October 2013

SHELL CANADA ENERGY

Appendix 3.6: Chronic Effects Benchmarks

REPORT

Project Number: 13-1346-0001

Golder Associates

Table of Contents

TABLES

FIGURES

1.0 INTRODUCTION

This appendix describes the assessment methods and results of Chronic Effects Benchmark (CEB) derivations applied to the aquatic health assessment of the Environmental Impact Assessment (EIA) for the Pierre River Mine (PRM) Project. This appendix further supports the Aquatic Resources Assessment presented in Appendix 1 and 2 of this submission.

This appendix provides CEBs for all compounds considered in the aquatics assessment with the exception of those which have status as a nutrient, were evaluated using an alternate constituent as a surrogate parameter, or had very low concentrations predicted by modelling. This document builds upon previous iterations of CEB development, which began with CEBs which were developed for numerous constituents as part of the EIA, including:

- aluminum, antimony, arsenic, barium, beryllium, boron, cadmium, chromium (III) cobalt, copper, iron, molybdenum, silver, strontium, vanadium, and polycyclic aromatic hydrocarbon groups 1, 2, 3, 5, 6 and 7.

Revisions to the original CEB derivation were prepared in response to technical information requested of Shell Canada Energy (Shell) during discussions with Environment Canada in 2011, culminating in refinements and additions to CEBs and submission in May 2012. The expanded list of CEBs in May 2012 included the following, in addition to those already included from the EIA:

- lead, manganese, mercury, nickel, zinc, PAH group 4, 8 and 9, ammonia, naphthenic acids (labile, refractory, and total), sulphide, sulphate, total dissolved solids, total phenolics.

Subsequent to the EIA and May 2012 updates, further refinements to the CEB derivations have been incorporated for eight constituents, reflecting recent advances in scientific information regarding these constituents. This appendix incorporates new information that has been obtained for the following constituents:

- aluminum, arsenic, cadmium, iron, strontium, sulphate, total dissolved solids, and total phenolics.

As CEBs for these eight constituents incorporate recent updates to derivations that are presented in previous submissions, the level of documentation (details of derivation) is greater for these constituents. The remaining constituents have been retained as submitted in May 2012, and have been included herein (unaltered) for the sake of completeness.

2.0 CHRONIC EFFECTS BENCHMARKS

Aquatic health assessments for oil sands developments have traditionally applied a combination of generic federal guidelines from the Canadian Council of Ministers of the Environment (CCME 1999a; updates to 2011) and derivations of site-specific criteria using species sensitivity distributions (CCME 2003a; Posthuma et al. 2002). The EIA included derivations of CEBs, as described in Volume 4B, Appendix 4-2, Section 3. However, while these CEBs were being derived, CCME (2007a) developed a refined stepwise procedure for site-specific derivations that is now preferred by regulators and that provides a consistent framework for future evaluations. This appendix presents updated CEBs that follow the approach described by CCME (2007a). The CEB derivations also include additional data points; therefore, the CEBs described herein supersede those presented in the EIA.

2.1 Updated Canadian Council of Ministers of the Environment Protocol

In the CCME (2007a) protocol, two approaches for deriving water quality guidelines are provided. These approaches depend on the quality and amount of data available for each constituent:

- **Statistical Extrapolation Method** – This approach uses data from multiple species to derive the final guideline and uses the Species Sensitivity Distribution (SSD). This approach establishes an acceptable response (effect) size, fits suitable endpoint data to a specified model, and calculates an exposure concentration that protects a specified percentage of species (e.g., 95%).
- **Lowest Threshold Method** – If the data are insufficient to model an SSD curve, then a second- or third-tier guideline may be developed considering the lowest toxicity value from a high-quality study (and applying an uncertainty or safety factor). This approach is based on the original federal guideline development protocol (CCME 1991).

The CCME (2007a) protocol provides guidance on derivation of different guideline types, considering the volume and quality of environmental and toxicological data available. Type A guidelines, which incorporate the SSD approach, are preferred because they consider the range of thresholds derived from valid studies, such that guidelines are not unduly influenced by a single anomalous result. Where inadequate or insufficient toxicity data exist for deriving a Type A guideline, Type B1 or Type B2 guidelines, which consider the lowest relevant individual toxicological endpoint available, can be derived. At present, there is no protocol for deriving guidelines when the minimum toxicity data requirement for a Type B guideline is not met.

2.2 Application

This appendix provides the technical rationale for the development of region-specific CEBs for oil sands developments, with an emphasis on the PRM. The overarching goal is to provide a system for aquatic effects benchmark development that includes the following:

- is consistent with updated Canadian (i.e., CCME 2007a) federal guidance for development of aquatic effect thresholds;
- is customized to general water quality constituents that are reflective of the Athabasca Oil Sands Region;
- makes appropriate use of available and relevant toxicity information;
- coordinates derivations across multiple projects for consistency and efficiency; and
- applies technically defensible assumptions in the derivation procedures.

The CEBs developed in this appendix have general applicability to the Athabasca Oil Sands Region. However, some of the CEBs are refined through customization to watercourse-specific water concentrations. The PRM receiving environment encompasses watersheds in the PRM LSA, and includes the following environmental characteristics that were considered for site-specific customization of CEBs:

- PRM LSA medians: pH = 7.5, temperature = 5°C, hardness = 143 mg/L $CaCO_3$, DOC = 21.8 mg/L.

2.3 Screening of Constituents for Chronic Effects Benchmark Development

The CEB evaluation began by considering the full list of water quality assessment constituents (Table 2.3-1). Predicted concentrations of these constituents were compared to generic federal Water Quality Guidelines (WQGs; CCME 2007b), if available.

Water quality guidelines represent levels that, if met in any surface water, will provide a high level of protection to aquatic life. In this assessment, the *Canadian Water Quality Guidelines for the Protection of Aquatic Life* were used; these conservative guidelines are intended to "protect all forms of aquatic life and all aspects of the aquatic life cycles, including the most sensitive life stage of the most sensitive species over the long term" (CCME 1999a). In other words, exceedance of a water quality guideline indicates that adverse effects may be possible, but not necessarily likely.

Table 2.3-1 Water Quality Assessment Constituents Considered for the Assessment

Assessment Constituents[a][b]		
Metals	**PAH Components**	**Other Components**
Aluminum	Polycyclic Aromatic Hydrocarbons (PAH) group 1	Ammonia
Antimony	PAH group 2	Naphthenic acids (labile and refractory)
Arsenic	PAH group 3	Sulphate
Barium	PAH group 4	Sulphide
Beryllium	PAH group 5	Tainting potential
Boron	PAH group 6	Temperature
Cadmium	PAH group 7	Total dissolved solids
Calcium[c]	PAH group 8	Total nitrogen[c]
Chloride[c]	PAH group 9	Total phenolics
Chromium		Total phosphorus[c]
Cobalt		Toxicity - acute
Copper		Toxicity - chronic
Dissolved Organic Carbon[c]		
Iron		
Lead		
Magnesium[c]		
Manganese		
Mercury		
Molybdenum		
Monomer[d]		
Nickel Selenium[c]		
Potassium[c]		
Silver		
Sodium		
Strontium		
Vanadium		
Zinc		

(a) All listed metals are considered to be total metals.

(b) For a discussion of individual Polycyclic Aromatic Hydrocarbons (PAHs) and linkage to PAH group assignments, see Table 2.5-1.

(c) Constituent modelled in the EIA but no chronic effect benchmark developed, due to status as a nutrient or evaluation using an alternate constituent.

(d) Constituent modelled in the EIA but no chronic effect benchmark developed, due to low concentrations predicted by modelling.

Note: Table is the same as EIA, Volume 4A, Section 6.5, Table 6.5-1.

To further focus the development of CEBs, a screening procedure was applied to identify the following categories of constituents:

- CEBs Available – CEBs already developed from other oil sands developments using the CCME (2007a) procedure, and considered to be relevant to the assessment.
- CEBs Unavailable – CEBs not previously derived for other oil sands developments, or that required refinement to be relevant to the assessment.
- CEBs Not Required – Constituents that were consistently below screening-level water quality guidelines.

A constituent in either of the first two categories was carried forward unless it was excluded from further consideration for one of the following reasons:

- the constituent in question has been shown to have limited potential to affect aquatic health (i.e., innocuous constituents);
- the constituent in question is more appropriately assessed through a tissue-based benchmark than a water-based benchmark; or
- the constituent in question is a component of another constituent, which is a more suitable focus point for the analysis.

Accordingly, the following constituents from Table 2.3-1 were excluded during the first step of the screening process:

- selenium;
- monomer, due to low concentrations predicted by modelling;
- individual nutrients and major ions (e.g., calcium, chloride, magnesium, potassium, sulphate, total nitrogen, total phosphorus);
- non-chemical constituents (i.e., fish tainting potential, toxicity – acute and chronic); and
- dissolved constituents.

These constituents, and the specific rationales for their exclusion from CEB development, are discussed below.

Selenium

Consistent with the current state of the science of selenium toxicology, and recognizing that selenium elicits effects on reproduction due to maternal transfer (Chapman et al. 2010), a water-based CEB was not developed for selenium. Rather, the potential for effects to aquatic health due to predicted selenium concentrations were assessed through predicted tissue concentrations in the indirect exposure – changes to fish tissue quality assessment.

Monomer

The modelling predicted that instream concentrations of monomer would be essentially zero at all assessment nodes and during all snapshots (EIA, Volume 4B, Appendix 4-7). Therefore, a benchmark was not derived for this constituent.

Individual Nutrients and Major Ions

Phosphorus and nitrogen compounds are nutrients that can exert adverse effects at high concentrations via eutrophication. These nutrients were not evaluated as individual constituents requiring CEB development because potential effects related to eutrophication are assessed separately in the EIA, particularly in terms of the effect on Dissolved Oxygen (DO) demand. The water from muskeg drainage and overburden dewatering has the potential to contain high levels of oxygen-consuming organic constituents and low DO levels.

The effect of PRM on DO levels was discussed in the EIA, Volume 4A, Section 6.5, and an eutrophication assessment was provided in the EIA, Volume 4A, Section 6.6. Notably, because watercourses in the PRM LSA are relatively nutrient-rich, they are less sensitive to nutrient inputs relative to oligotrophic streams. As such, negligible eutrophication effects on aquatic biota are expected in these watercourses under EIA Application Case conditions. Nevertheless, nitrate and ammonia were screened for toxicity effects using WQGs.

Furthermore, calcium, chloride, magnesium, potassium and sulphate were excluded from CEB development because these individual ions are components of Total Dissolved Solids (TDS), another modelled constituent included in the assessment. For TDS, the mixture of constituents was evaluated using a conservative CEB, which is protective against effects of the individual constituents in the mixture.

Predicted changes in sulphate concentrations are expected to have a negligible effect on aquatic biota given the moderately hard waters in these watercourses (which ameliorate sulphate toxicity), and the low predicted concentrations relative to recently derived effects-based water quality limits proposed by the Alberta Government (AENV 2008).

Non-chemical Constituents

Taint in fish is defined as an abnormal odour or flavour detected in the edible tissue (LeBlanc et al. 2000). Tainting thresholds were converted to Tainting Potential Units (TPU), which were then compared to standardized benchmarks for the evaluation of fish tainting. Similarly, the assessment constituents related to toxic effects (acute and chronic toxicity) in operational and reclamation water were converted to toxic units (toxic unit - acute [TU_a] and toxic unit - chronic [TU_c]), as described in EIA, Volume 4A, Section 6.5.2.7. The TU and TPU assessment methods do not require development of site-specific CEBs.

Dissolved Constituents

No CEBs were developed for the dissolved form of metals, metalloids or non-metals because they are a component of the corresponding total metal concentrations. Total metal measurements provide a more conservative basis for assessment than dissolved metals. Some site-specific considerations (i.e., toxicity-modifying factors) implicitly account for the proportion of constituent that is expected to be dissolved, but derivation of CEBs for the dissolved phase was not conducted.

2.4 Assessment Methods

2.4.1 General Approach

The general procedure for CEB development followed a three-step process which included creating a toxicological database for each constituent, analyzing the available data, and deriving an HC_5 value. An HC_5 value denotes a concentration that is hazardous to no more than 5% of species in the community. Where insufficient data were available, a value was conservatively derived using the lowest reported chronic toxicity test results (Figure 2.4-1). Chronic effect benchmarks were required for any constituent that was carried through the initial screening procedure described in Section 2.3.

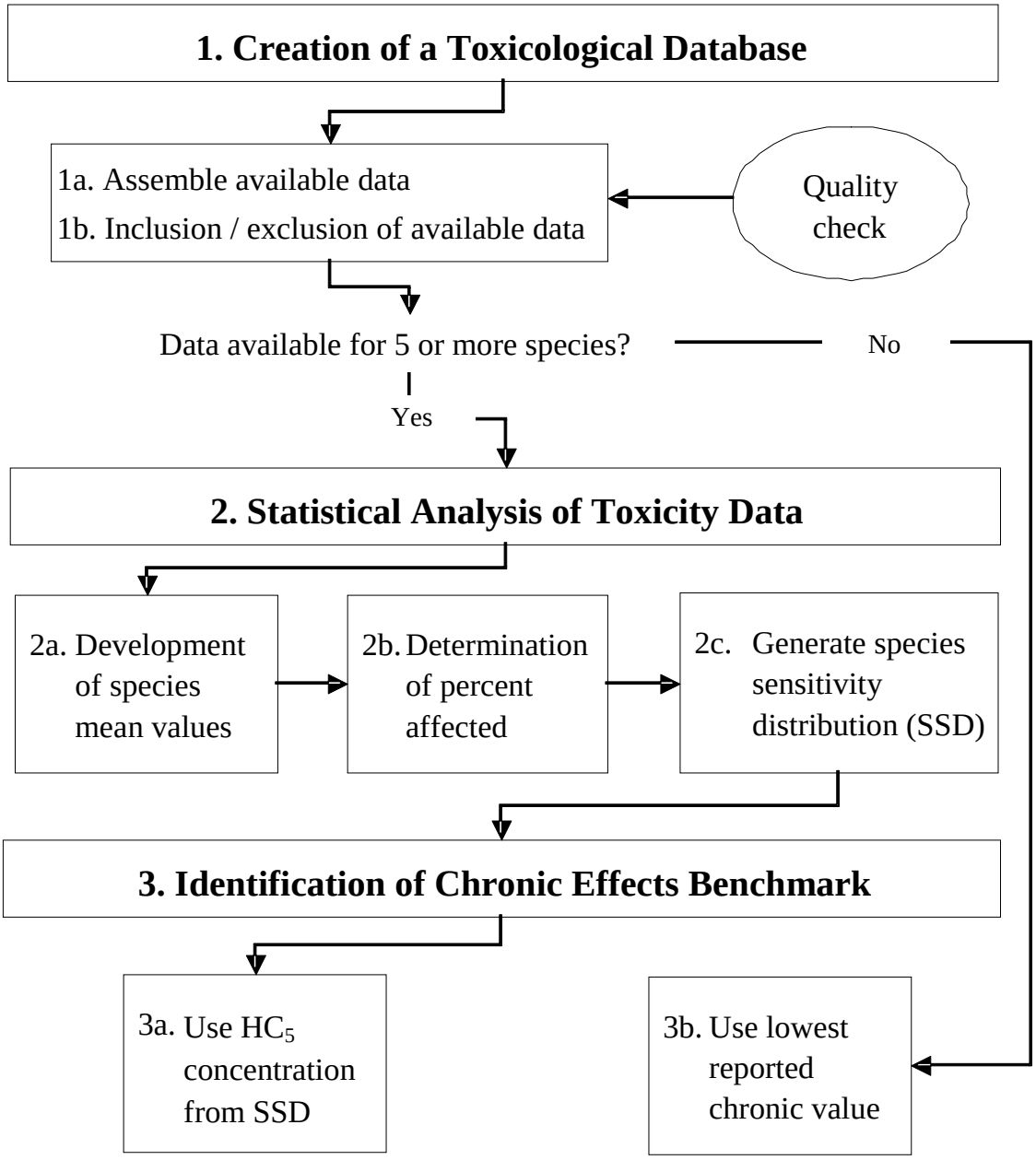
The CEBs represent constituent concentrations above which changes to aquatic health could occur on the scale of individual organisms. The benchmarks are less conservative (i.e., more realistic) than generic WQGs, but retain a level of conservatism for the evaluation of population-level effects, which would require concentrations to be higher than the CEBs. Consequently, the CEBs are considered to be conservative thresholds by which potential effects to aquatic health can be assessed.

Consistent with CCME (2007a), the SSD approach was selected as a preferred method to derive a CEB in acknowledgement that there are biological differences among species and that the variation among species sensitivities can be described by a statistical distribution. The distribution can then be used to define an environmental quality criterion, expressed as a concentration that is expected to be safe for the majority of species (Posthuma et al. 2002). The most commonly used criterion is referred to as the HC_5 value. A comparison of chronic, single species and experimental ecosystem data for metals, pesticides, surfactants and other organic and inorganic compounds has shown that the HC_5 is a conservative threshold for effects to aquatic ecosystems (Versteeg et al. 1999).

An SSD-type approach has been used to derive most of the current United States Environmental Protection Agency (U.S. EPA) water quality criteria for the protection of aquatic life. It has also been used by several European nations for deriving environmental quality criteria, and has been recommended as a standard ecological risk assessment technique by Suter and Barnthouse (1993), the Aquatic Risk Assessment and Mitigation Dialog Group (Baker et al. 1994), and the Water Environmental Research Foundation (Parkhurst et al. 1994).

The CCME has used an SSD approach to develop the Canadian water quality guidelines for ammonia and boron for the protection of aquatic life (CCME 2009, 2010). The CCME (2007a) recommends using this approach to develop other Canadian water quality guidelines for the protection of aquatic life. Although the approach has not yet been applied by CCME to the majority of the individual constituents for which WQGs exist for the protection of aquatic life, the basic concepts of the approach are transferable to site and region specific guideline derivations. Therefore, the basic principles of CCME (2007a) and many of the specific procedural rules were applied in deriving the CEBs.

Figure 2.4-1 Procedure Used to Identify Chronic Effects Benchmarks



Applying the SSD approach provides several advantages in CEB development, because it:

- enables more recent studies to be included in the toxicity database;
- enables exclusion of non-resident species with poor ecological relevance to the region; and
- facilitates the consideration of site-specific modifying factors in the screening of relevant toxicity studies.

These considerations improve the relevance of the CEB to the region.

One disadvantage of the region-specific customization of CEBs is related to the reduction of sample size that occurs when studies are excluded based on regional relevance. Whereas the freshwater SSDs used in the development of generic WQGs can incorporate toxicity test results for numerous species from many ecosystems, the region-specific CEBs filter the toxicity data for relevance. In most cases, this filtering reduces the number of species and endpoints available, and sometimes results in insufficient data for development of an SSD. To compensate for this problem, several mitigating assumptions were applied:

- The screening of organisms for regional relevance to the SSD was not highly restrictive (i.e., all freshwater organisms found in Canada were retained, with tropical and subtropical species excluded).
- Some subchronic test endpoints that did not meet the test duration constraints of the CCME (2007a) derivation protocol were retained to maintain suitable sample sizes for SSD development.
- Where toxicity datasets were screened for relevance to water quality constituents in regional waterbodies, a liberal acceptance range was applied to avoid premature exclusion of data. For example, water hardness in the range of 50 to 450 mg/L $CaCO_3$ was considered relevant to the Oil Sands Region, with only the extremes of soft and hard water excluded.

The above approach provided CEB derivations that are applicable to numerous waterbodies in the Oil Sands Region. Where the above assumptions had the potential to affect the conservatism and uncertainty of the derived CEBs, the derivation procedure qualitatively considered additional factors and context, such as:

- the direction of suspected bias (e.g., inclusion of aluminum toxicity data at pH 6.5 would tend to reduce CEBs relative to neutral pH conditions);
- the relation of the CEB to the most sensitive and relevant chronic toxicity endpoint;
- other toxicity modifying factors (e.g., high DOC) that were not explicitly included in the derivation, but that may influence site-specific bioavailability;
- the effect size of test endpoints (e.g., EC_{25} or LC_{10}) used to derive the SSD, particularly for those endpoints close to the fifth percentile of the distribution; and
- supporting lines of evidence from other toxicological models and literature not directly incorporated in the SSD.

2.5 Procedure

The following subsections outline the technical procedures for the three-step derivation process shown in Figure 2.4-1.

2.5.1 Step 1: Creation of a Toxicological Database

2.5.1.1 Step 1a: Assemble Available Data

Metals and Metalloids

Available chronic toxicological data for each constituent were summarized, with a focus on data for algae, invertebrates and fish (following the recommendations of CCME 2007a). The development of each toxicity database began with an examination of primary chronic toxicity data from fact sheets used to derive relevant *Canadian Water Quality Guidelines* (CCME 1999a, 2007b). The toxicity database was then expanded by querying the AQUIRE and ECOTOX databases administered by the U.S. EPA (2007a), and by searching for other available peer-reviewed scientific literature from journal databases (e.g., Cambridge Scientific Abstracts, PubMed). This review was focused on the period after the previous guideline derivations.

The resulting database contained data with various test endpoints, such as mortality, reduced survival, growth, or reproduction, derived from subchronic and chronic studies. The toxicity database contains primary and secondary data that meet the requirements of U.S. EPA and CCME guideline development protocols (CCME 2007a; Stephan et al. 1985).

All life stages were included in the toxicity database; however, for aquatic invertebrates and amphibians with terrestrial adult stages (e.g., non-biting midge), only the aquatic phases were included in the recent SSD analyses. Although the database does not include all available data, it contains primary data that meet the requirements of U.S. EPA and CCME guideline development protocols (CCME 2007a; Stephan et al. 1985).

Polycyclic Aromatic Hydrocarbons

The procedure for developing a toxicity database for Polycyclic Aromatic Hydrocarbons (PAHs) began with the selection of indicator PAH compounds spanning a range of molecular structures, including:

- anthracene;
- fluoranthene;
- fluorene;
- naphthalene;
- phenanthrene; and
- pyrene.

The PRM CEBs assessment included compound-specific derivations for these six indicator PAHs, with the objective of comparing the SSD-based CEBs to the threshold values for chronic toxicity summarized in McGrath and DiToro (2009). The latter work entailed an assessment of the Target Lipid Model (TLM) for toxicity assessment of Type I narcotic chemicals. The numeric water quality guidelines in McGrath and DiToro (2009) are equivalent to a HC_5 (i.e., hazard concentration to 5% of the tested species, or the concentration that protects 95% of the tested species) and therefore compatible with the level of protection specified in CCME (2007a). The

TLM derivation procedure differs from the SSD approach; however, the former applies toxic units as a metric for expressing the toxicity of multiple PAHs present in a mixture, and applies an acute to chronic ratio for estimation of chronic sublethal effects.

The TLM has been validated and demonstrated to correctly predict (within the uncertainty bounds) the onset of sublethal effects of edemas, haemorrhaging, and other abnormalities in early life-stage exposure of organisms to PAHs. The authors conclude that computed HC_5 values were lower than No Observed Effect Concentrations (NOECs) based on growth, reproduction, and mortality endpoints and sublethal effects. The TLM procedure, therefore, provides an independent validation of the SSD applied to individual PAH toxicity distributions. Once the comparability of the two derivation methods was assessed (and confirmed to be acceptable), remaining PAH groups were assigned CEBs extrapolated from the TLM (Table 2.5-1). This validation step provided an added level of confidence in the CEBs for PAHs, and helped to compensate for limitations in the datasets for specific PAH compounds.

The development of the toxicological database for the individual PAHs began with the examination of the toxicity data for PAHs summarized in McGrath and DiToro (2009). Review of the reported toxicological literature in that study included 34 studies that examined both chronic and acute toxicity of PAHs. Additional studies were also obtained by querying the ECOTOX database (U.S. EPA 2007a) and used to bolster the data set. Test endpoints included mortality, growth and/or reproduction for several species of algae, invertebrate, amphibians and fish.

Table 2.5-1 Compounds and Indicators Included in Polycyclic Aromatic Hydrocarbon Groups

Group	Constituent	Indicator Constituent for CEB
PAH Group 1	dibenzo(a,h)anthracene; benzo(a)pyrene; C_1 substituted benzo(b&k) fluoranthene/ **benzo(a)pyrene**; C_2 substituted benzo(b&k) fluoranthene/benzo(a)pyrene	**benzo(a)pyrene** - HC_5 from McGrath and DiToro (2009)
PAH Group 2	benzo(a)anthracene; benzo(b)fluoranthene; benzo(a)anthracene/chrysene; C_1 substituted benzo(a)anthracene/chrysene; C_2 substituted benzo(a)anthracene/chrysene; benzo(b&j)fluoranthene; benzo(b&k)fluoranthene; Indeno(c,d-123)pyrene	**7,12-dimethylbenzo(a)anthracene** - HC_5 from McGrath and DiToro (2009)
PAH Group 3	benzo(g,h,i)perylene; **chrysene**; carbazole; C_1 substituted carbazole; C_2 substituted carbazole; benzo(j)fluoranthene; benzo(k)fluoranthene	**chrysene** - HC_5 from McGrath and DiToro (2009)
PAH Group 4	**acenaphthene**; C_1 substituted acenaphthene; acenaphthylene	**acenaphthene** - HC_5 from McGrath and DiToro (2009)
PAH Group 5	**anthracene**; **phenanthrene**; C_1 substituted phenanthrene/anthracene; C_2 substituted phenanthrene/anthracene; C_3 substituted phenanthrene/anthracene; C_4 substituted phenanthrene/anthracene; 1-methyl-7-isopropyl-phenanthrene (retene)	**anthracene** - HC_5 from SSD (this study)
PAH Group 6	**biphenyl**; C_1 substituted biphenyl; C_2 substituted biphenyl; C_3 substituted biphenyl	**biphenyl** - HC_5 from McGrath and DiToro (2009)
PAH Group 7	**fluoranthene**; **fluorene**; C_1 substituted fluorene; C_2 substituted fluorene; C_3 substituted fluorene; dibenzothiophene; C_1 substituted dibenzothiophene; C_2 substituted dibenzothiophene; C_3 substituted dibenzothiophene; C_4 substituted dibenzothiophene	**fluoranthene** - HC_5 from SSD (this study)
PAH Group 8	**naphthalene**; C_1 substituted naphthalenes; C_2 substituted naphthalenes; C_3 substituted naphthalenes; C_4 substituted naphthalenes	**naphthalene** - HC_5 from SSD (this study)
PAH Group 9	C_1 substituted fluoranthene/pyrene; C_2 substituted fluoranthene/pyrene; C_3 substituted fluoranthene/pyrene; **pyrene**	**pyrene** - HC_5 from SSD (this study)

Notes: Table is the same as the EIA, Volume 4A, Section 6.5, Table 6.5-2, with updates to align with recent projects in the region.

Bold text indicates indicator PAHs used to represent the specified group.

Bold underlined text indicates indicator PAHs for which a compound-specific SSD was developed. Where multiple individual CEBs were derived for a PAH group, the most conservative (lowest) HC_5 for any individual compound in the group was applied as the indicator constituent.

2.5.1.2 Step 1b: Inclusion/Exclusion of Available Data

Available studies were screened for inclusion or exclusion based on CCME (2007a) guidance for developing site-specific water quality objectives. Test acceptability was confirmed by consulting the original publication or report, reviewing the test assessment methods and verifying the test results. The applicable rules were as follows:

- Toxicity data on species that are known to occur in Canadian waters or may occur at the site were included, whereas toxicity data on species that are not known to occur in Canada were excluded.
- Endpoints included data for growth, mortality and reproduction. Non-traditional endpoints with uncertain biological relevance (e.g., biochemical endpoints, swimming speed) were excluded.
- Tests using field-collected organisms or performed in the field were excluded due to uncontrolled sources of variability (e.g., life history of organisms unknown, exposure conditions not controlled, chemical mixtures present).
- Only studies with freshwater exposure to the constituent in question were included. Experiments in which test organisms exposure occurred through injections or diet were excluded.
- Studies were also excluded if exposure concentrations (e.g., nominal concentrations, extrapolated concentrations) or test duration were not reported.
- Studies evaluating synergistic, or antagonistic effects of chemicals or compensatory responses of organisms (such as tolerance [acclimation, adaptation], or reduced density-dependent mortality among juveniles) were excluded.
- Included studies must have followed good laboratory practices (e.g., presence of control group), a defensible experimental design, and accepted statistical procedures for data analysis.
- If a member of a family of freshwater fish may occur at the site, then toxicity data from any fish species within that family were retained in the database.
- If a member of a family of amphibians may occur at the site, then toxicity data from any amphibian species within that family were retained in the database.
- If a member of a class of freshwater invertebrates may occur at the site, then toxicity data from that invertebrate class were retained in the toxicity database.
- If a member of a phylum of freshwater algae may occur at the site, then toxicity data from that phylum were retained in the database.
- Tests greater than 96 hours in duration were considered to be chronic for most species. This decision rule differs somewhat from the CCME (2007a) guidance, which specifies a seven-day minimum duration for a chronic designation. During the review it was observed that some tests (such as the three-brood *Ceriodaphnia dubia* reproduction test) could fall below the 7-day threshold due to the variations in the speed of reproduction of test cultures. This endpoint is considered to be a suitable chronic endpoint even where time to three broods falls below seven days. Some additional exceptions were made to the guidance on test duration, such as for metals exposures to the nematode *Caenorhabditis elegans*. For this organism,

tests 96 hours in duration were considered to be chronic because of their relatively short life span (Carleton-Dodds 2010, pers. comm.).

Site-Specific Screening

The CCME (2007a) derivation protocol emphasizes the importance of toxicity modifying factors in the development of water quality guidelines. It is acceptable, indeed preferable, to account explicitly for the physico-chemical factors that mediate the bioavailability or toxicity of constituents. In the context of PRM, it was desirable to screen the toxicity database to include only those studies that are reflective of the general water quality constituents expected in the PRM LSA, which are representative of the receiving environment.

Metal speciation in aquatic environments and potential toxicity to aquatic organisms is highly influenced by water quality variables such as pH and water hardness. To derive a site-specific chronic benchmark for metals, studies with no reported pH and water hardness, or with pH and water hardness values outside the expected range of values encountered in the study area (pH = 6.5 to 9.5 and hardness = 50 to 450 mg/L $CaCO_3$) were excluded. These ranges were considered sufficiently robust to include a range of conditions over the duration of PRM, while simultaneously excluding extremes of these constituents that would not be relevant during the operational life or closure stages of PRM. In addition, for CEBs based on hardness-dependent equations, a central tendency value of approximately 150 mg/L $CaCO_3$ was applied. Use of this hardness value results in conservative benchmarks for PRM because water hardness is generally slightly higher in the PRM LSA. For copper, the CEB derived from the Biotic Ligand Model (BLM) was customized to site-specific hardness, DOC and pH.

Photo-enhanced toxicity of PAHs has been demonstrated in laboratory and in a few in situ studies, and the mechanism of toxic action for this process has been well described (Boese et al. 1998; Harrison 2008). Given a certain combination of chemical exposure conditions and lighting conditions (specifically the ultraviolet [UV] light spectrum and degree of light exposure to animal skin surface during contaminant bioaccumulation) photo-enhanced toxicity of PAHs can significantly increase toxicity beyond normal levels. However, in most realistic environmental exposures, this phenomenon is ameliorated by physical, chemical and biotic factors (McDonald and Chapman 2002). Whereas the difference between UV and non-UV LC_{50} values is at least two orders of magnitude for non-arthropods in laboratory comparisons (Harrison 2008), such laboratory bioassays have been criticized for faults such as exaggerated UV exposure and PAH bioavailability. Swartz et al. (1997) stated that “the importance of phototoxicity in the derivation of PAH Sediment Quality Criteria (SQC) may ultimately be determined by the photoecology of benthic ecosystems.” Few if any studies clearly and directly implicate PAH phototoxicity with adverse ecological effects in field populations (McDonald and Chapman 2002).

Whereas the ecological relevance of phototoxicity in field communities remains in question, a number of site-specific factors that mitigate against the influence of photo-enhanced toxicity of PAHs will be present in the scenarios modelled for the EIA. For example, the configuration of pit lakes incorporates an increase in water depth, and watercourses in the LSA are naturally high in the concentrations of water quality constituents such as turbidity, suspended solids and DOC, which result in attenuation of the UV light spectrum. Therefore, PAHs phototoxicity-based endpoints were not included in the SSD derivation. This exclusion matches the approach of McGrath and DiToro (2009) that excludes photo-enhanced toxicity endpoints.

Given that the toxicity of PAHs to aquatic organisms is not known to be influenced by water quality variables such as pH and hardness, all exposure conditions for these constituents were considered appropriate for inclusion in the SSD derivation.

Endpoint Selection

For statistical endpoints, the preference ranking (i.e., the most preferred acceptable to the least preferred acceptable endpoint) was conducted following guidance from CCME (2007a):

- EC_x/IC_x representing a no-effects threshold;
- EC_{10}/IC_{10};
- EC_{11-25}/IC_{11-25};
- Maximum Allowable Toxicant Concentration (MATC), which was estimated using the geometric mean of the NOEC and the Lowest Observed Effects Concentration (LOEC);
- NOEC;
- LOEC;
- IC_{26-49} or EC_{26-49}; and
- IC_{50}/EC_{50}.

Regression-based endpoints, such as Inhibiting Concentration (IC), Effect Concentration (EC) or Lethal Concentration (LC) values, were given preference over hypothesis-based endpoints, such as LOEC and NOEC values. They are also the endpoints favoured by CCME (2007a) and U.S. EPA (2007a). The IC_{10}/EC_{10} results were given first priority because these results represent a conservative threshold for no negative effect, and are derived by regression analysis. If a study yielded multiple results for a single endpoint, then the results were reduced to a single measurement to avoid biasing the database toward the results of a single study.

Generally, effects on no more than 20% of exposed individuals is considered to be an acceptable threshold level for negative effects (CCME 2007a), and current risk assessment guidance recommended the use of IC_{20}/EC_{20} as permissible level of effects (Suter et al. 1995). However, IC_{25}/EC_{25} values are commonly reported in the literature, and such variations are considered to be within the range of natural variability often observed in the field among normal, unexposed populations. Therefore, in the absence of an IC_{10}/EC_{10} result, IC_{11-25} or EC_{11-25} values were used.

In some instances, the primary literature reported NOEC/LOEC concentrations without corresponding EC_x/IC_x values, but also provided tabular or graphical data that permitted estimation of the EC_x/IC_x. In these cases, the preference for an effect-size based endpoint outweighed the small uncertainty associated with interpolation or estimation from the raw data. The results of a concentration-response model fit to the study data (i.e., smoothed results) were used preferentially to single concentration test data for estimating EC_x/IC_x.

2.5.2 Step 2: Statistical Analysis of Toxicity Data

Statistical analysis of the assembled data points (and associated model fitting) was completed if data were available for five or more species. The statistical analysis consisted of the following:

- developing species mean values;
- ranking the species mean values to determine percent affected; and
- fitting a statistical distribution to the available dataset, if appropriate.

Species means were calculated by taking the geometric mean of the individual test results. The geometric mean, as opposed to the arithmetic mean, was used to limit the bias of high test results. Species mean values were then ranked from lowest to highest, and the percent of species affected was calculated based on dividing the rank assigned to each species mean by the total number of species. Logistic relationships between the percent of species affected and concentration of the constituent in question were evaluated using SigmaPlot version 11.0 (SPSS 2002).

Minimum data requirements for SSDs that have been recommended by various authorities range from 3 to more than 20 (Suter et al. 2002). The CCME (2007a) specifies a minimum of seven species, but other jurisdictions differ (e.g., Danish soil quality criteria require a minimum of five species [Suter et al. 2002]). Species sensitivity derivations developed with more species are likely more robust. However, the benefit of basing the CEB on all relevant and available toxicity data (i.e., in an SSD) was considered to outweigh the potential increase in uncertainty arising from having relatively few data.

2.5.3 Step 3: Identification of Chronic Effects Benchmark

Step 3a – Derivation of the HC_5 Concentration

After an appropriate regression model was developed, the HC_5 value was calculated using the model equation. The HC_5 value was then used as the CEB for the constituent in question. In addition, the HC_{20} value and HC_{50} value were reported to provide context on the shape and steepness of the concentration-response curve.

Step 3b – Selection of the Lowest Reported Chronic Value

For several constituents, toxicity data were available for fewer than five species. Chronic effects benchmarks for these constituents were based on the lowest chronic toxicity result present in the constituent-specific toxicity database.

Previously Evaluated Constituents

The list of constituents requiring new SSD derivation was refined to consider the previous work conducted for similar developments such as Syncrude (2009) and Total (2010). These developments provided numerous water quality guidelines that were already consistent with the CCME (2007a) derivation procedure. The CEBs drawn from the technical derivations found in previous assessments included: antimony, barium, beryllium, cadmium, chromium, iron, lead, manganese, molybdenum, strontium and vanadium. Although these derivations were not all site- or region-specific, many of these CEBs were considered to be applicable to PRM as preliminary CEBs. Additional refinement of these CEBs would only be warranted if the predicted concentrations exceeded the CEBs. For completeness, the full derivations for these constituents are included in this appendix.

2.6 Chronic Effects Benchmark Results for Metals

2.6.1 Aluminum

Aluminum can be found in the natural environment generally associated with igneous rocks (composed of alumino-silicate materials), bauxite materials (composed of aluminum hydroxides), and cryolite (Staley and Haupin 1992). Aluminum can also react and form complexes with chloride, fluoride, sulphate, nitrate, phosphate, and negatively charged compounds such as humic materials and clay (ATSDR 2008). Based on natural factors that can ameliorate aluminum toxicity under circumneutral conditions, aluminum "is not a toxicological problem in the majority of freshwater environments" (Wilson 2012, p 70). This element does not readily bioconcentrate in aquatic organisms (Rosseland et al. 1990).

