# SCIENTIFIC CRITERIA DOCUMENT FOR THE DEVELOPMENT OF THE CANADIAN SOIL QUALITY GUIDELINES FOR THE PROTECTION OF ENVIRONMENTAL AND HUMAN HEALTH

## Hexavalent, trivalent and total chromium

# NOTE TO READERS

The Canadian Council of Ministers of the Environment (CCME) is the primary minister-led intergovernmental forum for collective action on environmental issues of national and international concern.

This scientific supporting document provides the background information and rationale for the development of Canadian environmental soil quality guidelines for chromium for the protection of human health. The information in this document is current as of 2022, when the document was revised and updated. For further technical information regarding these guidelines, please contact:

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# EXECUTIVE SUMMARY

Canadian Environmental Quality Guidelines, developed under the auspices of the Canadian Council of Ministers of the Environment (CCME), are numerical concentrations or narrative statements describing the levels of toxic substances or other parameters in soil that are recommended to provide a healthy, functioning ecosystem capable of sustaining the existing and likely future uses of a site by ecological receptors and humans. Canadian Soil Quality Guidelines can be used as the basis for the consistent assessment and remediation of contaminated sites in Canada.

This scientific criteria document provides the background information and rationale for the derivation of soil quality guidelines for hexavalent chromium (Cr(VI)) and trivalent chromium (Cr(III)) for the protection of human health. It contains a review of information on Cr(VI) and Cr(III) with respect to their chemical and physical properties, sources and emissions in Canada, distribution and behaviour in the environment, and toxicological effects in experimental animals and humans. This information was used to derive soil quality guidelines for Cr(VI) (Table 1) and Cr(III) (Table 2) to protect human health for four types of land use—agricultural, residential and parkland, commercial, and industrial—from exposures through three types of exposure pathways: required pathways (direct contact), applicable pathways (indoor air, groundwater, and produce, meat and milk ingestion), and check mechanisms (off-site migration of substances). Human-health-based soil quality guidelines ($SoQG_{HH}$) were derived separately for Cr(VI) and Cr(III) because they do not have the same effects on human health. Soil Quality Guidelines for the protection of environmental health ($SoQG_E$) for Cr(VI) and total chromium (Cr(T)) (Table 1; Table 2) are taken from the 1997 Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health – Chromium (CCME 1999). The soil quality guidelines for the protection of human health provided herein were derived according to the procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006).

In many circumstances, it may be possible to measure total chromium Cr(T) in soil and compare the result to the $SoQG_{HH}$ for Cr(III), because the majority of environmental Cr is expected to be present as Cr(III) compounds (Sections 2.2 and 3.6); however, analytical measurement of Cr(VI) in soil is strongly recommended for any site potentially contaminated by activities involving Cr(VI). Conversely, where speciated data are available, Cr(III) data may be compared to the $SoQG_E$ for Cr(T) for the same reason.

Speciated results for Cr(VI) should be compared to the $SoQG_{HH}$ provided for Cr(VI) while the Cr(III) results may be compared to the $SoQG_{HH}$ for Cr(III).

These human-health-based soil quality guidelines are intended as general guidance. Site-specific conditions should be considered in the application of these values.

212 **Table 1. Soil quality guidelines for hexavalent chromium (Cr(VI)) in surface soil**
213 **(mg·kg dry weight [dw]$^{-1}$)**

| | Land use | | | |
|---|---|---|---|---|
| | **Agricultural** | **Residential/ Parkland** | **Commercial** | **Industrial** |
| **Guideline**[a, b] | **0.4** | **0.4** | **1.4** | **1.4** |
| $SoQG_{HH}$ | | | | |
| ILCR $10^{-6}$ | 18 | 18 | 18 | 18 |
| ILCR $10^{-5}$ | 70 | 70 | 110 | 170 |
| $SoQG_E$[c] | 0.4 | 0.4 | 1.4 | 1.4 |

**Notes:** $SoQG_E$ = soil quality guideline for environmental health; $SoQG_{HH}$ = soil quality guideline for human health; ILCR = incremental lifetime cancer risk. Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

[a] See table 8 and 10 for more details on selection of $SoQG_{HH}$ and $SoQG_E$, including component values and check values.

[b] Data are sufficient and adequate to calculate an $SoQG_{HH}$ and a provisional $SoQG_E$. The soil quality guideline is the lower of the two and represents fully integrated guidelines.

[c] $SoQG_E$ taken from CCME (1999 update).

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215 **Table 2. Soil quality guidelines for trivalent chromium (Cr(III)) and total chromium**
216 **(Cr(T)) in surface soil (mg·kg dw$^{-1}$)**

| | Land use | | | |
|---|---|---|---|---|
| | **Agricultural** | **Residential/ Parkland** | **Commercial** | **Industrial** |
| **Guideline**[a, b] | **64** | **64** | **87** | **87** |
| $SoQG_{HH}$ (Cr(III)) | 26 000 | 26 000 | 86 000 | 96 000 |
| $SoQG_E$[c] (Cr(T)) | 64 | 64 | 87 | 87 |

**Notes:** $SoQG_E$ = soil quality guideline for environmental health; $SoQG_{HH}$ = soil quality guideline for human health. Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

[a] See tables 9 and 11 for more details on selection of $SoQG_{HH}$ and $SoQG_E$, including component values and check values.

[b] Data are sufficient and adequate to calculate an $SoQG_{HH}$ and an $SoQG_E$. The soil quality guideline is the lower of the two and they represent fully integrated guidelines.

[c] $SoQG_E$ taken from CCME (1999 update).

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# 1. INTRODUCTION

Canadian Soil Quality Guidelines are numerical concentrations or narrative statements that specify the levels of toxic substances or other parameters in soil that are recommended to maintain, improve or protect environmental quality and human health. They are developed using formal protocols to ensure nationally consistent, scientifically defensible values. The guidelines are nationally endorsed through the Canadian Council of Ministers of the Environment (CCME).

This report reviews the chemical and physical properties of chromium, its sources and emissions in Canada, its environmental fate and behaviour, and its effects on the health of humans and experimental animals. This information is used to derive guidelines for chromium to protect human receptors, according to the processes outlined in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006) for agricultural, residential and parkland, commercial, and industrial land uses. In addition, various check mechanisms that consider indirect pathways of exposure (e.g., off-site migration of contaminants via wind and water erosion), as elaborated in the Protocol (CCME 2006), are used to ensure the protection of resources and receptors not otherwise considered in the derivation of soil quality guidelines. Guidelines for the protection of ecological receptors, developed in 1997 and 1999 (CCME 1999), are included in this document for the purpose of selection of generic Soil Quality Guidelines (SoQG) that are considered protective of both human and ecological receptors. Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

Chromium occurs in the environment in two forms: trivalent chromium, Cr(III), and hexavalent chromium, Cr(VI). In soils, it occurs primarily as Cr(III), unless the soils have been polluted with Cr(VI)-contaminated wastes. Since Cr(III) and Cr(VI) have different effects on human health, human-health-based soil quality guidelines should be derived for each form; however, current practices favour the analysis of Cr(T) in soil, while Cr(VI) is only measured in cases where there is a known or suspected Cr(VI) source. On this basis, human-health-based soil quality guidelines were derived separately for Cr(III) and Cr(VI). Under this approach, it may be possible to measure Cr(T) in soil and compare the results to the $SoQG_{HH}$ for Cr(III), while analytical measurement of Cr(VI) in soil would be strongly recommended for any site potentially contaminated by activities involving Cr(VI).

In such cases, the speciated results for Cr(VI) may be compared with the $SoQG_{HH}$ provided for Cr(VI) while the Cr(T) or Cr(III) results may be compared to Cr(III).

The following derived $SoQG_{HH}$ values should be considered for general guidance purposes. Every attempt was made to provide a conservative estimate that could be applied to any area in Canada, but site-specific information (such as local background concentrations) should always be considered in the application of these guidelines. Since guidelines may be applied differently in

various jurisdictions, the reader should consult appropriate authorities for the laws and regulations of the jurisdiction in which they are working for applicable implementation procedures.

# 2. BACKGROUND INFORMATION

## 2.1 Physical and Chemical Properties

Chromium has an atomic number of 24 and is the first element in Group 6 of the periodic table and is a member of the first transition series. Its relative atomic mass is 51.996 (Anderson 1981). There are four naturally occurring isotopes—$^{50}$Cr, $^{52}$Cr, $^{53}$Cr and $^{54}$Cr—with relative abundances of 4.31, 83.76, 9.55 and 2.38%, respectively (Kumral 2007; Nriagu and Kabir 1995). The longest-lived radioactive isotope is $^{51}$Cr, with a half-life of 27.7 days (Ferguson *et al.* 2011).

Chromium has nine different oxidation states, from -2 to +6, but the common oxidation states are +2, +3 and +6 (Kumral 2007).

Elemental chromium (Cr(0)) does not appear in nature (Shupack 1991); rather, chromium is most commonly found in the trivalent (Cr III) state, and can occur in ores such as chromite ($FeCr_2O_4$) (Agency for Toxic Substances and Disease Registry [ATSDR] 2012; Environment Canada [EC] and Health Canada [HC] 1994). Cr(VI) only occurs naturally in crocoite ($PbCrO_4$) (ATSDR 2012). The highest concentrations are in basic and ultrabasic igneous rocks, with much lower concentrations in granitic (siliceous) igneous rocks. For industrial use, chromium ore is converted to either the metal (Cr(0)) or the chromate (Cr(VI)) form (ATSDR 2012).

Elemental chromium (Cr(0); CAS No. 7440-47-3) is a hard, brittle and lustrous steel-grey metal. Due to a number of attributes, including durability (resistant to corrosion, wear, temperature and decay), strength, hardness, hygiene and colour, elemental chromium is commonly used as an alloy in stainless steel and chrome-plated objects (Kumral 2007; Nriagu and Kabir 1995).

Only the two most common states of chromium—trivalent, Cr(III), and hexavalent, Cr(VI)—are discussed in detail in this report. These two forms show different physico-chemical properties which affect their biochemical reactivity. Cr(VI) compounds are generally more soluble, mobile and bioavailable than Cr(III) species (Kumral 2007). Under ambient conditions, the other oxidation states are not stable enough to be of environmental or toxicological importance.

In soil, redox reactions can interconvert Cr(III) and Cr(VI). At very low pH, or in the presence of organic matter or ferrous iron, Cr(VI) is likely to be reduced to Cr(III); however, at 3 to 10.1 pH, Cr(VI) is more stable and, if manganese is present in soil, it is possible for Cr(III) to be oxidized to Cr(VI) (Rai *et al*. 1989; Shupack 1991).

Cr(III) is more likely to sorb to clay and organic matter in soils than is Cr(VI), and sorption of Cr(III) is increased by a higher pH, whereas Cr(VI) sorption to soil was not found to be related to

pH. Cr(VI) leaches out of soil or disposed material in landfills more easily than does Cr(III) (Choppala *et al.* 2010; 2012). For this reason, Cr(VI) can also be more readily taken up into plant matter (Kumral 2007). Both oxidation states of chromium may exist at a single site without them existing in consistent ratios from one medium to the next.

Chemical and physical properties of Cr(0) and compounds of Cr(III) and Cr(VI) are presented in Table 3 and Table 4, respectively.

### *2.1.1 Chromium Speciation*

#### 2.1.1.1 Trivalent Chromium

The trivalent form of chromium (Cr(III)) is generally considered the most thermodynamically stable oxidation state under ambient redox conditions. Considerable energy is required to convert Cr(III) to a lower or higher oxidation state (Shupack 1991; ATSDR 2012).

The Cr(III) ion has a strong tendency to form stable complexes with the oxygen, nitrogen or sulphur in organic ligands (Taylor *et al*. 1979; Saleh *et al*. 1989; Shupack 1991). It also sorbs readily to clays (Choppala *et al*. 2010; 2012). In water, the ionic form of Cr(III), $Cr^{3+}$, predominates (HC 2016). Above pH 4, Cr(III) in water forms hydroxide complexes: $CrOH^{2+}$, $CrOH_2^-$, $CrOH_3$ and $CrOH_4^-$ (HC 2016; Rai *et al*. 1989). The dominant hydroxo species in water at pH values ranging from 3.8 to 6.3 is $CrOH^{2+}$, $CrOH_3^0$ dominates at pH 6.3 to 11.5, while, at pH >11.5, Cr(III) is transformed into the soluble tetrahydroxo complex, $CrOH_4^-$ (Rai *et al*. 1989). The hydroxides, oxides and phosphates tend to be insoluble (Nriagu *et al*. 1993). The formation of stable complexes between Cr(III) and amino acids, peptides and other ligands can prevent the precipitation of Cr(III) at pH values where it would otherwise precipitate (United States Environmental Protection Agency [US EPA] 1990).

Different chemical and physical processes, such as hydrolysis, complexation, redox reactions and adsorption, influence the presence, concentration and forms of Cr(III) in the environment. Rai *et al*. (1989) indicate that, in the absence of complexing agents, Cr(III) exists as hexa-aquachromium (3+) and its hydrolysis products. In natural waters, Cr(III) is present as hydrolyzed $CrH_2O_4OH_2^+$ and its complexes, and even adsorbed on colloidal matter (Kimbrough *et al.* 1999).

#### 2.1.1.2 Hexavalent Chromium

Anthropogenic pollution is the principal source of Cr(VI) in the environment. Cr(VI) is produced during the reduction of chromite ore to obtain chromium metal (World Health Organization [WHO] 1988; Shupack 1991). Cr(VI) rarely occurs naturally, as it can be reduced to other oxidation states in the presence of organic matter under many environmental conditions (US EPA 1984c; Bartlett and James 1988; Hammond 2002).

Cr(VI) (also known as chromate) is a strong oxidizing agent, and therefore is not stable in the environment unless redox potential is high (Rai *et al*. 1989). It forms different tetrahedral oxo species—$CrO_4^{2-}$, $HCrO_4^-$, or $Cr_2O_7^{2-}$—depending on the pH of the medium and the Cr(VI) concentration (Kumral 2007).

At pH >1, deprotonated forms of Cr(VI) are seen. Between pH of 1 and 6, $HCrO_4^-$ is the predominant form and only $CrO_4^{2-}$ ions exist in solution throughout the concentration range at pH >7 (Cotton and Wilkinson 1980; Greenwood and Earnshaw 1984).

In solution, Cr(VI) exists as an anion and thus is quite mobile in the environment. The dissolved Cr(VI) species are hydrochromate (HCrO), dichromate ($Cr_2O_4^-$) and chromate ($HCrO_4^-$) (Saleh *et al*. 1989); however, Cr(VI) oxyanions are readily reduced to trivalent forms by electron donors such as organic matter or reduced inorganic species, which are ubiquitous in soil, water and atmospheric systems (Stollenwerk and Grove 1985).

**Table 3. Identity and some physical and chemical properties of chromium and selected trivalent chromium (Cr(III)) compounds**

| Property | Chromium | Chromic acetate, hexahydrate | Chromic chloride | Chromic chloride hexahydrate | Chromic oxide | Chromic phosphate | Chromic phosphate hexahydrate | Chromic sulphate | Chromic sulphate octadeca-hydrate |
|---|---|---|---|---|---|---|---|---|---|
| Empirical formula | Cr | $Cr(C_2H_3O_2)_3 \cdot 6H_2O$ | $CrCl_3$ | $CrCl_3 \cdot 6H_2O$ | $Cr_2O_3$ | $CrPO_4$ | $CrPO_4 \cdot 6H_2O$ | $Cr_2(SO_4)_3$ | $Cr_2(SO_4)_3 \cdot 18H_2O$ |
| Molecular weight (g/mol) | 51.996 | 337.220 | 158.355 | 266.446 | 151.990 | 146.967 | 255.059 | 392.180 | 716.2 |
| CAS # | 7440-47-3 | 1066-30-4 | 10025-73-7 | 10060-12-5 | 1308-38-9 | 7789-04-0 | 84359-31-9 | 10101-53-8 | 13520-66-6 |
| Physical state | steel grey, lustrous and hard | blue needles | lustrous, red-violet, crystals | dark green, monoclinic crystals | light to dark-green fine hexagonal crystals | blue orthorhombic crystals | violet crystals | red powder | violet crystals |
| Melting point (°C) | 1907 | NR | 827 | 83 | 2432 | >1800 | decomposes at >500 | decomposes at >700 | decomposes at 115 |
| Boiling point (°C) | 2671 | NR | decomposes at 1300 | NR | ≈3000 | NR | NR | NR | NR |
| Density ($g/cm^3$) at ~21°C | 7.15 | NR | 2.76 | 1.76 | 5.22 | 4.6 | 2.121 | 3.1 | 1.7 |
| Solubility in water | $<10^{-8}$ mol/L | soluble | slightly soluble | 58.5 g/100 mL at 25 °C | insoluble | insoluble | Insoluble | soluble | soluble, reactive in water |
| Other solubilities | reacts with dilute acid | | | soluble in ethanol; slightly soluble in acetone | insoluble in ethanol; slightly soluble in acid and alkali | insoluble in acid and aqua regia | soluble in acid and alkali | very soluble in acid | |

Key: NR = not reported

Adapted from Hazardous Substances Data Bank (HSDB) (1987), US EPA (1990), Katz and Salem (1994) and Rumble (2018).

337 **Table 4. Identity and some physical and chemical properties of selected hexavalent chromium (Cr(VI)) compounds**

| Property | Ammonium chromate | Ammonium dichromate | Barium chromate | Chromium (VI) trioxide | Lead chromate | Mercury (II) chromate | Potassium chromate | Potassium dichromate | Sodium chromate | Sodium dichromate |
|---|---|---|---|---|---|---|---|---|---|---|
| Empirical formula | $(NH_4)_2CrO_4$ | $(NH_4)_2Cr_2O_7$ | $BaCrO_4$ | $CrO_3$ | $PbCrO_4$ | $HgCrO_4$ | $K_2CrO_4$ | $K_2Cr_2O_7$ | $Na_2CrO_4$ | $Na_2Cr_2O_7$ |
| Molecular weight (g/mol) | 152.71 | 252.065 | 253.21 | 99.994 | 323.2 | 316.58 | 194.191 | 294.185 | 161.974 | 261.968 |
| CAS # | 7788-98-7 | 7789-09-5 | 10294-40-3 | 1333-82-0 | 7758-97-6 | 13444-75-2 | 7789-00-6 | 7778-50-9 | 7775-11-3 | 10588-01-9 |
| Physical state | yellow crystals | bright orange-red monoclinic crystals | yellow orthorhombic crystals | red orthorhombic crystal | yellow-orange monoclinic crystal | red, monoclinic crystals | yellow orthorhombic crystals | orange-red triclinic crystals | yellow, deliquescent orthorhombic crystals | reddish, somewhat deliquescent crystals |
| Melting point (°C) | decomposes at 185 | decomposes at 180 | decomposes at 210 | 197 | 844 | NR | 971 | 398 | 792 | 357 |
| Boiling point (°C) | decomposes | decomposes | decomposes | decomposes at ≈250°C | decomposes | NR | NR | decomposes at 500°C | NR | decomposes at 400°C |
| Density ($g/cm^3$) at ~21°C | 1.90 | 1.155 | 4.50 | 2.70 | 6.12 | 6.06 | 2.732 | 2.678 | 2.72 | NR |
| Solubility in water | 40.5 g/100 mL at 30 °C | 30.8 g/100 mL at 15 °C | $3.4\times10^{-4}$ g/100 mL at 160 °C | 67.45 g/100 mL at 100 °C | $5.8\times10^{-4}$ g/100 mL at 25°C | slightly soluble | 62.9 g/100 mL at 20°C | 4.9 g/100 mL at 0°C 102 g/100 mL at 100°C | 87.3 g/100 mL at 30°C | very soluble |
| Other solubilities | soluble in acetone and methanol; insoluble in ethanol | | reacts with acid | | soluble in alkali and dilute acid | | | | soluble in ethanol | |

338 Key: NR = not reported

339 Adapted from HSDB (1987), US EPA (1990), Katz and Salem (1994) and Rumble (2018).

## 2.2 Geochemical Occurrence

Chromium is typically present only in trace amounts in rock (i.e., <3400 mg/kg) with an average concentration of 125 mg/kg in the continental crust (Adriano 2001); it ranks 17th in elemental abundance (Hem [1970], as cited in Izbicki *et al.* [2008]). More than 40 chromium-containing minerals have been identified. Trace amounts of chromium oxides, ubiquitous in the Earth's crust (Haluschak *et al*. 1998), account for the colouring of many minerals, such as rubies and emeralds (Shupack 1991).

Chromium is found in ultrabasic and basic rock. Among the minerals containing chromium as a major constituent, chromite ($FeCr_2O_4$) is the most common in crustal rock, found primarily in ultrabasic rock, such as serpentines (Nriagu and Kabir 1995). Natural chromium levels in igneous rock vary from 13 mg/kg (granitic rock) to 1800 mg/kg (ultramafic/basic and serpentine protolith rock) (Oze *et al*. 2004). Chromium retention increases with decreasing grain size, and thus chromium is usually found in higher concentrations in shales than in sandstones (EC 1996).

In central Saskatchewan, average concentrations of Cr(T) in cretaceous sedimentary rocks were 20 mg/kg (Bearpaw), 22 mg/kg (Judith River), 25 mg/kg (White Specks), 11 mg/kg (Viking and Joli Fou), 10 mg/kg (Lower Colorado and Mannville) and 9 mg/kg (Big River) (Dunn 1990).

The calculated background chromium concentrations in Québec were 85 mg/kg (St. Lawrence Lowlands Sector), 75 mg/kg (Appalachians Sector), 45 mg/kg (Grenville Sector), 85 mg/kg (Superior and Rae Sector), and 80 mg/kg (Labrador Trough Sector) (Ministère du Développement Durable, de l'Environnement et des Parcs [MDDEP] 1998).

## 2.3 Analytical Methods

During sampling and extraction, Cr(III) and Cr(VI) may interconvert depending on the procedure, which chromium compounds are present, and the chemistry of the matrix. Special handling of samples in the laboratory is necessary; stainless steel equipment (8 to 20% chromium) should not be used for biological sample processing due to the risk of sample contamination (WHO 2006) and attention must be paid to sample pre-treatment to ensure that the original chromium species do not change during manipulation due to instability. The accuracy of older environmental data (Sturgeon *et al.* 1987), particularly food (Kumpulainen 1992) and body tissues and fluids data (Fishbein 1984; Anderson 1987), is questionable due to relatively high detection limits.

In summary, the fundamental redox chemistry of chromium that allows for the interconversion of Cr(III) and Cr(VI) in the environment is also responsible for chromium reduction during sampling and extraction with all the general analytical procedures commonly used for metals.

### 2.3.1 Total Chromium

CCME (2016a; b; c; d) provides guidance on recommended sampling and analytical methods for the environmentally available portion of total metals. These methods are the most pertinent for the evaluation of environmental exposures to metals.

By far the most common approach to determine Cr(T) is the use of hot acids to destroy chemical and physical bonds between chromium and the sample matrix and convert chromium into water-soluble forms, leaving the bulk of non-target elements as solids that can be filtered out or oxidized to gases. Once solubilized, chromium can be analyzed with a variety of instruments (refer to CCME 2016d).

CCME (2016d) recommends the following analytical methods for the determination of Cr(T) in soil and sediment samples:

- Inductively coupled plasma-atomic emission spectrometry (ICP-AES) (US EPA Method 6010C) (US EPA 2007a)
- Inductively coupled plasma-mass spectrometry (ICP-MS) (US EPA Method 6020A) (US EPA 1998a)
- Flame atomic absorption spectrophotometry (FAAS) (US EPA Method 7000B) (US EPA 2007b)
- Graphite furnace atomic absorption spectrophotometry (GFAA) (US EPA Method 7010) (US EPA 1998b)
- Several methods from other international and Canadian agencies.

