Ministry of Environment
Province of British Columbia

Ambient Water Quality Guidelines for Cadmium

Technical Report

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Water Protection & Sustainability Branch
Environmental Sustainability and Strategic Policy Division
BC Ministry of Environment

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List of Abbreviations

ANZECC	Australia and New Zealand Environment and Conservation Council
ASTM	American Society for Testing and Materials
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation Factor
BC	British Columbia
BCF	Bioconcentration Factor
BC MOE	British Columbia Ministry of Environment
BLM	Biotic Ligand Model
Ca	Calcium
$CaCO_3$	Calcium Carbonate
C	Celsius
CCC	Criteria Continuous Concentration
CCME	Canadian Council of Ministers of the Environment
CI_{95}	95% Confidence Interval
CMC	Criteria Maximum Concentration
-d	Day
DOM	Dissolved Organic Matter
DW	Dry Weight
EC_x	Effective Concentration Affecting x% of the Population
EDTA	Ethylenediaminetetraacetic acid
EQS	Environmental Quality Standard
F_1	First Filial Generation
g/cm^3	Grams per Cubic Centimetre
-h	Hour
H	Hydrogen
IC_x	Inhibitory Concentration Causing an x% Inhibition in Tested Organisms
kg	Kilogram
LC_x	Lethal Concentration Affecting x% of the Population
LOEC	Lowest Observed Effect Concentration
MATC	Maximum Allowable Toxicant Concentration
MDL	Method Detection Limit
mg/L	Milligrams per Litre
MST	Median Survival Time
µg/g	Micrograms per Gram
µg/L	Micrograms per Litre
Na	Sodium
NOEC	No Observed Effect Concentration
NOM	Natural Organic Matter
NTU	Nephelometric Turbidity Units
PQL	Practical Quantitation Limit
ROS	Regression on Order Statistics
TDS	Total Dissolved Solids
TSS	Total Suspended Solids
USEPA	United States Environmental Protection Agency
WHO	World Health Organization
WQG	Water Quality Guideline
WW	Wet Weight

Executive Summary

Cadmium (Cd) has been identified as a metal of major importance by multiple agencies because of its toxicity to humans and wildlife. In aquatic ecosystems, Cd interferes with the uptake of calcium by organisms, resulting in cellular damage, decreases in metabolic activity, and adverse effects on osmoregulation. There is also evidence of Cd bioaccumulation through ingestion of contaminated prey. The results of Cd toxicity on freshwater aquatic organisms include increased mortality, decreased growth, and decreased reproductive capacity and success.

Cd enters aquatic ecosystems through natural processes, such as the weathering of minerals and forest fires, or through human activities such as mining and other land-use activities. Cd concentrations in natural (i.e., unimpacted) surface waters and groundwater are generally less than 1 µg/L Cd; however, some areas may contain naturally elevated cadmium levels due to the geological composition of the area. In BC, Cd concentrations are generally lower in southern regions compared to central and northern regions.

The British Columbia Ministry of Environment (BC MOE) develops ambient water quality guidelines (WQGs) to assess and manage the health, safety and sustainability of British Columbia's (BC's) aquatic resources. Guidelines are developed to protect aquatic life, wildlife, agriculture (irrigation and livestock watering), drinking water sources, and recreation and aesthetics.

In BC, the development of WQGs for aquatic life is directed by the following guiding principles:

- WQGs are science-based and intended for generic provincial application;
- WQGs do not account for site-specific conditions or socio-economic factors;
- All components of the aquatic ecosystem (e.g., algae, macrophytes, invertebrates, amphibians, and fish) are considered when data are available;
- Where data are available but limited, interim WQGs may be developed; and,
- All forms of aquatic life and all aquatic stages of their life cycle are to be protected during indefinite exposures.

WQGs for the protection of aquatic life represent levels that are protective of the most sensitive species at its most sensitive life-stage during both short-term and long-term exposures. The short-term maximum guideline represents a level that should not be exceeded at any given time (i.e., instantaneous maximum) to prevent severe short-term effects, whereas the long-term average guideline represents a level that the 30-day average concentration (calculated as the mean of 5 samples within 30 days) should not be exceeded to prevent chronic effects.

In 1996, the Canadian Council of Ministers of the Environment (CCME) proposed ambient freshwater WQGs for total Cd and the BC MOE adopted this guideline as a

working WQG. Since that time, additional studies on the toxicity of Cd to aquatic life have been conducted and published. In addition, CCME has updated the WQG for Cd (CCME 2014a). This report reviews the most recent science on Cd toxicity in the freshwater environment and uses this information to derive updated WQGs for Cd.

Water hardness influences cadmium toxicity by directly competing for binding sites on organism tissues such as gills. The working Cd WQG was a hardness-based guideline, which used hardness levels measured at the site to determine appropriate WQG levels. This approach is maintained in the updated Cd WQG. The updated Cd WQG has changed from the working Cd WQG in that it is now based on dissolved cadmium, rather than total Cd. Dissolved Cd was chosen for the following reasons:

- Dissolved Cd is the more bioavailable and ecologically relevant form;
- Concentrations of total Cd in BC waters are highly variable and depend on water flow and the associated concentrations of suspended sediment; and,
- Published toxicity tests were conducted using dissolved salts, and as such are best represented as dissolved Cd.

The results of published studies were used to derive relationships between Cd toxicity and water hardness. The following equation is recommended to calculate short-term maximum WQGs for water with different water hardness concentrations:

$$WQG_{SHORT\text{-}TERM} = e^{[1.03 * \ln(Hss) - 5.274]}$$

Where: H_{SS} = site-specific water hardness (mg/L $CaCO_3$).

At a water hardness of 50 mg/L $CaCO_3$, the recommended short-term maximum WQG for dissolved Cd to protect freshwater aquatic life is 0.288 µg/L.

The following equation is recommended to calculate long-term average WQGs for water with different water hardness concentrations:

$$WQG_{LONG\text{-}TERM} = e^{[0.736 * \ln(Hss) - 4.943]}$$

Where: H_{SS} = site-specific water hardness (mg/L $CaCO_3$).

At a water hardness of 50 mg/L $CaCO_3$, the recommended long-term (30-d) average WQG for dissolved Cd to protect freshwater aquatic life is 0.127 µg/L.

A summary of the short-term maximum and long-term average WQGs at various water hardness levels is presented in Table ES 1. Table ES 2 presents the working WQGs for marine water, sediment (both marine and freshwater), livestock watering, and irrigation.

Table ES 1. Summary of Recommended Water Quality Guidelines for Cadmium.

Guideline Type/Equation	British Columbia WQG (µg/L Cd) for Dissolved Cadmium at Varying Water Hardness				
	Lower Bound[A]	50 mg/L $CaCO_3$	180 mg/L $CaCO_3$	320 mg/L $CaCO_3$	Upper Bound[B,C]
Short-Term Maximum WQG					
$WQG_{Short\text{-}term} = e^{[1.03 * \ln(H_{ss}) - 5.274]}$	0.0380	0.288	1.08	1.95	2.80
Long-Term Average WQG					
$WQG_{Long\text{-}term} = e^{[0.736 * \ln(H_{ss}) - 4.943]}$	0.0176	0.127	0.326	0.457[B]	0.457

[A] The lower bound for the short-term maximum guideline is 7 mg/L $CaCO_3$; the lower bound for the long-term average guideline is 3.4 mg/L $CaCO_3$.
[B] The upper bound for the short-term maximum guideline is 455 mg/L $CaCO_3$; the upper bound for the long-term average guideline is 285 mg/L $CaCO_3$.
[C] When water hardness is greater than the upper bound (i.e., highest water hardness tested), a site-specific assessment may be required.

Table ES 2. Summary of Working Water Quality Guidelines for Total Cadmium.

Guideline Type	Working WQG for Total Cadmium[A]
Water (µg/L) - Marine	0.12
Sediment (µg/g) - Freshwater	
Interim Sediment Quality Guideline (ISQG)	0.6
Probable Effect Level (PEL)	3.5
Sediment (µg/g) Marine	
Interim Sediment Quality Guideline (ISQG)	0.7
Probable Effect Level (PEL)	4.2
Livestock watering (µg/L)	80
Irrigation (µg/L)	5.1

[A] A Compendium of Working WQGs for BC (BC MOE 2006); adopted from CCME (2014b).

It is important to note that, while water hardness is considered to be a primary factor in ameliorating Cd toxicity, other factors also play a role. The ameliorating effects of hardness are tightly linked to the pH and alkalinity of the system in natural surface waters; therefore, due care should be applied in systems in which water hardness is influenced by anthropogenic activities.

1.0 Introduction

The BC Ministry of Environment (BC MOE) develops province-wide ambient water quality guidelines (WQGs) for substances or physical attributes that are important for managing both fresh and marine surface waters of BC. This work has the following goals:

- To provide protection of the most sensitive aquatic life form and most sensitive life stage indefinitely;
- To provide a basis for the evaluation of data on water, sediment, and biota for water quality and environmental impact assessments;
- To provide a basis for the establishment of site-specific ambient water quality objectives;
- To provide a basis for identifying areas where degraded conditions may exist and warrant further evaluation;
- To provide a basis for establishing wastewater discharge limits; and,
- To report to the public on the state of water quality and promote water stewardship.

BC WQGs are science-based and intended for generic provincial application. They do not account for site-specific conditions or socio-economic factors. All components of the aquatic ecosystem (e.g., algae, macrophytes, invertebrates, amphibians, and fish) are considered if the data are available. Where data are available but limited, interim guidelines may be developed.

The approach to develop guidelines for aquatic life reflects the guiding principles that all forms of aquatic life and all aquatic stages of their life cycle are to be protected during indefinite exposure. For some substances both a short-term maximum and a 30-day average (long-term) guideline are recommended as provincial WQGs, provided sufficient toxicological data are available. Both conditions should be met to protect aquatic life.

The goal of freshwater aquatic life guidelines is the protection and maintenance of all forms of aquatic life and all life stages in the freshwater environment. Therefore, it is essential that, at a minimum, data for fish, invertebrates, and plants be included in the guidelines derivation process. Data from amphibians are also highly desirable. Guidelines or interim guidelines may also include studies involving species not required in the minimum data set (e.g., protozoa, bacteria) when reasonable justification exists.

It should be noted that there are several sources of uncertainty when it comes to developing WQGs and therefore it is necessary to apply uncertainty factors. Sources of uncertainty include:

- Laboratory to field differences;
- Single to multiple contaminants (additive, synergistic, antagonistic effects);
- Toxicity of metabolites;
- Intra and inter-species differences (limited species to conduct tests on, which may not include the most sensitive species);
- Indirect effects (e.g., foodweb dynamics);
- Whole life-cycle vs. partial life-cycle (many toxicity studies are only conducted on partial life-cycles and it can be difficult to determine the most sensitive life stage);
- Delayed effects;
- Impacts of climate change (species may be more vulnerable with additional stressors); and,
- Other stressors including cumulative effects.

The appropriate uncertainty factor to be applied is decided on a case-by-case basis and is based on data quality and quantity, toxicity of the contaminant, severity of toxic effects, and bioaccumulation potential (BC MOE 2012). The use of an uncertainty factor is a policy decision; however, scientific judgment is used when determining the value.

Presently, WQGs do not have any direct legal standing. They are intended as a tool to provide policy direction to those making decisions affecting water quality provided that they do not allow legislated effluent standards to be exceeded. WQGs can be used to establish the allowable limits in waste discharges. These limits are set out in waste management permits, approvals, plans, or operating certificates which do have legal standing. An exceedance of the WQGs presented in this document does not imply that unacceptable risks are present, but that the potential for adverse effects is increased and additional investigation and monitoring should be conducted.

Cadmium (Cd) has been identified as a metal of major importance by multiple agencies because of its toxicity to humans and wildlife (e.g., Agency for Toxic Substances and Disease Registry, Environment Canada, Health Canada, and U.S. Environmental Protection Agency). In experiments with duckweed (*Lemna minor*), Cd was determined to be the most toxic of the four metals tested, including copper, nickel, and zinc (Drost *et al.* 2007). Cd is also known to be toxic to algae (e.g., Canton and Slooff 1982; Benhra *et al.* 1997; Baer *et al.* 1999; Källqvist 2009), invertebrates (e.g., Suedel *et al.* 1997; Watts and Pascoe 2000; Black 2001; Felten *et al.* 2008; Wang *et al.* 2010; Mebane *et al.* 2012), fish (e.g., Eaton *et al.* 1978; Suedel *et al.* 1997; Hansen *et al.* 2002a; Besser *et al.* 2007; Brinkman and Vieira 2008; Mebane *et al.* 2012), and amphibians (e.g., Canton and Slooff 1982; Ferrari *et al.* 1993; Nebeker *et al.* 1995).

Cd is released into the environment from both natural and anthropogenic sources. Small amounts of Cd enter the environment from the natural weathering of minerals, forest fires, volcanic emissions, generation of sea salt aerosols, and other natural processes. Major anthropogenic sources of Cd in the environment include non-ferrous metal mining and smelting operations, fossil fuel combustion, application of phosphate fertilizers, and waste incineration and disposal (ATSDR 2012). Furthermore, up until the early 1990s an unknown quantity of Cd had been administered through three turf grass production fungicides that contained Cd (CCME 1999).

In 2010, it was estimated that 2,670 kg of Cd were released into Canadian waters as a result of domestic anthropogenic activities. The largest proportion originated from water, sewage, and other systems, releasing 45% (1,213 kg) of the national total (Environment Canada 2012). Pulp, paper, and paperboard mills released 26% (705 kg), whereas 12% (321 kg) originated from non-ferrous production and processing (excluding aluminum). Other sources included foundries, metal ore mining, iron and steel mills, ferro-alloy manufacturing, and the manufacturing of petroleum and coal products (Environment Canada 2012). Sources in Ontario, BC, and Quebec had the highest releases of Cd, contributing 31% (831 kg), 27% (730 kg), and 26% (704 kg) to the national total, respectively. In BC, wastewater treatment plants were responsible for the majority of Cd releases, while non-ferrous production/processing, and pulp, paper, and paperboard mills were also important contributors (Environment Canada 2012).

The purpose of this report is to compile and review the key current scientific literature related to the toxicity of Cd to freshwater aquatic life. The information from these key studies was evaluated for its applicability for deriving short-term maximum and long-term average WQGs for the protection of freshwater aquatic life for the province of BC using the guidance provided in BC MOE (2012). This report is organized into a number of sections to facilitate the development of the WQGs, including:

- Introduction;
- Physical and Chemical Properties of Cadmium;
- Analysis of Cadmium in Environmental Samples;
- Environmental Concentrations of Cadmium in British Columbia Waters;
- Environmental Fate and Transport;
- Bioaccumulation and Bioconcentration of Cadmium in the Aquatic Environment;
- Toxicity of Cadmium to Freshwater Aquatic Organisms;

- Water Quality Guidelines from Other Jurisdictions; and,
- Derivation of Water Quality Guidelines for Cadmium in British Columbia.

2.0 Physical and Chemical Properties of Cadmium

Cd is a soft, silver-white metal, with an atomic number of 48 and a molar mass of 112.4 g (Budavari *et al.* 1989). Often associated with zinc, lead, and copper ores, Cd is found in the earth's crust at concentrations ranging from 0.1 to 0.5 mg/kg (ATSDR 2012). Cd is usually found in rocks as a minor component of mineral sulphides, especially zinc sulphides such as sphalerite and wurtzite (Nriagu 1980).

Elemental Cd has a density of 8.65 g/cm^3 at 25^{o}C, a melting point of 321^{o}C and a boiling point of 765^{o}C (ATSDR 2012). Cd can occur in two oxidation states: elemental Cd and as the divalent Cd ion (Cd^{2+}). However, Cd typically occurs in nature in its ionic form or in metal-ligand complexes. Although elemental Cd is insoluble in water, several of its salts such as cadmium chloride ($CdCl_2$), cadmium nitrate ($Cd[NO_3]_2$), and cadmium sulphate ($CdSO_4$) are freely soluble in water (Budavari *et al.* 1989). Some water-insoluble compounds such as cadmium oxide (CdO), cadmium sulphide (CdS), and cadmium carbonate ($CdCO_3$) may be solubilized under strong acidic or oxidizing conditions (WHO 1992) and are typically found as insoluble complexes in the environment. The physical and chemical properties of Cd and key salts of environmental relevance are found in Table 1.

Table 1. Physical and chemical properties of key cadmium species in the aquatic environment.

Compound	Chemical Formula	Molecular Mass (g)	Density (g/cm^3)	Melting Point (°C)	Boiling Point (°C)	Water Solubility (g/L)	Reference
Cadmium	Cd	112.41	8.65 @ 25°C	321	765	Insoluble	ATSDR (2012)
Cadmium carbonate	$CdCO_3$	172.42	4.58 @ 20°C	357 (decomposes)	ND	Insoluble	ATSDR (2012)
Cadmium chloride[A]	$CdCl_2$	183.32	4.047 @ 25°C	568	960	1400 @ 20°C	WHO (1992); ATSDR (2012)
Cadmium hydroxide	$Cd(OH)_2$	146.41	4.79 @ 25°C	300 (decomposes)	ND	0.0026 @ 26°C	WHO (1992)
Cadmium nitrate[A]	$Cd(NO_3)_2$	236.40	ND	59.5	132	Soluble	NJDHSS (2008)
Cadmium sulphate[A]	$CdSO_4$	208.47	4.69 @ 20°C	1000	ND	755 @ 0°C	WHO (1992); ATSDR (2012)

ATSDR = Agency for Toxic Substances and Disease Registry; ND = no data; NJDHSS = New Jersey Department of Health and Senior Services; WHO = World Health Organization.
[A] Commonly used in toxicity tests.

3.0 Analysis of Cadmium in Environmental Samples

A variety of instruments and associated methods are used to measure Cd in environmental samples (e.g., water samples), each with their own associated cost, efficiency, and sensitivity of detection for the target analyte. Methods such as flame atomic absorption spectrometry (FAAS; detection limit around 1.0 µg/L) and graphite furnace atomic absorption spectrometry (GFAAS; detection limit around 0.01 µg/L) detect one element at a time, while inductively coupled plasma atomic emission spectrometry (ICP-AES) and inductively coupled plasma mass spectrometry (ICP-MS) are commonly employed by laboratories and can detect multiple elements in a sample. The method detection limits (MDLs) typically attained by ICP-AES range between 0.1 and 1.0 µg/L, while the more sensitive method of ICP-MS can attain MDLs of 0.001 to 0.01 µg/L (Thermo Elemental 2001). While the MDL is defined as "the minimum concentration of a substance that can be measured and reported with 99% confidence that the analyte concentration is greater than zero and is determined from analysis of a sample in a given matrix containing the analyte" (CFR 2011), the practical quantitation limit (PQL) is defined as "the lowest achievable level of analytical quantitation during routine

laboratory operating conditions within specified limits of precision and accuracy" (USEPA 1985a). In practice, the PQL has been defined as a concentration 5-10 times the MDL (USEPA 1985a). Importantly, appropriate MDLs need to be achieved for monitoring programs that are designed to evaluate water quality conditions relative to ambient WQGs. Therefore, MDLs should be, at minimum, five times below the ambient WQG to ensure a higher level of precision and accuracy. However, in cases where laboratories have defined PQLs for the substance of interest, it is recommended that the PQL be at or below the ambient WQG.

Cd may be analysed in a water sample as either the dissolved or total fraction. Analysis of dissolved Cd includes the dissolved metal only (i.e., the fraction that passes through a 0.45 µm filter), while the analysis of total Cd includes the dissolved fraction and any Cd associated with particulates (e.g., suspended sediments) in the sample. While neither method provides information on the speciation of metals (e.g., ionic Cd) in the sample, the dissolved fraction provides a better estimate of bioavailable Cd (Campbell 1995; Paquin *et al.* 2002) as Cd associated with suspended sediments (and therefore not available for uptake) is excluded from the measurement. However, while measurements of dissolved metals in water may provide a more effective basis for evaluating the potential effects of metals on fish and aquatic life, such measurements do not provide comprehensive data for evaluating risks to aquatic life (i.e., only through direct toxicity) associated with exposure to metals or for managing discharges of metals into the environment. Therefore, monitoring of both dissolved and total Cd in the aquatic environment may be useful to address study questions because:

- WQGs based on the dissolved fraction are not designed to account for contamination due to loading of particulate metals to the water column and bottom sediments; and,
- WQGs based on the dissolved fraction are not designed to protect against accumulation through diet.

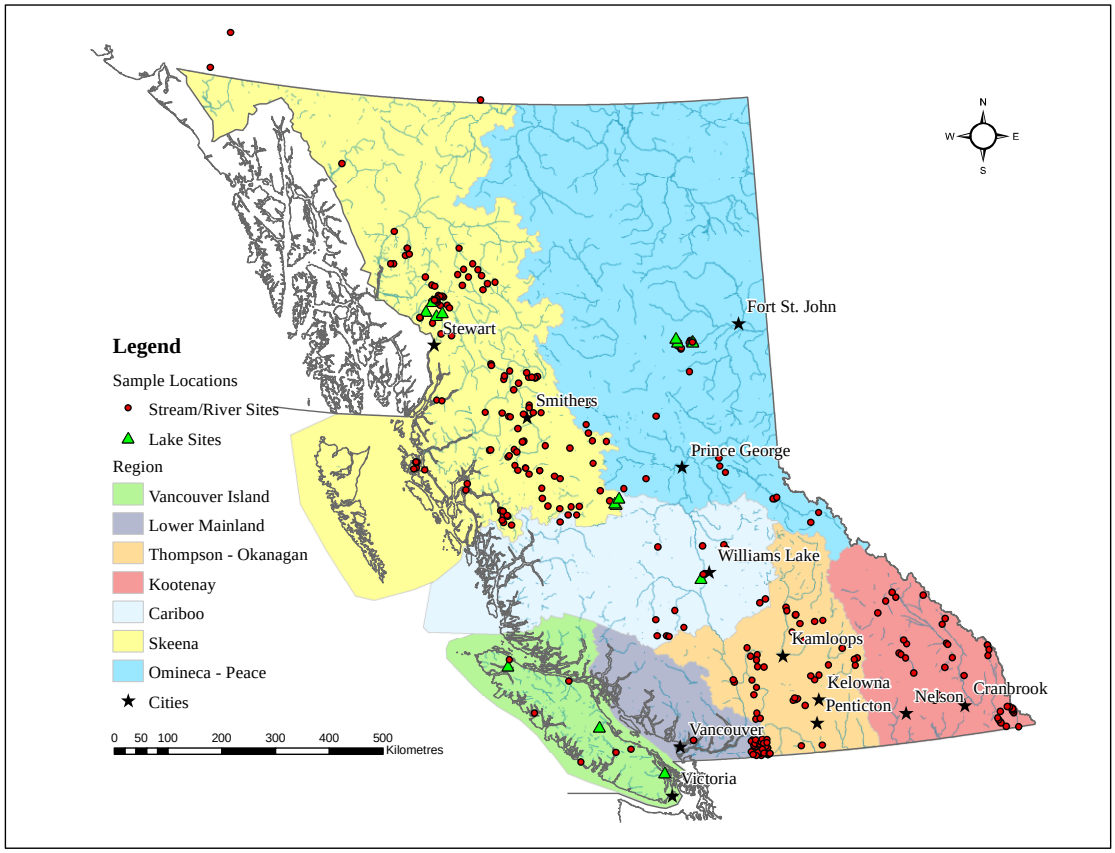
4.0 Environmental Concentrations of Cadmium in British Columbia Waters

Cd may enter aquatic ecosystems as a result of natural processes such as the weathering of minerals, forest fires, volcanic emissions, generation of sea salt aerosols, and other natural phenomena. While the concentration of Cd in natural (i.e., un-impacted) surface waters and groundwater is generally less than 1 µg/L Cd (ATSDR 2012), some areas contain naturally elevated levels of Cd due to the geological composition of the area. Additionally, the variability in background concentrations of Cd is determined by local climate, soil, water quantity and flow regime, and water and sediment quality conditions (e.g., pH and redox potential). It is therefore useful to estimate the environmental concentrations of Cd in BC waters to provide context for the development of WQGs.