Aluminum exhibits greater toxicity in acidic pH (< 6) and alkaline (pH >8) conditions (Freeman and Everhart 1971; Hamilton and Haines 1995). The majority of studies encountered during the literature review focused on toxicity of aluminum in water of pH less than 6.5. Only those studies conducted at pH ≥ 6.5 were considered in the SSD derivation, as the receiving environment for PRM is circumneutral in pH. It has also been demonstrated that hardness can act as a modifying factor in moderately alkaline water (Gundersen et al. 1994). However, the influence of hardness on toxicity in circumneutral water is uncertain. Hardness data were not reported in many of the available chronic studies; therefore, toxicity studies were not screened on the basis of hardness. Although DOC and phosphate are also potentially important toxicity modifying factors due to their influence on complexation of aluminum, these parameters were not explicitly considered for screening chronic studies because they were not reported in the majority of the studies reviewed.

Inclusion of the majority of toxicity data provides a conservative assessment method because the protective effect of toxicity modifying factors has not been incorporated. Recent publications indicate that the complexation of aluminum under natural conditions yields reduced bioavailability and toxicity relative to the test conditions used in laboratory exposures (Trenfield et al. 2012; Wilson 2012). Gensemer and Playle (1999) review factors that ameliorate toxicity of aluminum to freshwater aquatic life, including complexation to dissolved organic matter, hardness-dependent amelioration, and antagonistic (protective) effects of other elements including calcium, fluoride, and silicon. However, the available database of toxicity values is difficult to evaluate in terms of these factors, at least in quantitative terms. Derivation of an aluminum benchmark is "restricted by our limited understanding of Al bioavailability" under site-specific conditions; in particular, the toxicity of the aluminate ion $Al(OH)^{4-}$, which predominates at pH > 7, is very poorly understood (Gensemer and Playle 1999).

Sufficient acceptable data were available to derive an SSD for aluminum. Chronic toxicity data were available for three fish species (including two salmonids and one non-salmonid), four invertebrate species (three crustaceans), and five plant and algae species. The available chronic toxicity data indicate that fish are the most sensitive taxonomic group to aluminum at circumneutral pH. Effects concentrations for fish ranged from a 7 day embryo-large survival MATC of 7.7 µg/L for the goldfish (Birge 1978) to a 45 day growth IC_{42} of 514 µg/L for rainbow trout (Freeman and Everhart 1971). Invertebrate toxicity to aluminum was highly variable, ranging from a 7 day LC_{50} of 89 µg/L for the amphipod *Hyalella azteca* (Borgmann et al. 2005) to an 8 day reproduction MATC of 7,700 µg/L for the water flea *Ceriodaphnia dubia* (Call et al. 1984). Algae and plants were the least sensitive to aluminum, with Species Mean Chronic Values (SMCVs) ranging from 400 to 54,000 µg/L.

Two species exhibited lower SMCVs than the CCME WQG and strongly influence the derived value: the goldfish *Carassius auratus* and the amphipod *Hyalella azteca*. The most sensitive species in the SSD was the goldfish

(Birge 1978). The uncertainty and ecological relevance of this endpoint were considered to determine whether the derived HC_5 was unduly driven by a false positive or unrepresentative data. Although goldfish are not native to the study area, they were initially retained as a surrogate for species in the carp family. The MATC for goldfish was calculated as the geometric mean of the LC_1 and LC_{50} reported by Birge from a 7 day embryo-larval test. This MATC of 7.7 is within the range of background aluminum concentration in northern Canadian lakes, and is therefore considered overly conservative for inclusion in the SSD.

The Birge study included testing of a number of other metals, in addition to aluminum. A review of the U.S. EPA's water quality criteria for aluminum, arsenic, cadmium, chromium, copper, and selenium revealed that the corresponding data from this study was listed as 'other data' but were not included in the datasets used for criteria derivation; no reason was given for this exclusion. The Birge (1978) and Birge et al. (1979, 1980a;1980b) data have been found to yield anomalously low toxic concentrations for numerous microelements (Shell 2012) and were excluded from the SSD for molybdenum by De Schamphelaere et al. (2010) on the basis of quality control screening. Therefore, the results from these experiments were considered questionable and were subsequently removed.

The second most sensitive species in the SSD was the amphipod, *Hyalella azteca* (Borgmann et al. 2005). *H. azteca* was originally retained as a surrogate for the invertebrate class Amphipoda, species of which have been identified in the PRM LSA. Borgmann et al. (2005) conducted the 7-d amphipod tests to determine the effects of aluminum on survival in waters at two different hardness levels. In soft water (hardness of 17 mg/L), the 7-d LC_{50} was 89 µg/L and in the higher-hardness Lake Ontario water (hardness of 169 mg/L), the 7-d LC_{50} was >3,500 µg/L. A less conservative and more realistic exposure regime relevant to PRM would consider intermediate hardness (i.e., between the hardness levels referenced above). The geometric mean of the two LC_{50} values is 558 µg/L, similar to chronic toxicity values observed for other aquatic invertebrates, and a more reasonable reflection of the hardness conditions found in the PRM receiving environment.

The SSD excluded data from Birge (1978) and considered the Borgmann et al. (2005) result with the geometric mean chronic LC_{50} of 558 µg/L. The endpoints that were used to generate the revised SSD for aluminum are summarized in Table 2.6-1.

Table 2.6-1 Available Chronic Freshwater Toxicity Data for Aluminum Retained for Species Sensitivity Distribution

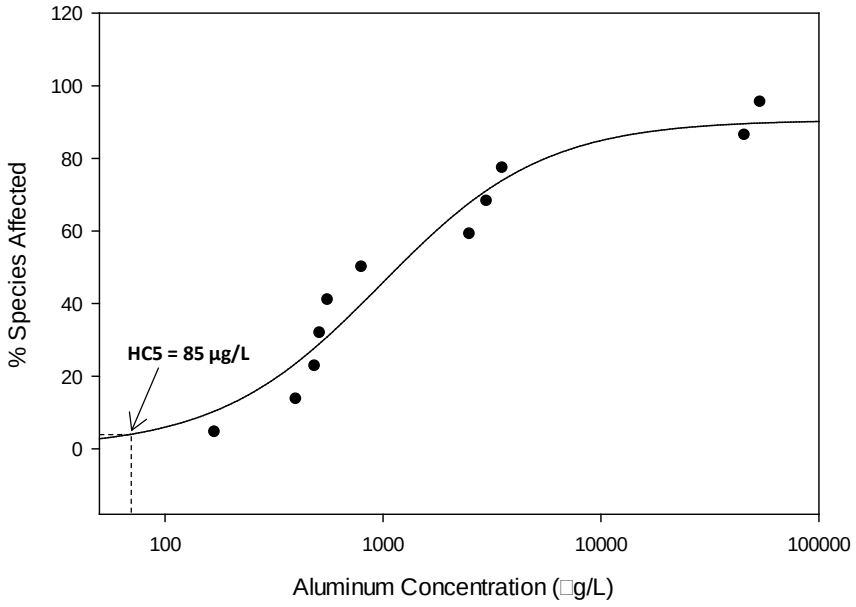
Test Species	Species Common Name	Species Mean Chronic Value (SMCV)	Rank	% Species Affected
Salvelinus fontinalis	brook trout	169	1	4.5
Pseudokirchneriella subcapitata	green algae	400	2	13.6
Daphnia magna	water flea	487	3	22.7
Oncorhynchus mykiss	rainbow trout	514	4	31.8
Hyalella azteca	amphipod	558	5	40.9
Tanytarsus dissimilis	midge	800	6	50
Myriophyllum spicatum	Eurasian watermilfoil	2,500	7	59.1
Ceratophyllum demersum	hornwort	3,000	8	68.2
Ceriodaphnia dubia	water flea	3,536	9	77.3
Lemna minor	duckweed	45,700	10	86.4
Chlorella vulgaris	green algae	54,000	11	95.5

An additional update to the CEB derivation for aluminum entailed the use of the Hazen plotting position method, whereby the species mean chronic values were ranked from lowest to highest, and the percent of species affected was calculated using the following equation:

$$\text{Percent Affected} = (X - 0.5) / N$$

where X is the species rank, with 1 being the most sensitive species, and N is the total number of species in the database. The correction factor of 0.5 was used (Aldenberg et al. 2002) to create symmetry in cumulative probability (i.e., median ranked species will be associated with 50% affected) and to acknowledge that the concentration affecting the highest ranked species is not necessarily associated with adverse effects to the entire aquatic community. SigmaPlot software was used to fit the chronic data to a curve for the SSD, using a logistic four-parameter model. However, visual inspection of the model (Figure 2.6-1) indicated poor model fit in the lower tail of the distribution. The three lowest SMCVs were associated with a lower percentage of species affected than predicted by the curve. Thus, the predicted HC_5 is lower than that represented by the data. Use of other models did not yield an improved fit. The lowest reliable SMCV (169 µg/L for brook trout) was associated with an effect to 4.5% of species. Therefore, the existing CCME WQG of 100 µg/L is sufficiently low to protect the most sensitive species included in the SSD. Given the uncertainty associated with individual endpoints and with model fitting, the small difference between the lowest SMCV and the CCME WQG was considered insufficient to warrant replacement of the WQG.

Figure 2.6-1 Species Sensitivity Distribution Curve for Aluminum



One of the conceptual advantages of the SSD assessment method is that the underlying dataset can be screened for Environmental Toxicity Modifying Factors (ETMFs) of relevance to PRM. However, in the case of aluminum, it was not possible to control for all parameters that could influence bioavailability and toxicity without

diminishing the quantity of data available for developing an SSD. Only pH was explicitly considered in the SSD derivation, and other ETMFs (e.g., hardness, DOC) could not be quantitatively considered due to lack of data reported in the toxicity studies. Among the studies with reported hardness, most of the hardness concentrations were in the range of 30 mg/L to 50 mg/L (as $CaCO_3$), which is lower than the typical hardness found in the PRM receiving environment. Therefore, exclusion of hardness as a screening factor may influence the outcome of the SSD. The behavior and fate of aluminum in freshwater systems is highly complex and dependent on several variables. Aluminum can react and form complexes with various ions (e.g., chloride, fluoride, sulphate, nitrate, and phosphate), as well as DOC (ATSDR 2008; Wilson 2012), which may serve to reduce the bioavailability and subsequent toxicity to aquatic organisms in receiving water containing these constituents. Inorganic monomeric aluminum species (i.e., dissolved or labile aluminum) are considered the most toxic forms of aluminum (Wilson 2012). Organic and complexed aluminum is less available and therefore less toxic to aquatic life. All of the studies reviewed used relatively soluble and available forms of aluminum (i.e., inorganic aluminum salts) in the test treatments. Most of the studies reviewed did not distinguish between dissolved and total aluminum when reporting results. Therefore, the SSD generated based on these laboratory studies is likely conservative; in other words yields an HC_5 value likely to be substantially lower than the concentration causing responses to 5% of species in field exposures.

Differences in the aging of aluminum stock solutions before their dilution and delivery to toxicity test exposure tanks can also influence toxicity, and insufficiently long aging periods can result in overly conservative test results (Wilson 2012). Information on the aging of stock solutions prior to test initiation was not provided in the studies reviewed.

The updated analysis of toxicity data for aluminum provided sufficient information to revise the SSD. The HC_5 value for the SSD was close to the CCME water quality guideline, and therefore the guideline was retained as the CEB. However, in applying this CEB of 100 µg/L, several factors were identified that suggest that the guideline is highly conservative and may greatly overstate the toxicity of aluminum in natural waters. In particular, the mineral-associated, organic, and complexed forms of aluminum that would dominate in site waters would be substantially reduced in bioavailability relative to the dissolved forms used in published toxicity studies.

2.6.2 Antimony

Two forms of antimony (Sb) can exist in the dissolved phase (ATSDR 1997); however, most antimony released into waterways is associated with particulate matter. Dissolved antimony (Sb^{3+}) occurs under moderately oxidizing conditions, whereas dissolved antimony (Sb^{5+}) predominates in highly oxidizing environments (NWQMS 2000). The toxicity of antimony depends largely upon its chemical form and oxidation state, with Sb^{3+} exerting greater toxicity than Sb^{5+} (Hou and Narasaki 1999). Consequently, most toxicity studies focus on Sb^{3+}.

Insufficient data were available to create an SSD for antimony. The lowest reported toxicity value was a 30-day NOEC of 7.5 µg/L for survival and growth of fathead minnow embryos (LeBlanc and Dean 1984); however, this NOEC was unbounded (i.e., a LOEC could not be calculated in the study). It was also very low in comparison to other toxicity estimates. Therefore, the next lowest reported toxicity value of 157 µg/L was selected for use as the CEB for antimony. This value is based on a 28-day LC_{10} test result generated using rainbow trout (Birge et al. 1979, 1980a) and is considered to be conservative, because it is based on the more toxic form (i.e., Sb^{3+}).

2.6.3 Arsenic

Arsenic (As) is widely distributed in the natural environment. This element has four oxidation states: As^{3-}, As^{0}, As^{3+}, and As^{5+}; however, in aquatic systems, inorganic arsenic occurs primarily as As^{5+} and As^{3+}. The valency and chemical form of arsenic have a large influence on its behaviour and toxicity in aquatic systems. In general, the inorganic forms of arsenic (arsenate [As^{5+}] and arsenite [As^{3+}]) are the most toxic forms, whereas methylated compounds (e.g., methylarsonate) are less toxic, and more complex organoarsenic compounds (e.g., arsenobetaines) are generally non-toxic (Leverone 2007). Some studies have demonstrated that arsenate (As^{5+}) is less toxic to algae and invertebrates compared to arsenite (As^{3+}) (Borgmann et al. 1980; Naumann et al. 2007). The pentavalent form (As^{5+}) predominates under oxidizing conditions and As^{3+} predominates under reducing conditions, and the former conditions are more applicable to the PRM receiving environment; therefore toxicity data for arsenate were preferentially selected for inclusion in the SSD to reflect site specific conditions[1]. Additionally, the bioavailability of arsenic can be reduced by the presence of natural organic matter, phosphorus, metal sulphides, and iron and aluminum oxides (Redman and Macalady 2003; Reuther 1992; Senn and Hemond 2004).

Data were available for a wide range of species. Some species were excluded from the SSD based on lack of presence at northern sites (e.g., toads, sunfish, catfish). Overall, chronic toxicity data for 24 species were retained for the SSD (Table 2.6-2): four fish species (one salmonid and three non-salmonids), eight invertebrate species (six crustaceans and two gastropods), ten algae species, and two aquatic plant species (duckweed). CCME (2007a) recommends the inclusion of a stonefly, mayfly or caddisfly species in the SSD, and one relevant study was identified for these groups. Spehar et al. (1980) reported that no effects were observed to the stonefly species *Pteronarcys dorsata* exposed to 973 µg/L of arsenate. However, because this was the highest concentration tested (i.e., an unbounded NOEC), the true effects benchmark is not known. Therefore, this result was not included in the SSD. These results do suggest, however, that stoneflies are not particularly sensitive to arsenic, given that SMCVs equal to or lower than 973 µg/L were calculated for numerous other invertebrate species, including water fleas, amphipods, and snails.

The SMCVs in Table 2.6-2 suggest that algae are among the most sensitive species to arsenic, with algal species comprising four of the five most sensitive species. Algae also exhibited the highest variability in sensitivity among species, with SMCVs ranging from 10 µg/L to 100,000 µg/L. Fish were generally less sensitive to arsenic, with the exception of the goldfish, which ranked fourth out of 24 species. Rainbow trout and fathead minnow, in contrast, were among the least sensitive species to arsenic. The goldfish data were taken from Birge et al. (1978). As discussed for aluminum (Section 2.6.1), the Birge study has generated questionable and anomalous results for several parameters and was not included in the datasets used for criteria derivation by the U.S. EPA. Birge (1978) reported an LC_{10} of 134 µg/L for rainbow trout as part of the same series of studies. This value was much lower than the chronic endpoints reported in other studies (9,640 µg/L EC_{20} for growth [Rankin and Dixon 1994]; 4,243 µg/L MATC for growth [Speyer 1975]). Invertebrate species exhibited less variation in their sensitivity to arsenic, with SMCVs ranging from 100 µg/L for *Daphnia pulex* to 1,420 µg/L for *Ceriodaphnia dubia*.

[1] Speciation results were not used for screening fish data due to a lack of arsenate data; therefore both arsenate and arsenite endpoint data are included for fish.

Table 2.6-2 Available Chronic Freshwater Toxicity Data for Arsenic

Test Species	Common Name	SMCV	Rank	% Affected
Scenedesmus obliquus	green algae	10	1	2.1
Melorisa granulata	diatom	75	2.5	8.3
Ochromonas vallesiaca	algae	75	2.5	8.3
Carassius auratus	goldfish	87	4	15
Ankistrodesmus falcatus	green algae	100	5.5	21
Daphnia pulex	water flea	100	5.5	21
Lemna gibba	inflated duckweed	224	7	27
Cryptomonas erosa	algae	225	8	31
A. testudineus	climbing perch	500	9	35
Hyalella azteca	amphipod	581	10	40
Gammarus pseudolimnaeus	amphipod	973	12	48
Helisorna campanulata	snail	973	12	48
Stagnicola emarginata	snail	973	12	48
Daphnia magna	water flea	1,070	14	56
Cyclops vernalis	copepod	1,380	15	60
Ceriodaphnia dubia	water flea	1,420	16	65
Oncorhynchus mykiss	rainbow trout	1,763	17	69
Anabaena variabilis	phytoplankton	2,250	18.5	75
Chlamydomonas reinhardtii	green algae	2,250	18.5	75
Pimephales promelas	fathead minnow	2,589	20	81
Scenedesmus quadricauda	algae	5,490	21	85
Lemna minor	duckweed	8,709	22	90
Pseudokirchnerella subcapitata	green algae	25,000	23	94
Microcoleus vaginatus	green algae	100,000	24	98

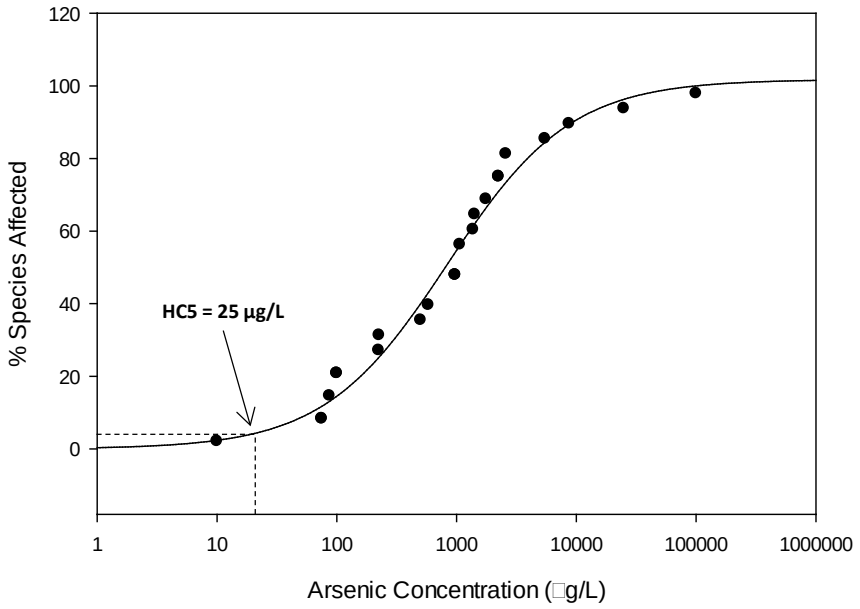
The logistic model was used to derive an SSD for arsenic, based on the data shown in Table 2.6-2. The model provided a good fit to the data (r^2 = 0.98), and followed the form:

$$y = \frac{102.6}{1 + \left(\frac{x}{876}\right)^{-0.793}}$$

where: y = percent of aquatic community affected; and
x = arsenic concentration (µg/L).

The resulting HC_5 was 25 µg total As/L (Figure 2.6-2). Visual inspection of Figure 2.6-2 confirms a good model fit, including those results in the lower tail of the distribution that sets the HC_5.

Figure 2.6-2 Species Sensitivity Distribution Curve for Arsenic



Overall, the dataset for arsenic was considered very robust, as it included 24 species from various trophic levels, and fully satisfied the data requirements of CCME (2007a). Site-specific ETMFs were captured to some extent through screening of chemical exposures (e.g., excluding arsenite data for algae and invertebrates). Speciation results were not used for screening fish data due to a lack of arsenate data. Only one suitable entry was included for arsenate: a 30-day EC_{18} for growth of 1,500 µg/L for fathead minnow (De Foe 1982). This result was approximately three times lower than the EC_{13} for arsenite (4,467 µg/L) for the same species reported by Spehar and Fiandt (1986). Sufficient data are not available to evaluate the relative toxicity of arsenite and arsenate to fish; inclusion of both forms was prudent considering the comparability of toxicity results for fathead minnow.

The goldfish and rainbow trout toxicity data from Birge et al. (1978, 1979) are considered highly conservative and may be false positives, for reasons discussed previously. The SSD derivation was repeated with the exclusion of the Birge data to determine the sensitivity of the HC_5 to these potential outliers. The amended SSD resulted in an HC_5 of 29 µg/L, which is only slightly higher than the HC_5 of 25 µg/L including all data. As the Birge data did not substantially influence the SSD equation, they were conservatively retained.

The reanalysis of arsenic toxicity data provided sufficient data to develop an SSD-based CEB of 25 µg/L. This CEB is protective of sensitive taxonomic groups, including algae, and is approximately four times lower than the benchmarks for toxicity of fish and aquatic invertebrates. The CEB incorporates some, but not all, of the toxicity modifying factors for arsenic, and is expected to be a conservative indication of the site-specific benchmark for aquatic toxicity.

2.6.4 Barium

The acetate, nitrate and halide salts of barium are soluble in water, but the carbonate, chromate, fluoride, oxalate, phosphate and sulphate salts are relatively insoluble. The aqueous solubility of barium compounds increases as pH decreases. Organometallic barium compounds are ionic and are hydrolyzed in water. The concentration of barium ions in natural aquatic systems is limited by naturally occurring anions, such as sulphates and carbonates, and by the possible adsorption of barium ions onto metal oxides and hydroxides.

Insufficient data were available to develop an SSD for barium. The lowest reported toxicity value of 5,800 µg/L was, therefore, conservatively selected for use as the CEB for barium. This value is based on an EC_{16} reproduction test result generated using the water flea *Daphnia magna* (Biesinger and Christensen 1972).

2.6.5 Beryllium

Beryllium toxicity and speciation varies with pH changes in the environment. Formation of solid beryllium hydroxide ($Be[OH]_2$) occurs in most aquatic systems with ranges of pH 6 to 8. Beryllium can also form insoluble carbonates and soluble beryllium sulphates in aquatic environments.

Insufficient data were available for beryllium to develop an SSD. The lowest reported toxicity value of 5.3 µg/L was, therefore, conservatively selected for use as the CEB for beryllium. This value is based on a 28-day MATC for reproduction in *Daphnia magna* (Kimball 1978).

2.6.6 Boron

The CCME water quality guideline for the protection of aquatic life is 1.5 mg/L of boron for long-term exposures (CCME 2009). This value was derived based on an SSD of long-term toxicity endpoints, which yielded a fifth percentile value (HC_5) of 1.5 mg/L with confidence limits of 1.2 mg/L and 1.7 mg/L. As this derivation included a number of long-term endpoints for fish, invertebrates, plants and amphibians, the HC_5 derivation is considered consistent with the CCME (2007a) derivation procedure. Many of the endpoints were based on NOECs, LOECs or MATCs (which do not convey the effect size and are not preferred endpoints); however, there is not a compelling reason to discard the CCME analysis for an alternative derivation.

Toxicity modifying factors were not applied to the boron guideline because evidence for the influence of water hardness on toxicity is mixed. Some species exhibit reduced toxicity at high hardness, whereas others show no response. Although reduced toxicity has been indicated in natural waters and in waters with elevated organic carbon content, there is not sufficient information to quantify the relationship (CCME 2009).

2.6.7 Cadmium

Cadmium (Cd) is usually found as a mineral combined with other elements, such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulphur (cadmium sulphate, cadmium sulphide). It may exist in water as a hydrated ion, as inorganic complexes (such as carbonates, hydroxides, chlorides or sulphates) or as organic complexes with humic acids (OECD 1994). Cadmium may enter aquatic systems through weathering and erosion of soils and bedrock, atmospheric deposition, direct discharge from industrial operations, leakage from landfalls and contaminated sites, and the dispersive use of sludge and fertilizers in agriculture. The predominant dissolved form of cadmium in freshwater is the cadmium ion (Cd^{2+}), which is the form that is most bioavailable to aquatic biota (Wright and Welbourn 1994). Upon entry to the aquatic ecosystem, cadmium tends to partition to particulate matter and dissolved organic matter, reducing concentrations of the free ion in the water column, thereby lowering its bioavailability (Jonnalagadda and Rao 1993).

Modifying factors, such as hardness, salinity, pH and DO concentrations, can have a profound effect on cadmium toxicity to aquatic plants and animals. Ions, such as hydrogen and calcium, may compete with cadmium, resulting in reduced cadmium uptake and toxicity (Wright and Welbourn 1994). The toxicity of cadmium to fish is strongly affected by hardness, mainly because of competition for anionic binding sites at the gills between cadmium ions and ions responsible for hardness (i.e., calcium and magnesium) (Parametrix 1995). Because the free divalent cadmium ion (Cd^{2+}) is the most toxic form, water quality factors that sequester or otherwise limit uptake of cadmium ions across biological membranes will reduce toxicity to aquatic life (CCME 2012). There are three main processes that limit the uptake of divalent cadmium ions at respiratory surfaces, where most uptake occurs: (1) competition effects, particularly from calcium [Ca^{2+}] and magnesium [Mg^{2+}] in surface water; (2) complexation of Cd^{2+} ions with suspended particles, colloidal materials, and/or dissolved organic matter in the water column, rendering them unavailable for uptake; and (3) changes to the form of cadmium, which can occur at extremes in pH (CCME 2012; Roch and Maly 1979).

Both Canada and the United States have developed chronic water quality guidelines for cadmium. Both the CCME (2012) and U.S. EPA (2001) derivations incorporate hardness-toxicity relationships and apply the 5th percentile of a species sensitivity distribution method. The Canadian Council of Ministers of the Environment (CCME) has recently issued (in draft form) a revised derivation document for cadmium based on compilations of recently published data (CCME 2012). The long-term exposure guidelines for medium hardness and high hardness water conditions (which bracket the range of conditions found in the Pierre River watershed) are:

- medium (120 mg/L): 0.21 µg/L Cd;
- hard (180 mg/L): 0.29 µg/L Cd; and
- Project-specific (150 mg/L): 0.25 µg/L Cd.

Based on a combination of the above national water quality derivation documents and an independent search of the literature, effect concentrations were compiled and screened using the CCME (2007a) derivation guidance, as shown in Table 2.6-3. The species mean chronic toxicity values from this compilation are depicted in Figure 2.6-3. The lowest SMCV was 0.25 for the amphipod *Hyalella azteca* (0.25 µg/L Cd; normalized to 50 mg/L hardness). Once adjusted to a hardness level representative of the PRM receiving environment (150 mg/L), and using the hardness conversion factor of CCME (2012), the most sensitive SMCV equals 0.62 µg/L Cd. Based on the strong representation of freshwater aquatic organisms in the data set, no application factor is required.

From the above information, the range of project-specific CEBs is from 0.25 to 0.62 µg/L Cd, once adjusted for site-specific hardness conditions.

Table 2.6-3 Summary of Compiled Chronic (Long-term) Freshwater Aquatic Toxicity Tests With Cadmium Exposures, Indicating Preferred Test Endpoint for Each Study, and Species Mean Values

Species Common Name	Scientific Name	Life Stage	Duration [b]	Endpoint	Observed Effect	Test Hardness [mg/L]	Observed Effect Concentration [µg/L]	Effect Concentration [µg/L] at 50 mg/L Hardness [a]	Species Mean Chronic Value [µg/L] at 50 mg/L Hardness [a]	pH
Amphipod - gammarid	*Echinogammarus meridionalis*	Adult	6 d	MATC	Feeding inhibition	263	5.2	1.3	1.3	8.0
Amphipod - gammarid	*Gammarus pulex*	Adult	5 d	LOEC/L	Mortality	269	7.5	1.9	1.9	7.2
Amphipod - scud	*Hyalella azteca*	7-8 d old	28 d	IC25	Biomass, decrease in	280	0.5	0.1	0.3	7.8
		Juvenile	7 d	LC50	Mortality	18	0.2	0.4		7.4 (6.4-8.5)
		<7 d old	42 d	LC50	Mortality	130	0.5	0.2		7.90-9.00
		7-8 d old	42 d	MATC	Mortality	163	0.7	0.3		7.90 (0.10)
		Juvenile	14 d	MATC	Mortality	17	0.2	0.4		5.50-7.70
Oligochaete	*Aeolosoma headleyi*	Juvenile	14 d	MATC	Population growth	168	40.1	14.7	14.7	NR
Fatmucket	*Lampsilis siliquoidea*	Juvenile	28 d	IC10	Length	44	4.6	5.1	5.1	7.20-7.60
Mayfly	*Rhithrogena hageni*	Nymph	10 d	EC10	Mortality	48	2,570.0	2,660.0	2,660.0	7.66
Dragonfly	*Pachydiplax longipennis*	Larva	7 d	NOEC/L	Survival	120	100,000.0	48,353.0	48,353.0	6.24
Water flea	*Ceriodaphnia reticulata*	Less than 24 hrs	7 d	MATC	Reproduction - young per adult	240	0.4	0.1	0.7	8.0 +- 0.3
		Less than 24 hrs	9 d	MATC	Reproduction	67	4.9	3.8		7.2-7.8
Water flea	*Ceriodaphnia dubia*	Not reported	14 d	MATC	Reproduction	17	2.0	4.9	4.9	5.5-7.7
Water flea	*Daphnia magna*	Adult	7 d	EC10	Reproduction - brood mass	179	0.1	0.1	0.3	8.07 +- 0.07
		Less than 24 hrs	21 d	EC16	Reproduction	45	0.2	0.2		7.74 (7.4-8.2)
		Not reported	21 d	MATC	Reproduction - young per adult	130	0.6	0.3		-
		Less than 24 hrs	21 d	MATC	Reproduction - young per survivor	103	0.2	0.1		7.9 +- 0.3
		Less than 24 hrs	14 d	EC50	Reproduction - young per adult	240	3.5	1.0		8.0 +- 0.3
		24 h	21 d	NOEC/L	Reproduction	250	0.6	0.2		8.0 +- 0.2
		Not reported	7 d	MATC	Mortality	78	7.1	4.9		6.9-8.3
		Neonate	7 d	MATC	Growth	90	1.2	0.7		-
Water flea	*Daphnia pulex*	Less than 24 hrs	42 d	MATC	Reproduction - brood size	230	7.4	2.1	3.1	8.3-9.0
		Less than 24 hrs	58 d	MATC	Reproduction - brood size	106	7.1	3.8		8.5
		Less than 24 hrs	42 d	MATC	Reproduction - brood size	58	3.6	3.2		8.3-9.0
		Less than 24 hrs	14 d	MATC	Reproduction - young per adult	240	13.7	3.7		8.0 +- 0.3
European shrimp	*Atyaephyra desmarestii*	Adult	6 d	MATC	Feeding inhibition	263	5.2	1.3	1.3	7.9
Great pond snail	*Lymnaea stagnalis*	Adult	4 weeks	NOEC/L	Growth	284	80.0	19.0	18.9	6.7-8.1
Marsh snail	*Lymnaea palustris*	Adult	4 weeks	EC50	Growth	284	58.2	13.8	13.8	6.7-8.1
Midge	*Chironomus riparius*	1st instar	17 d	MATC	Mortality	98	47.4	27.1	27.1	7.6
Midge	*Chironomus tentans*	Less than 24 hrs	60 d	IC25	Hatching success	280	4.0	1.0	15.3	7.8
		2nd instar	14 d	LOEC/L	Growth	17	100.0	244.8		5.5-7.7
Duckweed	*Lemna minor*	Not reported	7 d	EC50	Growth rate	166	214.0	79.0	79.0	5.5 +- 0.2
Green algae	*Ankistrodesmus falcatus*	Population	96 h	NOEC/L	Growth	118	10.0	4.9	4.9	7.7 +- 0.5
Green algae	*Pseudokirchneriella subcapitata*	Population	96 h	NOEC/L	Growth	118	5.0	2.5	5.4	7.7 +- 0.5
		Population	72 h	EC50	Growth	250	43.5	11.4		8.1
		Population	72 h	Mean EC10	Growth rate	50	5.7	5.7		-
Northwestern salamander	*Ambystoma gracile*	Larva	24 d	MATC	Weight	45	97.2	106.1	106.1	6.8
Atlantic salmon	*Salmo salar*	Egg	496 d	LOEC/L	Weight and Length	28	0.5	0.8	0.8	7.3 (6.8-7.5)
Brook trout	*Salvelinus fontinalis*	Larva	126 d	MATC	Biomass, decrease in	45	2.0	2.2	2.7	7.6 (7.2-7.8)
		Fry	60 d	MATC	Weight	37	1.9	2.5		6.5-7.2
		Mixed	1100 d	MATC	Mortality	44	3.2	3.6		7.0-8.0
Brown trout	*Salmo trutta*	Swim-up fry	30 d	IC20	Biomass, decrease in	29	0.9	1.4	1.3	7.5 (0.1)
		Larva	61 d	NOEC/L	Biomass, decrease in	45	1.1	1.2		7.6 (7.2-7.8)

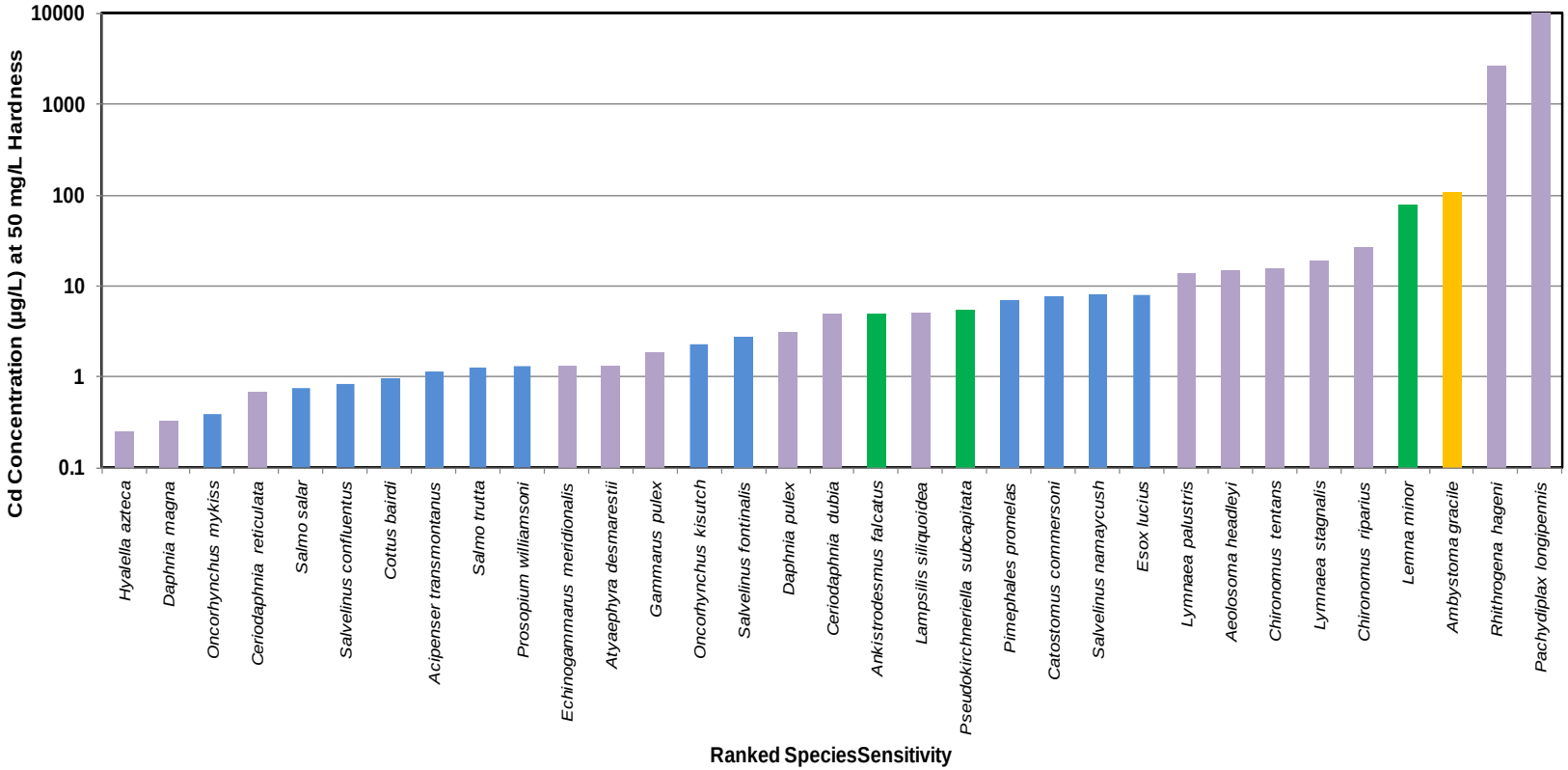
Table 2.6-3 Summary of Compiled Chronic (Long-term) Freshwater Aquatic Toxicity Tests With Cadmium Exposures, Indicating Preferred Test Endpoint for Each Study, and Species Mean Values (continued)

Species Common Name	Scientific Name	Life Stage	Duration [(b)]	Endpoint	Observed Effect	Test Hardness [mg/L]	Observed Effect Concentration [µg/L]	Effect Concentration [µg/L] at 50 mg/L Hardness[(a)]	Species Mean Chronic Value [µg/L] at 50 mg/L Hardness[(a)]	pH
Bull trout	*Salvelinus confluentus*	Juvenile	55 d	MATC	Growth	31	0.6	0.8	0.8	7.6
Coho salmon	*Oncorhynchus kisutch*	Embryo	27 d	MATC	Biomass, decrease in	45	2.1	2.3	2.3	7.6 (7.2-7.8)
Fathead minnow	*Pimephales promelas*	Juvenile	32 d	MATC	Mortality	67	18.9	14.8	6.9	7.2-7.8
		Nor reported	32 d	MATC	Mortality	44	10.0	11.1		6.0-8.1
		Juvenile (4 to 6 days)	7 d	MATC	Mortality	278	9.2	2.2		8.4-8.6
		Fry	250 d	MATC	Mortality	204	39.2	12.2		7.6
		Larva	10 d	MATC	Mortality	17	1.4	3.4		5.5-7.7
Lake trout	*Salvelinus namaycush*	Larva	64 d	MATC	Biomass, decrease in	45	7.4	8.0	8.0	7.6 (7.2-7.8)
Mottled sculpin	*Cottus bairdi*	Swim-up fry	21 d	LC50	Mortality	104	1.7	1.0	1.0	8.2
Northern pike	*Esox lucius*	Embryo	35 d	MATC	Biomass, decrease in	45	7.4	8.0	8.0	7.6 (7.2-7.8)
Rainbow trout	*Oncorhynchus mykiss*	Adult	455 d	MATC	Reproduction - delay in oogenesis	250	0.9	0.2	0.4	7.4-8.0
		Unknown	100 d	NOEC/L	Mortality	217	3.6	1.1		6.9
		Early life stage	62 d	EC10	Weight	29	0.2	0.2		7.2
White Sucker	*Catostomus commersoni*	Embryo	40 h	MATC	Biomass, decrease in	45	7.1	7.8	7.8	7.6 (7.2-7.8)
Sturgeon	*Acipenser transmontanus*	Fry	58 d	LC20	Mortality	70.0	1.5	1.1	1.1	7.9 +-0.2
Mountain whitefish	*Prosopium williamsoni*	Embryo and fry	90 d	IC10	Weight, biomass	48	1.3	1.3	1.3	6.8+-0.18

(a) If adjusted from another hardness, value was calculated using the following equation: 10 ^ (LOG_{10}(EFFECT conc)-(0.83)*(LOG_{10}(measured water hardness)-LOG_{10}(desired water hardness)).