CCME (2016d) recommends the use of the following methods for the determination of Cr(T) in water and wastewater samples:

- Axially viewed inductively coupled plasma-atomic emission spectrometry (Method 200.5, revision 4.2) (US EPA 1994a)
- Inductively coupled plasma-atomic emission spectrometry (Method 200.7, revision 4.4) (US EPA 1994a)
- Inductively coupled plasma-mass spectrometry (Method 200.8, revision 5.4) (US EPA 1994a)
- Stabilized temperature graphic furnace atomic absorption (Method 200.9, revision 2.2) (US EPA 1994a)
- Ultrasonic nebulization inductively coupled plasma-atomic emission spectrometry (Method 200.15, revision 1.2) (US EPA 1994a)
- Several methods from other international and Canadian agencies.

Several techniques (ICP-MS, ICP and GFAA) have historically been used by laboratories to test for Cr(T) at levels below the current HC drinking water quality guideline of 50 µg/L (HC 2014). Graphite furnace techniques are reliable to levels below 10 µg/L but are generally inadequate for

measurements in the <3 µg/L range. When properly performed, ICP-MS is accurate to 1 µg/L Cr(T) in water (Eaton *et al.* 2001).

### *2.3.2 Chromium Speciation*

#### 2.3.2.1 Trivalent chromium

Cr(III) is calculated from the difference between Cr(T) and Cr(VI). When reporting method detection levels (MDL) or lower reporting limits (LRL) for samples determined this way, normally the MDL and LRL for Cr(T) are used. However, if Cr(VI) is ≥1/3 Cr(T), the confidence of detection is reduced and the limit of detection should be increased to reflect this uncertainty. Refer to CCME (2016a; d) for guidance.

#### 2.3.2.2 Hexavalent chromium

Sample preparation (digestion) for the determination of Cr(VI) in soils and other solids differs from that of total metals. CCME (2016d) recommends the use of US EPA Method 3060A, a performance-based method using alkaline digestion (US EPA 1996a).

CCME (2016d) recommends the following analytical methods for the determination of Cr(VI) in soil and sediment samples:

- Chromium, hexavalent (colorimetric) (US EPA Method 7196A) (US EPA 1992)
- Determination of hexavalent chromium in drinking water, groundwater and industrial wastewater effluents by ion chromatography (US EPA Method 7199) (US EPA 1996b)
- Several methods from other international and Canadian agencies.

CCME (2016d) recommends the use of the following methods for the determination of Cr(VI) in water and wastewater samples:

- Determination of dissolved hexavalent chromium in drinking water, groundwater and industrial wastewater effluents by ion chromatography (US EPA Method 218.6, revision 3.3) (US EPA 1994b)
- Determination of hexavalent chromium in drinking water by ion chromatography with post-column derivatization and UV-visible spectroscopic detection (US EPA Method 218.7) (US EPA 2011)
- Determination of hexavalent chromium by ion chromatography (US EPA Method 1636) (US EPA 1996*c*)
- Several methods from other international and Canadian agencies.

For soil and sediment samples, a minimum 2.5 g sample is subjected to an alkaline digestion with continuous stirring prior to analysis. The extract must be analyzed within seven days of extraction.

For the determination of dissolved Cr(VI), aqueous samples are field-filtered and preserved to achieve a 28-day holding time (US EPA 1994b; Standard Methods 2011).

Methods have also been developed to measure Cr(VI) in welding fumes (ATSDR 2012), with detection by spectrophotometry alone (Method 7600, DL=0.05 µg/sample) or by spectrophotometry coupled with chromatography (Method 7604, DL=3.5 µg/sample) (National Institute for Occupational Safety and Health [NIOSH] 1994a; b).

Cr(III) and Cr(VI) in solution may be measured simultaneously using a single probe to detect differences in diffusive gradients through thin films. The probe uses a polyacrylamide hydrogel overlying a layer of resin embedded in gel. Cr(III) species accumulate exclusively in the resin layer, while Cr(VI) species equilibrate with both hydrogel and resin layers. Detection limits of 0.008 µg/L for Cr(III) and 0.3 µg/L for Cr(VI) have been achieved (Ernstberger *et al*. 2002). A solvent extraction-atomic spectrometric technique for Cr(VI), based on chromium's reaction with diphenylcarbazide, reduced the detection limit to 0.024 µg/L (Castillo *et al*. 2002).

Chromium-contaminated water, soil leachate and soil samples can be analyzed using HPLC methods with diode array detection (HPLC-DAD). This method also differentiates between $CrO_4^-$ and $Cr_2O_4^{2-}$. The detection limits are 4 µg/L for Cr(VI) and 7 µg/L for Cr(III) (Cathum *et al*. 2002).

### *2.3.3 Analysis of Biological Materials*

For determining low levels of chromium in biological samples, the four most frequently used methods are NAA, mass spectrometry (MS), graphite spark atomic emission and GFAA (ATSDR 2012).

## 2.4 Production and Uses in Canada

The only commercial source of chromium is chromite ore. The bulk (95%) of shipping-grade chromite is concentrated in Kazakhstan and South Africa, with the remaining production located in Zimbabwe, Russia, Turkey, Albania, the Philippines, Finland, Brazil and Iran (United States Geological Survey [USGS] 2012). World chromite ore production of 14.294 Mt-gross weight (4.3 Mt-Cr) is used by the metallurgical (11.292 Mt-Cr Ore), chemical (1.858 Mt-Cr Ore) and refractory (1.144 Mt-Cr Ore) industries (Papp 1995).

Ore-grade chromite has been identified at more than 250 locations in Canada, with the ore resources estimated to be about 20 million tonnes (EC and HC 1994). The principal deposits occur in Québec, Ontario, British Columbia, Manitoba, and Newfoundland and Labrador. The ores in the first three provinces are low-grade ($Cr_2O_3$ content <25%). The Newfoundland and Labrador deposits are of medium grade ($Cr_2O_3$ content of up to 53% with a Cr/Fe ratio of 2.7:1); however, they occur in isolated locations (EC and HC 1994). Domestic ores were mined in the past when

there were limitations on imports. Canada now depends on foreign sources for all its chromium requirements. Natural Resources Canada (NRCan) reported chromium mineral imports of 60 301 kg in 2008, 50 599 kg in 2007 and 49 009 kg in 2006, as well as exports of 1921 kg in 2008, 1759 kg in 2007, and 2733 kg in 2006 (NRCan 2009).

Pure chromium metal is produced via two commercial production methods. The aluminothermic process uses powdered chromic oxide and granulated aluminium in an exothermic process to produce metallic chromium and aluminium oxide. The more popular silicothermic process uses an electric arc furnace to heat chromic oxide, silicon and lime to produce molten chromium metal and a slag rich in silicon dioxide (Shupack 1991).

The high melting point and chemical stability of chromite ore and chromium alloys provide corrosion resistance to acids and bases at high temperatures. Copper-chromium alloys are used in electrical applications that require high strength and good conductivity, while copper-nickel-chromium alloys are used in marine equipment requiring corrosion resistance (Nriagu and Kabir 1995). The automobile industry is a major user of chromium alloys in the form of stainless-steel components, catalytic converters, chrome trims and other control and decorative systems. Chromium-containing superalloys with high heat resistance are used in aircraft engines and other aerospace equipment (Nriagu and Kabir 1995).

The major chromium compounds and their uses are presented in Appendix 7. Chromium is used in the production of fungicides, drilling muds, water treatment, textiles, catalysts, synthetic rubies for lasers, chromium dioxide magnetic tapes, medicine (labelling of red blood cells), toner for copy machines, montan wax (also known as lignite wax or OP wax), vitamin K, and as a mordant in wool dyeing, photography, and the manufacture of activated carbon (Taylor *et al*. 1979; US EPA 1984a; Nriagu and Kabir 1995; Yeates *et al*. 1994; ATSDR 2012). Chromium is also used in the manufacture of refractory bricks, furnace linings, mortars, ramming mixtures for domestic iron and steel, portland cement, glass, castables, and coating materials to close pores and to join bricks in furnaces (US EPA 1984a; Nriagu and Kabir 1995; ATSDR 2012).

Another important use of chromium is wood treatment with aqueous copper chrome arsenate (CCA). Application rates range from 4 to 24 kg/m$^3$ depending upon the type of wood and the intended use. Over 100 000 tonnes of CCA are traded worldwide annually. Historically in Canada, approximately 65% of the total production of CCA was utilized for residential construction with retentions of 4 to 6.4 kg/m$^3$. Commercial products, such as poles (retentions of 6.4 to 9.6 kg/m$^3$) and marine products (retentions of 24 to 40 kg/m$^3$), are treated in lesser quantities (EC 2002). The use of CCA for wood treatment has been prohibited for residential (but not industrial) purposes since 2004 in Canada (HC 2005). Similar restrictions have been put in place in other countries, such as the United States (in 2003) and Australia (in 2006) (US EPA 2003; Australian Pesticides and Veterinary Medicines Authority 2005).

## 2.5 Sources and Concentrations in the Canadian Environment

### *2.5.1 Natural Sources*

Chromium occurs naturally in small amounts in rocks and soils as relatively inert solid phase Cr(III) and is released into the aquatic environment in limited quantities by weathering and erosion. It has been estimated that more than 70% of chromium in the environment comes from anthropogenic sources (Merian 1984).

The species of chromium present in environmental media are rarely identified. Most studies of chromium distribution in the Canadian environment have measured only Cr(T).

### *2.5.2 Anthropogenic Sources*

Chromium is used in a wide variety of manufactured products in Canada, and likely enters the aquatic environment from many industrial sources.

While there is presently no chromium mined in Canada, data on releases for 1988 indicate that nonferrous base metal smelters and refineries throughout Canada discharged liquid effluents containing approximately 2 tonnes of chromium (EC and HC 1994). Chromium smelting slag contains 2 to 12 % chromium, and the pulverization of dry slag and ore converts Cr(III) to Cr(VI) (MiningWatch Canada 2012). Environment and Climate Change Canada (ECCC)'s National Pollutant Release Inventory (NPRI) database indicates that in 2015, approximately 9.5 tonnes of Cr(T) were released into the air, 31 tonnes into land and 1.6 tonnes into Canadian waters (ECCC 2016). The Municipal/Industrial Strategy for Abatement (MISA) program estimated releases of approximately 7.7 tonnes per year of chromium into provincial waters (St. Marys River, Lake Erie and Lake Ontario) over approximately 12 months in 1989 and 1990 from Ontario iron and steel plants (Ontario Ministry of the Environment [OMOE] 1991). Treated effluents from these iron and steel mills typically contained 10 to 26 µg/L of chromium.

Most of the chromium entering municipal wastewaters annually is removed as sludge (OMOE 1988), some of which is applied to Canadian agricultural lands (CCME 2012). Samples of sewage sludge proposed for use as soil ameliorants in India found one sludge to have significant amounts of chromium (in the range of 19 to 50 mg/kg); however, it was present as relatively benign Cr(III) in all samples (Martin *et al*. 2003).

Chromium can also enter soil via the application of organic matter and fertilizers. In one study, manure was found to contain Cr(T) concentrations <20 mg/kg (Centre de recherche industrielle du Québec [CRIQ] 1994) and yields in agricultural lime were found to be <5 mg /kg; however, another study reported that cow manure contains Cr(T) concentrations of approximately 56 mg/kg (dw) (Webber and Singh 1995). Phosphate-containing fertilizers contain Cr(T) concentrations between 60 and 250 mg/kg (Hébert 1998), with reported concentrations from Canadian studies

generally below 100 mg/kg (Hébert 1998; Mermut *et al*. 1996; Webber and Singh 1995). Chromium in fertilizers is available to plant roots at a rate of 7 to 13% (as determined by the diethylenetriaminepentaacetic acid [DTPA] extractable method) (Mermut *et al*. 1996).

European household wastes typically contain between 50 and 100 µg/L chromium (EC and HC 1994). Assuming similar concentrations in the 12 million tonnes of solid waste generated each year by Canadian households, estimated annual disposal was 600 to 1200 tonnes.

Process effluents released from petroleum refineries in Ontario in 1988 and 1989 contained six-month mean Cr(T) concentrations ranging from 87 to 126 µg/L, approximately 10% of which was Cr(VI) (OMOE 1989; 1990). Based on these data, more than one tonne of chromium was discharged annually in liquid effluent from petroleum refineries into the St. Clair River and Lake Ontario.

Ferrochromium production is the most important industrial source of atmospheric chromium (US EPA 1984b), but other processes, such as ore refining, chemical and refractory processing, cement production and automobile catalytic converters and brake linings, also contribute to chromium in the atmosphere.

### *2.5.3 Ambient Air*

Since 1984 the National Air Pollution Surveillance (NAPS) Network has measured fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) ambient particles in air from across Canada. $PM_{2.5}$ has been found to be a stronger predictor of mortality than $PM_{10-2.5}$; specifically, sulphate, iron and chromium in $PM_{2.5}$ are associated with mortality (chromium having the strongest association) (Burnett *et al.* 2000). The NAPS data set most relevant to chromium exposure via inhalation is the respirable size fraction (i.e., $PM_{2.5}$) analyzed by ICP-MS (inductively coupled plasma mass spectroscopy) following acid ($HNO_3$) digestion. This data set is available for download from the NAPS website (EC 2013).

Although NAPS ICP-MS data were only available for British Columbia, Ontario, Québec and New Brunswick at the time of estimated daily intake (EDI) development, these data were preferable to using data from non-Canadian sources to the develop the concentration profile of chromium in ambient air. Data from 2003 to 2009 were used to develop the EDIs from inhalation of ambient air for the Canadian population. The chromium content of $PM_{2.5}$ ranged from $1.41 \times 10^{-5}$ to $1.58 \times 10^{-2}$ µg/m$^3$ (arithmetic mean = $5.16 \times 10^{-4}$ µg/m$^3$; SD = $5.69 \times 10^{-4}$ µg/m$^3$; n = 3054) (EC 2013).

Annual national average Cr(T) concentrations in rural and non-industrial urban areas of between $3 \times 10^{-3}$ and $4 \times 10^{-3}$ µg/m$^3$ (OMOE 2009) were estimated from NAPS $PM_{10}$ data; however, in urban centres with local sources, annual averages were between $3 \times 10^{-3}$ and $11 \times 10^{-3}$ µg/m$^3$. The range of median (50th percentile) 24-hour average concentrations were $2.6 \times 0^{-3}$ to $5 \times 10^{-3}$ µg/m$^3$.

The 95th percentile of 24-hour average concentrations can be in the range of $1.1 \times 10^{-2}$ to $3.0 \times 10^{-2}$ µg/m$^3$. No data from widespread measurements of Cr(VI) in ambient air were available (OMOE 2004).

Other studies (summarized in Appendix 1) provide concentrations in ambient air away from potential point sources. These studies were not used to calculate the estimate of typical concentrations in ambient air in Canada.

### *2.5.4 Indoor Air*

Due to technical difficulties associated with monitoring very low concentrations of metals in air samples, there are a limited number of studies reporting chromium concentrations in indoor air. Additionally, there is no published Canadian database for indoor air concentrations of chromium (Rasmussen *et al*. 2006).

Based on the two Canadian studies (Alberta Health 1998; Bell *et al*. 1994), four American studies (Adgate *et al*. 2007; Finley *et al*. 1993; Graney *et al*. 2004; Van Winkle and Scheff 2001), one Belgian study (Stranger *et al*. 2009) and one Singaporean study (Balasubramanian and Lee 2007), the arithmetic mean background concentration for total chromium in indoor air was estimated at 2 ng/m$^3$ (SD = 15 ng/m$^3$, n = 836). These data were used to develop the indoor air inhalation EDI values used in the SQG calculations. The EDI methodology is further discussed in Section 5.2.

Hexavalent chromium was measured in a Canadian pilot study (Bell and Hipfner 1997). Indoor air Cr(VI) concentrations ranged from 0.07 to 0.62 ng/m$^3$, with a geometric mean of 0.2 ng/m$^3$. There was a significant difference between outdoor and indoor air concentrations. Lower indoor air concentrations could be explained by the air exchange rate for a typical home (≈1× every three hours) and by higher indoor temperatures (Bell and Hipfner 1997). These study results contributed to the analysis carried out to determine the fraction of Cr(VI) (as compared to total chromium) in indoor air (HC 2017b).

Tobacco smoke is one of the greatest sources of indoor inhalable particles. The elements associated with tobacco smoke (S, K, Cr, Ni, Zn, As, Cd and Pb) were predominantly present in the fine fraction, which has a strong influence on health (Slezakova *et al.* 2009). In indoor environments influenced by tobacco smoke, Cr concentrations were increased by 15 to 680% and 20 to 250% in $PM_{10}$ and $PM_{2.5}$, respectively.

A summary of background concentrations for indoor air is provided in Appendix 1.

### 2.5.5 Indoor Dust

Two studies were identified that provide measured values for various Canadian cities (Rasmussen *et al*. 2001; 2013). As the larger Canadian data set (Rasmussen *et al.* 2013) was published subsequent to the work carried out to determine the EDI, data from the United States and other developed countries were included in the EDI calculations.

Based on one Canadian study (Rasmussen *et al*. 2001), the National Human Exposure Assessment Survey (NHEXAS) database, and additional literature sources—four American studies (Freeman *et al*. 1997; 2000; Lioy *et al*. 1992; Stern *et al*. 1992), two Australian studies (Chattopadhyay *et al*. 2003; Davis and Gulson 2005), one Polish study (Lisiewicz *et al*. 2000), one Bahraini study (Madany *et al*. 1994), one Turkish study (Turkoglu *et al*. 2004), one Omani study (Yaghi and Abdul-Wahab 2004) and one German study (Seifert *et al*. 2000)—the estimated background concentration in Canadian indoor dust was 81.1 mg/kg (arithmetic mean, SD = 136.2 mg/kg, n = 5,740). This value was used to calculate the EDI. Note that loading values (i.e., $mg/m^2$) were not included in the calculation of the background concentration of chromium in indoor dust (HC 2012a). Rasmussen *et al*. (2013) provide a higher estimate of average Canadian indoor dust concentrations (117 mg/kg). This value falls within the range of values used to calculate the EDI; however, these data were not used to calculate the EDI, as they were not available at that time.

Fan *et al.* (2009) and Lioy (2010) suggested that the Cr(VI) in house dust may be partially attributed to wooden furniture and building materials and to tobacco smoke.

A summary of Canadian studies in which chromium was measured in indoor dust is provided in Appendix 1.

### 2.5.6 Soil

Chromium is a naturally occurring element with typical soil concentrations of 80 to 200 mg/kg (Nriagu and Kabir 1995). McKeague and Wolynetz (1980) indicate that Canadian soils have a range of 10 to 100 mg/kg chromium based on 173 samples from across Canada.

Data from geological surveys conducted by both the Geological Survey of Canada (GSC) and the New Brunswick Department of Natural Resources (NBDNR) were used to develop the concentration distribution of chromium in Canadian soil (NRCan 2010a; b). These data are representative of <63µm grain size till samples, not the surface soil that is most likely to impact public health. Samples from Newfoundland and Labrador, New Brunswick, Québec, Nunavut, the Northwest Territories, Manitoba, Saskatchewan, Alberta and British Columbia were analyzed by AAS/ICP-AES following *aqua-regia* digestion (partial digestion by HCl and $HNO_3$). OMOE (1993) Ontario Typical Range (OTR) values for concentrations of various inorganics and organics in soil were not used to develop the concentration profile for chromium in soil.

Based on these data, the Cr(T) background soil concentration (BSC) was estimated at 42 mg/kg (arithmetic mean, SD = 45.5 mg/kg, n = 7398). Based on the estimated fractionation data described in Section 3.6.4, the BSC for Cr(VI) is 0.84 mg/kg. These data were used to develop the soil exposure EDI values used in the SQG calculation. The EDI methodology is further discussed in Section 5.2.

Dodd *et al.* (2017) reported Cr(T) concentrations in soil collected from reference locations across Canada. For soils in the top 5 cm (i.e., the layer most relevant to humans and many animals) a mean Cr(T) concentration of 15 mg/kg was reported (95% UCLM = 17 mg/kg; n = 532). On the other hand, the Cr(T) concentrations were greater, with a mean of 28 mg/kg (95% UCLM = 32 mg/kg; n = 532) in C-horizon soils (i.e., unconsolidated material underlying the solum or A and B horizons). Cr(T) concentrations varied across Canada with the greatest concentrations reported in the C-horizon soils from Newfoundland and Labrador, New Brunswick and Québec. Dodd *et al.* (2017) have suggested that higher C-horizon concentrations in these provinces may be reflective of weathering and other natural soil-forming processes. A summary of available soil concentrations can be found in Appendix 1.

### *2.5.7 Surface Water*

Natural background concentrations of chromium in surface water bodies and groundwater aquifers are a function of regional geology, mineral weathering processes, sediment loading rates and precipitation patterns. Average Cr(T) concentrations (including dissolved and particulate phase Cr(III) and Cr(VI)) in non-affected surface and marine waters are generally <1.0 µg/L (HC 2016). Concentrations in seawater are generally lower than in lakes and rivers, with concentrations ranging from 0.04 to 0.7 µg/L (Slooff *et al.* 1990; WHO 2005).

Dissolved Cr(T) is the parameter most often analyzed in surface and groundwater. It is generally assumed that Cr(III) is not likely to be present in waters of ≥pH 5 because of the low solubility of the hydrated oxide (HC 1986).

The available information is insufficient to permit a complete inventory of chromium loadings to Canadian surface waters; however, chromium concentrations are usually lower than 10 µg/L. An average concentration of chromium in Canadian surface waters was not determined for the purposes of setting soil quality guidelines for human health. Surface water used as a source for drinking water is addressed in the drinking water portion of this section.

A summary of available background concentrations in surface water is provided in Appendix 1.

### 2.5.8 Groundwater

Groundwater data are available from some Canadian provinces (Appendix 1). Based on the summary of Provincial Groundwater Monitoring Information System data used to develop the Ontario Background Site Condition Standards for groundwater, chromium concentrations in groundwater ranged from 0.5 ug/L to 106 ug/L with a 97.5$^{th}$ percentile of 11.4 (OMOE 2011).

A summary of select background concentrations in groundwater from Canada and the US is provided in Appendix 1.

### 2.5.9 Drinking Water

Databases from three Canadian provinces were consulted to obtain data on background concentrations in Canadian drinking water. Estimated mean chromium concentrations in drinking water were 1.75 µg/L (SD = 2.55 µg/L, n = 3800, range = 0.25–46.2 µg/L) for Ontario, based on 1998–2007 data (OMOE 2010), 1.5 µg/L (SD = 5.1 µg/L, n = 2504, range = 0.015–210 µg/L) for Saskatchewan, based on 2000 to 2009 data (Government of Saskatchewan 2008), and 1.4 µg/L (SD = 3.1 µg/L, n = 8329, range = 0.5–240 µg/L) for Newfoundland and Labrador, based on 2000 to 2009 data (GNL 2009).

Based on the above data, a background Canadian drinking water chromium concentration of 1.49 µg/L (arithmetic mean, SD = 3.4 µg/L, n = 14 633) was estimated. These data were used to develop the drinking water EDI values (Appendix 5) used in the SQG calculation. Data from other provinces were not available to include in estimating the national background value. The EDI methodology is further discussed in Section 5.2. More recent data are available on background Canadian drinking water concentrations. Considering that the newer data indicate similar concentrations, the EDI was not updated.