4.1 Methods for Estimating Background Concentrations of Cadmium in British Columbia

In order to estimate the background levels of Cd in BC waters, data sets containing samples collected from 343 reference sites throughout the province were compiled from various sources (including Environment Canada, BC MOE, and the private sector) for evaluation (Figure 1; Appendix 1). These samples were collected to support multiple programs including BC MOE and Environment Canada water quality monitoring programs, the Canadian Aquatic Biomonitoring Network (CABIN), source-water monitoring programs, and baseline monitoring programs to support environmental assessments, and thus, were deemed appropriate for estimating background concentrations of Cd in BC waters. The compiled data sets included information on water quality variables (total and dissolved Cd, turbidity and/or total suspended solids [TSS], and hardness) and sampling information (i.e., sample collection date and geographic coordinates). Estimates of the mean concentration of Cd at each of the evaluated stations were summarized within each of the seven BC MOE administrative

Figure 1. Locations of reference sites used to estimate background concentrations of cadmium in B.C. waters.



regions (i.e., Vancouver Island, Lower Mainland, Thompson-Okanagan, Kootenay, Cariboo, Skeena, and Omineca-Peace). Laboratory replicates were identified during data compilation and were incorporated as a sample average. Field replicates were treated as discrete samples and were not averaged.

To facilitate the characterization of background concentrations of total and dissolved Cd in BC, the mean concentration of Cd was calculated for each station prior to determining the distribution of Cd concentrations in regions and in the province. Conducting the analysis in this way gives equal weighting to each station used in the analysis. To calculate station means using data containing non-detect concentrations, the regression on order statistics (ROS) method was employed to estimate each mean where at least some of the samples were non-detect data (Huston and Juarez-Colunga 2009). However, due

to the high number of non-detect concentrations present at some of the stations (i.e., > 80% of samples), some level of uncertainty exists in the calculated station means. In cases where all samples collected at a particular station were below detection limits, the highest detection limit present in the samples at that station was selected to represent the estimated station mean. The mean concentrations for stations in which all samples had detected concentrations were calculated as arithmetic means. A single dataset was created from the individual samples collected at the 343 reference sites, which included the mean total and dissolved concentrations by station for all collected samples.

The characterization of Cd concentrations at the regional level was derived by calculating the minimum and maximum of the station means. In cases where all stations within a region were represented by station means below detection limits, the minimum and maximum detection limits of the data within that region were used to determine the range.

4.2 Background Levels of Cadmium in British Columbia

The processed (i.e., screened) data set consisted of 2,680 total Cd samples (Table 2) and 671 dissolved Cd samples (Table 3), which were used for estimating the range of background concentrations of Cd in each of the seven BC MOE administrative regions. A summary of the range of environmental concentrations in each of these regions is provided in the following sections and in Table 4.

4.2.1 Vancouver Island

Cd concentrations collected from 9 stations were used to estimate background concentrations on Vancouver Island. The mean total Cd concentrations by station were among the lowest in the province at both lentic and lotic sites. Mean total Cd concentrations by station in lotic systems of Vancouver Island ranged from 0.00287 to 0.0165 µg/L (n = 9 stations); the mean dissolved Cd concentrations ranged from 0.00275 to 0.0160 µg/L (n = 8 stations).

Table 2. Summary of data used to estimate background concentrations of total cadmium at select reference stations in British Columbia by region[A].

Data Source	# Stations	# Samples	Date Range	Concentration Range (µg/L)	DL Range (µg/L)	% Samples < DL
Vancouver Island						
BCMOE	8	153	2001-2011	<0.005 - 0.1	0.005 - 0.01	71.9
EC (Fed-Prov sites)	1	70	2006-2011	0.001 - 0.027	NA	0
All data	*9*	*223*	*2001-2011*	*0.001 - 0.1*	*0.005 - 0.01*	*49.3*
Lower Mainland						
BCMOE	2	4	2002	<0.01 - 0.06	0.01	25.0
CABIN	9	9	2008	<0.01 - 0.04	0.01	88.9
All data	*11*	*13*	*2002-2008*	*<0.01 - 0.06*	*0.01*	*69.2*
Thompson-Okanagan						
BCMOE	7	47	1999- 2012	<0.006 - 1.69	0.006 - 0.01	17.0
CABIN	47	52	2007-2012	<0.005 - 0.082	0.005 - 0.01	44.2
All data	*54*	*99*	*1999-2012*	*<0.005 - 1.69*	*0.005 - 0.01*	*31.3*
Kootenay						
EC (Fed-Prov sites)	6	893	1985-2012	<0.001 - 1	0.001	3.47
CABIN	31	39	2007-2011	<0.005 - 0.062	0.005 - 0.01	46.2
All data	*37*	*932*	*1985-2012*	*<0.001 - 1*	*0.001 - 0.01*	*5.26*
Cariboo						
BCMOE	15	281	1998-2012	<0.005 - 0.65	0.005 - 0.05	67.6
CABIN	2	3	2006-2011	<0.01 - 0.012	0.01 - 0.01	66.7
All data	*17*	*284*	*1998-2012*	*<0.005 - 0.65*	*0.005 - 0.05*	*67.6*
Skeena						
CABIN	111	162	2003-2011	0.0006 - 0.151	0.005 - 0.01	66.0
EC (Fed-Prov sites)	3	407	2003-2012	<0.001 - 0.248	0.001 - 0.001	1.23
Seabridge Gold (KSM Mine)	17	93	2009	<0.01 - 0.176	0.01 - 0.03	39.8
TTM Resources (Chu Molybdenum Mine)	3	4	2008-2009	<0.017 - 0.0218	0.017 - 0.017	75.0
All data	*134*	*666*	*2003-2012*	*0.0006 - 0.248*	*0.001 - 0.03*	*22.8*
Omineca - Peace						
BCMOE	5	32	2002-2006	<0.01 - 0.04	0.01 - 0.01	90.6
EC (Fed-Prov sites)	1	200	2003-2012	<0.001 - 0.014	0.001 - 0.001	3.50
CABIN	8	8	2005-2006	<0.01 - 0.02	0.01 - 0.01	87.5
Cardero Coal (Carbon Creek)	19	93	2011-2012	0.011 - 0.117	0.05 - 0.05	6.45
CKD Mines (Gething Coal)	12	122	2006-2011	<0.0085 - 0.488	0.0085 - 0.025	46.7
Xstrata Coal (Goodrich Coal)	1	8	2011	0.015 - 0.029	NA	0
All data	*46*	*463*	*2002-2012*	*<0.001 - 0.488*	*0.001 - 0.05*	*22.9*

BCMOE = British Columbia Ministry of Environment; CABIN = Canadian Aquatic Biomonitoring Network; DL = Detection Limit; EC = Environment Canada; Fed-Prov = Federal-Provincial monitoring sites.
[A] Summary does not include data that were excluded during the screening process.

Table 3. Summary of data used to estimate background concentrations of dissolved cadmium at select reference stations in British Columbia by region[A].

Data Source	# Stations	# Samples	Date Range	Concentration Range (µg/L)	DL Range (µg/L)	% Samples < DL
Vancouver Island						
BCMOE	8	130	2001-2010	<0.005 - 0.09	0.005 - 0.01	74.6
Lower Mainland						
CABIN	24	25	2007-2008	<0.01 - 0.07	0.01 - 0.01	96.0
Thompson-Okanagan						
CABIN	22	22	2007-2008	<0.01 - 0.03	0.01 - 0.01	77.3
Kootenay						
EC (Fed-Prov sites)	1	3	2003-2004	0.003 - 0.009	NA	0
Cariboo						
BCMOE	15	142	1999-2012	<0.005 - 0.145	0.005 - 0.017	73.9
Skeena						
Seabridge Gold (KSM Mine)	17	94	2009	<0.01 - 0.057	0.01 - 0.017	74.5
Omineca - Peace						
BCMOE	5	32	2002-2006	<0.01 - 0.03	0.01 - 0.01	87.5
Cardero Coal (Carbon Creek)	19	93	2011-2012	0.007 - 0.071	0.01 - 0.05	14.0
CKD Mines (Gething Coal)	12	122	2006-2011	<0.0085 - 0.054	0.0085 - 0.025	67.2
Xstrata Coal (Goodrich Coal)	1	8	2011	0.014 - 0.022	NA	0
All data	*37*	*255*	*2002-2012*	*0.007 - 0.071*	*0.0085 - 0.05*	*48.2*

BCMOE = British Columbia Ministry of Environment; CABIN = Canadian Aquatic Biomonitoring Network; DL = Detection Limit; EC = Environment Canada; Fed-Prov = Federal-Provincial monitoring sites.
[A] Summary does not include data that were excluded during the screening process.

Table 4. Range of the station mean total and dissolved cadmium concentrations for each of the seven administrative regions in British Columbia.

Cadmium Fraction	Number of Stations	Station Mean Cd Concentration (µg/L)	
		Minimum	Maximum
Vancouver Island			
Total	9	0.00287	0.0165
Dissolved	8	0.00275	0.0160
Lower Mainland			
Total	11	< 0.01	0.0500
Dissolved	24	< 0.01	0.0700
Thompson - Okanagan			
Total	54	< 0.005	0.173
Dissolved	22	< 0.01	0.0300
Kootenay			
Total	37	0.00435	0.115
Dissolved	1	0.00600	0.00600
Cariboo			
Total	17	0.00345	0.132
Dissolved	15	0.00372	0.145
Skeena			
Total	134	< 0.005	0.151
Dissolved	17	< 0.01	0.0300
Omineca - Peace			
Total	46	0.00397	0.123
Dissolved	37	< 0.00850	0.0558

The detection limits for samples used in the analysis of background data from the Vancouver Island region ranged from 0.005 to 0.01 µg/L for both total and dissolved Cd. Overall, 49% of the samples analysed for total Cd fell below the detection limit, whereas 75% of the samples for the dissolved fraction were below detection limits.

4.2.2 Lower Mainland

Background Cd concentrations for the Lower Mainland region were estimated using data collected at 28 stations. The mean total Cd concentrations by station were among the lowest in the province for lotic sites. Mean total Cd concentrations by station ranged

from < 0.01 to 0.0500 µg/L (n = 11 stations) while mean dissolved Cd concentrations by station ranged from < 0.01 to 0.0700 µg/L (n = 24 stations).

The detection limit for all samples analysed from the Lower Mainland was 0.01 µg/L; overall, 69% of the samples analysed for total Cd were below the detection limit, whereas 96% of the samples for the dissolved fraction fell below the detection limit.

4.2.3 Thompson-Okanagan

Data from 63 stations provided information on the background concentrations of Cd in the Thompson-Okanagan region. Mean concentrations of total Cd by station ranged from < 0.005 to 0.173 µg/L for all data (n = 54 stations). Mean concentrations of dissolved Cd by station ranged from < 0.01 to 0.0300 µg/L (n = 22 stations).

Detection limits for samples used to estimate background Cd concentrations in the Thompson-Okanagan region ranged from 0.005 to 0.01 µg/L for total Cd and the detection limit was 0.01 µg/L for the dissolved fraction. Overall, 31% of the samples analysed for total Cd fell below the detection limit, whereas 77% of the data for dissolved Cd were below the detection limit.

4.2.4 Kootenay

Data collected from 37 reference sites were used to estimate background Cd concentrations in the Kootenay region. Mean total Cd concentrations by station ranged from 0.00435 to 0.115 µg/L (n = 37 stations) when all data were considered. Dissolved Cd data were only provided for one station with the estimated mean concentration for this station being 0.006 µg/L.

Detection limits of samples used to estimate background Cd concentrations in the Kootenay region ranged from 0.001 to 0.01 µg/L. Overall, only 5% of the samples analysed for total Cd fell below the detection limit, whereas all samples analysed for dissolved Cd were above the detection limit.

4.2.5 Cariboo

Data used to estimate background Cd concentrations for the Cariboo region were collected from 17 reference sites. Compared to rivers and creeks in the other regions, mean total Cd concentrations in the Cariboo region were among the highest in samples collected. In addition, the concentrations of dissolved Cd measured in all samples collected were among the highest in the province. Mean concentrations of total Cd by station ranged from 0.00345 to 0.132 µg/L for all data (n = 17 stations). Mean concentrations of dissolved Cd by station ranged from 0.0372 to 0.145 µg/L (n = 15 stations).

Detection limits for samples used to estimate background Cd concentrations in the Cariboo region ranged from 0.005 to 0.05 µg/L for total Cd and 0.005 to 0.017 µg/L for dissolved Cd. Overall, 68% of samples analysed for total Cd fell below the detection limit, whereas 74% of samples were below the detection limit for dissolved Cd.

4.2.6 Skeena

Data collected from 127 lotic and 7 lentic reference stations provided information on the background concentrations of Cd in the Skeena region, which were among the highest in the province for total Cd. Mean concentrations of total Cd by station ranged from < 0.005 to 0.151 µg/L for all data (n = 134 stations). Mean concentrations of dissolved Cd by station ranged from < 0.01 to 0.03 µg/L (n = 17 stations).

Detection limits for samples used in the analysis of background Cd concentrations in the Skeena region ranged from 0.001 to 0.03 µg/L for total Cd and from 0.01 to 0.017 µg/L for dissolved Cd. Overall, 23% of the samples analysed for total Cd fell below the detection limit, whereas 75% of the samples for the dissolved Cd fraction were below detection limits.

4.2.7 Omineca-Peace

Data collected from 46 reference sites were used to estimate background Cd concentrations in the Omineca-Peace region, which were among the highest in the province for total Cd data collected. Mean total Cd concentrations by station ranged from 0.00397 to 0.123 µg/L (n = 46 stations) when all data were considered. Station mean dissolved Cd concentrations ranged from < 0.0085 to 0.0558 µg/L.

Detection limits for samples used to estimate background Cd concentrations in the Omineca-Peace region ranged from 0.001 to 0.05 µg/L for total Cd and 0.0085 to 0.05 µg/L for dissolved Cd. Overall, 23% of samples analysed for total Cd fell below the detection limit and 48% of samples analysed for dissolved Cd were below detection limits.

4.2.8 Summary

In general, Cd concentrations were found to be lower in southern BC compared to central and northern regions, which may be attributed to variability in local rock composition and ambient conditions such as climate, soil type, pH, and water quantity and flow regime. In general, the mean total Cd concentrations by station were the lowest on Vancouver Island and in the Kootenay region, whereas the highest concentrations were found in samples collected in the Cariboo, Skeena, and Omineca-Peace regions. Overall, mean total Cd concentrations by station in BC waters ranged from 0.00287 to 0.173 µg/L. Mean dissolved Cd concentrations by station ranged from 0.00275 µg/L to 0.145 µg/L (Table 4).

5.0 Environmental Fate and Transport

Cd is released into the environment through a variety of natural processes and anthropogenic activities. Although Cd and Cd compounds are typically non-volatile, combustion processes may emit Cd into the atmosphere as part of oxide, chloride, and sulphate complexes. These particles can be transported over long distances (hundreds to

thousands of kilometres) before being deposited onto the soil or surface water (ATSDR 2012). In addition, Cd can be released onto soils through the application of phosphate fertilizers (ATSDR 2012). Dependent upon the pH of the soil and the abundance of organic matter, Cd can become immobilized in the soils, becoming available for uptake to plants and into the food web (ASTDR 2012). However, Cd may become more mobile under low pH conditions and be transported into surface waters and groundwater.

Compared to most other heavy metals, Cd is relatively mobile in aquatic systems. In water, Cd typically exists as a hydrated ion or as a component of ionic complexes with other substances (e.g., cadmium chloride, cadmium nitrate, and cadmium sulphate; ASTDR 2012), which migrate easily in the water column. Under high pH (i.e., alkaline) conditions however, Cd can form insoluble complexes with carbonate ($CdCO_3$) or hydroxide ($Cd[OH]_2$) and settle out in bottom sediments (Hahne and Kroontje 1973; Guegen *et al.* 2003). Additionally, in organic-rich waters, Cd readily adsorbs to humic acids and other organic substances (USEPA 1979).

In sediments, Cd is primarily found as a component of insoluble complexes (e.g., $CdCO_3$) or adsorbed to organic matter (e.g., humic acid ligands). In addition, bacteria in sediment may play a role in the partitioning of Cd to sediments from the water column (Burke and Pfister 1988). The potential for Cd to re-mobilize from bottom sediments to the water column is determined by a number of factors. Cd that partitions into sediments by complexation with carbonate minerals or co-precipitation with hydrous iron oxides is less likely to re-mobilize by turbulence (i.e., disturbance of the sediments). However, Cd that is adsorbed to sediment surfaces (e.g., clay or organic matter) is more readily released to a dissolved state. In addition, Cd (as well as other metals) may disassociate from sediments under certain conditions (USEPA 1979).

The degree of Cd toxicity to aquatic biota is dependent on its bioavailability in the water column. The Cd ion is readily taken up by multiple aquatic organisms (including algae, plants, aquatic invertebrates, fish, and amphibians) by interfering (i.e., competing) with

calcium ions for Ca^{2+} receptors on the tissues of organisms (Verbost *et al.* 1987; 1989; Playle *et al.*1993a; 1993b; Playle 1998). Generally, ambient conditions that favour the ionic form of Cd result in conditions that are most toxic to aquatic species (Campbell 1995). Further, there is some evidence to support that in addition to uptake (i.e., bioconcentration) of Cd through direct contact (e.g., at the gill-water interface), bioaccumulation of Cd through ingestion of contaminated food sources may contribute to the Cd body burden and thus toxicity in fish (Farag *et al.* 1994; Woodward *et al.* 1995) and invertebrates (van Hattum *et al.* 1989). However, there is no evidence to support the possibility that Cd biomagnifies in the aquatic environment (i.e., increases in concentration in higher trophic levels).

6.0 Bioaccumulation and Bioconcentration of Cadmium in the Aquatic Environment

Field measurements of contaminants in water, sediment, and tissue provide information on the concentrations of the contaminant in the aquatic environment. However, these measurements provide little information about the bioaccumulation or bioconcentration of the contaminants in the tissues of aquatic organisms and the potential transfer of those contaminants to higher trophic levels. Bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) express the ratio of contaminant concentration in the ambient environment to the contaminant concentration within an organism (Arnot and Gobas 2006). The BCF estimates the relationship between the uptake and retention of a chemical by an aquatic organism from the ambient water to excretion of that chemical (Barron 1990; Meylan *et al.* 1999; Arnot and Gobas 2006), whereas BAFs incorporate the concentrations of a chemical from all surrounding media (i.e., water, sediment, and food; Arnot and Gobas 2006). Therefore, bioconcentration and bioaccumulation express the competing rates of chemical uptake and loss by aquatic organisms, which depend on a number of factors such as the concentration and properties of the chemical (i.e., speciation), ambient conditions (e.g., hardness, temperature, salinity) and the type of exposure (Taylor 1983). Organism physiology (i.e., mechanisms of uptake, excretion,

and detoxification) and food web structure are also important factors. A BCF is typically calculated from data derived in laboratory studies, while a BAF is usually calculated using field measurements (Arnot and Gobas 2006), but can be calculated from properly designed laboratory studies. Although BCFs and BAFs are important for some contaminants, they are not recommended for use with metals (McGeer *et al*. 2003; Fairbrother *et al.* 2007). A review of the literature found that BCFs for Cd had high variability and were inversely related to exposure concentration (McGeer *et al.* 2003).

The purpose of this section is to provide information on the studies that have been conducted to investigate the potential for bioconcentration and bioaccumulation of Cd. While the general consensus in the literature suggests that aquatic plants uptake Cd readily, there is considerable variability in calculated BCFs. Phytoplankton have been found to uptake Cd rapidly, adversely affecting growth and photosynthesis (Hutchinson 1973; Klass *et al.* 1974; Cossa 1976; Conway and Williams 1979). Bioconcentration factors for phytoplankton of up to 24,000 (unspecified moisture basis) have been reported, with values typically decreasing at the highest range of exposure levels (Cain *et al.* 1980; Conway and Williams 1979; Ferard *et al.* 1983). For example, Cain *et al.* (1980) reported BCFs between 329 and 4,900 (dry weight [DW] basis) in 14-d exposures of freshwater phytoplankton to Cd concentrations of 10 to 2,000 µg/L Cd, and found that maximum uptake efficiency occurred at the lower concentration rather than the concentration that resulted in the greatest accumulation. Ferard *et al.* (1983) reported BCFs for phytoplankton ranging from 1,850 to 3,000 (DW basis) after a 10-d exposure to 10 to 250 µg/L Cd. Conway and Williams (1979) found markedly higher BCFs of 3,500 to 24,400 (unspecified moisture basis) in concentrations of 0.05 to 8.5 µg/L Cd; in this short-term study, the initial sorption was observed during the first 5-10 minutes of Cd exposure (Conway and Williams 1979). Investigations have found the uptake of Cd by aquatic plants to quickly reach steady-state concentrations, with the accumulation of Cd in roots to be greater than the accumulation in leaves (e.g., Giesy *et al.* 1981). A mixture of algae and small crustaceans, in a channel microcosm study, also exhibited rapid uptake of Cd, reaching steady-state in less than 23 days (Giesy *et al.* 1981). The BCF calculated

from the data was 7,200 when the community was exposed to 5 µg/L Cd and 5,800 when exposed to 10 µg/L Cd (DW basis; Giesy *et al.* 1981).

Invertebrates tend to exhibit larger BCFs at lower Cd water concentrations (Marshall 1978; Spehar *et al.* 1978; Giesy *et al.* 1981). When the cladoceran, *Daphnia galeata mendotae,* was exposed to four different concentrations of Cd (1, 2, 4, 8 µg/L) for 22 weeks, the BCF decreased from 17,600 to 6,463 (DW basis) as the water concentration increased (Marshall 1978). Though it was not stated whether steady-state was reached, the long period of exposure (22 weeks) suggests that the BCFs were calculated when concentrations were at steady-state (Marshall 1978; ASTM 2012a). In an experiment exposing stoneflies (*Pteronarcys dorsata*), caddisflies (*Hydropsyche betteni*), and snails (*Physa integra*) to concentrations ranging from 3 to 238 µg/L Cd, Spehar *et al.* (1978) found an increase in whole-body Cd concentration as the exposure water concentration increased, with the whole-body concentrations (DW) ranging from 600 to 30,000 times greater than the associated water concentrations. While it was not stated whether steady-state was reached, BCFs calculated for stoneflies after 28 days of exposure ranged from 798 in exposure chambers with a concentration of 238 µg/L Cd to 4,096 in exposure chambers with a concentration of 8.3 µg/L Cd. For caddisflies, these BCFs ranged from 1,260 at 238 µg/L Cd to 31,667 at 3 µg/L Cd, while for snails the BCFs ranged from 6,024 at 8.3 µg/L Cd to 13,667 at 3 µg/L Cd (analysis of graphical data; DW basis; Spehar *et al.* 1978). Giesy *et al.* (1981) obtained similar BAFs (DW basis), with concentrations of Cd between 820 and 17,600 times greater in the bodies of beetles (Coleoptera), dragonflies (Anisoptera), damselflies (Zygoptera), midges (Chironomidae and Ceratopogonidae), and mayflies (Ephemeroptera) than in the water of exposure chambers with concentrations of 5 or 10 µg/L Cd. These BAFs were highest in the detritivores and herbivores (Ephemeroptera and Chironomidae; Giesy *et al.* 1981).