(b) d= days and h = hours.

Figure 2.6-3 Distribution of Species Geometric Mean Chronic Effects Benchmarks From Multiple Tests of Cadmium Toxicity to Freshwater Aquatic Life



Bars are coloured to represent major taxonomic groups (invertebrates, fish, algae and macrophytes, amphibians). All endpoint data have been standardized to common hardness level of 50 mg/L calcium carbonate.

2.6.8 Chromium

Chromium (Cr) can exist in nine different oxidation forms; however, it is found most commonly in the trivalent (Cr^{3+}) and hexavalent (Cr^{6+}) states in the environment. Cr^{3+} oxidizes slowly to chromium Cr^{6+}, although Cr^{6+} is more soluble (U.S. EPA 1984). As such, Cr^{3+} dominates in reducing environments such as sediments and wetlands, whereas Cr^{6+} is the primary species found in surface water and aerobic soils (CCME 1999b). Cr^{6+} is more toxic to aquatic life than Cr^{3+}, and thus is typically addressed separately in water quality guidelines (e.g., CCME 1999b). Cr^{3+} is more toxic in soft water than in hard water, whereas hardness does not affect toxicity of Cr^{6+} (U.S. EPA 1984). Given the different toxicity profiles, separate CEBs were developed for Cr^{3+} and Cr^{6+}.

Sufficient data were available to develop an SSD for Cr^{6+}. The logistic model provided a good fit to the data ($r^2 = 0.97$), and followed the form:

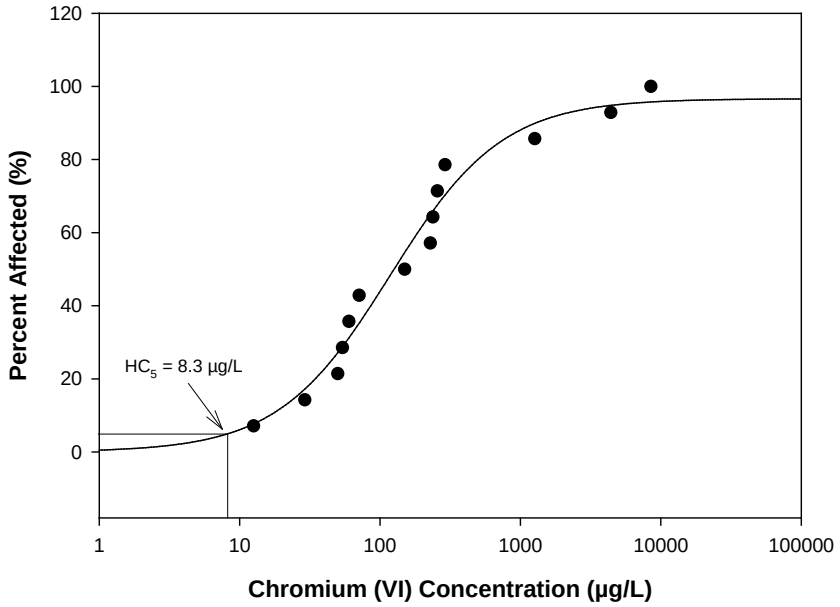
$$y = \frac{96.6}{1 + \left(\frac{x}{118.1}\right)^{-1.09}}$$

where: y = percent of aquatic community affected; and

x = chromium (Cr^{6+}) concentration (µg/L).

The resulting HC_5 based on the logistic regression model was 8.3 µg/L (Figure 2.6-4). The SSD was derived using chronic toxicity data for 14 aquatic species.

Figure 2.6-4 Species Sensitivity Distribution Curve for Hexavalent Chromium



Insufficient chronic toxicity data were available for chromium Cr^{3+} to develop an SSD. The lowest reported toxicity value of 89 µg/L was therefore selected for use as the CEB for Cr^{3+}. This value is based on a LOEC for survival of rainbow trout embryos exposed until 30-day post-swim-up (Stevens and Chapman 1984). This toxicity value is associated with soft water (i.e., hardness of 25 mg/L as $CaCO_3$) and therefore would be a conservative estimate of the toxicity threshold for PRM.

2.6.9 Cobalt

Cobalt can exist in six oxidation states; however, the most common states in the aquatic environment are cobalt (III) and cobalt (II), which form numerous organic and inorganic salts. Like most metals, the solubility of cobalt is highly dependent on its form. While cobaltous carbonate is highly insoluble in water, several salts, such as cobalt chloride ($CoCl_2$), are highly soluble. Cobalt is essential in trace amounts, and it forms part of the vitamin B-12.

The British Columbia Ministry of Water, Land and Air Protection (BC MWLAP) recently evaluated the toxicological literature for cobalt (BC MWLAP 2004), and determined that invertebrates are more sensitive than fish to cobalt exposure. These data, although insufficient to develop an SSD-based threshold, were considered adequate to develop a benchmark based on the most sensitive chronic freshwater endpoints (freshwater crustaceans). Thus, the chronic toxicity data reported by BC MWLAP (2004) for species relevant to PRM area were applied to define the CEB for cobalt, using the geometric mean of *Daphnia* and *Ceriodaphnia* endpoints. The derivation also included an application factor of 0.5 to account for the difference between an effect and no-effect level; this value was justified by BC MWLAP (2004) on the basis of experimental observations and the essential nutrient status of cobalt. The resulting CEB was set to 4 µg/L. This value is close to the reproductive NOEC test result generated using the water flea *Daphnia magna* (Kimball 1978).

2.6.10 Copper

In natural waters, copper (Cu) occurs primarily as the divalent cupric ion in free and complex forms. The cupric ion (Cu^{2+}) is the most readily available (Suedel et al. 1996), and is highly reactive, forming complexes and precipitates with organic and inorganic constituents and suspended solids in the water column (U.S. EPA 1985). Copper can be toxic to aquatic life, but at low concentrations it is an essential nutrient for both aquatic plants and animals (U.S. EPA 1985).

Water quality can also affect the toxicity and bioavailability of copper to aquatic life. Generally, as water hardness increases, toxicity decreases. Water hardness in natural waters is controlled by the presence of calcium and magnesium, which compete with metal cations for binding sites on the gills of aquatic organisms (ICME 1995).

2.6.10.1 Background to Copper Water Quality Guidelines

A default screening concentration (conservative water quality guideline) of 2 µg/L is available from CCME (2011) for protection of aquatic life, but this value is substantially overprotective for PRM.

A hardness-adjusted value can be derived from the following formula:

- copper chronic effect guideline in µg/L = $e^{(0.8545[\ln Hardness]-1.465)} \times 0.2$

For a site-specific water hardness of 143 to 149 mg/L $CaCO_3$, the calculated CCME copper guideline would be 3.21 to 3.32 µg/L Cu. Even when adjusted for hardness, this guideline is considered overprotective. Like the CCME, the U.S. EPA (2007b) has developed hardness adjusted benchmarks for copper. Specifically, except possibly where a locally important species is very sensitive, freshwater aquatic organisms should be protected if:

- the four-day average concentration of copper does not exceed the numerical value (in µg/L) given by the equation:

  a. Criterion Continuous Concentration (CCC) = $e^{(0.8545\ [\ln(\text{Hardness})]\ -1.702)} \times 0.960$
  more than once every three years on the average

- the one-hour average concentration does not exceed the numerical value (in µg/L) given by the equation:

  b. Criterion Maximum Concentration (CMC) = $e^{(0.9422\ [\ln(\text{Hardness})]\ -1.700)} \times 0.960$
  more than once every three years on the average

For a water hardness of 150 mg/L $CaCO_3$, reflective of PRM LSA watercourses, the calculated hardness-based U.S. EPA copper guideline (CCC) is 13 µg/L Cu.

The above U.S. EPA aquatic life benchmarks for metals address the reported effects of hardness on copper toxicity using empirical regressions of toxic concentrations versus hardness for available toxicity data across a wide range of hardness values (Stephan et al. 1985). However, these regressions have certain limitations. Most notably, these regressions apply best to waters in which the correlations among hardness, pH and alkalinity are similar to the data used in the regressions. The separate effects of these factors are not addressed for exposure conditions in which these correlations are different. In addition, some physicochemical factors affecting metal toxicity, such as organic carbon, are not addressed at all.

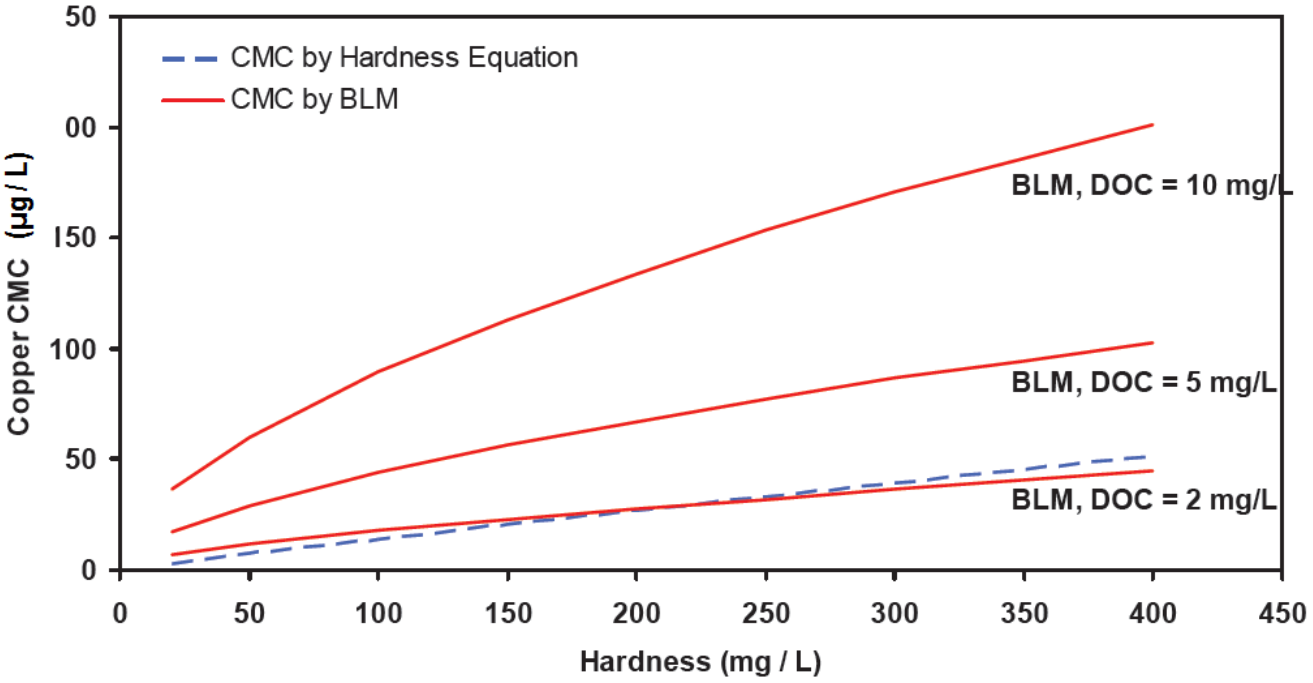
Because copper toxicity has been reported to vary markedly because of various physicochemical characteristics of the exposure water, a simple hardness-based derivation is not appropriate. Some of the physicochemical factors that need to be considered are temperature, dissolved organic compounds, suspended particles, pH and various inorganic cations and anions, including those composing hardness and alkalinity. These factors influence the bioavailability of copper with hard water, turbid water and circumneutral pH having lower bioavailability relative to soft, clear, acidic water.

2.6.10.2 The Biotic Ligand Model

The Biotic Ligand Model (BLM) provides a means to account for the effect of water chemistry, in addition to hardness, on metal bioavailability and toxicity (i.e., hardness, pH and DOC are used for the BLM). In this manner, one of the principal weaknesses of hardness-based guidelines can be resolved. The U.S. EPA (2007b) has developed the BLM procedure for copper, and the updated freshwater benchmark derivations using the BLM are based on the general guidelines set forth in Stephan et al. (1985).

One of the more sensitive constituents in the BLM is DOC in the water. As shown in Figure 2.6-5 (from U.S. EPA 2007b), the toxicity of copper decreases significantly when DOC rises to 10 mg/L and beyond. Furthermore, the divergence between the BLM and the hardness-based equation increases for high DOC concentrations.

Figure 2.6-5 Criterion Maximum Concentration Calculated Using Hardness Equation and Biotic Ligand Model



Source: (U.S. EPA 2007b).

Full specification of the BLM requires parameterization of numerous water quality constituents, including alkalinity, ion concentrations, sulphides, and other constituents. However, U.S. EPA (2007b) has provided a simplified version of the BLM for copper, in which benchmarks can be estimated from approximate values of three driving constituents, specifically pH, hardness and DOC. The simplified BLM calculations provided in U.S. EPA (2007b) assume that alkalinity was correlated with pH, and that other major ions are correlated with hardness based on observed correlations in U.S. EPA synthetic water recipes.

The output from the copper BLM (U.S. EPA 2007b, Appendix G), assuming alkalinity and pH are proportional to hardness is excerpted in Table 2.6-4. The pH of 7.5 is considered representative of the receiving environment; typical hardness (149 mg/L $CaCO_3$ and DOC 22 mg/L or greater) apply to the streams to which the CEBs are being applied. Representative water quality benchmark values using the BLM and the hardness equation assessment methods are listed in Table 2.6-4. The benchmarks are considered equivalent to the CMC values because they are based on short-term effects, rather than continuous chronic exposure.

Given the site-specific water quality conditions for PRM, the most applicable CMC from Table 2.6-4 is 82.4 µg/L Cu (BLM), which is about four times greater than the hardness-based CMC of 21.7 µg/L Cu. This CMC again shows the strong influence of DOC on copper toxicity.

Table 2.6-4 Criterion Maximum Concentration Calculated Using Hardness Equation and Biotic Ligand Model

pH	Hardness [mg/L $CaCO_3$]	Dissolved Organic Carbon [mg/L]	Hardness Equation Based Water Quality Criterion for Copper[(a)] [µg/L]	BLM Based Instantaneous Water Quality Criterion for Copper [µg/L]
75	40	2	5.9	7.9
		4	5.9	15.8
		8	5.9	32.4
		16	5.9	67.3
	80	2	11.3	8.7
		4	11.3	17.4
		8	11.3	35.3
		16	11.3	72.5
	159	2	21.7	10.1
		4	21.7	20.1
		8	21.7	40.5
		16	21.7	82.4
	317	2	41.5	12.0
		4	41.5	23.9
		8	41.5	47.8
		16	41.5	96.8

(a) CMC calculated using hardness equation and BLM for subset of pH, hardness and DOC conditions relevant to PRM (U.S. EPA 2007b).

2.6.10.3 Derivation of Chronic Effects Benchmark for Copper

To maintain the degree of environmental protection inherent in a true CEB, it is necessary to convert the above benchmark from a CMC to a CCC. Unfortunately, U.S. EPA (2007b) could not be used directly to derive a chronic copper BLM (i.e., BLM-based CCC) because of data limitations. The minimum eight family data requirements for chronic toxicity data were not met to calculate the final chronic value by the fifth percentile method. Instead, an Acute to Chronic Ratio (ACR) approach was recommended.

This approach assumes that the acute BLM reasonably approximates the bioavailability relationships for chronic toxicity. The U.S. EPA (2007b) documented limited data available regarding effects of water chemistry on sublethal effects and chronic lethality for copper, and noted similar effects of organic matter, alkalinity, pH, hardness and ions to the acute BLM. Therefore, extrapolation of the BLM approach through use of an ACR is considered acceptable.

Following the U.S. EPA's guidance for guideline derivation, the final ACR for copper of 3.22 was calculated as the geometric mean of the ACRs for sensitive freshwater species, *Ceriodaphnia dubia*, *Daphnia magna*, *D. pulex*, *Oncorhynchus tshawytscha*, and *O. mykiss* along with the one saltwater ACR for *C. variegatus* (U.S. EPA 2007b). Based on the normalization to water chemistry conditions specified for PRM, the freshwater site-specific acute guideline is 82.4 µg/L which, divided by the final ACR of 3.22, results in a freshwater CEB of 25.6 µg/L Cu.

The generic CCME copper guideline and U.S. EPA's earlier freshwater copper benchmark recommendations are hardness-dependent values. These default guidelines, although conservative, are expected to substantially overstate copper toxicity for the stream conditions anticipated for PRM. As concluded by U.S. EPA (2007b), a

BLM-based benchmark calculation procedure is expected to yield a more appropriate benchmark because it accounts for the important water chemistry factors that affect toxicity, including DOC complexation, where the hardness-only correction does not.

A freshwater CEB of 26 µg/L Cu is recommended based on the BLM approach. This value is still considered conservative given that the input DOC of 16 mg/L is lower than the estimated value of 22 mg/L for the receiving waters (PRM LSA).

2.6.11 Iron

In the aquatic environment, iron (Fe) exists in two primary forms: soluble ferrous (Fe^{2+}) iron and insoluble ferric (Fe^{3+}) iron. Oxidation-reduction reactions determine the chemical behaviour of iron in the aquatic environment. In aerobic systems, the vast majority of iron is present in water as insoluble ferric ion, which is largely non-toxic. Under anaerobic conditions, soluble ferrous iron can form. The oxidation of Fe^{2+} (the more toxic species) to insoluble Fe^{3+} oxyhydroxides is influenced by pH, redox potential, dissolved oxygen, the amount and type of dissolved organic matter, and other environmental factors, with acidic, non-humic rivers generally having the highest proportion of iron present in the dissolved form (Vuori 1995). Whereas dissolved iron has the potential to cause toxicity in the traditional sense (i.e., uptake of sufficient dose to cause an adverse effect via the site of toxic action), insoluble iron present in a suspended particulate state can cause adverse effects via physical mechanisms such as smothering of sensitive bottom habitats or reduced visibility. Measurements of total iron in water include both the dissolved and particulate phase in proportions determined by the physical-chemical properties outlined above.

The CCME WQG for total iron (0.3 mg/L) was published by CCREM (1987) based on a benchmark developed by the International Joint Commission and the Ontario Ministry of the Environment. Although details of the derivation methods were unclear, the value is based on the concentration of iron in water that could result in precipitation of iron hydroxides on stream substrates and potentially smother habitat, rather than toxicological responses. Accordingly, this WQG would be protective of all potential exposure conditions, but would be over-protective for most conditions.

The first step in CEB derivation for iron was to consider the speciation(s) of iron relevant to the PRM receiving environment. Based on the oxygenated environment in the receiving waterbodies, iron is expected to exist predominantly as insoluble ferric iron rather than the dissolved iron used in most laboratory toxicity tests. Although periodic anoxia has been observed in the LSA of both PRM, dissolved oxygen levels tend to be high, and iron exists predominantly as insoluble ferric iron. In most field situations, however, the effect of iron as a toxicant is through a physical effect associated with the settling of iron precipitates (e.g., ferric hydroxide). Because the circumneutral conditions in watercourses in the LSA are not amenable to production of significant concentrations of stable Fe^{2+}, the iron screening guideline described above is highly conservative.

It is challenging to obtain meaningful toxicity data for iron, and particularly for ferric iron. Difficulties have been encountered in conducting toxicity tests for iron, due to the decrease in pH that results from adding ferric chloride to test solutions (Phippen et al. 2008). To quantify dissolved iron toxicity, tests need to be conducted at a low enough pH to retain iron in the dissolved state, while also not too low to cause pH-driven toxicity. However, these conditions are often not representative of most receiving environments, where much of the iron is present in the particulate form. Consequently, laboratory toxicity data for iron are conservative compared to real-world conditions.

Based on the unique properties of iron, CEB derivation is more complex relative to other metals. Therefore, to develop a CEB for ferric iron, three primary lines of evidence were applied:

- Toxicity-based SSD - a compilation of ferric iron toxicity data was evaluated, using similar procedures as for the other constituents. Adverse effects in these experiments are likely to be strongly influenced by physical effects of iron, as precipitates were observed during the exposures.
- Supporting toxicology research by the BC Ministry of Environment (Phippen et al. 2008).
- Field Studies - a review of North American field studies was conducted, considering sites where iron was the dominant contaminant of concern and where relationships between iron concentrations and ecological responses were documented.

2.6.11.1 Toxicity-Based Species Sensitivity Distribution

From the literature, data were available for a wide range of species. Overall, chronic toxicity data for 10 species were retained for the SSD (Table 2.6-5). These included: three fish species (two salmonids and one non-salmonid); five invertebrate species (including two crustaceans); one alga; and one aquatic macrophyte (duckweed). CCME (2007a) recommends the inclusion of a stonefly, mayfly, or caddisfly species in the SSD, and multiple endpoint data for the mayfly *Leptophlebia marginataone* were available for this purpose. Overall, the data met the requirements of Type A derivations following the CCME (2007a) protocol.

Table 2.6-5 Available Chronic Freshwater Toxicity Data for Ferric Iron

Test Species	Common Name	Species Mean Chronic Value [mg/L]	Rank	% Affected
Pimephales promelas	fathead minnow	1.5	1	5
Daphnia longispina	water flea	1.7	2	15
Oncorhynchus kisutch	coho salmon	3.1	3	25
Lemna minor	duckweed	3.7	4	35
Daphnia magna	water flea	4.4	5	45
Chlorella vulgaris	green algae	4.5	6	55
Leptophlebia marginata	mayfly	8.2	7	65
Salvelinus fontinalis	brook trout	9.0	8	75
Tubifex tubifex	annelid worm	17.8	9	85
Asellus aquaticus	isopod	124.0	10	95

The chronic data summarized in Table 2.6-5 were used to generate an SSD for ferric iron. The logistic model provided a good fit to the data (r^2 = 0.98), and followed the form:

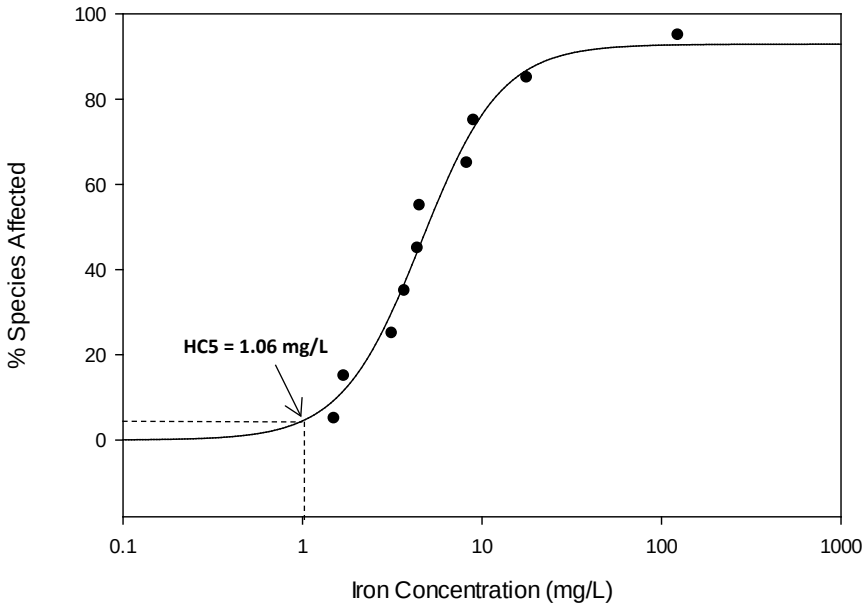
$$y = \frac{92.9}{1 + \left(\frac{x}{4.58}\right)^{-1.96}}$$

where: y = percent of aquatic community affected; and

x = total iron concentration (mg/L), assumed to be 100% ferric iron.

The resulting HC_5 was 1.06 mg Fe/L (Figure 2.6-6). Visual inspection of Figure 2.6-6 confirms a good model fit, including the lower tail of the distribution that sets the HC_5. The calculated HC_5 is sufficiently low to protect the most sensitive species (fathead minnow).

Figure 2.6-6 Species Sensitivity Distribution Curve for Ferric Iron



2.6.11.2 British Columbia Ministry of Environment Testing

In 1997, a suite of toxicity tests was conducted by the British Columbia Ministry of Environment (Phippen et al. 2008) to address uncertainties in the toxicological literature with respect to iron. These test results have been summarized separately from the studies used to develop the SSD because some of the testing attributes are not amenable to SSD evaluation, including high frequency of unbounded effect concentrations, incorporation of acute test endpoints, and lack of detail documented for some endpoint data.

Several freshwater toxicity tests were conducted, including the following biota: rainbow trout, amphipod *Hyalella azteca*, midge *Chironomus tentans*, crustacean *Daphnia magna*, and Microtox bioluminescence. Rainbow trout testing involved both standard assessment of juvenile survival and early life stage development tests using newly fertilized eggs. Tests were conducted over multiple hardness concentrations. Tests were conducted through administration of ferric chloride (Fe^{3+}) and tests were maintained at a minimum pH level of 5.0 to avoid major confounding toxicity attributable to pH effects (although differences attribute to pH drift in the range of 5.0 to 7.0 could have been present). This constraint limited the concentration of ferric chloride that could be administered. In tests with soft water, the LC_{50} values for all species exceeded the maximum concentration of iron that could be delivered to the organisms (5.2 mg/L to 7 mg/L). The exception was for *Hyalella azteca*, which exhibited an LC_{50} of 3.5 mg/L.

Separate tests of well water with hardness of approximately 100 mg/L $CaCO_3$ were conducted, which are more representative of hardness conditions in the PRM LSA. These tests generally confirmed the soft water results, but also included testing with the alga *Selanastrum capricornutum*, and incorporated chronic testing with *Daphnia magna*. The MATC (geometric mean of NOEC and LOEC) from the chronic Daphnia test was 7.5 mg/L, and the alga *Selanastrum capricornutum* yielded an effect benchmark of 3.6 mg/L (Phippen et al. 2008). These results provided supporting information on the range of toxicity observed in controlled laboratory experiments. Iron precipitates were observed at the bottom of the test vessels during the experiments, such that observed effects may reflect physical effects of ferric precipitates.

2.6.11.3 Field Study Results

Adverse effects from iron can occur in the field due to settling of iron-hydroxide precipitates, which may displace organisms that are sensitive to the physical (smothering) effect of these particulates. It is difficult to simulate realistic environmental conditions of iron precipitation and environmental fate in the laboratory, prompting several investigators to directly assess the effect of iron under field conditions. Most toxicity observed in laboratory tests with iron exposures has been attributed to motion inhibiting or smothering effects of ferric hydroxides or iron-humus precipitates on gills, eggs, or other sensitive surfaces (Vuori 1995); as such, the assessment of indirect (physical) effects under realistic natural exposure environments may provide better insight into the toxicity of ferric iron.

Linton et al. (2007) conducted a bioassessment-based analysis of iron toxicity, using a quantile regression to model the decline in maximum abundance of taxa along a gradient of increasing iron concentration. The model was applied to a large volume of field data obtained by the West Virginia Department of Environmental Protection, and was based on compilations of numerous stream orders, with benthic invertebrate collections obtained from kick-net samples. The interpretation of effects was conducted through derivation of Field Effect Concentrations (FECs) for total iron associated with benchmarks for environmental impairment. The authors derived an FEC20 value of 1.8 mg/L that represents a 20% reduction in organism abundance relative to reference stations. The authors also developed a similar benchmark of 1.74 mg/L designed to protect against impairment of the overall community; this was equal to the point at which a 50% reduction in the fourth most sensitive individual taxonomic group was observed. Linton et al. (2007) also derived benchmarks for iron that align with West Virginia Stream Condition Index (SCI) target values. For an SCI value of 78, corresponding to a rating of "highly comparable to reference sites", a concentration of 1.25 mg/L total iron was required. A less protective benchmark, associated with an SCI score of 68 (comparable to "below-average reference sites") was found to be 5.3 mg/L total iron.

Brenner et al. (2004) reported the results of environmental monitoring of iron-laden discharges from mining origin in a Pennsylvania freshwater stream environment. Accumulation of iron in sediments was identified as a potential stressor to the aquatic invertebrate community, principally through the effect of settling of iron hydroxide precipitates. The pH conditions of the mine discharge and the receiving environment were slightly lower than the PRM LSA (e.g., mean pH in discharges ranged from 6.50 to 7.27). Multiple indicators of biological condition (biotic index, abundance, taxonomic richness) exhibited a strong and statistically significant relationship to total iron concentrations in the stream. Species richness and the number of organisms were reduced at total iron concentrations of 4 mg/L or greater, with effect sizes of 20% to 50% in the range of 4 to 6 mg/L Fe. The 4 mg/L Fe concentration can be interpreted as a benchmark for the site based on approximate 20% reductions to the biotic index, abundance, and taxonomic richness endpoints.

2.6.11.4 Chronic Effects Benchmark Summary

The combined results of the lines of evidence discussed above indicate strongly that the default WQG for iron (0.35 mg/L) is overprotective in the context of the PRM LSA. The WQG, which is based primarily upon adverse effects from dissolved iron (ferrous), is inapplicable to a site condition for which the vast majority of iron will occur in a ferric state.

The three lines of evidence discussed above provide different, but complementary, evidence for the establishment of a protective benchmark for ferric iron. Specifically:

- The SSD evaluation indicates that a total iron concentration of 1.1 mg/L Fe (HC_5) will be protective against both direct and indirect toxicological effects of ferric iron. This analysis assumes that the conditions in the laboratory (e.g., physical smothering) represent plausible conditions in the field.
- The supplemental toxicity evaluation conducted by Phippen et al. (2008) indicates that sensitive organisms are affected at concentrations of approximately 3.5 mg/L total iron or greater. Most organisms were unaffected at much higher concentrations (i.e., beyond maximum tested concentrations) although testing included both acute and chronic endpoints. There is also uncertainty regarding the relative influence of pH and iron concentration in these tests.
- Results of field assessments indicate benchmarks in broad agreement with toxicity-based benchmarks. The FEC20 value of 1.8 mg/L (Linton et al. 2007) and the approximate 20% inhibition for field responses of 4 mg/L (Brenner et al. 2004) both reflect slight biological alteration of benthic communities. These field studies may not be directly reflective of biological conditions in the PRM, but reflect observed relationships between ferric iron exposure and biological responses in natural communities.

Based on the above, a CEB for total iron of 1.5 mg/L is proposed; this value was derived as the average of the SSD-based benchmark (HC_5) and the lower of the two field-based IC_{20} values. The above lines of evidence suggest that physical responses are the toxicity driver for ferric iron; laboratory tests of the direct toxicity of ferrous and ferric iron on freshwater fish and invertebrates generally exceed 2 to 5 mg/L (Linton et al. 2007). Iron in the insoluble ferric state may remain in solution or precipitate to bottom sediments depending on the hydrodynamics of the receiving environment (Phippen et al. 2008). If formation of iron precipitates (ferric hydroxide) does not occur in PRM area, the above benchmark will be substantially overprotective.

2.6.12 Lead

The generic CCME (2007b) guideline for lead is 1 µg/L. However, CCME (1999a, with updates to 2011) provides a hardness-based derivation, which corresponds to 5.0 to 5.3 µg/L at a site-specific hardness range of 143 to 149 mg/L $CaCO_3$. The latter was selected as the CEB for lead, and no SSD-based derivation was necessary for PRM due to the low concentrations of lead predicted by the water quality model.

2.6.13 Manganese

Manganese (Mn) exists in oxidation states ranging from Mn^{3-} to Mn^{7+}, of which divalent and trivalent manganese are the more important forms in aquatic systems (CCREM 1987). Sufficient data were available to develop an SSD for manganese (Table 2.6-6), as there were chronic toxicity data for five species, including two fish species (one salmonid and one non-salmonid) and three invertebrate species (including two crustaceans).

The logistic model provided a good fit to the data ($r^2 = 0.94$), and followed the form:

$$y = \frac{89.9}{1 + \left(\frac{x}{4110}\right)^{-2.7}}$$

where: y = percent of aquatic community affected; and

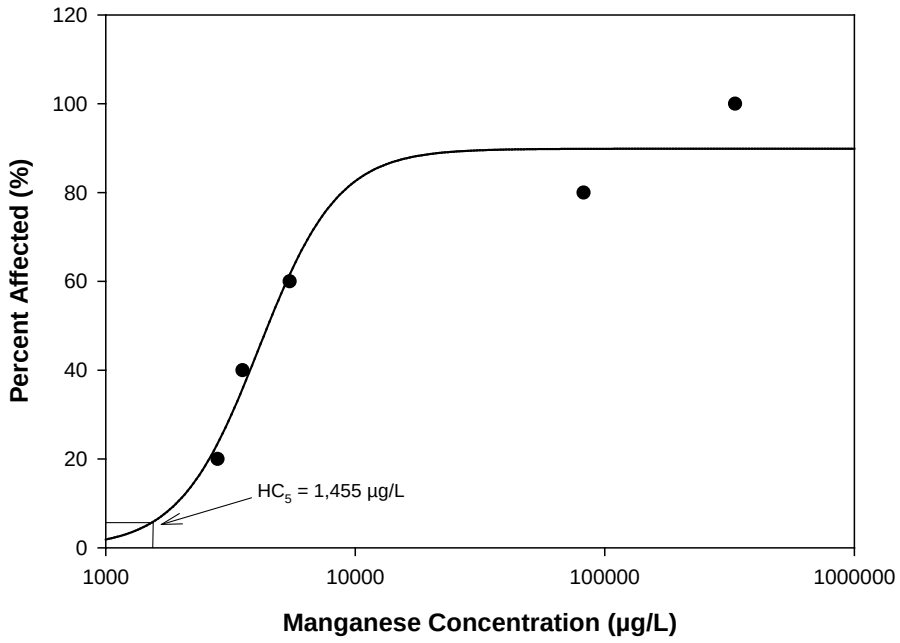
x = manganese concentration (µg/L).

Table 2.6-6 Available Chronic Freshwater Toxicity Data for Manganese

Species Scientific Name	Species Common Name	Chronic Value [µg/L]	Rank	Percent Affected [%]
Oncorhynchus mykiss	Rainbow trout	2,806	1	20%
Pimephales promelas	Fathead minnow	3,535	2	40%
Daphnia magna	Water flea	5,463	3	60%
Tubifex tubifex	Worm	46,151	4	80%
Asellus aquaticus	Isopod	333,000	5	100%

The resulting HC_5 based on the logistic regression model was 1,455 µg/L (Figure 2.6-7). This value is approximately half the species mean chronic value for the most sensitive organism (rainbow trout). The latter was derived from 28-day chronic toxicity studies (Birge 1978; Birge et al. 1979).

Figure 2.6-7 Species Sensitivity Distribution Curve for Manganese



2.6.14 Mercury

In surface waters, mercury (Hg) can exist in the mercuric (Hg^{2+}) and mercurous (Hg^{1+}) states. Mercuric mercury can form complexes with organic compounds and organic carbon and is the predominant form of mercury present in surface waters (ATSDR 1999). The presence of soluble sulphide may facilitate the removal of mercury ions from the water column by the formation of insoluble mercuric sulphide salts and hence, reducing the availability of mercury to aquatic organisms. The activity of sulphide ions decreases under acidic conditions, thus inhibiting the formation of mercuric sulphide and favouring the formation of methylmercury (Bjornberg et al. 1988). The formation of methylmercury is the most important transformation process in the environmental fate of mercury in surface waters. Any form of mercury entering surface waters can be converted by sulphur-reducing bacteria to methylmercuric ions, especially under anaerobic conditions. Abiotic reduction of inorganic mercury to metallic mercury can also occur in aqueous systems, particularly in the presence of soluble humic constituents (ATSDR 1999).

2.6.15 Aquatic Benchmark Derivation

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for mercury is provided in Table 2.6-7. The SSD was conducted using chronic toxicity data for 15 aquatic species. Sufficient toxicity data were available for mercury to develop a SSD, with a logistic regression model providing a good fit to the observed data (r^2 = 0.98) (Figure 2.6-8). The logistic model followed the form:

$$y = \frac{192.42}{1 + \left(\frac{x}{2345.9}\right)^{-0.33}}$$

Where: y = percent of aquatic species affected; and

x = mercury concentration (µg/L).