A summary of chromium concentrations in drinking water is provided in Appendix 1.

### 2.5.10 Sediments

Lakes that receive significant industrial and municipal effluents often show high chromium accumulation in their sediments. An approximate fourfold increase in the average chromium concentration in sediments in Lake Simcoe, Ontario, is estimated to have occurred since about 1850, which can only be attributed to the heavy urban and industrial development in its watershed (Johnson and Nicholls 1988).

Elevated concentrations, two- to fourfold above local background levels, have also historically been reported in sediments from Belledune and Dalhousie Harbours in New Brunswick, the Saguenay Fjord and the St. Lawrence River in Québec, the Detroit River and Lake Ontario off the

Niagara River in Ontario, and the Fraser River drainage basin in British Columbia (EC and HC 1994).

Freshwater sediments in many parts of Canada are contaminated with chromium from industrial sources. The most severely affected sites in Ontario include the St. Marys River system, with a concentration of 31 000 mg/kg in Tannery Bay (OMOE and MDNR 1992), and 5120 mg/kg in the Welland River downstream from a steel manufacturing plant (Dickman *et al*. 1990).

A summary of available background concentrations used in previous guidelines for sediments is provided in Appendix 1.

### *2.5.11 Precipitation*

A summary of available precipitation data used in previous guidelines for sediments is provided in Appendix 1.

### *2.5.12 Biota Used as Human Food*

A summary of available background concentrations in biota used as human food is provided in Appendix 1. Mean Cr(T) concentrations in moose muscle (0.256 µg/g dw), liver (0.52 µg/g dw) and kidney (0.22 µg/g dw from 1994 to 2001) were reported from the Yukon Territory (Gamberg *et al*. 2005). Studies in ungulates from 2002 to 2003 in the same region (Gamberg 2004) reported mean Cr(T) concentrations ranging from 0.90 to 2.81 µg/g dw (caribou kidneys) and means of 0.80 µg/g dw (elk kidney), 0.79 µg/g dw in 2002 and 0.93 µg/g dw in 2003 (moose kidney), and 0.87 µg/g dw (mule deer kidney). In fish from the northeastern United States, Yeardley *et al*. (1998) reported average Cr(T) concentrations of 0.19 µg/g dw, while Ramelow *et al*. (1989) reported bivalve concentrations ranging from <0.1 to 6.8 µg/g dw. Chromium concentrations in biota used as food are included in the dataset used to generate the EDI, as discussed in Section 5.2.

### *2.5.13 Commercial Food*

Health Canada's Food Directorate has measured concentrations of various metals and corresponding dietary intakes for Canadians as part of Health Canada's Total Diet Study; however, as chromium was not included in the TDS, data on concentrations of chromium in food were obtained from other authoritative sources.

The data from the NHEXAS were considered to provide the best estimate of food concentrations and these data were used exclusively to derive the food EDIs for chromium. The NHEXAS raw data was downloaded from the Human Exposure Database System (US EPA 2009).

The EDIs of chromium from food used in the SoQG calculation are shown in Appendix 5. The EDI methodology is further discussed in Section 3.6.7 and 5.2.

A summary of estimated chromium intake via food ingestion for additional sources (including international data) used in previous guidelines is included in Appendix 1.

More recent Canadian data on chromium in food (2012–2013), provided through annual residue monitoring by the Canadian Food Inspection Agency (CFIA 2013), was found to be consistent with the EDI estimates based on NHEXAS. These data are included as well in Appendix 1.

### *2.5.14 Human Breast Milk*

No published Canadian studies on chromium in human breast milk were identified. Based on two American studies (Casey and Hambidge 1984; Casey *et al*. 1985), one Emirati study (Abdulrazzaq *et al*. 2008) and one Japanese study (Yoshida *et al*. 2008), the chromium concentration in human breast milk was estimated at 0.59 µg/L (arithmetic mean, SD = 1.1 µg/L, n = 648). These data were used to develop the chromium EDI values used in the SoQG calculation. The EDI methodology is further discussed in Section 5.2.

### *2.5.15 Consumer Products*

Chromated copper arsenate (CCA; $CrO_3$•$CuO$•$As_2O_5$) is a chemical mixture used as a pesticide to protect wood against decay-causing organisms. The three common waterborne formulations of CCA, designated as CCA types A, B and C, have varying proportions of the active ingredients (Chou *et al.* 2007). The composition of these formulations, as weight percent (wt%) on an oxide basis, is 35.3 to 65.5 % hexavalent chromium trioxide ($CrO_3$), 18.1 to 19.6 % copper oxide (CuO) and 16.4 to 45.1% arsenic pentoxide ($As_2O_5$) (Chou *et al*. 2007). The preservatives are supplied as pastes or water-based concentrates that are diluted to between 1 and 10% w/w total salts (Cocker *et al.* 2006). Between 4 and 24 kg/m$^3$ is delivered to the wood, dependent upon the type of wood and its intended use (European Chemicals Bureau 2005). Higher application rates are used in commercial products such as poles (6.4 to 9.6 kg/m$^3$) and marine products (24 to 40 kg/m$^3$) (EC 2002).

Chromium-based alloys, i.e., cobalt-chromium-molybdenum, are used in the fabrication of knee, ankle and hip replacements (Pierce and Goodkind 1989) and higher-than-average chromium levels in body fluids have been reported following implantation of such metal prostheses (Coleman *et al*. 1973; Harding *et al*. 2002; Iavicoli *et al*. 2006). Three studies assessing 313 total hip replacement patients showed 4.4 to 100 times higher chromium concentrations in blood or serum than in the control group (Lhotka *et al.* 2003; Milosev *et al*. 2005; Dahlstrand *et al*. 2009). Most chromium was associated with serum or plasma, while a minor amount was associated with red blood cells

(Walter *et al*. 2008). Nickel-chromium alloy, used in dental restorations and considered to be inert, has been reported to ionize over time (Pierce and Goodkind 1989; Wolfaardt and Peters 1992).

Chromium is also used in costume jewellery as a coating over other metals.

Frank *et al*. (1987) reported chromium mean concentrations of 1.11 to 2.28 µg/g (dw) in cured Ontario tobacco leaves, which are the main constituent (90%) of Canadian domestic cigarette brands (Rickert and Kaiserman 1994). An average 1.47 µg Cr/g tobacco was reported by Rickert (1992) for Canadian cigarettes purchased in 1988. Cigarette tobacco grown in the US contains ≤6.3 µg Cr/g (ATSDR 2012). Assuming 10% of the chromium in cigarettes is transferred to mainstream smoke and that one cigarette contains 1 g of tobacco (Rickert 1992), the smoking of one cigarette would result in 0.15 µg of chromium in the mainstream smoke (0.15 µg/smoked cigarette). This estimate is within the reported range of 0.0002 to 0.5 µg/smoked cigarette measured in mainstream smoke (n = 23 types of cigarettes). Data from particulate and gaseous phases suggest that chromium may be primarily present in the particulate phase. The highest concentrations (0.3 to 0.5 µg/cigarette) were found in smoke from a non-filtered Soviet cigarette (Smith *et al*. 1997). According to Sógor *et al.* (1998), 15% of Cr(III) converts to Cr(VI) at cigarette burning temperature and about 0.8 to 1.2% of the original chromium content of the cigarette is present as Cr(VI) in the smoke.

No Canadian data were found regarding the consumption of nutritional supplements and vitamins. Multivitamin and multimineral supplements sold in Canada generally contain 50 µg chromium or less. Chromium-only supplements such as chromium picolinate usually contain 200 µg chromium per tablet. No data were located regarding dietary supplements used by children.

Chromium has also been reported in a variety of cosmetics such as lipsticks, eye shadows and skin creams at concentrations ranging from 0.52 to 15.3 mg/kg (Sneyers *et al*. 2009). In Asia, Sneyers *et al.* (2009) reported undetectable concentrations in soaps, while bronzing powder had concentrations ranging from <0.15 to 46.1 mg/kg. Cosmetics samples from the Egyptian market had average concentrations of 2151 mg/kg (El-Shazly *et al.* 2004).

North American and European household cleaning products, such as detergents, have been reported to contain concentrations up to 10 µg/g. Concentrations ranged from not detectable to 7.8 mg/L in dishwashing liquids, 0.04 to 10 mg/L in cleaners, 0.1 to 0.7 mg/L in bleaches and 0.1 to 0.3 mg/L in textile softeners (Nava *et al*. 1987; Basketter *et al*. 1993).



## 3. ENVIRONMENTAL FATE AND BEHAVIOUR

Once released into the environment, all five Cr(VI) compounds behave similarly. They give rise to the same ions in solution: $CrO^{2}_{4}{}^{-}$, $HCrO^{-}_{4}$ and $Cr_2O^{7}_{4}{}^{-}$. The first of these is the main species at

higher pH (>7), while the other two will be present at lower pH (the dichromate ion only at concentrations >0.4 g/L chromium).

The difference between the observed toxicity of Cr(VI) and Cr(III) species can largely be attributed to differences in bioavailability. In the environment, Cr(III) tends to be associated with relatively inert solid phases whereas Cr(VI) tends to form quite soluble compounds and does not readily adsorb onto particulate matter (EC and HC 1994). Cr(III) can therefore accumulate and persist in sediments and soils, but its availability for uptake by biota may be limited. However, labile forms of Cr(III) may be oxidized photochemically to Cr(VI) in aerobic surface waters. Cr(VI), in contrast, can persist in bioavailable form in aerobic surface waters and soil pore waters (EC and HC 1994), although it tends to be reduced to the less mobile form of Cr(III) under anaerobic conditions.

Both abiotic and biotic redox reactions govern the degradation and transformation reactions of chromium compounds in the environment. These processes are summarized in the following sections. However, since the end-products of the biotic and abiotic processes are essentially the same, it is difficult to unambiguously separate the two processes.

Cr(VI) can be reduced to Cr(III) under anaerobic conditions including reactions with iron (II), sulphides, organic matter and anaerobic microorganisms (European Chemicals Bureau 2005). Reduction of Cr(VI) is expected to occur most rapidly in acidic soils with high iron, sulphide or organic carbon contents. Under such conditions, Cr(VI) may be completely reduced to Cr(III) within a few hours.

Under aerobic conditions and at a pH of around 7 or 8 and above, Cr(VI) appears to be more stable, particularly when low concentrations of reductants such as iron (II) are present (Adriano 2001).

Elemental chromium is not biodegradable and consequently is persistent in the environment (Bartlett 1991; ATSDR 2012). Nearly all of the chromium in soils (excluding those contaminated with Cr(VI)) (Bartlett and James 1988), sediments (excluding those immediately below the interface with overlying aerobic waters) (Nriagu *et al*. 1993) and biological tissues (Anderson 1981; Nieboer and Jusys 1988) is likely to be present as Cr(III).

Results from studies in Canada and elsewhere indicate that Cr(VI) is the predominant form of dissolved chromium in surface waters. At normal drinking water pH (~7), Cr(III) is generally insoluble (Costa and Klein 2006). Elevated dissolved Cr(III) concentration could nevertheless be present in some deep, anoxic waters and waters receiving waste containing Cr(III).

Chromium oxidation and reduction processes must be considered in risk assessment. In addition to standard modifying parameters (such as pH, organic matter and cation exchange capacity), the oxidation (valence) state of the chromium species determines its mobility, bioavailability, uptake kinetics and toxicity, and hence determines the overall exposure risk.

## 3.1 Atmosphere

Due to the extremely high boiling point of chromium (2676 °C), gaseous chromium is rarely encountered. The atmospheric transformation and transport of chromium largely occurs in the liquid and solid phases (i.e., droplets and particles) or, more generally, aerosols (WHO 1988; Davidson and Wu 1989; Nriagu 1990; Seigneur and Constantinou 1995).

Little is known about the atmospheric persistence of Cr(VI). It has been suggested that Cr(VI) reacts in the atmosphere with available organic matter; however, there is no information available on the atmospheric reactions of Cr(VI) or Cr(III). Chromium is removed from the atmosphere by physical deposition processes. Measurements show that most chromium deposition occurs through wet deposition (CARB 1985).

The chemical form of chromium in air depends on the source of emissions. Naturally occurring gaseous forms are rare (ATSDR 2012). Chromium released from natural sources is probably in the trivalent form, but both Cr(III) and Cr(VI) can be present in anthropogenic emissions. Cr(III) oxides are expected near fossil fuel combustion and ore processing plants, while Cr(VI) species are generally found near chromate manufacturing and processing plants (EC and HC 1994). However, following release into the atmosphere, Cr(VI) reacts with dust particles and is eventually reduced to Cr(III) (US EPA 1990); it may be reduced to Cr(III) at a significant rate by vanadium, Fe(II) ion, bisulphite and As(III) ion. Conversely, Cr(III), if present as a salt other than Cr(III) oxide, may be oxidized to Cr(VI) in the presence of at least 1% manganese oxide (US EPA 1990). The estimated half-life for atmospheric reduction of Cr(VI) to Cr(III) is reported to range from 16 hours to approximately five days (Kimbrough *et al*. 1999). The median diameter of chromium-containing particulates in ambient air ranges from 1.5 to 1.9 μm (US EPA 1984a), with a deposition velocity of ≈0.5 cm/second (Schroeder *et al*. 1987).

Deposition is the principal mechanism by which chromium is removed from the atmosphere and distributed to terrestrial and aquatic ecosystems. Both Cr(III) and Cr(VI) are removed from the air by precipitation (wet deposition) and atmospheric fallout (dry deposition). The estimated atmospheric residence time for chromium is less than 14 days (Nriagu *et al*. 1988), while ATSDR (2012) indicates that particulate chromium is expected to be removed from the atmosphere in <10 days, which is similar to the residence time of particles with equivalent mass and median diameters. Smaller particles (<10 μm in diameter: $PM_{10}$) may remain airborne for longer periods of time and be transported a considerable distance from the source (US EPA 1984a; b; 1990). Based on a troposphere-to-stratosphere turnover time of 30 years, atmospheric particles with a residence time of <10 days are not expected to transport from the troposphere to the stratosphere (ATSDR 2012). Generally, the smaller the particles, the farther they are transported. Mean annual deposition rates for chromium in remote, rural and urban areas are <0.2, 0.5 to 3, and 5 to 15 mg/m$^3$, respectively (Pacyna and Nriagu 1988).

## 3.2 Water

Chromium exists in its two stable oxidation states, Cr(III) and Cr(VI), in natural waters. The presence and ratio between these two forms is dependent on chemical and photochemical redox transformation, precipitation and dissolution, and adsorption and desorption reactions (Kumral 2007). Dissolved Cr(T) is the form most measured in surface and groundwater.

In contrast to Cr(III), Cr(VI) is not readily adsorbed to surfaces and, given that many of its salts are soluble, much of the Cr(VI) released to aerobic surface waters is present as hydrochromate, chromate and dichromate ionic species (Rai *et al*. 1989). At pH 5 or above, Cr(III) is not likely present in water, because of the low solubility of the hydrated oxide (HC 1986). However, Cr(III) concentrations can be elevated in some deep anoxic waters and in water receiving direct discharges of Cr(III)-containing wastes. Depending on local hydrogeological conditions and the distribution of dissolved oxygen, Cr(VI) concentrations may vary considerably with depth (Ball and Izbicki 2004). In preliminary studies, both Cr(III) and Cr(VI) were shown to exist in surface waters. The conversion of Cr(III) to Cr(VI) in natural lake waters is very slow (HC 1986).

The European Chemicals Bureau (2005) provided suspended matter-water partition coefficients ($Kp_{susp}$) of 2000 L/kg and 30 000 L/kg under acidic conditions, and 200 L/kg and 300 000 L/kg for under alkaline conditions for Cr(VI) and Cr(III), respectively. Studies in aerobic surface waters indicated Cr(VI):Cr(III) ratios of 1:1 to 2:1 (EC and HC 1994). Few oxidants are able to convert Cr(III) to Cr(VI) and such oxidation is normally very slow. However, it has been suggested that labile (including dissolved and colloidal) forms of Cr(III) can be converted to Cr(VI) relatively quickly by strong oxidants (e.g., $H_2O_2$) produced photochemically in aerobic surface waters (Pettine and Millero 1990; Pettine *et al*. 1991; Nriagu *et al*. 1993). By itself, dissolved oxygen in natural waters did not cause any measurable oxidation of Cr(III) to Cr(VI) in 128 days (Saleh *et al*. 1989).

Frey *et al*. (2004) indicate that, while Cr(T) occurs equally in surface waters and groundwaters, Cr(VI) is relatively insoluble and not found to nearly the same degree in groundwater. Dissolved oxygen may control the concentration and speciation of chromium in groundwater. Where dissolved oxygen concentrations were less than 1 mg/L in Mojave Desert groundwater, Cr(III) was found to be the predominant form of dissolved chromium. Its median concentration was estimated at 0.1 µg/L, owing to its low solubility in near-neutral pH water (Ball and Izbicki 2004).

In the absence of organic matter or appropriate reducing agents, aqueous Cr(VI), present as complexed soluble anions, can persist indefinitely in the aquatic environment. However, as a strong oxidizing agent, Cr(VI) reacts readily with dissolved organic molecules to form Cr(III). Dissolved Cr(VI) can be also be converted to Cr(III) by a host of reducing agents such as $S^{2-}$, Fe(II), fulvic acid, organic compounds with low molecular weights, and proteins, and is thus removed from solution. This is common in deeper anaerobic waters (Nriagu *et al*. 1993). The effectiveness of these reducing agents varies with pH, redox conditions and Cr(T) concentrations

(Nriagu *et al*. 1993). The reduction of Cr(VI) by sulphide or Fe(II) ions occurs rapidly under anaerobic conditions; the reduction half-lives range from instantaneous to a few days (Saleh *et al*. 1989). In deep anoxic water, colloidal Cr(III) can be the dominant chromium species (EC and HC 1994). A small amount of Cr(VI) can also be taken up by plankton and released as Cr(III) at lower depths where oxygen is depleted (EC and HC 1994).

## 3.3 Sediment

The same processes that govern the distribution of chromium in natural waters, such as redox potential, precipitation and adsorption, also govern the distribution of chromium in sediments (European Chemicals Bureau 2005). Nearly all of the chromium present in sediments is likely to be Cr(III), except for those sediments present immediately below the interface with overlying aerobic waters (Nriagu *et al*. 1993), where some Cr(III) can be oxidized by manganese oxides and hydroxides present at the sediment-water interface (Saleh *et al*. 1989; Bartlett and James 1988).

In surface water bodies, dissolved Cr(III) may be removed from the water column by precipitation, adsorption onto suspended particles, and the formation of oxide, hydroxide and phosphate complexes (e.g., $CrO_3 \bullet H_2O$), which ultimately settle to the sediment phase (Cranston and Murray 1978; Taylor *et al*. 1979). However, due to its strong affinity for oxygen, nitrogen and sulphur-containing ligands, Cr(III) can also form stable complexes with many dissolved or colloidal organic and inorganic ligands, which are relatively unaffected by adsorption and precipitation reactions and can thus remain in the water column (Masscheleyn *et al*. 1992; Irwin *et al.* 1997). Reduction of Cr(VI) in organic sediments is slow, and depends on the type and amount of organic material and on the redox conditions in the water. The reduction half-life of Cr(VI) in water containing soil and sediment ranged from four to 140 days (Saleh *et al*. 1989).

It has been suggested that Cr(VI) in sediment can be released to the overlying waters, especially by bioturbation processes (EC and HC 1994). Estimated adsorption partition coefficients between water and sediments ($Kp_{sed}$) for acidic and alkaline conditions were $Kp_{sed}$ = 1000 L/kg and 11 000 L/kg and $Kp_{sed}$ = 100 L/kg and 120 000 L/kg for Cr(VI) and Cr(III), respectively (European Chemicals Bureau 2005).

## 3.4 Soil

The fate of chromium in soil is greatly dependent upon its speciation, which is a function of redox potential and soil pH (ATSDR 2012).

Cr(III) dominates in most unpolluted soils (Bartlett and James 1988; Katz and Salem 1994). Cr(III) is present primarily as insoluble hydroxides and oxides and adsorbed to clay particles, soil organic matter, metal oxyhydroxides and other negatively charged particles (EC 1996), although it can exist in complexes with inorganic and organic ligands (Puls *et al*. 1994; McGrath 1995).

Consequently, Cr(III) is considered relatively immobile and stable in most soils (CCME 1999) and generally does not leach from soil to groundwater (CCME 1997). Cr(III) solids, such as oxide ($Cr_2O_3$) and phosphate complexes (e.g., $CrPO_4$), are practically insoluble at pH >4 (CCME 1997) and show increased sorption and immobilization with increasing soil pH (Puls *et al*. 1994; CCME 1999). However, gradual mobilization by acid leaching (podzolization) has been reported (Bartlett and James 1988), and oxidation to Cr(VI) can occur under specific environmental conditions (MiningWatch 2012).

The redox potential of the Cr(VI)/Cr(III) couple is quite high at +1.33 eV (Shanker *et al*. 2005) and only a small percentage of the Cr(III) in soils is normally present in oxidizable forms (Bartlett and James 1988). Relatively few oxidants are known to mediate the conversion of Cr(III) to Cr(VI) in the soil environment. Oxidation has been reported with manganese oxide, dissolved oxygen and soil water activity (Rai *et al*. 1989), although dissolved oxygen-mediated oxidation is much slower than with manganese oxides (EC 1996). Manganese oxides present in fresh, moist, non-acid, aerobic soil samples serve as the electron link between Cr(III) and oxygen in the atmosphere; the amount of Cr(III) oxidized to Cr(VI) is proportional to the manganese reduced (Bartlett and James 1979). The proposed oxidation mechanism is sequential adsorption of Cr(III) onto $MnO_2$ surface sites, oxidation by surface $Mn^{+4}$ and then desorption of Cr(VI) (Puls *et al*. 1994). The rate of oxidation increases with decreasing pH and with increasing ratios of surface area to solution volume (Eary and Rai 1989). Decreasing pH results in increased Cr(III) solubility, enabling increased contact with the oxidizing agent (Bartlett 1991). Abiotic oxidation of Cr(III) to Cr(VI) is also facilitated by the presence of moisture and small amounts of organic matter, and it can be enhanced by elevated surface soil temperatures, as may occur in brush fires (Bartlett 1991; Panichev *et al*. 2008).

Cr(III) oxidation is not observed in soil samples that have been dried and stored for extended periods of time (Bartlett and James 1979; Bartlett 1991). It is not clear whether this is due to physico-chemical alterations within the soil matrix or to reduced microbial activity.

Cr(VI) added to or formed in soils can be removed from soil solution by uptake into living organisms, adsorption, reduction to relatively immobile Cr(III) (Bartlett and James 1988) or leaching resulting in transfer to groundwater, where it is quite stable and can have a long residence time (Prokisch *et al*. 1997).

Factors influencing the reduction of Cr(VI) to Cr(III) in soil include soil pH, the presence of electron donors (such as organic matter or ferrous ions), and soil oxygen levels. Cr(VI) reduction increases with decreasing soil pH (Bartlett and Kimble 1976; Bloomfield and Pruden 1980; McGrath 1995; Bartlett 1991; Eary and Rai 1991). Soil pH affects the degree of positive and negative charge on the surfaces of soil colloids, thus directly influencing the availability of electron donors (Bartlett and James 1988). Rai *et al*. (1989) concluded that acidic soil solutions enhance the release of Fe(II) ions from soil minerals, which increases Cr(VI) reduction.

