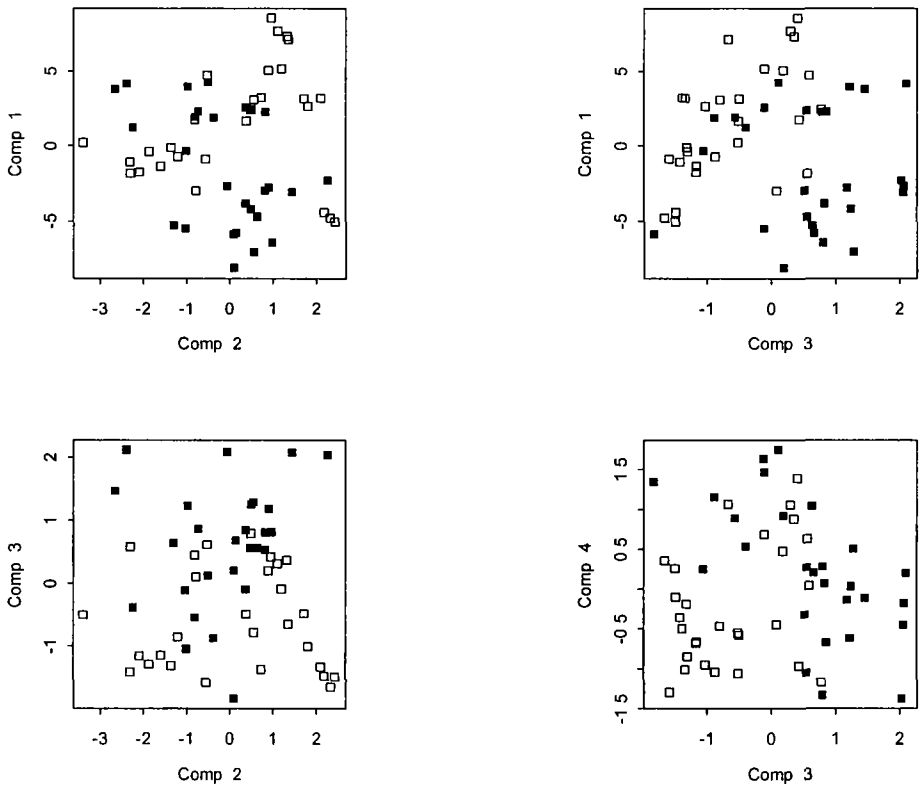

Figure E6A-11. Scatterplots among first four principal components. Open squares are chemistry only stations (n=28); filled squares are the synoptic chemistry/bioassay stations (n=27)

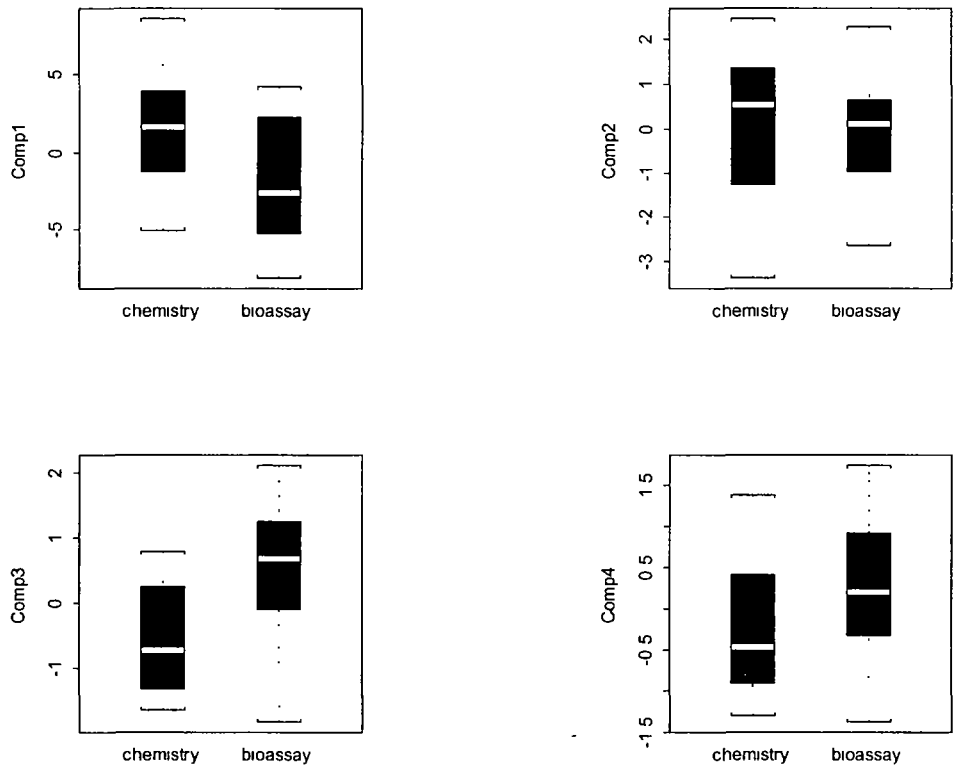

Figure E6A-12. Boxplots showing the distribution of values for the principal components 1-4, comparing chemistry only and synoptic chemistry/bioassay stations.