Table 2.6-7 Available Chronic Freshwater Toxicity Data for Mercury

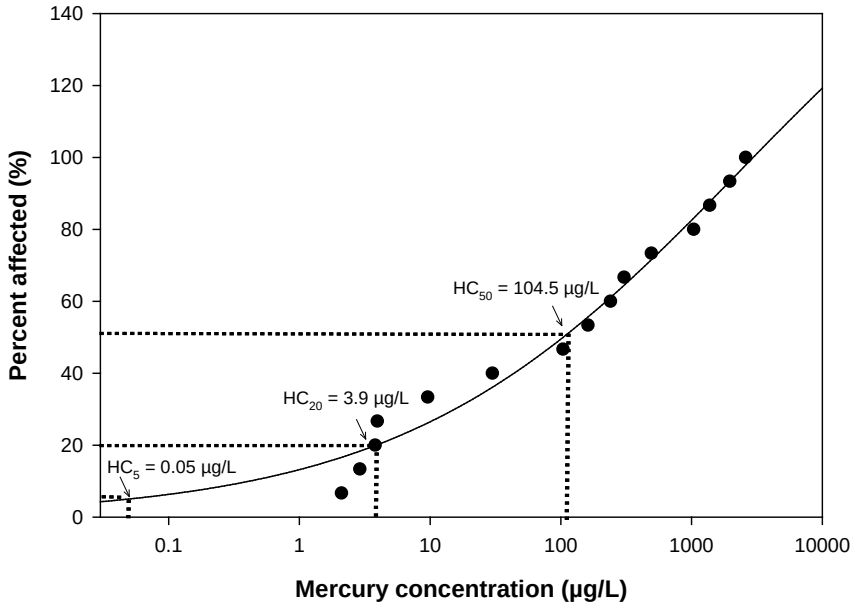
Species Scientific Name	Species Common Name	Chronic Value [µg/L]	Rank	Percent Affected [%]
Hyalella azteca	scud (amphipod)	2.1	1	6.7
Ceriodaphnia reticulata	water flea	2.9	2	13.3
Daphnia pulex	water flea	3.8	3	20.0
Oncorhynchus mykiss	rainbow trout	4.0	4	26.7
Daphnia magna	water flea	9.6	5	33.3
Gammarus sp.	amphipod	30.0	6	40.0
Tubifex tubifex	worm	104.0	7	46.7
Cypris sp.	ostracod	161.0	8	53.3
Daphnia sp.	water flea	240.0	9	60.0
Chironomus sp.	midge	305.0	10	66.7
Chironomus riparius	midge	493.0	11	73.3
Amnicola sp.	snail	1,039.0	12	80.0
Nais sp.	snail	1,378.0	13	86.7
Zygoptera	damselfly	1,960.0	14	93.3
Trichoptera	caddisfly	2,592.0	15	100.0

Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for mercury were estimated to be 0.05, 3.9 and 104.5 µg/L, respectively (Figure 2.6-8).

The mercury water quality guideline of 0.026 µg/L proposed by CCME (2003b) was derived by dividing the LOEC of 0.26 µg Hg/L reported by Snarski and Olson (1982) in juvenile fathead minnows by a safety factor of 10. The percentage of affected species calculated by plotting the proposed guideline value of 0.026 µg Hg/L against the site-specific mercury SSD curve was 4%. Therefore, the proposed CCME guideline for mercury appears to be conservative given the aquatic species and water quality conditions encountered in the study area.

The above CEB should be interpreted with caution because aqueous mercury is a relatively poor indicator of mercury toxicity. As a bioaccumulative constituent, the toxic effects of mercury are best evaluated through a comparison of aquatic organism tissues to critical tissue burdens.

Figure 2.6-8 Water-Based Species Sensitivity Distribution Curve for Mercury



2.6.15.1 Tissue Benchmark Derivation

Because mercury is a strongly bioaccumulative constituent, and biomagnifies in the aquatic food web, the CEB development for mercury was supplemented with an analysis of tissue-based effects observed in freshwater aquatic life. This procedure entailed compiling information that related the exposure of invertebrates and fish to methylmercury and total mercury (measured as a wet weight concentration of mercury in their body tissues) to adverse effects in survival, growth, and reproductive endpoints.

The unique chemical partitioning properties of mercury warranted separate derivations for invertebrates and fish. In fish tissues, mercury is found predominantly in the methylated form, with approximately 95% or more as methylmercury (Wiener and Spry 1996). For this reason, it is common practice to assume that total mercury and methylmercury in fish tissue are equivalent for the purposes of deriving concentration-response relationships. In contrast, the proportion of methylmercury in invertebrate tissues is more variable, with methylmercury usually predominating, but sometimes dropping below 30% of the total mercury present. In general, the proportion of mercury found in organic forms increases with progression up the food web.

The procedure used to develop threshold mercury body residues for mercury entailed the following:

- compilation of literature studies that linked the concentrations of mercury in freshwater organisms to chronic lethal and sublethal response endpoints;
- conversion of response data to a normalized percent effect by scaling exposure responses to the background/control response and expressing results as a percentage impairment for the specified endpoint;
- examining data for systematic differences in sensitivity for certain endpoint types or groups of taxa;

- developing a threshold tissue concentration that is protective of both fish and invertebrates, through separate analyses of the statistical fit to the most sensitive endpoint(s) for each biota type; and
- comparison of derived values to ecological protection thresholds proposed by other authors.

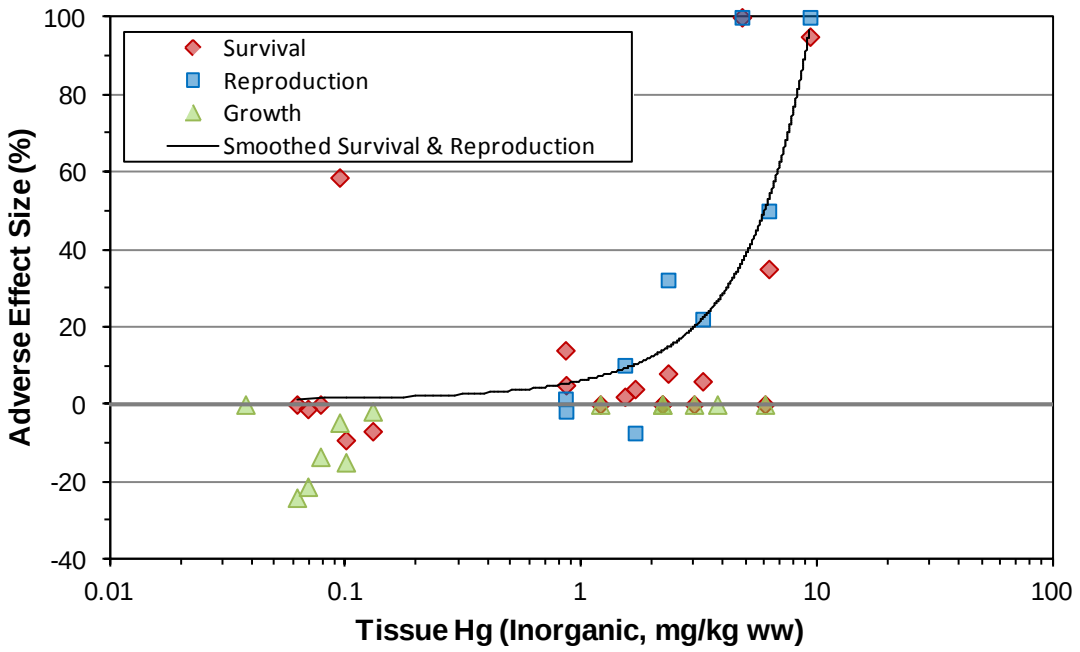
The data sources included studies that were identified in numerous compendia of tissue effect thresholds (Beckvar et al. 2005; EVS 1999; Jarvinen and Ankley 1999; Sandheinrich and Wiener 2011; Wiener and Spry 1996) and were supported by the results of an online literature review.

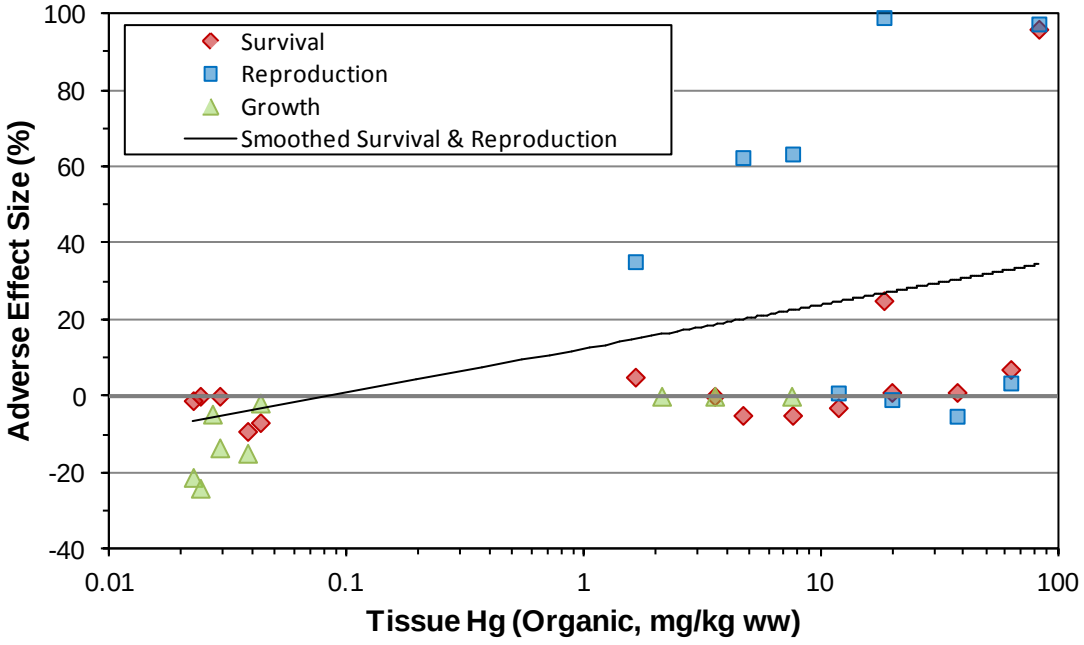
Mercury Effects in Invertebrates

The concentration-response relationship for invertebrate experiments where the tissue mercury burden was measured in inorganic (upper pane) and organic (lower pane) forms is presented in Figure 2.6-9. The data represent responses to multiple test species including mussels, snails, mayflies and daphnids. For both inorganic and organic mercury, no significant responses were observed for the growth endpoint. However, survival and reproduction were adversely affected at high tissue concentrations of mercury. The data vary substantially, reflecting the range of experimental designs and test species, and negative effect levels were observed in some treatments (i.e., indication of a positive response to exposure, likely attributable to natural variability).

Despite the variability in individual results, a smoothed model was fit to the combined survival and reproduction data for the concentration-response data for both in-organic and organic mercury. Whereas the onset of toxic responses appears to occur in the low mg/kg range for both forms of mercury, the steepness of the concentration-response was greater for the inorganic mercury model, with very large responses observed near 10 mg/kg Hg. Based on the overall response profile shown in Figure 2.6-9, a threshold mercury concentration of 3 mg/kg ww total mercury, or 2 mg/kg ww methylmercury, is proposed as a tissue-based CEB for PRM. These concentrations were chosen based on the estimate of the IC_{20} from the combined data for the most sensitive endpoints (reproduction and survival).

Figure 2.6-9 Concentration-Response for Mercury in Freshwater Invertebrate Tissues





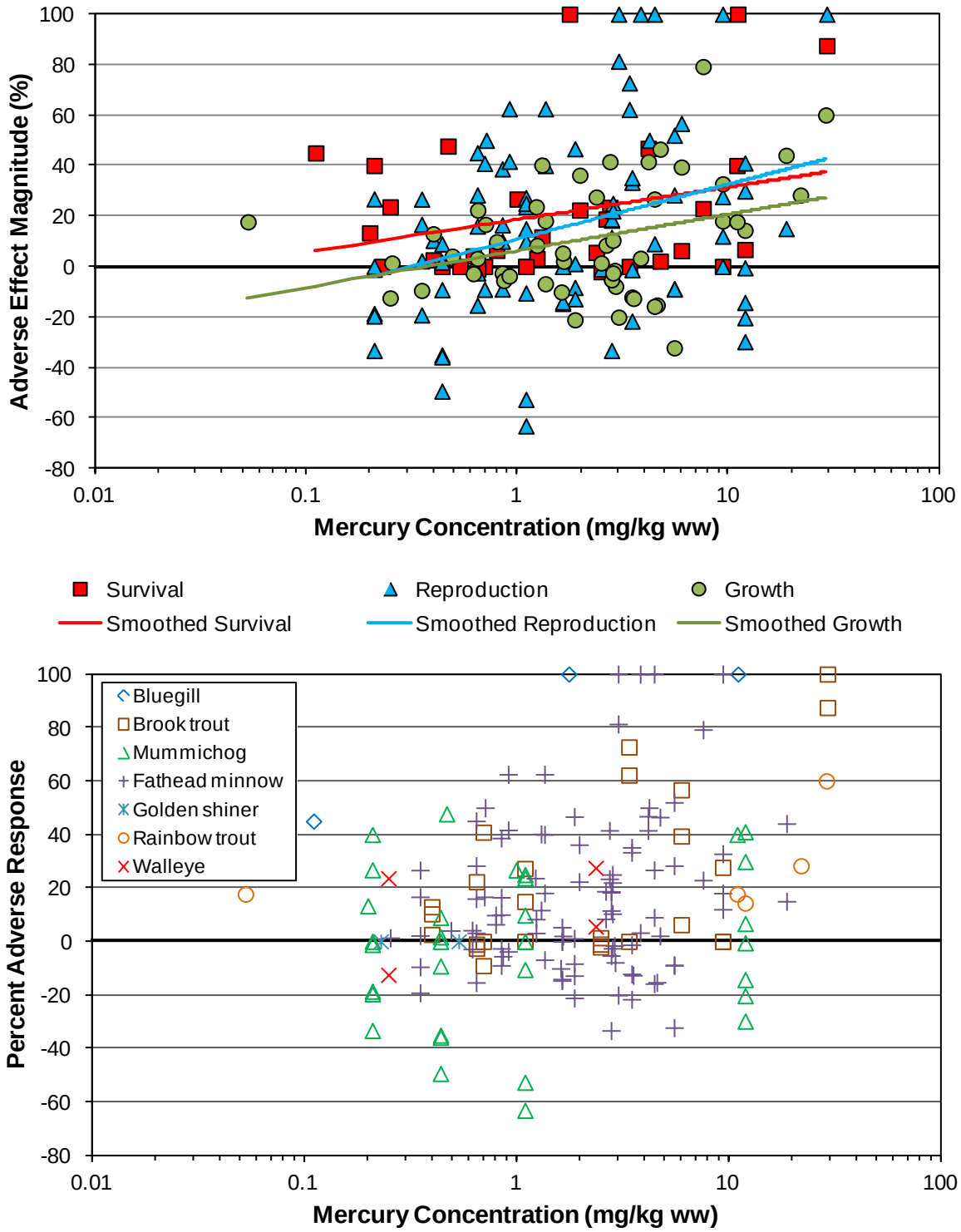
Mercury Effects in Fish

A scatterplot of the tissue effects data for freshwater fish, subdivided into the three main endpoint types (survival, growth and reproduction) (Figure 2.6-10, upper pane). Unlike invertebrates, there were no major differences in the sensitivity of these three endpoints, with the data highly overlapping and similar model fit (using a logarithmic regression for all endpoints). Again, there is substantial variability in individual data points, reflecting the range of experimental designs and test species, along with natural variability within experiments. However, an increase in response rate with increasing mercury concentration is evident for all three endpoints. Furthermore, the sensitivity of fish to mercury is greater than for invertebrates. At the concentration of 3 mg/kg used for invertebrates, numerous studies have indicated large adverse responses, and the mean (modelled) response for survival and reproduction endpoints exceeds 20%. Although development of a precise threshold is confounded by the high variability, it appears that the magnitude and frequency of adverse responses increases at concentrations close to 1.0 mg/kg total mercury.

Using an IC_{20} assessment method applied to the most sensitive endpoint, a concentration of 1.0 mg/kg total mercury was identified. However, the uncertainty associated with the data variability may warrant additional conservatism. Accordingly, a fish tissue CEB of 0.5 mg/kg was selected, which represents a lower level of mean response for survival, and a mean response close to zero for growth and reproduction.

As an additional check on the acceptability of the fish tissue CEB of 0.5 mg/kg, the data were plotted on the basis of species (Figure 2.6-10, lower pane). Mummichog appear to be insensitive to mercury exposure, with no meaningful increase in the incidence of adverse effects across the range of exposures tested. The remaining species (with sufficient data for comparison) suggest similar sensitivity to mercury exposure.

Figure 2.6-10 Concentration-Response for Mercury in Freshwater Fish Tissues



Comparison to Other Reviews

Because the CEB for the protection of fish is more sensitive than for protection of invertebrates, and because fish tissue concentrations tend to be higher (due to biomagnification), the fish CEB is likely to be the driver for overall environmental protection. The following authors have developed tissue-based thresholds for protection of fish; therefore their results were compared with the CEB based on evaluation of concentration-response data:

- Beckvar et al. (2005) – The authors applied a systematic method for deriving protective (i.e., unlikely to have adverse effects) tissue residue-effect concentrations in fish using decision rules that were formulated to provide guidance on selecting studies and obtaining data in a consistent manner. Paired no-effect and low-effect whole-body residue concentrations in fish were identified for mercury and four analytical approaches of simple ranking, empirical percentile, tissue Threshold-Effect Level (t-TEL), and cumulative distribution function were explored. A whole-body mercury t-TEL of 0.2 mg/kg ww was derived, based largely on sublethal endpoints (growth, reproduction, development, behaviour), and was determined to be protective of juvenile and adult fish. The difference between the t-TEL of 0.2 mg/kg ww and the CEB of 0.5 mg/kg ww is partly attributable to the inclusion by Beckvar et al. (2005) of behavioural test endpoints (such as foraging behaviour and predator avoidance) and partly to a recognized "consistent bias toward protective tissue concentrations." Importantly, the authors cautioned against using tissue residue information from only one study of a species that is closely related to the species of interest. This method supports the meta-analysis of multiple species and experimental conditions to help account for inter-experimental variability.

- Wiener and Spry (1996) – The authors summarize the information on mercury toxicity to fish available up to the mid 1990s. The review indicated that concentrations of total mercury associated with lethal and sublethal effects in freshwater fish were expected in the concentration range of 5 to 10 mg/kg ww in whole fish, with signs of overt toxicity not expected below this range. Although it was suggested that sensitive species may require a threshold in the low mg/kg range for protection against ecological impairment, there was no indication of adverse responses below 1.0 mg/kg. The authors also conducted an analysis of existing fish egg concentration studies, and concluded that while adverse effects tended to occur at lower concentrations in eggs relative to adult tissue, these differences were compensated by the lower average concentrations in eggs relative to adult body burdens. The study for PRM suggests that the threshold concentration range proposed by Wiener and Spry (1996) is too high, and this opinion is shared by other authors based on more recent scientific findings.

- Sandheinrich and Wiener (2011) – The authors summarize a range of fish toxicity studies including evidence for mercury responses associated with biochemistry, gene expression, behaviour, reproduction, histology, and growth endpoints. This compilation serves as an update to the Wiener and Spry (1996) publication, and emphasizes that recent research has led to a reassessment of the concentration threshold previously thought to be protective. The authors conclude, based on an examination of a wide array of laboratory experiments and field studies, that freshwater fish are adversely affected at tissue concentrations of methylmercury below 1.0 mg/kg ww. They conclude that changes in biochemical processes, damage to cells and tissues, and reduced reproduction can occur at methylmercury concentrations of about 0.3 to 0.7 mg/kg ww in the whole body, and about 0.5 to 1.2 mg/kg ww in axial muscle tissues. The midpoint of the whole body threshold range proposed by Sandheinrich and Wiener (2011) is equal to the CEB proposed for this assessment of 0.5 mg/kg ww. Furthermore, the lower end of the 0.3 to 0.7 mg/kg ww range appears to be associated with endpoints that have more uncertain ecological relevance. For

example, the data presented in Sandheinrich and Wiener (2011) suggest that behavioural responses occur at concentrations slightly lower than reproduction, growth, and developmental responses. Furthermore, biochemistry and gene expression responses can occur at low concentrations, but these responses are indicators of environmental exposure that do not necessarily translate into ecologically significant effects. Given the differences in the choice of endpoints used in this study versus Sandheinrich and Wiener (2011), the findings are in very strong agreement.

- EVS Solutions Inc. (EVS 1999) – Eleven studies, focusing on laboratory experiments that dosed with either methylmercury or mercuric chloride in food or water, were reviewed to identify the range of mercury concentrations associated with adverse effects on survival, growth, and reproductive success in fish. Similar to this study, the authors found that the effects and no-effects concentrations were overlapping. No effects concentrations for sublethal test endpoints ranged from 0.25 mg/kg ww in juvenile walleye to 16 mg/kg ww in Japanese medaka eggs. The most common endpoints assessed were growth, survival, and hatching and spawning success, and larval growth and survival appeared to be the most sensitive endpoints. EVS (1999) did not provide a point estimate of the threshold tissue residue-effect concentration; however, their results were in general agreement with the overall findings in this report.

Overall, the results in this report appear to be in alignment with the work of others, particularly recent work investigating sublethal chronic responses of fish to mercury. On this basis the proposed 0.5 mg/kg ww threshold for whole body fish tissue concentrations was retained as a protective CEB for PRM.

2.6.16 Molybdenum

Molybdenum (Mo) is widely distributed in the natural environment and occurs primarily as molybdenite (MoS_2) and molybdate (MoO_4^{2-}) (CCREM 1987). Molybdenum enters the aquatic environment naturally through weathering of igneous and sedimentary rocks. The largest anthropogenic source is through the use of molybdenum-containing fertilizer; however other sources include mining and milling of molybdenum, use of molybdenum products, the mining and milling of uranium and copper ores, and the burning of fossil fuels.

Molybdenum occurs in several oxidation states including Mo^{2+}, Mo^{3+}, Mo^{4+}, Mo^{5+} and Mo^{6+}. The tetravalent and hexavalent states are the most common in nature. Dissolved molybdenum occurs mainly as molybdenum sulphide (MoS_2), molybdate (MoO_4^{2-}) and bimolybdate ($HMoO_4^-$). At a pH greater than 7 the molybdate anion is the predominant form, whereas at pH less than 7 polymeric species form (CCME 1999a). Water solubility is largely dependent on pH with molybdenum remaining in solution at pH greater than 5, and forming complexes with iron and aluminum at pH less than 5. Molybdenum bioaccumulates in aquatic organisms such as algae, vascular plants, and soft tissues of fish (CCREM 1987).

The current Canadian Water Quality Guideline promulgated by CCME (1999a, updates to 2011) for the protection of freshwater aquatic life for molybdenum is 73 µg/L. It was derived by multiplying the lowest chronic toxicity value, a 28-day LC_{50} of 0.73 mg/L for rainbow trout (Birge 1978; Birge et al. 1979, 1980a) by a safety factor of 0.1 (CCME 1999a).

A summary of the toxicity database used to derive a CEB for molybdenum is provided in Table 2.6-8.

Table 2.6-8 Available Chronic Freshwater Toxicity Data for Molybdenum

Species Scientific Name	Species Common Name	Chronic Value [mg/L]	Rank	Percent Affected [%]
Onchorhynchus mykiss	rainbow trout	43	1	13
Ceriodaphnia dubia	water flea	51	2	25
Pimephales promelas	fathead minnow	60	3	38
Pseudokirchneriella subcapitata	green algae	74	4	50
Daphnia magna	water flea	89	5	63
Chironomus riparius	midge	121	6	75
Lymnaea stagnalis	great pond snail	211	7	88
Lemna minor	duckweed	242	8	100

The SSD was developed using chronic toxicity data for eight freshwater species. Importantly, the Birge (1978) and Birge et al. (1979, 1980a) data were categorized as unacceptable based on the protocol for guideline derivation provided by the CCME (2007a). These Birge studies have been identified to have data reliability issues by several authors, including De Schamphelaere et al.'s (2010) evaluation of molybdenum toxicity data, which provides a detailed discussion of quality assessment and screening. In brief, the test assessment methods were poorly described, the molybdenum salt that was used in the testing was not reported, molybdenum concentrations were measured but not reported, physiochemical parameters were measured but not reported, and survival of controls was not reported. As well, there is a discrepancy between the results of these studies and the majority of the literature which demonstrate low toxicity of molybdenum to fish. Similar discrepancies have been observed for other trace contaminants evaluated by Birge (1978) and Birge et al. (1979, 1980a), and data from these studies have been eliminated as unreliable on this basis.

In 2005, Davies et al. (2005) replicated the experiments of Birge (1978) and Birge et al. (1979, 1980a) in an attempt to resolve the discrepancy described above, using established protocols for chronic toxicity testing. The reproduced tests could not replicate the high molybdenum toxicity reported by Birge (1978) or Birge et al. (1979, 1980a), and the results were comparable to other studies reporting low toxicity of molybdenum to fish. For these reasons, as well as those noted above, the Birge (1978) and Birge et al. (1979, 1980a) data were not included in the SSD.

Until recently, the data availability for molybdenum toxicity was poor, such that reliable SSD derivations could not be performed. Recognizing these limitations, the International Molybdenum Association sponsored a suite of chronic toxicity tests using multiple freshwater species, with the express purpose of developing an SSD. De Schamphelaere et al. (2010) presents the results of these ecotoxicity experiments, which were conducted to support the derivation of a freshwater probable-no-effect-concentration for molybdenum using the European SSD framework. Ten species were tested, including fish, amphibians, aquatic invertebrates, aquatic plants and algae. These data, as well as the data of GEI (2009) were primarily used to develop the SSD for molybdenum. The SSD analysis in this report differs slightly from that of De Schamphelaere et al. (2010) due to subtle differences in the derivation procedure (CCME versus Europe), and due to the inclusion of additional data from a literature review.

The chronic effect levels provided by De Schamphelaere et al. (2010) represent dissolved molybdenum concentrations, and therefore represent underestimates of the total molybdenum concentrations modelled for PRM. However, both total and dissolved concentrations were measured in the study and dissolved concentrations were generally within 10% of total concentrations. Because these differences were small in magnitude, the chronic effect levels derived for dissolved molybdenum in the De Schamphelaere et al. (2010) study were conservatively applied to total molybdenum.

Sufficient toxicity data were available for molybdenum to develop an SSD with the exception that reliable data were only available for two fish species (rainbow trout and fathead minnow), and the CCME requires three fish species (CCME 2007a). Data are available for other fish species, including coho salmon, white sucker and northern pike; however, the coho salmon is not native to Alberta and the data available for the latter species represent unbounded toxicity values. These data were not included in the SSD, although qualitative consideration of the results supports the conclusion that freshwater molybdenum toxicity is overstated by the current Canadian Water Quality Guideline (i.e., no effects were observed at concentrations greater than the guideline).

A logarithmic regression model provided a good fit to the data (r^2= 0.99) (Figure 2.6-10). The logarithm model followed the form:

$$y = y_o + a \ln x + b(\ln x)^2$$

Where: y = percent of aquatic species affected;

y_o = -536.7646;

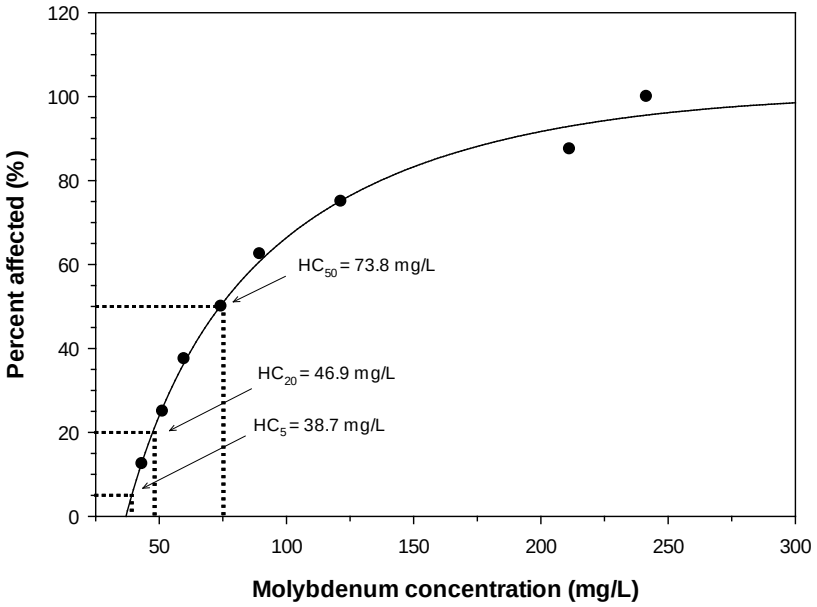
a = 213.1047;

b = -17.8343; and

x = molybdenum concentration (mg/L).

Based on the logarithm regression model, the HC_5, HC_{20} and HC_{50} for molybdenum were estimated to be 38.7, 46.9 and 73.8 mg/L, respectively (Figure 2.6-11). The HC_5 value corresponds well with the $HC_{5.5}$ value developed by De Schamphelaere (2010) of 38.2 mg/L. The HC_5 value of 38.7 mg/L is proposed as the CEB for molybdenum.

Figure 2.6-11 Species Sensitivity Distribution Curve for Molybdenum



2.6.17 Nickel

Nickel (Ni) is widely distributed in the natural environment. This element can exist in five oxidation states (Ni^{1-}, Ni^{0}, Ni^{2+}, Ni^{3+}, and Ni^{4+}); however, the most common form in the aquatic environment is nickel hexahydrate ($[Ni(H_2O)_6]^{2+}$). Nickel is known to form strong, soluble complexes with hydroxide (OH^-), sulphate (SO_4^{2-}), and bicarbonate (HCO^{3-}) whereas formation of insoluble nickel precipitates may occur in the presence of iron and manganese oxides (ATSDR 2005a). These complexation and precipitation processes are pH dependent (Richter and Theis 1980). Water hardness in natural waters is controlled by the presence of calcium (Ca^{2+}) and magnesium (Mg^{2+}), which competes with metal cations for absorption in aquatic organisms (ATSDR 2005a). Water hardness can therefore affect the toxicity and bioavailability of nickel to aquatic organisms. Generally, as water hardness increases, toxicity decreases. Accordingly, the CCME guideline (CCME 1999a, updates to 2011) provides an on-line equation to adjust the guideline for nickel to local water hardness.

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for nickel is shown in Table 2.6-9. The SSD curve was conducted using chronic toxicity data for 27 aquatic species. Sufficient toxicity data were available for nickel to develop a SSD, with a logistic regression model providing a good fit to the observed data ($r^2 = 0.95$) (Figure 2.6-11). The logistic model followed the form:

$$y = \frac{121.21}{1 + \left(\frac{x}{6304.59}\right)^{-0.61}}$$

Where: y = percent of aquatic species affected; and

x = nickel concentration (µg/L).

Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for nickel were estimated to be 37, 445 and 3,538 µg/L, respectively (Figure 2.6-12).

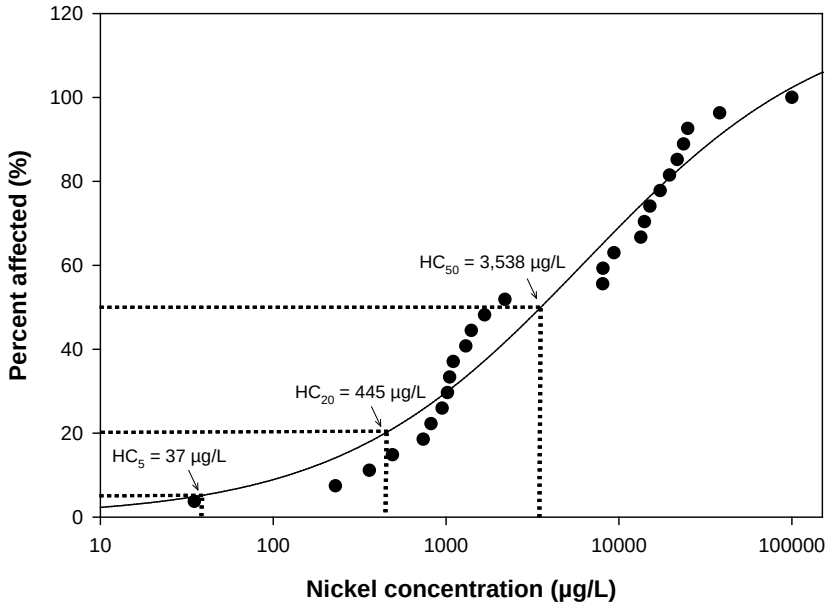
The CCME guideline for nickel is hardness dependent. Calculated nickel concentrations using the hardness-specific formula provided by CCME (1999a; updates to 2011) and based on the plausible range of water hardness for the study area were 56.7 µg/L (water hardness of 50 mg/L $CaCO_3$) and 299.7 µg/L (water hardness of 450 mg/L $CaCO_3$). For a central tendency hardness range of 143 to 149 mg/L $CaCO_3$, the nickel guideline becomes 125.4 to 129.4 µg/L.

The hardness-adjusted CCME water quality guideline value of 125.4 µg/L (lower end of range) was retained as the CEB for nickel; this value corresponds to the approximate HC_{10} of the SSD using all relevant nickel toxicity data. Only one species (rainbow trout) yielded a mean chronic toxicity value below the CEB.

Table 2.6-9 Available Chronic Freshwater Toxicity Data for Nickel

Species Scientific Name	Species Common Name	Chronic Value [µg/L]	Rank	Percent Affected [%]
Oncorhynchus mykiss	rainbow trout	35	1	3.7
Ceriodaphnia dubia	water flea	229	2	7.4
Paramecium bursaria	ciliate	360	3	11.1
Paramecium caudatum	ciliate	490	4	14.8
Spirostomum ambiguum	protozoa	737	5	18.5
Hyalella azteca	scud (amphipod)	819	6	22.2
Daphnia magna	water flea	950	7	25.9
Stylonychia pustulata	protozoa	1,020	8	29.6
Dexiostoma campyla	freshwater ciliate	1,050	9	33.3
Holosticha kessleri	protozoa	1,100	10	37.0
Paramecium putrinum	ciliate	1,300	11	40.7
Glaucoma scintillans	protozoa	1,400	12	44.4
Loxodes striatus	protozoa	1,670	13	48.2
Planorbella trivolvis	snail, marsh rams-horn	2,191	14	51.9
Pimephales promelas	fathead minnow	8,058	15	55.6
Morone saxatilis	striped bass	8,083	16	59.3
Chironomus sp.	midge	9,366	17	63.0
Anguilla rostrata	American eel	13,392	18	66.7
Gammarus sp.	scud (amphipod)	14,057	19	70.4
Nais sp.	oligochaete	15,114	20	74.1
Amnicola sp.	spire snail	17,313	21	77.8
Daphnia pulex	water flea	19,640	22	81.5
Tubifex tubifex	tubificid worm	21,720	23	85.2
Zygoptera	damselfly order	23,658	24	88.9
Chironomus riparius	midge	25,000	25	92.6
Trichoptera	caddisfly order	38,232	26	96.3
Gammarus fasciatus	scud (amphipod)	100,000	27	100.0

Figure 2.6-12 Species Sensitivity Distribution Curve for Nickel



2.6.18 Silver

Silver (Ag) has a relatively low abundance but is widely distributed in the earth's crust (CCREM 1987). It occurs naturally in ores and elemental form (CCREM 1987). It is released to the environment from natural and anthropogenic sources. Mining, silverware manufacturing and the photographic/imaging industry are the greatest anthropogenic contributors (Purcell and Peters 1998). It has been estimated that one-fourth to two-fifths of the silver released to the environment enters the aquatic component (Scow et al. 1981).

Silver may exist in the aquatic environment in several chemical forms. However, only the free ionic form of silver (Ag^+) is toxic to freshwater organisms (Hogstrand and Wood 1998). In natural waters, the free ionic form is present only at very low concentrations because it readily reacts (binds) with different inorganic and organic ligands that are present in the freshwater environment, including chloride, DOC and sulphides. Silver sulphide is the principal naturally occurring silver compound and is the likely fate of silver discharged into any environment containing a source of sulphide (Kramer et al. 2002). These bound forms of silver are much less toxic than silver nitrate ($AgNO_3$), the form which is frequently used in toxicity testing because it dissociates freely in solution to yield large amounts of Ag^+, representing a worst-case simulation of toxicity in the natural environment (Davies et al. 1978; LeBlanc et al. 1984). Recognizing this, recent research has focused on characterizing the role of such ligands in mitigating chronic silver toxicity in the freshwater environment, and development of a BLM for prediction of chronic silver toxicity, analogous to the BLM described above for copper.

The current Canadian Water Quality Guideline for the protection of freshwater aquatic life for silver is 0.1 µg/L (CCME 2007b), which was originally derived using the procedure of CCREM (1987) and has not changed since that date. This value is primarily based on the studies of Davies et al. (1978) and Nebeker et al. (1983). In an early life stage test using rainbow trout, Davies et al. (1978) reported a decrease in growth (length) at a silver

concentration of 0.17 µg/L. In an early life stage test using steelhead trout, Nebeker et al. (1983) reported a decrease in growth (length) at a silver concentration of 0.1 µg/L.

A summary of the toxicity database used to derive a CEB for silver is provided in Table 2.6-10.