Crayfish (*Cambarus latimanus*) exposed to 5 and 10 µg/L Cd for 5 months were found to have whole-body BAFs of 2,980 and 2,196, respectively (DW basis; Thorp *et al.* 1979). In comparison, crayfish (*Procambarus acutus acutus*) exposed to 5 and 10 µg/L Cd for

21 days accumulated significantly more Cd in the gills than controls, but not in dorsal tail-muscle tissue (Dickson *et al.* 1982). Although it was not stated whether steady-state was reached, based on the water Cd concentrations, the BCFs (using gill tissue) were 40,808 for crayfish exposed to 5 µg/L Cd and 17,936 in 10 µg/L Cd exposures (DW basis). However, despite the large increase in gill Cd concentration, there was not a significant difference in mortality or molting between Cd-exposed and control crayfish (Dickson *et al.* 1982). Differences in Cd uptake between different tissue types were also seen in the freshwater mussel *Dreissena polymorpha* (Herwig *et al.* 1989). After 4 weeks of exposure to 100 µg/L Cd, the Cd concentration in the shell had reached a steady-state concentration of approximately 30 µg/g Cd (DW). However, the Cd concentration in the soft body tissue increased linearly over the exposure period, to about 290 µg/g Cd (DW; Herwig *et al.* 1989). Although 4 weeks is generally considered a reasonable length of time for reaching steady-state (ASTM 2012a), the linear increase in Cd concentration shown in Herwig *et al.* (1989) implies that the BCF calculated in this case would not be accurate. An experiment exposing isopods (*Asellus aquaticus*) to Cd through food and water also did not reach steady-state (van Hattum *et al.* 1989) after a 30-d exposure. However, using a first order one-compartment bioaccumulation model, van Hattum *et al.* (1989) estimated the steady-state Cd concentration in *A. aquaticus* and calculated a BCF of 17,560 ± 9,960 and a BAF of 0.082 ± 0.068 (both DW), where the BAF was considered to represent the accumulation of Cd from food alone. Over 89% of the Cd uptake was estimated to be from direct contact with water, except for in the treatment with high Cd in the food source, when food and water each contributed 50% of the Cd taken up by *A. aquaticus* (van Hattum *et al.* 1989).

In fish, Cd appears to preferentially accumulate in certain organs relative to the whole body overall. A study exposing rainbow trout (*Oncorhynchus mykiss*) fingerlings to 4 µg/L Cd for 10 weeks found the highest Cd concentrations in the kidney, followed by the liver and the gills (Kumada *et al.* 1980). Similarly, when Benoit *et al.* (1976) exposed 3 generations of brook trout (*Salvelinus fontinalis*) to Cd, the first generation was exposed for 38 weeks and the highest concentration (about 50 µg/g, DW) of Cd was found in the

kidney, followed by the liver and gills. Cd appeared to have reached steady-state in the kidney, giving a BCF of about 14,000 (DW basis) for that organ for fish exposed to 3.4 µg/L Cd (Benoit *et al.* 1976). In second generation fish exposed to 3.4 µg/L Cd for 70 weeks from fertilization, the kidney again had the highest Cd concentration, with a BCF of about 19,000 (DW basis). In the third generation, only whole-body tissue residues were analysed; the BCF of these juveniles exposed to 3.4 µg/L Cd was 371, while for fish exposed to 0.9 µg/L Cd the BCF was 756 (DW basis; Benoit *et al.* 1976).

By modelling uptake and elimination rates, Giesy *et al.* (1981) calculated the steady-state concentrations of Cd in mosquitofish (*Gambusia affinis*) after 139 days. Using these numbers, the BAFs were 7,156 and 6,100 for fish exposed to 5 and 10 µg/L Cd, respectively (DW basis; Giesy *et al.* 1981). In contrast, fathead minnows (*Pimephales promelas*) exposed to 48.8 µg/L Cd reached a steady-state concentration of approximately 7 µg/g in whole-body tissue after 50 days, giving a BCF of around 150 (though the whole-body concentration peaked after 46 days at 10.7 µg/L; Sullivan *et al.* 1978). Hansen *et al.* (2002b) found that juvenile bull trout (*Salvelinus confluentus*) exposed to a range of Cd concentrations (0.052 to 0.786 µg/L Cd) accumulated increased amounts of Cd with increasing water concentration at each time point sampled (20, 40, and 55 days of exposure). The concentration of Cd also increased over time within a given exposure concentration, and steady-state was not reached after 55 days in the higher concentrations. However, the 2 lowest exposure concentrations resulted in similar whole-body Cd concentrations at all 3 sampling times; the BCFs in exposures of 0.052 and 0.089 µg/L Cd were approximately 3,000 and 2,000, respectively (DW basis; Hansen *et al.* 2002b). Exposing rainbow trout to higher Cd concentrations (3 and 10 µg/L Cd) for 30 days resulted in lower whole-body BCFs (167 and 120, respectively; wet weight basis; Hollis *et al.* 1999). The gills accumulated a greater proportion of Cd on a per gram basis, with BCFs of 2,000 and 1,200 (weight wet basis) for fish exposed to 3 and 10 µg/L Cd, respectively (Hollis *et al.* 1999). Although steady-state was not reached in this case, the 30-d exposure period was longer than the 28 days recommended for calculating a BCF for fish (ASTM 2012a).

In an even longer-term experiment, Cearley and Coleman (1974) found that Cd concentrations in largemouth bass (*Micropterus salmoides*) and bluegill (*Lepomis macrochirus*) were at steady-state when sampled at 2 and 4 months of exposure. When exposed to 8 µg/L Cd, the BCFs for the bass gills and body carcass were 2.0, while for the internal organs, the BCF was 2.75. For all body components the BCF was less than one when the fish were exposed to 80 µg/L Cd. Only the whole body of the bluegill was analysed; the calculated BCF was 1.75 and 2.12 when exposed to 8 and 850 µg/L Cd, respectively (all BCFs on a DW basis; Cearley and Coleman 1974). These numbers were all calculated using body concentrations estimated from graphs, so it is possible that these BCFs are not as accurate as those reported from other studies. In another long-term study, Giles (1988) looked at Cd accumulation in several organs in rainbow trout over 178 days. The gills accumulated the most Cd, while Cd in the muscle did not increase significantly compared to the control in both Cd treatments. Based on the calculated average whole-body Cd concentrations, the BAFs (DW basis) for this study were 36 for the 3.6 µg/L Cd exposure and 30 for the 6.4 µg/L Cd exposure, although the whole-body Cd burden may not have reached steady-state (Giles 1988).

There is some evidence that it is important to consider ingestion of Cd from food sources rather than only considering direct contact with water. Farag *et al.* (1994) exposed adult rainbow trout to a mixture of metals, including Cd, through water and/or food. They found significantly more Cd in the gills and liver of fish exposed to metals through water than in controls or in fish exposed through food only. However, there was significantly more Cd in the stomach and pyloric caeca in fish exposed through food than in the control (i.e., no exposure through food or water) suggesting that the ingestion of Cd-contaminated prey may play a role in Cd body burden (Farag *et al.* 1994). In addition, rainbow trout and brown trout exposed to metal-contaminated food for 88 days post-hatch had significantly decreased weight compared to control fish; only brown trout weighed significantly less than controls when exposed to contaminants through water alone (Woodward *et al.* 1995). After 84 days of exposure, feeding activity was decreased in rainbow trout fed contaminated food. As well, the whole body concentration of Cd in

brown trout was significantly increased after 88 days due to exposure through food and water, but rainbow trout only accumulated Cd from the water exposure (Woodward *et al.* 1995). Kraemer *et al.* (2006) compared Cd uptake in yellow perch (*Perca flavescens)* from food with uptake from water. Evidence for uptake through water was stronger than uptake through food, though there was some accumulation of Cd from the diet. However, statistical comparisons of the treatments could not be completed due to high mortality in the reference and test treatments. In addition, water hardness in the test and reference lakes was not measured, which may have influenced waterborne Cd uptake (Kraemer *et al.* 2006). Nonetheless, *in vivo* and *in vitro* studies with yellow perch suggested that Cd uptake is controlled at the gut rather than at the gills (Klinck *et al.* 2007). An *in vitro* study with rainbow trout found that the presence of excess iron decreased Cd accumulation in some parts of the gastro-intestinal tract, and iron absorption in the mucosal epithelium was inhibited in the presence of Cd (Kwong and Niyogi 2009). Therefore, it appears that Cd enters the fish gastro-intestinal tract through the same pathway as iron, and a diet rich in iron might protect fish from Cd uptake and toxicity (Kwong and Niyogi 2009).

Although there is some evidence of Cd accumulation from the diet (Farag *et al.* 1994; Woodward *et al.* 1995), there is little evidence to support that Cd biomagnifies in the food web (i.e., increases in concentration with increasing trophic level; Nfon *et al.* 2008). When looking at Cd transfer through multiple trophic levels (algae, cladocerans, fish), Ferard *et al.* (1983) only found evidence of lower trophic levels exhibiting bioaccumulation; the alga *Chlorella vulgaris* had a mean BCF of approximately 2,200 (DW basis). While the concentration of Cd in the daphnids increased as the Cd concentration in the algae they were exposed to increased, they did not accumulate as much Cd as the algae on a µg/g basis (BAFs < 1 on a DW basis). Fish (*Leucaspius delineatus*) feeding on the algae and daphnids did not show a relationship between Cd exposure and tissue concentration; fish accumulated less than 1 µg/g DW of Cd at all Cd exposure levels (Ferard *et al.* 1983). Similarly, Williams and Giesy (1978) and Hatakeyama and Yasuno (1982) found that fish took up more Cd through contaminated

water than food, suggesting Cd does not biomagnify with increasing trophic level. Nfon *et al.* (2009) found no evidence of Cd biomagnification in a pelagic food chain in the Baltic Sea; in fact, Cd was diluted in tissues as trophic levels increased from zooplankton to herring. Direct exposure from water is the primary pathway for Cd toxicity. Therefore, the WQGs presented in this document were developed based on direct exposure toxicity data.

7.0 Toxicity of Cadmium to Freshwater Aquatic Organisms

Cd has been identified as a metal of major importance by multiple agencies including the Agency for Toxic Substances and Disease Registry, Environment Canada, Health Canada, and the U.S. Environmental Protection Agency due to its toxicity to humans and wildlife. The following sections provide information on the mode of toxic action of Cd in freshwater aquatic organisms, and the factors which affect the toxicity and bioavailability of Cd in the aquatic environment. Typically, values presented in the following sections reflect dissolved concentrations of Cd from the dissolution of Cd salts. To facilitate the assessment of the toxicological data to derive short-term maximum and long-term average WQGs for Cd, all of the toxicological data were normalized to a standard hardness of 50 mg/L $CaCO_3$. The details and justification of the normalization procedure are presented in Section 9.3.

7.1 Mode of Toxicity

Cd is known to interfere with the uptake of calcium at calcium binding sites of aquatic organisms (Verbost *et al.* 1987; 1989; Reid and McDonald 1988), which is linked to adverse effects which include increased mortality, decreased growth, and decreased reproductive capacity and success.

In aquatic plants and algae, Cd uptake causes adverse effects by inhibiting photosynthesis, growth, and chlorophyll synthesis, therefore decreasing water and nutrient uptake (Faller *et al.* 2005). In aquatic invertebrates, Cd inhibits the influx of

calcium, causing cellular damages (Soegianto *et al.* 1999), and affects osmoregulation (Felton *et al.* 2008). In fish, Cd inhibits calcium uptake at the gills (Verbost *et al.* 1987; 1989). The magnitude of calcium inhibition depends on the concentration, bioavailability in the aquatic environment, and exposure time (Verbost *et al.* 1989).

7.2 Toxicity to Algae and Aquatic Plants

There have only been a few studies completed for evaluating the toxicity of Cd to algae and aquatic plants. However, the applicable data have been acquired and evaluated for the purpose of deriving a short-term maximum and/or long-term average WQG for Cd. While Cd is generally considered to be a non-essential metal as it has no known biological/biochemical functions in plants (Drost *et al.* 2007), it is taken up by plants quite readily. The uptake of Cd may affect metabolic activities, leading to adverse effects including inhibition of photosynthesis and growth, and a decrease in water and nutrient uptake (Faller *et al.* 2005). Kwan and Smith (1991) suggested that Cd competes with calcium for binding sites in plants, interfering with calcium uptake; however, their experiment showed mixed results. A later study by Faller *et al.* (2005) reported that cadmium (Cd^{2+}) binds to calcium (Ca^{2+}) receptors during photoactivation, interfering with photosynthesis. Many plants (including aquatic plants) can become acclimated to elevated levels of Cd by producing phytochelatins, which bind with the cadmium ions, effectively forming complexes that mitigate the competitive nature of the ions (Grill *et al.* 1985; Kobayashi *et al.* 2006). Similarly, in tests conducted with the algae, *Selenastrum capricornutum* (now known as *Pseudokirchneriella subcapitata*), reductions in growth had recovered with the addition of the chelating agent ethylenediaminetetraacetic acid (EDTA; Thompson and Couture 1990).

Duckweed (*L. minor*), a common freshwater macrophyte in BC waters, was studied in 72-hour (h) and 7-day (d) exposures (Drost *et al.* 2007). In the 72-h exposure, an effective concentration affecting 50% of the population (EC_{50}) for growth rate of 393 µg/L Cd was observed. In the same study, long-term exposure to Cd resulted in a 7-d

EC_{50} of 214 µg/L Cd. Both experiments were conducted in exposures with a water hardness of 166 mg/L $CaCO_3$. However, the growth rate of duckweed recovered to that observed in control exposures after being removed from the treatment for an additional 72-h. While growth rates recovered, the concentration of Cd in the tissues of the plant did not decrease (i.e., there was no depuration of the Cd; Drost *et al.* 2007). In a study conducted with the green alga, *P. subcapitata,* Källqvist (2009) found that the observed 72-h EC_{50} for growth ranged from 9.4 to 199 µg/L Cd over an increasing range of water hardness (i.e., 3.4 to 46.2 mg/L $CaCO_3$).

7.3 Toxicity to Aquatic Invertebrates

Toxicity of Cd to aquatic invertebrates is highly variable between taxonomic groups. Within a taxonomic group, variability in observed effects is often dependent on factors including life-stage, measured endpoint (e.g., reproduction), duration of exposure, and water quality in the laboratory exposures (e.g., water hardness). In aquatic invertebrates, the adsorption of Cd causes cellular damages that include structural changes to the epithelial cells and decreases in the number of apical microvilli, basal infoldings, and mitochondria of crustacean gills (Soegianto *et al*. 1999). Cd also inhibits the influx of calcium, by competing for binding-sites at the environment-tissue interface, which affects osmoregulation (Felton *et al.* 2008). To facilitate the assessment of toxicity of Cd to aquatic invertebrates, a summary of the short-term and long-term studies are discussed separately in the following sections.

7.3.1 Short-Term Toxicity to Aquatic Invertebrates

Adverse effects from short-term exposure to Cd in aquatic invertebrates vary greatly between the taxonomic groups. Crustaceans (e.g., cladocerans and amphipods) are among the most sensitive groups along with the hydra, *Hydra viridissima*, and various mussel species, while the aquatic life-stage of various insects including the midges, mayflies, and stoneflies are more tolerant during short-term exposure (McCahon and

Pascoe 1988; Suedel *et al.* 1997; Ward and Robinson 2005; Shaw *et al.* 2006; Brinkman and Johnston 2008).

From an assessment of the literature, the cladoceran, *D. magna*, was one of the most sensitive invertebrate species to Cd toxicity. In toxicity tests that were conducted in exposures with water hardness of 160 to 180 mg/L $CaCO_3$, the reported 48-h lethal concentration affecting 50% of the population (LC_{50}) was 26 µg/L Cd (95% confidence interval [CI_{95}] not reported; Ward and Robinson 2005). However, in the same study, the authors reported that *D. magna* exhibited a wide range of sensitivity to Cd, depending on the genetic strain used in each of the eight tests. The 48-h LC_{50} in the eights tests ranged from 26 to > 120 µg/L Cd. The amphipod, *Hyalella azteca*, was also found to be sensitive in short-term exposures to Cd. Suedel *et al.* (1997) reported a 96-h LC_{50} of 2.8 µg/L Cd (CI_{95}: 2.4 - 3.3 µg/L Cd) in toxicity tests performed at a water hardness between 6 and 28 mg/L $CaCO_3$. Holdway *et al.* (2001) reported a 96-h LC_{50} of 3.0 µg/L Cd (CI_{95} not reported) for the hydra, *H. viridissima*, in toxicity tests conducted at a water hardness between 19 and 20 mg/L $CaCO_3$. Mussels were also determined to be relatively sensitive to Cd exposure. For example, Black (2001) reported a 96-h LC_{50} of 19.1 µg/L Cd (CI_{95} not reported) in exposures performed at a hardness of 84 mg/L $CaCO_3$.

Generally, the aquatic life-stage of insects such as mayflies, midges, caddisflies, and stoneflies is relatively more tolerant of short-term exposure to Cd. Mebane *et al.* (2012) performed toxicity tests with the mayfly larvae, *Baetis tricaudatus*, in exposures with water hardness between 21 and 59 mg/L $CaCO_3$. The 96-h LC_{50} reported from the test ranged between 16 µg/L Cd (CI_{95}: 1 - 314 µg/L Cd) and > 444 µg/L Cd. In the same study, another species of mayfly, *Rithrogena* sp., was tested in similar exposures; the observed 96-h LC_{50} ranged from > 50 to 157 µg/L Cd (CI_{95}: 90 - 273 µg/L Cd). In another study with the mayfly, *R. hageni*, Brinkman and Johnson (2008) reported a comparatively higher 96-h LC_{50} of 10,500 µg/L Cd (CI_{95} not reported), in exposures with water hardness of 48 mg/L $CaCO_3$. In toxicity tests performed by Watts and Pascoe (2008), two species of midge larvae (*Chironomus tentans* [now known as *Chironomus*

dilutus] and *Chironomus riparius*) were tested in exposures with water hardness of 114 mg/L $CaCO_3$. The 48-h LC_{50}s reported from the toxicity tests were 9,340 µg/L Cd (3,400 - 25,300 µg/L Cd) for *C. dilutus* and 2,620 µg/L Cd (CI_{95}: 1,800 - 3,800 µg/L Cd) for *C. riparius*. Results from other studies with the midge, *C. dilutus*, showed similar results with a 48-h LC_{50} of 29,560 µg/L Cd (CI_{95}: 21,250 - 37,870 µg/L Cd) in exposures with water hardness between 6 and 28 mg/L $CaCO_3$.

7.3.2 Long-Term Toxicity to Aquatic Invertebrates

In the studies of long-term exposure to Cd that were evaluated, crustaceans (e.g., cladocerans and amphipods) were the most sensitive aquatic invertebrate species. Elnabarawy *et al.* (1986) reported a 7-d EC_{16} for reproduction of 0.2 µg/L Cd for *Ceriodaphnia reticulata* in exposures conducted with water hardness of 240 mg/L $CaCO_3$. In the same study, the 14-d EC_{16} for reproduction for *D. pulex* was also reported as 0.2 µg/L Cd (CIs not reported), however these estimates are considered less reliable due to limitations of the study design and statistical analyses. Chadwick Ecological Consultants, Inc. (2004) also determined that reproduction in cladocerans was more sensitive in long-term exposures than other endpoints such as survival. The 21-d inhibitory concentration causing a 20% inhibition in tested organisms (IC_{20}) for reproduction in *D. magna* was reported as 2.23 µg/L Cd in exposures with water hardness of 99 mg/L $CaCO_3$.

In long-term exposures to Cd, biomass was also determined to be relatively more sensitive than survival, indicating that sub-lethal concentrations of Cd in aquatic habitats can have adverse effects on aquatic invertebrates. In tests conducted with the amphipod, *H. azteca*, Ingersoll and Kemble (2001) reported a 28-d IC_{25} for biomass of 0.51 µg/L Cd (CI_{95} not reported) compared to a 28-d LC_{25} of 2.1 µg/L Cd (CI_{95} not reported) in exposures performed at a water hardness of 280 mg/L $CaCO_3$. Similar results were reported from Chadwick Ecological Consultants, Inc. (2004), where the 28-d IC_{20} for biomass in tests conducted with *H. azteca* ranged from 0.50 to 0.76 µg/L Cd (CI_{95} not reported) in exposures with water hardness of 126 and 153 mg/L $CaCO_3$, respectively.

In a study completed by Suedel *et al.* (1997), the long-term exposure of *H. azteca*, *D. magna*, and *C. reticulata* to Cd was evaluated. Of the crustaceans, *H. azteca* was determined to be the most sensitive to Cd in both 7-d and 14-d exposures. The lowest observed effect concentration (LOEC) for survival reported in the study for the 14-d test was 0.25 µg/L Cd. In exposures with the cladoceran, *C. reticulata*, conducted at a similar water hardness (between 6 and 28 mg/L $CaCO_3$), the 14-d LOEC for survival was reported as 13 µg/L Cd. In tests with the cladoceran, *D. magna*, exposures with a higher water hardness (between 69 and 87 mg/L $CaCO_3$) were used. The 7-d LOEC for survival was reported as 10 µg/L.

Similar to the results of the assessment of short-term toxicity to aquatic invertebrates, it was found that the aquatic life-stage of insects such as midges and mayflies are relatively more tolerant of long-term exposure to Cd. Ingersoll and Kemble (2001) performed life-cycle tests (60-d) with the midge, *C. dilutus*. The life-cycle tests are designed to evaluate multiple endpoints, including developmental (e.g., survival, growth, and emergence) and reproductive (e.g., fecundity) endpoints. The authors reported that the percent of eggs hatched was the most sensitive endpoint in the test. The IC_{25} was reported as 4.0 µg/L Cd (CI_{95} not reported) at a water hardness of 280 mg/L $CaCO_3$. For the developmental endpoints, individual weight was observed to be the most sensitive of the endpoints measured with an IC_{25} of 9.9 µg/L Cd (CI_{95} not reported). In 10-d tests with the mayfly, *R. hageni*, Brinkman and Johnson (2008) measured the effects of long-term exposure to Cd on moulting rate and survival. The authors observed a LOEC for both moulting rate and survival of 1,880 µg/L Cd in exposures with water hardness of 48 mg/L $CaCO_3$.

In addition to the aquatic life-stage of insects, mussels also exhibited a relative tolerance to long-term exposure to Cd. Lasee (1991) observed a LOEC for anterior shell length in *Lampsilis ventricosa* of 10 µg/L in tests using a water hardness of 145 mg/L $CaCO_3$. Similar results were obtained from a study completed by Wang *et al.* (2010); the reported IC_{20} in the 28-d test with *L. siliquoidea* was 5.0 µg/L Cd in exposures with a water hardness of 47 mg/L $CaCO_3$.