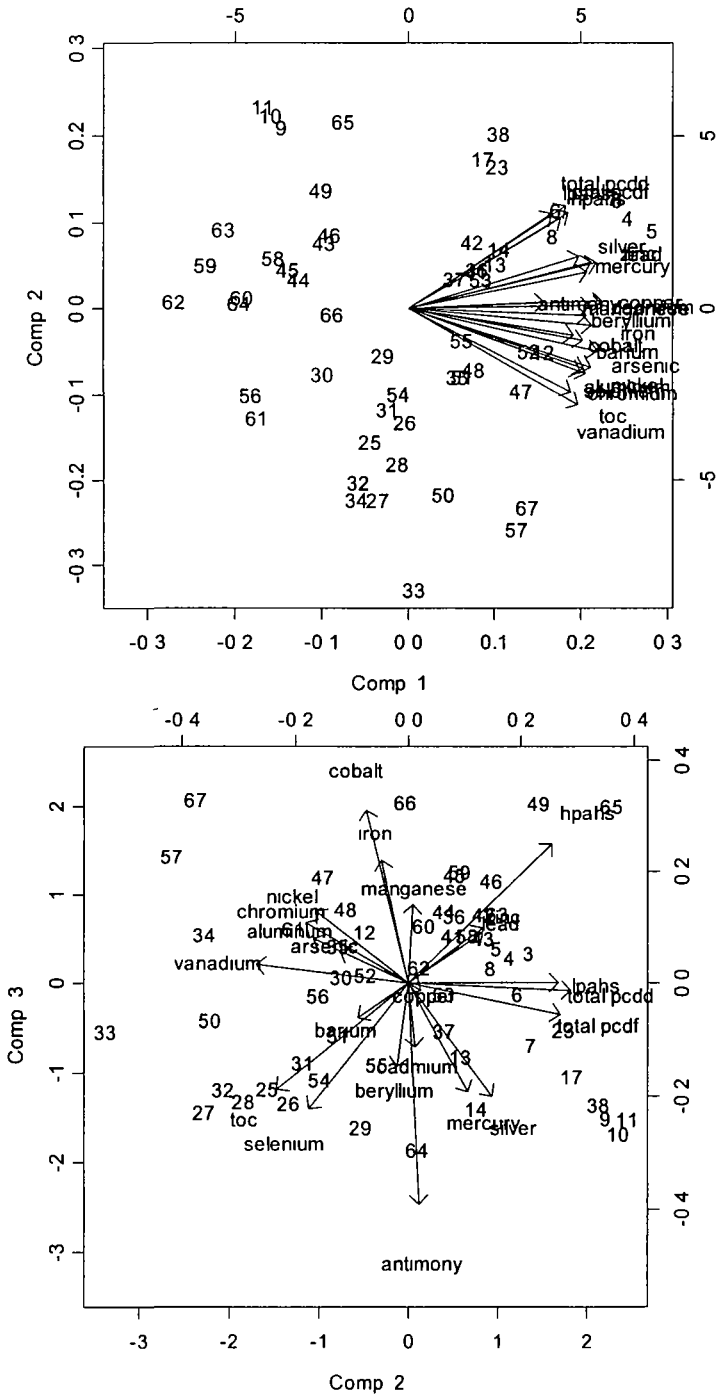

Figure E6A-13. Biplots for PCA for components 1 and 2 (left) and components 2 and 3 (right). Stations are shown by unique number; numbers < 40 are the chemistry only stations, and numbers > 40 are the synoptic chemistry/bioassay stations The location of data points in these plots correspond to the locations shown by symbol in Figure 2