Table 2.6-10 Available Chronic Freshwater Toxicity Data for Silver

Species Scientific Name	Species Common Name	Chronic Value [µg/L]	Rank	Percent Affected [%]
Salmo trutta	brown trout	0.2	1	7
Daphnia pulex	water flea	0.6	2	14
Isonychia bicolor	mayfly	0.7	3	21
Ceriodaphnia reticulata	water flea	0.8	4	29
Oncorhynchus mykiss	rainbow trout	1.0	5	36
Pimephales promelas	fathead minnow	1.0	6	43
Stenonema modestum	mayfly	2.5	7	50
Hyalella azteca	amphipod	2.5	8	57
Daphnia magna	water flea	4.5	9	64
Lemna minor	duckweed	6.0	10	71
Ceriodaphnia dubia	water flea	9.5	11	79
Pseudokirchneriella subcapitata	green algae	10.0	12	86
Chlorella vulgaris	green algae	23.0	13	93
Chironomus tentans	midge	63.0	14	100

The SSD was developed using chronic toxicity data for 14 freshwater species. Sufficient toxicity data were available for silver to develop an SSD, with a logistic regression model providing a good fit to the data (r^2= 0.98) (Figure 2.6-13). The logistic model followed the form:

$$y = y_o + \frac{a}{1 + \left(\frac{x}{x_o}\right)^b}$$

Where: y = percent of aquatic species affected;

a = 130.7799;

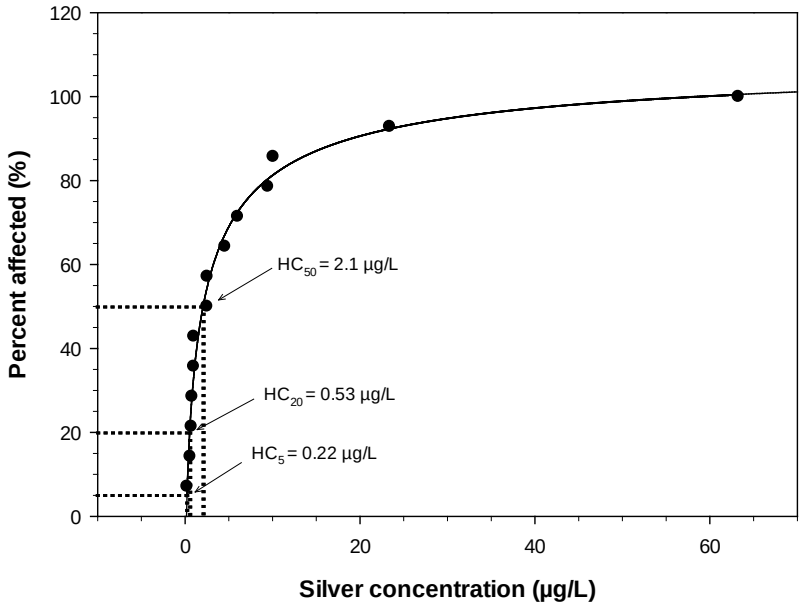
b = -0.698;

x_o = 1.6198;

y_o = -20.8465; and

x = silver concentration (µg/L).

Figure 2.6-13 Species Sensitivity Distribution Curve for Silver



Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for silver were estimated to be 0.22, 0.53 and 2.1 µg/L, respectively (Figure 2.6-13). The HC_5 of 0.22 µg/L is proposed as the CEB for silver. This value is close to the lowest species mean chronic value (for brown trout) and thus is protective of the most sensitive freshwater aquatic life.

2.6.19 Strontium

Strontium (Sr) can exist in two oxidation states: Sr^0 and Sr^{2+}. Under normal environmental conditions, only the Sr^{2+} oxidation state is stable enough to be of practical importance, since strontium readily reacts with both water and oxygen (Cotton and Wilkinson 1980; Hibbins 1997). There are 26 isotopes of strontium, four of which occur naturally. Naturally occurring strontium is not radioactive and is either referred to as stable strontium or simply as strontium.

Previous assessments of strontium toxicity have identified that, although sufficient data are available to develop an SSD for strontium, the logistic model could not provide a good fit to the data. The apparent reason for the lack of model fit was the disparity between the majority of chronic toxicity estimates (in the range 34,000 µg/L to 465,000 µg/L), and the very low values reported by Birge et al. (1979, 1980a) for rainbow trout embryos (49 µg/L and 200 µg/L). These disparities warranted a detailed critical assessment of the primary data prior to CEB derivation, particularly given concerns regarding data reliability previously identified for the Birge et al. (1979) study reference (compare molybdenum CEB derivation above). In addition, recent confirmatory testing has been conducted using modern protocols for toxicity testing, chronic test durations, and test organisms previously identified to be sensitive to strontium toxicity. Accordingly, this section presents a more detailed presentation of literature findings than is provided for other metals. For a technical summary similar in detail to other metals, the reader is referred to Section 2.6.19.6 of this appendix.

2.6.19.1 Water Quality Guidelines

National water quality guidelines for strontium for protection of freshwater aquatic life are currently not available in Canada or the United States.

Ecometrix (2011) proposed a Water Quality Objective (WQO) for strontium in a northern Canadian lake (Snap Lake) of 500 µg/L; the same value was also proposed as an environmental quality criterion for treated mine effluent, making no allowance for effluent mixing or dilution following discharge. This value was calculated as the geometric mean of the two lowest LC_{50} values in their database:[2] a 28-d LC_{50} for rainbow trout of 250 µg/L (Birge et al. 1980a) and a 7-d LC_{50} for the amphipod *Hyalella azteca* of 1,000 µg/L (Borgmann et al. 2005). Ecometrix (2011) identified potential technical issues with both these test results, which are discussed further below.

Hull (2008) provided a collection of worksheets showing calculations used to develop acute and chronic values for strontium for the Michigan Department of Environmental Quality (MDEQ 2008), although it was not clear whether these had been formally adopted as state water quality standards. Development of these water quality benchmarks for strontium involved rejecting all of the data available in the literature at the time, and relying on data from six unpublished studies. A Tier I Final Acute Value (FAV) of 80,600 µg/L was calculated using acute data from six studies; the FAV was divided by two to obtain an acute benchmark, the Aquatic Maximum Value (AMV), of 40,300 µg/L. Chronic toxicity data from one test with *Ceriodaphnia dubia* and one test with fathead minnow (Cook 2008, cited in Hull 2008), plus acute-to-chronic ratios, were used to calculate a Tier II Final Chronic Value (FCV) of 21,000 µg/L as a chronic benchmark for strontium. According to Chowdhury and Blust (2011), that chronic threshold was also adopted by Ohio (Ohio EPA 2009) and Quebec.

The Indiana Department of Environmental Management (IDEM 2001) calculated Tier II acute and chronic values for strontium using acute data from two studies with *Daphnia magna* and *Tubifex tubifex* (Khangarot 1991; Khangarot and Ray 1989). It appears that the Genus Mean Acute Value (GMAV) for *Daphnia magna* was calculated incorrectly because it used 24-h and 48-h LC_{50}s from the same test. Calculation of the acute and chronic values involved use of application factors and a default acute-to-chronic ratio because of the lack of data, resulting in a Tier II acute value of 4,800 µg/L and chronic value of 530 µg/L.

Given the limitations of the above guideline derivations in terms of data quality and consideration of available data, it was determined that recalculation of a guideline using the SSD assessment method is a preferred method for establishment of a CEB for strontium.

[2] The LC*p* is the concentration of test material estimated to be lethal to a specific percentage ("*p*") of the test organisms. The LC_{50}, or median lethal concentration, is the concentration estimated to be lethal to 50% of the test organisms.

2.6.19.2 Fish Toxicity

Pacholski (2009) conducted a 21-d test with juvenile rainbow trout *Oncorhynchus mykiss*; additional details of the test procedures and endpoint calculations were provided in HydroQual (2009, 2013 pers. comm.)[3]. Test fish were approximately 0.3 to 0.5 g wet weight at test initiation, and the exposure system was static-renewal with weekly replacement of test solutions. Control survival after 21 d was 90%, and the results were corrected for control responses. Survival was the only endpoint measured, and the endpoints reported were an LC_{10} of 67,000 µg/L, an LC_{20} of 110,000 µg/L, and an LC_{50} of 286,000 µg/L. The LC_{10} of 67,000 µg/L was considered to be an acceptable low-effect concentration and was therefore used for the CEB determination.

Birge (1978) conducted a 28-d test with rainbow trout, from fertilization through to four days post-hatch; results of this study were also reported in Birge et al. (1979). The exposure system was static-renewal, with replacement of test solutions every 12 hours. Control performance was not reported, but the results were corrected for control responses. Survival was the only endpoint measured; an LC_{01} of 6.0 µg/L and an LC_{50} of 200 µg/L were reported. The LC_{01} was considered to be too conservative an estimate of a no-effect concentration for the CEB, as the CCME (2007a) methodology allows for up to a 10% effect for that estimate, and was also problematic because it was within the range of baseline and/or background strontium concentrations associated with reference Canadian lakes and European and US streams. Conversely, the LC_{50} was considered to be insensitive and therefore unsuitable for the CEB; CCME (2007a) notes that if lethal endpoints are used as low-effect concentrations for the CEB, their effect level should be between 11% and 25%. A Maximum Acceptable Toxicant Concentration (MATC) of 35 µg/L was calculated as the geometric mean of the LC_{01} and LC_{50} concentrations. Results from this study were over 1,000 times more sensitive than reported by Pacholski (2009) for juveniles of the same species.

Birge et al. (1980a) reported results for a 28-d test with rainbow trout, conducted from fertilization through to four days post-hatch; results of this study were also reported in Birge et al. (1981). The exposure system was static-renewal, with replacement of test solutions every 12 hours. Control survival was 83% to 96%, which was acceptable for this type of test, and the results were corrected for control responses. Survival was the only endpoint measured, and the endpoints reported were an LC_{01} of 13 µg/L, an LC_{10} of 49 µg/L, and an LC_{50} of 250 µg/L. Results from this study were consistent with those reported by Birge (1978), but were at or below baseline/background concentrations associated with northern Canadian lakes and lower than background concentrations reported for European and US streams.

Birge et al. (1980a) noted that their point estimates were calculated by a different method than used in previous studies. Given the similarity in results reported by Birge (1978) and Birge et al. (1980a) for the 28-d rainbow trout test, and the lack of details about test methodologies, there is uncertainty as to whether these represent results from two separate tests or results from a single test calculated by different assessment methods. To provide a conservative approach to developing the strontium CEB, it has been assumed that they represent two separate tests. Because the results from the Birge (1978) and Birge et al. (1980a,b) rainbow trout tests indicated a much greater sensitivity to strontium than reflected in other toxicity test results, additional rainbow

[3] HydroQual (2009) conducted toxicity tests with freshwater algae, invertebrates, and fish in support of Pacholski (2009), but only reported point estimates based on 25% and 50% effect levels for each test. For the purpose of developing the strontium CEB proposed in this report, HydroQual was subsequently requested to provide point estimates based on the 10% and 20% effect levels for each those tests (HydroQual 2013, pers. comm.).

trout Early Life Stage (ELS) tests were conducted to determine whether these results were reproducible and could be relied upon (see Section 2.6.19.5).

Birge (1978) conducted a 7-d test with the goldfish *Carassius auratus*, from fertilization through to four days post-hatch; results of this study were also reported in Birge et al. (1979). The exposure system was static-renewal, with replacement of test solutions every 12 hours. Control performance was not reported, but the results were corrected for control responses. Survival was the only endpoint measured; an LC_{01} of 45.3 µg/L and an LC_{50} of 8,580 µg/L were reported. For reasons previously described, an MATC of 623 µg/L was calculated as the geometric mean of the LC_{01} and LC_{50}. This MATC was within the range of background concentrations reported for European and US streams.

Pacholski (2009) conducted a standard 7-d survival and growth test with the larval stage (<24-h old) of the fathead minnow *Pimephales promelas*; additional testing details and endpoint calculations were provided in HydroQual (2009; 2013, pers. comm.). Control performance was acceptable, and the results were corrected for the control responses. For survival, the endpoints reported were an LC_{10} of 255,000 µg/L, an LC_{20} of 276,000 µg/L, and a 7-d LC_{50} of 354,000 µg/L. For growth (expressed as increased dry weight), the endpoints were reported as an IC_{10} of 263,000 µg/L and an IC_{20} of 304,000 µg/L[4]. The IC_{10} of 263,000 µg/L was used for the CEB determination.

Hull (2008) provided a collection of worksheets showing calculations used to develop acute and chronic values for strontium for the Michigan Department of Environmental Quality, including tabulated toxicity data from several unpublished reports. Hull (2008) used survival data from a 7-d fathead minnow test conducted by Cook (2008), but only reported the No Observed Effect Concentration[5] (NOEC; 92,870 µg/L) and Lowest Observed Effect Concentration (LOEC; 188,750 µg/L) that were used to calculate an MATC of 132,390 µg/L. Copies of summary tables and bench sheets (Cook 2013, pers. comm.) were used to confirm testing details and calculate point estimates that would be more suitable for use in the CEB determination. The Cook (2008) fathead minnow test was a standard 7-d larval survival and growth test; control performance was acceptable and the results were corrected for control responses. For survival, the LC_{20} and LC_{50} were >92,870 µg/L. For growth (expressed as increased dry weight), the IC_{10} was <13,440 µg/L and the IC_{20} was 17,420 µg/L. Because the IC_{10} for growth was lower than the lowest test concentration, and therefore could not be estimated accurately, the IC_{20} of 17,420 µg/L was included in the CEB determination. Survival and growth results from the Cook (2008) 7-d fathead minnow test were more sensitive than the Pacholski (2009) 7-d fathead minnow results. However, survival results from Cook (2008) were similar to the Pacholski (2009) results for juvenile rainbow trout, and also consistent with results from three acute 96-h LC_{50}s for fathead minnow that ranged from 140,180 to 228,470 µg/L (Hull 2008).

Jones (1939) conducted toxicity tests with stickleback (*Gasterosteus aculeatus*), to determine a survival curve for strontium. The test duration was 10 days, and test solutions were renewed daily. The lethal time to 50% mortality (LT_{50}) for strontium was 1,200,000 µg/L for this 10-d exposure; this result was included in the CEB determination.

[4] The ICp is the inhibiting concentration for a specified percentage ("p") effect on a continuous endpoint such as growth or reproduction. For example, the IC_{10} is the concentration of test material estimated to cause a 10% reduction in growth or reproduction of the test species.
[5] The NOEC is the highest test concentration where there is no statistically significant difference in mean response relative to the control. The LOEC is the lowest test concentrations where there is a statistically significant difference in mean response relative to the control.

Schroder et al. (1995) reported that a 24-h immersion in a strontium chloride solution is used for marking chum salmon (*Oncorhynchus keta*) and sockeye salmon (*O. nerka*) fry prior to their release in the wild. The strontium is deposited in calcified tissues and can easily be detected in otoliths when the fish are older. For that study, chum salmon fry were exposed to three strontium concentrations (120,000, 1,200,000, or 9,000,000 µg/L) for 24 h, reared for 34 d on a standard hatchery diet, then sacrificed for analyses. Control mortality was 1%, mortality in the 1,200,000 µg/L treatment was 2%, and mortality in the 9,000,000 µg/L treatment was 7%. The geometric mean of these two results was used for the CEB determination. In a second experiment, sockeye salmon were immersed in a 5,000,000 µg/L strontium solution for 24 h and then reared for 21 months on a standard hatchery diet to determine how long the marked fish could be distinguished from unmarked fish. Although mortality data were not provided for the second experiment, it was presumed that survival was sufficiently high during the 21 month rearing period to provide meaningful test results. However, this information was not used in the CEB determination.

2.6.19.3 Invertebrate Toxicity

Biesinger and Christensen (1972) conducted 21-d tests with the water flea, *Daphnia magna*, to determine effects of strontium exposure on survival and reproduction. They reported a 21-d LC_{50} of 86,000 µg/L for survival and a 21-d EC_{50} of 60,000 µg/L for reproduction[6]. In addition, an EC_{16} of 42,000 µg/L was calculated for reproduction, to represent the lowest effect size that could be distinguished from variability associated with the control responses. This EC_{16} was used for the CEB determination.

Pacholski (2009) conducted a 21-d survival and reproduction test with *Daphnia magna*, and reported an LC_{50} of 122,000 µg/L for survival; the IC_{10} was 23,000 µg/L and the IC_{20} was 35,000 µg/L for reproduction. Control performance was acceptable, and results were corrected for control responses. Additional details regarding testing and endpoint calculations were provided in HydroQual (2009; 2013 pers.comm.). The IC_{10} and IC_{20} for reproduction were lower than the EC_{16} from the Biesinger and Christensen (1972) study; the IC_{10} was included for the CEB determination.

Cook (2008; cited in Hull 2008) conducted a 6-d survival and reproduction test with the water flea, *Ceriodaphnia dubia*. Hull (2008) only reported the NOEC and LOEC for reproduction as 24,570 and 45,890 µg/L, respectively, and used those values to calculate an MATC of 33,578 µg/L. Point estimates more suitable for use in the strontium CEB determination were determined using data provided by Cook (2013, pers.comm.). The LC_{50} for survival was 92,870 µg/L, and the IC_{10} and IC_{20} for reproduction were 22,920 µg/L and 33,610 µg/L, respectively. The IC_{10} for reproduction was included for the CEB determination.

Pacholski (2009) conducted a 6-d survival and reproduction test with *Ceriodaphnia dubia*, and reported an LC_{50} of 206,000 µg/L for survival, and an IC_{10} of 2,866 µg/L and an IC_{20} of 11,160 µg/L for reproduction. Additional details regarding testing and endpoint calculations were provided in HydroQual (2009; 2013 pers.comm.). Control performance was acceptable, and results were corrected for control responses. Mean reproduction fluctuated among the lower test concentrations, and therefore the IC_{20} of 11,160 µg/L was considered to be a more representative endpoint for use in the CEB determination.

[6] The EC*p* is the concentration of test material estimated to cause an adverse effect other than lethality to a specific percentage ("*p*") of the test organisms. The EC50, or median effective concentration, is the concentration estimated to cause an effect to 50% of the test organisms.

Borgmann et al. (2005) conducted 7-d tests with the amphipod, *Hyalella azteca*, to determine the effects of strontium on survival. The primary objective of this study was to assess the toxicity of 63 elements in waters at two different hardnesses, and therefore a number of elements were only tested at a few concentrations, starting at 1,000 µg/L and then testing at higher or lower concentrations depending on the initial result. This was the case for strontium, which was not tested at a full dilution series that would have allowed for determination of LC_{20} or LC_{50} values. In soft water, the 7-d LC_{50} was >1,000 µg/L; there was 18% mortality at 315 µg/L but only 12% mortality at 1,000 µg/L. In higher-hardness water, the 7-d LC_{50} was >3,150 µg/L, and there was only 7% mortality at 1,000 µg/L. The authors reported that control survival was at least 80%, which is reasonable for this test method. However, the results were not corrected for the control responses and, given that the survival results that were reported for strontium were all at least 80% it is possible that, with correction for the control responses, these effect sizes would have been smaller or even non-existent. This is supported by the fact that Hull (2008) reported a 48-h LC_{50} of 198,011 µg/L from an acute *Hyalella azteca* test, a concentration almost 200 times higher than that reported by Borgmann et al. (2005). To address the uncertainty regarding these results, additional toxicity testing was conducted with *Hyalella azteca* (see Section 2.6.19.5).

Boutet and Chaisemartin (1973) reported a 30-d LC_{50} of 390,000 µg/L for white clawed crayfish, *Austropolmobius pallipes pallipes*, and a 30-d LC_{50} of 860,000 µg/L for spinycheek crayfish, *Orconectes limonus*. Both these results were included for the CEB determination.

Suzuki (1959) conducted 10-d tests with mosquito larvae, *Culex pipiens paliens*, to determine the time required to reach 50% effect levels using different concentrations of strontium. The EC_{50} for emergence occurred at approximately 6.9 days and was 553,000 µg/L. The EC_{50} for pupation occurred at approximately 4.1 days and was 5,530 µg/L, but the time required to reach this endpoint was inconsistent for the range of test concentrations. The EC_{50} of 553,000 µg/L for emergence was considered to be more representative and was therefore included for the CEB determination.

Jones (1940) conducted 48-h tests with the planarian, *Polycelis nigra*, and reported LT_{50}s of 3,500,000 and 6,000,000 µg/L for two different strontium salts. The author considered this endpoint to be the threshold of toxicity because with only a slight dilution survival was extended considerably. Both of these results were included for the CEB determination.

2.6.19.4 Other Organism Toxicity

Pacholski (2009) conducted a standard 72-h algal growth test with the alga *Pseudokirchneriella subcapitata*; additional testing details and endpoint calculations were provided in HydroQual (2009; 2013 pers. comm.). Control performance was acceptable, and the results were corrected for the control responses. The 72-h IC_{10} was 36,000 µg/L and the IC_{20} was 47,000 µg/L; the IC_{10} was used for the CEB determination. The algae demonstrated a hormetic response, with growth stimulation occurring at strontium concentrations up to 23,000 µg/L, but inhibition of growth at higher concentrations.

Birge (1978) conducted a 7-d test with the narrow-mouthed toad, *Gastrophryne carolinensis*, from fertilization through to four days post-hatch; results of this study were also reported in Birge et al. (1979). The exposure system was static-renewal, with replacement of test solutions every 12 hours. Control performance was not reported, but the results were corrected for control responses. Survival was the only endpoint measured; an LC_{01} of 2.4 µg/L and an LC_{50} of 160 µg/L were reported. The LC_{01} was considered to be too conservative for use as a no-effect concentration, and the LC_{50} was not conservative enough as a low-effect concentration. An

MATC of 20 µg/L was calculated as the geometric mean of the LC_{01} and LC_{50}. This study was excluded from the CEB determination, because the test endpoints were close to baseline/background strontium concentrations associated with European and US streams.

2.6.19.5 Recent Confirmatory Toxicity Studies

Results of the chronic toxicity studies summarized above indicate two sets of studies contributing uncertainty to the strontium CEB determination. The 28-d rainbow trout test results reported by Birge (1978) and Birge et al. (1980a, 1980b) were orders of magnitude lower than other test results performed with a range of aquatic species. Therefore, additional rainbow trout Early Life Stage (ELS) tests were recently conducted to determine whether those test results were reproducible. Furthermore, the 7-d *Hyalella azteca* tests conducted by Borgmann et al. (2005) did not include high enough strontium concentrations to calculate point estimates, and therefore additional testing with *Hyalella azteca* was performed to determine sensitivity to higher strontium concentrations. Results from these additional toxicity tests (discussed below, and summarized in Table 2.6-11) were added to the chronic toxicity data set used for the strontium CEB determination.

a. Rainbow Trout Early Life Stage Toxicity Tests

Nautilus (2013) conducted two Rainbow Trout ELS tests to repeat the tests reported in Birge (1978) and Birge et al. (1980), in order to establish whether those results were repeatable, and to determine the relative sensitivity of rainbow trout to strontium. The tests were conducted under two water quality regimes: one with water hardness similar to that used by Birge and colleagues (approximately 100 mg/L as $CaCO_3$); and, a second test in water with a lower hardness (approximately 12 mg/L as $CaCO_3$). Testing was conducted under these two hardness regimes because it was anticipated that sensitivity to strontium may change in response to calcium concentrations in the water. The proximity of calcium and strontium to each other on the periodic table suggests that they may share similar properties that could result in interactions by competitive exclusion at uptake sites on the fish gill.

Table 2.6-11 Summary of Recent Freshwater Chronic Strontium Toxicity Testing

Endpoint		Effect Concentration for Survival to Hatch	Effect Concentration for Normally Developed Surviving Fry
Rainbow Trout - Soft Water			
Point Estimates (µg/L measured Sr)	EC_{50}	>157,500 µg/L	>157,500 µg/L
	EC_{20}	98,500 µg/L	101,400 µg/L
	EC_{10}	75,200 µg/L	77,800 µg/L
Rainbow Trout - Moderately Hard Water			
Point Estimates (µg/L measured Sr)	EC_{50}	>151,100 µg/L	>151,100 µg/L
	EC_{20}	>151,100 µg/L	>151,100 µg/L
	EC_{10}	>151,100 µg/L	>151,100 µg/L
Hyalella azteca		**Survival [%]**	**Biomass [mg/ind]**
Point Estimates (mg/L measured Sr)	LC_{50}	176,800 µg/L	Not applicable
	IC_{50}	Not applicable	79,600 µg/L
	IC_{20}	Not applicable	43,000 µg/L
	IC_{10}	Not applicable	31,200 µg/L

Source: Nautilus 2013.

SD = standard deviation.

Test assessment methods were intended to match those used by Birge (1978), with the following exceptions:

- test solutions were renewed every 24 h, rather than every 12 h;
- the number of eggs exposed per concentration was 120, rather than 150;
- the test temperature was 14°C ± 1°C, rather than 13°C ± 0.5°C; and
- the test ended seven days after 50% of the control fish had hatched (32 day exposure overall), rather than four days following hatch (28 day exposure overall).

These procedural differences were implemented to provide consistency with the standard Environment Canada (1998) test protocol. They were considered minor and would not be anticipated to result in any difference in sensitivity between the tests. If anything, the use of a slightly higher test temperature and longer test duration would have been expected to result in lower (more sensitive) test endpoints than those reported by Birge and colleagues; however, this was not the case.

Control performance was acceptable for both tests, and results were corrected for mean control responses. The endpoints measured were survival to hatching, and normal development of surviving fry. There was very little difference between these two endpoints, as almost all of the surviving fish developed normally. Because of small differences in the concentration-response patterns for each test endpoint, the point estimates for survival were slightly lower than those for normal development and were therefore given priority for the CEB determination.

Rainbow trout were more sensitive to strontium in very soft water, when exposed at the embryo-larval stage. In the test with very soft water, the survival endpoints were an LC_{10} of 75,200 µg/L, an LC_{20} of 98,500 µg/L, and an LC_{50} of >157,500 µg/L. In contrast, the corresponding survival endpoints for the test performed with moderately hard water, which is more representative of the PRM receiving environment, were all >151,100 µg/L. These point estimates were more than 1,000 times higher than those reported by Birge (1978) and Birge et al. (1980a,b), but were similar to the results reported by Pacholski (2009) for a 21-d test with rainbow trout fry. The LC_{10} of 75,200 µg/L in very soft water was used for the CEB determination; this was a conservative assessment method because Pierre River receiving waters have a higher hardness.

b. Hyalella azteca Survival and Growth Test

Nautilus (2012) conducted a toxicity test with the amphipod, *Hyalella azteca*, to obtain more clearly defined point estimates than those reported by Borgmann et al. (2005). Whereas Borgmann et al. (2005) conducted 7-d tests with survival as the only test endpoint, this additional testing was conducted using a 14-d test duration in order to measure effects on both survival and growth (Environment Canada 1997). The amphipods were exposed to strontium-spiked test solutions in test containers with a clean sediment substrate. Control performance was acceptable, and results were corrected for mean control responses. The endpoints measured were survival and growth (dry weight). For survival, the LC_{50} was 176,800 µg/L. For growth, the IC_{10} was 31,200 µg/L and the IC_{20} was 43,000 µg/L. These point estimates were at least an order of magnitude higher than those previously reported by Borgmann et al. (2005). The IC_{10} of 31,200 µg/L was included for the CEB determination.

2.6.19.6 Chronic Effects Benchmark Derivations

Tests with three species were excluded from the CEB calculation: goldfish (Birge 1978; Birge et al. 1979); rainbow trout (Birge 1978; Birge et al. 1979); and older testing with *Hyalella azteca* (Borgmann et al. 2005). Goldfish are not native to North America, and the tests by Birge and colleagues for this species produced results that overlapped with background strontium concentrations (i.e., were questionable). The tests conducted by Birge and colleagues with rainbow trout were not reproducible, also overlapped background concentrations, and had previously been considered unreliable. These two studies by Birge and colleagues also reported results for testing of a number of other metals, in addition to strontium. A review of the U.S. EPA water quality criteria for aluminum, arsenic, cadmium, chromium, copper, and selenium revealed that the corresponding data from these two studies were listed as 'other data' but were not included in the datasets used for criteria derivation; no reason was given for this exclusion. Ecometrix (2011) stated: "There is evidence for other metals that the Birge et al. tests are not reproducible…confidence in the trout result is low." Thus, the more recent data for rainbow trout were used instead in the CEB calculation.

The *Hyalella azteca* tests by Borgmann et al. (2005), when redone using additional test concentrations and an additional endpoint, provided less uncertain data for this amphipod. The study design and data processing used by Borgmann et al. (2005) were such that clearly defined point estimates could not be determined and the responses that were reported were likely overly conservative because they were not corrected for potentially similar control responses. Ecometrix (2011) stated that these results, like the results of the studies by Birge and colleagues discussed above, were low outliers in the literature. In contrast, Nautilus (2012) reported that effects on *Hyalella* only occurred at concentrations at least 30 times higher than those reported by Borgmann et al. (2005). These more recent data were used in the CEB calculation.

The endpoints that were used to generate the SSD for strontium are summarized in Table 2.6-12 along with the data from the Birge (1978), Birge et al. (1980a,b), and Borgmann et al. (2005) studies replaced by the rainbow trout studies by Pacholski (2009) and Nautilus (2013), and the *Hyalella azteca* study by Nautilus (2012). Data from 10 chronic studies with 12 species (representing 4 fish, 7 invertebrates, and 1 algal species) were used for this calculation.

An additional update to the CEB derivation entailed the use of the Hazen plotting position method, whereby the species mean chronic values were ranked from lowest to highest, and the percent of species affected was calculated using the following equation:

$$\text{Percent Affected} = (X - 0.5) / N$$

where: X = species rank;

1 = most sensitive species; and

N = total number of species in the database.

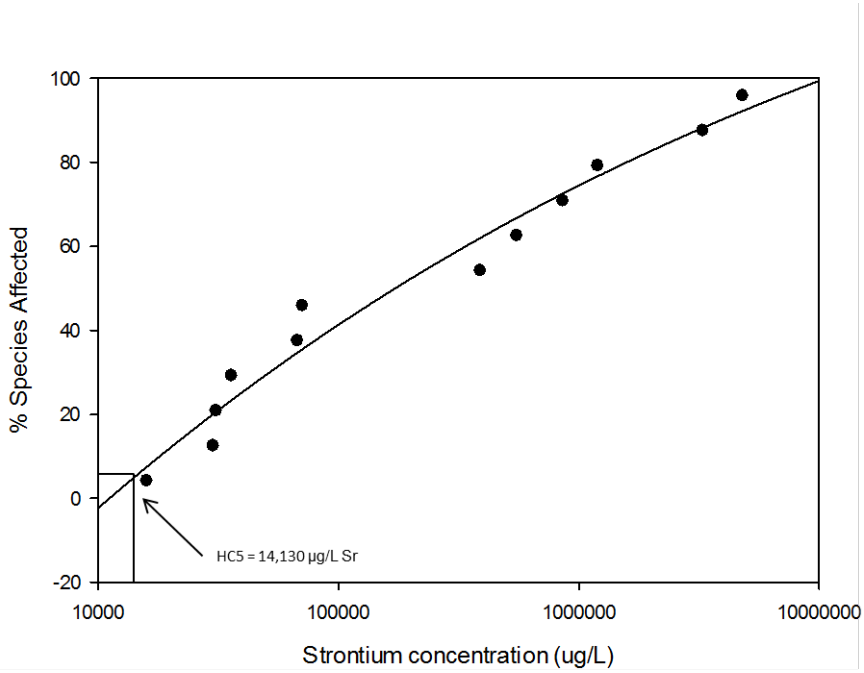
Table 2.6-12 Summary of Freshwater Chronic Toxicity Data for Strontium Retained for Species Sensitivity Distribution

Citation	Test Species	Common Name	Endpoint	Strontium Concentration [µg/L]	Species Mean Value [µg/L]	Rank	Percent Affected [%]
Pacholski (2009); HydroQual (2009, 2013)	*Ceriodaphnia dubia*	water flea	IC_{20}	11,160	15,993	1	4
Cook (2008) as cited in Hull (2008); Cook (2013)	*Ceriodaphnia dubia*	water flea	IC_{10}	22,920			
Nautilus (2012)	*Hyalella azteca*	amphipod	IC_{10}	30,240	30,240	2	13
Pacholski (2009); HydroQual (2009, 2013)	*Daphnia magna*	water flea	IC_{10}	23,000	31,081	3	21
Biesinger and Christensen (1972)	*Daphnia magna*	water flea	EC_{16}	42,000			
Pacholski (2009); HydroQual (2009, 2013)	*Pseudokirchneriella subcapitata*	green algae	IC_{10}	36,000	36,000	4	29
Cook (2008) as cited in Hull (2008); Cook (2013)	*Pimephales promelas*	fathead minnow	IC_{20}	17,420	67,686	5	38
Pacholski (2009); HydroQual (2009, 2013)	*Pimephales promelas*	fathead minnow	IC_{10}	263,000			
Pacholski (2009); HydroQual (2009, 2013)	*Oncorhynchus mykiss*	rainbow trout	LC_{10}	67,000	70,982	6	46
Nautilus (2013)	*Oncorhynchus mykiss*	rainbow trout	LC_{10}	75,200			
Boutet and Chaisemartin (1973)	*Austropotamobius pallipes pallipes*	white-clawed crayfish	LC_{50}	390,000	390,000	7	54
Suzuki (1959)	*Culex pipiens paliens*	mosquito	EC_{50}	553,000	553,000	8	63
Boutet and Chaisemartin (1973)	*Orconectes limosus*	spinycheek crayfish	LC_{50}	860,000	860,000	9	71
Jones (1939)	*Gasterosteus aculeatus L.*	threespine stickleback	LT_{50}	1,200,000	1,200,000	10	79
Schroder et al. (1995)	*Oncorhynchus keta*	chum salmon	NOEC	1,200,000	3,286,335	11	88
Schroder et al. (1995)	*Oncorhynchus keta*	chum salmon	LC_{06}	9,000,000			
Jones (1940)	*Polycelis nigra*	planarian	LT_{50}	3,500,000	4,806,246	12	96
Jones (1940)	*Polycelis nigra*	planarian	LT_{50}	6,600,000			

NOEC = no observed effect concentration.

The correction factor of 0.5 was used (Aldenberg et al. 2002) to create symmetry in cumulative probability (i.e., median ranked species will be associated with 50% affected) and to acknowledge that the concentration affecting the highest ranked species is not necessarily associated with adverse effects to the entire aquatic community. SigmaPlot software was used to fit the chronic data to a curve for the SSD, using a logistic four-parameter model. The CCME (2007a) approach for WQG derivation is to use the intercept of the fifth (5^{th}) percentile of the SSD as the WQG, with the intent that this hazardous concentration to 5% of species (HC_5) will provide protection to 95% of the aquatic species. The SSD curve for this dataset, and the associated HC_5 of 14,130 µg/L is shown in Figure 2.6-14.

Figure 2.6-14 Species Sensitivity Distribution Curve for Strontium



The HC_5 of 14,130 µg/L is a more realistic chronic threshold than the WQO of 500 µg/L calculated by Ecometrix (2011) using the geometric mean of the unreliable (as demonstrated in repeat testing) Birge et al. (1980a) and Borgmann et al. (2005) studies. This chronic threshold of 14,130 µg/L is also conservative when considered relative to the endpoints used to generate it. The six lowest SMCVs used to generate this chronic threshold ranged from approximately 16,000 to 71,000 µg/L and were calculated from point estimates that represented effect levels between 10% and 20%, with the majority being 10% effect levels. The above chronic threshold of 14,130 µg/L is also lower than the chronic threshold adopted for strontium by the US states of Michigan and Ohio (Hull 2008; MDEQ 2008; Ohio EPA 2009) and subsequently by Quebec (Chowdhury and Blust 2012): 21,000 µg/L.

A recent review of the homeostasis and toxicology of strontium (Chowdhury and Blust 2011) found that "Sr in the environment is not generally considered a concern to aquatic organisms. The only known case is the Kola region of Russia, where many lakes are heavily contaminated with Sr from nearby metal mines, and the fish living in the lakes are characterized by high concentrations of tissue Sr in association with skeletal abnormalities (Moiseenko and Kudryavtseva 2001)." Calcium and strontium share many common pathways; strontium uptake and toxicity decrease as calcium concentrations increase (Chowdhury and Blust 2011). This was evident in the results reported by Nautilus (2013) for rainbow trout ELS tests at two different water hardnesses; strontium was less toxic at the higher hardness. The elevated hardness of PRM receiving waters relative to many of the toxicity endpoints used in the CEB derivation provide an additional level of conservatism.

The updated assessment of strontium toxicity has incorporated advances in the understanding of strontium toxicity to freshwater aquatic life. The results of recent standardized testing using published protocols has addressed uncertainty in historical testing results, and has characterized effects to sensitive taxa including

rainbow trout early life stages and crustaceans. The HC_5 of 14.1 mg/L Sr from the revised SSD provides a more reliable basis for screening potential toxicity due to strontium.

2.6.20 Vanadium

The transport and speciation of vanadium (V) in water is influenced by pH, redox potential and the presence of particulate matter. In fresh water, vanadium generally exists in solution as the vanadyl ion, (V^{4+}) under reducing conditions and the vanadate ion (V^{5+}) under oxidizing conditions, or as an integral part of, or adsorbed onto, particulate matter (Wehrli and Stumm 1989). The partitioning of vanadium between water and sediment is strongly influenced by the presence of particulates in the water. Both vanadate and vanadyl species are known to bind strongly to mineral or biogenic surfaces by adsorption or complexing (Wehrli and Stumm 1989).

Insufficient data were available to develop an SSD for vanadium. The lowest reported toxicity value of 33.8 µg/L was, therefore, selected for use as the CEB for vanadium. This value is based on a 28-day LC_{10} test result (Birge et al. 1979, 1980a) with rainbow trout embryos. As discussed earlier in the context of molybdenum and strontium CEB derivations, the Birge et al. test endpoints should be interpreted with caution due to evidence for other metals that the tests are not reproducible.

2.6.21 Zinc

Zinc (Zn) compounds can be found in rocks, certain minerals, and carbonate sediments. As a result of weathering of these materials, soluble compounds of zinc may be released into aquatic systems. Urban runoff, mine drainage, and industrial effluents may be also a source of zinc to receiving aquatic systems. Zinc exists in the aquatic environment as Zn^{2+} oxidation state with zinc hydroxide ($Zn[OH]_3$) as the main form present in natural waters (ATSDR 2005b). This element frequently forms complexes with a variety of organic and inorganic compounds (e.g., humic acids). The stability of these zinc complexes depends on the pH of the water with a higher dissociation rate occurring as the pH decreases (Guy and Chakrabarti 1976).