7.4 Toxicity to Fish

Toxicity of Cd to fish is highly variable between taxonomic groups. Within a taxonomic group, variability in observed effects is often dependent on factors including the measured endpoint (e.g., reproduction), duration of exposure, water quality in the laboratory exposures (e.g., water hardness), and life-stage. For example, with the exception of newly-hatched steelhead trout (*O. mykiss*) alevins, which were more tolerant to Cd exposure, toxicity was greater for the early life-stages (i.e., swim-up and parr) than for smolts (Chapman 1978). Similarly, Reid and McDonald (1988) found Cd to be more toxic to juvenile rainbow trout relative to adult fish. In fish, as in other aquatic organisms, Cd inhibits the influx of calcium ions through receptor sites in the gills, which may lead to cellular impairment and/or death (Verbost *et al.* 1987; 1989). To facilitate the assessment of toxicity of Cd to fish, a summary of the short-term and long-term studies are discussed in the following sections.

7.4.1 Short-Term Toxicity to Fish

Cd is highly toxic to salmonid fish during short-term exposure, and in particular, to the rainbow trout (*O. mykiss*; e.g., Buhl and Hamilton 1991; Davies *et al.* 1993; Hansen *et al.* 2002a; Sloman *et al.* 2003; Besser *et al.* 2007; Mebane *et al.* 2012). Of the studies examined from the primary literature, rainbow trout was the most sensitive fish species, exhibiting a relatively wide sensitivity range depending on life-stage and water quality (e.g., water hardness, pH, and water temperature). Hansen *et al.* (2002a) ran a series of toxicity tests with rainbow trout fry in exposures with varied water hardness (29.3 to 89.3 mg/L $CaCO_3$), pH (6.5 to 7.5), and water temperature (7.6 to 12.1°C). The reported 120-h LC_{50} ranged from 0.35 (CI_{95}: 0.33 - 0.37 µg/L Cd) to 2.07 µg/L Cd (CI_{95}: 1.83 - 2.35 µg/L Cd). Similarly, Mebane *et al.* (2012) reported a 96-h LC_{50} for fry that ranged between 0.34 (CI_{95}: control - 0.50 µg/L Cd) and < 2.9 µg/L Cd. Cuisimano (1986) also reported a similar 96-h LC_{50} in exposures with similar pH (between 5.7 and 7.0) and very soft water (9.2 mg/L $CaCO_3$) of between < 0.5 and 0.7 µg/L Cd (CI_{95}: 0.6 - 0.8 µg/L Cd). The authors reported a markedly higher 96-h LC_{50} of 28 µg/L Cd (CI_{95}: 22 - 37 µg/L Cd)

in exposures with pH 4.7. Other salmonids were notably more tolerant of Cd in short-term exposures. Hansen *et al.* (2002a) reported a 120-h LC_{50} for the bull trout, *S. confluentus*, that ranged from 0.83 (CI_{95}: 0.76 - 0.91 µg/L Cd) to 5.23 µg/L Cd (CI_{95}: 4.63 - 5.89 µg/L Cd) in exposures with varied water hardness (29.3 to 89.3 mg/L $CaCO_3$), pH (6.5 to 7.6), and water temperature (7.6 to 12.1°C). Chapman (1978) conducted a study that evaluated the effects of short-term exposure to Cd on the chinook salmon, *Oncorhynchus tshawytscha*. The 96-h LC_{50} reported from the test varied depending on the life-stage of the fish. The most sensitive of the life-stages was the swim-up stage. The reported 96-h LC_{50} in exposures with water hardness of 23 mg/L $CaCO_3$ was 1.8 µg/L Cd (CI_{95}: 1.7 - 2.0 µg/L Cd). The least sensitive of the life stages was the newly-hatched alevin with a reported 96-h LC_{50} of > 26 µg/L Cd.

Generally, non-salmonid fish were as sensitive to Cd during short-term exposures as the salmonid fish, with the exception of rainbow trout. The shorthead sculpin (*Cottus confusus*) exhibited the lowest 96-h LC_{50} of the non-salmonid fishes of 0.93 µg/L Cd (CI_{95}: 0.62 - 1.4 µg/L Cd; Mebane *et al.* 2012). Newly-hatched mottled sculpin (*Cottus bairdi*) were also relatively sensitive to short-term exposure to Cd with a reported 96-h LC_{50} of 2.9 µg/L Cd (CI_{95}: 2.2 - 3.8 µg/L Cd; Besser *et al.* 2007). Palawski *et al.* (1985) reported a 96-h LC_{50} for the striped bass (*Morone saxtilis*) that ranged from 4 (CI_{95}: 3 - 6 µg/L Cd) to 75 µg/L Cd (CI_{95}: 59 - 96 µg/L Cd) in exposures with increasing hardness (40 to 455 mg/L $CaCO_3$).

7.4.2 Long-Term Toxicity to Fish

In long-term toxicity studies, the rainbow trout was the most sensitive fish species to Cd. Other salmonid and non-salmonid fish exhibited similar sensitivities to long-term exposure to Cd. Of the endpoints tested, growth was observed to be more sensitive than survival, indicating that sub-lethal concentrations of Cd in aquatic habitats can have adverse effects on fish. In addition, few studies were available on the effects of Cd exposure on physiology or behaviour of fish, but the results of these studies indicated that these endpoints were not as sensitive relative to growth.

In toxicity tests with the early life-stage rainbow trout, Mebane *et al.* (2008) reported a 62-d LOEC for growth (i.e., weight) of 0.16 µg/L Cd in exposures with water hardness of 29.4 mg/L $CaCO_3$; however, the authors reported that no clear dose-response relationship was apparent for this endpoint. Comparatively, the 62-d LC_{10} from the same test was reported as 1.6 µg/L Cd (CI_{95} not reported). Besser *et al.* (2007) conducted 28-d toxicity tests with rainbow trout at the swim-up stage. The authors reported that the 28-d LOECs for biomass and survival were the same, 2.7 µg/L Cd in exposures with water hardness of 103 mg/L $CaCO_3$. Scott *et al.* (1993) reported from tests with juvenile rainbow trout, that in 7-d exposures with water hardness of 120 mg/L $CaCO_3$, the LOEC for behaviour (i.e., predator avoidance) was 2 µg/L Cd. Hansen *et al.* (2002b) reported a 55-d LOEC for growth and survival in bull trout (*S. confluentus*) of 0.787 µg/L Cd in exposures with a water hardness of 30.6 mg/L $CaCO_3$. Brown trout (*Salmo trutta*) exhibited a similar response in 30-d exposures with water hardness of 29.2 mg/L $CaCO_3$ with a reported IC_{20} for biomass of 0.87 µg/L Cd (CI_{95} not reported; Brinkman and Hansen 2007). However, the IC_{20} was markedly higher in additional tests from the same study that were conducted with higher water hardness of 67.6 and 151 mg/L $CaCO_3$. The IC_{20}s reported from these tests were 2.18 and 6.62 µg/L Cd (CI_{95} not reported). Of the salmonids evaluated from the primary literature, coho salmon (*Oncorhynchus kisutch*) were found to be the least sensitive to long-term Cd exposure. In 27-d exposures with water hardness of 45 mg/L $CaCO_3$, Eaton *et al.* (1978) reported a LOEC for biomass of 3.4 µg/L Cd. In longer exposures (i.e., 47- and 82-d), the reported LOEC was 12.5 µg/L Cd.

Sub-lethal effects were also observed in non-salmonid fish, including the mottled sculpin (*C. bairdi*). Besser *et al.* (2007) reported a LOEC for growth of 1.3 µg/L Cd in 21-d exposures with water hardness of 103 mg/L $CaCO_3$. Similarly, the 21-d LOEC for survival was also reported as 1.3 µg/L Cd. Castillo and Longley (2001) conducted a study to evaluate the effects of long-term exposure to Cd on the fathead minnow. Growth was determined to be more sensitive in the tests. The 7-d LOEC for growth was reported as 8.0 µg/L Cd in exposures with water hardness of 292 mg/L $CaCO_3$. Comparatively, the 7-d LOEC for survival ranged between 16.5 and 213.3 µg/L Cd in exposures with

water hardness varying between 261 and 285 mg/L $CaCO_3$. Although few long-term exposure studies have been completed with lake trout (*Salvelinus namaycush*), northern pike (*Esox lucius*), white sturgeon (*Acipenser transmontanus*), and white sucker (*C. commersoni*), these species appear to be less sensitive to Cd, reporting no low-effect values at the low end of the range exhibited by other species. Eaton *et al.* (1978) reported a 41- and 74-d LOEC for early life-stage biomass of 12.3 µg/L Cd in exposures with water hardness of 45 mg/L $CaCO_3$. In the same study, a similar result was reported for early life-stage northern pike with a 35-d LOEC for biomass of 12.9 µg/L Cd observed. For the early life-stage of the white sucker, a 40-d LOEC for biomass of 12.0 µg/L Cd was reported. Vardy *et al.* (2011) reported a 27-d LC_{20} for early life-stage white sturgeon of 8.7 µg/L Cd (CI_{95}: 7.9 - 9.5 µg/L Cd) in exposures with water hardness of 70 mg/L $CaCO_3$.

7.5 Toxicity to Amphibians

Very few studies have been conducted on the toxicity of Cd to amphibians. Nebeker *et al.* (1995) reported a 96-h LC_{50} for the northwestern salamander (*Ambystoma gracile*) of 468 µg/L Cd in exposures with water hardness of 45 mg/L $CaCO_3$. Sub-lethal concentrations of Cd in long-term (i.e., 10- and 24-d) exposures from the same study resulted in a LOEC for growth (weight) of 227 µg/L Cd and a LOEC of 193 µg/L Cd for growth (weight), respectively.

7.6 Factors Affecting the Bioavailability and Toxicity of Cadmium

Cd is most toxic to aquatic organisms in its ionic form (i.e., Cd^{2+}), as it interacts with ion receptors (i.e., ligands) at the environment-tissue interface. Therefore, factors that influence the uptake of the Cd ion by aquatic organisms and factors that affect the bioavailability of the Cd ion in the water column and associated sediments determine the magnitude of toxicity to freshwater aquatic organisms. The ambient water quality conditions that influence the toxicity and bioavailability of Cd include hardness, alkalinity, pH, dissolved organic matter (DOM), and temperature.

7.6.1 Hardness and Alkalinity

Water hardness and related factors (including the concentration of major cations) influence the toxicity of specific metals (i.e., divalent metals) by directly competing with those metals for binding with Ca^{2+} and Na^{+} channels on organism tissues (such as gills). Additionally, other cations including Na^{+} and H^{+} provide additional competition with Cd (and other divalent metals) for binding on tissue receptors.

Alkalinity is a measure of the buffering capacity (i.e., ability to neutralize acids) of the system and is a function of the concentration of carbonate (CO_3^{2-}). Carbonate will bind with Cd (and other metals) to form insoluble metal complexes that settle out onto the sediments, reducing the bioavailability of the metal in the system. Hardness and alkalinity (as well as pH) are often correlated in aquatic systems, as one of the primary constituents of hardness in natural waters is $CaCO_3$. Hardness and alkalinity both reduce the toxicity of some divalent metals (such as Cd) by competing with the metal for Ca^{2+} binding sites and providing a ligand for complexation, thus reducing the bioavailability of Cd in the system. In the majority of the studies compiled, both hardness and alkalinity increased in the exposure chamber, making it difficult to discern the relative contribution of metal competition and metal complexation on the observed toxicity in the experiment.

Källqvist (2009) explored the relationship between water hardness and toxicity of Cd to the green alga (*P. subcapitata*) in low-hardness systems (characteristic of the Fennoscandian region of Europe). The study looked at the effects of Cd exposure on the growth rate of the algae. The observed EC_{50} values increased with increasing hardness, even at the relatively low water hardness in the exposures (3 treatments: 6.2, 16.2, and 46.2 mg/L $CaCO_3$). The 72-h EC_{50} for growth ranged from 29 µg/L Cd (95% confidence interval [CI_{95}]: 26 - 33 µg/L Cd) to 199 µg/L Cd (CI_{95}: 158 - 265 µg/L Cd; Källqvist 2009). The exposures were spiked with Ca^{2+} to obtain the desired water hardness, and thus the reduction in toxicity can be attributed primarily to the increased competition of the Ca^{2+} ions with Cd^{2+} (i.e., reduced uptake of Cd) rather than the reduction in bioavailability caused by complexation with CO_3^{2-}.

Chapman *et al.* (1980) looked at the effects of water hardness on the short-term and long-term toxicity of *Daphnia magna* in water-only exposures. The 48-h LC_{50} values calculated from the results increased by 4.9 times as hardness increased from 51 to 209 mg/L $CaCO_3$. While other water quality conditions remained similar in the treatments, both alkalinity and pH (to a lesser degree) increased, resulting in the potential of both to contribute to the reduced toxicity observed. Similar results were obtained in long-term exposures. The 21-d maximum acceptable toxicant concentration (MATC) for reproduction increased from 0.15 to 0.44 µg/L Cd as hardness increased from 53 to 209 mg/L $CaCO_3$ (Chapman *et al.* 1980).

Several studies have looked at the relationship between short-term and long-term toxicity of Cd at varying water hardness. For example, in a study conducted by Brinkman and Hansen (2007), the 96-h LC_{50} for brown trout (*Salma trutta*) fry increased from 1.23 to 10.1 µg/L Cd with increasing water hardness (from 30.6 to 151 mg/L $CaCO_3$), while other water quality variables, except alkalinity, remained constant. Similar results were observed in long-term tests where early life stage (egg to 41 days post-hatch) exposures with brown trout showed increasing MATCs for swim-up survival (from 3.52 to 13.6 µg/L Cd) with increasing hardness. Similarly, toxicity tests with fry resulted in 30-d MATCs for survival ranging from 1.02 to 6.54 µg/L Cd as hardness increased from 30 to 150 mg/L $CaCO_3$. Further, the IC_{20} (endpoint: biomass) for early life stage fish increased from 2.22 to 13.6 µg/L Cd (CI_{95} not reported) and in fry increased from 0.87 to 6.62 µg/L Cd (CI_{95} not reported) over the 3 hardness treatments (Brinkman and Hansen 2007).
Palawski (1985) designed a study to assess the sensitivity of striped bass (*Morone saxatilis*) to Cd under varying water quality conditions. The 96-h LC_{50} of 4 µg/L Cd (CI_{95}: 3 - 6 µg/L Cd) in 40 mg/L $CaCO_3$ water hardness increased to 75 µg/L Cd (CI_{95}: 59 - 96 µg/L Cd) in exposure chambers with hardness of 455 mg/L $CaCO_3$.
However, Davies *et al.* (1993) showed that the ameliorating effects of hardness were greatly reduced when magnesium alone was used to increase hardness. The 96-h LC_{50} was calculated from toxicity tests in exposures with water hardness of 50, 200, and 400 mg/L $CaCO_3$. In these exposure chambers, alkalinity was held constant at 30 mg/L.

While the observed LC_{50}, which ranged from 3.08 µg/L Cd (CI_{95}: 2.80 - 3.39 µg/L Cd) to 5.92 µg/L Cd (CI_{95}: 4.34 - 9.11 µg/L Cd), increased as water hardness increased, a statistically significant effect was not observed.

7.6.2 pH

The pH of the aquatic system affects both the uptake of Cd and bioavailability of the Cd ion. In acidic waters, concentrations of the free Cd^{2+} ion can account for up to 90% of total Cd due to the dissociation of Cd from metal-ligand complexes (Campbell and Stokes 1985). This is supported by models showing that the Cd^{2+} ion is the predominant form of Cd in acidic conditions (i.e., pH < 7), but forms insoluble complexes with carbonate (CO_3^{2-}) and hydroxide (OH^-) as pH increases above 7 (Bervoets and Blust 2000; Gueguen *et al.* 2003). At low pH, the increased concentration of hydrogen ions (H^+) increases competition with Cd for binding sites on cell surfaces and may also affect the membrane potential of the cell, both of which can reduce the toxicity of Cd (Campbell and Stokes 1985). However, at pH above 7, the Cd ion is less bioavailable and therefore exposure is limited.

Several studies support the hypothesis that Cd is most toxic between pH 6 and 7. Kwan and Smith (1991) observed that the uptake of Cd by the aquatic plant *L. minor* was greatest at pH 6, suggesting that Cd is most toxic at this pH. In the same experiment, uptake was reduced in exposures with pH above 8 and below 4. Hahne and Kroontje (1973) also reported that in exposures with pH greater than 8, Cd forms insoluble hydroxide complexes, which greatly reduce the bioavailability of the metal. Further, in an experiment with the alga, *Stichococcus bacillaris*, the toxicity of Cd was studied under varying pH. The largest decrease in growth (approximately 60% of control) and chlorophyll *a* content (25-30% of control) was observed between pH 6 and 7. At pH 3, the toxic effects of Cd were greatly reduced (Skowronski *et al.* 1991). The results of these studies corroborate the observations in other studies with algae that increased adsorption and uptake of Cd occurs as pH increases from 4 to 7.5 (Skowronski 1986a; 1986b).

A similar trend has also been observed in experiments with fish. Cuismano *et al.* (1986) reported 96-h LC_{50} values for steelhead trout in soft water ranging from 28 µg/L Cd (CI_{95}: 22 - 37 µg/L Cd) at pH 4.7 to < 0.5 µg/L Cd at pH 7.0. Similarly, in long-term exposures (i.e., 7 days) the LC_{50} for steelhead trout decreased from 6.3 µg/L Cd (CI_{95}: 4.6 - 8.9 µg/L Cd) to < 0.5 µg/L Cd as pH increased from 4.7 to 7.0 (Cuismano *et al.* 1986). Mortality after 24 hours was also greater for juvenile rainbow trout exposed to Cd at pH 7.8 (28.6%) compared to pH 4.8 (0%) in hard water, as well as in soft water (34.8% and 19.0%, respectively; Reid and McDonald 1988). In a study with rainbow trout and bull trout, Hansen *et al.* (2002a) reported increased toxicity at pH 7.5 relative to pH 6.5. In this study, the 120-h LC_{50} for rainbow trout increased from 0.53 µg/L Cd (CI_{95}: 0.48 - 0.59 µg/L Cd) at pH 7.5 to 0.84 µg/L Cd (CI_{95}: 0.76 - 0.93 µg/L Cd) at pH 6.5. For bull trout, the 120-h LC_{50} increased from 0.83 µg/L Cd (CI_{95}: 0.76 - 0.91 µg/L Cd) at pH 7.5 to 2.41 µg/L Cd (CI_{95}: 2.15 - 2.70 µg/L Cd) at pH 6.5.

However, other studies have reported contrasting results. Schubauer-Berigan *et al.* (1993) ran short-term toxicity tests with the cladoceran, *Ceriodaphnia dubia*; the amphipod, *H. azteca*; the fathead minnow, *P. promelas*; and, the oligochaete, *Lumbriculus variegatus* at three pH levels: 6.3, 7.3, and 8.3. Cd was most toxic to *C. dubia* and *H. azteca* at pH 8.3 and least toxic at pH 6.3, while toxicity to *P. promelas* and *L. variegatus* remained constant with increasing pH. Similarly, Musko *et al.* (1990) found that Cd was more toxic to the amphipod *Gammarus fossarum* at pH 8.5 than 6.0. In addition, Cd was more acutely toxic to *H. azteca* at pH 5 than 6, while damselflies (*Enallagma* sp.) were more sensitive to Cd at pH 3.5 than 4.5 (Mackie 1989). In contrast, the clams *Pisidium casertanum* and *P. compressum,* and the snail *Amnicola limosa* exhibited higher mortality when exposed to Cd at pH 4.5 than 3.5 (Mackie 1989).
In insects, Cd uptake is likely a mixture of internal intake and adsorption to the exoskeleton. Uptake was greater in *Ephemeroptera* at pH 7 than pH 5 (Gerhardt 1990). However, survival was greater at pH 7 than pH 5, suggesting most Cd was taken up in the exoskeleton and did not affect the organism (Gerhardt 1990). In general, Cd uptake over 6 hours in the midge, *C. riparius,* increased as pH increased from 5.5 to 9.0, before

decreasing at pH 10, though the magnitude of uptake depended on the pH in the acclimation chambers (Bervoets and Blust 2000). The authors suggest that physiological changes occur in the larvae during acclimation that influence the uptake of Cd during exposure (i.e., that pH influences Cd uptake as well as metal speciation).

7.6.3 Dissolved Organic Matter

Dissolved organic matter (DOM), including humic acid and fulvic acid, may reduce the bioavailability of Cd by binding to free Cd^{2+} ions, but competition for the DOM binding sites from other cations (e.g., Ca^{2+} and Mg^{2+}) may reduce Cd binding efficacy. While the binding of calcium with DOM may also inhibit the ameliorating effects of water hardness, the calcium ions also compete for the binding sites at the environment-tissue interface. Penttinen *et al.* (1998) showed that Cd did not bind to DOM as readily in conditions with elevated water hardness. Thus, at low water hardness, toxicity to *D. magna* was reduced in the presence of DOM. At higher water hardness, less Cd was bound to the DOM, but toxicity to *D. magna* was comparatively low due to the ameliorating effects of the increased Ca^{2+} ions. Similar results were observed in studies with the zebrafish (*Danio rerio*), where survival at low hardness was relatively higher compared to treatments with elevated water hardness (Meinelt *et al.* 2001).

In contrast, Winner (1984) found that the addition of 1.5 mg/L of humic acid increased the toxicity of Cd to daphnids; the observed 72-h LC_{50} decreased from 87.8 to 71.1 µg/L Cd. Survival in long-term exposures (i.e., 20 to 42 days) also decreased in treatments with humic acid (Winner 1984; Winner and Gauss 1986). However, the addition of humic acid did not affect Cd bioaccumulation over 7 days (Winner and Gauss 1986). Winner and Gauss (1986) suggested that this discrepancy between toxicity and bioaccumulation could be due to adsorption of Cd at the gill surface, rather than accumulating in the body tissue. Oikari *et al.* (1992) also found that humic acid increased the short-term toxicity of Cd to *D. magna* (48-h LC_{50} of 12 µg/L Cd compared to 99 µg/L Cd with no humic acid; CI_{95} not reported). In another study, the addition of humic acid (50 mg/L) had mixed results on toxicity tests with *Daphnia pulex*.

Stackhouse and Benson (1988) reported that the addition of humic acid significantly increased ($p < 0.05$) the LC_{50} across all measured exposure periods. For example, the 24-h LC_{50} increased from 179.9 µg/L Cd (CI_{95}: 159.9 - 203.8 µg/L Cd) to 739.3 µg/L Cd (CI_{95}: 680.1 - 816.8 µg/L Cd) with the addition of humic acid. Similarly, the 96-h LC_{50} increased from 32.4 µg/L Cd (CI_{95}: 29.6-35.7 µg/L Cd) to 112.6 µg/L Cd (CI_{95}: 101.2-127.9 µg/L Cd) with the addition of humic acid (Stackhouse and Benson 1988). However, the addition of smaller volumes of humic acid (i.e., 0.5 and 5 mg/L) increased, decreased, or did not significantly change the LC_{50}, depending on the exposure period measured (Stackhouse and Benson 1988). In a separate test, the concentration of free Cd ions decreased as the volume of humic acid increased, suggesting that the decrease in observed toxicity reported in the 50 mg/L humic acid treatment was due to the formation of metal-ligand complexes (Stackhouse and Benson 1988). Supporting this, the bioaccumulation of Cd in *D. magna* after 96 hours was significantly decreased ($p < 0.05$) with 50 mg/L humic acid compared to the control (24.6 ± 0.9 µg/g Cd DW compared to 65.0 ± 5.0 µg/g Cd DW; Stackhouse and Benson 1989).