Cr(VI) to Cr(III) reduction is significantly slowed in soils lacking appropriate electron donors such as ferrous iron minerals, silicate minerals, reduced sulphur species and soil organic matter (Palmer and Wittbrodt 1991). Bartlett and Kimble (1976) found no evidence of Cr(VI) reduction in soils with very low organic matter content (<0.05%). Cr(VI) reduction increased linearly with increasing soil humus content. Cr(VI) reduction by Fe(II) could be an important fate process in subsoils, where organic matter content is typically low (Bartlett and James 1988; Eary and Rai 1991). Reduction of Cr(VI) is enhanced under anaerobic conditions, such as within waterlogged soils (Bloomfield and Pruden 1980; Bartlett 1991; Losi *et al*. 1994a). Since oxygen is an electron acceptor, it is believed to inhibit Cr(VI) reduction through direct competition for electron donors (Losi *et al*. 1994b). Waterlogged soils may also enhance chromium reduction because of increased $CO_2$ trapping, which tends to lower soil pH (Losi *et al*. 1994a). Furthermore, soil microbial activity may indirectly influence Cr(VI) reduction by decreasing soil oxygen concentrations and increasing $CO_2$ levels (Losi *et al*. 1994a).

Cr(III) is strongly adsorbed to clay particles, soil organic matter, metal oxyhydroxides and other negatively charged particles. The solubility of Cr(III) is enhanced by chelation to low-molecular-weight organic compounds such as citric or fulvic acids (Bartlett and James 1988). Average Cr(III) retention in mineral soils was reported to be lower (23%) than in highly organic soils (78%) (Balasoiu *et al*. 2001). Cr(III) is strongly adsorbed to both kaolinite and montmorillonite clays <pH 4. At pH 4 to 5, the combination of adsorption and precipitation renders Cr(III) immobile in most soils (CCME 1999). Since clay surfaces become more negatively charged as pH increases, Cr(III) adsorption increases with increasing soil pH. Estimated adsorption partition coefficients ($Kp_{soil}$) for acidic soils were 50 L/kg and 800 L/kg, and 2 L/kg and 15 000 L/kg for alkaline soils for Cr(VI) and Cr(III), respectively (European Chemicals Bureau 2005).

Cr(VI) solids, except $BaCrO_4$, are soluble within the soil environment. Although not readily adsorbed to most surfaces, Cr(VI) adsorbs to clay minerals through surface complexation reactions with inorganic hydroxyl groups such as iron and aluminum oxides, or along the edges of layer silicates (Rai *et al*. 1989; Zachara *et al*. 1989). Cr(VI) adsorption increases with decreasing pH as a result of the protonation of surface hydroxyl sites (Zachara *et al*. 1988; 1989) and in proportion to iron and aluminium oxide concentrations, while the presence of $SO_4^{2-}$ and dissolved inorganic carbon depresses Cr(VI) adsorption (Zachara *et al*. 1989). In general, highly weathered soils dominated by oxide-rich colloids adsorb more Cr(VI) than less weathered acid soils (Bartlett and James 1988; Bartlett 1991). Competition for adsorption has been shown with phosphate, carbonate and sulphate (Puls *et al*. 1994). Adsorption can inhibit or completely prevent the reduction of Cr(VI) to Cr(III) in some soils (Bartlett and James 1988). Cr(VI) that is not adsorbed or reduced to Cr(III) remains highly mobile within the soil profile. At pH >8.5, Cr(VI) is completely mobile and can readily leach to groundwater. Increasing soil pH by liming or the addition of phosphate fertilizers would likely result in the remobilization of adsorbed Cr(VI) (Bartlett and James 1988).

## 3.5 Bioconcentration and Bioaccumulation

As with most elements, the bioavailability of chromium depends upon its chemical speciation and its adsorption to environmental matrices (WHO 2006).

The bioavailability of Cr(III) to freshwater invertebrates (*Daphnia pulex*) decreased with the addition of humic acid (Ramelow *et al*. 1989). This decrease in bioavailability was attributed to the lower availability of the free form of the metal due to its complexation with humic acid. Based on this information, chromium is not expected to biomagnify in the aquatic food chain.

Reported bioaccumulation factors for chromium in aquatic and terrestrial species are summarized in Appendix 8.

## 3.6 Assumed Fractionation of Hexavalent Chromium (Cr(VI)) to Total Chromium (Cr(T))

The methodology for estimating the fraction of Cr(VI) in relation to Cr(T) is summarized below. HC provides a more detailed discussion (HC 2012a; 2017b).

### *3.6.1 Outdoor Air*

From ambient air studies, Cr(VI) was <1% of total airborne chromium in remote (rural) areas, but between 10 and 40% of Cr(T) in urban and industrial areas with known chromium sources (Bell and Hipfner 1997).

Studies in Windsor and Hamilton, Ontario, indicated that approximately 20 to 25% of ambient airborne Cr(T) was Cr(VI), with 24-hour average Cr(VI) concentrations between 0.1 and 6 ng/m$^3$ (geometric mean = 0.55 ng/m$^3$) (Bell and Hipfner 1997). A size fractionation study suggested that the majority of Cr(VI) was in the inhalable fraction (Bell and Hipfner 1997).

Similar concentrations were measured in meadowland background air concentrations. Concentrations ranged from 0.2 to 3.8 ng/m$^3$ Cr(VI) (mean = 1.2 ng/m$^3$) and 1.5 to 10 ng/m$^3$ Cr(T) (mean = 4.5 ng/m$^3$), with a calculated mean Cr(VI)/Cr(T) ratio of 26.6%, and were considered similar to those measured elsewhere in the US (Scott *et al*. 1997).

Results from a monitoring study representing industrial and rural background sites (JCDH 2009) showed a mean fraction concentration Cr(VI)/Cr(T) of 0.7% (range = 0.86 to 2.71%; n = 62; R = 0.52 and $R^2$ = 0.26).

Urban and industrial data indicate that 25% is a reasonable fraction for Cr(VI). It represents the upper end of the available Canadian data (Bell and Hipfner 1997) and is comparable to urban and industrial air data from other countries. For rural air masses, 1% was chosen to represent the Cr(VI)

fraction from Cr(T) measurements in air based on data from Bell and Hipfner (1997), which is similar to the lower range from studies which included rural air samples.

According to the 2006 Canadian Census of Population, 80% of Canadians live in urban or industrial areas (Statistics Canada 2014). Based on the data above, a value of 20% (1% × 0.20 + 25% × 0.80) is suggested to represent the fraction of Cr(VI) in measurements of Cr(T) in air samples. This value represents the estimated percentage of the population living in rural areas and urban or industrial areas.

### *3.6.2 Indoor Air*

Mean ratios of Cr(VI) to total chromium in US studies were reported by Sheehan *et al*. (1992), Falerios *et al*. (1992), Finley *et al*. (1993) and in one Canadian study (Bell and Hipfner 1997).

Based on these references, a suggested Cr(VI) fraction of 20% is recommended for indoor air and considered adequately conservative. This is in line with the geometric mean from Sheehan *et al*. (1992) study and similar to the Cr(VI)/Cr(T) ratio measured in the Canadian study with respect to outdoor air (Bell and Hipfner 1997).

### *3.6.3 Dust*

According to Stern *et al.* (2010) there was no statistically significant difference in Cr(VI) concentrations in dust between homes located near a pollution source and those considered to be unexposed. Cr(VI) was found in the dust of all homes sampled. In homes near the chromate plants, the mean (± standard deviation) Cr(VI) concentration for all samples was 3.9 (±7.0) µg/g compared to 4.6 (±7.8) µg/g for all background home samples.

According to Fan *et al*. (2009), the mean ratio of Cr(VI)/Cr(T) was 8% (range = 1 to 20%), which was estimated based on replicate samples from the same location (n = 10). However, Lioy and Gochfeld (2008) obtained a median value of 9% (n = 31) and a mean value of 12% (range = 0.3 to 51%).

According to Bell and Hipfner (1997), the majority of the airborne Cr(VI) was in the inhalable size fraction. Assuming that most Cr(VI) in dust is found in the respirable size fraction, the recommended Cr(VI)/Cr(T) ratio is 10%, which is at the higher end of the means in the Fan *et al*. (2009) and Lioy and Gochfeld (2008) studies.

### *3.6.4 Soil*

The main source of chromium in natural soils is weathering of parent materials. The relation between Cr(III) and Cr(VI) depends strongly on pH and oxidative soil properties, but Cr(III) is the

dominant species (Kotaś and Stasicka 2000). In soils, Cr is mainly present as insoluble $CrOH_{3aq}$ or as Cr(III) adsorbed to soil components, which prevents leaching into groundwater or uptake by plants. Soil pH strongly influences the dominant chromium form; in acidic soils (pH<4), it is primarily present as $CrH_2O_6^{3-}$, whereas at pH <5.5, it is primarily present as the hydrolysis product, $CrOH^{2+}_{aq}$.

Considering that the Cr(III) oxidation state is the most stable and chromium is mined only as chromite ($FeCr_2O_4$) ore, the recommended Cr(VI)/Cr(T) ratio is 2%. This value is based on the only Canadian reference, "Ontario typical range for chemical parameters in soil", (OMOE 1993), which indicates that the Cr(VI)/Cr(T) fraction is 2%.

### *3.6.5 Drinking Water*

Cr(VI) is found in both groundwater and surface water, although to a lesser extent in surface water (AWWA 2013). Eaton *et al.* (2001) demonstrated that typically, for California drinking water, more than 90% of chromium is present as Cr(VI).

Several studies on chromium occurrence in the environment indicate that Cr(T) concentrations in groundwater can be primarily as Cr(VI) (AWWA 2013). Analysis of Cr(T) and Cr(VI) from well water and distribution system water monitoring in Wisconsin, US, demonstrated that that 82 to 98% of Cr(T) was Cr(VI) (Madison Water Utility 2011).

Considering that the concentration of chromium in household tap water may be higher than in supply water due to the corrosion of chromium-containing pipes (Ohanian 1986), it is recommended that the fraction of Cr(VI) in drinking water be estimated at 100% for the calculation of soil quality guidelines for Cr(VI).

### *3.6.6 Breast Milk*

Data specific to Cr(VI) in breast milk were not available. There is evidence that any chromium that is absorbed into the body (after ingestion, inhalation, *etc.*) will be reduced rapidly to Cr(III) after contact with reducing agents in biological fluids and tissues (Paustenbach *et al.* 2003). Therefore, it was assumed that there was essentially no Cr(VI) (0%) in breast milk.

### *3.6.7 Food*

A limited survey in 12 food samples revealed that 11 to 63% of Cr(T) was present as Cr(VI) (Schroeder *et al*. 1962). A study by Soares *et al*. (2010) found that Cr(VI) accounts for 12 and 13% of Cr(T) in white and whole wheat bread, respectively. Another study of breads and cereals in France showed similar percentages (≈15%) of Cr(VI) (Sykula-Zajak and Pawlak 2012), while

another did not detect Cr(VI) in any of the samples of diverse foodstuffs analyzed (Vacchina 2015). Hughes *et al*. (1994) state that Cr(III) is the dominant form. Based on the results observed in the limited available data summarized above in the current assessment, it was assumed that Cr(VI) comprises 10% of Cr(T) in food, for the purpose of deriving the $SoQG_{HH}$. Data are provided in Appendix 1.

The estimate of Cr(VI) content in various environmental media is as follows:

- 20% in outdoor and indoor air
- 100% in drinking water
- 2% in soil
- 10% in indoor dust and food
- 0% in breast milk.

# 4. BEHAVIOUR AND EFFECTS IN HUMANS AND NON-HUMAN MAMMALIAN SPECIES

## 4.1 Overview

The behaviour and effects of Cr(III) and Cr(VI) in humans and non-human mammalian species have been the subject of several key toxicological reviews, including HC (2016; in derivation of a drinking water quality guideline), EC and HC (1994; in estimation of a cancer potency factor for use in Priority Substance List assessments), the ATSDR (2012; in derivation of minimal risk levels), and the US EPA (1998c; d; 2010; as part of the US EPA Integrated Risk Information System (IRIS) database). Based on these reviews and additional, recently published studies, section 4.0 presents information on the mode of action, toxicokinetics and health effects of Cr(III) and Cr(VI), in experimental animals and humans. Wherever possible, the data presented specify the oxidation state of the chromium compound in question.

As the soil quality guidelines for human health are derived from toxicological reference values (TRVs) for chronic exposure, the key studies used by different agencies in the development of TRVs are described in some detail, while supporting information is presented in a more summarized form. In all cases, the reader should refer to the original study reports for more detailed information on dosing patterns, dose conversions and other experimental conditions.

## 4.2 Essentiality and Mode of Toxicity

### *4.2.1 Essential Nutrient Status of Chromium*

Although there is evidence that Cr(III) plays a beneficial role in glucose metabolism and regulation (IOM 2006), there is no clear consensus that the human body requires this element.

Chromium was found to aid in the regulation of glucose metabolism and lipids (Broadhurst and Domenico 2006); however, such benefits were found only in diabetics or patients receiving total parenteral nutrition. No measurable benefits, such as the popularly promoted conversion of fat into muscle, have been demonstrated in healthy people (Pittler *et al*. 2003; Trumbo and Ellwood 2006; Vincent and Stallings 2007; Balk *et al*. 2007). An evaluation performed by the US Food and Drug Administration (FDA) concluded that while chromium picolinate supplementation was safe (only one study (Cefalu *et al*. 1999), with a small number of subjects, supported a role for chromium picolinate in reducing the risk of insulin resistance), the existence of such a relationship between chromium picolinate and either insulin resistance or type 2 diabetes is considered uncertain (Trumbo and Ellwood 2006).

The Institute of Medicine (IOM) of the US National Academies of Sciences, Engineering and Medicine (now the Health and Medicine Division) has found that the current data are inadequate to determine an estimated average requirement (EAR) or a tolerable upper limit (UL) for chromium. However, adequate intakes (AIs) have been proposed (IOM 2006), reflecting estimates of average chromium intake from well-balanced diets. These AIs, expressed in µg/day of chromium, vary from 0.2 µg/day (infants) to 45 µg/day (lactating women). The 2006 AIs are lower than the estimated safe and adequate daily dietary intakes (ESADDIs) previously recommended (IOM 2001).

The recommended intakes of chromium are based on an AI that reflects the observed mean chromium intake of infants, toddlers, children, teens and adults. For a more detailed breakdown of AI with respect to age and sex, see Appendix 9.

A survey conducted in Maryland in 1998 revealed that the concentration of chromium found in several commercially available chromium picolinate products varied from 25 to 200 µg chromium (National Toxicology Program [NTP] 2012). In Canada, dietary supplements for adults (excluding food-like forms such as bars, chewing gums and beverages) can provide 2.2 to 500 µg/day (HC 2007). The hypothesis that normal subjects are deficient in chromium has served as the cornerstone of the chromium supplement industry. However, this conclusion was derived from comparison of "normal" dietary intakes to an estimated "safe and adequate daily dietary intake" of 50 to 200 µg/day (a previously recommended value by the National Academy of Sciences) and not from the clinical demonstration of symptoms of chromium deficiency. The data from the clinical studies indicate that dietary intake below 50 to 200 µg/day is sufficient to maintain positive chromium balances (Stearns *et al*. 1995).

### *4.2.2 Modes of Toxicity*

This section provides an overview of the mechanisms of Cr(III) and Cr(VI) toxicity by inhalation and oral routes. The summary is drawn from the information provided in section 4.3, as well as

in sections 4.8 and 4.10 with respect to carcinogenicity. More details can also be found in HC (2016) and literature referenced therein.

The mechanisms of chromium toxicity and carcinogenicity are complex. The oxidation state of the chromium atom is a major determinant of its toxic potency, Cr(VI) being more potent than Cr(III).

This difference in toxic potency may be explained by both molecular structure (influencing cellular uptake) and redox potential, described as follows:

- Due to differences in molecular structure, Cr(VI) enters cells more rapidly than Cr(III). At physiological pH, Cr(VI) exists as the tetrahedral chromate anion, resembling the forms of other natural anions (e.g., sulphate and phosphate) which are permeable across non-selective membrane channels. In contrast, Cr(III) forms octahedral complexes and cannot easily enter through these channels. Thus, lack of penetration through cell membranes may, at least in part, explain the lower toxicity of Cr(III) compared to Cr(VI). It follows that extracellular reduction of Cr(VI) to Cr(III) may result in the decreased penetration of chromium into cells, and therefore, decreased toxicity.
- The higher redox potential of Cr(VI) contributes to its higher toxic potency relative to Cr(III), because once it is taken into cells, Cr(VI) is rapidly reduced to Cr(III), with Cr(V) and Cr(IV) as intermediates. It is believed that many of the deleterious effects of chromium on cells, including lipid peroxidation and alterations in cellular communication, signalling pathways and the cytoskeleton, are due to the oxygen radical species generated by Cr(VI), Cr(V) and Cr(IV). In addition, the newly formed intracellular Cr(III) can form deleterious complexes with critical target macromolecules, including peptides, proteins and DNA.

The solubility of Cr(III) and Cr(VI) compounds (extracellular dissolution) is an important factor in the toxicity of the compound, as this influences absorption via oral, inhalation and dermal exposure routes. With respect to the inhalation of chromium particles, the location of particle deposition in the lung also influences toxicity. For example, with respect to Cr(VI)-induced carcinogenesis, higher chromium concentrations and more precancerous bronchial lesions have been observed at bronchial bifurcation in chromate workers. The carcinogenic potential of Cr(VI) is thought by many researchers to be dose-dependent in a non-linear fashion and, in risk assessment, would be best represented by a threshold model. At sufficiently low doses the capacity for detoxification in the gastrointestinal (GI) tract or lung may be adequate to reduce all Cr(VI) to Cr(III), this local capacity being significantly greater in the GI tract than in the lung.

In oral exposure animal studies (see Section 4.5.1), the most sensitive health effect observed is diffuse hyperplasia of the small intestine in mice. This occurs at lower doses than those resulting in an increase in small intestine tumours. The hyperplasia (increased cell turnover due to cellular damage) is often a precursor to cancer, as prolonged cellular regeneration may lead to accumulated damage to DNA, which in turn results in tumour growth. The supporting evidence for this

threshold mode of cancer for ingestion of Cr(VI) is described in further detail in Section 4.8.1.1 and by HC (2016).

The data from epidemiological studies in which workers are exposed to Cr(VI) primarily via inhalation are consistent with both threshold and non-threshold models, and the supporting evidence from animal inhalation studies for a threshold model of carcinogenicity is less extensive than that for oral exposures. As the non-threshold model provides a more conservative risk estimate, this model has been most often applied by public health organizations for the development of guidelines.

## 4.3 Toxicokinetics

### *4.3.1 Absorption*

The water solubility and oxidation state of the different chromium compounds are key determinants in their absorption rates via oral, inhalation, and dermal exposure routes. Both Cr(III) and Cr(VI) form compounds with a wide range of water solubilities.

#### 4.3.1.1 Oral

Absorption of both Cr(III) and Cr(VI) following oral administration is low in both experimental animals and humans. Absorption from the gastrointestinal (GI) tract occurs mainly in the upper small intestine (WHO 1988; Kerger *et al*. 1996a; ATSDR 2012) and may be up to four times higher in insulin-dependent diabetic patients (WHO 1988).

In general, soluble Cr compounds (e.g., $CrCl_3$) are better absorbed than insoluble forms (e.g., $CrCO_3$), and soluble Cr(VI) compounds (e.g., $K_2Cr_2O_7$) are better absorbed than soluble Cr(III) compounds, with up to 10 times greater absorption in humans reported in a study of Cr(VI) compared to Cr(III) (Kerger *et al*. 1996a; ATSDR 2012).

While absorption of dietary chromium (Cr(III)) reported in the literature generally varies from 0.4 to 2%, absorption of Cr(III) ingested as picolinate (a dietary supplement) may be higher, with values ranging from 0.7 to 5.2% in humans (Anderson and Kozlovsky 1985; WHO 1988; Stearns *et al*. 1995). In another study, absorption of Cr(III) picolinate in eight human volunteers was reported to be 2.8±1.14% (Gargas *et al*. 1994) compared to an absorption fraction of 0.4% for dietary chromium or dietary supplements of Cr(III) chloride (Anderson *et al*. 1983).

Facilitated transport may be responsible, in part, for the greater absorption of Cr(VI) as compared to Cr(III). Under physiological conditions, Cr(VI), as chromate, is isostructural with sulfate and phosphate; Cr(VI) therefore readily enters many types of cells by means of the anion-exchange carrier pathway that also transports phosphate and sulfate (Wiegand *et al*. 1985).

The gastrointestinal uptake of Cr(VI) is reported to be variable and largely dependent on the rate of GI reduction, especially under fasting conditions, while the fractional absorption of Cr(III) appears to be fairly consistent across subjects in different studies. Gastric reduction of Cr(VI) to Cr(III) would be expected to reduce the amount of Cr(VI) absorbed directly in this form. However, Cr(VI) reduction in the GI tract may also explain the higher absorption of ingested Cr(VI) compared to inorganic Cr(III). This process likely involves the formation of Cr(III) complexes that are more readily absorbed than inorganic Cr(III) forms due to their higher solubility. It has also been suggested that absorption from the GI tract is so rapid that it is able to compete effectively with reduction in the stomach. Cr(VI) which escapes reduction in the stomach and intestine can enter portal venous blood and be subject to reduction in the plasma, red blood cells and liver. With the exception of very high doses (e.g., fatal poisonings), it is expected that essentially all the chromium entering the systemic circulation will be reduced to Cr(III) (Kerger *et al*. 1997; O'Flaherty *et al*. 2001; Kirman *et al.* 2013).

Given the importance of gastrointestinal reduction on the absorption of chromium, it is not surprising that absorption is strongly influenced by various factors related to the diet. Absorption of both Cr(III) and Cr(VI) decreases when dietary intake increases, and increases during fasting (MacKenzie *et al*. 1959; ATSDR 2012). Interactions with dietary components may enhance or retard chromium absorption. For example, in rats, co-administration of $^{51}CrCl_3$ with phytate and with oxalate significantly decreased and markedly increased, respectively, chromium absorption (Chen *et al.* 1973). The timing of meals and the nature and amount of foods ingested may also influence absorption through Cr(VI) gastrointestinal reduction, with enhanced reducing capacity occurring after meals (De Flora *et al*. 1987). Orange juice and other low-pH foodstuffs and certain metals are efficient reducers of Cr(VI) to Cr(III) (Kerger *et al*. 1996a; b; Costa 1997). Cr(III) organic complexes formed by adding Cr(VI) to orange juice were absorbed three times more readily and exhibited a urinary excretion half-life almost double that observed for the ingestion of Cr(III) trichloride (Kerger *et al.* 1996a).

**Oral Absorption of Chromium from Soil**

Tests of *in vitro* bioaccessibility are carried out to measure chemical dissolution from a medium (e.g., soil at a contaminated site) in a solution simulating conditions in the gastrointestinal tract. When *in vitro* tests are sufficiently validated with *in vivo* studies, they may be used to estimate relative bioavailability, or the bioavailability of the chemical in the medium of concern (e.g., soil) versus the chemical's bioavailability in the media (e.g., drinking water, food) administered in the key toxicological study used to develop the TRV (HC 2017a).

The *in vitro* bioaccessibility of chromium in 12 soil samples collected near in-service CCA-treated poles (total chromium concentrations 26 ± 2.1 to 394 ± 53 mg/kg soil) ranged from below detection (<0.3%) to 33 ± 18% (mean ± SD: 8.5 ± 10%). Bioaccessible chromium was negatively correlated with silt content ($r^2 = 0.39$, $p < 0.05$) (Pouschat and Zagury 2008).

Dodd *et al.* (2017) measured the *in vitro* bioaccessibility of total chromium in soils collected across Canada. The mean bioaccessibility of total chromium in soil was 3.6% in the 0- to 5-cm layer (n = 14) and 7.3% in C-horizon soils (n = 164).