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for zinc is shown in Table 2.6-13. The SSD was conducted using chronic toxicity data for 14 aquatic species. Sufficient toxicity data were available for zinc to develop a SSD, with a logistic regression model providing a good fit to the observed data ($r^2 = 0.98$) (Figure 2.6-15). The logistic model followed the form:

$$y = \frac{118.67}{1 + \left(\frac{x}{814.05}\right)^{-1.76}}$$

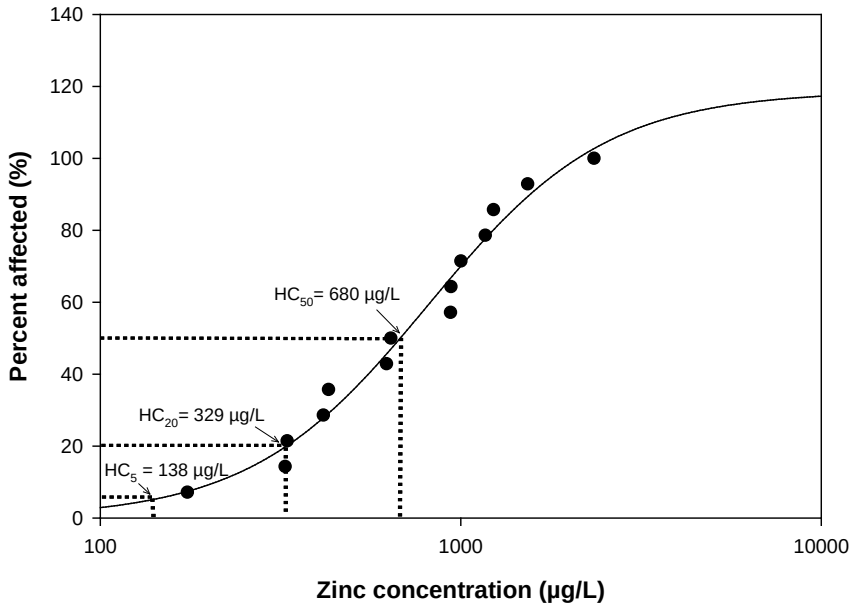
Where: y = percent of aquatic species affected; and

x = zinc concentration (µg/L).

Table 2.6-13 Available Chronic Freshwater Aquatic Toxicity Data for Zinc

Species Scientific Name	Species Common Name	Chronic Value [µg/L]	Rank	Percent Affected [%]
Hyalella azteca	scud (amphipod)	174	1	7.1
Daphnia magna	water flea	326	2	14.3
Salvelinus confluentus	bull trout	330	3	21.4
Ceriodaphnia dubia	water flea	416	4	28.6
Morone saxatilis	striped bass	430	5	35.7
Daphnia longispina	water flea	623	6	42.9
Oncorhynchus clarki	cutthroat trout	640	7	50.0
Ceriodaphnia reticulata	water flea	937	8	57.1
Simocephalus vetulus	water flea	939	9	64.3
Daphnia galeata	water flea	1,001	10	71.4
Simocephalus exspinosus	cladoceran	1,171	11	78.6
Oncorhynchus mykiss	rainbow trout	1,233	12	85.7
Ceriodaphnia pulchella	water flea	1,534	13	92.9
Pimephales promelas	fathead minnow	2,344	14	100.0

Figure 2.6-15 Species Sensitivity Distribution Curve for Zinc



Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for zinc were estimated to be 138, 329 and 680 µg/L, respectively (Figure 2.6-15).

The toxicity benchmark for zinc reported by CCME (1999a; updates to 2011) is 30 µg/L. However, guidance is not clearly provided on how this toxicity benchmark was derived. The percentage of affected species calculated

by plotting the proposed guideline value of 30 µg Zn/L using the site-specific SSD curve derived in this report was less than 1%. Therefore, the proposed CCME guideline for zinc appears to be conservative given the aquatic species and water quality conditions encountered in the study area.

2.7 Chronic Effects Benchmark Results for Polycyclic Aromatic Hydrocarbons

This section presents the results of the individual CEB derivations for the following indicator Polycyclic Aromatic Hydrocarbon (PAH) compounds:

- anthracene;
- fluoranthene;
- fluorene;
- naphthalene;
- phenanthrene; and
- pyrene.

The results from these derivations were combined with the information in Table 2.5-1 to assign CEBs to the PAH Groups 1 through 9.

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of organic compounds that contain two or more benzene rings in their structure. Polycyclic aromatic hydrocarbons are ubiquitous in terrestrial, atmospheric and aquatic environments (ATSDR 1995). In general, PAHs can be grouped as low molecular weight (less than four rings; including acenaphthene, acenaphthylene, anthracene, fluorene, 1-methylnaphthalene, 2-methylnaphthalene, naphthalene and phenanthrene), and high molecular weight (four or more rings; including benzo(a)anthracene, benzo(a)pyrene, benzo(b&j)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, indeno(1,2,3-cd)pyrene and pyrene). These compounds can enter aquatic systems through atmospheric deposition and from discharges of industrial effluents, municipal waste water, and improper disposal of used motor oil. Transport and partitioning (e.g., Henry's law constant, soil organic carbon-water partitioning coefficient [K_{OC}] values, and n-octanol/water partition coefficient [K_{OW}] values) of PAHs in aquatic environments are generally related to their molecular weights (ATSDR 1995).

Once in the aquatic environment, PAHs compounds can be removed from the water column by volatilization to the atmosphere (primarily low molecular PAHs), by binding to suspended particles or sediments, or by being accumulated by aquatic biota (McGrath and DiToro 2009). Due to their low solubility and high affinity for organic carbon, PAHs are primarily found sorbed to particles (e.g., humic acids) in aquatic systems. Polycyclic aromatic hydrocarbons can be also chemically transformed by photo-oxidation into metabolites that may be more carcinogenic and toxic than the parental compound (Oris and Giesy 1986). This process and its associated phototoxicity has been observed mainly under laboratory conditions and rarely studied in natural environments, thus the ecological relevance of PAHs photo-oxidation is still unclear (McDonald and Chapman 2002).

Aquatic organisms can accumulate PAHs directly from water, sediments or food with bioconcentration factors generally ranging from 10 to 10,000 (Eisler 1987). In general, bioconcentration is greater for the higher molecular weight than for the lower molecular weight PAHs. Polycyclic aromatic hydrocarbons biomagnification has not been reported to occur in aquatic organisms because of the rapid metabolism and high elimination rate of these compounds in aquatic organisms (Eisler 1987).

2.7.1 Anthracene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for anthracene is show in Table 2.7-1. The SSD was conducted using chronic toxicity data for seven aquatic species. Sufficient toxicity data were available for anthracene to develop a SSD, with a logistic regression model providing good fit to the data (r^2 = 0.90) (Figure 2.7-1). The logistic model followed the form:

$$y = \frac{86.93}{1 + \left(\frac{x}{14.37}\right)^{-2.95}}$$

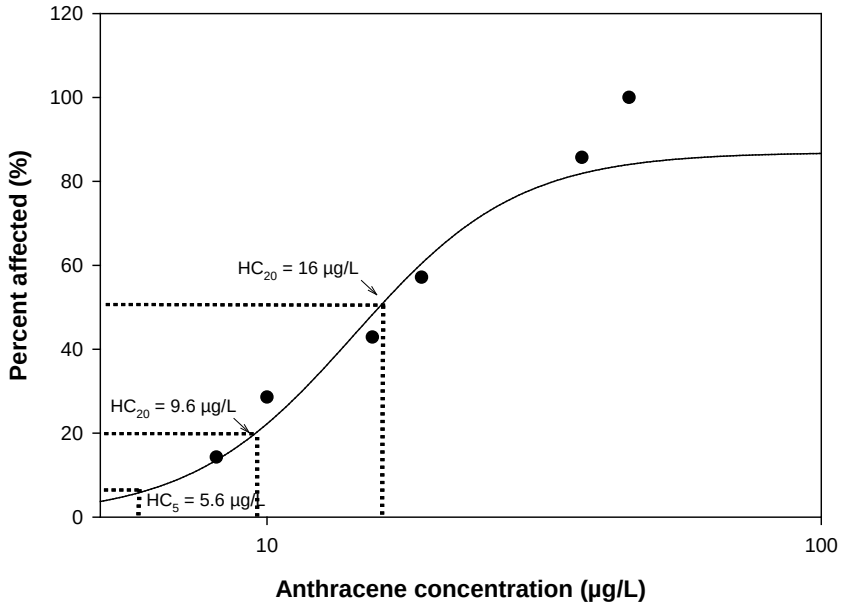
Where: y = percent of aquatic species affected; and

x = anthracene concentration (µg/L).

Table 2.7-1 Available Chronic Freshwater Aquatic Toxicity Data for Anthracene

Species Scientific Name	Species Common Name	Ranking	Chronic Value [µg/L]	Percent Affected [%]
Daphnia magna	water flea	1	8.1	14.3
Hydrilla verticillata	hydrilla	2	10.0	28.6
Pimephales promelas	fathead minnow	3	15.5	42.9
Selenastrum capricornutum	green algae	4	19.4	57.1
Daphnia pulex	water flea	5	32.7	71.4
Lumbriculus variegatus	oligochaete, worm	6	37.0	85.7
Aedes taeniorhynchus	mosquito	7	45.0	100.0

Figure 2.7-1 Species Sensitivity Distribution Curve for Anthracene



Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for anthracene were estimated to be 5.6, 9.6 and 16 µg/L, respectively (Figure 2.7-1).

The HC_5 for anthracene reported in this study was two times lower than the HC_5 reported by McGrath and DiToro (2009). The SSD-based derivations correspond strongly to the TLM for toxicity assessment of Type I narcotic chemicals (Table 2.7-2). The small differences in the predicted HC_5 concentration may be explained by the inclusion/exclusion criteria to derive SSD relationships followed in this report, such as inclusion of studies to better represent site-specific conditions and the exclusion of aquatic species not relevant to Canadian waters.

Table 2.7-2 Predicted Chronic HC_5 Values for Polycyclic Aromatic Hydrocarbons

Source	Anthracene [µg/L]	Fluoranthene [µg/L]	Fluorene [µg/L]	Naphthalene [µg/L]	Phenanthrene [µg/L]	Pyrene [µg/L]
McGrath and Di Toro (2009)	11.3	3.7	39.9	132.0	10.4	3.6
This report (Site-specific SSD)	5.6	5.9	17.1	32.0	6.7	2.3

The interim water quality guideline for anthracene reported by CCME (1999a; updates to 2011) is 0.012 µg/L. This guideline was derived by multiplying the 15-minute LT_{50} phototoxicity value reported by Allred and Giesy (1985) for *Daphnia pulex* by a safety factor of 0.01 (CCME 1991). The use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

2.7.2 Fluoranthene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for fluoranthene is provided in Table 2.7-3. The SSD for fluoranthene was conducted using chronic toxicity data for 19 aquatic species. Sufficient toxicity data were available for fluoranthene to develop a SSD, with a logistic regression model providing a good fit to the observed data (r^2 = 0.98) (Figure 2.7-2). The logistic model followed the form:

$$y = \frac{126.94}{1 + \left(\frac{x}{93.70}\right)^{-1.15}}$$

Where: y = percent of aquatic species affected; and

x = fluoranthene concentration (µg/L).

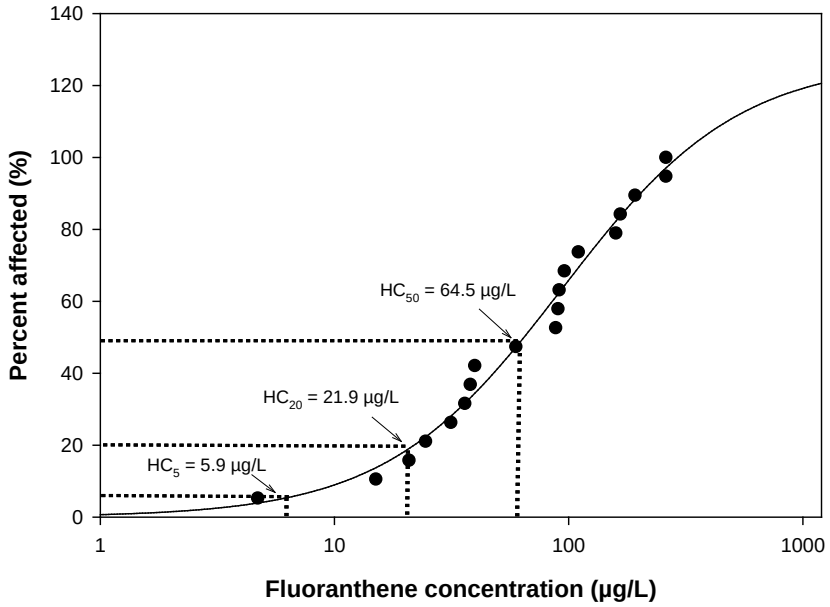
Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for fluoranthene were estimated to be 5.9, 21.9 and 64.5 µg/L, respectively (Figure 2.7-2). The HC_5 for fluoranthene reported in this study was slightly higher than the HC_5 reported by McGrath and DiToro (2009) (Table 2.7-2).

The interim water quality guideline for fluoranthene reported by CCME (1999a; updates to 2011) is 0.04 µg/L. This guideline was derived by multiplying the acute LC_{50} of 4 µg/L reported by Kagan et al. (1985) in *Daphnia magna* exposed to fluoranthene and UV light by a safety factor of 0.01 (CCME 1991). The use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

Table 2.7-3 Available Chronic Freshwater Aquatic Toxicity Data for Fluoranthene

Species Scientific Name	Species Common Name	Rank	Chronic Value [µg/L]	Percent Affected [%]
Lumbriculus variegatus	oligochaete, worm	1	5	5.3
Pimephales promelas	fathead minnow	2	15	10.5
Hyalella azteca	scud (amphipod)	3	21	15.8
Chironomus tentans	midge	4	25	21.1
Daphnia magna	water flea	5	31	26.3
Ictalurus punctatus	channel catfish	6	36	31.6
Anabaena flosaquae	blue-green algae	7	38	36.8
Ceriodaphnia dubia	water flea	8	40	42.1
Rana catesbeiana	bullfrog	9	60	47.4
Chironomus riparius	midge	10	88	52.6
Diporeia sp.	scud (amphipod)	11	90	57.9
Oncorhynchus mykiss	rainbow trout	12	91	63.2
Gammarus pulex	amphipod	13	96	68.4
Utterbackia imbecillis	paper pondshell	14	110	73.7
Stylaria lacustris	oligochaete	15	159	79.0
Lemna minor	duckweed	16	166	84.2
Scenedesmus subspicatus	algae	17	192	89.5
Lemna gibba	inflated duckweed	18	260	94.7
Pseudokirchneriella subcapitata	algae	19	260	100.0

Figure 2.7-2 Species Sensitivity Distribution Curve for Fluoranthene



2.7.3 Fluorene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for fluorene is provided in Table 2.7-4. The SSD for fluorene was conducted using chronic toxicity data for 15 aquatic species. Sufficient toxicity data were available for fluorene to develop a SSD, with a logistic regression model providing a good fit to the observed data (r^2 = 0.95) (Figure 2.7-3). The logistic model followed the form:

$$y = \frac{97.48}{1 + \left(\frac{x}{634.62}\right)^{-0.81}}$$

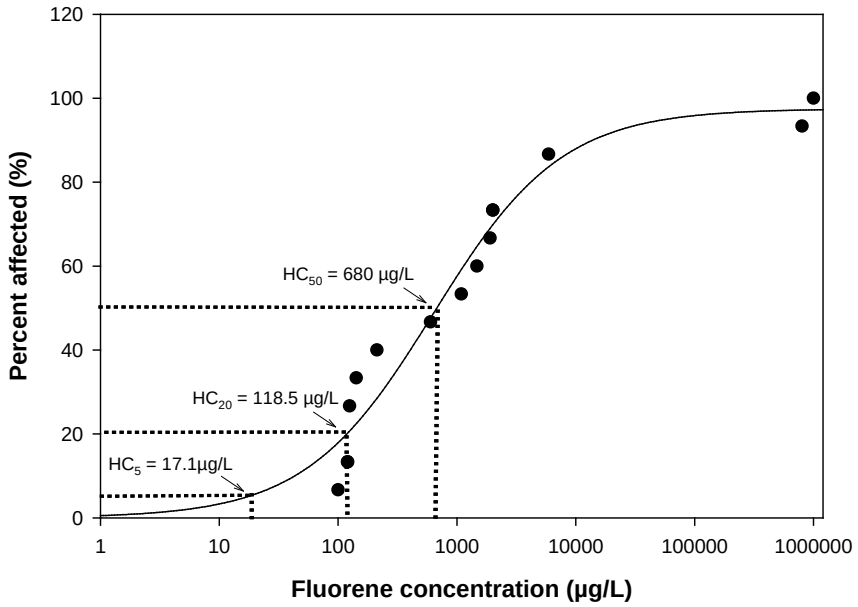
Where: y = percent of aquatic species affected; and

x = fluorene concentration (µg/L).

Table 2.7-4 Available Chronic Freshwater Aquatic Toxicity Data for Fluorene

Species Scientific Name	Species Common Name	Rank	Chronic Value [µg/L]	Percent Affected [%]
Lumbriculus variegatus	oligochaete, worm	1	100	6.7
Micropterus salmoides	largemouth bass	2	120	13.3
Plankton	plankton	2	120	13.3
Daphnia magna	water flea	4	125	26.7
Chironomus riparius	midge	5	142	33.3
Daphnia pulex	water flea	6	212	40.0
Gammarus pseudolimnaeus	scud (amphipod)	7	600	46.7
Anabaena flosaquae	blue-green algae	8	1,089	53.3
Oncorhynchus mykiss	rainbow trout	9	1,473	60.0
M. potosensis	snail	10	1,900	66.7
Invertebrates	invertebrates	11	2,000	73.3
Lemna gibba	inflated duckweed	11	2,000	73.3
Hexagenia bilineata	mayfly	13	5,900	86.7
Selenastrum capricornutum	green algae	14	800,000	93.3
Pimephales promelas	fathead minnow	15	1,000,000	100.0

Figure 2.7-3 Species Sensitivity Distribution Curve for Fluorene



Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for fluorene were estimated to be 17.1, 118.5 and 680 µg/L, respectively (Figure 2.7-3). The HC_5 for fluorene reported in this study was a factor of two lower than the HC_5 reported by McGrath and DiToro (2009) (Table 2.7-2).

The interim water quality guideline for fluorene reported by CCME (1999a; updates to 2011) is 3 µg/L. This guideline was derived by multiplying the LOEC of 125 µg/L (nominal concentration) reported by Finger et al.

(1985) for phototoxicity responses to *Daphnia magna* by a safety factor of 0.1 (CCME 1991). The result was then multiplied by a correction factor of 0.24 because the measured fluorene concentration during the chronic tests with daphnids was 24% of the reported nominal LOEC concentration of 125 µg/L. However, the use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

2.7.4 Naphthalene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for naphthalene is provided in Table 2.7-5. The SSD was conducted using chronic toxicity data for 20 aquatic species. Sufficient toxicity data were available for naphthalene to develop a SSD, with a logistic regression model providing strong fit to the observed data ($r^2 = 0.98$) (Figure 2.7-4). The logistic model followed the form:

$$y = \frac{101.65}{1 + \left(\frac{x}{1480}\right)^{-0.77}}$$

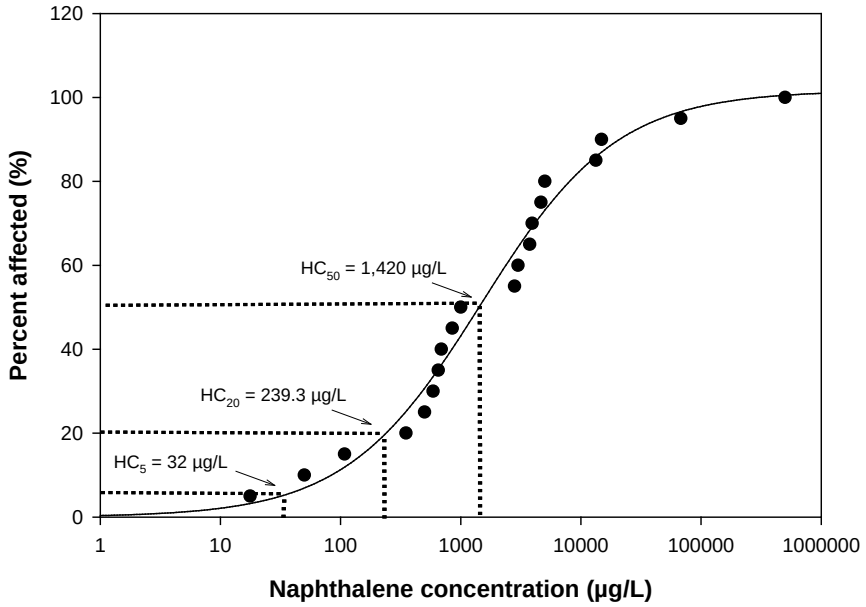
Where: y = percent of aquatic species affected; and
x = naphthalene concentration (µg/L).

Table 2.7-5 Available Chronic Freshwater Aquatic Toxicity Data for Naphthalene

Species Scientific Name	Species Common Name	Rank	Chronic Value [µg/L]	Percent Affected [%]
Oncorhynchus mykiss	rainbow trout	1	18	5
Tanytarsus dissimilis	midge	2	50	10
Oncorhynchus kisutch	coho salmon	3	108	15
Chlamydomonas angulosa	green algae	4	350	20
Lemna gibba	inflated duckweed	5	500	25
Micropterus salmoides	largemouth bass	6	589	30
Pimephales promelas	fathead minnow	7	650	35
Daphnia magna	water flea	8	690	40
Anacystis aeruginosa	blue-green algae	9	850	45
Somatochlora cingulata	dragonfly	10	1,000	50
Chironomus tentans	midge	11	2,810	55
Chlorella vulgaris	green algae	12	3,000	60
Chlamydomonas moewusii	green algae	13	3,750	65
Gammarus minus	scud (amphipod)	14	3,930	70
Daphnia pulex	water flea	15	4,660	75
Physa gyrina	pouch snail	16	5,020	80
Chironomus attenuatus	midge	17	13,321	85
Anabaena flosaquae	blue-green algae	18	14,851	90
Diaptomus forbesi	calanoid copepod	19	67,800	95
Selenastrum capricornutum	green algae	20	500,000	100

Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for naphthalene were estimated to be 32, 239.3 and 1,420 µg/L, respectively (Figure 2.7-4). The HC_5 for naphthalene reported in this study was four times lower than the HC_5 reported by McGrath and DiToro (2009) (Table 2.7-2).

Figure 2.7-4 Species Sensitivity Distribution Curve for Naphthalene



The interim water quality guideline for naphthalene reported by CCME (1999a; updates to 2011) is 1.1 µg/L. This guideline was derived by multiplying the LOEC (survival) of 11 µg/L reported by Black et al. (1983) for rainbow trout embryos by a safety factor of 0.1 (CCME 1991). However, the use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

2.7.5 Phenanthrene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for phenanthrene is provided in Table 2.7-6. The SSD was conducted using chronic toxicity data for 17 aquatic species. Sufficient toxicity data were available for phenanthrene to develop a SSD, with a logistic regression model providing a good fit to the observed data (r^2 = 0.97) (Figure 2.7-5). The logistic model followed the form:

$$y = \frac{142.06}{1 + \left(\frac{x}{454.38}\right)^{-0.78}}$$

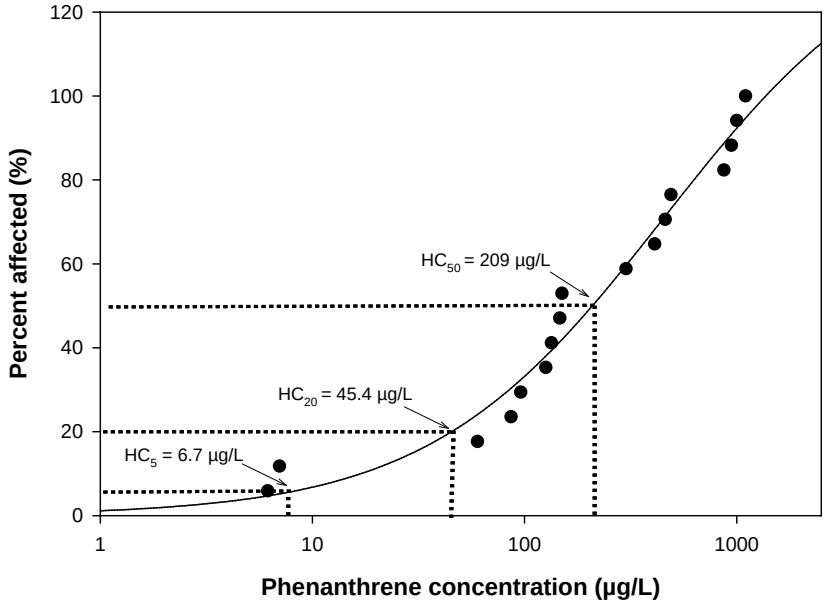
Where: y = percent of aquatic species affected; and

x = phenanthrene concentration (µg/L).

Table 2.7-6 Available Chronic Freshwater Aquatic Toxicity Data for Phenanthrene

Species Scientific Name	Species Common Name	Rank	Chronic Value [µg/L]	Percent Affected [%]
Oncorhynchus mykiss	rainbow trout	1	6	5.9
Daphnia magna	water flea	2	7	11.8
Daphnia pulex	water flea	3	60	17.7
Ceriodaphnia dubia	water flea	4	86	23.5
Hydra sp.	hydroid	5	96	29.4
Gammarus pseudolimnaeus	amphipod	6	126	35.3
Anabaena flosaquae	blue-green algae	7	134	41.2
Micropterus salmoides	largemouth bass	8	147	47.1
Anacystis aeruginosa	blue-green algae	9	150	52.9
Gammarus pulex	amphipod	10	301	58.8
Selenastrum capricornutum	green algae	11	410	64.7
Gammarus minus	amphipod	12	460	70.6
Chironomus tentans	midge	13	490	76.5
Nitzschia palea	diatom	14	870	82.4
Chlamydomonas angulosa	green algae	15	944	88.2
Lemna gibba	inflated duckweed	16	1,000	94.1
Chlorella vulgaris	green algae	17	1,100	100.0

Figure 2.7-5 Species Sensitivity Distribution Curve for Phenanthrene



Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for phenanthrene were estimated to be 6.7, 45.4 and 209 µg/L, respectively (Figure 2.7-5). The HC_5 for phenanthrene reported in this study was slightly lower than the HC_5 reported by McGrath and DiToro (2009) (Table 2.7-2).

The interim water quality guideline for phenanthrene reported by CCME (1999a; updates to 2011) is 0.4 µg/L. This guideline was derived by multiplying the phototoxicity LOEC (survival) of 4 µg/L reported by Black et al. (1983) for rainbow trout by a safety factor of 0.1 (CCME 1991). Therefore, the proposed CCME guideline for phenanthrene appears to be over-conservative given the aquatic species and water quality conditions encountered in the study area. The use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

2.7.6 Pyrene

A summary of the toxicity database used to derive a site-specific chronic toxicological benchmark for pyrene is shown in Table 2.7-7. The SSD was conducted using chronic toxicity data for five aquatic species. Sufficient toxicity data were available for pyrene to develop a SSD, with a logistic regression model providing a good fit to the observed data (r^2 = 0.97) (Figure 2.7-6). The logistic model followed the form:

$$y = \frac{105.09}{1+\left(\frac{x}{15.73}\right)^{-2.10}}$$

Where: y = percent of aquatic species affected; and

x = pyrene concentration (µg/L).

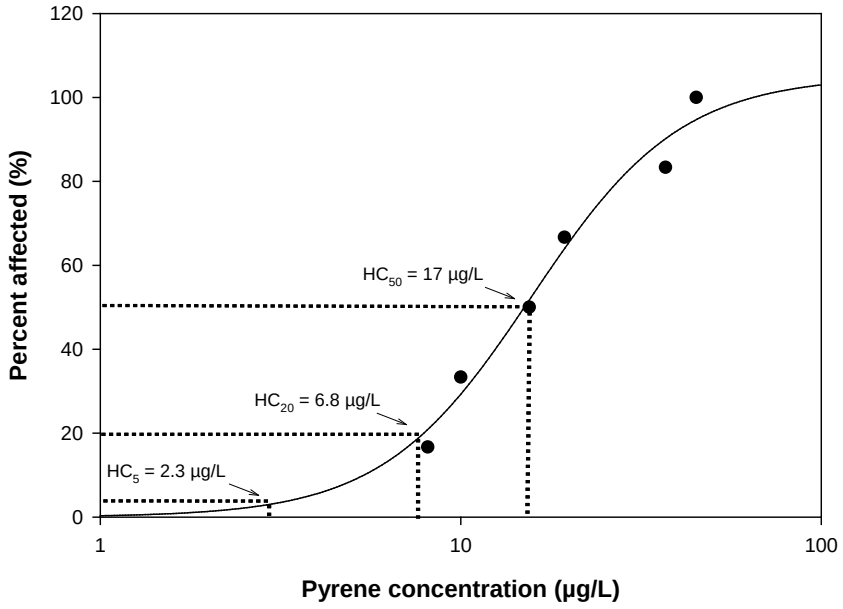
Table 2.7-7 Available Chronic Freshwater Aquatic Toxicity Data for Pyrene

Species Scientific Name	Species Common Name	Rank	Chronic Value [µg/L]	Percent Affected [%]
Lumbriculus variegatus	oligochaete, worm	1	5.0	20
Ceriodaphnia dubia	water flea	2	18.5	40
Daphnia magna	water flea	3	22.0	60
Gammarus pulex	amphipod	4	27.1	80
Chlamydomonas angulosa	algae	5	132.0	100

Based on the logistic regression model, the HC_5, HC_{20} and HC_{50} for pyrene were estimated to be 2.3, 6.8 and 17 µg/L, respectively (Figure 2.7-6). The HC_5 for pyrene reported in this study was slightly lower than the HC_5 of 3.6 µg/L reported by McGrath and DiToro (2009) (Table 2.7-2).

The interim water quality guideline for pyrene reported by CCME (1999a; updates to 2011) is 0.025 µg/L. This guideline was derived by multiplying the acute value (LC_{50}) of 2.5 µg/L for phototoxicity to mosquito larvae (*A. aegypti*) (Kagan and Kagan 1986) by a safety factor of 0.01 (CCME 1991). The lowest acute value for phototoxicity to mosquitoes, prior to application of safety factors, is similar to the HC_5 for non-phototoxicity endpoints. Therefore, the proposed CCME guideline for pyrene appears to be over-conservative given the aquatic species and water quality conditions encountered in the region. The use of a phototoxicity endpoint to characterize site-specific toxicity for PRM is not considered to be ecologically relevant.

Figure 2.7-6 Species Sensitivity Distribution Curve for Pyrene



2.7.7 Application of Polycyclic Aromatic Hydrocarbon Compounds to Groups

As shown in Table 2.7-2, the correspondence is strong between the SSD-based derivations and the TLM for toxicity assessment of Type I narcotic chemicals (McGrath and DiToro 2009). For five of the six compounds evaluated in this study, the SSD approach yielded a lower CEB, indicating that the SSD approach is expected to provide an appropriately conservative threshold for chronic toxic responses. The difference between the two assessment methods was less than a factor of two for most of the individual PAHs. This broad agreement provides validation of the McGrath and DiToro (2009) chronic benchmarks for application to PAH Groups for which no individual PAH was evaluated using the SSD method.

2.8 Chronic Effects Benchmark Results for Other Constituents

2.8.1 Ammonia

Ammonia can act as both a nutrient and a toxicant. The toxicity of ammonia is strongly influenced by ammonia speciation, which in turn is influenced by environmental parameters including pH, temperature and ionic strength. The CCME (2010) freshwater guideline for ammonia is for unionized ammonia (0.019 mg/L) rather than total concentration.

Data collected from the Oil Sands Region indicate that the majority of the nitrogen (N) present in process-affected waters is in the form of ammonia, with some organic nitrogen and little or no nitrate or nitrite. In aerobic environments, ammonia is transformed to nitrate or used for growth by aquatic plants.

As the PRM data will likely consist of total ammonia concentrations, it is necessary to consider the site-specific temperature and pH, which are the primary determinants of ammonia speciation. Using the temperature interval

(5°C to 10°C) and pH (7.5) values that are representative of the LSA for PRM, CCME (2010) indicates a water quality guideline range of 3.26 to 4.84 mg/L ammonia (NH_3) for the protection of aquatic life. This number can be converted to total ammonia nitrogen by multiplying by 0.8224, yielding a water quality guideline range of 2.68 to 3.98 mg/L N.

Note that as the temperature rises, the guideline decreases, such that the guideline during summer months (chronic exposure) would decline to about 2.0 mg/L N. To be conservative, the latter number was used as the CEB.

2.8.2 Naphthenic Acids

Naphthenic acids are found at concentrations ranging from 20 to 120 mg/L in oil sands process waters of northeastern Alberta (Clemente et al. 2004). These compounds have been identified as the agent responsible for most of the acute toxicity observed in tailings water and other process-affected waters (MacKinnon and Boerger 1986; Verbeek et al. 1993). Naphthenic acids act as surfactants, interfering with normal gas exchange across gill membranes in fish. Exposure can result in acute narcosis, which means that lethality occurs before thresholds for sublethal toxicity endpoints such as reduced growth or reproduction in aquatic organisms are reached. Environment Canada has indicated that testing is presently underway to derive thresholds for naphthenic acids, but there are currently no North American guidelines for the protection of aquatic life for naphthenic acids, because of a lack of sufficient chronic toxicity data for exposure to naphthenic acids and their fractionated components.

Most of the studies documented in the literature are for acute effects, including acute toxicity to freshwater fish, invertebrates, and algae; these data show that naphthenic acids are moderately to highly toxic to fish and other aquatic life. Fish are particularly sensitive to the toxic effects of naphthenic acids relative to other aquatic organisms (Nero et al. 2006a). Studies documented by API (2003) and Maxxam (2010) indicate acute toxicity to multiple species in the 1 to 5 mg/L range, including zebra fish (*Brachydanio rerio*) embryos and three-spine sticklebacks (*Gasterosteus aculeatus*), whereas other species have much higher thresholds. Other authors report LC_{50} values for naphthenic acids that range from 4 to 78 mg/L, depending on the species, water hardness, water temperature, length of exposure, and dissolved oxygen concentration (Dokholyan and Magomedov 1983; Verbeek et al. 1993). The age of the process affected water is also an important consideration because a portion of the naphthenic acids readily degrade (Herman et al. 1994; MacKinnon and Boerger 1986; Holowenko et al. 2002; Nero et al. 2006a,b). The degradation of naphthenic acids results in lower toxicity, as shown by studies of fresh versus aged tailings pond water (Holowenko et al. 2002).

Total naphthenic acids can be classified as two fractions, i.e., labile (degradable) naphthenic acids and refractory (non-degradable) naphthenic acids and as discussed below, these fractions have markedly different toxicity characteristics. The water quality assessment predicted total naphthenic acid concentrations as well as the concentrations of labile and refractory naphthenic acids.

The American Petroleum Institute (API 2003) determined that additional studies would be necessary to adequately characterize the hazard of naphthenic acids, and proposed toxicity testing of fish, aquatic invertebrates, and algae to address the potential aquatic toxicity of these materials. Currently, there is insufficient information to develop a chronic effect benchmark using the SSD method; therefore benchmarks must be developed from consideration of the most sensitive individual endpoints documented in published

studies. Separate benchmarks are proposed for labile and refractory fractions of the total naphthenic acid mixture. The available chronic toxicity information for labile and refractory naphthenic acids is discussed below.

Relative Toxicity of Labile and Refractory Naphthenic Acids

The labile, lower molecular weight fraction is primarily responsible for whole effluent toxicity, and is selectively degraded from mixtures of naphthenic acids during aerobic biodegradation (Clemente et al. 2004; Holowenko et al. 2002). Due to the changes in the composition of naphthenic acid mixtures, the most relevant toxicity endpoints for labile naphthenic acids are derived from studies of oil sands process-affected water. Oil sands process-affected water incorporates the degradation of the mixture (i.e., reduction in the labile fraction), in contrast to the fresh commercial mixtures where degradation has not occurred.

A sensitive study of naphthenic acid toxicity to freshwater fish was conducted by Nero et al. (2006a). This study entailed exposure of yellow perch to naphthenic acids extracted from both oil sands process-affected water (ENA) and commercial mixtures (CNA) over a chronic exposure period. At sublethal concentrations (0.9 mg/L CNA; 1.7 mg/L ENA) the study yielded elevated incidence of gill anomalies, and these effect concentrations were four-fold lower than those causing acute toxicity (Nero et al. 2006a). The CNA mixture was observed to be more acutely toxic than the ENA mixture, which is expected because no aging/degradation from the parent mixture had occurred. The results from Nero et al. (2006a) also indicated that young of the year yellow perch exposed to laboratory conditions were more sensitive than similar fish exposed in the field, based on comparison to a study of adult yellow perch and goldfish exposure to reclaimed waters with elevated levels of naphthenic acids (24 mg/L) and salinity (Nero et al. 2006a, b). The authors postulate that the differing sensitivity may be attributed to differences in chemical composition attributable to the age of the oil sands process-affected waters – the ENA used for the laboratory study was extracted from relatively fresh oil sands tailings relative to the naphthenic acids used in the field studies, which had naturally degraded for about 12 years (Nero et al. 2006a). Thus the relatively fresh oil sands process-affected water used in the Nero et al. (2006a) study would still be expected to have a high proportion of the more toxic, labile naphthenic acids.

Other studies have also indicated lower toxicity of naturally degraded naphthenic acids. For example, Kavanagh et al. (2011) demonstrated that oil sands process-affected waters (Syncrude) that had been aged for over 15 years adversely affected the reproductive physiology of fathead minnows when naphthenic acid concentrations were greater than 25 mg/L. However, in the same study, reproduction was not impaired in oil sands process-affected waters with 19.2 mg/L total naphthenic acid.

Proposed Chronic Effects Benchmarks

The preferred toxicological endpoints in aquatic assessments are survival, growth, reproduction and development. If the gill anomalies documented by Nero et al. (2006a, b) are conservatively assumed to be ecologically relevant developmental effects, the 0.9 mg/L concentration represents the lowest threshold concentration identified in the literature for total naphthenic acid mixtures containing a high proportion of labile naphthenic acids. However, this concentration may be over-conservative because it was based on a commercial mixture, rather than an extract from actual oil sands tailings for which a higher threshold of 1.7 mg/L was determined. To account for this uncertainty, the 0.9 mg/L threshold was rounded up to 1 mg/L to provide a conservative threshold for the labile (more toxic) fraction of the naphthenic acid mixture.

Although there are fewer data available for developing a CEB for refractory naphthenic acids, an approximate threshold for refractory naphthenic acids would be 19 mg/L, which is below the no-effect level from Kavanagh et al. (2011) and below the effects levels of 24 mg/L observed by Nero et al. (2006b) and 25 mg/L observed by Kavanagh et al. (2011) using aged samples.