The presence of DOM has also been shown to reduce the toxicity of Cd to algae. Koukal *et al.* (2003) found that the addition of 1 mg/L humic acid significantly decreased ($p<0.05$) photosynthetic inhibition of *P. subcapitata* exposed to 200 µg/L Cd (33.2 to 45% inhibition in treatments with humic acid versus 52% inhibition without humic acid). The observed decrease in toxicity was even more pronounced when 5 mg/L of humic acid was added (9.7 to 26.7% inhibition; Koukal *et al.* 2003). However, the addition of fulvic acids (from the Suwannee River in the United States) did not reduce Cd toxicity to *P. subcapitata* (Koukal *et al.* 2003). The authors suggested that Cd and fulvic-acid complexes dissociate more readily than Cd and humic-acid complexes. Secondly, they proposed that fulvic acids do not adsorb to algae as well as humic acids, suggesting that fulvic acids do not shield the cell membranes from Cd as well (Koukal *et al.* 2003).

In experiments with fish, DOM was also found to reduce the toxicity of Cd. In a study with rainbow trout exposed to copper and Cd in soft water for 15 days, survival increased

from 70% to 87% with the addition of 5 mg/L DOM; survival further increased to 100% with the addition of 10 and 20 mg/L DOM (Hollis *et al.* 1996). However, Cd binding at the gill sites did not differ significantly between treatments. Similarly, Hollis *et al.* (1996) observed that in exposures with 5 mg/L DOM and various metals, a significantly increased concentration of Cd at the gill binding sites of trout was observed relative to the control treatments, but significantly less Cd on the gills than in exposures with metals alone. This suggests that other metals may have increased DOM binding efficacy relative to Cd. However, when juvenile rainbow trout were exposed to a mixture of six metals for 74 hours, the addition of up to 10 mg/L natural organic matter (NOM) did not decrease Cd adsorption on the gills, though it did increase survival of the fish (Richards *et al.* 2001). Using three different sources of NOM, Richards *et al.* (2001) found that adding allochthonous NOM resulted in the highest fish survival.

In a 144-h multi-factor experiment designed to evaluate the effects of calcium and DOM on Cd toxicity, zebrafish embryos and larvae were exposed to multiple levels of Cd, calcium, and DOM. Survival was highest in the high calcium, no DOM treatment (90 - 100% survival) over the entire range of Cd treatments (0 - 9.3 μg/L Cd; Meinelt *et al.* 2001). The high calcium with DOM treatment produced similar results, however survival was reduced (40% after 144 h) in the 9.3 μg/L Cd exposure. Survival was lowest in the low calcium without DOM treatment (reaching 0% in the 4.2 and 9.3 μg/L Cd treatments (Meinelt *et al.* 2001). In this study, Meinelt *et al.* (2001) showed that both DOM and calcium contribute to reducing the toxicity of Cd, but interactions between DOM and calcium need to be considered.

7.6.4 Temperature

As many aquatic organisms are ectotherms, water temperature may affect the toxicity of Cd. Changes in water temperature can alter feeding activity, swimming speed, metabolic rate, and physiological state (Donker *et al.* 1998). Elevated water temperature may increase metabolic demand and respiration, increasing the potential uptake of contaminants through the gills (Cairns *et al.* 1975). Several researchers have investigated

the hypothesis that toxicity of Cd increases with increasing temperature. For example, Edgren and Notter (1980) found that perch (*Perca fluviatilis*) fingerlings kept at 15°C accumulated radioactive Cd (Cd-115) in greater quantities (BCF: 17) than fish kept at 5°C (BCF: 7.5) after 39 days. After exposure to 0.5 µg/L Cd for an additional 25 days, 12 fish were euthanized and analysed. Cd accumulated primarily in the kidney (9.0 mg/kg Cd DW), and to a lesser degree in the liver (5.0 mg/kg Cd DW) and gills (3.6 mg/kg Cd DW; Edgren and Notter 1980). In another study, Yang and Chen (1996) observed that the kidney, liver, and gills of the Japanese eel (*Anguilla japonica)* showed greater accumulation of Cd when exposed to higher temperatures. For example, the concentration of Cd in the kidney measured approximately 5 mg/kg Cd WW in the 15°C treatment compared to approximately 33 mg/kg wet weight (WW) in the 30°C treatment over the same exposure period. Similarly, Cd uptake in the freshwater plant *L. minor* increased as temperature increased from 4°C (approximately 1 µmol/g DW after 48-h) to 34°C (approximately 8 µmol/g DW after 48-h; Kwan and Smith 1991). Cd uptake was also about 50% greater in the algae *P. subcapitata* at 20°C compared to 2°C (Errécalde and Campbell 2000). Another study reported an increase in bioaccumulation of Cd in the Asiatic clam (*Corbicula fluminea*) when exposed to Cd at 21°C relative to the treatment at 9°C (Graney *et al.* 1984). In addition, Lewis and Horning (1991) reported that in treatments with elevated water temperature (i.e., 26°C), the 24-h and 48-h LC_{50} were significantly lower compared with treatments at 20°C.

Heugens *et al.* (2003) performed multiple experiments to assess the effect of temperature on Cd toxicity to *D. magna*. Short-term (i.e., 48-h) toxicity tests were performed using multiple treatments between 10 and 35°C. The results showed no control mortality at temperatures of 26°C and below, almost 80% control mortality at 32°C, and 100% control mortality at 35°C. The 48-h LC_{50} decreased from approximately 1,200 µg/L Cd at 10°C to approximately 50 µg/L Cd at 32°C. In a separate test, the uptake rate of Cd in *D. magna* in 45-h exposures was significantly greater at 20°C (2.56 mg/kg DW/hour) than at 10°C (1.06 mg/kg DW/hour). The authors also estimated a lethal toxicity threshold concentration for *D. magna* at 26°C of 51.6 mg/kg Cd DW; CI_{95}: 42-61 mg/kg

Cd DW) compared to 270 mg/kg Cd DW (CI_{95}: 220-330 mg/kg Cd DW) at 10°C. Therefore, they concluded that changes in sensitivity to Cd, along with increased accumulation kinetics, occur at higher temperatures and play a part in increasing toxicity of Cd to daphnids.

7.6.5 Acclimation

Acclimation refers to the exposure of organisms to sub-lethal concentrations of a toxicant over time. In some cases, the organism becomes more resistant (i.e., tolerant) to higher, normally lethal, concentrations of the toxicant. The resistance is often due to physiological changes in the organism (Klerks and Weis 1987). While no studies were found that evaluated the effects of acclimation on chronic exposure to Cd, studies on the effects of acclimation on acute exposure were compiled. Benson and Birge (1985) designed an experiment to evaluate the potential acclimation of fathead minnows (*P. promelas*) to Cd. Fish collected from a flyash pond (mean concentration: 0.46 µg/L Cd) were tested along with hatchery-reared fish (mean concentration: 0.28 µg/L Cd). The observed 24-h LC_{50} was significantly greater ($p < 0.05$) in fish collected from the flyash pond (6.06 mg/L Cd; CI_{95}: 5.12 - 7.29 mg/L Cd) compared to hatchery-reared fish (4.03 mg/L Cd; CI_{95}: 2.71 - 4.99 mg/L Cd). However, the 96-h LC_{50} was not significantly different (3.89 mg/L Cd; CI_{95}: 3.23-4.47 versus 3.06 mg/L Cd; CI_{95}: 2.00-3.81). Nonetheless, the median lethal time (LT_{50}) was significantly less in the treatments with the hatchery-reared fish compared with the fish collected from the flyash pond. Specifically, in the 6 mg/L exposure treatment, the LT_{50} for the acclimated fish was 50 h (CI_{95}: 29.4 - 85.0 h) compared to the LT_{50} in the hatchery-reared fish (6.8 h; CI_{95}: 4.3 - 10.8 h). A similar trend was also observed at higher exposure concentrations. Interestingly, when fish from the flyash pond were held in clean water prior to testing the 96-h LC_{50} decreased from 9.5 mg/L Cd (deacclimation period: 0 d) to 7.5 mg/L Cd (deacclimation period: 14 d). The holding period did not affect the hatchery-reared fish; the observed LC_{50} remained around 3 mg/L Cd. Benson and Birge (1985) also exposed hatchery-reared fish to 10 µg/L Cd for 35 d to determine if these fish would become acclimated to the increased Cd concentration. Indeed, the 96-h LC_{50} was significantly

greater (2.88 mg/L Cd; CI_{95}: 2.07 - 3.72 mg/L Cd) than in the control treatment (1.71 mg/L Cd; CI_{95}: 1.15 - 2.19 mg/L Cd).

Exposure of white suckers (*Catostomus commersoni*) to 0.41 or 0.73 mg/L Cd for one week prior to toxicity testing resulted in a greater LC_{50} relative to fish in the control treatment (i.e., no acclimation period; Duncan and Klaverkamp 1983). For example, the 96-h LC_{50} in the control treatment was 1.0 mg/L Cd. In the 0.41 mg/L acclimation treatment, the reported 96-h LC_{50} was 1.9 mg/L Cd. For fish acclimated to 0.71 mg/L Cd, the 96-h LC_{50} was 2.2 mg/L Cd. In the same study, the fish acclimated to 0.73 mg/L Cd also exhibited a longer median survival time (MST; 18 h) relative to fish that were not acclimated to Cd (8.5 h).

Juvenile rainbow trout exposed to 3 or 10 µg/L Cd for 30 days exhibited increased resistance to Cd toxicity compared to fish that were not acclimated to Cd (Hollis *et al.* 1993). The 96-h LC_{50} in the 3 µg/L Cd acclimation treatment was 286 µg/L Cd, whereas the 96-h LC_{50} in the 10 µg/L Cd acclimation treatment was 242 µg/L Cd. The 96-h LC_{50} for the fish that were not acclimated was markedly less (22 ± 12 µg/L Cd). While 30% mortality was observed in the acclimation chambers during the first three days of the exposure to 10 µg/L Cd, less than 1% mortality was observed in the 3 µg/L Cd chambers. The authors hypothesized that physiological changes, increasing Cd storage and detoxification efficiency as well as greater resistance of gill processes are possible mechanisms for acclimation to Cd. Other authors have reported similar results with rainbow trout. Stubblefield *et al.* (1999) exposed juvenile and adult rainbow trout to sub-lethal levels of Cd for 21 days. Acclimated juvenile trout fish increased their Cd tolerance by a factor of 1.4 to 2.0, while acclimated adult rainbow trout exhibited a 15 to 20 times increase in Cd tolerance.

Fewer studies on acclimation of aquatic invertebrates to Cd have been found in the primary literature. Stuhlbacher and Maltby (1992) found that acclimating the amphipod *Gammarus pulex* to 5, 10, or 20 µg/L Cd for 24 hours resulted in a significantly greater (*p*

< 0.05) 48-h EC_{50} for mortality or immobility than unexposed amphipods. The observed 48-h EC_{50} increased from 1.4 to 2.2 with increasing acclimation concentration. However, exposing the amphipods to 50.4 µg/L Cd prior to toxicity testing resulted in a significantly lower ($p < 0.05$) 48-h EC_{50} (1.05 mg/L Cd) relative to control. In the same study, increasing the acclimation period to seven days in the 10 µg/L Cd treatment did not result in an increased tolerance to Cd exposure.

7.6.6 Biotic Ligand Model for Cadmium

The biotic-ligand model (BLM) is an approach that integrates multiple factors that may influence the toxicity of Cd. This approach accounts for the competition between certain metals (e.g., Cd and zinc), cations (including the constituents of hardness) and other naturally occurring ligands (including DOM) to develop a tool for incorporating site-specific water quality conditions into the assessment of metals toxicity (Paquin *et al.* 2002; Niyogi and Wood 2004).

While the effects of varying water hardness on metals toxicity is often considered to be a primary factor for ameliorating the toxicity of metals such as Cd, other factors such as alkalinity, pH, and specific ions have been found to be critical to mediating toxicity of Cd in laboratory studies (Paquin *et al.* 2002; Niyogi and Wood 2004). Further, abiotic ligands (e.g., DOM) may bind with free Cd, reducing its bioavailability. However, there is currently no short-term or long-term BLM for Cd that has been adopted by other jurisdictions for the development of WQGs. In addition, the existing toxicological studies typically do not provide the requisite information (i.e., model inputs) for the BLM that could be applied to evaluate toxicological data from multiple species for use in adapting WQGs to site-specific conditions.

8.0 Water Quality Guidelines from Other Jurisdictions

As noted in Section 4.0, the environmental concentrations of water quality variables (e.g., Cd) are reflective of many local characteristics (e.g., soil type, pH, hydrological regimes,

and water hardness). This highlights the importance of developing WQGs taking into consideration local conditions in conjunction with WQGs for the protection of aquatic life developed in other jurisdictions. A comparison of the WQGs for Cd from other jurisdictions is provided in Table 5.

8.1 Canadian Council of Ministers of the Environment Water Quality Guidelines

The CCME is the national body that promulgates WQGs for the protection of aquatic life. The CCME WQG for short-term exposure to total Cd was developed using the site-specific water hardness (as mg/L $CaCO_3$) and the following equation:

$$\text{CCME WQG } (\mu g/L \text{ Cd}) = 10^{1.016[\log(\text{hardness})]-1.71}$$

Table 5. Summary of water quality guidelines from other jurisdictions.

Guideline Type/Jurisdiction[A]	Water Quality Guideline for Cadmium (µg/L)		
	50 mg/L $CaCO_3$	180 mg/L $CaCO_3$	320 mg/L $CaCO_3$
Short-Term Water Quality Guideline			
CCME [B]	1.0	3.8	6.8
Ontario [B]	0.1	0.5	0.5
USEPA [C]	1.0	3.8	6.8
Australia/New Zealand [B]	0.32	0.99	1.6
European Union [C]	0.60	0.90	1.5
Long-Term Water Quality Guideline			
CCME [B]	0.09	0.26	0.37
USEPA [C]	0.15	0.39	0.60
European Union [C]	0.09	0.15	0.25

[A] See Section 8.0 for details on the application of the WQGs.
[B] This guideline applies to the total cadmium concentration.
[C] This guideline applies to the dissolved cadmium concentration.

At water hardness of 50 mg/L $CaCO_3$, the WQG is 1.0 µg/L (CCME 2014a). The CCME short-term hardness equation is valid for hardness between 5.3 and 360 $CaCO_3$ mg/L. At

hardness < 5.3 mg/L $CaCO_3$, the short-term WQG is 0.11 µg/L, while at hardness > 360 mg/L $CaCO_3$ the WQG is 7.7 µg/L.

The CCME WQG for long-term exposure to total Cd was developed using the site-specific water hardness (as mg/L $CaCO_3$) and the following equation:

$$\text{CCME WQG (µg/L Cd)} = 10^{0.83[\log(\text{hardness})]-2.46}$$

At water hardness of 50 mg/L $CaCO_3$, the WQG is 0.09 µg/L (CCME 2014a). The CCME long-term hardness equation is valid for hardness between 17 and 280 $CaCO_3$ mg/L. At hardness < 17 mg/L $CaCO_3$, the long-term WQG is 0.04 µg/L, while at hardness > 280 mg/L $CaCO_3$ the WQG is 0.37 µg/L.

8.2 Provincial Water Quality Guidelines

Provinces of Canada typically develop province-specific WQGs or adopt the WQG from another jurisdiction (e.g., CCME). The Ontario Ministry of Environment has set policies to manage Ontario's water resources, including providing Provincial Water Quality Objectives (PWQOs) for surface water to protect aquatic life (OMOEE 1994). The PWQO for total Cd is 0.2 µg/L, however an interim PWQO is under development. The interim PWQO for total Cd is 0.1 µg/L for water with hardness between 0 and 100 mg/L $CaCO_3$ and 0.5 µg/L for water with hardness > 100 mg/L $CaCO_3$ (OMOEE 1994). Saskatchewan has adopted the CCME WQGs, including the WQG for total Cd (Saskatchewan Environment 2006) while the province of Quebec has adopted the United States Environmental Protection Agency (USEPA) Cd water quality criteria (WQC) for protection of aquatic life (MDDEFP 2002). Alberta has not developed a WQG for Cd; however, it has adopted the use of both the CCME and USEPA WQGs for protection of aquatic life (Alberta Environment 1999).

8.3 United States Environmental Protection Agency Water Quality Criteria

The USEPA has recommended acute (i.e., short-term) and chronic (i.e., long-term) national WQC for the protection of aquatic life for use with the dissolved metal concentration (USEPA 2013). The WQC for Cd are based on water hardness and calculated using the following equations:

$$CMC\ (\mu g/L) = [1.136672 - (\ln(hardness)*0.041838)] * e^{1.0166[\ln(hardness)] - 3.924}$$

$$CCC\ (\mu g/L) = [1.101672 - (\ln(hardness)*0.041838)] * e^{0.7409[\ln(hardness)] - 4.719}$$

Where: CMC = Criteria Maximum Concentration
CCC = Criteria Continuous Concentration

At a hardness of 50 mg/L $CaCO_3$, the USEPA acute WQC (i.e., CMC) for dissolved Cd would be 1.0 µg/L. The chronic WQC (i.e., CCC) for dissolved Cd would be 0.15 µg/L. The USEPA is in the process of updating their 2001 Cd criteria (Gallagher personal communication, 2013).

8.4 Other Water Quality Guidelines

Australia and New Zealand have joint WQGs that are described as trigger values; that is, they trigger a response if exceeded (ANZECC 2000a; 2000b). To protect 95% of aquatic life, ANZECC (2000a; 2000b) has developed a high reliability trigger value for Cd of 0.2 µg/L, which applies to waters with hardness of 30 mg/L $CaCO_3$. The trigger value to protect 99% of aquatic life is 0.06 µg/L Cd and is recommended for use for slightly-moderately disturbed ecosystems for protection of cladoceran species (ANZECC 2000b). The trigger value for Cd can be adjusted to the site-specific water hardness using:

$$\text{Hardness-Modified Trigger Value} * (hardness/30)^{0.89}$$

At a water hardness of 50 mg/L $CaCO_3$ the 95% protection level trigger value is 0.32 µg/L. ANZECC (2000a) generally recommends using the 95% protection level for slightly-moderately disturbed ecosystems (considered typical systems), though they provide guidance for 90% and 80% protection levels as well for use at highly disturbed sites (ANZECC 2000a). To protect 90% of aquatic life, the trigger value for Cd is 0.4 µg/L, while the 80% protection level is 0.8 µg/L (at a hardness of 30 mg/L $CaCO_3$; ANZECC 2000a).

The European Union's Water Framework Directive provides a list of priority substances to monitor and control in water, and has set out Environmental Quality Standards (EQSs) for these priority substances defined as concentrations that should not be exceeded (EU 2008). For Cd, the EQSs are separated by five water hardness classes. The Cd annual average EQSs (calculated as the average Cd concentration over a year) for inland surface waters are as follows:

- 0.08 µg/L (hardness < 40 mg/L $CaCO_3$);
- 0.08 µg/L (hardness 40 to < 50 mg/L $CaCO_3$);
- 0.09 µg/L (hardness 50 to < 100 mg/L $CaCO_3$);
- 0.15 µg/L (hardness 100 to < 200 mg/L $CaCO_3$); and**,**
- 0.25 µg/L (hardness ≥ 200 mg/L $CaCO_3$).

The Cd maximum allowable concentration EQSs for inland surface waters are:

- 0.45 µg/L (hardness < 40 mg/L $CaCO_3$);
- 0.45 µg/L (hardness 40 to < 50 mg/L $CaCO_3$);
- 0.6 µg/L (hardness 50 to < 100 mg/L $CaCO_3$);
- 0.9 µg/L (hardness 100 to < 200 mg/L $CaCO_3$); and,

- 1.5 µg/L (hardness ≥ 200 mg/L $CaCO_3$).

9.0 Derivation of Water Quality Guidelines for Cadmium in British Columbia

The derivation of WQGs for dissolved Cd for BC to protect freshwater aquatic life followed the protocol of BC MOE (2012). To facilitate the development of WQGs, scientific literature on the toxicity of Cd to freshwater aquatic life was compiled and evaluated. Studies were evaluated to identify high-quality data (i.e., data which meet the criteria outlined in BC MOE [2012]), which were used in the formation of the short-term maximum and long-term average guidelines. The toxicological data were adjusted to account for site-specific toxicity-modifying factors (i.e., water hardness) by normalizing to 50 mg/L $CaCO_3$, based on the relationship between water hardness and toxicity from select studies. Consistent with guidance in BC MOE (2012), the lowest value from a primary study was selected to form the preliminary guideline. The minimum uncertainty factor of 2 was applied to the guideline to account for incomplete knowledge or various uncertainties in the toxicity data.

9.1 Acquisition of Toxicological Data for Cadmium

The current scientific literature was reviewed, and studies that evaluated the toxicity of Cd to freshwater aquatic organisms under laboratory conditions were compiled from literature reviews conducted between October 2012 and October 2014. Information on the toxicity of Cd to aquatic organisms was compiled from multiple sources including recent WQG derivation documents (USEPA 2001 and CCME 2014a), scientific databases, and personal communications with researchers investigating the toxicity of metals to aquatic life. The compilation of relevant studies focussed on species relevant to BC and ensured that an appropriate number of studies were obtained to meet the minimum data requirements for WQG derivation (BC MOE 2012). These studies were subsequently classified for their appropriateness. Summaries of the data and

bibliographical information for each of the studies used in the evaluation are reported in Appendix 2.

9.2 Evaluation and Classification of Toxicological Data

The evaluation of toxicological data followed the BC protocol for guidelines development (BC MOE 2012). Each of the compiled studies was evaluated based on test procedures and experimental design, to ensure that only those studies that were scientifically sound and of high-quality were used in the WQG derivation. Toxicological studies were classified as primary, secondary, or unacceptable. While the primary studies were used to derive the short-term maximum and long-term average WQG, toxicological data from both primary and secondary studies were considered in the development of the guideline.

Information on the test species, test conditions, experimental design, chemical and physical properties of the test water, statistical analyses, and negative control performance were reviewed. The key points for determining the classification (i.e., primary, secondary, or unacceptable) are summarized below.

To be considered as primary data, studies must have included the following (BC MOE 2012):

- Cd concentrations measured and reported at a minimum at the beginning and end of the exposure period;
- Flow-through or renewal test, unless a static test included evidence that Cd concentrations did not change and environmental conditions were acceptable throughout the test;
- Two or more replicate test chambers per concentration (ASTM 2012b; 2012c);

- Ecologically relevant endpoints (i.e., related to growth, reproduction, hatching success, or survival);
- Acceptable negative control performance reported (less than 10% mortality for short-term [96-h] tests and less than the ASTM International test acceptability criteria for the appropriate species and test for long-term exposures);
- Measurements of temperature, pH, dissolved oxygen, and water hardness reported and results are acceptable (e.g., dissolved oxygen met the requirements in ASTM E729 for short-term tests [ASTM 2012b] and ASTM E1241 for early life-stage tests [ASTM 2012c]); and,
- Appropriate statistical analyses were applied.