Morman *et al.* (2009) also reported total chromium bioaccessibility values of 0.5 to 7.5% from soils across Canada and the US.

Due to a lack of *in vivo* validation data, as well as a lack of information on speciation, the above bioaccessibility values were not quantitatively integrated into the development of the SoQG$_{HH}$ (Section 5.1).

#### 4.3.1.2 Inhalation

In humans, the absorption of Cr(III) and Cr(VI) via inhalation has been studied primarily for occupational exposures. Both Cr(III) and Cr(VI) compounds can be absorbed through the lung, as shown by biomonitoring data in urine, serum and tissues of humans with occupational exposure to soluble chromium compounds in air (Cavalleri and Minoia 1985; Gylseth *et al*. 1977; Kiilunen *et al*. 1983; Mancuso 1997a; b; Minoia and Cavalleri 1988; Randall and Gibson 1987; Tossavainen *et al*. 1980). Particle size and the solubility of the chromium species influence absorption following inhalation exposure.

Soluble Cr(VI) is more readily absorbed from the respiratory system than soluble Cr(III) although both Cr(VI) and Cr(III) associated with insoluble particles are subject to phagocytosis by macrophages (Cohen 2009). Mucociliary clearance and alveolar macrophages would remove a variable proportion of the inhaled Cr particles. However, with increasing Cr-particle exposure, the capacity of clearance mechanisms would be exceeded (Proctor *et al.* 2014).

Reduction of Cr(VI) to Cr(III) in the respiratory tract constitutes a line of defence against pulmonary chromium toxicity, occurring in the epithelial lining fluid, pulmonary alveolar macrophages, bronchial tree and peripheral lung parenchyma cells (US EPA 2010; De Flora *et al*. 1997; De Flora 2000). The reduction capacity of the respiratory tract, however, has been estimated to be substantially less than that of the gastrointestinal tract (Proctor *et al*. 2014). This lower reduction capacity can explain the greater absorption of Cr(VI) through inhalation as compared to absorption via the gastrointestinal tract, with an estimated 20 to 30% of highly water-soluble Cr(VI) entering the bloodstream following inhalation exposure (European Chemicals Bureau 2005).

#### 4.3.1.3 Dermal

Cr(VI) can penetrate skin more readily than Cr(III), but absorption of Cr(VI) is normally limited because of the reduction of Cr(VI) to Cr(III) on the skin (Cohen 2009). Absorption may be

considerably increased if the skin is damaged, as has been documented in chromic acid burns in industrial workers (Cohen 2009; ATSDR 2012). In contrast to chromium salts, chromium metal is not considered likely to penetrate intact human skin under normal physiological conditions (Larese *et al*. 2007).

HC (2010), in its federal contaminated site risk assessment guidance, recommends using relative absorption factors (RAFs) - dermal relative to oral - of 0.1 both for Cr(T) (most of which would be Cr(III) form) and for Cr(VI).

### *4.3.2 Distribution*

Tissue distribution of chromium depends on several factors including the chemical form, solubility and route of exposure. Cr(III) in the bloodstream is mainly transported bound to transport proteins. In contrast, Cr(VI) can readily cross the red blood cell (RBC) membrane and bind to hemoglobin (Cohen 2009; Kirman *et al.* 2013).

Inside the RBC, Cr(VI) is reduced to Cr(III) by glutathione or hemoglobin (O'Flaherty *et al*. 2001). Reduction of Cr(VI) in the plasma is thought to be low (Korallus *et al*. 1984; Minoia and Cavalleri 1988; Corbett *et al*. 1998). The reduction process in the bloodstream results in a low concentration of Cr(VI) in the cell, favouring continued passage of Cr(VI) from the extracellular milieu into the cell (O'Flaherty *et al.* 2001). The newly formed Cr(III) in the RBC is bound by hemoglobin (Gray and Sterling 1950; Wiegand *et al*. 1988) or low molecular weight ligands, likely glutathione (O'Flaherty *et al*. 2001), and is slowly lost from the cell (half-life of approximately 30 days, mean residence time 43 days) (Eadie and Brown 1955; Read 1954).

When Cr(III) salts are administered by oral or inhalation routes, it is presumed to be present in plasma as a stable mix of organic complexes with amino acids, other low-molecular-weight organic acids, and proteins, primarily globulins. The small fraction of chromium complexed with low-molecular-weight ligands is considered able to traverse membranes and diffuse into and out of plasma, blood and cells (O'Flaherty 1996; O'Flaherty *et al*. 2001; Paustenbach *et al*. 2003; Kerger *et al*. 1996a; 1997).

Once in the bloodstream, absorbed chromium may be widely distributed throughout the body. The iron-transport protein, transferrin, maintains chromium levels in the blood and transfers chromium to tissues in an insulin-responsive manner. Absorbed chromium distributes to nearly all tissues, with the highest concentrations found in kidney and liver. Bone is also a major chromium storage organ and may contribute to the long-term retention kinetics of chromium (ATSDR 2012). Total blood chromium concentration may not be a good indicator of tissue chromium accumulation since diffusible chromium is rapidly removed from blood and absorbed into tissues (Okada *et al.* 1983).

Transplacental transfer of chromium occurs in humans, as demonstrated in pregnant women with metal-on-metal hip arthroplasty (Ziaee *et al*. 2007). Placental transport in rats was shown to vary with the chemical form of chromium (Mertz *et al*. 1969) and varied with the timing of the exposure during pregnancy (Cohen 2009).

### *4.3.3 Metabolism*

Reduction of Cr(VI) to Cr(III) occurs in body fluids (including alveolar fluids), RBCs and nucleated cells, and may occur at various sites in the cell, including cytoplasm, endoplasmic reticulum, mitochondria or the nucleus. Small molecules (glutathione, cysteine and ascorbate), soluble proteins (haemoglobin and glutathione reductase) and microsomal proteins (NADPH-cytochrome P-450 reductase and cytochrome P-450 transport systems) are thought to be implicated in the intracellular reduction of Cr(VI) to Cr(III) (Connett and Wetterham 1983). After crossing cellular membranes, Cr(VI) tends to be metabolized either directly or via intermediates in a network of mechanisms. This leads to the generation of reduced chromium species (Cr(V), Cr(IV) and Cr(III)) and reactive oxygen species (ROS) such as radicals of oxygen, carbon and sulphur (Connett and Wetterham 1983; De Flora 2000; Sugden and Stearns 2000; Costa and Klein 2006).

Reduction may result either in activation or detoxification depending on the nature of the cellular components reducing Cr(VI), the site of the intracellular reaction and its proximity to DNA (Bianchi and Levis 1988). Reduction of Cr(VI) to Cr(III) in the cell is thought to be a prerequisite for genotoxic action of chromium salts. Intracellular Cr(III) binds DNA more efficiently than Cr(VI), and chromium-reduced intermediates (especially Cr(V)) are suspected to play a role in chromium genotoxicity and carcinogenicity through reaction with other cellular components, resulting in the generation of ROS (Norseth 1986; Stearns *et al*. 1994; De Flora 2000; Sugden and Stearns 2000; Sugden and Martin 2002; Zhitkovich 2005). Although uptake of Cr(III) salts by cells is low, uptake of Cr(III) is high when it is complexed with a ligand; considering that such complexation occurs *in vivo* (e.g., after ingestion of Cr(III) salts), it is likely that Cr(III) can be genotoxic *in vivo* (Norseth 1986; Kerger *et al*. 1996a). Mechanisms involved in the formation of chromium-DNA adducts are detailed in a review by Zhitkovitch (2005). Cr(III) resulting from Cr(VI) reduction reacts with other cellular constituents such as proteins and thus is likely involved in cytotoxicity. Apoptosis induced by chromium may be due to either Cr(VI) or repaired DNA adducts, which can both activate the p53 apoptosis pathway. On the other hand, ROS may inhibit apoptosis (Costa and Klein 2006).

Alternatively, reduction is a detoxification process when it occurs far away from DNA and the ROS can be trapped by a large number of ligands, nucleophiles and antioxidants which are present in the intracellular environment (De Flora 2000).

### 4.3.4 Elimination

Chromium absorbed via inhalation, whether originating from Cr(III) or Cr(VI) exposure, is largely eliminated from the body in the urine as Cr(III), but secondary excretion of smaller amounts (2 to 20%) may occur via the bile and feces (Suzuki *et al.* 1984; Cohen 2009).

Chromium absorbed following ingestion of Cr(VI), as $K_2Cr_2O_7$, appears to have a slower elimination rate (half-life ≈ 40 hours) than when absorbed following the ingestion of soluble Cr(III) (as $CrCl_3$, half-life ≈ 10 hours) (Kerger *et al*. 1996a). Chromium absorbed by tissues, especially after the ingestion of complexes such as picolinate, may be retained for longer periods (Stearns *et al*. 1995; Kerger *et al*. 1996a). Most of the unabsorbed portion of ingested chromium is excreted in the feces. Chromium supplementation in pregnant women has been shown to increase urinary chromium excretion, which is thought to be representative of serum chromium concentration. However, no relationship between breast milk chromium concentration and dietary intake or serum concentration was observed (Anderson *et al.* 1993; Mohamedeshah *et al.* 1998).

### 4.3.5 Toxicokinetic Models

Physiologically based pharmacokinetic (PBPK) models have been developed for ingestion of Cr(VI) and Cr(III) by rats (e.g., O'Flaherty 1996), rats and mice (e.g., Kirman *et al*. 2012) and humans (e.g., O'Flaherty *et al*. 2001; Kirman *et al*. 2013). More recent reports have refined the rodent and human models with respect to initial reduction in the gastrointestinal tract (Sasso and Schlosser 2015; Kirman *et al.* 2016).

#### 4.3.5.1 PBPK Models for Experimental Animals

The O'Flaherty (1996) model for chromium ingestion in rats is based on previous PBPK models developed to model the fate of bone-seeking elements (O'Flaherty 1991a; b; c). Bone-seeking elements have prolonged residence times in the body, with elimination kinetics largely determined by the balance among excretion, bone uptake, and release from bone; PBPK models for such elements must incorporate the characteristics of bone, bone growth, and growth and maturation of the organism over time (O'Flaherty 1988).

The rat lead model was adapted to model the kinetics of Cr(III) and Cr(VI) (O'Flaherty 1996) with input values for absorption, reduction and distribution obtained from published data (Mertz *et al.* 1965; Visek *et al.* 1953; MacKenzie *et al.* 1958; 1959). Final model calibrations were performed using oral and intratracheal kinetic studies in rats given soluble Cr(III) and Cr(VI) salts (Bragt and van Dura 1983; Weber 1983; Edel and Sabbioni 1985).

The model allowed for absorption of Cr(III) or Cr(VI) from the lung or GI tract and included five organ compartments: the liver, kidney, and other well-perfused tissues; bone and other poorly

perfused tissues; plasma; red blood cells; and an additional holding compartment for urine, to account for delays in excretion seen experimentally.

The model was validated by comparing the predicted results to those obtained for a Cr(VI) inhalation study (Langård *et al*. 1978). Although the model was calibrated with only oral data, O'Flaherty felt the model predicted Cr(III) and Cr(VI) kinetics reasonably well for both oral and inhalation exposures over the short or long term; however, two major limitations were identified. First, bioaccessiblity and bioavailability data were available for only a few chemically defined salts; pulmonary and oral bioavailability may prove to be the most important determinant of the toxicity of environmental chromium sources (as noted by Stearns *et al.* 1995). Second, the author argued that the role of bone as a reservoir and continuing source of internal exposure to chromium needs to be examined, and the mechanisms by which chromium is incorporated into bone as well as the dependence of bone chromium uptake on age and physiologic status also need to be clarified.

The model developed by Kirman *et al.* (2012) expands PBPK modelling of chromium kinetics to mice, as well as rats; however, it is restricted to oral exposure. The modelling of the GI tract was refined to include multiple compartments and reduction of Cr (VI) to Cr(III), modelled as a second-order, pH-dependent process. The authors found that the model predicted, within a factor of 3, chromium tissue concentrations in over 80% of the data points evaluated, performing better than the O'Flaherty (1996) model with respect to the results of a 2008 rat cancer assay. Modeling results also provided an explanation for differences between mice and rats, consistent with the results of carcinogenicity assays.

#### 4.3.5.2 Human PBPK Models

The O'Flaherty (1991a; b; c) rat lead model was adapted to model chromium kinetics in humans (O'Flaherty 1993). Specific adjustments were incorporated to account for the larger fraction of bone, the smaller proportion of bone to bone marrow, the more extensive structural remodelling of mature bone in larger animals, bone growth during early life, and the loss of bone as a result of aging (O'Flaherty 1991c; 1993; 1995; 2001).

The O'Flaherty (2001) model incorporated chromium-specific parameters, including differential absorption of Cr(III) and Cr(VI), rapid reduction of Cr(VI) to Cr(III) in all body fluids and tissues, modest incorporation into surface bone (with uptake and loss controlled by age-related bone turnover) and the co-existence of mechanisms for renal chromium excretion and retention at ambient exposures. The model was calibrated with human blood and urine concentrations, following oral exposure to inorganic Cr(III) and Cr(VI) salts, from Finley *et al*. (1997), Kerger *et al*. (1996a) and Paustenbach *et al*. (1996).

The kinetics of chromium are dependent on the route of administration, the chemical form and solubility; the model may not completely reflect the kinetics of chromium following environmental

exposures. However, the authors considered the model usable for an ambient or moderately elevated intake of chromium, where urinary clearance is set at a constant of 1 to 2 L of plasma per day and GI absorption rate set at constants of 0.25 per day for Cr(III) and 2.5 per day for Cr(VI).

The Kirman *et al.* (2013) model was adapted from the rodent model (Kirman *et al.* 2012) to model reduction as a second-order, pH-dependent process, based on studies of Cr(VI) reduction in human stomach fluid. For model development, toxicokinetic data for Cr(T) were identified from the scientific literature.

One difference between the Kirman *et al.* (2013) and O'Flaherty (2001) models is the treatment of individual data from human studies. Individuals are considered separately in the O'Flaherty (2001) model, whereas Kirman *et al.* (2013) averaged data across individuals in the same study to determine model parameters. Assessing the extent of variation in model parameters would therefore be useful in risk assessments using this Kirman *et al.* (2013) model.

### *4.3.6 Human Tissues and Body Fluids*

Few reliable data are available regarding chromium concentrations in tissues, organs and body fluids, but chromium is normally widely distributed throughout the human body in low concentrations, with the highest concentrations found in hilar lymph nodes and the lungs (Anderson 1987). Chromium concentrations have been reported to increase with exposure (Teraoka 1981), and to vary depending on age, gender, health status, and geographic location (presumably due at least in part to differing intake rates) (EC and HC 1994).

Higher-than-average levels of chromium have been measured in the pulmonary tissue of smokers. Lung concentrations of chromium were significantly higher in smokers who died of lung cancer than in non-smokers (Raithel *et al*. 1989). Similarly, chromium concentrations in the lungs of current or ex-smokers were 3.3 to 3.7 times higher than in those of non-smokers (Pääkkö *et al*. 1989); there was no significant difference between current and ex-smokers and there was a significant positive correlation between chromium content in lung and age.

Increased chromium concentrations may also be present in the lungs of occupationally exposed workers. For instance, chromium concentrations measured in two deceased stainless-steel welders were markedly elevated, with values 7 to 130 times higher than median concentrations reported in 30 subjects who were not occupationally exposed (Raithel *et al*. 1993).

In a review, Anderson (1987) reported that blood chromium concentrations increase with strenuous exercise, glucose challenge and chromium supplements, and decrease with pregnancy and during acute infectious illness. This author also reported that urine chromium concentrations can reflect dietary intake and have been reported to increase in individuals with diabetes and with glucose loading, strenuous running and physical trauma. Chromium partitions to blood components based

on species, with CrVI being the only species to partition to red blood cells (RBCs). Excretion kinetics are also highly variable between plasma and RBCs. Additionally, there are few datasets available for chromium blood concentrations in the general population (EFSA 2014).

Urinary chromium excretion reflects absorption over the previous one or two days only. Assuming no source of excessive exposure, urinary chromium levels are typically less than 2 µg/L for a 24-hour period (ATSDR 2012).

A summary of available background concentrations in human tissues and body fluids is provided in Appendix 1.

## 4.4 Acute Toxicity

The acute toxicity of Cr(III) and Cr(VI) in both experimental animals and humans is briefly summarized below for each exposure route. Much of the data for humans comes from case reports of accidental occupational exposures. HC (2016) also provides a toxicological review of chromium toxicity with additional details.

### *4.4.1 Mammalian (Non-Human)*

#### 4.4.1.1 Oral Exposure

In general, the acute toxicity of chromium compounds in experimental animals increases with solubility in water, with Cr(VI) being more toxic than Cr(III).

Reported $LD_{50}$ values for Cr(III) compounds range between 183 and 422 mg Cr(III)/kg for soluble compounds administered to rats and mice and 2365 mg Cr(III)/kg for the less soluble chromium acetate (Fairhurst and Minty 1989; ATSDR 2012).

In contrast, exposure to soluble Cr(VI) compounds, such as potassium dichromate, sodium dichromate, ammonium dichromate and sodium chromate in rats, has generated $LD_{50}$ values ranging from 13 to 20 mg Cr(VI)/kg for females and 23 to 28 mg Cr(VI)/kg for males (Fairhurst and Minty 1989; ATSDR 2012). $LD_{50}$ values for the less soluble compounds are higher (27 to 59 mg Cr(VI)/kg for chromium trioxide in rats and 70 to 91 mg Cr(VI)/kg in mice) (European Chemicals Bureau 2005). $LD_{50}$ values for other compounds were even higher: 811 mg Cr(VI)/kg for strontium chromate in male rats and 108 mg and 249 mg Cr(VI)/kg of calcium chromate for female and male rats, respectively (ATSDR 2012). Signs of toxicity included hypoactivity, lacrimation and diarrhoea, and necropsy revealed pulmonary congestion and corrosion of mucosa in the GI tract (European Chemicals Bureau 2005; ATSDR 2012).

#### 4.4.1.2 Inhalation Exposure

No $LC_{50}$ values for Cr(III) compounds were identified in the literature.

The $LC_{50}$ values for Cr(VI) compounds (sodium chromate, sodium dichromate, potassium dichromate and ammonium dichromate) reported by ATSDR (2012) vary between 33 and 82 mg Cr(VI)/$m^3$ for male rats and between 29 and 45 mg Cr(VI)/$m^3$ for female rats. Higher $LC_{50}$ values have been reported for chromium trioxide: 87 and 137 mg Cr(VI)/$m^3$ for female and male rats, respectively (ATSDR 2012). Other compounds revealed $LC_{50}$ in rats of the same order of magnitude: 99 mg Cr(VI)/$m^3$ for potassium dichromate aerosols, 200 mg/$m^3$ for ammonium dichromate and 104 mg/$m^3$ for sodium chromate. The acute toxic effects observed included reduced body weight, respiratory distress, lung irritation, inflammation and oedema, and tracheal epithelium necrosis (European Chemicals Bureau 2005).

#### 4.4.1.3 Dermal Exposure

No dermal $LD_{50}$ values for Cr(III) compounds have been identified in the literature.

Standard dermal testing in rabbits with Cr(VI) compounds produced $LD_{50}$ values ranging from 380 to 770 mg Cr(VI)/kg (European Chemicals Bureau 2005), with an $LD_{50}$ of 30 mg Cr(VI)/kg reported for chromium trioxide. Acute dermal toxicity was manifested as dermal necrosis, oedema, erythema, diarrhoea and hypoactivity (ATSDR 2012).

#### 4.4.1.4 Sensitization and Irritation

Contact with chromium is known to induce two types of dermatitis: allergic contact dermatitis (ACD) and non-allergic irritation, which may progress to ulceration of the skin. Irritation may occur at relatively large doses or at low concentrations under particular conditions (e.g., abraded skin, high humidity, etc.) and depending on the compound and vehicle in which it was applied.

Trivalent chromium diffuses through the skin at a much lower rate than Cr(VI). For this reason, it is thought to be less toxic to the skin; however, both Cr(III) and Cr(VI) have induced sensitization and allergic reactions in multiple species (Samitz and Epstein 1962; Gad *et al*. 1986; Samitz 1970; Fairhurst and Minty 1989; Shara *et al*. 2005).

It is thought that Cr(VI) is reduced to Cr(III) within the skin where it binds to the immune cells, eliciting the toxic response (Shelnutt *et al*. 2007).

### *4.4.2 Human Studies*

#### 4.4.2.1 Oral Exposure

Little information is available concerning the acute effects of chromium exposure in humans. Anecdotal reports of chromium intoxication include a small number of fatalities from oral ingestion. In all cases, highly water-soluble forms were implicated and doses, when estimated and reported, were in the range of 4 to 360 mg Cr(VI)/kg (ATSDR 2012).

No apparent clinical changes or health effects were reported in several studies in which human volunteers were exposed to 0.03 to 4 mg/day of Cr(VI) via drinking water (approximately 0.0004 to 0.06 mg Cr(VI)/kg) for at least three days (Paustenbach *et al*. 1996; Finley *et al*. 1997; Kerger *et al*. 1997) or to a single dose (5 mg or ≈0.07 mg/kg) of Cr(III) or Cr(VI) via drinking water or orange juice (Kerger *et al*. 1996b).

#### 4.4.2.2 Inhalation Exposure

Case reports suggest that brief exposure to highly soluble forms of Cr(VI) causes irritation of the upper respiratory tract, nasal mucous membranes and facial skin and causes a number of pulmonary symptoms (Fairhurst and Minty 1989). In the three-month period following acute occupational exposure to "massive amounts" of chromium trioxide fumes, abdominal or substernal pain as well as anorexia and weight loss were reported (Meyers 1950).

A study of 2357 chromate production workers reported nasal irritation and nasal ulceration in the majority of subjects (mean exposure of 0.048 to 0.054 mg $CrO_3/m^3$) (Gibb *et al*. 2000a).

#### 4.4.2.3 Dermal Exposure

In addition to skin burns, severe kidney damage, liver damage and gastrointestinal symptoms were observed in workers who had been accidentally sprayed or splashed with highly soluble Cr(VI) compounds (Fairhurst and Minty 1989). Application of Cr(VI) to the skin of patients (as chromic acid salts or ointments containing potassium chromate) was shown to be fatal in a few cases (ATSDR 2012). No effects were reported in human volunteers (n = 4) immersed in a bath containing 22 mg Cr(VI)/L for three hours (Corbett *et al*. 1997).

#### 4.4.2.4 Ocular Exposure

Congestion of the conjunctiva, discharge, corneal scar and burns were reported in chromate production workers as a result of accidental splashes (ATSDR 2012).

#### 4.4.2.5 Sensitization and Irritation

Chromium is recognized as one of the most common human sensitizing agents, resulting in respiratory and dermal effects. As indicated in Section 4.4.1.4, contact with chromium is known to induce two types of dermatitis. Allergic contact dermatitis (ACD) has been reported in numerous case reports and epidemiological studies of previously sensitized, occupationally or non-occupationally exposed populations. ACD typically involves Cr(VI), as greater exposure to Cr(III) is required to produce dermal effects, which tend towards irritant effects (Paustenbach *et al*. 2003; Shelnutt *et al*. 2007). Although a combination of inhalation, oral and dermal routes are probably the cause of initial sensitization in an occupational setting, information on the exposure levels that produce sensitization by the inhaled route was not identified (ATSDR 2012).