The 1 mg/L threshold for labile naphthenic acids is considered appropriate for screening of total and labile naphthenic acids while the 19 mg/L threshold is considered applicable to refractory naphthenic acids.

2.8.3 Sulphide

Hydrogen sulphide can be highly toxic in the short term to aquatic life. Long-term exposure of fish to sub-lethal levels can cause slower growth, increase in mortality, and reduction in fecundity. Wang and Chapman (1999) summarize aquatic toxicity data for sulphides to benthic invertebrates. The most sensitive endpoint evaluated was Oseid and Smith (1974); it measured mortality in a long-term (65 to 105 day) exposure to freshwater amphipods of the genus *Gammarus*. The chronic no effect level was 2 µg/L, whereas other studies reported thresholds for toxicity that were at least 10-fold greater. As such, 2 µg/L represents a conservative CEB screening threshold, particularly given that chronic elevated exposures of sulphide in the water column are unlikely to be maintained given that sulphides are rapidly volatilized.

2.8.4 Dissolved Solids

Total Dissolved Solids (TDS) is a measure of the combined content of all inorganic and organic constituents contained in a liquid, including molecular, ionized and colloidal suspended matter. The principal inorganic anions dissolved in water include carbonates, chlorides, sulphates and nitrates, and the principal cations are sodium, potassium, calcium and magnesium (IDNR 2003).

The operational definition of TDS is that the solids must be small enough to survive filtration through a sieve the size of 2 µm. Total dissolved solids are distinct from Total Suspended Solids (TSS), in that the latter cannot pass through a sieve of 2 µm and yet remain suspended in solution.

The CCME (2002) provides water quality guidelines for the protection of aquatic life, but these guidelines apply to turbidity and TSS, rather than TDS. A Canadian TDS guideline for drinking water quality of 500 mg/L exists (Health Canada 2007), but it is for protection of aesthetic values for human use, not for protection of aquatic life. For aquatic life endpoints, several jurisdictions in the United States report thresholds ranging from 250 to 2,500 mg/L. For example, the Iowa Department of Natural Resources recently repealed a TDS standard of 1,000 mg/L for aquatic life (IDNR 2009). The standard was repealed in favour of guidelines for specific ions such as chloride and sulphate.

2.8.4.1 Literature Review

The most ecologically relevant studies that were reviewed for this report and were related to the effects of mining-related TDS on freshwater aquatic life are for elevated TDS levels in the vicinity of two northern Canadian mines.

Red Dog Mine, Alaska

A series of TDS investigations were conducted at the Red Dog Mine in Alaska (Brix and Grosell 2005). These studies were conducted based on questions regarding the ecological relevance of the site-specific water quality standards for Mainstem Red Dog Creek of:

- 1,500 mg/L TDS during non-spawning periods of salmonids; and
- 500 mg/L TDS during spawning based on State of Alaska Water Quality Regulations.

The Brix and Grosell (2005) studies emphasized fertilization effects on two fish species (Arctic grayling [*Thymallus arcticus*] and Dolly Varden char [*Salvelinus malma*]) across a gradient of TDS exposures. The test was an early life stage (embryo) test of up to 72 hours duration, which was considered long enough to assess successful fertilization for this sensitive life stage. Previous work by Stekoll et al. (2003a, b) considered fertilization success, embryo development, hatching success, and larval growth and survival, but indicated that fertilization success was the most sensitive endpoint of those evaluated despite the short test duration.

For an EC_{20} and the reproductive endpoint, a geometric mean of available toxicity values was calculated by Brix and Grosell (2005) to derive a species mean value. Species mean values of 1,357 and 1,779 mg/L TDS were estimated for Arctic grayling and Dolly Varden, respectively. On this basis, the authors concluded that the current site-specific limit of 1,500 mg/L TDS (or a value near it), is appropriate for the protection of this endpoint in salmonids.

Snap Lake Mine, Northwest Territories

Golder (2011) provides a detailed literature review of the effects of TDS on freshwater aquatic biota. The review was conducted as follow up to Golder (2003), which reported on potential effects of increased TDS on aquatic communities in Snap Lake. The results of the literature review indicate that freshwater fish and benthic invertebrates can tolerate exposures to TDS that substantially exceed the lower-bound guideline of 500 mg/L TDS from the State of Alaska Water Quality Regulations (a value commonly used as a conservative screening value).

The literature review identified a species geomean for the most sensitive fish species (for chronic tests in the review) of 1,000 mg/L TDS. The literature review also indicates that freshwater benthic invertebrates are generally tolerant of TDS at this higher concentration. The only taxa group that indicated some adverse responses at 1,000 mg/L TDS was zooplankton, and the sensitivity of the various taxa was highly variable.

Other Studies of Total Dissolved Solids Toxicity

Weber-Scannell and Duffy (2007) reviewed available literature to compile data on toxicity relating TDS to fish, invertebrates and plants/algae. The review indicates that toxicity to freshwater aquatic life is variable and depends on species and ionic composition. The observed toxicity of ions to freshwater aquatic life at concentrations below 1,000 mg/L was generally associated with the more toxic ions such as chloride and potassium, which are not applicable to environmental mixtures that typify the source waters for PRM. Moreover, many of the reviewed studies investigated the toxicity of a single constituent (or ion) of TDS (e.g., Ca^{2+}), and therefore many of the reported effect concentrations represent a proportional concentration of the total TDS concentration.

Ketola et al. (1988) investigated the effect of waters with high concentrations of gypsum (calcium sulphate) on survival of salmonid embryos during the water hardening stage of embryo development. The authors found that very hard water containing high concentrations of calcium (520 mg/L or greater), which corresponds to a TDS concentration of approximately 1,750 mg/L, significantly reduced egg survival of Atlantic salmon, brook trout, and rainbow trout. Survival of trout eggs was significantly increased when the high-gypsum water was treated to reduce the calcium content. This treatment did not reduce the sulphate content of the water, which was approximately 1,000 mg/L.

A compilation of available TDS freshwater toxicity data is provided in Table 2.8-1.

Table 2.8-1 Summary of Effects of Total Dissolved Solids on Freshwater Aquatic Biota

Species Scientific Name	Effects Concentration [mg/L]	Endpoint Details	Dominant TDS Components	Source
Phytoplankton				
Nitzschia linearis	3,200	120-hr EC_{50} for growth	$CaSO_4$	Patrick et al. 1968
Selenastrum capriconutum	>2,000	96-hr NOEC for growth	TDS (50% sulphate)	Chapman et al. 2000
Selenastrum capriconutum	551	72-hr EC_{20} for growth	TDS (70% sulphate)	LeBlond and Duffy 2001
Invertebrates				
Ceriodaphnia dubia	1,692	LC_{50}	not specified	Weber-Scannell and Duffy 2007
Ceriodaphnia dubia	>1,913	48-hr LC_{50}	$CaSO_4$	Mount et al. 1997
Ceriodaphnia dubia	1,139	6-7 d IC_{50} for reproduction	TDS	Lasier and Hardin 2010
Ceriodaphnia dubia	1,569	6-7 d IC_{50} for reproduction	TDS	Lasier and Hardin 2010
Chironomus tentans	1,134	10-d NOEC for growth	TDS (50% sulphate)	Chapman et al. 2000
Chironomus tentans	2,089	10-d LOEC (growth reduced by 45%); NOEC for survival	TDS (50% sulphate)	Chapman et al. 2000
Chironomus tentans	2,240	10-d NOEC for growth	TDS (50% sulphate)	Chapman et al. 2000
Chironomus tentans	1,220	10-d NOEC for survival	TDS (50% sulphate)	Chapman et al. 2000
Chironomus tentans	1,750	10-d LOEC for survival	TDS (50% sulphate)	Chapman et al. 2000
Chironomus tentans	2,035	10-d LC_{50}	$CaSO_4$	Weber-Scannell and Jacobs 2001
Chironomus tentans	1,598	IC_{20} for growth	$CaSO_4$	Weber-Scannell and Jacobs 2001
Cyclops abyssorum prealpinus	7,000	EC_{50}	Ca^{2+}	Weber-Scannell and Duffy 2007
Daphnia magna	1,692	LC_{50}	not specified	Weber-Scannell and Duffy 2007
Daphnia magna	>1,968	48-hr LC_{50}	$CaSO_4$	Mount et al. 1997
Daphnia pulex	499	48-hr EC_{50}	Ca^{2+}	Weber-Scannell and Duffy 2007
Hexagenia bilineata	1,230	30-d LOEC (74% survival)	K, Li, Mg, Mo, Na, SO_4, NO_3	Woodward et al. (1985)
Mysidopsis bahia	927	96-hr LC_{50}	Ca^{2+}	Weber-Scannell and Duffy 2007
Tubifex tubifex	814	EC_{50}	Ca^{2+}	Weber-Scannell and Duffy 2007
Fish				
Oncorhynchus mykiss	1,999	7-d NOEC for embryo viability	TDS (70% sulphate)	Chapman et al. 2000
Oncorhynchus mykiss	1,999	7-d NOEC for mortality and growth	TDS (70% sulphate)	Chapman et al. 2000
Oncorhynchus mykiss	2,080	7-d NOEC for mortality and growth	TDS (50% sulphate)	Chapman et al. 2000

Table 2.8-1 Summary of Effects of Total Dissolved Solids on Freshwater Aquatic Biota (continued)

Species Scientific Name	Effects Concentration [mg/L]	Endpoint Details	Dominant TDS Components	Source
Oncorhynchus mykiss	1,500	Mortality	$CaSO_4$	Ketola et al. 1988
Pimephales promelas	>1,968	96-h LC_{50}	$CaSO_4$	Mount et al. 1997
Pimephales promelas	2,270	30-d LOEC (88% survival)	K, Li, Mg, Mo, Na, SO_4, NO_3	Woodward et al. (1985)
Salmo salar	1,500	Mortality	$CaSO_4$	Ketola et al. 1988
Salvelinus fontinalis	2,220	Mortality	$CaSO_4$	Ketola et al. 1988
Salvelinus malma	>1,779	SMV of 24-h EC_{20} for reproduction (fertilization)	TDS ($CaSO_4$)	Brix and Grosell 2005
Salvelinus malma	964	14-h LOEC for reproduction (water absorption)	TDS ($CaSO_4$)	Brix et al. 2010
Salvelinus malma	585	14-h NOEC for reproduction (water absorption)	TDS ($CaSO_4$)	Brix et al. 2010
Thymallus arcticus	1,357	SMV of 72-h EC_{20} for reproduction (fertilization)	TDS ($CaSO_4$)	Brix and Grosell 2005
Thymallus arcticus	1,402	14-h LOEC for reproduction (water absorption)	TDS ($CaSO_4$)	Brix et al. 2010
Thymallus arcticus	784	14-h NOEC for reproduction (water absorption)	TDS ($CaSO_4$)	Brix et al. 2010
Salvelinus malma	585	14-h NOEC for reproduction (water absorption)	TDS ($CaSO_4$)	Brix et al. 2010

Notes: EC_x = effective concentration to x% of test organism, IC_x = inhibiting concentration to x% of test organism, LC_x = lethal concentration to x% of test organism, LOEC = lowest observed effect concentration, NOEC = no observed effect concentration, SMV = species mean value.

2.8.4.2 Chronic Effects Benchmark Development

Because the data on TDS effects to aquatic life reflect a wide range of water quality types (i.e., compositions of major ions differ greatly among the reported experiments), it is not considered appropriate to develop an SSD-based CEB for this parameter. Mount et al. (1997) found that, when multiple cations are present, they tend to be less toxic than comparable exposures with a single dominant cation; therefore, many of the individual results from Table 2.8-1 would overstate TDS effects from the mixtures found in the PRM receiving environment. As an alternative to the SSD method, a value of 1,000 mg/L TDS has been selected as the CEB based on the weight of evidence from the above information, as summarized below.

The literature review of fish and benthic invertebrate toxicity indicates that significant adverse effects are generally not expected for these species at concentrations below 1,000 mg/L TDS. The review indicates that potential effects to sensitive algal species (*Pseudokirchneriella subcapitata*, formerly *Selenastrum capricornutum*) can sometimes be observed below 1,000 mg/L TDS. However, these responses are not consistently observed in experiments (Chapman et al. 2000; Weber-Scannell and Duffy 2007). This species was also included in the toxicity program conducted by Nautilus Environmental Inc. to test sulphate toxicity, and was found to be less sensitive than other freshwater aquatic species tested (Elphick et al. 2011). Potential toxicity to algae would likely be mitigated by the chemical composition of the site-specific TDS mixture, and the relative tolerance of other algae (i.e., a viable community would be present even at 1,000 mg/L). Effects to invertebrates were also observed below 1,000 mg/L, but these toxicity studies focused on the calcium ion, and a simple stoichiometric conversion would result in an effects concentration for TDS that would be far above the CEB. The LOEC of 964 mg/L in salmonid embryos reported by Brix et al. (2010) is close to the proposed CEB and as discussed previously is regarded as a conservative estimate of the benchmark for toxicity to sensitive fish toxicity

endpoints. This revised screening concentration exceeds the maximum TDS estimate for all nodes at all snapshots. Therefore, the predicted changes in TDS levels in the LSA, resulting from either PRM or approved developments, would have a negligible effect on aquatic health. This conclusion applies over the operational phase of PRM and into the Far Future.

One of the factors influencing the toxicity of the TDS mixture is the composition of major ions. The principal inorganic anions dissolved in water include carbonates, chlorides, sulphates and nitrates, and the principal cations are sodium, potassium, calcium and magnesium (IDNR 2003). Some patterns of relative toxicity of different ions to freshwater biota have been observed, with a general decrease in toxicity observed across the following ions: potassium > carbonate = magnesium > chloride > sulphate > calcium = sodium (IDNR 2009). For this reason, jurisdictions including the Iowa Department of Natural Resources have moved toward assessment of these specific toxic ions. Importantly, the composition of source waters for PRM indicates relatively low proportions of these ions. Based on the composition of source waters, which includes a combination of natural watercourses and process-affected water, the composition of this water during the peak concentration would be about: 10% sodium, 10% calcium, 40% bicarbonate, 30% sulphate and 10% other minor constituents. The sodium, calcium and bicarbonate ions (comprising 60%) are not expected to be as toxic as the sulphate and chloride portions of TDS that comprise a minority of the mixture.

In conclusion, the CEB of 1,000 mg/L TDS provides suitable protection of the aquatic community based on the site-specific composition of TDS in the receiving environment of PRM.

2.8.5 Sulphate

The derivation of a CEB for sulphate was based on the recently updated BC WQG document (Mays and Nordic 2013), which is considered a robust and up-to-date assessment of sulphate toxicity. Although other data are available in the literature, the toxicity tests used to develop the BC WQG explicitly evaluated hardness-dependence of sulphate toxicity, used current toxicity testing protocols, and evaluated a range of freshwater species relevant to sensitive species in the receiving waters of the LSA. Accordingly, the information used to develop the proposed WQGs included the following three suites of recent tests conducted specifically to address uncertainties from previous work:

- Tests conducted by Environment Canada at the Pacific Environment Science Centre; these tests included seven species of representative fish, invertebrates, algae, and amphibians.
- Tests conducted at Simon Fraser University by Dr. Chris Kennedy and colleagues; these tests included additional replication of a sensitive reproductive endpoint in rainbow trout.
- Tests conducted by Nautilus Environmental Inc. and subsequently published by Elphick et al. (2011); these tests included nine species and representation of invertebrates, fish, algae, moss, and an amphibian.

The above toxicity data were evaluated by Dr. Carl Swartz at Simon Fraser University, and maximum likelihood estimation methods were used to fit various models, including incorporation of hardness effect as applicable. The outcome was model-averaged estimates of benchmark concentrations of sulphate (mg/L SO_4^{2-}) for multiple hardness levels and levels of effect. These model averaged estimates were used as the basis of proposed WQGs by Meays and Nordin (2013). Meays and Nordin (2013) recognized that rainbow trout (21-d embryo to alevin life stages) are sensitive to sulphate exposures and, like many other species tested, they provide

evidence of amelioration of sulphate toxicity with increasing water hardness. The guidelines developed by Meays and Nordin (2013) are:

- very soft water (0-30 mg/L $CaCO_3$) - 128 mg/L SO_4^{2-};
- soft to moderately soft water (31-75 mg/L $CaCO_3$) - 218 mg/L SO_4^{2-};
- moderately soft/hard to hard (76-180) - 309 mg/L SO_4^{2-};
- very hard (181-250) - 429 mg/L SO_4^{2-}; and
- very hard (>250) - need to determine based on site water.

Based on the site-specific hardness of approximately 150 mg/L, the sulphate CEB for PRM would be 309 mg/L.

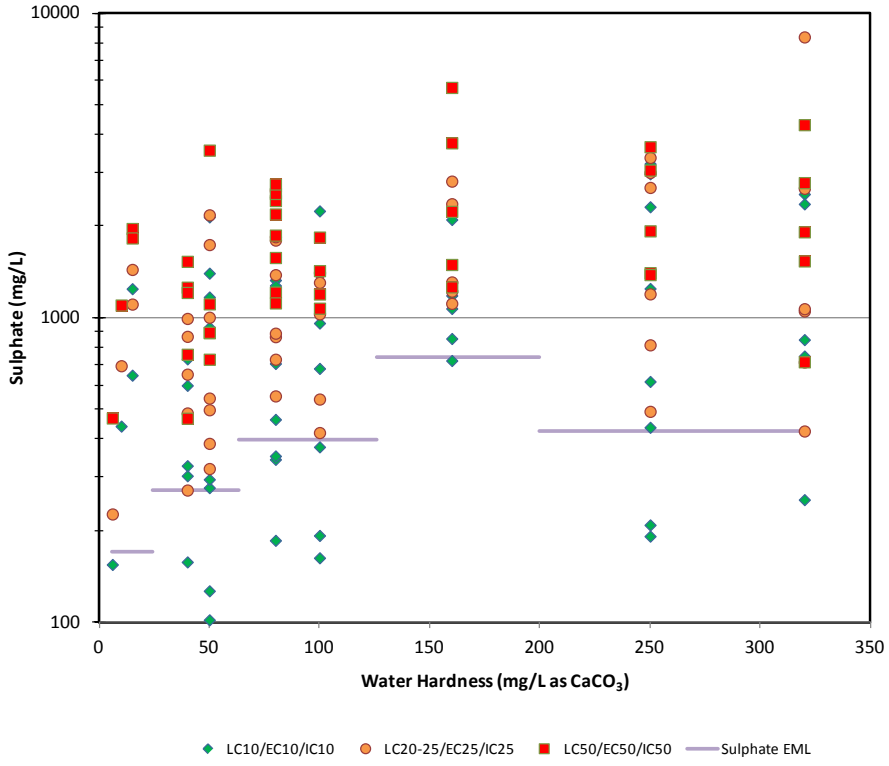
Although the Meays and Nordin (2013) analysis incorporates a number of significant improvements over previous derivations, some limitations remain. Therefore, an alternate derivation was conducted with the following revisions:

- Screening of lower-bound chronic toxicity values - The proposed sulphate WQGs are based on chronic rainbow trout toxicity, with the 30-d average guideline set equal to the 21-d LC_{20} from the 2011 Kennedy study, with the application of a safety factor of 2. Because the rainbow trout endpoint was not the most sensitive test endpoint across all hardness levels, consideration was given to all 20% to 25% effect benchmarks, and the lowest value (irrespective of species) was selected as the candidate benchmark value for each range of hardness conditions. This approach provides a similar level of overall protection for all ranges of hardness.
- Safety factor approach - The application of a universal safety factor of 2 for all hardness levels, while pushing the WQG below the lowest value of the 20% to 25% effect benchmarks (required for protection of biological endpoints), is arbitrary and does not address the variability in the sensitive test endpoints. Furthermore, although Meays and Nordin (2013) correctly identified that many of the 10% effect concentrations have wide confidence bounds, there are some cases where endpoints exhibit confidence bands that have suitable precision for benchmark development. Accordingly, where available, reliable (precise) EC_{10} results were used explicitly as an alternative to the safety factor approach. Specifically, the effects benchmark was selected as the lower of the following: (a) lowest effect benchmark representing a 20% to 25% effect size within each hardness range; or (b) the upper confidence limit of an effect benchmark representing a 10% effect size within each hardness range.
- Break-points between hardness levels - The selection of hardness levels representing transitions between categories of hardness do not reflect the pattern of hardness-dependence in the underlying data. To address this limitation, the effects results data were grouped into hardness categories based on the clustering of effects data, and using the geometric means between tested hardness levels to establish break-points between five hardness categories.

- Treatment of high hardness levels - The BC WQGs do not provide a basis for screening of sulphate concentrations associated with high hardness conditions (greater than 250 mg/L). To address this limitation, test results from very high hardness conditions were aggregated, including the 320 mg/L $CaCO_3$ treatments that were excluded in the draft BC WQG derivation. Consideration of potential osmotic stress at higher sulphate concentrations was also considered through screening to TDS benchmarks

A revaluation of the underlying toxicity data, showing the alternate CEBs in relation to the distribution of chronic effects data is provided in Figure 2.8-1. Based on the hardness conditions in the LSA, the corresponding alternate CEB is 743 mg/L SO_4^{2-}. Accordingly, the CEB for sulphate is expressed as a range between 309 mg/L SO_4^{2-} (BC guideline based) and 743 mg/L SO_4^{2-} (based on observed effect sizes).

Figure 2.8-1 *Model-averaged Sulphate Toxicity Endpoints From Three Investigations of Sulphate Toxicity and Relationship to Water Hardness With Comparison to Candidate Environmental Management Levels for Sulphate*



Source: Meays and Nordin 2013.

2.8.6 Total Phenolics

Phenols are organic compounds that contain one or more hydroxyl groups and other substituents on an aromatic ring, and have been described using various terms, i.e., phenol, phenols, total phenols, phenolics, total phenolics, and phenolic compounds (Breton et al. 2003). The term phenols relates to a suite of related chemicals including monohydric phenols (including phenol, cresols and xylenols) and dihydric phenols (including catechols, resorcinol and quinol). However, the majority of toxicity data available for guideline derivation are for phenol.

2.8.6.1 Background and Guidelines

This constituent is considered to be more toxic than other phenolic constituents; therefore the assumption of additive toxicity for phenols is conservative. Phenols are highly soluble in water and are not expected to significantly bioconcentrate in aquatic biota, but are subject to photo-oxidation, oxidation, and microbial degradation processes. The toxicity of phenols to freshwater biota can be affected by pH, temperature, dissolved oxygen, and water hardness (CCME 1999c).

Industrial and municipal effluents represent major sources of phenols in the Canadian environment, while trace concentrations are naturally released to the aquatic environment through the natural decomposition of aquatic vegetation (CCME 1999c). Phenols have a short half-life so the highest concentrations are typically reported close to an effluent discharge or the natural phenol source. Concentrations of phenols in surface waters not influenced by anthropogenic activities are typically < 2 µg/L (Breton et al. 2003; CCME 1999c).

The proportion of monohydric phenols (including phenol, cresols and xylenols) and dihydric phenols (including catechols, resorcinol and quinol) present as total phenols, will affect toxicity, given that the various mono- and dihydric phenols report differential toxicity (Devi and Sastry 1987). Phenol is considered to be more toxic than other phenolic constituents and is the phenolic substance for which the most toxicity data are available. The CCME WQG for the protection of freshwater aquatic life for phenols (mono- and dihydric phenols) is 4.0 µg/L (CCME 1999c). The guideline is derived by multiplying the 9-d LC_{50} of 40 µg/L for the leopard frog (Birge et al. 1980b) by a safety factor of 0.1 (CCME 1999c). This test was based on the embryo larval stage of the leopard frog, which was found to be the most sensitive receptor to phenols. Fish were less sensitive to phenols with 27-d LC_{50}s of 0.07 and 0.12 mg/L reported for rainbow trout by Birge et al. (1979) and Milleman et al. (1984).

2.8.6.2 Chronic Effect Benchmark Development

As one of the constituents listed on the second Canadian Priority Substances List, phenol underwent an probabilistic ecological risk assessment by Breton et al. (2003) to determine the potential for harmful effects on the environment, the environment on which human life depends, or human health. As part of this assessment, Breton et al. (2003) selected a Critical Toxicity Value (CTV) that represented a "*quantitative expression (e.g., IC_{25}) of low toxic effect on the measurement endpoint*", based on the available toxicological information concerning the chronic toxicity of phenol to freshwater aquatic biota. Rainbow trout were identified as the most sensitive aquatic species, and so the CTV selected by Breton et al. (2003) was the 27-day LC_{25} of 0.01 mg/L for the embryo-larval stage of rainbow trout (Birge et al. 1979).

Breton et al. (2003) concluded that the concentration-response curves for the early life-stage of rainbow trout was similar to that of the early life-stage of leopard frog, and so the probabilistic risk analysis would be expected to yield similar results for both species. A rainbow trout CTV was selected over a leopard frog CTV, because rainbow trout appeared to be more sensitive to phenol exposure at lower concentrations.

Breton et al. (2003) also estimated exposure values for phenol in the receiving waters of industrial and municipal discharges. Consideration was given to the amount of phenol in the total phenolics measurement typically reported in the assessment of receiving water quality. Breton et al. (2003) estimated that the amount of phenol in the total phenolics measurement to be 11% for the petroleum refining and products sector. For CEB development, no application factor has been applied to the rainbow trout CTV of 0.01 mg/L phenol, in recognition that the estimated percentage of phenol in the total phenolics measurement in likely to be low. Furthermore, the environmental half-lives of phenols are short such that the highest exposures will occur in receiving waters near point sources. Concentrations of phenols are expected to decrease in the receiving environment quickly due to bacterial breakdown.

Based on the above information, the rainbow trout CTV of 0.01 mg/L phenol, has been adopted as the CEB for total phenols.

2.9 Selected Chronic Effects Benchmarks

The CEBs developed for PRM, along with the derivation basis for the selected value, are summarized in Table 2.9-1.

Table 2.9-1 Chronic Effects Benchmarks Used in the Aquatic Health Assessment

Constituent	Units	Chronic Effects Benchmark		Basis for Benchmark
		Environmental Impact Assessment	August 2013 Pierre River Mine[(a)]	
Metals				
aluminum	mg/L	0.680	0.150	Most sensitive test endpoint from aluminum toxicity studies conducted in pH and hardness conditions applicable to the site (MATC for survival of goldfish exposed through egg-to-fry life stages). No safety factor applied because of anomalous nature of toxicity data from Birge (1978) and Birge et al. (1980a,b).
antimony	mg/L	0.580	0.157	Lowest reported toxicity value (28-day LC_{10} with rainbow trout) from Birge et al. (1979)
arsenic	mg/L	0.191	0.025 to 0.029	HC_5 from updated chronic SSD with 24 species, which fully satisfied the requirements of CCME (2007a). Assumes that chemical speciation would be dominated by arsenate.
barium	mg/L	5.800	5.800	Lowest reported toxicity value (21-day EC_{16} for reproduction with *Daphnia magna*) from Biesinger and Christensen (1972)
beryllium	mg/L	0.0053	0.0053	Lowest reported toxicity value (28-day MATC for reproduction with *Daphnia magna*) from Kimball (1978)
boron	mg/L	8.157	1.500	HC_5 from SSD (CCME 2009)
cadmium	mg/L	0.00042	0.00025 to 0.00062	Draft Revised CCME (2012) Guideline for Protection of Aquatic Life, freshwater, long-term exposure guideline - using hardness of 150 mg/L. Upper value is species mean chronic value for most sensitive species, adjusted to hardness of 150 mg/L.
chromium (III)	mg/L	0.00248	0.089	The lowest reported toxicity value of 89 µg/L was selected for use as the CEB for Cr^{3+}. This value is based on a LOEC for survival of rainbow trout embryos exposed until 30-day post-swim-up (Stevens and Chapman 1984).
chromium (VI)	mg/L	-	0.0083	HC_5 from SSD (this study)

Table 2.9-1 Chronic Effects Benchmarks Used in the Aquatic Health Assessment (continued)

Constituent	Units	Chronic Effects Benchmark		Basis for Benchmark
		Environmental Impact Assessment	August 2013 Pierre River Mine[(a)]	
cobalt	mg/L	0.0093	0.004	Recommended guideline from BC MWLAP (2004) based on sensitive invertebrate test endpoints.
copper	mg/L	0.073	0.0256	U.S. EPA (2007b) biotic ligand model (BLM) predictions normalized to water chemistry conditions specified for PRM, with application of acute-to-chronic ratio 3.22.
iron	mg/L	0.57	1.50	Weight of evidence from multiple lines of evidence, including HC_5 from SSD fit to ferric iron toxicity data, toxicity test data from experiments conducted by the BC Ministry of Environment (Phippen et al. 2008), and field based assessments (Brenner et al. 2004; Linton et al. 2007)
lead	mg/L	-	0.005	Hardness-based CCME guideline (assuming 143 mg/L $CaCO_3$)
manganese	mg/L		1.46	Species sensitivity distribution using chronic toxicity data from five species)
mercury	mg/L	-	0.00005	HC_5 from SSD (this study)
mercury (invertebrate)	mg/kg ww tissue	-	3.0	This study (concentration-response analysis)
mercury (fish)	mg/kg ww tissue	-	0.5	This study (concentration-response analysis)
molybdenum	mg/L	0.703	38.7	HC_5 from SSD (this study)
nickel	mg/L	-	0.125	Hardness-adjusted water quality guideline value of 125.4 µg/L CCME (1999a).
silver	mg/L	0.00017	0.00022	HC_5 from SSD (this study)
strontium	mg/L	0.2	14.1	HC_5 from revised SSD incorporating new literature data and results of recent testing on strontium toxicity to rainbow trout early life stages and *Hyalella azteca* (Nautilus 2013).
vanadium	mg/L	0.16	0.0338	Lowest reported toxicity value (28-day LC_{10} with rainbow trout) from Birge et al. (1979, 1980a)
zinc	mg/L	-	0.138	HC_5 from SSD (this study)
PAH Components				
PAH Group 1	µg/L	0.15	0.281	McGrath and DiToro (2009) benzo(a)pyrene HC_5
PAH Group 2	µg/L	0.18	0.278	McGrath and DiToro (2009) 7,12-dimethylbenzo(a)anthracene HC_5
PAH Group 3	µg/L	0.07	0.99	McGrath and DiToro (2009) chrysene HC_5
PAH Group 4	µg/L	-	41.5	McGrath and DiToro (2009) acenaphthene HC_5
PAH Group 5	µg/L	0.12	5.6	Anthracene HC_5 from SSD (this study)
PAH Group 6	µg/L	29	64	McGrath and DiToro (2009) biphenyl HC_5
PAH Group 7	µg/L	0.4	5.9	Fluoranthene HC_5 from SSD (this study)
PAH Group 8	µg/L	-	32	Naphthalene HC_5 from SSD (this study)
PAH Group 9	µg/L	-	2.3	Pyrene HC_5 from SSD (this study)
Other Components				
ammonia	mg/L N	-	2.0	CCME (2010), using the site-specific summer water temperature (15°C) and pH (7.5)
naphthenic acids - refractory	mg/L	-	19	No-effect level from Kavanagh et al. (2011) for process-affected waters
naphthenic acids – labile	mg/L	-	1.0	Conservative CEB based on lower bound of chronic toxicity to freshwater organisms from aged oil sands process waters

Table 2.9-1 Chronic Effects Benchmarks Used in the Aquatic Health Assessment (continued)

Constituent	Units	Chronic Effects Benchmark		Basis for Benchmark
		Environmental Impact Assessment	August 2013 Pierre River Mine[(a)]	
sulphide	mg/L	-	0.002	Most sensitive chronic no-effect level from literature review by Wang and Chapman (1999)
sulphate	mg/L	-	309 to 743	BC sulphate water quality guideline (Meays and Nordin 2013) for moderately hard to hard water conditions; also, recalculation of benchmark for hard water conditions, based on model-averaged sulphate toxicity endpoints from three investigations of sulphate toxicity and relationship to water hardness.
total dissolved solids	mg/L	-	1,000	Meta-analysis of TDS toxicity data for soft and moderately hard waters
total phenolics	mg/L	-	0.01	Breton et al. (2003) selected a critical toxicity value (CTV) based on the available toxicological information concerning the chronic toxicity of phenol to freshwater aquatic biota. Rainbow trout were identified as the most sensitive aquatic species, and the CTV was the 27-day LC_{25} of 0.01 mg/L for the embryo-larval stage of rainbow trout (Birge et al. 1979).

(a) CEBs for Pierre River Mine developed assuming water quality characteristics representative of PRM receiving waters. From the May 2012 submission, the PRM Local Study Area (LSA) medians were pH = 7.5; temperature = 5°C; hardness = 143 mg/L $CaCO_3$, and DOC = 21.8 mg/L.

Note: Substances marked in grey highlighting indicate revisions from May 2012 submission.

- = Benchmark not derived.

3.0 REFERENCES

AENV (Alberta Environment). 2008. *Muskeg River Interim Management Framework for Water Quantity and Quality*. ISBN 978-0-7785-7630-3. June 2008. Edmonton, AB, Canada. 72 pp.

Aldenberg, T., T.S. Jaworska, and J.P. Traas. 2002. *Normal species sensitivity distributions and probabilistic ecological risk assessment*. In Posthuma L, Suter II GW, Traas TP (eds), Species Sensitivity Distributions in Ecotoxicology. CRC Press, Boca Raton, FL, USA, pp 49-102.

Allred, P.M. and J.P. Giesy. 1985. *Solar radiation-induced toxicity of anthracene to* Daphnia pulex. Environ Toxicol Chem 4:219–226.

API (American Petroleum Institute). 2003. *High Production Volume (HPV) Chemical Challenge Program: Test Plan for Reclaimed Substances: Streams Containing Naphthenic Acids, Phenolics, Disulfides, Acids or Caustics.* Submitted to the U.S. EPA by the American Petroleum Institute, Petroleum HPV Testing Group, Washington, DC, USA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. *Toxicological profile for polycyclic aromatic hydrocarbons.* United States Department of Health and Human Services. Public Health Service, Atlanta, GA, USA. 458 pp.

ATSDR. 1997. *Toxicological Profiles on CD-ROM.* United States Public Health Service. Atlanta, GA, USA.

ATSDR. 1999. *Toxicological profile for mercury.* United States Department of Health and Human Services. Public Health Service, Atlanta, GA, USA. 610 pp.

ATSDR. 2005a. *Toxicological profile for nickel.* United States Department of Health and Human Services. Public Health Service, Atlanta, GA, USA. 351 pp.

ATSDR. 2005b. *Toxicological profile for zinc.* United States Department of Health and Human Services. Public Health Service, Atlanta, GA, USA. 607 pp.

ATSDR. 2008. *Toxicological profile for aluminum.* United States Department of Health and Human Services. Public Health Service, Atlanta, GA, USA. 310 pp.

Baker, J.L., A.C. Barefoot, L.E. Beasley, L. Burns, P. Caukins, J. Clark, R.L. Feulner, J.P. Giesy, R.L. Graney, R. Griggs, H. Jacoby, D. Laskowski, A. Maciorowski, E. Mihaich, H. Nelson, R. Parrish, R.E. Siefert, K.R. Solomon, W. van der Schalie. 1994. *Final Report: Aquatic Risk Assessment and Mitigation Dialog Group.* SETAC Press. Pensacola, FL, USA.

BC MWLAP (British Columbia Ministry of Water, Land and Air Protection). 2004. *Technical Report – Water Quality Guidelines for Cobalt*. BC MWLAP. Victoria, BC, Canada. Available at: http://www.env.gov.bc.ca/wat/wq/BCguidelines/cobalt/cobalt_tech.pdf

Beckvar, N., T.M. Dillon and L.B. Read. 2005. *Approaches for Linking Whole-body Fish Tissue Residues of Mercury or DDT to Biological Effect Thresholds*. Environ Toxicol Chem 24(8):2094-2105.

Biesinger, K.E. and G.M. Christensen. 1972. *Effects of various metals on survival, growth, reproduction and metabolism of Daphnia magna*. J Fish Res Board Can 29:1691-1700.

Birge, W.J. 1978. *Aquatic toxicology of trace elements of coal and fly ash*. In J.H. Thorp, J.W. Gibbons (eds), Energy and Environmental Stress in Aquatic Systems: Selected Papers From a Symposium Held at Augusta, Georgia, November 2-4, 1977. Dep Energy Symp Ser 48:219-240.

Birge, W.J., J.A. Black and A.G. Westerman. 1979. *Evaluation of aquatic pollutants using fish and amphibian eggs as bioassay organisms*. In S.W. Nielsen, G. Migaki, D.G. Scarpelli (eds), Animals as Monitors of Environmental Pollutants. National Academy of Science, Washington, DC, pp 108-118.

Birge, W.J., J.A. Black and A.G. Westerman and J.E. Hudson. 1980a. *Aquatic toxicity tests on inorganic elements occurring in oil shale.* In Gale, C. (ed.)., Oil Shale Symposium: Sampling, Analysis and Quality Assurance. United States Environmental Protection Agency, Cincinnati, OH, USA. pp. 519-534.

Birge, W.J., J.A. Black and R. Kuehne. 1980b. *Effects of organic compounds on amphibian reproduction.* University of Kentucky, Water Resources Institute, Lexington, KY.

Bjornberg A, L. Hakanson and K. Lundbergh. 1988. *A theory on the mechanisms regulating the bioavailability of mercury in natural waters.* Environ Pollut 49:53-61.

Black, J.A., W.J. Birge, A.G. Westerman and P.C. Francis. 1983. *Comparative aquatic toxicology of aromatic hydrocarbons.* Fundam Appl Toxicol 3:353–358.

Boese, B.L., J.O. Lamberson, R.C. Swartz, R. Ozretich R and F. Cole. 1998 *Photoinduced toxicity of PAHs and alkylated PAHs to a marine in faunal amphipod (Rhepoxynius abronius).* Arch Environ Contam Toxicol 34:235-240.

Borgmann, U., R. Cove, abd C. Loveridge. 1980. *Effects of metals on the biomass production kinetics of freshwater copepods.* Can J Fish Aquat Sci 37:567-575.