Guidance for secondary data includes (BC MOE 2012):

- Toxicity tests may incorporate the exposure of test organisms to additional stressors or effects (e.g., low temperature, lack of food, exposure of F_1 generation);
- Calculated (vs. measured) concentrations of Cd in exposure chambers are acceptable;
- Static (non-renewal) tests are acceptable;
- Pseudoreplication (e.g., only one test chamber with multiple organisms inside per concentration) is acceptable;
- Broader array of endpoints is acceptable, though endpoints need to be ecologically relevant (e.g., pathological, behavioural, and physiological endpoints, in addition to the primary data endpoints); and,
- Must meet the same requirements for negative control performance, water quality measurements, and statistical analyses as primary data sets.

If any of the conditions for primary or secondary data were not met, the data were given a ranking of unacceptable (BC MOE 2012). A summary of all short-term and long-term primary and secondary data is provided in Appendix 2 (Tables A2.1 and A2.2). A list of the studies that were ranked as unacceptable is provided in Table A2.3.

9.3 Normalization of the Toxicological Data to a Standard Hardness

Studies classified as primary and secondary were evaluated for their potential use in developing relationships between Cd toxicity and water hardness. Short-term and long-term exposure data were extracted from these studies and compiled.

9.3.1 Data Evaluation

To facilitate the normalization of raw toxicological data to a standard hardness prior to guideline derivation, short-term and long-term toxicological data were evaluated independently. To determine the relationship between toxicity and water hardness in short-term exposures, the LC_{50}s from studies that tested a range of water hardness were identified. To develop the relationship for toxicity and water hardness in long-term exposures, MATCs or other similar low-effect values for survival and growth were identified. Data were grouped by species and life-history stage and then evaluated to ensure that the experimental conditions (i.e., experimental design, life-stage, exposure duration, and water quality parameters such as temperature and pH) were similar between studies within the data set for a particular species. Studies with experimental conditions that differed (i.e., introduced confounding factors) were excluded from the analysis. Criteria for using data in the evaluation included:

- Primary or secondary classification;
- No additional stressors were placed on the organisms;
- The effect value must have been defined (i.e., undefined LC_x, EC_x, or long-term values that were estimated to be higher than the highest tested concentration were not included); and,

- A minimum of 3 data points for each species over a range of water hardness must have been tested.

While studies that were designed to explicitly evaluate the relationship between toxicity and water hardness were preferred, no studies on the relationship between short-term toxicity and water hardness for aquatic invertebrates met the data evaluation criteria. Chapman *et al.* (1980) designed an experiment to develop and evaluate the relationship between toxicity and water hardness using the cladoceran, *D. magna*. While this study failed to meet the evaluation criteria outlined in Section 9.2 (control mortality was not reported), the study was specifically designed to investigate the relative toxicity of Cd to *D. magna* (i.e., estimate of the slope) at varying water hardness under experimental conditions, and was therefore included in the analysis.

9.3.2 Development of the Relationship between Short-Term Toxicity and Water Hardness

Eight studies were used to develop a relationship between short-term toxicity and water hardness (Appendix 2; Table A2.4). A total of 6 species, comprising 39 data points, were used in the evaluation (striped bass, *M. saxatilis*, n = 3; brown trout, *Salmo trutta*, n = 3; bull trout, *S. confluentus*, n = 6; cutthroat trout, *O. clarkii lewisi*, n = 3; rainbow trout, *O. mykiss*, n = 19; water flea, *D. magna*, n = 5; Figure 2).

Using the methods described in USEPA (1985b), USEPA (2001), and Mebane (2010), the raw toxicity and hardness data were transformed using the natural log prior to application of the linear regression model to each species. The results of the regression analysis are presented in Table 6. The derived slope for the individual species ranged from 0.912 (rainbow trout) to 1.48 (bull trout), with coefficients of determination (i.e., R^2) ranging from 0.270 (rainbow trout) to 0.998 (brown trout). The higher variability in the rainbow trout model may be explained by the inclusion of multiple life-stages in the data set. These data (i.e., the rainbow trout effects data) were pooled because limited data for rainbow trout were available for each life-stage at multiple water hardness

concentrations. The ANCOVA procedure (Zar 1999) was employed to develop a common (i.e., pooled) slope that could be used to normalize the toxicological data that had been compiled from the primary and secondary studies evaluated. The first step of the ANCOVA procedure was to use an *F*-test to test that the estimated slopes from the individual species were not statistically different. The results of this test showed that the individual slopes were not statistically different ($F = 0.190$, $p = 0.902$) and therefore, the common slope was used. Using the pooled data to establish a relationship between short-term toxicity (represented by an LC_{50}) and water hardness, the common slope was

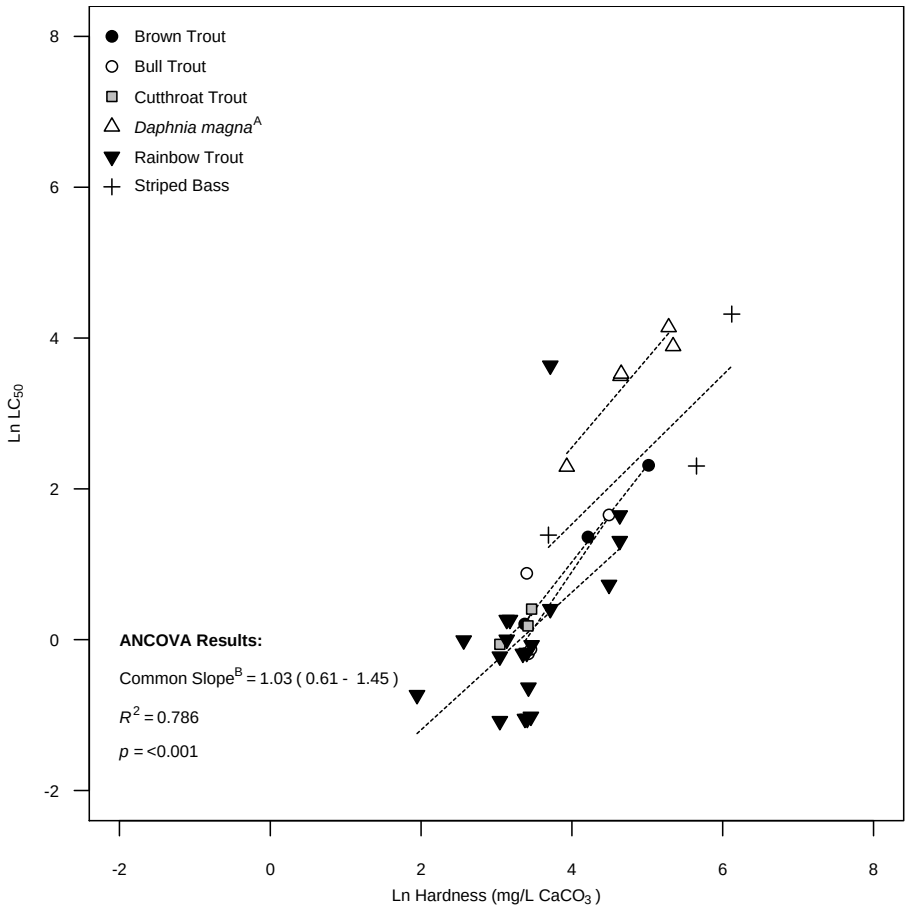
Table 6. Estimate of the slope for use in developing a hardness-modifying conversion factor for short-term toxicity of cadmium based on water-only toxicity tests with various aquatic species.

Receptor Group / Species	Life Stage	Test Duration	Effect Level and Endpoint	n	Slope Estimation			R^2	*p*	Reference
					Slope	95% LCL	95% UCL			
Fish										
Morone saxatilis (Striped bass)	63-d old	96-h	LC_{50}; Mortality	3	0.99	-6.78	8.76	0.723	0.353	Palawski (1985)
Fish - Salmonids										
Salmo trutta (Brown trout)	Fry	96-h	LC_{50}; Mortality	3	1.28	0.582	1.98	0.998	0.0273*	Brinkman & Hansen (2007)
Salvelinus confluentus (Bull trout)	Fry	120-h	LC_{50}; Mortality	6	1.48	0.123	2.84	0.698	0.0385*	Hansen *et al.* (2002a)
Oncorhynchus clarkia lewisi (Cutthroat trout)	YOY	96-h	LC_{50}; Mortality	3	0.94	-4.00	5.88	0.853	0.250	Mebane *et al.* (2012)
Oncorhynchus mykiss (Rainbow trout)	Fry	120-h	LC_{50}; Mortality	19	0.912	0.144	1.68	0.270	0.0227*	Hansen *et al*. (2002a)
	Fry	96-h	LC_{50}; Mortality							Mebane *et al*. (2012)
	Alevin, Juv	96-h	LC_{50}; Mortality							Buhl & Hamilton (1991)
	Swim-up, Juv	96-h	LC_{50}; Mortality							Besser *et al.* (2007)
	Swim-up, Parr	96-h	LC_{50}; Mortality							Chapman (1978)
Invertebrates										
Daphnia magna (Water flea)	< 24-h old	48-h	LC_{50}; Mortality	5	1.18	0.519	1.84	0.915	0.0108*	Chapman *et al*. (1980)
Common Slope (All species)[A]				**39**	**1.03**	**0.61**	**1.45**	**0.786**	**< 0.001*** **	
Common Slope (All significant relationships)[A]				**33**	**1.04**	**0.54**	**1.54**	**0.758**	**< 0.001*** **	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; LCL = lower confidence limit; UCL = upper confidence limit; d = day; h = hour; juv = juvenile; LC = lethal concentration; n = number; YOY = young-of-year.

[A] Common (i.e., pooled) slope estimated using the ANCOVA method (Zar 1999; USEPA 2001).

Figure 2. Relationship between short-term toxicity of cadmium and ambient water hardness in water-only toxicity tests with various aquatic species.



A. Only the Daphnia magna study by Chapman et al. (1980) was included in the analysis to minimize variability in the data resulting from varying experimental conditions and genetic strains.
B. Estimated common (i.e., pooled) slope of all species and associated 95% confidence intervals.

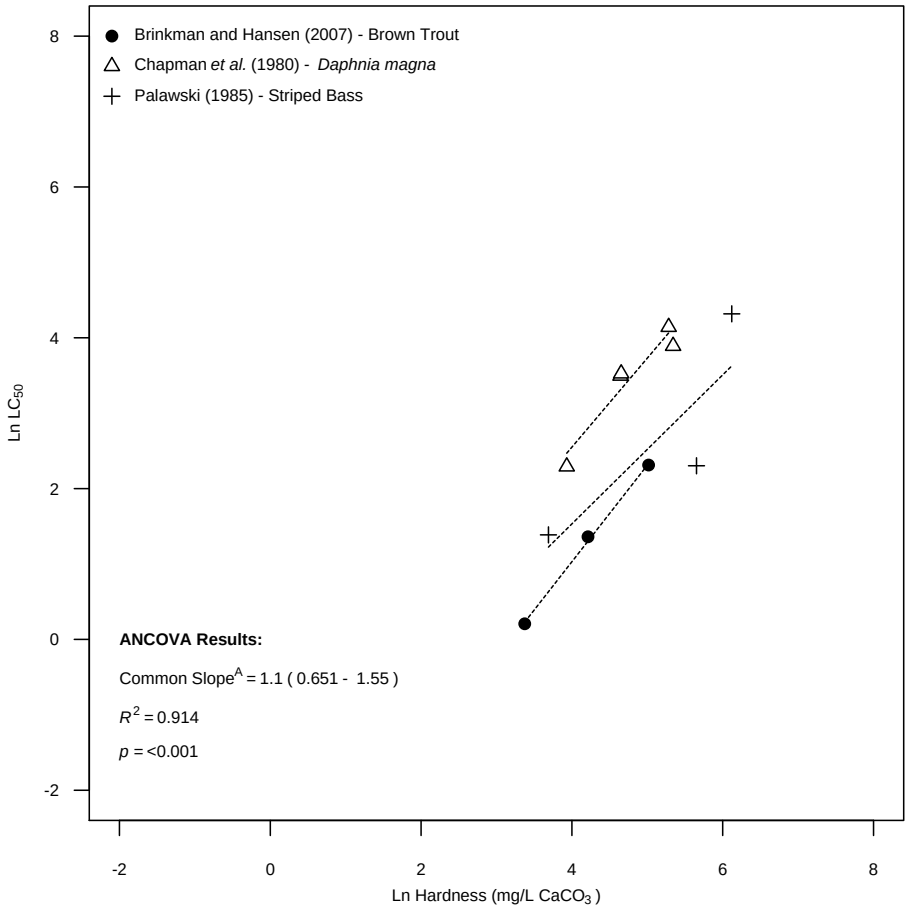
estimated to be 1.03 (n = 39, R^2 = 0.786, p < 0.001) with a 95% confidence interval around the slope of 0.61 to 1.45. Using the common slope, the raw toxicological data can be normalized to a standard hardness (i.e., 50 mg/L $CaCO_3$) using the following equation:

$$EC_x \text{ (at 50 mg/L } CaCO_3) = e^{[((\ln(50) - \ln(\text{Measured Hardness})) * 1.03) + \ln(\text{Measured ECx})]}$$

The relationship between short-term toxicity of Cd and water hardness was developed by incorporating the results of toxicity tests from multiple studies to capture the inherent

variability in response at varying water hardness. However, the influence of additional water quality variables, experimental conditions, or mixed life-stages may influence the resulting pooled slope. To test the influence of these factors on the slope estimate, a sensitivity analysis was performed using only those studies for which the intent of the experimental design was to test the change in the short-term response to Cd toxicity at varying water hardness. The LC_{50}s from 3 studies on 3 separate species were identified and a revised slope estimate was determined using the same process (Figure 3). The original slope estimate (1.03) and the estimated pooled slope from the sensitivity analysis (1.04) were in agreement, and thus the original estimated slope was used in the analysis.

Figure 3. Validation analysis of the relationship between short-term toxicity of cadmium and ambient water hardness in individual water-only toxicity tests with various aquatic species.



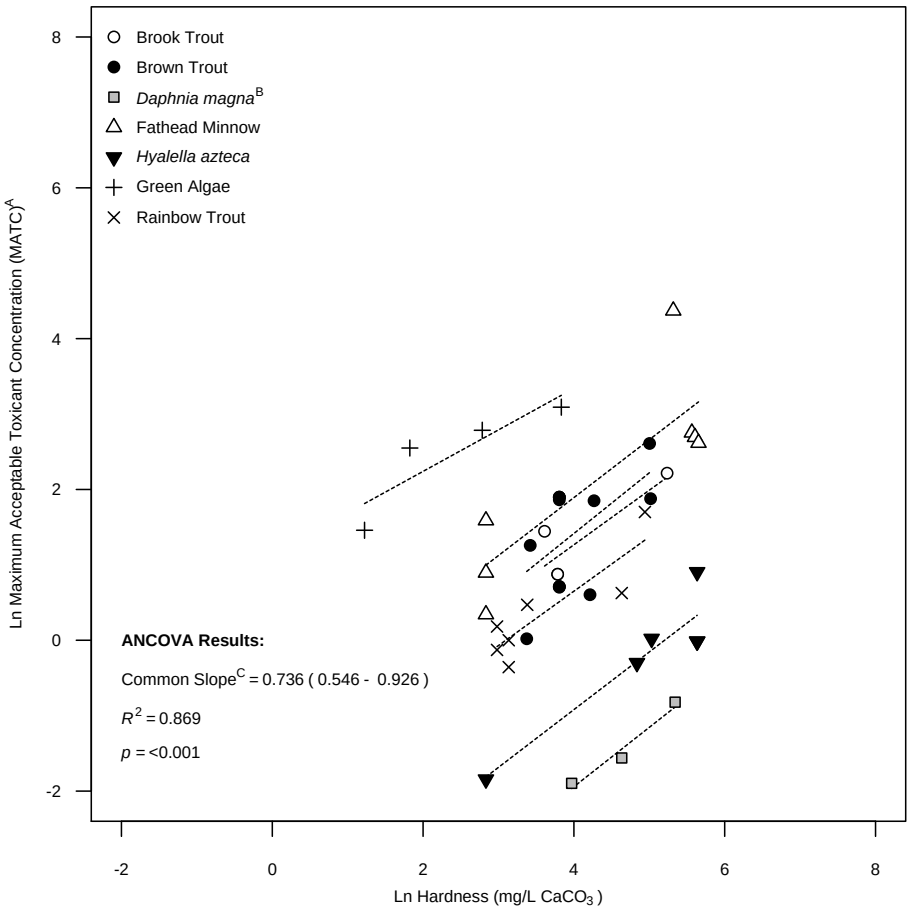
A. Estimated common (i.e., pooled) slope of all species and associated 95% confidence intervals.

9.3.3 Development of the Relationship between Long-Term Toxicity and Water Hardness

Observed effect values from 15 studies were used to evaluate the relationship between water hardness and long-term toxicity (Table A2.5). The majority of the studies used hypothesis testing in the evaluation of toxicity. Therefore, the data used in the analysis of the relationship between long-term toxicity and water hardness were limited to MATCs for survival to ensure that equivalent endpoints were available for each species across a range of water hardness concentrations. For two studies, MATCs for growth and/or biomass were used in order to supplement the data set and complete the analysis (brown trout, *S. trutta*, n = 4; brook trout, n = 5), which facilitated the evaluation of the relationship between toxicity and water hardness for both sub-lethal and lethal effects. Additionally, the estimated LC_{10} (steelhead trout/rainbow trout, n = 2), EC_{20} (rainbow trout, n = 1), and EC_{20} (green algae, n = 4) were used as no MATCs were calculated in these studies. The variability from the use of multiple endpoints within a species data set was not found to be significant. A total of 7 species, comprising 44 data points, were used in the evaluation (fathead minnow, n = 7; rainbow trout, n = 7; brown trout, n = 10; brook trout, n = 7; *H. azteca*, n = 6; *D. magna*, n = 3; green algae, n = 4; Figure 4).

The methods for developing the relationship were consistent with the methods followed to develop the short-term relationship. Linear regression models were applied to each of the individual species. The results of the regression analysis are presented in Table 7. The individual slopes ranged from 0.550 (green algae) to 0.804 (brown trout), with coefficients of determination (i.e., R^2) ranging from 0.347 (brown trout) to 0.963 (*D. magna*). The ANCOVA procedure (Zar 1999) was used to develop a common (i.e., pooled) slope to normalize the toxicological data that were compiled from the primary and secondary studies. The first step of the ANCOVA procedure was to use the *F*-test to test that the estimated slopes for the individual species were not statistically different. It was found that the slopes were not statistically different ($F = 0.0846$, $p = 0.997$) and

Figure 4. Relationship between long-term toxicity of cadmium and ambient water hardness in water-only toxicity tests with various aquatic species.



A. MATCs were calculated as the geometric mean of the NOEC and LOEC for survival from individual toxicity tests, except for the use of the EC_{20} [rainbow trout; n = 1; Mebane *et al.* (2008) and green algae; n = 4; Källqvist (2009)], LC_{10} [steelhead/rainbow trout; n = 2; Chapman (1978)], the MATC for biomass [brown trout; n = 4; brook trout; n =3; Eaton *et al.* (1978)], and the MATC for growth [brook trout; n = 2; Benoit *et al.* (1976)].
B. Only the *Daphnia magna* study by Chapman *et al.* (1980) was included in the analysis to minimize variability in the data resulting from varying experimental conditions and genetic strains.
C. Estimated common (i.e., pooled) slope of all species and associated 95% confidence intervals.

Table 7. Estimate of the slope for use in developing a hardness-modifying conversion factor for long-term toxicity of cadmium based on water-only toxicity tests with various aquatic species.

Receptor Group / Species	Life Stage	Test Duration	Effect Level and Endpoint	n	Slope Estimation			R^2	*p*	Reference
					Slope	95% LCL	95% UCL			
Plants/Algae										
Pseudokirchneriella subcapitata (Green algae)	NA	72-h	EC_{20}; Growth Rate	4	0.55	-0.331	1.43	0.783	0.115	Källqvist (2009)
Fish										
Pimephales promelas (Fathead minnow)	Larvae	7-d	MATC; Survival	7	0.768	0.159	1.38	0.677	0.0230*	Castillo & Longley (2001)
	Fry	30-d	MATC; Survival							Pickering & Gast (1972)
	NR	7-, 10-, 14-d	MATC; Survival							Suedel *et al.* (1997)
Fish - Salmonids										
Salvelinus fontinalis (Brook trout)	Juv	84-, 112-d	MATC; Growth	7	0.724	-0.217	1.66	0.439	0.105	Benoit *et al.* (1976)
	ESL	55-, 89-, 150-d	MATC; Biomass							Eaton *et al.* (1978)
	Fry	60-d	MATC; Survival							Sauter *et al.* (1976)
Salmo trutta (Brown trout)	Fry, ELS	30-, 55-d	MATC; Survival	10	0.804	-0.0969	1.7	0.347	0.0734	Brinkman & Hansen (2007)
	ELS	31-, 63-, 83-, 110-d	MATC; Biomass							Eaton *et al.* (1978)
Oncorhynchus mykiss (Rainbow trout)	Swim-up	28-d	MATC; Survival	7	0.720	0.260	1.18	0.763	0.0102*	Besser *et al.* (2007)
	Juv	30-d	MATC; Survival							Hollis *et al.* (1999)
	ESL	53-, 62-, 69-d	MATC; Survival							Mebane *et al.* (2008)
	Swim-up, Parr	8.3-d	LC_{10}; Survival							Chapman (1978)
Invertebrates										
Daphnia magna (Water flea)	< 24-h old	21-d	MATC; Reproductionl	3	0.787	-1.18	2.76	0.963	0.124	Chapman *et al.* (1980)
Hyalella azteca (Amphipod)	7-8 d old	28-d	MATC; Survival	6	0.765	0.323	1.21	0.853	0.00849**	CEC, Inc. (2004)
	7-8 d old	28-, 35-, 42-d	MATC; Survival							Ingersoll & Kemble (2001)
	NR	14-d	MATC; Survival							Suedel *et al.* (1997)
Common Slope (All species)[A]				**44**	**0.736**	**0.546**	**0.926**	**0.869**	**< 0.001*** **	
Common Slope (All significant relationships)[A]				**20**	**0.758**	**0.514**	**1.000**	**0.876**	**< 0.001*** **	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. LCL = lower confidence limit; UCL = upper confidence limit; CEC, Inc. = Chadwick Ecological Consultants, Inc.; d = day; ESL = early life stage; h = hour; juv = juvenile; LC = lethal concentration; MATC = maximum acceptable toxicant concentration; n = number; NR = not reported.