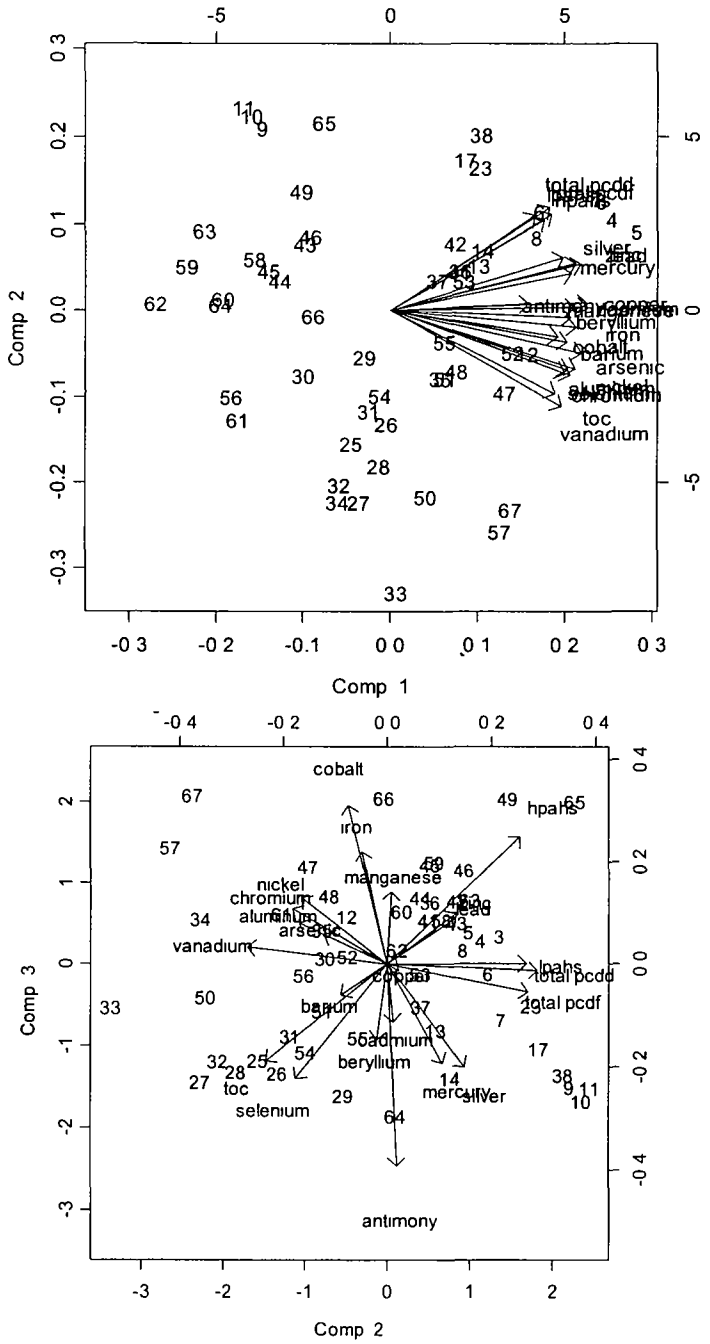

Figure E6A-13 Biplots for PCA for components 1 and 2 (left) and components 2 and 3 (right). Stations are shown by unique number; numbers < 40 are the chemistry only stations, and numbers > 40 are the synoptic chemistry/bioassay stations. The location of data points in these plots correspond to the locations shown by symbol in Figure 2

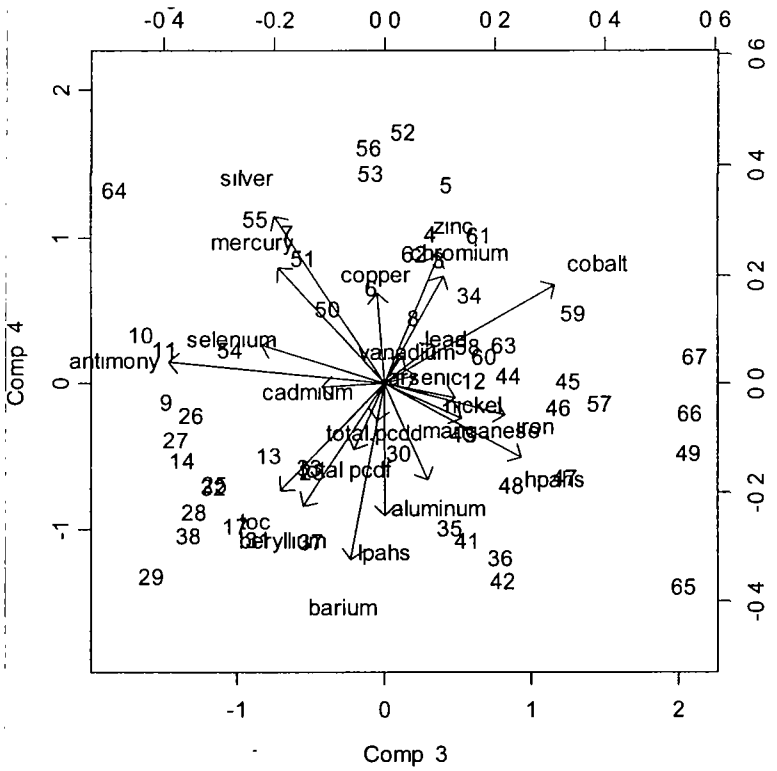

Figure E6A-14. Biplot for PCA for components 3 and 4. Stations are shown by unique number; numbers < 40 are the chemistry only stations, and numbers > 40 are the synoptic chemistry/bioassay stations. The location of data points in these plots correspond to the locations shown by symbol in Figure 2.