Borgmann, U, Y. Couillard, P. Doyle and D.G. Dixon. 2005. *Toxicity of sixty-three metals and metalloids to Hyalella azteca at two levels of water hardness.* Environ Toxicol Chem 24:641-652.

Boutet, C. and C. Chaisemartin. 1973. *Specific toxic properties of metallic salts in Austropotamobius pallipes pallipes and Orconectes limosus (Proprietes toxiques specifiques des sels metalliques chez Austropotamobius pallipes pallipes et Orconectes limosu)*. C R Seances Soc Biol Fil 167: 1933-1938.

Brenner, F.J., S. Hedglin, S. Alexander, and S. Busler. 2004. *Chemical and Biological Analysis of Fox Run Watershed, Mercer County, Pennsylvania*. Proceedings from the National Meeting of the American Society of Mining and Reclamation (ASMR), Morgantown, WV, USA.

Breton, R.L., R.S. Teed, and D.R.J. Moore. 2003. *An Ecological Risk Assessment of Phenol in the Aquatic Environment*. Human Ecoll Risk Assess 9:549-568.

Brix, K.V. and M. Grosell. 2005. *Report on the Effects of Total Dissolved Solids on Arctic Grayling and Dolly Varden Fertilization Success.* Prepared for Teck Cominco, Red Dog Mine, Kotzebue, AK by the University of Miami, Miami, FL, USA. Available at: http://www.dec.state.ak.us/water/wqsar/wqs/pdfs/final_Brix_TDS_report.pdf

Brix, K.V., R. Gerdes, N. Curry, A. Kasper and M. Grosell. 2010. *The effects of total dissolved solids on egg fertilization and water hardening in two salmonids – Arctic Grayling (Thymallus arcticus) and Dolly Varden (Salvelinus malma)*. Aquat Toxicol 97:109-115.

Call, D.J., L.T. Brooke, C.A. Lindberg, T.P. Markee, D.J. McCauley, and S.H. Poirier. 1984. *Toxicity of Aluminum to Freshwater Organisms in Water of pH 6.5-8.5*. Technical Report Project No. 549-238-RT-WRD. Prepared for Camp Dresser and Mckee, Annandale, VA, USA.

CCME (Canadian Council of Ministers of the Environment). 1991. *Appendix IX— A protocol for the derivation of water quality guidelines for the protection of aquatic life (April 1991).* In: Canadian water quality guidelines, Canadian Council of Resource and Environment Ministers, 1987. Prepared by the Task Force on Water Quality Guidelines. [Updated and reprinted with minor revisions and editorial changes in Canadian environmental quality guidelines, Chapter 4, Canadian Council of Ministers of the Environment, 1999, Winnipeg, MB]

CCME. 1999a. (with updates to 2011). *Canadian Environmental Quality Guidelines.* Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: http://ceqg-rcqe.ccme.ca/

CCME. 1999b. *Canadian water quality guidelines for the protection of aquatic life: Chromium – hexavalent chromium and trivalent chromium.* In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: http://ceqg-rcqe.ccme.ca/download/en/165/

CCME. 1999c. *Canadian water quality guidelines for the protection of aquatic life: Mono- and dihydric phenols.* In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: http://ceqg-rcqe.ccme.ca/download/en/204/

CCME. 2002. *Canadian water quality guidelines for the protection of aquatic life: Total particulate matter.* In: Canadian Environmental Quality Guidelines, 1999. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. 2002 update. Available at: http://ceqg-rcqe.ccme.ca/download/en/217/

CCME. 2003a. *Guidance on the site-specific application of water quality guidelines in Canada: Procedures for deriving numerical water quality objectives.* In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada.

CCME. 2003b. *Canadian water quality guidelines for the protection of aquatic life: Mercury - Inorganic mercury and methylmercury.* In: Canadian Environmental Quality Guidelines, 1999. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. 2003 update. Available at: http://ceqg-rcqe.ccme.ca/download/en/191/

CCME. 2007a. *A protocol for the derivation of water quality guidelines for the protection of aquatic life 2007.* In: Canadian Environmental Quality Guidelines, Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: ceqg-rcqe.ccme.ca/download/en/220/

CCME. 2007b. *Canadian Water Quality Guidelines for the Protection of Aquatic Life: Summary Table. Update 7.1. December 2007. I*n: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: http://www.ccme.ca/assets/pdf/aql_summary_7.1_en.pdf

CCME. 2009. *Canadian Water Quality Guidelines for the Protection of Aquatic Life: Boron.* In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: ceqg-rcqe.ccme.ca/download/en/324/

CCME. 2010. *Canadian Water Quality Guidelines for the Protection of Aquatic Life: Ammonia.* In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada. Available at: ceqg-rcqe.ccme.ca/download/en/141/

CCME. 2011. *Canadian Environmental Quality Guidelines.* Canadian Water Quality Guidelines for the Protection of Aquatic Life – Copper. Available at: http://st-ts.ccme.ca/?lang=en&factsheet=71#aql_fresh_concentration.

CCME. 2012. *Canadian Water Quality Guidelines for the Protection of Aquatic Life: Cadmium*. Draft Scientific Criteria Document. Canadian Council of Ministers of the Environment. Winnipeg, MB, Canada.

CCREM (Canadian Council of Resource and Environment Ministers). 1987. *Canadian Water Quality Guidelines of the Canadian Council of Resource and Environment Ministers (CCREM)*. Winnipeg, MB, Canada. Prepared by the Task Force on Water Quality Guidelines of the Canadian Council of Resource and Environment Ministers. March 1987.

Chapman, P.M., H. Bailey and E. Canaria. 2000. *Toxicity of total dissolved solids associated with two mine effluents to chironomid larvae and early life stages of rainbow trout*. Environ Toxicol Chem 19: 210-214.

Chapman, P.M., W.J. Adams, M.L. Brooks, C.G. Delos, S.N. Luoma, W.A. Maher, H.M. Ohlendorf, T.S. Presser and D.P. Shaw. 2010. *Ecological Assessment of Selenium in the Aquatic Environment.* Society of Environmental Toxicology and Chemistry (SETAC). Pensacola, FL, USA.

Chowdhury, M.J. and R. Blust. 2011. *Strontium*. In C.M. Wood, A.P. Farrell, C.J. Brauner (eds), Homeostasis and Toxicology of Non-Essential Metals. Volume 31 B, Fish Physiology Series. Elsevier, New York, NY, pp 351-390.

Clemente, J.S., M.D. MacKinnon and P.M. Fedorak. 2004. *Aerobic biodegradation of two commercial naphthenic acids preparations.* Environ Sci Technol 38:1009-1016.

Cook, D. 2008. *Chemical-Specific Toxicity Tests to Calculate Tier II Acute-to-Chronic Ratios (ACRs) for Lithium Chloride (LiCl) and Strontium Chloride ($SrCl_2$) Using Fathead Minnow and Ceriodaphnia dubia*. Unpublished report by Global Environmental Consulting LLC.

Cotton, F.A. and G. Wilkinson (eds). 1980. *Advanced Inorganic Chemistry: A Comprehensive Text.* John Wiley & Sons. New York, NY, USA. 1396 pp.

Davies, P.H., J.P. Goettl and J.R. Sinley. 1978. *Toxicity of silver to rainbow trout (Salmo gairdneri)*. Water Res 12:113-117.

Davies, T.D., J. Pickard and K.J. Hall. 2005. *Acute molybdenum toxicity to rainbow trout and other fish.* J Environ Eng Sci 4:481-485.

De Foe, D.L. 1982. *Arsenic (V) Test Results*. Memorandum to Spehar RL, U.S. EPA, Duluth, MN, USA. July 9, 1982.

De Schamphelaere, K.A.C., W. Stubblefield, P. Rodriguez, K. Vleminckx, C.R. and Janssen. 2010. *The chronic toxicity of molybdate to freshwater organisms. I. Generating reliable effects data*. Sci Tot Environ 408:5362-5371.

Devi, C.R., and C.A. Sastry. 1987. *Toxicity of phenols to fish*. Indian J. Environ. Prot. 7(4):271-283.

Dokholyan, V.K. and A.K. Magomedov. 1983. *Effect of sodium naphthenate on survival and some physiological-biochemical parameters of some fishes*. Journal of Ichthyology. 23(6):125–132.

Ecometrix. 2011. *Suggested Effluent Quality Criteria for the Snap Lake Diamond Mine*. Memorandum from Don Hart and Ian Collins (Ecometrix Inc.) to the MVLWB. October 19, 2011.

Eisler, R. 1987. *Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: A synoptic review.* Laurel, MD: US Fish and Wildlife Service, Patuxent Wildlife Research Center.

Elphick, J.R., M. Davies, G. Gilron, E.C. Canaria, B. Lo and H.C. Bailey. 2011. *An aquatic toxicological evaluation of sulfate: the case for considering hardness as a modifying factor in setting water quality guidelines*. Environ Toxicol Chem 30:247-53.

Environment Canada. 1997. *Biological Test Method: Test for Survival and Growth in Sediment Using the Freshwater Amphipod Hyalella azteca*. Environmental Protection Series EPS 1/RM/33. Method Development and Application Section, Environmental Technology Centre, Ottawa, ON, Canada.

Environment Canada. 1998. *Biological Test Method: Toxicity Tests Using Early Life Stages of Salmonid Fish (Rainbow Trout)*. Second Edition. EPS/1/RM/28. Method Development and Application Section, Environmental Technology Centre, Ottawa, ON, Canada.

EVS (EVS Solutions Inc). 1999. *Review of Tissue Residue Effects Data for Tributylin, Mercury, and Polychlorinated Biphenyls*. Prepared for the Port of Seattle, Lockheed Martin Corporation, Todd Shipyards, Seattle, WA.

Finger, S.E., E.F. Little, M.G. Henry, J.F. Fairchild and T.P. Boyle. 1985. *Comparison of laboratory and field assessment of fluorene: Part I. Effects of fluorene on the survival, growth, reproduction, and behavior of aquatic organisms in laboratory tests.* In: Boyle, TP, ed., Validation and predictability of laboratory methods for assessing the fate and effects of contaminants in aquatic ecosystems, ASTM STP 865. Philadelphia PA.

Freeman, R.A. and W.H. Everhart. 1971. *Toxicity of aluminum hydroxide complexes in neutral and basic media to rainbow trout*. Trans Am Fish Soc 100: 644-658.

GEI (GEI Consultants). 2009. *New molybdenum toxicity data, Appendix A of ambient water quality standards for molybdenum*. Exhibit D of Chevron Mining Inc's notice of intent to present direct testimony of S.P. Canton in the matter of the triennial review of water quality standards for interstate and intrastate waters, 20.6.4 NMAC; 2009.

Gensemer, R.W. and R.C. Playle RC. 1999. *The bioavailability and toxicity of aluminum in aquatic environments*. Crit Rev Env Sci Tec 29: 315-450.

Golder (Golder Associates Ltd.). 2003. *Report on Potential Effects of Increased Total Dissolved Solids on Aquatic Communities in Snap Lake.* Submitted to Department of Fisheries and Oceans Canada and the Mackenzie Valley Environmental Review Board.

Golder. 2011. *Total Dissolved Solids (TDS) Study – Task 1 – Literature Review - Final.* Technical Memorandum prepared by Cathy McPherson and James Dwyer (Golder Associates, Burnaby BC) for Jason Ash (De Beers Canada Inc.). April 6, 2011.

Gunderson, D.T., S. Bustaman, W.K. Seim, and L.R. Curtis. 1994. *pH, hardness and humic acid influence aluminum toxicity to rainbow trout (Oncorhynchus mykiss) in weakly alkaline waters*. Can J Fish Aquat Sci 51:1345-1355.

Guy, R.D. and C.L. Chakrabarti. 1976. *Studies of metal-organic interactions in model systems pertaining to natural waters.* Can J Chem 54:2600-2611.

Hamilton, S.J. and T.A. Haines. 1995. *Influence of fluoride and aluminum toxicity to Atlantic salmon* (*Salmo salar*). Can J Fish Aquat Sci 52:2432-2444.

Harrison, J. 2008. *Benthic macroinvertebrate sensitivity to PAH phototoxicity: patterns and causes.* APS 402 Dissertation. University of Sheffield's Ecotoxicology Group. Candidate no: 040196002. Available at: http://aps.group.shef.ac.uk/level-4-web-sites/l4-students-08/harrison-jesse/aps402-dissertation.pdf

Health Canada. 2007. *Guidelines for Canadian Drinking Water Quality: Chemical and Physical Parameters.* Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/doc_sup-appui/sum_guide-res_recom/chemical-chimiques_e.html#4.

Herman, D.C., P.M. Fedorak, M.D. MacKinnon and J.W. Costernon. 1994. *Biodegradation of naphthenic acids by microbial populations indigenous to oil sands tailings.* Canadian Journal of Microbiology 40(6):467-77.

Hibbins, S.G. 1997. *Strontium and strontium compounds.* In: Kroschwitz, J.I. and M. Howe-Grant (eds.). Kirk-Othmer Encyclopaedia of Chemical Technology, Volume 22. John Wiley & Sons. New York, NY, USA. pp. 947-955.

Hogstrand, C., C.M. Wood. 1998. *Toward a better understanding of the bioavailability, physiology and toxicity of silver in fish: Implications for water quality criteria*. Environ Toxicol Chem 17:547-561.

Holowenko, F.M., M.D. MacKinnon and P.M. Fedorak. 2002. *Characterization of naphthenic acids in oil sands wastewaters by gas chromatography-mass spectrometry.* Water Res 36:2843-2855.

Hou, H and H. Narasaki. 1999. *Differential determination of antimony (III) and antimony (V) in river water by hydride-generation inductively coupled plasma mass spectrometry.* Anal Sci 15:911-914.

Hull, C. 2008. *Rule 57 Aquatic Values Data Sheet*. Michigan Department of Environmental Quality. June 2008. Available at: http://www.epa.gov/Region5/glic/pdfs/ mi_al_487_06162008.pdf. Accessed September 16, 2011.

HydroQual (HydroQual Laboratories Ltd.). 2009. *Aquatic Toxicity Assessment on Strontium*. Reported prepared for Golder Associates Ltd., Calgary, AB, by HydroQual Laboratories Ltd., Calgary, AB. May 8, 2009.

ICME (International Council on Metals and the Environment). 1995. *Persistence, bio-accumulation and toxicity of metals and metal compounds.* ICME. Ottawa, ON, Canada.

IDEM (Indiana Department of Environmental Management). 2001. *Tier II Acute and Chronic Aquatic Life Values. Strontium*. Office of Water Quality, Water Quality Standards Section, Indianapolis, IN, USA.

IDNR (Iowa Department of Natural Resources). 2003. *Issue Paper – Total Dissolved Solids (TDS).* Available at: http://www.albuw.ait.ac.th/Group_R/TDS/report-4/Database/Ref%20materials/ tdsissue.pdf

IDNR. 2009. *Water Quality Standards Review: Chloride, Sulfate and Total Dissolved Solids*. Consultation Package. February 9, 2009. Available at: http://www.dnr.mo.gov/env/wpp/rules/rir/so4-cl-ws_review_idnr_so4-cl.pdf

Jarvinen, A.W. and G.T. Ankley. 1999. *Linkage of Effects to Tissue Residues: Development of a Comprehensive Database for Aquatic Organisms Exposed to Inorganic and Organic Chemicals*. SETAC Technical Publication Series, Pensacola, FL.

Jones, J.R.E. 1939. *The relation between the electrolytic solution pressures of the metals and their toxicity to the stickleback (Gasterosteus aculeatus L.)*. J Exp Biol 16: 425-437.

Jones, J.R.E. 1940. *A further study of the relation between toxicity and solution pressure, with Polycelis nigra as test animal*. J Exp Biol 17: 408-415.

Jonnalagadda, S.B. and P.V. Rao. 1993. *Toxicity, bioavailability and metal speciation.* Comp Biochem Physio Ser C (Pharmacology, Toxicology and Endocrinology) 106:585-595.

Kagan, J, E.D. Kagan, I.A. Kagan, P.A. Kagan and S. Quigley. 1985. *The phototoxicity of non-carcinogenic polycyclic aromatic hydrocarbons in aquatic organisms.* Chemosphere 14:1829–1834.

Kagan, J. and E.D. Kagan. 1986. *The toxicity of benzo(a)pyrene and pyrene in the mosquito Aedes aegypti, in the dark and in the presence of ultraviolet light*. Chemosphere 15:243–251.

Kavanagh, R.J., R.A. Frank, K.D. Oakes, M.R. Servos, R.F. Young, P.M. Fedorak, M.D. MacKinnon, K.R. Solomon, D.G. Dixon and G. Van Der Kraak. 2011. *Fathead minnow (Pimephales promelas) reproduction is impaired in aged oil sands process-affected waters.* Aquat Toxicol 101:214-220.

Ketola, H.G., D. Longacre, A. Greulich, L. Phetterplace and R. Lashomb. 1988. *High calcium concentration in water increases mortality of salmon and trout eggs*. Prog Fish Culturist 50:129-135.

Khangarot, B.S. 1991. *Toxicity of metals to a freshwater tubificid worm*, *Tubifex tubifex* (*Müller*). Bull Environ Contam Toxicol 46: 906-912.

Khangarot, B.S. and P.K. Ray. 1989. *Investigation of correlation between physicochemical properties of metals and their toxicity to the water flea Daphnia magna Straus*. Ecotox Environ Saf 18: 109-120.

Kimball, G. 1978. *The effects of lesser known metals and one organic to fathead minnows (Pimephales promelas) and Daphnia magna.* Department of Entomology, Fisheries and Wildlife, University of Minnesota, USA.

Kramer, J.R., G. Benoit, K.C. Bowles, D.M. DiToro, R.T. Herrin, G.W. Luther III, H. Manolopoulos, K.A. Robillard, M.M. Shafer and J.R. Shaw. 2002. *Environmental Chemistry of Silver*. In: Andren, A.W. and Bober, T.W. (eds.), Silver in the Environment: Transport, Fate and Effects. Research Findings of the Argentum International Conference Series 1993-2000. pp. 1-20.

Lasier, P.J. and I.R. Hardin. 2010. *Observed and predicted reproduction of Ceriodaphnia dubia exposed to chloride, sulphate, and bicarbonate*. Environ Toxicol Chem 29:347-358.

LeBlanc, E.L., L.W. Leger, and R.J. LeBlanc. 2000. *Final Draft: Evaluation of the potential for Oil Sands CT water to cause taint in laboratory exposed rainbow trout (Onchorhynchus mykiss) using a selected, screened, trained sensory evaluation panel.* Prepared for HydroQual Laboratories Ltd.

LeBlanc, G.A. and J.W. Dean. 1984. *Antimony and thallium toxicity to embryos and larvae of fathead minnows (Pimephales promelas).* Bull Environ Contam Toxicol 32:565-569.

LeBlanc, G.C., J.D. Mastone, A.P. Paradice, B.F. Wilson, H.B. Lockhart Jr. and K.A. Robillard. 1984. *The influence of speciation on the toxicity of silver to fathead minnow (Pimephales promelas)*. Environ Toxicol Chem 3:37-46.

LeBlond, J.B. and L.K. Duffy. 2001. *Toxicity assessment of total dissolved solids in effluent of Alaskan mines using 22-h chronic Microtox® and Selenastrum capricornutum assays*. Sci Total Environ 217: 49-59.

Leverone, J.R. 2007. *Development of New Technologies for Removal of Arsenic from Water Supplies through Biomimicry of Natural Systems*. Final Report. Mote Marine Laboratory, Sarasota, FL, USA. Prepared for Economic Development Corporation, Sarasota County, Sarasota, FL, USA.

Linton, T.K., M.A.W. Pacheco, D.O. McIntyre, W.H. Clement, and J. Goodrich-Mahoney. 2007. *Development of bioassessment-based benchmarks for iron*. Environ Toxicol Chem 26: 1291-1298.

MacKinnon, M. and H. Boerger. 1986. *Description of two treatment methods for detoxifying oil sands tailings pond water.* Water Pollution Research Journal of Canada 21:496-512.

Maxxam. 2010. *A review of the nature of naphthenic acid occurrence toxicity and fate in refinery and oil sands extraction wastewaters.* Power Point presentation prepared by Dr. Curtis Eickhoff, presented at Second International Oil Sands Tailings Conference. Internet: http://ostrfdownload.civil.ualberta.ca/2nd%20International%20Oil%20Sands%20Tailings%20Conference%202010/IOSTC%202010%20Dec%207%20presentations/Eickhoff%20et%20al.pdf

McDonald, B.G. and P.M. Chapman. 2002. *PAH phototoxicity – an ecologically irrelevant phenomenon?* Mar Poll Bull 44:1321-1326.

McGrath, J.A. and D.M. DiToro. 2009. *Validation of the target lipid model for toxicity assessment of residual petroleum constituents: monocyclic and polycyclic aromatic hydrocarbons.* Environ Toxicol Chem 28:1130-1148.

MDEQ (Michigan Department of Environmental Quality). 2008. *Rule 57 Water Quality Values 2008-12-10*. Surface Water Quality Assessment Section.

Meays, C. and R. Nordin. 2013. *Ambient Water Quality Guidelines for Sulphate - Technical Appendix Update*. Prepared by: Meays C and Nordin R, Water Protection & Sustainability Branch, Environmental Sustainability and Strategic Policy Division, BC Ministry of Environment. April 2013.

Millemann, R.E., W.J., Birge, J.A. Black, R.M. Cushman, K.L. Daniels, P.J. Franco, J.M. Giddings, J.F. McCarthy, and A.J. Stewart. 1984. *Comparative acute toxicology to aquatic organisms of components of coal-derived synthetic fuels*. Trans Am Fish Soc 113:74-85.

Moissenko, T.I. and L.P. Kudryavtseva. 2001*. Trace metal accumulation and fish pathologies in areas affected by mining and metallurgical enterprises in the Kola Region, Russia*. Environ Pollut 114:285-297.

Mount, D.R., D.D. Gulley, J.R. Hockett, T.D. Garrison and J.M. Evans. 1997. *Statistical Models to Predict the Toxicity of Major Ions to Ceriodaphnia dubia, Daphnia magna and Pimephales promelas (fathead minnows)*. Environ Toxicol Chem 16:2009-2019.

Naumann, B., M. Eberius, and K-J Appenroth. 2007. *Growth rate based dose-response relationships and EC-values of ten heavy metals using the duckweed growth inhibition test (ISO 20079) with Lemna minor L*. Clone St. J Plant Physiol 164:1656-1664.

Nautilus (Nautilus Environmental). 2012. *Evaluation of the Chronic Toxicity of Strontium to Hyalella azteca*. Laboratory report for Golder Associates Ltd., Burnaby, BC, Canada. November 26, 2012.

Nautilus. 2013. *Evaluation of the Chronic Toxicity of Strontium to Early Life Stages of Rainbow Trout*. Laboratory report for Golder Associates Ltd., Burnaby, BC, Canada. January 11, 2013.

Nebeker, A.V, C.K. McAuliffe, R. Mshar and D.G. Stevens. 1983. *Toxicity of silver to steelhead and rainbow trout, fathead minnows and Daphnia magna*. Environmental Toxicology and Chemistry 2:95-104.

Nero, V, A. Farwell, L.E. Lee, T. Van Meer, M.D. MacKinnon and D.G. Dixon. 2006a. *The effects of salinity on naphthenic acid toxicity to yellow perch: gill and liver histopathology.* Ecotoxicol Environ Saf 65:252-264.

Nero V., A. Farwell, A. Lister, G. Van Der Kraak, L.E.J. Lee, T. Van Meer, M.D. MacKinnon and D.G. Dixon. 2006b. *Gill and liver histopathological changes in yellow perch (Perca flavescens) and goldfish (Carassius auratus) exposed to oil sands process-affected water*. Ecotoxicol. Environ. Saf. 63: 265–377.

NWQMS (National Water Quality Management Strategy). 2000. *Australian and New Zealand Guidelines for Fresh and Marine Water Quality.* Prepared by Australia and New Zealand Environment and Conservation Council (ANZECC) and Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ).

OECD (Organisation for Economic Co-operation and Development). 1994. *Risk Reduction Monograph No. 5: Cadmium.* OECD Environment Directorate. Paris, France.

Ohio EPA (Ohio Environmental Protection Agency). 2009. *Lake Erie Basin Aquatic Life and Human Health Tier I Criteria, Tier II Values and Screening Values (SV)*. Chapter 3745-1 of the Ohio Administrative Code. Division of Surface Water.

Oris, J.T. and J.P. Giesy (Jr). 1986. *Photoinduced toxicity of anthracene to juvenile bluegill sunfish (Lepomis macrochirus Rafinesque): photoperiod effects and predictive hazard evaluation.* Environ Toxicol Chem 5:761-768.

Oseid, D.M. and L.L. Smith Jr. 1974. *Chronic toxicity of hydrogen sulfide to Gammarus pseudolimnaeus.* Am Fish Soc Trans 103:819-822.

Pacholski, L. 2009. *Toxicity of Stable Strontium in Surface Freshwaters of the Oil Sands Region in Alberta, Canada – Deriving an Aquatic Guideline and Refined Chronic Effects Benchmark*. University of Ulster, Londonderry, Northern Ireland.

Parametrix (Parametrix, Inc.). 1995. *Persistence, bioaccumulation and toxicity of metals and metal compounds.* Prepared for the International Council on Metals and the Environment. Ottawa, ON. 93 pp.

Parkhurst, B.R., W. Warren-Hicks, R.D. Cardwell, J. Volosin, T. Etchison, J.B. Butcher and S.M. Covington. 1994. *Methodology for Aquatic Ecological Risk Assessment.* Contract No. RP91-AER-1. Prepared for Water Environment Research Foundation. Alexandria, VA, USA.

Patrick, R., J. Cairns Jr., A. Scheier. 1968. *The relative sensitivity of diatoms, snails, and fish to twenty common constituents of industrial wastes*. Prog Fish Culturist 30:137-140.

Posthuma, L, G.W. Suter II and T.P. Traas. 2002. *Species Sensitivity Distributions in Ecotoxicology.* Lewis Publishers, Boca Raton, FL, USA.

Phippen, B. C. Horvath, R. Nordin and N. Nagpal. 2008. *Ambient Water Quality Guidelines for Iron*. Prepared for Science and Information Branch, Water Stewardship Division, British Columbia Ministry of Environment, February 28, 2008.

Purcell, T.W. and J.J. Peters. 1998. *Sources of silver in the environment*. Environ Toxicol Chem 17:539-546.

Rankin, M.G and D.G. Dixon. 1994. *Acute and chronic toxicity of waterborne arsenite to rainbow trout (Oncorhynchus mykiss)*. Can J Fish Aquat Sci 51:372-380.

Redman, A. and D. Macalady D. 2003. *Speciation of Inorganic Arsenic in Natural and Synthetic Waters: Redox Transformations and Ligand-Metal Complexation*. Poster presentation at the Twenty-Fourth Annual Meeting of the Society of Environmental Toxicology and Chemistry, November 9-13, Austin, TX, USA.

Reuther, R. 1992. *Arsenic introduced into a littoral freshwater model ecosystem*. Sci Total Environ 115: 219–237.

Richter, R.O. and T.L. Theis. 1980. *Nickel. In: Nriagu JO (ed.), Nickel in the Environment.* New York, NY: John Wiley and Sons, Inc. pp. 189-202.

Roch, M. and E.J. Maly. 1979. *Relationship of cadmium-induced hypocalcemia with mortality in rainbow trout (Salmo gairdneri) and the influence of temperature on toxicity*. J Res Fish Board Canada 36):297-1303.

Rosseland, B.O., T.D. Eidhuset, and M. Staurnes. 1990. *Environmental effects of aluminum.* Environ Geochem Health 12:17-27.

Sandheinrich, M.B, and J.G. Wiener. 2011. *Methylmercury in Freshwater Fish: Recent Advances in Assessing Toxicity of Environmentally Relevant Exposures*. Chapter 4 in Beyer, N.W. and J.P. Meador (eds). 2011. Environmental Contaminants in Biota, Interpreting Tissue Concentrations (2nd Edition). CRC Press, Taylor and Francis Group, Boca Raton, Florida.

Schroder, S.L., C.M. Knudsen, E.C. Volk. 1995. *Marking salmon fry with strontium chloride solutions*. Can J Fish Aquat Sci 52: 1141-1149.

Scow, K., M. Goyer, L. Nelken, P. Cruse and K. Moss. 1981. *Exposure risk assessment for silver*. Technical Report. US Environmental Protection Agency, Washington, DC, USA.

Senn, D.B. and H.F. Hemond. 2004. *Particulate arsenic and iron during anoxia in a eutrophic, urban lake*. Environ Toxicol Chem 23: 1610-1616.

Shell (Shell Canada Energy). 2012. *Jackpine Mine Expansion Project Submission of Information to the Joint Review Panel*, May 2012. Re: Jackpine Mine Expansion Project CEAR No. 10-05-59540 and Response to Supplemental Information Requests of January 30, 2012. Submitted to the Canadian Environmental Assessment Agency and the Jackpine Mine Expansion Panel. Prepared by Shell Canada Limited, Golder Associates Limited and Nichols Applied Management. Calgary, AB.

Snarski, V.M. and G.F. Olson. 1982. *Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (Pimephales promelas).* Aquat Toxicol 2:143-156.

Spehar, R.L., J.T. Fiandt, R.L. Anderson, and D.L. DeFoe. 1980. *Comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish*. Arch Environ Contam Toxicol 9: 53-63.

Spehar, R.L. and J.T. Fiandt. 1986. *Acute and chronic effects of water quality on mixtures of three aquatic species*. Environ Toxicol Chem 5: 917-931.

Speyer, M.R. 1975. *Some Effects of Chronic Combined Arsenic and Cyanide Poisoning on the Physiology of Rainbow Trout.* MS Thesis. Concordia University, Montreal, QB, Canada.

SPSS (SPSS Inc.). 2002. *SigmaPlot 8.02.* Chicago, Illinois.

Staley, J.T. and W. Haupin. 1992. *Aluminum and aluminum alloys.* In: Kroschwitz, J.I. and M. Howe-Grant (eds.), Kirk-Othmer Encyclopedia of Chemical Technology. Vol. 2: Alkanolamines to antibiotics (glycopeptides). New York: John Wiley & Sons, Inc. pp. 248-249.

Stekoll, M., W. Smoker, I. Wang and B. Failor. 2003a. *Salmon as a bioassay model of effects of total dissolved solids*. Final report for ASTF grant #98-1-012. Juneau, Alaska, University of Alaska - Fairbanks: 87 pp.

Stekoll, M, W. Smoker, I. Wang and W. Hayes. 2003b. *Final report on the effects of total dissolved solids on fertilization rates of salmonids in the Red Dog Mine Area.* Juneau, Alaska, University of Alaska - Fairbanks: 27 pp.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman and W.A. Brungs. 1985. *Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses*. PB85-227049. National Technical Information Service, Springfield, VA, USA.

Stevens, D.G. and G.A. Chapman. 1984. *Toxicity of trivalent chromium to early life stages of steelhead trout.* Environ Toxicol Chem 3:125-133.

Suedel, B.C., E. Deaver and J.H. Rodgers. 1996. *Experimental factors that may affect toxicity of aqueous and sediment-bound copper to freshwater organisms.* Arch Environ Contam Toxicol 30:40-46.

Suter, G.W. II and L. Barnthouse. 1993. *Assessment concepts.* In: Suter, G.W. II (ed.), Ecological Risk Assessment. Lewis Publishers. Chelsea, MI, USA. pp. 21-47.

Suter, G.W. II, B.W. Cornaby, C.T. Hadden, R.N. Hull RN, M. Stack and F.A. Safran. 1995. *An approach for balancing health and ecological risks at hazardous waste sites.* Risk Anal 15:221–231.

Suter, G.W. II, T.P. Traas and L. Posthuma. 2002. *Issues and practices in the derivation and use of species sensitivity distributions.* In: Posthuma L, G.W. Suter II and T.P. Traas (eds), Species Sensitivity Distributions in Ecotoxicology. Lewis Publishers, New York, NY, USA.

Suzuki, K. 1959. *The toxic influence of heavy metal salts upon mosquito larvae*. Hokkaido Univ J Fac Sci Ser 6: 196-209.

Swartz, R.C., S.P. Ferraro J.O. Lamberson, F.A. Cole, R.J. Ozretich, B.L. Boese, D.W. Schults, M. Behrenfeld and G.T. Ankley. 1997. *Photoactivation and toxicity of mixtures of polycyclic aromatic hydrocarbon compounds in marine sediment.* Environ Toxicol Chem 16:2151-2157.

Syncrude (Syncrude Canada Ltd.). 2009. *Syncrude Aurora South Project 2009 Environmental Update*. Submitted to the Alberta Energy Resources Conservation Board. December 2009. Calgary, AB.

Total (Total E&P Joslyn Ltd.). 2010. *Joslyn North Mine Project 2010 Additional Information - Project Update*. Submitted to Government of Canada-Energy Resources Conservation Board Joint Review Panel. February 2010. Calgary, AB.

Trenfield, M.A., S.J. Markich, J.C. Ng, B. Noller, and R.A. van Dam. 2012. *Dissolved organic carbon reduces the toxicity of aluminum to three tropical freshwater organisms*. Environ Toxicol Chem 31: 427-436.

U.S. EPA (United States Environmental Protection Agency). 1984. *Ambient water quality criteria for chromium – 1984.* EPA 440/5-84-029. Office of Water Regulations and Standards, Criteria and Standards Division. Washington, DC, USA.

U.S. EPA. 1985. *Ambient water quality criteria for copper* – 1984. EPA 440/5-84-031. Office of Water Regulations and Standards, Criteria and Standards Division. Washington, DC, USA.

U.S. EPA. 2001. *Update of ambient water quality criteria for cadmium*. EPA-822-R-01-001. National Technical Information Service, Springfield, VA, USA.

U.S. EPA. 2007a. ECOTOX Release 4.0. *The ECOTOX (ECOTOXicology) database of single chemical toxicity information for aquatic and terrestrial life.* Available at: http://cfpub.epa.gov/ecotox/ecotox_home.cfm

U.S. EPA. 2007b. *Aquatic life ambient freshwater quality criteria – copper, 2007 revision*. EPA-822-R-07-001. U.S. EPA Office of Water. Washington, DC, USA. 204 pp.

Verbeek, R.C., W. MacKay W and M. MacKinnon. 1993. *Oil Sands: Our Petroleum Future Conference, Edmonton, Alberta. April 4-7, 1993.* Alberta Oil Sands Technology and Research Authority. Edmonton, AB.

Versteeg, D.J., S.E. Belanger and G.J. Carr. 1999. *Understanding single species and model ecosystem sensitivity: data-based comparison.* Environ Toxicol Chem 18:1329-1346.

Vuori, K-M. 1995. *Direct and indirect effects or iron on river ecosystems.* Ann Zool Fennici 32:317-329.

Wang, F. and P.M. Chapman. 1999. *Biological implications of sulfide in sediment—a review focusing on sediment toxicity.* Environ Toxicol Chem 18:2526–2532.

Weber-Scannell, P.K. and L.K. Duffy. 2007. *Effects of Total Dissolved Solids on Aquatic Organisms: A Review of Literature and Recommendation for Salmonid Species*. Am J Environ Sci 3: 1-6.

Weber-Scannell, P.K. and L.L. Jacobs. 2001. *Effects of Total Dissolved Solids on Aquatic Organisms: A Literature Review*. Technical Report No. 01-06. Alaska Department of Fish and Game, Division of Habitat and Restoration. June 2001.

Wehrli, B. and W. Stumm. 1989. *Vanadyl in natural waters: adsorption and hydrolysis promote oxygenation.* Geochimica et Cosmochimica Acta. 53:69-77.

Wiener, J.G. and D.J. Spry. 1996. *Toxicological significance of mercury in freshwater fish*. In Beyer, N.W., G.H. Heinz, A.W. Redmon-Norwood (eds), *Environmental Contaminants in Wildlife, Interpreting Tissue Concentrations*. CRC Press, Boca Raton, Florida. pp. 297-339.

Wilson, R.W. 2012. *Aluminum*. In Wood CM, Farrell AP, Brauner CJ (eds), Homeostasis and Toxicology of Non-Essential Metals. Fish Physiology Volume 31B, Academic Press/Elsevier, New York, NY, USA, pp 67-123.

Woodward D.F, R.G. Riley, M.G. Henry, J.S. Meyer and T.R. Garland. 1985. *Leaching of retorted oil shale: assessing the toxicity to Colorado squawfish, fathead minnows, and two food-chain organisms*. Trans Am Fish Soc 114:887-894.

Wright, D.A. and P.M. Welbourn. 1994. *Cadmium in the aquatic environment: a review of ecological, physiological, and toxicological effects on biota.* Environ Rev 2:187-214.

3.1 Personal Communications

Carleton-Dodds, I. 2010. Personal communication. Electronic mail from I. Carleton-Dodds (Senior Ecotoxicologist, HydroQual Laboratories Ltd., Calgary, AB, Canada) to Kerrie Serben (Environmental Scientist, Golder Associates, Saskatoon SK). October 15, 2010.

Cook, D. 2013. Personal communication. Electronic mail from Dave Cook (Global Environmental Consulting LLC) to Cathy McPherson (Environmental Scientist, Golder Associates Ltd.). February 5, 2013.

HydroQual. 2013. Personal communication. Electronic mail from Laura Pacholski (now Golder Associates Ltd) to Cathy McPherson (Environmental Scientist, Golder Associates Ltd.), February 1, 2013.

As a global, employee-owned organisation with over 50 years of experience, Golder Associates is driven by our purpose to engineer earth's development while preserving earth's integrity. We deliver solutions that help our clients achieve their sustainable development goals by providing a wide range of independent consulting, design and construction services in our specialist areas of earth, environment and energy.

For more information, visit golder.com

Africa	+ 27 11 254 4800
Asia	+ 86 21 6258 5522
Australasia	+ 61 3 8862 3500
Europe	+ 356 21 42 30 20
North America	+ 1 800 275 3281
South America	+ 56 2 2616 2000

solutions@golder.com
www.golder.com

Golder Associates Ltd.
102, 2535 - 3rd Avenue S.E.
Calgary, Alberta, T2A 7W5
Canada
T: +1 (403) 299 5600

Golder Associates