[A] Common (i.e., pooled) slope estimated using the ANCOVA method (Zar 1999; USEPA 2001)

therefore the common slope was used. Using the pooled data to establish a relationship between long-term toxicity and water hardness, the common slope was estimated to be 0.736 (n = 44, R^2 = 0.869, $p < 0.001$) with a 95% confidence interval around the slope of 0.546 to 0.926. Using the common slope, the raw toxicological data can be normalized to a standard hardness (i.e., 50 mg/L $CaCO_3$) using the following equation:

$$EC_x \text{ (at 50 mg/L } CaCO_3) = e^{[((\ln(50) - \ln(\text{Measured Hardness})) * 0.736) + \ln(\text{Measured ECx})]}$$

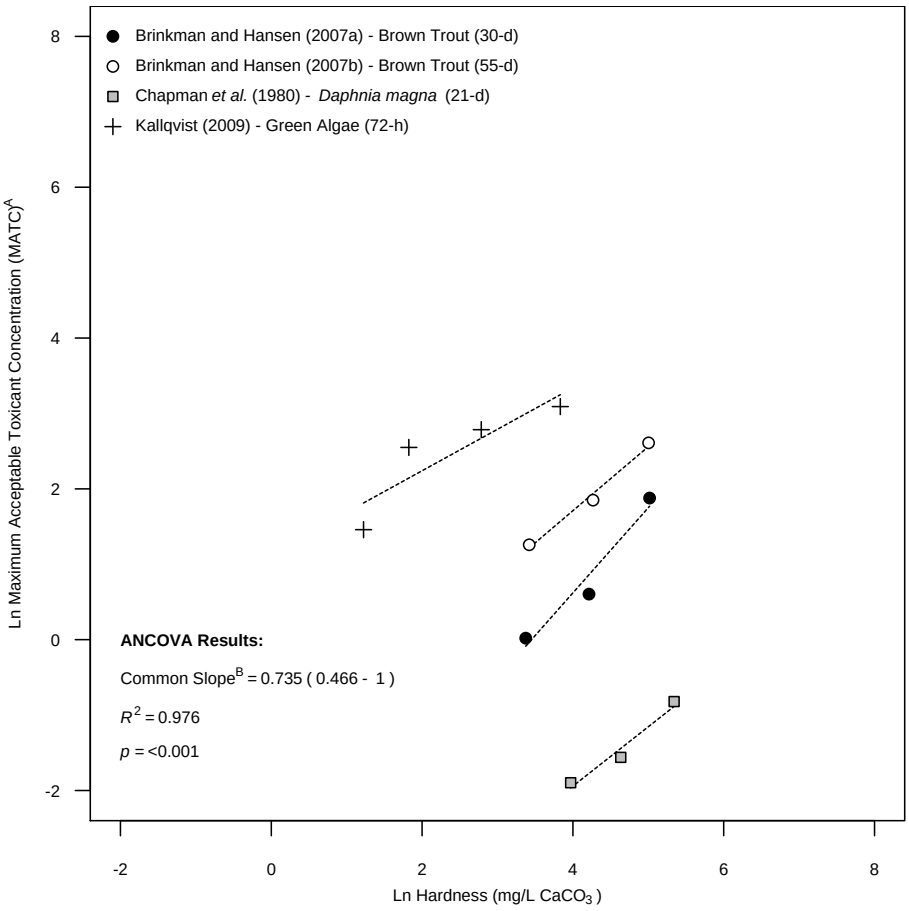
The relationship between long-term toxicity of Cd and water hardness was developed by incorporating the results of toxicity tests from multiple studies to capture the inherent variability in response at varying water hardness. However, the influence of additional water quality variables, experimental conditions, or life-stages may influence the resulting pooled slope. To test the influence of these factors on the slope estimate, a sensitivity analysis was performed using only those studies for which the intent was to test the change in response to Cd toxicity at varying water hardness. The MATCs from 4 studies on 3 species were identified and a revised slope estimate was determined using the same process (Figure 5). The original slope estimate (0.736) and the estimated pooled slope from the validation analysis (0.735) were in agreement, and thus the original estimated slope was used in the analysis.

9.3.4 Evaluation of Developed Relationships for Normalizing Toxicity to a Standard Hardness

To facilitate the development of a short-term maximum and long-term average WQG for Cd, the toxicological data used in the derivation process were normalized to a standard hardness using procedures in USEPA (1985b), USEPA (2001), and Mebane (2010).

The derived slopes for normalizing the toxicological data to a standard hardness were compared to slopes estimated using similar approaches (USEPA 2001; Mebane 2010;

Figure 5. Validation analysis of the relationship between long-term toxicity of cadmium and ambient water hardness in individual water-only toxicity tests with various aquatic species.



A. MATCs were calculated as the geometric mean of the NOEC and LOEC for survival from individual toxicity tests, except for the use of the EC_{20} [green algae; n = 4; Källqvist (2009)].
B. Estimated common (i.e., pooled) slope of all species and associated 95% confidence intervals.

Table 8). Using the approach described in Section 9.3.2, the estimated slope for normalizing the short-term toxicological data was 1.03 (0.610 - 1.45). This slope was similar to that calculated for the update of the USEPA WQC for Cd of 1.02 (0.975 - 1.06; USEPA 2001) as well as the slope calculated by CCME of 1.016 (CCME 2014a). However, this slope was slightly higher than that which was derived by Mebane (2010) using additional data. The slope derived in this case was 0.84 (0.676 - 1.00). The derived slope estimate used to develop the short-term maximum WQG for BC is similar

to the theoretical slope of 1.0 derived in Meyer (1999), and is determined to be a robust method for normalizing the short-term toxicological data.

Table 8. Comparison of slope estimates used in developing a hardness-modifying conversion factor for short-term and long-term toxicity of cadmium based on water-only toxicity tests with various aquatic species.

Exposure Period / Agency	Slope Estimation			R^2	P^A	Reference
	Slope	95% Lower Confidence Limit	95% Upper Confidence Limit			
Short-Term						
BCMOE	1.03	0.61	1.45	0.786	< 0.001***	This document
USGS	0.840	0.676	1.00	0.978	< 0.01**	Mebane (2010)
USEPA	1.017	0.975	1.059	0.967	< 0.05*	USEPA (2001)
CCME	1.016	NA	NA	0.966	NA	CCME (2014a)
Long-Term						
BCMOE	0.736	0.546	0.926	0.869	< 0.001***	This document
USGS	0.625	0.533	0.716	0.991	< 0.001***	Mebane (2010)
USEPA	0.741	0.336	1.146	0.994	< 0.05*	USEPA (2001)
CCME	0.83	NA	NA	0.985	NA	CCME (2014a)

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. BCMOE = British Columbia Ministry of the Environment; USGS = United States Geological Survey; USEPA = United States Environmental Protection Agency; CCME = Canadian Council of Ministers of the Environment; NA = not applicable.
[A] *p*-value as it was reported in the document; values should not be used to compare relative significance between methods.

Using the approach described in Section 9.3.3, the estimated slope for normalizing the long-term toxicological data was 0.736 (0.546 - 0.926). This slope was similar to that calculated for the update of the USEPA WQC for Cd of 0.741 (0.336 - 1.15; USEPA 2001), the slope derived by CCME of 0.83 (CCME 2014a), and that which was derived by Mebane (2010) using additional data of 0.625 (0.533 - 0.716), and is determined to be a robust method for normalizing the long-term toxicological data.

9.4 Derivation of Water Quality Guidelines

A total of 20 short-term and 28 long-term studies were classified as primary studies. These data provided sufficient information to meet the minimum data requirements for

developing a short-term maximum and long-term average WQG for Cd as outlined in BC MOE (2012) and summarized below:

- 3 studies on freshwater fish species resident in BC, including two cold-water species;
- 2 studies on aquatic invertebrates from different classes, including one planktonic species resident in BC;
- 1 study on a freshwater vascular plant or algal species resident in BC (data for long-term exposure only were available); and,
- When available, toxicological data on amphibians should be included.

The WQGs for Cd were developed based on the results of toxicity tests in water-only exposures under laboratory conditions. While there is some evidence to support that the bioaccumulation of Cd (i.e., through the ingestion of contaminated prey and direct contact with water) can contribute to the overall body burden in some species and/or trophic levels, dietary studies have generally indicated that the contribution is not significant. Further, the mechanism for bioaccumulation and conditions under which bioaccumulation is a contributing factor in Cd body burden have not been clearly established.

The laboratory studies evaluated in the derivation process used cadmium chloride ($CdCl_2$), cadmium nitrate ($Cd[NO_3]_2$), or cadmium sulphate ($CdSO_4$). All values reported in this document are reported as dissolved Cd, and as such, the WQG will be determined for this form.

9.4.1 Short-Term Maximum Water Quality Guidelines

The 20 primary short-term studies contained data on 9 resident fish species (including 6 cold-water species), 13 resident invertebrate species, and 1 resident amphibian species (Table 9). These data were considered sufficient to meet the minimum data requirements

Table 9. Studies used to meet the data requirements for developing a short-term water quality guideline.

Species (all resident in BC)	Dur.	Normalized LC_{50} (µg/L; CI)[A]	Reference
Fish - Non-Salmonid Species			
Cottus bairdi	96-h	1.38 (1.05 - 1.81)	Besser *et al.* (2007)
Cottus confusus	96-h	2.27 (1.52 - 3.42)	Mebane *et al.* (2012)
Pimephales promelas	48-h	27.0 (23.4 - 30.7)	Suedel *et al.* (1997)
Fish - Salmonid Species			
Oncorhynchus clarkii lewisi	96-h	2.00 (1.75 - 2.33)	Mebane *et al.* (2012)
Oncorhynchus mykiss	96-h	1.76 (1.52 - 2.04)	Besser *et al.* (2007)
Oncorhynchus mykiss	96-h	2.23 (1.78 - 2.45)	Chapman (1978)
Oncorhynchus mykiss	96-h	4.00 (3.43 - 4.57)	Cusimano *et al.* (1986)
Oncorhynchus mykiss	5-d	0.576 (0.528 - 0.64)	Hansen *et al.* (2002a)
Oncorhynchus mykiss	96-h	0.831 (0 - 1.22)	Mebane *et al.* (2012)
Oncorhynchus tshawytscha	96-h	4.01 (3.78 - 4.45)	Chapman (1978)
Prosopium williamsoni	96-h	4.92 (4.58 - 5.29)	Brinkman & Vieira (2008)
Salmo trutta	96-h	2.14 (NR)	Brinkman & Hansen (2007)
Salvelinus confluentus	5-d	1.37 (1.26 - 1.50)	Hansen *et al.* (2002a)
Invertebrates			
Baetis tricaudatus	96-h	13.5 (0.843 - 265)	Mebane *et al.* (2012)
Chironomus riparius	96-h	753 (428 - 1330)	Watts & Pascoe (2000)
Chironomus dilutus	48-h	89,800 (64,600 - 115,000)	Suedel *et al.* (1997)
Chironomus dilutus	96-h	719 (342 - 1450)	Watts & Pascoe (2000)
Rhithrogena sp.	96-h	321 (184 - 557)	Mebane *et al.* (2012)
Rhithrogena hageni	96-h	11,000 (NR)	Brinkman & Johnston (2008)
Gammarus pulex	96-h	15.7 (NR)	Felten *et al.* (2008)
Hyalella azteca	96-h	8.51 (7.29 - 10.0)	Suedel *et al.* (1997)
Ceriodaphnia dubia	48-h	37.3 (NR)	Black (2001)
Ceriodaphnia dubia	48-h	39.6 (26.9 - 48.1)	Shaw *et al.* (2006)
Ceriodaphnia dubia	96-h	51.3 (41.9 - 60.8)	Suedel *et al.* (1997)
Daphnia ambigua	48-h	12.7 (7.07 - 15.6)	Shaw *et al.* (2006)
Daphnia magna	48-h	73.5 (51.1 - 106)	Schuytema *et al.* (1984)
Daphnia magna	48-h	127 (67.9 - 283)	Shaw *et al.* (2006)
Daphnia magna	96-h	8.03 (7.27 - 8.79)	Suedel *et al.* (1997)
Daphnia pulex	48-h	56.6 (46.7 - 63.6)	Shaw *et al.* (2006)
Simocephalus serrulatus	96-h	33.0 (25.4 - 42.9)	Giesy *et al.* (1977)
Orconectes virilis	96-h	12,000 (9,220 - 15,500)	Mirenda (1986)
Amphibians			
Ambystoma gracile	96-h	522 (NR)	Nebeker *et al.* (1995)

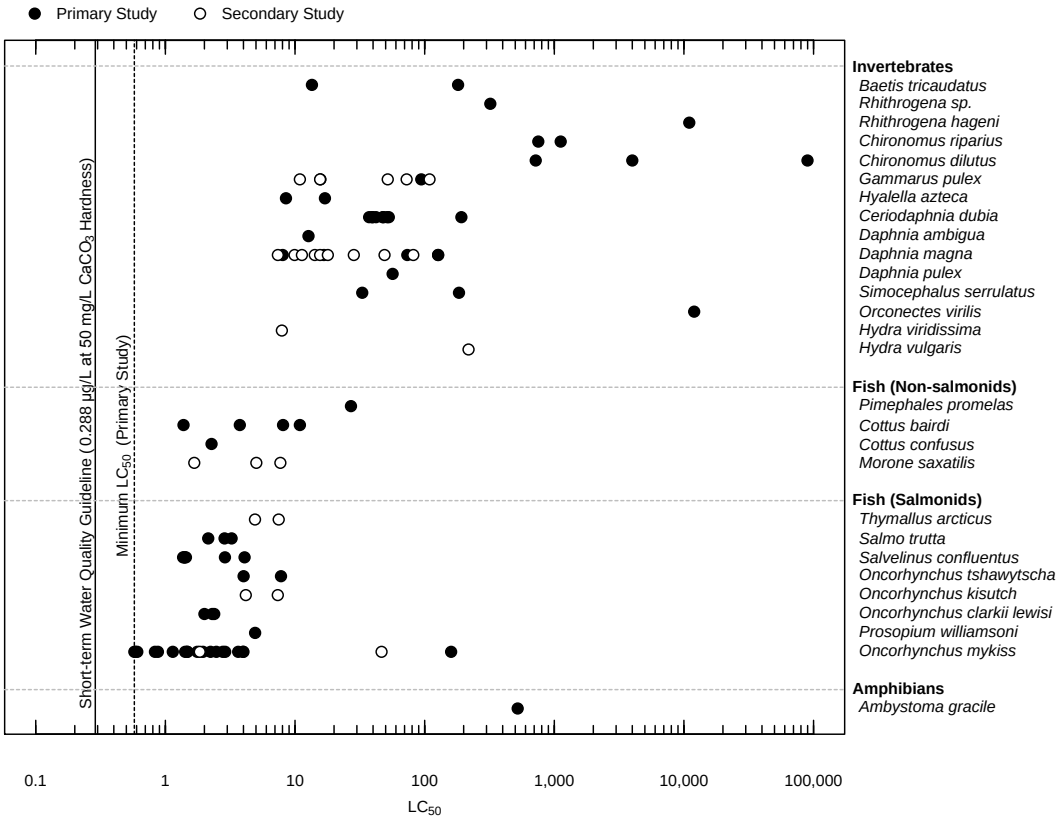
Dur = duration; LC = lethal concentration; CI = confidence interval; -h = hour; -d = day; NR = Not Reported.
[A] The toxicity test endpoint corresponding to the lowest normalized LC_{50} is reported.

for the development of a short-term maximum WQG. While no toxicity data were available that met the short-term exposure criteria for algae or aquatic plants, a comparison of the derived short-term maximum WQG and the long-term exposure data

for algae and aquatic plants showed that the derived short-term maximum WQG would be protective of these groups.

The primary and secondary studies generated 101 defined LC_{50} values for species resident in BC. These effect concentrations were normalized to a standard hardness of 50 mg/L $CaCO_3$ using the equation reported in Section 9.3.2. These normalized values were then plotted to show the distribution of effect values by species (Figure 6). The lowest

Figure 6. Distribution of LC_{50} values[A] from the primary and secondary studies used to determine the short-term toxicity of cadmium in freshwater environments.



A. All values are normalized to a hardness of 50 mg/L $CaCO_3$.

effect value from a primary study (0.576 µg/L; LC_{50} for rainbow trout fry; Hansen *et al.* 2002a) was selected to support the derivation of a short-term maximum WQG.

In order to derive a WQG that is protective of the most sensitive species and life stage in BC, uncertainty around the lowest effect value was taken into account. The minimum uncertainty factor of 2 was applied to the selected effect value to derive the WQG. As stated in BC MOE (2012), sources of uncertainty in the protectiveness of a lowest effect level from a laboratory study include:

- Laboratory to field differences;
- Interactive effects of multiple contaminants;
- Toxicity of metabolites;
- Intra-specific and inter-specific differences in sensitivity;
- Indirect effects (e.g., associated with bioaccumulation);
- Duration of exposure, relative to the life-cycle of the species;
- Delayed effects;
- Presence of other stressors (e.g., habitat loss); and,
- Impacts of climate change.

The minimum uncertainty factor of 2 for the short-term maximum guideline is supported by the mean ratio of the LC_{50} to the LC_{10} from a short-term exposure study using rainbow trout (the most sensitive species; Mebane 2012). The results of the toxicity tests conducted by Mebane (2012) and used to calculate the ratios (in parentheses) are:

- Test 9; LC_{50}: 0.48 µg/L Cd, LC_{10}: 0.38 µg/L Cd (1.26);
- Test 10; LC_{50}: 0.99 µg/L Cd, LC_{10}: 0.66 µg/L Cd (1.50);
- Test 11; LC_{50}: 1.30 µg/L Cd, LC_{10}: 0.92 µg/L Cd (1.41);
- Test 13; LC_{50}: 0.93 µg/L Cd, LC_{10}: 0.57 µg/L Cd (1.63);
- Test 14; LC_{50}: 0.83 µg/L Cd, LC_{10}: 0.58 µg/L Cd (1.43); and,
- Test 15; LC_{50}: 0.34 µg/L Cd, LC_{10}: 0.11 µg/L Cd (3.09).

Based on the results of 6 tests, the ratio of the LC_{50} to the LC_{10} ranged from 1.26 to 3.09 (geometric mean = 1.64). Therefore, the minimum uncertainty factor of 2 was considered to be protective of the most sensitive species against short-term effects on survival (i.e., < 10% mortality). The resultant WQG of 0.288 µg/L at 50 mg/L $CaCO_3$ is also protective against adverse effects on behaviour in rainbow trout (e.g., Sloman *et al.* 2003).

The recommended short-term maximum WQG for Cd (0.288 µg/L) is directly applicable to waters with a hardness of 50 mg/L $CaCO_3$. The following equation is recommended to calculate short-term maximum WQGs for other waters:

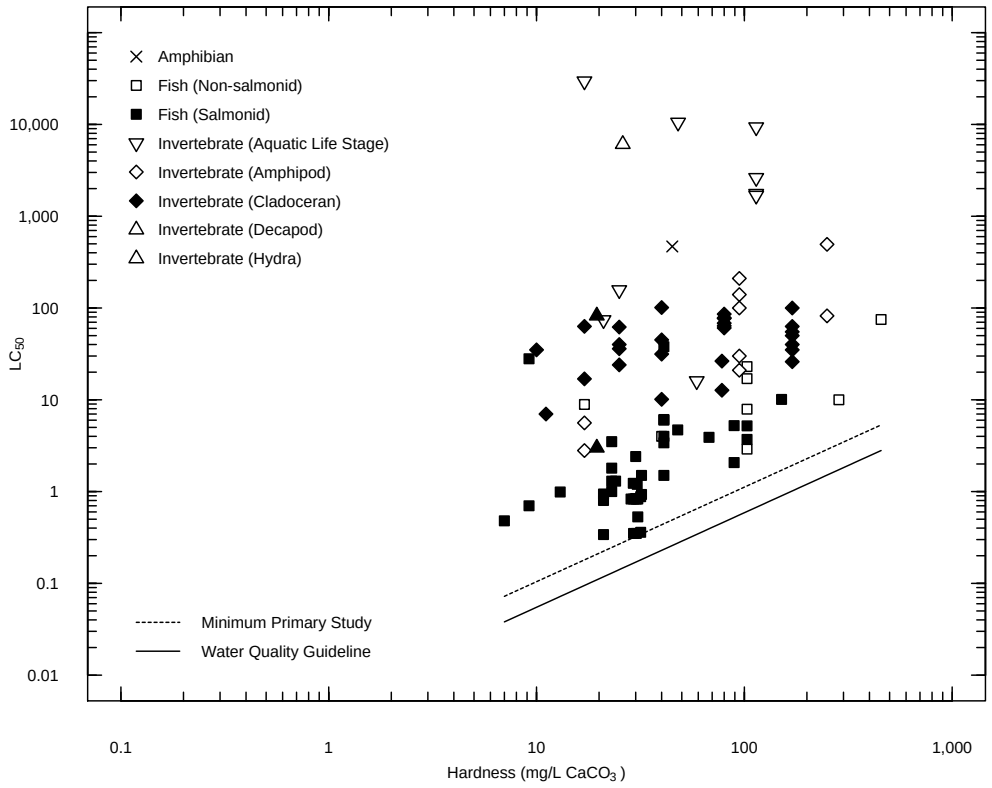
$$WQG_{SHORT\text{-}TERM} = e^{[1.03 * \ln(Hss) - 5.274]}$$

Where: H_{SS} = site-specific water hardness (mg/L $CaCO_3$)

The short-term maximum WQGs derived using this equation were evaluated against the raw toxicological data presented in the primary and secondary studies (Figure 7). Because none of the reported effect values for Cd were less than the short-term WQG, it was concluded that the recommended short-term WQG was protective of a diverse range of organisms and water quality conditions (i.e., as indicated by water hardness).

As the range of water hardness in the water-only toxicity tests used to derive the relationship between short-term toxicity and hardness was limited to between 7 and 455 mg/L $CaCO_3$, the site-specific WQG should be bounded to this range. In waters with water hardness below 7 mg/L $CaCO_3$, the guideline should be calculated with a water hardness of 7 mg/L $CaCO_3$. In conditions with water hardness above 455 mg/L $CaCO_3$, a site-specific assessment may be required.

Figure 7. Distribution of responses from water-only toxicity tests classified as primary and secondary studies relative to the short-term maximum guideline for cadmium.



9.4.2 Long-Term Average Water Quality Guidelines

The 28 primary long-term studies contained data on 1 BC resident aquatic plant species, 1 resident algal species, 13 resident fish species (including 8 salmonid species), 11 resident invertebrate species, and 1 resident amphibian species (Table 10). These data were considered sufficient to meet the minimum data requirements for the development of a long-term average WQG. In addition, a total of 15 studies provided secondary long-term data that were used to support the development of the guideline.

The primary and secondary studies were used to compile 394 effect values for species resident in BC. These results were normalized to a hardness of 50 mg/L CaCO3 using

Table 10. Studies used to meet the data requirements for developing a long-term water quality guideline.