Dermal irritation due to chromium exposure may occur at relatively high doses (compared to those that induce sensitization and ACD) or at low concentrations under particular conditions (e.g., cut or abraded skin or other defects in the protective epidermis, such as high humidity). Minimum concentrations associated with dermatitis range from 10 to 25 mg/L (Shelnutt *et al*. 2007). Irritation can occur in both chromium-sensitized and non-chromium-sensitized people and can result in "chromium ulcers" or "chrome holes." In occupational exposures, chromium ulcers are mainly reported on the extremities of cement workers and the nasal septum of those exposed to chromic acid vapours (e.g., lithographers) (Shelnutt *et al*. 2007). The lowest concentration required to produce an ulcer is unknown.

## 4.5 Subchronic and Chronic Toxicity

Data are presented here on the mammalian and human toxicity of Cr(III) and Cr(VI) for a range of target organs and different exposure routes, for subchronic and chronic exposure periods. Note that reproductive and developmental toxicity, mutagenicity and genotoxicity, and carcinogenicity are presented in separate sections (Sections 4.6, 4.7 and 4.8, respectively). HC (2016) also provides a toxicological review of chromium toxicity with additional details.

### *4.5.1 Mammalian (Non-Human) Toxicology*

#### 4.5.1.1 Oral Exposure

While subchronic and chronic exposure studies for Cr(III) and Cr(VI) ingestion have been carried out on rodents since the mid-1960s, the most thorough investigations have been conducted by the US National Toxicology Program (NTP 1996a; b; 1997; 2007; 2008; 2010). The doses tested in earlier studies generally fell in the ranges of the NTP studies. Thus, Sections 4.5.1.1.1.1 and 4.5.1.1.1.2 focus on the NTP investigations. Details are also included for a study by Ivankovic and Preussmann (1975) since this study provided the basis for the US EPA (1998d) derivation of a Cr(III) oral reference dose.

#### 4.5.1.1.1 Trivalent Chromium

Subchronic Exposure

NTP (2010) subchronic exposure studies in male and female rats and mice exposed orally to five dietary levels did not elucidate any non-neoplastic or neoplastic effects. Therefore, no observed adverse effects levels (NOAELs) were developed from these studies (>506 mg Cr(III)/kg bw/day for rats and 1420 mg Cr(III)/kg bw/day for mice).

Chronic Exposure

Similarly to the subchronic studies, NTP (2010) and Ivankovic and Preussmann (1975) studies in rats and mice orally exposed to three dietary levels showed no signs of toxicity. Given the absence of observed treatment effects, NOAELs of >1468 mg Cr(III)/kg bw/day (as chromic oxide) (Ivankovic and Pueussmann 1975) and 313 mg Cr(III)/kg bw/day (as chromium picolinate) were identified (NTP 2010), whereas the NOAEL for mice was set at >783 mg Cr(III)/kg bw/day (as chromium picolinate (NTP 2010).

#### 4.5.1.1.2 Hexavalent Chromium

Subchronic Exposure

NTP conducted several studies in rats and mice exposed to potassium dichromate in the diet (including a multigenerational study) (NTP 1996a; b) and to sodium dichromate dihydrate (chromic acid) in water (NTP 2007). Animals were exposed to four or five concentrations in the diet or drinking water followed by a recovery period.

The reproductive studies on exposure via the diet resulted in the identification of NOAELs (100 ppm in rats and mice [NTP 1996a] and 1.4 mg Cr(VI)/kg bw/day [NTP 1996b]), although some mild, reversible effects on mean corpuscular volume (MCV) and mean corpuscular haemoglobin (MCH) were observed in rats and mice at the highest doses. These effects were considered suggestive of a possible bone marrow or erythroid response (NTP 1996a; b). In the second study (NTP 1996b), cytoplasmic vacuolization in the hepatocytes was also noted in the highest three dose groups. In the multigenerational study, decreases in mean absolute liver weights were observed in animals in the $F_0$ group receiving a mid-range dose, while females in the $F_1$ group administered the lowest dose showed slight haematological effects (NTP 1997). A lowest observed adverse effects level (LOAEL) of approximately 6.9 mg Cr(VI)/kg bw/day was identified for this study.

In the two-part drinking water exposure study (NTP 2007), rats and three strains of mice were first compared over several dose levels. In this first study, reduced body weights were observed in the

two highest dose groups for rats and the four highest dose groups for mice. Additionally, non-neoplastic lesions in the stomach at the highest dose level (rat) and duodenum at all dose levels (mice) were observed. Subsequently, a comparative toxicity study was performed which observed effects in the duodenum and the mesenteric lymph nodes of different mice strains and at different mid-range dose levels and above. No consistent evidence of the hepatic effects observed in the 1996 study (NTP 1996b) were observed in the 2007 study; however, haematological effects were observed in all three strains at mid-range dose levels and above. LOAELs were identified from these studies: 3.1 mg Cr(VI)/kg bw/day for duodenum effects observed in study 1 and 2.8 mg Cr(VI)/kg bw/day, also with effects in the duodenum, in study 2 (NTP 2007).

Chronic Exposure

NTP (2008) completed a two-year carcinogenicity study of sodium dichromate dihydrate (chromic acid) administered to male and female rats and mice at five dose levels in drinking water. Adverse effects were observed in both sexes and both species. In rats, effects included liver lesions and increased histiocytic cellular infiltration of the small intestine, mesenteric and pancreatic lymph nodes after exposure to mid-level doses. In mice, the incidence of diffuse epithelial hyperplasia in the duodenum was significantly increased. The LOAEL (0.4 mg Cr(VI)/kg bw/day) was based on this effect. Other effects in mice included additional duodenal effects as well as effects in the jejunum, liver, and mesenteric and pancreatic lymph nodes.

### 4.5.1.2 Inhalation Exposure

#### 4.5.1.2.1 Trivalent Chromium

Subchronic Exposure

Several studies have assessed inhalation exposure to different Cr(III) compounds as aerosols (Johansson *et al*. 1986a; b; Derelanko *et al*. 1999). Morphological and macrophage changes were observed in rabbits (Johansson *et al*. 1986a; b), whereas in rats, chromic sulphate resulted in more severe effects (e.g., effects in the nasal cavity, larynx, lungs and mediastinal lymph nodes, including hyperplasia, accumulation of foreign material and infiltration on lung tissue) in comparison to exposure to chromic oxide, whose effects were similar to other dust exposures (Derelanko *et al*. 1999). No other histopathological changes were observed in this study. Derelanko *et al*. (1999) indicated that 4.4 $mg/m^3$ as chromic oxide is near the NOAEL for subchronic exposures.

Chronic Exposure

No studies were located regarding chronic toxicity of Cr(III) compounds in animals via inhalation.

#### 4.5.1.2.2 Hexavalent Chromium

Subchronic Exposure

Pneumocyte toxicity has been demonstrated in short-term or subchronic inhalation investigations of Cr(VI) on the lung or immunological parameters (Johansson *et al.* 1986a; b; Glaser *et al.* 1985; 1990; US EPA 2010). Observed effects included inflammatory changes in the respiratory tract and alterations in macrophage response and morphology at 180 µg Cr(VI)/$m^3$ and above, although some effects on macrophage function and immunological response have been reported at lower concentrations (EC and HC 1994). US EPA (2010) selected Glaser *et al.* (1985; 1990) as the principal studies for the development of the Cr(VI) particulate reference concentration (RfC).

In a more recent subchronic study, a NOAEL of 0.20 mg/$m^3$ was identified in rats exposed to Cr(VI) as chromium trioxide aerosols (Kim *et al.* 2004).

No gastrointestinal or renal effects were observed in rats subchronically exposed to Cr(VI) via inhalation (Glaser *et al*. 1985).

Chronic Exposure

Effects on the respiratory system have been reported in rats, mice, guinea pigs, rabbits and hamsters after exposure to sodium chromate, sodium dichromate, potassium dichromate, calcium chromate and ground chromium roast material (Glaser *et al.* 1986; 1988; Steffee and Baetjer 1965; Nettesheim *et al.* 1971; Adachi *et al.* 1986; Adachi 1987; US EPA 2010). However, many long-term inhalation studies were limited by small group sizes, inadequate histopathological examination or single exposure levels.

Glaser *et al.* (1986; 1988) observed hepatic effects in rats exposed to sodium dichromate, whereas studies of renal and adrenal gland effects in rats have generally not shown any evidence of effects after exposure to chromium dichromate or a mixture of chromium trioxide and chromium oxide (Glaser *et al.* 1986; 1988). Some incidence of GI tract effects were reported after exposure to calcium chromate; however, details describing the lesions were not reported (Nettesheim *et al.* 1971).

#### 4.5.1.2.3 Mixture of Trivalent and Hexavalent Chromium

Glaser *et al.* (1986; 1988) reported a LOEL of 0.1 mg/$m^3$ based on respiratory effects after exposure to a 3:2 mixture of Cr(VI) and Cr(III). Haematological effects were also observed after exposure to the mixture, whereas exposure to Cr(VI) alone also led to observed hepatic effects that were not present after exposure to the mixture.

#### 4.5.1.3 Dermal Exposure

4.5.1.3.1 Subchronic Exposure to Trivalent and Hexavalent Chromium

Only one study on the effects of Cr(VI) following dermal exposure was identified. Systemic effects were indicated after rats were exposed to an aqueous solution of potassium dichromate on their skin. Effects such as local inflammation, increased concentrations of glycoproteins in skin and serum, and increased levels of serotonin in skin and liver were observed (WHO 1988).

4.5.1.3.2 Chronic Exposure to Trivalent and Hexavalent Chromium

No chronic dermal studies were identified in the literature.

### *4.5.2 Human Toxicology*

The following sections present human data on exposure to Cr(III) and Cr(VI). The focus is on epidemiological studies (occupational or environmental) rather than individual case studies, as these are the most relevant to the derivation of toxicological reference values. Moreover, only studies in which exposures to Cr(III) and Cr(VI) are reported separately are included. HC (2016) contains additional details on the toxicological review of chromium.

#### 4.5.2.1 Oral Exposure

4.5.2.1.1 Trivalent Chromium

No data were identified regarding chronic toxicity of oral exposure to Cr(III) compounds in humans.

4.5.2.1.2 Hexavalent Chromium

Abdominal and haematological effects were reported in a cross-sectional study, after environmental exposure via drinking water contaminated with Cr(VI) from a nearby chromium alloy plant in China (Zhang and Li 1987). The drinking water concentration was established at 20 mg Cr(VI)/L, which is equivalent to a dose of 0.57 mg Cr(VI)/kg bw/day. This study also reported elevated mortality rates for stomach and lung cancer in communities with Cr(VI)-contaminated well water when compared with unexposed regions. However, this ecological study did not report statistical measures of association or individual exposures, nor did it account for numerous confounding factors.

### 4.5.2.2 Inhalation Exposure

#### 4.5.2.2.1 Effects on the Respiratory Tract

Trivalent Chromium

Information regarding the effects of inhaled Cr(III) on the respiratory tract is limited. Occupational studies of Cr(III) oxide or Cr(III) sulphate production workers have shown inconsistent results (ATSDR 2012; Huvinen *et al*. 1996; 2002a; b) and did not always account for confounding factors.

Similarly, studies on hematological disorders were inconsistent, showing either no observed effects (ATSDR 2012) or suspected effects on haemoglobin levels (Kornhauser *et al.* 2002).

There is no evidence that exposure to metallic chromium (Cr(0)) or Cr(III)) is associated with renal dysfunction, based on the results of a small number of studies (EC and HC 1994).

Hexavalent Chromium

A variety of non-neoplastic effects on the respiratory system have been reported in several epidemiological studies of occupationally exposed populations.

Occupations such as chrome plating, welding and chromate production expose workers to Cr(VI) (US EPA 2010). Several occupational studies have reported increases in lung cancer after occupation inhalation (HC 2016), while evidence for links to other cancers (gastro-intestinal tract, larynx, kidney, prostate, bladder, brain, small intestine, genital organs, Hodgkin's disease, lymphoma and leukemia) are less conclusive (HC 2016).

Non-neoplastic respiratory effects (nasal ulceration and septum perforation, coughing, sneezing, rhinorrhea, nasal itching, soreness, epistaxis, nasal irritation and bleeding, throat irritation, phlegm production, haemoptysis, bronchial asthma and bronchitis, chronic rhinitis, and rhinitis with bronchitis) have been associated with subchronic inhalation exposure to Cr(VI) as chromic acid mist and other forms (e.g., dichromate dust) at concentrations >0.001 mg/m$^3$ (Kleinfeld and Rosso 1965; Haslian *et al*. 1967; Gomes 1972; Cohen *et al.* 1974; Lucas and Kamkowski 1975; NIOSH 2008; Royle 1975; Lee and Goh 1988; Kuo *et al.* 1997; Kitamura *et al.* 2003). Other non-neoplastic effects involving the GI tract, kidneys and liver have also been observed in chromate production and chrome plating workers (Wang *et al.* 2011a; b; ATSDR 2012), as have excess deaths related to mental, psychoneurotic and personality disorders (Gibb *et al*. 2000b). No increased mortality due to non-malignant respiratory disease was reported in studies in welders exposed to Cr(VI), painters using chromate pigments, chrome leather tanners exposed to Cr(VI), stone masons using cement containing Cr(VI), ferrochromium workers exposed to both Cr(VI) and Cr(III), or stainless steel workers exposed to metallic chromium (Svensson *et al.* 1989). Evidence for these effects was not observed in all studies (EC and HC 1994).

## 4.6 Reproductive and Developmental Toxicity

### *4.6.1 Mammalian (Non-Human)*

Several studies assessed the reproductive or developmental toxicity of Cr(III) or Cr(VI) compounds in experimental animals exposed via inhalation or ingestion, which are the routes most relevant to environmental exposures.

#### 4.6.1.1 Trivalent Chromium

In mice, chromium picolinate-related skeletal defects were observed in one study (Bailey *et al*. 2006). However, these results were not reproduced in a subsequent study conducted under similar conditions (Bailey *et al*. 2008) and no maternal toxicity was observed in these studies. Additionally, no significant effects on neurological development were found, although Cr(III) as picolinic acid showed some effects on some developmental indictors.

#### 4.6.1.2 Hexavalent Chromium

The effects of Cr(VI) have been investigated in primates, rabbits, rats and mice (HC 2016). Effects on male reproductive toxicity (particularly on spermatogenesis) occurred in all species, at ≥2.1 mg Cr(VI)/kg-bw/day (Zahid *et al*. 1990; Subramanian *et al*. 2006; Yousef *et al*. 2006; ATSDR 2012). These effects were observed to be dose- and duration-dependent and appeared to be reversible in some studies with an extended recovery period (Subramanian *et al*. 2006). Effects on reproduction in female mice (placental effects and fetal resorption and loss) were observed only at doses of ≥46 mg Cr(VI)/kg bw/day (Junaid *et al*. 1996; Murthy *et al*. 1996; Trivedi *et al*. 1989; NTP 2007), and not in a lower-dose multigenerational study (NTP 1997). Effects on ovarian development, steroidogenesis and pituitary hormone synthesis in developing rats were observed after lactational exposure to potassium dichromate at higher concentrations than those causing the effects observed in mice (Banu *et al*. 2008).

### *4.6.2 Human*

Effects on the male reproductive system (particularly on spermatogenesis) have also been observed in humans occupationally exposed to chromium compounds (Li *et al*. 2001; Kumar *et al*. 2005; ATSDR 2012), although study details were not always fully reported. Investigation into effects on female reproduction effects is limited to very few studies of the spouses of occupationally exposed men. One large study did not observe an increased risk of spontaneous abortion among the spouses of steel welders compared to controls over a ten-year period (Hjollund *et al*. 1995).

## 4.7 Mutagenicity and Genotoxicity

### *4.7.1 Non-Human*

#### 4.7.1.1 Trivalent Chromium

Some genotoxic effects have been induced by Cr(III) in *in vitro* studies using bacteria, yeast and mammalian cells or sub-cellular systems, including DNA deletions and DNA damage (Bagchi *et al*. 1995; Hassoun and Stohs 1995; Kirpnick-Sobol *et al*. 2006; ATSDR 2012). The genotoxicity of Cr(III) is generally reduced in comparison to Cr(VI) (ATSDR 2012); however, Cr(III) induced more DNA deletions than Cr(VI) in one study (Kirpnick-Sobol *et al*. 2006). Evidence of chromosomal aberrations was equivocal. Cr(III) compounds did not induce sister chromatid exchange (ATSDR 2012). On the other hand, the Committee on Mutagenicity (COM 2004) and the Expert Group on Vitamins and Minerals (EVM 2003) consider chromium picolinate supplements non-mutagenic based on negative *in vitro* studies and therefore no futher *in vivo* studies were required.

In a review of the literature published between 1990 and 2004, Eastmond *et al.* (2008) found that Cr(III) gentoxicity studies are frequently conflicting. The results of *in vivo* tests using mammalian cell cultures are highly dependent on the Cr(III) compound tested, and *in vivo* systems often don't indicate toxicity where *in vitro* studies showed toxic effects. NTP (2010) conducted both *in vitro* and *in vivo* tests on chromium picolinate. In standard *in vitro* assays, no clear evidence of genotoxicity was identified. In male mice, no increase in the frequency of micronucleated normochromatic erythrocytes (NCEs) was observed. In females, a small non-significant increase in micronucleated NCEs was observed at the highest dose, but this result was considered equivocal, and the absence of increase in micronucleated polychromatic erythrocytes (PCEs) indicates that these concentrations did not induce bone marrow toxicity.

In mice, DNA crosslinks and strand breaks were not detected after interperitoneal injection of $CrCl_3$ (Cupo and Wetterhahn 1985). Similarly, chromium chloride had no effect in the *Drosophila melanogaster* wing spot test, which detects both somatic recombination and mutational events (Amrani *et al*. 1999) or in micronucleus formation in bone marrow cells in mice administered $CrCl_3$ (Itoh and Shimada 1996).

#### 4.7.1.2 Hexavalent Chromium

Hexavalent chromium compounds have consistently led to positive *in vitro* and *in vivo* genotoxicity assay results (Sedman *et al*. 2006; ATSDR 2012); however, results from *in vitro* assays or parenteral injection of Cr(VI) should be interpreted with caution since the genotoxic potential of Cr(VI) is influenced by various factors in the whole organism (e.g., availability of

Cr(VI) to target cells, influence of toxicokinetic and metabolic mechanisms) (De Flora 2000; De Flora *et al*. 2006).

Intracellular reduction of Cr(VI) to Cr(V), Cr(IV) and, ultimately, Cr(III) is believed to be required for genotoxicity (IARC 1990; De Flora 2000; Sugden and Stearns 2000; ATSDR 2012).

De Flora *et al.* (2008) indicated that the majority of observed genotoxic effects of Cr(VI) have been documented *in vitro*.

*In vivo*, Cr(VI) did not induce DNA damage in the GI tract of mice after oral administration, in contrast to results in gastrointestinal cells *in vitro* (De Flora *et al*. 2008). In another study, with oral administration of Cr(VI) in drinking water, DNA deletions were observed in the offspring of treated mice in a concentration-dependent manner (Kirpnick-Sobol *et al*. 2006). NTP (2007) reported mixed results in micronucleus tests completed in various strains of mice exposed to sodium dichromate dihydrate via drinking water. While one study in one strain of mice observed increases in MN-NCE in male mice correlated to drinking water concentrations, two other studies with two other mice strains did not observe any mononuclear increases. De Flora *et al*. (2006) also studied Cr(VI) clastogenic effects through various routes of exposures and various exposure periods. As in the later study, no GI tract effects were observed after oral exposure, while some evidence of genotoxicity was observed following interperitoneal injection. The lack of GI tract effects after high exposures to Cr(VI) may be explained by the GI tract's highly efficient Cr(VI) detoxification process (De Flora *et al.* 2006).

### *4.7.2 Human*

Numerous studies have assessed the genotoxicity of chromium compounds in occupationally exposed humans and in human cell lines. No studies were located evaluating genotoxic effects in humans after oral exposure to chromium or its compounds (ATSDR 2012).

#### **4.7.2.1 Trivalent Chromium**

4.7.2.1.1 Occupational Studies

No increase in the number of chromosomal aberrations (CAs) or sister chromatid exchanges (SCEs) was found in the peripheral lymphocytes of tannery workers exposed to Cr(III), and plasma and urine concentrations were equivalent to the control group (Hamamy *et al.* 1987). However, Medeiros *et al.* (2003) did observe an increase in micronuclei frequency and DNA-protein crosslinks in the peripheral lymphocytes of tannery workers primarily exposed to Cr(III) compounds.

4.7.2.1.2 In Vitro Studies

Positive responses were found in human lymphocytes *in vitro.* The genotoxic potential of Cr(III) was several orders lower than that of Cr(VI) in the same systems (Nakamuro *et al.* 1978; Stella *et al.* 1982), and for micronuclei and DNA damage (ATSDR 2012). Some authors indicate that positive results with Cr(III) in intact cells could be due to artefacts (De Flora *et al.* 1990; IARC 1990). Due to its relative inability to cross cell membranes, exposure to Cr(III) compounds is less toxic than Cr(VI) compounds; however, when Cr(III) is formed by intracellular reduction from Cr(VI), or when Cr(III) is reacted with DNA in subcellular systems, it causes more DNA damage and mutations than Cr(VI) (Bridgewater *et al.* 1994a; b; 1998; Fornace *et al.* 1981; Snow 1991; Snow and Xu 1989).

### 4.7.2.2 Hexavalent Chromium

4.7.2.2.1 Occupational Studies

Increased levels of CAs and SCEs in the peripheral lymphocytes of electroplating workers, stainless steel welders and ferrochromium workers exposed to Cr(VI) were reported in several studies (Deng *et al.* 1988; Koshi *et al.* 1984; Lai *et al.* 1998; Sarto *et al.* 1982; Stella *et al.* 1982; Werfel *et al.* 1998; Halasova *et al.* 2001; Wu *et al.* 2001; Gambelunghe *et al.* 2003), while other studies reported no increase in CAs or SCEs (Husgafvel-Pursiainen *et al.* 1982; Littorin *et al.* 1983; Nagaya 1986; Nagaya *et al.* 1991; Benova *et al.* 2002). However, some of the studies with positive genotoxic results have limitations (Sarto *et al.* 1982; Koshi *et al.* 1984; Deng *et al.* 1988; Lai *et al.* 1998; ATSDR 2012). Similar indications of genotoxicity (DNA breaks) were found in chromium plating workers (Gambelunghe *et al.* 2003) and welders (DNA-protein crosslinks) (Medeiros *et al.* 2003). While increases in micronuclear frequencies were reported in two studies of chromium electroplating workers (Benova *et al.* 2002; Vaglenov *et al.* 1999).

Conversely, no elevations in DNA strand breaks or hydroxylation of deoxyguanosine were detected in lymphocytes of bichromate production workers (Gao *et al.* 1994) and no correlation between unscheduled DNA synthesis in pleural mesothelial cells and chromium urine concentrations was found in six chromium plating workers (Pilliere *et al*. 1992).

4.7.2.2.2 In Vitro Studies

Sodium chromate has been shown to be genotoxic in human cell lines: Ha *et al.* (2003; 2004) observed DNA double-strand breaks in cultured human dermal fibroblasts, while Holmes *et al.* (2006) and Wise *et al.* (2006) showed concentration-dependent chromosome damage in cultured human bronchial fibroblasts and bronchial epithelial cells, and Wise *et al.* (2006) observed disruption of mitosis in cultured human bronchial fibroblasts.