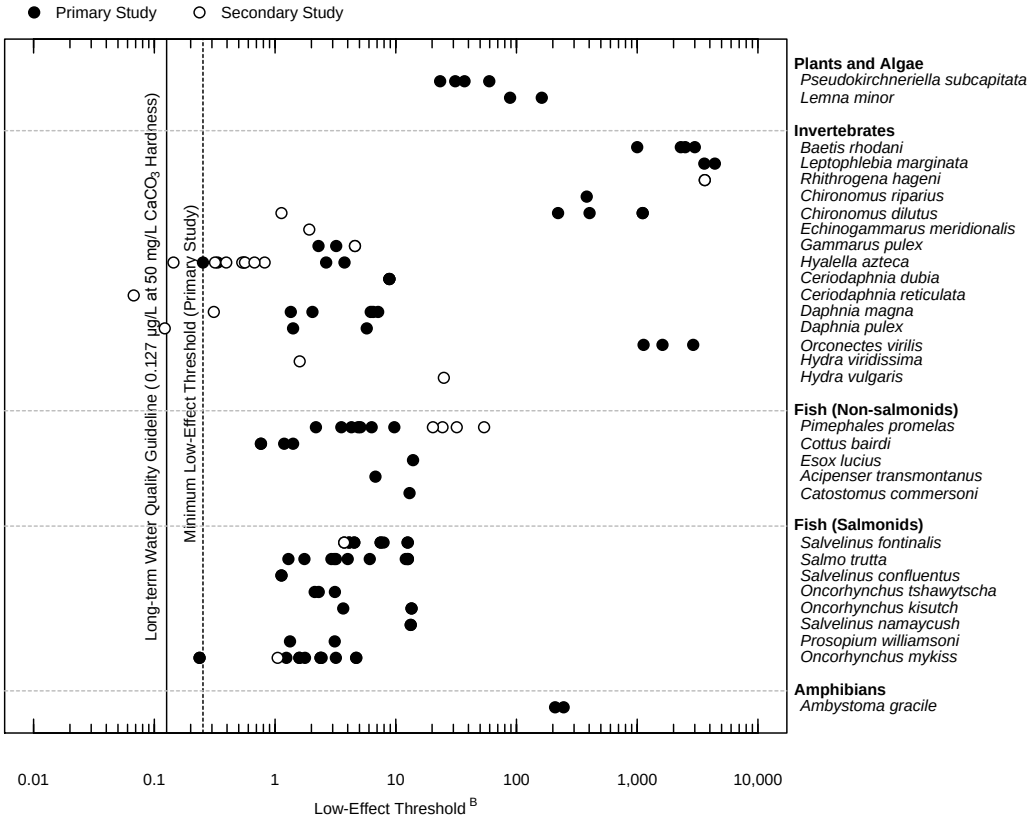
Receptor Group / Species (all resident in BC)	Selected Toxicity Test Endpoint[A]	Dur.	Normalized Effect Value (µg/L; CI)	Reference
Fish – Non-Salmonid Species				
Catostomus commersoni	LOEC; bio	40-d	13	Eaton *et al*. (1978)
Cottus bairdi	LOEC; bio	21-d	0.764	Besser *et al*. (2007)
Esox lucius	LOEC; bio	35-d	13.9	Eaton *et al.* (1978)
Pimephales promelas	LOEC; gro	7-d	2.18	Castillo & Longley (2001)
Pimephales promelas	LC_{50}	10-d	3.54 (2.88 - 4.42)	Suedel *et al*. (1997)
Acipenser transmontanus	LC_{20}	27-d	6.79 (6.17 - 7.42)	Vardy *et al.* (2011)
Fish – Salmonid Species				
Oncorhynchus kisutch	LOEC; bio	27-d	3.67	Eaton *et al.* (1978)
Oncorhynchus mykiss	LOEC; bio	28-d	1.59	Besser *et al.* (2007)
Oncorhynchus mykiss	LC_{10}	200-h	1.24 (1.06 - 1.42)	Chapman (1978)
Oncorhynchus mykiss	LC_{50}	7-d	2.43 (2.09 - 2.78)	Cusimano *et al.* (1986)
Oncorhynchus mykiss	LOEC; mor	30-d	4.69	Hollis *et al.* (1999)
Oncorhynchus mykiss	LOEC; mor	30-d	3.19	Hollis *et al.* (2000b)
Oncorhynchus mykiss	LOEC; gro	62-d	0.237	Mebane *et al.* (2008)
Oncorhynchus tshawytscha	LC_{10}	200-h	2.13 (1.59 - 2.48)	Chapman (1978)
Oncorhynchus tshawytscha	LOEC; mor	120-d	3.13	Chapman (1982)
Prosopium williamsoni	IC_{20}; bio	90-d	1.33 (NR)	Brinkman & Vieira (2008)
Salmo trutta	IC_{20}; bio	30-d	1.29 (NR)	Brinkman & Hansen (2007)
Salmo trutta	LOEC; bio	63-d	4.00	Eaton *et al.* (1978)
Salvelinus confluentus	LOEC; gro	55-d	1.13	Hansen *et al*. (2002b)
Salvelinus fontinalis	LOEC; bio	150-d	4.11	Eaton *et al.* (1978)
Salvelinus fontinalis	LOEC; gro	60-d	4.53	Sauter *et al.* (1976)
Salvelinus namaycush	LOEC; bio	74-d	13.3	Eaton *et al.* (1978)
Invertebrates				
Baetis rhodani	LC_{50}	120-h	1000 (NR)	Gerhardt (1992)
Chironomus riparius	LC_{50}	10-d	382 (273 - 545)	Watts & Pascoe (2000)
Chironomus dilutus	LOEC; gro	14-d	221	Suedel *et al*. (1997)
Chironomus dilutus	LC_{50}	10-d	403 (273 - 600)	Watts & Pascoe (2000)
Leptophlebia marginata	LC_{50}	120-h	3600 (NR)	Gerhardt (1992)
Rhithrogena hageni	LOEC; mor	10-d	3,630	Brinkman & Johnston (2008)
Gammarus pulex	LOEC; mor	120-h	2.29	Felten *et al.* (2008)
Hyalella azteca	IC_{20}; bio	28-d	0.253 (0.0 - 1.11)	CEC, Inc. (2004)
Hyalella azteca	LOEC; mor	14-d	0.553	Suedel *et al.* (1997)
Ceriodaphnia dubia	LOEC; rep	14-d	8.85	Suedel *et al.* (1997)
Daphnia magna	IC_{20}; rep	21-d	1.35 (1.16 - 1.73)	CEC, Inc. (2004)
Daphnia magna	LC_{50}	14-d	6.20 (5.48 - 6.92)	Suedel *et al.* (1997)
Daphnia pulex	IC_{20}; mor	18-d	1.41 (0.358 - NC)	CEC, Inc. (2004)
Daphnia pulex	LOEC; mor	60-d	5.75	Ingersoll & Winner (1982)
Orconectes virilis	LC_{50}	14-d	1,130 (809 - 1,590)	Mirenda (1986)
Plants/Algae				
Lemna minor	EC_{50}; gro rate	7-d	88.5 (NR)	Drost *et al.* (2007)
Pseudokirchneriella subcapitata	EC_{20}; gro rate	72-h	23.3 (15.9 - 35.0)	Källqvist 2009
Amphibians				
Ambystoma gracile	LOEC; gro	24-d	209	Nebeker *et al*. (1995)

CI = confidence interval; Dur. = duration; CEC, Inc. = Chadwick Ecological Consultants, Inc.; EC = effective concentration; IC = inhibitory concentration; LC = lethal concentration; LOEC = lowest observed effect concentration; bio = biomass; gro = growth; mor = mortality; rep = reproduction; NR = not reported; NC = not calculable.

[A] The toxicity test endpoint corresponding to the lowest normalized effect value is reported.

the equation described in Section 9.3.3. These data included multiple endpoints and effect levels for the same species, life-stage, and test duration; therefore the data were sorted and only the endpoint-effect combinations that yielded the lowest effects thresholds (i.e., most sensitive) from each study were selected for further use in the guideline derivation. From this process, 116 low effect values were selected for deriving the WQG (Table A2.6). These values were plotted to show the distribution of effect values by species (Figure 8). The minimum effect value from a primary study (0.253

Figure 8. Distribution of low-effects threshold values[A,B] from the primary and secondary studies used to determine the long-term toxicity of cadmium in freshwater environments.



A. All values are normalized to a hardness of 50 mg/L $CaCO_3$.
B. All low−effect thresholds used in the analysis are presented in Table A2.6.

µg/L; IC20 for H. azteca biomass; Chadwick Ecological Consultants, Inc. 2004) was used to support the derivation of the long-term average WQG. Similarly, a LOEC of 0.237 was

reported for *O. mykiss* growth (Mebane et al. 2008); however, the authors reported that a clear dose-response relationship was not apparent and suggested that the endpoint was not a robust estimate of an effects threshold.

Sufficient data were not available in the literature to quantitatively evaluate the uncertainty associated with the lowest effect levels that were selected to support derivation of the long-term average WQG (0.253 µg/L Cd at 50 mg/L $CaCO_3$ hardness; Chadwick Ecological Consultants, Inc. 2004). However, data from a secondary study on cladocerans indicated that the reproduction of *D. pulex* in 14-d exposures or of *C. reticulata* in 7-d exposures has the potential to be adversely affected at Cd concentrations of 0.122 and 0.0675 µg/L Cd at 50 mg/L $CaCO_3$ hardness, respectively (Elnabaraway *et al.* 1986). However, there is a high level of uncertainty in the results of the Elnabaraway study (i.e., only nominal concentrations were reported and there were typographical errors in the form of Cd reported). In addition, data from a study that did not report all of the information required to receive a primary or secondary classification indicated that no adverse effects on the feeding rates or brood mass of *D. magna* were observed at 0.081 µg/L Cd at 50 mg/L $CaCO_3$ (Barata and Baird 2000). In another secondary study, Ingersoll and Kemble reported an IC_{25} for *H. azteca* biomass of 0.144 µg/L Cd at 50 mg/L $CaCO_3$; however, a clear dose-response relationship was not observed in the underlying data. When considered together with the other information contained in the toxicological data set, these data suggest that application of the minimum uncertainty factor of 2 to the selected lowest effect level is likely to result in a long-term average WQG that would protect the most sensitive life-stages of the most sensitive species of aquatic organisms in BC waters. Therefore, a long-term average WQG of 0.127 µg/L is recommended for Cd at a water hardness of 50 mg/L $CaCO_3$.

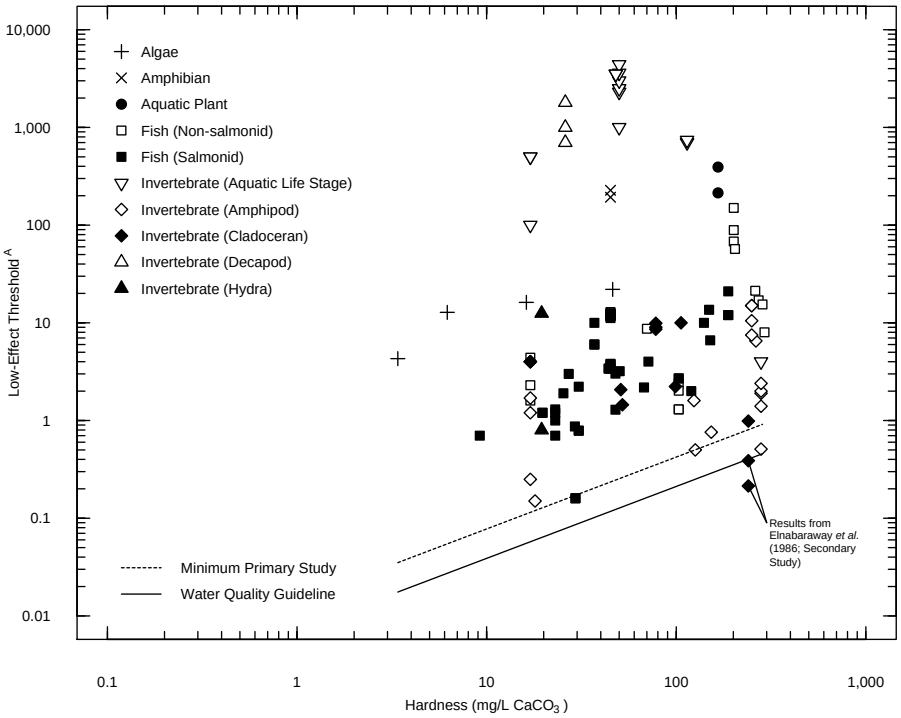
It is recommended that the long-term (30-d) average WQG for Cd to protect the most sensitive life-stage of the most sensitive species be adjusted to the site-specific water hardness using the following equation:

$$WQG_{LONG\text{-}TERM} = e^{[0.736 * \ln(Hss) - 4.943]}$$

Where: H_{SS} = site-specific water hardness (mg/L $CaCO_3$)

The derived long-term average WQG was evaluated against the raw toxicological data presented in the primary and secondary studies (Figure 9). It was determined that the recommended long-term WQG was protective of aquatic life across the full range of organisms and water quality conditions tested.

Figure 9. Distribution of responses from water-only toxicity tests classified as primary and secondary studies relative to the long-term average guideline for cadmium.



A. All low−effect thresholds used in the analysis are presented in Table A2.6.

As the range of water hardness in the water-only toxicity tests used to derive the relationship between long-term toxicity and hardness was limited to between 3.4 and 285 mg/L $CaCO_3$, the site-specific application of the WQG should be bounded to this range. In waters with water hardness below 3.4 mg/L $CaCO_3$, the guideline should be calculated

with a water hardness of 3.4 mg/L $CaCO_3$. In conditions with water hardness above 285 mg/L $CaCO_3$, a site-specific assessment may be required.

9.4.3 Evaluation of Uncertainty in the Water Quality Guidelines for Cadmium

The toxicological data that were used to establish the preliminary short-term maximum and 30-day average WQGs for Cd (i.e., the minimum effect values from the key primary studies) were both derived from regression-based estimates of the relationship between the concentration of Cd and organism response. As such, these regression-based effect values have an associated source of error around the parameter estimate. Specifically, the 95% confidence interval around the selected LC_{50} for rainbow trout fry of 0.36 µg/L Cd (at a hardness of 31.7 mg/L $CaCO_3$; 0.576 µg/L Cd when normalized to 50 mg/L $CaCO_3$) was calculated to be 0.33 to 0.40 µg/L Cd. The error around the selected LC_{50} is minimal and not expected to be a major source of uncertainty in the short-term maximum WQG. However, the error associated with the IC_{20} for the amphipod (*H. azteca*) of 0.5 µg/L Cd (at a hardness of 126 mg/L $CaCO_3$; 0.253 µg/L Cd when normalized to 50 mg/L $CaCO_3$) was reported to be between 0 and 1.11 µg/L Cd (normalized to 50 mg/L $CaCO_3$) in Chadwick Ecological Consultants, Inc. (2004). While the confidence interval around the endpoint estimate is relatively large, additional tests conducted by Chadwick Ecological Consultants, Inc. (2004) reported similar IC_{20} estimates, normalized to 50 mg/L $CaCO_3$ (IC_{20} for survival of 0.265 (0.143 – 0.499) µg/L Cd, IC_{20} for growth 0.335 (0.150 – 0.623) µg/L Cd, and an IC_{20} for survival of 0.400 (0.0878 – 0.505) µg/L Cd). An uncertainty factor of 2 has been applied to the long-term guideline and is expected to produce a conservative guideline.

The toxicological data compiled to develop the short-term maximum and long-term average WQG for Cd were normalized to a standard hardness of 50 mg/L $CaCO_3$ using an approach consistent with methods used by USEPA (2001) and Mebane (2010). As the estimated slopes of the relationship between water hardness and Cd toxicity were developed using a regression-based approach, the error associated with the slope estimate could be ascertained. The 95% confidence interval around the estimated slope of 1.03

used in this study was calculated to be 0.61 to 1.45. The error associated with this slope estimate was not expected to greatly influence the result of the WQG derivation for two reasons. First, the predicted slope established by Meyer (1999) for a mid-range of water hardness (20 to 200 mg/L $CaCO_3$) was determined to be 1.0, based on modelling of the competitive binding of cations in fish gills. Second, USEPA has developed a similar slope estimate (1.02; 0.975 - 1.06), that is consistent with the results of this analysis. However, using the 95% confidence interval derived from our slope to determine the bounds for the LC_{50} selected to support WQG development (i.e., 0.576 µg/L Cd at a hardness of 50 mg/L $CaCO_3$) results in a predicted range of 0.475 to 0.697 µg/L Cd; this is above the short-term maximum WQG of 0.288 µg/L Cd (at a hardness of 50 mg/L $CaCO_3$). Therefore, the error associated with the slope estimate for the short-term toxicological data is not expected to introduce potential risks to aquatic life.

Similarly, the 95% confidence interval around the estimated slope for normalizing the long-term data of 0.736 was calculated to be 0.546 to 0.926. This slope is similar to that calculated by USEPA (2001) and Mebane (2010), and is not expected to greatly influence the results of the WQG derivation. Application of the 95% confidence interval derived from the slope estimate to establish estimated bounds for the effect value selected to support long-term WQG development (0.253 µg/L Cd at a hardness of 50 mg/L $CaCO_3$) results in a predicted range of 0.212 to 0.302 µg/L Cd, which is above the final guideline of 0.127 µg/L Cd (at a hardness of 50 mg/L $CaCO_3$). Therefore, the error associated with the slope estimate for the long-term toxicological data is not expected to introduce potential risks to aquatic life.

The process used to derive the slope estimates for normalizing the toxicological data to a standard hardness involved transforming the raw toxicological data and water hardness using a natural log transformation. The calculation of a slope estimate and back-calculating a predicted effect value may introduce bias into the estimated effect value (Newman 1991). To determine the potential influence on the predicted effect value, the bias was calculated using the method provided in Newman (1991) for the pooled slope

estimates. It was found that the slope estimate could bias the predicted effect value downward (i.e., lower) by 34% for the short-term data, and 16% for the long-term data. However, the bias would not influence the relative position of the normalized effect values and therefore will not affect the derived short-term maximum and long-term average guidelines.

The short-term maximum and long-term average guidelines developed in this document were not derived using information that allows for the direct evaluation of the toxic effects of Cd in combination with other stressors (e.g., the possible synergistic or antagonistic interactions with other contaminants). Rather, the application of an uncertainty factor is meant to account for various uncertainties (including multiple stressors). Additional investigation may be needed at sites with multiple contaminants to ensure the protection of aquatic life. Importantly, the interactions and therefore, toxicity, of other metals that have similar modes of toxicity (e.g., copper, lead, and zinc) is not considered in this guideline. Therefore, site-specific assessments of water quality conditions should utilize toxic units models or similar approaches to evaluate the joint effects of multiple contaminants for substances that have similar modes of toxicity (e.g., USEPA 2005).

The short-term maximum and long-term average guidelines have been developed using the results of toxicity tests in water-only exposures using dissolved Cd salts, which are highly bioavailable under laboratory conditions (i.e., under neutral pH and low DOM). While the short-term maximum and long-term average guidelines have been developed by accounting for the ameliorating effects of hardness, site-specific water-quality conditions can further affect the toxicity of Cd to freshwater organisms. For example, acidic or alkaline systems are expected to affect the uptake and bioavailability of Cd. Furthermore, systems with elevated levels of DOM, especially ligands that readily bind with Cd, may reduce the bioavailability and therefore toxicity of Cd. Site-specific assessments may be conducted for water bodies with naturally high levels of DOM or atypical pH to account for their potential influence on Cd toxicity (BC MOE 2013).

The WQGs for Cd were developed based on the results of toxicity tests in water-only exposures under laboratory conditions. While there is some evidence indicating that bioaccumulation of Cd (i.e., through the ingestion of contaminated prey and direct contact with water) contributes to the overall body burden in some species and/or trophic levels, the mechanism for bioaccumulation and conditions under which bioaccumulation is a primary factor in Cd body burden have not been clearly established. Therefore, Cd body burden in systems that favour the bioaccumulation of Cd in freshwater aquatic organisms may be higher than expected based on direct exposure to water alone.

9.4.4 Application of Water Quality Guidelines for Cadmium

The WQGs for the protection of aquatic life represent levels that are meant to be protective of the most sensitive life-stage of the most sensitive species to either short-term or long-term exposure to Cd. The short-term maximum guideline represents a level that should not be exceeded at any given time (i.e., instantaneous maximum), whereas the long-term average guideline represents a level that the 30-day average concentration (calculated as the mean of 5 weekly samples within 30 days) should not exceed. In order to generate data with a high level of precision and accuracy for the evaluation of water quality conditions relative to the WQGs, it is recommended that MDLs should be, at minimum, five times below the ambient WQG to ensure this higher level of precision and accuracy. However, in cases where laboratories have defined PQLs, is recommended that the PQL be at or below the ambient WQG.

The WQGs for Cd have been developed as equations that take into account the water hardness of the system. Generally, the WQGs for Cd can be directly applied to water bodies in the province. However, as the ameliorating effects of hardness are tightly linked to the pH and alkalinity of the system in natural surface waters, due care should be taken in systems in which the ratio of ions that make up water hardness is significantly altered by anthropogenic discharge. In these systems, the recommended WQG may or may not be protective of all freshwater aquatic species. Furthermore, the recommended WQGs for Cd may or may not be suitable in waters that exhibit elevated levels of DOM,

elevated levels of total dissolved solids (TDS), atypical pH, or saltwater intrusion. In these cases, a site-specific assessment may be required.

The short-term maximum and long-term average WQGs were developed using toxicity results generated from laboratory studies in which aquatic organisms were exposed to water spiked with Cd salts that are highly bioavailable and exist in the exposure chambers in the dissolved fraction. Thus, the derived short-term maximum and long-term average WQGs apply to the measured dissolved Cd fraction in environmental samples. While consideration of dissolved metals does neglect the potential toxicity of particulate-associated metals, most studies indicate that the particulate fraction is substantially less toxic than the dissolved fraction. In addition, sediment quality guidelines (e.g., BC MOE 2006; CCME 2014b) provide a basis for evaluating the toxicity of particulate-associated metals directly (i.e., when metal-contaminated particulates settle out of the water column and become associated with bottom sediments). Tissue residue guidelines provide a basis for evaluating the significance of tissue-associated metals. As the concentrations of total Cd and other metals in BC waters are highly variable, depending in large measure on flow regime and associated changes in the levels of suspended sediments in the water, the application of WQGs (generated using toxicological data reflecting dissolved metal concentrations) to measured concentrations of total metals is likely to result in overly conservative evaluations of water quality conditions. Accordingly, the short-term maximum and long-term average WQGs derived herein are intended to be applied to the dissolved fraction of Cd in environmental samples.

9.4.5 Research and Development Needs

The majority of research on the toxicity of Cd has been completed on fish and aquatic invertebrates, and to a lesser degree, algae and aquatic plants. To better understand the impacts of exposure to Cd on aquatic species, more research is needed on the toxicity of metals (including Cd) on amphibians, especially species that are resident in BC. Amphibians are declining in number around the world (Wake and Vredenburg 2008),

making it especially important to better understand how contaminants like Cd affect them.

An important component in the evaluation of potential risks to aquatic life is the routine generation of reliable and usable data. As such, appropriate MDLs need to be achieved for monitoring programs that are designed to evaluate water quality conditions relative to ambient WQGs. Therefore, there is a need for establishing analytical methods that generate data with a high level of precision and accuracy. MDLs used should be, at minimum, five times below the ambient WQG to ensure this higher level of precision and accuracy. However, in cases where laboratories have defined PQLs, it is recommended that the PQL be at or below the ambient WQG.

While some research on the toxicity of chemical mixtures has been conducted (e.g., Mebane *et al.* 2012), the cumulative effects of multiple contaminants and the associated effects on aquatic life are not completely understood and are difficult to regulate without site-specific assessments of the chemical mixture. Interactions (including synergistic and antagonistic) between these chemicals may increase or decrease their toxicity. In addition, multiple contaminants have the potential to become multiple stressors to aquatic organisms. As such, research on the integration of WQGs in the context of stressor identification and evaluation in a mixture at a site is an area that requires further research.

Finally, the development and application of a biotic-ligand model for Cd have the potential to provide tools that more accurately predict the effects of ambient water quality on metal toxicity in fish, considered to be the most sensitive organisms to short-term exposure to Cd. The integration of the concentrations of metals, cations, and ligands into the assessment of metals toxicity is an important factor in determining how site-specific conditions influence the toxicity of the metals in the system, the cumulative effects of metals contamination, and the impacts on the aquatic life of the system.

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