## 4.8 Carcinogenicity

### *4.8.1 Mammalian (Non-Human)*

Several studies regarding the carcinogenicity of chromium compounds have been performed in experimental animals, including rats, mice, guinea pigs, hamsters and rabbits. Studies related to metallic chromium and inorganic Cr(III) salts were consistently negative, suggesting that Cr(0) and inorganic Cr(III) salts are not carcinogenic. Cr(VI) carcinogenicity studies found both positive and negative results; more specifically, the Cr(VI) compounds of intermediate solubility (i.e., calcium chromate, strontium chromate and basic zinc chromate) were generally found to be carcinogenic, and the route of administration greatly influenced the results, with the majority of positive results being obtained after subcutaneous, intramuscular, intraperitoneal, intrapleural or intrabronchial administration. Such routes are not representative of human exposure as they may bypass important detoxification mechanisms (e.g., reduction at the portal of entry, first pass in the liver, *etc.*) (De Flora 2000). The following subsections are based on the most appropriate studies available for determining TRVs, including two studies completed by NTP. HC provides further analysis of carcinogenicity (HC 2016).

Particulate chromates (<10 µm) are more persistent than soluble chromate compounds and are considered more highly carcinogenic. Carcinogenicity may be secondary to chronic inflammation or the promotion of oxidative processes leading to persistent chromosome damage and chromosome instability; in contrast, chromosome damage is repaired with chronic exposure to soluble Cr(VI) (Ortega *et al*. 2005, Holmes *et al*. 2006). Reduction of Cr(VI) to Cr(III) in the respiratory tract constitutes a line of defence against pulmonary chromium toxicity (US EPA 2010; De Flora *et al*. 1997; De Flora 2000). Newer data suggest that extracellular particle dissolution, rather than internalization, mediates lead chromate clastogenesis in human bronchial cells (Xie *et al*. 2004).

#### 4.8.1.1 Oral Studies

4.8.1.1.1 Trivalent Chromium

The only adequate study assessing Cr(III) carcinogenic potential is the NTP carcinogenicity study of chromium picolinate dietary supplements in rats and mice (NTP 2008). Increased incidence of pituitary gland adenomas, with no significant dose trend, was observed in male rats, but no increased incidence of neoplasms was observed in female rats. In mice, no neoplasms or lesions were observed. NTP (2008) concluded that evidence for chromium picolinate carcinogenicity in male rats was equivocal and that the study provided no evidence of carcinogenicity in mice.

#### 4.8.1.1.2 Hexavalent Chromium

Cr(VI) was shown to increase the incidence of oral cancers in male and female rats and cancer of the small intestine in male and female mice exposed to sodium dichromate dihydrate in drinking water (NTP 2008). The authors of the study and the technical reports review subcommittee considered these data clear evidence of the carcinogenic activity of sodium dichromate dihydrate in rats and mice of both sexes (NTP 2008).

Further investigation has been conducted to better understand the mode of action (MOA) of Cr(VI) in the intestine. Thompson *et al*. (2013) carried out an MOA analysis based on evidence from the literature and the results of a 90-day drinking water study in mice and presented evidence for the following sequence of key events: absorption of Cr(VI) from the intestinal lumen, toxicity to intestinal villi, regenerative hyperplasia in the intestinal epithelium lining, and increased mutations within the intestinal (crypt) stem cells, resulting in late-onset tumourigenesis. HC (2016), in its evaluation of this work and other evidence (e.g., reductive capacity of the gastrointestinal tract), concluded that the intestinal tumours in mice in the NTP (2008) study resulted from a threshold MOA, and that the guideline derived on the basis of a precursor event (hyperplasia) would be protective against cancer as well.

### 4.8.1.2 Inhalation Studies

#### 4.8.1.2.1 Trivalent Chromium

No studies were located regarding the toxicity of Cr(III) compounds by inhalation.

#### 4.8.1.2.2 Hexavalent Chromium

While limited in number, studies to date indicate that Cr(VI) is weakly carcinogenic in experimental animals exposed via inhalation. The available data are equivocal in small-scale investigations, although small increases in the incidence of lung tumours were observed in several studies (HC 2016). Borderline increases in the incidence of lung tumours, the significance of which was not always indicated, have been reported in larger investigations (Baetjer *et al.* 1959; Steffee and Baetjer 1965; Nettesheim *et al.* 1971).

Carcinogenic effects were also reported following intratracheal administration of soluble sodium dichromate and calcium chromate in rats (Steinhoff *et al*. 1986).

#### 4.8.1.2.3 Mixture of Trivalent and Hexavalent Chromium

Slight increases in the incidence of respiratory tumours or lymphosarcomas were observed in three studies in which rats and mice were exposed to a mixture of Cr(III) and Cr(VI) by inhalation.

These increases could not be clearly attributed to one chromium species (Glaser *et al*. 1986; Baetjer *et al*. 1959).

### *4.8.2 Human*

Carcinogenicity associated with exposure to chromium compounds has been widely investigated in occupational populations. For the purpose of deriving TRVs, only those studies in which exposure is clearly characterized with respect to Cr(III) and Cr(VI) are included.

#### 4.8.2.1 Trivalent Chromium

There are no studies examining the carcinogenicity of Cr(III) alone. Occupational inhalation exposure to chromium compounds including Cr(III) has been studied in the leather tanning, chromate production and ferrochromium industries, which generally included mixed exposure to Cr(III), Cr(VI) and Cr(0). Little consistent evidence was found of association between exposure to Cr(III) and cancer. IARC (1990) concluded that Cr(III) compounds are not classifiable as to their human carcinogenicity (Group 3).

#### 4.8.2.2 Hexavalent Chromium

##### 4.8.2.2.1 Oral Studies

Evidence of carcinogenicity risks has been observed in studies of Cr(VI) exposure in drinking water (Costa and Klein 2006; Sedman *et al.* 2006).

Four epidemiological investigations of cancer rates in populations exposed to Cr(VI) have been published to date with contrasting results. Three American studies (Axelsson and Rylander 1980; Bednar and Kies 1991; Fryzek *et al*. 2001) found no increase in several health outcomes in response to environmental exposures (sourced from a gas compressor facility, ferrochromium alloy industries or contaminated drinking water) and a weak significant negative correlation between exposure through drinking water and chronic lung disease (Bednar and Keis 1991). In contrast, cancer mortality rates were increased in Chinese residents living in an area with significant chromium contamination in drinking water compared to residents in uncontaminated regions (Zhang and Li 1987; Beaumont *et al*. 2008). Beaumont *et al.* (2008) reported a number of limitations in the study and additional confounding factors were not accounted for in the analysis.

##### 4.8.2.2.2 Inhalation Studies

The carcinogenicity of chromium compounds has been investigated in retrospective mortality studies of a wide range of occupationally exposed populations, including workers in chromate

production, chromate pigment production and use, chrome plating, stainless steel welding and ferrochromium alloy production (Buckell and Harvey 1951; Bidstrup and Case 1956; Sassi 1956; Taylor 1966; Enterline 1974; Ohsaki *et al*. 1978; Alderson *et al*. 1981; Davies *et al*. 1991; Pastides *et al*. 1994; Satoh *et al*. 1994; Rosenman and Stanbury 1996; Mancuso 1997a; Gibb *et al*. 2000b; Crump *et al*. 2003; Park *et al*. 2004; Park and Stayner 2006). Exposure to Cr(VI) was associated with increased risk of lung cancer, less consistently with stomach cancer, and not with other types of cancer (Welling *et al.* 2015; IARC 2012; Cole and Rodu 2005). The occupational studies investigating cancer in relation to Cr(VI) exposure, along with the evidence from experimental animal studies, led IARC (2012) to conclude that Cr(VI) compounds are carcinogenic to humans (Group 1).

The forms of Cr(VI) involved in carcinogenicity have not been identified. In a review of epidemiological studies involving chromate production workers, Kimbrough *et al*. (1999) suggest that the insoluble forms of Cr(VI) may be carcinogenic while soluble species are not carcinogenic. In contrast, increased risk of lung cancer was correlated with exposure to soluble Cr(VI) in the chrome plating industry (chromic acid mists) (Sorahan *et al*. 1987) and at a chromium chemical production plant (Hayes *et al*. 1979).

The major studies providing evidence of Cr(VI) carcinogenicity and used to derive quantitative risk estimates are described below.

Lung Cancer

**Painesville, Ohio Worker Cohort: Chromate Production**

Mancuso *et al*. (1975; 1997a) retrospectively estimated individual weighted average worker exposures to insoluble, soluble and total chromium based on duration of employment in particular occupations and departments within the Painsville chromate production plant. Soluble chromium was present primarily as Cr(VI) and insoluble chromium was chiefly Cr(III).

The authors concluded that lung cancer rates clearly increased with exposure to total chromium, and that additional findings strongly suggested that lung cancers could not be solely attributed to soluble chromium (mostly Cr(VI)). However, the potential role of Cr(III) in lung cancer development was not analyzed by multiple regression analysis, and measures of Cr(VI), Cr(III) and Cr(T) may be interdependent, precluding conclusions regarding the relationship between Cr(III) and lung cancer.

Using published data (Luippold *et al*. 2003; Proctor *et al*. 2003; 2004), Crump *et al*. (2003) studied a distinct cohort from the same plant to evaluate the dose-response relationship and assess the risk of lung cancer in relation to lifetime cumulative exposure and highest monthly exposure. A strong relationship between lung cancer mortality and cumulative Cr(VI) exposure was evident in the

highest exposure groups only. These results are consistent with two possible assumptions: first, there is a linear dose-response, and second, there is a threshold below which no increased risk of lung cancer is expected. However, potential misclassifications of exposure information limited the identification of a clear distinction between a linear and threshold dose-response. The unit risks (UR) established in this study were 0.00978 $(\mu g/m^3)^{-1}$ and 0.0125 $(\mu g/m^3)^{-1}$ for the relative and additive risk models, respectively. The authors preferred the relative risk model, as they judged it more consistent with the expected trend for lung cancer risk with age. These values are comparable to the US EPA (2010) UR of 0.012 $(\mu g/m^3)^{-1}$ based on Mancuso (1975).

**Baltimore, Maryland Worker Cohort: Chromate Production**

In an extension of the Hayes *et al*. (1979) study of a Baltimore chromate production plant, Gibb *et al*. (2000b) identified a sub-cohort of male workers first employed between 1950 and 1974 (after construction of a new mill and roast and bichromate plant). The inclusion of several short-term workers (<90 days) in this group was considered a strength of the Gibb *et al*. (2000b) study and a weakness by Crump *et al*. (2003). Cr(III) concentrations were estimated through the use of measured airborne Cr(VI) concentrations and the Cr(III):Cr(VI) ratio in settled dust approximately three years after the facility closed. Cumulative Cr(VI) exposure (but not cumulative Cr(III) exposure) was associated with an increased lung cancer risk (Gibb *et al.* 2000b).

Gibb *et al.* (2000b) did not derive inhalation URs based on the data for this cohort. However, the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) (2011) produced a range of inhalation URs ranging from 0.01 to 2 $(\mu g/m^3)^{-1}$ depending on whether the line is modelled using all four exposure categories (lowest potency) or the two lowest exposure categories (highest potency), based on the Gibb *et al.* (2000b) data. OEHHA noted several advantages to the Baltimore study, including the larger cohort and concurrent exposure measurements.

Stomach Cancer

Some authors and agencies have concluded that the evidence for an association between inhalation exposure to Cr(VI) and stomach cancer risk is inadequate to evaluate this relationship (IARC 2012; Cole and Rodu 2005).

A recent meta-analysis (Welling *et al.* 2015) of 56 cohort and case-control occupational studies analyzed data for both lung cancer and stomach cancer. The summary relative risk of stomach cancer was higher when the analysis was limited to those studies in which an increased risk of lung cancer was observed, as compared to the summary relative risk for all studies combined. The authors concluded that current evidence is suggestive that Cr(VI) is a stomach carcinogen in humans, consistent with tumour results reported in rodent studies.

## 4.9 Summary: Health Effects of Cr(III) and Cr(VI)

Limited information is available on the effects of chromium in people from an acute perspective (see Section 4.4). Lethality due to cardiovascular effects (severe hypovolemia) has been associated with high Cr(VI) intakes. Other acute effects from Cr(VI) ingestion include gastrointestinal tract, kidney and respiratory system effects. Sensitization and allergic contact dermatitis have been reported in workers and the general population. Little information is available on acute toxic effects of Cr(III); however, contact dermatitis has been reported in the general population.

Most subchronic and chronic chromium toxicity studies have involved Cr(VI) rather than Cr(III) (see Section 4.5). The most important studies to date are considered to be National Toxicology Program (NTP 2008) rat and mouse drinking studies involving subchronic and chronic Cr(VI) exposures and NTP (2010) rat and mouse dietary studies involving subchronic and chronic Cr(III) exposures. Generally speaking, toxicity studies have not found that chemical-related effects were associated with Cr(III) at the intake rates evaluated in most oral assays. On the other hand, Cr(VI) has been determined to cause gastrointestinal tract lesions in laboratory animals when administered via the diet.

In humans, non-neoplastic respiratory lesions and respiratory cancers have been reported in several epidemiological studies of workers exposed to airborne Cr(VI) (Sections 4.5 and 4.7). Non-cancer effects of the respiratory system include nasal lesions, throat irritation, rhinitis and decreased pulmonary function in chrome plating and chromate production workers. Various gastrointestinal effects (including gastric and duodenal ulcers and inflammation) may also be associated with inhaled Cr(VI), although hand-to-mouth transfer remains a possible exposure route for workers. Nevertheless, the key concern identified in the epidemiological studies was elevated lung cancer rates in chromate production workers. There has been no consistent indication that Cr(III) is carcinogenic via the inhalation route.

In animal studies, Cr(VI) was shown to be carcinogenic via the oral route. NTP (2008) found elevated gastric tumours in mice and rats chronically exposed to Cr(VI) via drinking water. NTP (2010) did not find any evidence of carcinogenic activity of Cr(III) administered via the diet. HC (2016), based on an analysis of evidence relevant to the Cr(VI) mode of action, considers the diffuse hyperplasia of the small intestine to be a precursor of tumour formation caused by Cr(VI).

With respect to effects on reproduction and development, Cr(VI) has been associated with effects in laboratory animals, both male (spermatogenesis and sperm motility) and female (ovarian development, steroidogenesis and pituitary hormone synthesis) (see Section 4.6). Two small epidemiological studies have suggested effects on sperm in male workers exposed to Cr(VI).

## 4.10 Toxicological Reference Values

The potency of chromium depends upon its oxidation state (Cr(III) vs. Cr(VI)) and the route of exposure. Although chromium is predominantly present as Cr(III) in many environmental media, it was considered prudent to develop $SoGQ_{HH}$ for both Cr(III) and Cr(VI).

For Cr(III), the following toxicological reference values (TRVs) were selected for the development of the $SoQG_{HH}$:

| | |
|---|---|
| Combined oral and dermal tolerable daily intake (TDI): | 1500 µg/kg bw/day |
| Inhalation tolerable concentration (TC): | 0.1 µg/m$^3$ |

For Cr(VI), the following TRVs were selected to be for the development of the $SoQG_{HH}$:

| | |
|---|---|
| Combined oral and dermal TDI: | 2.2 µg/kg bw/day |
| Inhalation tolerable TC: | 0.1 µg/m$^3$ |
| Inhalation unit risk (non-threshold effects): | $7.6 \times 10^{-2}$ (µg/m$^3$)$^{-1}$ |

The rationale for the selection of the TRVs is presented in the sections that follow.

### *4.10.1 Oral and Dermal Exposure to Cr(III)*

US EPA (1998d) provided a reference dose (RfD) based on a NOAEL in mice at the highest dose in a dietary study by Ivankovic and Preussman (1975). Based on this study, US EPA (1998d) established a NOAEL of 1468 mg Cr(III)/kg bw/day and then applied an uncertainty factor of 100 (10 for interspecies differences and 10 for intraspecies differences) and a further modifying factor of 10 (for database deficiencies, including lack of a non-rodent mammal study and concerns regarding reproductive impacts). The resulting US EPA (1998d) recommended reference dose (RfD) is 1500 µg Cr(III)/kg bw/day.

Although US EPA completed their assessment prior to the NTP (2010) chronic mouse and rat study, US EPA's Cr(III) oral TRV remains the most relevant of all major health agencies; at the current time, no other major agency has provided a TRV for evaluation of oral Cr(III) intake. Furthermore, the NTP (2010) studies reported no effects at any of the doses tested in mice and rats (their highest dose was 781 mg Cr(III)/kg bw/day) and, thus, does not provide any suggestion that the US EPA analysis is outdated from this perspective.

Overall, the TDI of 1500 µg Cr(III)/kg bw/day is considered to be protective of oral and dermal intakes; however, since Cr(III) may have a greater potency when airborne, a separate TRV is provided for the evaluation of inhalation intakes.

### *4.10.2 Inhalation Exposure to Cr(III)*

ATSDR (2012) provided a minimal risk level (MRL) of 0.1 µg/m$^3$ for protection against non-cancer effects resulting from inhalation exposures of intermediate duration. The MRL was developed for soluble Cr(III) particulates based on a subchronic (13-week) rat inhalation study by Derelanko *et al*. (1999). ATSDR (2012) estimated a LOAEL as a human effects concentration ($LOAEL_{HEC}$) of 0.04 mg Cr(III)/m$^3$. Applying an uncertainty factor of 300 (3 for interspecies differences when an HEC conversion was already applied; 10 for human variability; 10 for use of a LOAEL instead of a NOAEL), ATSDR (2012) estimated an MRL of 0.1 µg Cr(III)/m$^3$.

Although the ATSDR (2012) MRL was developed for exposures up to one year, it is considered to be the most appropriate TRV for evaluation of inhalation risks of Cr(III). The ATSDR MRL is similar to the chronic reference concentration (RfC) for Cr(VI) provided by US EPA (1998c), as discussed in Section 4.10.5. Thus, application of the ATSDR MRL essentially treats Cr(III) as if it were equipotent to Cr(VI) for non-cancer risk assessment. This is a conservative approach but considered appropriate based on the available evidence.

Overall, a TC of 0.1 µg Cr(III)/m$^3$ is considered to be protective against inhalation exposures.

### *4.10.3 Oral and Dermal Exposure to Cr(VI)*

HC (2016) derived a TDI of 2.2 µg Cr(VI)/kg bw/day for non-carcinogenic (threshold) effects related to Cr(VI) exposure via the oral route. Using the NTP (2008) mouse dataset and a critical effect of small intestine hyperplasia, a benchmark dose lower 95% confidence limit ($BMDL_{01}$) of 0.67 mg Cr(VI)/kg bw/day was estimated (Thompson *et al*. 2014; HC 2016). Under contract to Health Canada, Summit Toxicology (2014) then used a physiologically based pharmacokinetic (PBPK) model to convert the mouse $BMDL_{01}$ of 0.67 mg Cr(VI)/kg bw/day into a human equivalent dose (HED) of 0.054 mg Cr(VI)/kg bw/day. Applying an uncertainty factor of 25 (10 for intraspecies variability; 2.5 for interspecies differences where a PBPK model was applied to estimate an HED), HC (2016) estimated a TDI of 2.2 µg Cr(VI)/kg bw/day for protection against non-cancer effects.

HC (2016) concluded that the TDI of 2.2 µg/kg bw/day for Cr(VI) is protective against cancer effects. Although the NTP (2008) dataset indicated an increased rate of gastrointestinal tumours in exposed mice and rats, Health Canada, based on a mode of action analysis, considers the diffuse hyperplasia of the small intestine to be a precursor of tumour formation caused by Cr(VI). Consequently, since the HC (2016) TDI is protective against the critical effect of small intestine hyperplasia, it will also be protective against cancer effects. A separate TRV for carcinogenic effects via oral exposure was therefore not developed.

It is recognized that this TDI may be overly conservative to evaluate the dermal route (i.e., there is no evidence that dermal intake would contribute to the risk of small intestine hyperplasia); however, no dermal route TRV has been identified and the dermal route is considered to represent a relatively minor pathway (i.e., not very sensitive with respect to overall $SoQG_{HH}$ derivation).

Overall, the TDI of 2.2 µg/kg bw/day for Cr(VI) is considered to be protective against non-cancer and cancer effects via the oral and dermal routes.

### *4.10.4 Inhalation Exposure to Cr(VI) – Cancer Effects*

HC (EC and HC 1994) completed an evaluation of the carcinogenic potency of Cr(VI) *via* the inhalation route and estimated the tumourigenic concentration that may be associated with a 5% increase in tumour incidence (i.e., $TC_{05}$). The $TC_{05}$ was based on the Mancuso (1975) epidemiological study of lung cancer incidence among workers at the Painseville, Ohio chromate production plant. From this study, HC (EC and HC 1994) estimated the $TC_{05}$ for lung cancer at 4.6 µg/m$^3$ for Cr(total). Based on information from Mancuso (1975) suggesting 1/7$^{th}$ of the chromium at the chromate production plant was Cr(VI), HC (EC and HC 1994) estimated the $TC_{05}$ for lung cancer at 0.66 µg/m$^3$ for Cr(VI).

A $TC_{05}$ can be converted into an inhalation unit risk (IUR) according to the following equation:

$$\text{IUR } (\mu g/m^3) = \frac{0.05}{TC_{05}(\mu g/m^3)}$$

Thus, the IUR for Cr(VI) can be estimated as:

$$\text{IUR } (\mu g/m^3) = \frac{0.05}{(0.66\ \mu g/m^3)} = 7.6 \times 10^{-2} (\mu g/m^3)^{-1}$$

Consequently, an IUR of $7.6 \times 10^{-2}$ $(\mu g/m^3)^{-1}$ is considered equivalent to the EC and HC (1994) $TC_{05}$. This IUR corresponds to a risk-specific concentration (RSC) of $1.3 \times 10^{-4}$ µg/m$^3$ for an incremental lifetime cancer risk (ILCR) of 1 in 100 000 (i.e., RSC = ILCR ÷ IUR). The HC IUR is of the same order of magnitude as the US EPA (2010) IUR of $1.2 \times 10^{-2}$ $(\mu g/m^3)^{-1}$, which was also based on an analysis of the Mancuso (1975) data.

Later analyses of lung cancer risk among workers at the Painesville plant (Crump *et al.* 2003; Proctor *et al.* 2016) led to the derivation of an IUR of $9.78 \times 10^{-3}$ and $8.32 \times 10^{-3}$ $(\mu g/m^3)^{-1}$, respectively. The Crump *et al.* (2003) analysis is described in Section 4.8.2. These IURs correspond to RSCs of 1.0 to $1.2 \times 10^{-3}$ µg/m$^3$ for an ICLR of 1 in 100 000. The analysis by Proctor *et al.* (2016) used a similar methodology to Crump *et al.* (2003) but expanded the cohort size by including short-term workers and more recent workers to increase statistical power in the lower exposure range.

OEHHA estimated a range of IURs of $1.0 \times 10^{-2}$ to 2 $(\mu g/m^3)^{-1}$ based on its analysis of data from the Baltimore cohort (OEHHA 2011). This range corresponds to RSCs of $1 \times 10^{-3}$ and $5 \times 10^{-6}$ $\mu g/m^3$, respectively, for an ICLR of 1 in 100 000. Haney *et al.* (2014) also calculated IURs of $1.94 \times 10^{-3}$ and $2.56 \times 10^{-3}$ for the Painesville and Baltimore cohorts, respectively.

The two original studies (Painesville and Baltimore cohorts) exhibit various strengths and limitations. Depending on the models employed and the inclusion criteria, estimated IURs, even for a single cohort, vary over a range of three orders of magnitude. Based on the quality of the original study or the risk analysis methodology, no one cohort or single IUR estimate is viewed as clearly superior by authoritative public health agencies. The original HC IUR of $7.6 \times 10^{-2}$ $(\mu g/m^3)^{-1}$ was retained as it is considered to be appropriately conservative given the range of estimated cancer risks associated with Cr(VI) inhalation exposure.

### *4.10.5 Inhalation Exposure to Cr(VI) – Non-Cancer Effects*

For evaluation of non-cancer inhalation exposures, US EPA (1998c) developed an RfC of 0.1 $\mu g/m^3$ for Cr(VI) particulates. Using the Glaser *et al*. (1990) rat dataset, a critical effect of increased lactate dehydrogenase (LDH) in bronchioalveolar lavage fluid (BALF), and a regional deposited dose ratio (RDDR) of 2.16 to account for pharmacokinetic differences between rats and humans, Malsch *et al*. (1994) estimated a 95% lower confidence limit benchmark concentration of $3.4 \times 10^{-2}$ mg Cr(VI)/$m^3$ corresponding to a 10% relative increase in LDH in BALF ($BMC_{10}$). Applying a total uncertainty factor of 300 (3 for interspecies differences where a PBPK model has been applied to estimate an RDDR, 3 for use of a subchronic study, and 10 for intraspecies variability) to the $BMC_{10}$, Malsch *et al*. (1994) estimated an RfC of 0.1 $\mu g/m^3$, which was subsequently adopted by US EPA (1998c).

No other health agency has developed a chronic TRV for inhalation exposure. ATSDR (2012) provided an MRL of 0.3 $\mu g/m^3$ for protection against intermediate exposures to soluble chromium (i.e., 14 to 364 days) as well as an intermediate and chronic inhalation MRL of 0.005 $\mu g/m^3$ for exposure to Cr(VI) aerosols and mists, based on data from an occupational cohort. However, the US EPA value is preferred as it is associated with long-term exposure and with exposure to Cr(VI) particulate matter, which are more relevant to the application of the $SQG_{HH}$ as compared to shorter-term aerosol exposures.

Overall, a tolerable concentration (TC) of 0.1 µg Cr(VI)/$m^3$ is considered to be protective against non-cancer risks associated with inhalation exposures.

# 5. DERIVATION OF HUMAN HEALTH SOIL QUALITY GUIDELINES

Human health soil quality guidelines ($SoQG_{HH}$) are developed for agricultural, residential and parkland, commercial, and industrial land uses.

On the basis of the contrasting effects of the different chromium species on human health, separate $SoQG_{HH}$s were derived for Cr(VI) and Cr(III).

## 5.1 Protocol

For threshold effects, two key factors are considered in setting Canadian soil quality guidelines. First, it is recognized that, exclusive of hazardous waste sites or any other point source of pollution, everyone is exposed to a background level of contamination that cannot be avoided. For chromium, this background exposure[1] is attributed primarily to food (mainly as Cr(III)) and drinking water (mainly as Cr(VI)), except in breast-fed infants. In deriving soil guidelines for chromium based on threshold effects, the background EDI was deducted from the TDI. For non-threshold effects, background exposure was not accounted for because guidelines for the target incremental lifetime cancer risk (TILCR) refer to an additional cancer risk associated with the contaminated site.

Secondly, a multimedia approach to guideline development has evolved whereby guidelines for one medium are established recognising that guidelines for other media may also be required. Guidelines must be established in such a manner that total simultaneous exposure at the guideline levels for all media will not result in exposure which exceeds the TDI.

Therefore, to set soil guidelines for threshold contaminants, some portion of the residual TDI (TDI - EDI) must be attributed to each medium. As recommended by CCME (2006), 20% of the residual TDI for threshold effects was apportioned to each environmental medium, namely air, water, soil, food and consumer products.

For chromium, the adverse health effects differ depending on the route of exposure. For oral exposures, Cr (III) and Cr(VI) act only as threshold toxicants. For inhalation exposures, Cr(VI) acts as both a threshold and a non-threshold toxicant, while Cr(III) acts only as a threshold toxicant. Separate TRVs have been developed for Cr(III) and Cr(VI) threshold oral endpoints and Cr(III) threshold inhalation endpoints, while a non-threshold inhalation TRV was developed for Cr(VI). For this reason, separate SoQGs were developed for combined oral and dermal exposures, threshold effects from inhalation exposures, and non-threshold effects from inhalation exposures.

[1] Background exposure, accounted for by the estimated daily intake (EDI), should not be confused with background soil concentration (BSC). The BSC is included in all equations for determining SoQGs.

## 5.2 Estimated Daily Intake for Trivalent and Hexavalent Chromium

Estimated daily intakes (EDIs) for the Canadian population have been calculated based on chromium concentrations typically found in environmental media (see Section 2.5). In general, the EDI is an estimate of the total concurrent background exposure from all known or suspected sources via a multimedia exposure assessment for the average Canadian. It does not include exposures that may occur from a contaminated site, or activities that may result in increased exposure to substances that are not considered background (e.g., occupational exposure). The EDI calculation is illustrated in the following equation (CCME 2006).

$$EDI = \sum_{i=1}^{n} ED_i$$

The EDIs are expressed in units of µg/kg bw/day and are intended to represent the average exposure for the Canadian population. The general population was subdivided into five age classes: infants (birth to 6 months), toddlers (7 months to 4 years), school age children (5 to 11 years), teenagers (12 to 19 years) and adults (20 years and older). The media considered in calculating the EDI are ambient air, indoor air, indoor dust, soil, drinking water, food and breast milk. Consumer products were not included because data are very limited. The equation below illustrates the media and pathway-specific EDI calculation (CCME 2006).

$$\mathrm{ED_i} = \frac{\mathrm{C} \times \mathrm{CR} \times \mathrm{BF} \times \mathrm{EF}}{\mathrm{BW}}$$

Where:

$ED_i$ = exposure dose from pathway i (mg/kg-day)
C = contaminant concentration in medium (e.g., mg/L)
CR = media specific contact rate (e.g., L/day)
BF = bioavailability factor (unitless)
EF = exposure factor, which is the product of exposure frequency (events per year) and exposure duration (years per lifetime); unitless
BW = body weight (kg)

Concentrations of chromium in environmental media were obtained from governmental databases, scientific literature and grey literature, as outlined in Section 2.5, Appendix 1 and Appendix 2. Concentrations in the literature were identified as either Cr(T) or Cr(VI) (i.e., Cr(III) concentrations were generally not reported). On the basis of this literature review, the proportion of Cr(VI) to Cr(T) was estimated for each media (see Section 3.6). This allowed for the development of Cr(VI) concentration distributions based on Cr(T). Nevertheless, for the purposes of EDI calculations, the concentration of Cr(III) was considered to be equal to that reported for Cr(T) for all environmental media except drinking water, for which all chromium was assumed to be present as Cr(VI). This is considered to be reasonable and not overly conservative, as most Cr(T) in the environment is expected to be found in the Cr(III) form. Receptor characteristics and

intake rates for each age class were treated as probability distribution functions (PDFs) as described by HC (2010). PDFs were assumed to be lognormal, except for human breast milk intake and time spent outdoors. Due to limited data, a triangular distribution was used for human breast milk intake.

PDFs were generated for concentrations in environmental media, receptor characteristics and intake rates. These were used to generate EDI distributions by age group for each medium as well as a combined total EDI for all media and exposure routes. Receptor characteristics and intake rate distributions are presented in Appendix 3 and Appendix 4. Appendix 5 summarizes the EDIs for Cr(T) and Cr(VI) via all media for the five age groups.

The Cr(III) EDIs, assumed equal to Cr(T) EDIs, for adults, teenagers, children and toddlers, are 1.16, 1.21, 2.06, and 3.45 µg/kg bw/day (median values). Depending on whether infants are exclusively formula-fed or breastfed, or fed a mixture of breast milk, infant formula and table food, the Cr(III) EDIs for infants vary between 0.324 µg/kg bw/day (exclusively breastfed) and 4.04 µg/kg bw/day (non-breastfed).

The Cr(VI) EDIs for adults, teenagers, children and toddlers are 0.14, 0.14, 0.231 and 0.368 µg/kg bw/day (median values). Depending on whether infants are exclusively formula-fed or breastfed, or fed a mixture of breast milk, infant formula and table food, the Cr(VI) EDI for infants can vary between 0.0150 µg/kg bw/day (exclusively breastfed) and 0.443 µg/kg bw/day (non-breastfed).

Certain Canadian subpopulations may be exposed to higher levels of chromium, and naturally occurring high chromium concentrations in drinking water have been found at various locations. Consumption of such waters would be the most likely route for higher Canadian exposure. Consumption of food grown on soils containing high levels of chromium could also possibly increase exposure above the EDIs. In addition, people living near industrial areas associated with chromium emissions could be exposed to higher concentrations via their inhalation of ambient air. However, the current analysis suggests that next to food consumption, the direct soil contact pathways (incidental ingestion, inhalation and dermal contact) are small contributors to total chromium exposure.

## 5.3 Toxicity Reference Values Selected for Human Receptors

TRVs recommended for use in the derivation of $SoQG_{HH}$ for Cr(III) and Cr(VI) were selected based on the analysis of those proposed by major health agencies (see Section 4.9).

**Table 5. Recommended TRVs for the derivation of $SoQG_{HH}$ for Cr(III) and Cr(VI)**

| Species of chromium | Tolerable daily intake (TDI) (evaluation of oral and dermal intake) | Tolerable concentration (TC) (threshold effects: inhalation intake) | Inhalation unit risk (IUR) (non-threshold effects: inhalation intake) |
|---|---|---|---|
| Cr(III) | 1500 µg/kg bw/day | 0.1 µg/m$^3$ | Not applicable |
| Cr(VI) | 2.2 µg/kg bw/day | 0.1 µg/m$^3$ | 7.6 x 10$^{-2}$ (µg/m$^3$)$^{-1}$ |

## 5.4 Relative Absorption Factors for Chromium

RAFs are applied in two instances:

(1) when the critical toxicological study used to derive the TRV used a different exposure medium than that under consideration (e.g., food vs. soil) to account for the difference in contaminant absorption by the body from the two different media. They should be specific to the chemical form (e.g., Cr(VI) or Cr(III)).

(2) When the critical toxicological study used to derive the TRV used a different exposure route than that under consideration (e.g., oral vs. dermal).

Key Cr(VI) toxicity data were obtained from drinking water ingestion in animals (threshold and non-threshold effects), inhalation of chromium salts (threshold effects) in animals, and occupational inhalation exposure (non-threshold effects).

With respect to RAFs pertaining to the exposure medium, there is a lack of *in vivo* validation data on the bioavailability of Cr(III) and Cr(VI) adsorbed to soil particles (see Section 4.3.1.1). Thus, default RAFs of one (1) were used for both the oral and inhalation routes to derive the $SoQG_{HH}$. This assumes that there is no difference in Cr(III) or Cr(VI) bioavailability in soil relative to their bioavailability in the key studies used to derive the TRVs.

In the case of dermal exposure, there are no toxicity studies that could be used to derive a TRV specific to this route. A dermal RAF of 0.1 (based on HC 2012b) is used to derive the $SoQG_{HH}$ (see Section 5.5) for both Cr(III) and Cr(VI). This assumes that absorption via the skin is not greater than 10% of the absorption via ingestion.

## 5.5 Ingestion and Dermal Pathways

For dermal exposures, the oral TRV was applied, since no TRV has been derived specifically for this route. The CCME (2006) equation for the derivation of SoQGs for threshold chemicals was applied to the oral and dermal routes, as the oral TRVs for both Cr(III) and Cr(VI) are based on threshold effects only.

As soil ingestion and soil adherence to the skin depend more on discrete events or behaviours than on the number of hours spent on the site per day, no adjustment is made for daily exposures of less than 24 hours for the oral and dermal pathways. However, adjustments are made, in the case of commercial and industrial land uses, for exposure that is less than 365 days per year for threshold toxicants.

### *5.5.1 Agricultural and Residential and Parkland Land Uses*

To determine agricultural and residential and parkland soil guidelines, the toddler is the most appropriate receptor, due to a large exposure per unit mass.

In accordance with CCME guideline derivation procedures (CCME 2006), a guideline for soil ingestion and dermal contact, which applies to agricultural (residential use of farm property only) and residential and parkland soil is derived using the following equation:

$$\mathrm{SoQG_{DH}} = \frac{(\mathrm{TDI} - \mathrm{EDI}) \times \mathrm{SAF} \times \mathrm{BW}}{[(\mathrm{AF_G} \times \mathrm{SIR}) + (\mathrm{AF_s} \times \mathrm{SR})] \times \mathrm{ET}} + \mathrm{BSC}$$

Where:

$\mathrm{SoQG_{DH}}$ = direct human-health-based soil quality guideline (mg/kg)

TDI = tolerable daily intake by oral exposure = 1500 µg Cr(III)/kg bw/day (US EPA 1998d) (Section 4.10.1)

TDI = tolerable daily intake by oral exposure = 2.2 µg Cr(VI)/kg bw/day (HC 2016) (Section 4.10.3)

EDI = estimated daily intake = 3.45 µg Cr(III)/kg bw/day for toddler (Appendix 5)

EDI = estimated daily intake = 0.368 µg Cr(VI)/kg bw/day for toddler (Appendix 5)

SAF = soil allocation factor (20% by default) = 0.2 (CCME 2006)

BW = body weight for a toddler (16.5 kg) (CCME 2006)

$\mathrm{AF_G}$ = relative absorption factor from gut = 1 by default for both Cr(VI) and Cr(III)

SIR = soil ingestion rate for a toddler ($8 \times 10^{-5}$ kg/day) (CCME 2006)

$\mathrm{AF_S}$ = relative absorption factor for chromium across the skin (0.1 for Cr(VI) and Cr(III)) (Section 5.4)

SR = soil dermal contact rate for toddler = $6.88 \times 10^{-5}$ kg/day; (hand surface area of 0.043 $m^2$ × soil adherence factor of 0.001 kg/$m^2$/day) + (arm/leg surface area of 0.258 $m^2$ × soil adherence factor of 0.0001 kg/$m^2$/day) (all parameters from CCME 2006)

ET = exposure term 1 (unitless) = 1.0 (i.e., 7 d/wk, 52 wk/year assumed at the site (CCME 2006))

BSC = background soil concentration = 42 mg/kg for Cr(III) and 0.84 mg/kg for Cr(VI) (Section 2.5.4).

Therefore, applying the previously provided equation, the following $SQG_{DH}$ were estimated for agricultural, residential and parkland land uses:

- $SoQG_{DH}$ = 56 886, which is rounded up to 57 000 mg/kg Cr(III) in soil at agricultural and residential and parkland sites.
- $SoQG_{DH}$ = 70.43, which is rounded down to 70 mg/kg Cr(VI) in soil at agricultural and residential and parkland sites.

### *5.5.2 Commercial Land Use*

Commercial sites include such places as shopping malls and places of business. Access to the site is not restricted and, since some commercial properties may include daycare facilities, the critical receptor is the toddler. Commercial sites do not include any areas where manufacturing takes place or where individuals may reside.

Since access to commercial sites is assumed to be less than 24 hours, exposure assumptions are lower than for residential land use. Discretion should be used in employing the commercial land use classification—in scenarios where unrestricted 24-hour access by children or toddlers or residential occupancy by any individual are possible, the residential and parkland classification may be more appropriate.

The commercial land use calculation is the same as the agricultural and residential and parkland calculations, except for the ET, which is 0.66 (based on 5 days per week and 48 weeks per year). Therefore, applying the previously provided equation, the following $SoQG_{DH}$ were estimated for commercial land uses:

- $SoQG_{DH}$ = 86 256, which is rounded down to 86 000 mg/kg Cr(III) in soil at commercial sites.
- $SoQG_{DH}$ = 106.38, which is rounded up to 110 mg/kg Cr(VI) in soil at commercial sites.

### *5.5.3 Industrial Land Use*

Industrial lands typically have limited or restricted access such that adult occupational exposure will predominate. An example of industrial land use is a manufacturing plant. At industrial sites, children are assumed to be excluded as part of SoQG development. The most common exposure scenario is expected to be unintentional soil ingestion by an adult. The potential for off-site migration of substances (i.e., via soil and dust) should be evaluated for industrial land use scenarios. Exposure for an adult at an industrial site is assumed to be 10 hours a day, 5 days a week and 48 weeks a year.

The SoQG was estimated using the previously described equation and input assumptions described in Section 5.5.1, except for the following adult-specific values:

EDI = estimated daily intake = 1.16 µg Cr(III)/kg bw/day for adult (Appendix 5)
EDI = estimated daily intake = 0.14 µg Cr(VI)/kg bw/day for adult (Appendix 5)
BW = body weight for an adult = 70.7 kg (CCME 2006)
SIR = soil ingestion rate for an adult = $2 \times 10^{-5}$ kg/day (CCME 2006)
SR = soil dermal contact rate for an adult = $1.14 \times 10^{-4}$ kg/d: (hand surface area of 0.089 $m^2$ × soil adherence factor of 0.001 kg/$m^2$/day) + (arms surface area of 0.25 $m^2$ × soil adherence factor of 0.0001 kg/$m^2$/day) (all parameters from CCME 2006)
ET = exposure term = 0.66 [5 day/wk/7 × 48 wk/year/52 at the site (CCME 2006)].

Therefore, applying the previously provided equation, the following $SoQG_{DH}$ were estimated for industrial land uses:

- $SoQG_{DH}$ = 1 023 724, which is rounded down to 1 000 000 mg/kg Cr(III) in soil for industrial sites.
- $SoQG_{DH}$ = 1407, which is rounded down to 1400 mg/kg Cr(VI) in soil for industrial sites.

## 5.6 Inhalation Pathway (All Land Uses)

As discussed in Sections 4.10.4, 4.10.5 and 5.1, the Cr(III) and Cr(VI) inhalation TRVs are based on endpoints that are distinct from the oral route. Consequently, separate inhalation SoQGs were calculated. In the case of Cr(VI), it was necessary to consider both threshold and non-threshold TRV endpoints, but only the threshold TRV for Cr(III).

Cr(VI) and Cr(III) SoQGs for soil inhalation were developed by adapting the indoor air quality equations specified in CCME (2006) for both non-threshold and threshold substances.

The threshold SoQGs for Cr(III) and Cr(VI) based on inhalation of soil particles are calculated as follows:

$$\mathrm{SoQG_{DH-PI}} = \frac{\mathrm{TC} \times \mathrm{SAF}}{(\mathrm{DC} \times \mathrm{AF_L}) \times \mathrm{ET_1} \times \mathrm{ET_2}} + \mathrm{BSC}$$

(This is a mathematical re-arrangement of the CCME equation for threshold contaminants when the TRV is expressed as a tolerable concentration instead of a tolerable daily intake.)

Where:
$SoQG_{DH\text{-}PI}$ = direct human health-based soil quality guideline for particulate inhalation – threshold effects (mg/kg)
TC = tolerable concentration in air = 0.1 µg/$m^3$ for both Cr(III) and Cr(VI) (Section 4.9)
SAF = soil allocation factor = 20% by default (CCME 2006)

$AF_L$ = absorption factor from lung = 1 for both Cr(III) and Cr(VI) (100% assumed by default)
DC = dust concentration from re-suspension of soil = $7.6 \times 10^{-7}$ g/m$^3$ (HC 2021)
BSC = background soil concentration = 42 mg/kg for Cr(III) and 0.84 mg/kg for Cr(VI) (Appendix 2 and Sections 2.5.4 and 3.6.4)
$ET_1$ = exposure term 1 (unitless) = 0.66 for commercial and industrial land use (5/7 d/wk × 48/52 wk/yr at the site) (CCME 2006)
$ET_2$ = exposure term 2 (unitless) = 1 for residential land use–- 24/24 hr/day at the site; 0.42 for commercial and industrial land use–- 10/24 hr/day at the site (CCME 2006).

For Cr(VI), the $SoQG_{DH\text{-}PI}$ for inhalation of soil-borne particulates for protection of non-cancer risks is 26 317, rounded down to 26 000 mg/kg for agricultural and residential and parkland uses, and 94 790, rounded up to 95 000 mg/kg for commercial and industrial land uses.

For Cr(III), the $SoQG_{DH\text{-}PI}$ for inhalation of soil-borne particulates for protection of non-cancer risks is 26 358, rounded down to 26 000 mg/kg for agricultural and residential and parkland uses, and 95 831, rounded up to 96 000 mg/kg for commercial and industrial land uses.

The non-threshold Cr(VI) SoQG for inhalation of soil particles was calculated as follows:

$$SoQG_{DH-PI} = \frac{TILCR}{(DC \times UR \times AF_L) \times ET} + BSC$$

(This is a mathematical re-arrangement of the CCME equation for estimation of soil quality guidelines for non-threshold substances when the cancer potency factor is expressed as a unit risk.)

Where:
$SoQG_{DH\text{-}PI}$ = direct human health-based soil quality guideline for particulate inhalation—non-threshold effects (mg/kg)
TILCR = target incremental lifetime cancer risk ($1 \times 10^{-6}$ or $1 \times 10^{-5}$)
UR = unit risk = $7.6 \times 10^{-2}$ $(\mu g/m^3)^{-1}$ (Section 4.10.4)
$AF_L$ = relative absorption factor for lungs = 1 (100% assumed by default) (CCME 2006)
DC = dust concentration from re-suspension of soil = $7.6 \times 10^{-7}$ g/m$^3$ (CCME 2006)
ET = exposure term (unitless) =1 (i.e., continuous lifetime exposure for an individual)
BSC = background soil concentration = 0.84 mg/kg (Cr (VI)) (Section 2.5.6).

SoQGs are provided for incremental lifetime cancer risks (ILCR) of both $1 \times 10^{-6}$ and $1 \times 10^{-5}$. Given that the exposure period is greater than the likely latency period for most carcinogens, the CCME (2006) default ET for all land uses is one (1). The Cr(VI) inhalation $SoQG_{DH\text{-}PI}$ for soil-borne particulates for all land uses are 18 mg/kg (rounded down from 18.15 mg/kg) for an ILCR of $10^{-6}$ and 170 mg/kg (rounded down from 173.97) for an ILCR of $10^{-5}$.

A summary of SoQGs for soil particle inhalation (i.e., $SoQG_{DH\text{-}PIS}$) is provided in Table 6 for Cr(VI) and Table 7 for Cr(III) along with the overall SoQG for soil particle inhalation for each land use category.

**Table 6. Summary of human health soil quality guidelines for the inhalation of Cr(VI) soil particles**

| | Land use | | | |
|---|---|---|---|---|
| Target risk | Agricultural | Residential/parkland | Commercial | Industrial |
| **Separate inhalation Cr(VI) SoQGs protective against threshold or non-threshold effects[a] (mg/kg dw)** | | | | |
| Non-threshold ($1 \times 10^{-5}$) | 170 | 170 | 170 | 170 |
| Non-threshold ($1 \times 10^{-6}$) | 18 | 18 | 18 | 18 |
| Threshold | 26 000 | 26 000 | 96 000 | 65 000 |

[a] The SoQGs for soil particles are set at the lowest of the guideline values for the non-threshold and threshold endpoints. Since the non-threshold values for Cr(VI) at ILCRs of $1 \times 10^{-5}$ and $1 \times 10^{-6}$ are lower than the threshold values, the soil inhalation SoQGs are set at the non-threshold guideline values for all land uses.

**Table 7. Summary of human health soil quality guidelines for the inhalation of Cr(III) soil particles**

| | Land use | | | |
|---|---|---|---|---|
| Target risk | Agricultural | Residential/parkland | Commercial | Industrial |
| **Overall inhalation Cr(III) SoQG protective against threshold effects[a] (mg/kg dw)** | | | | |
| Threshold | 26 000 | 26 000 | 96 000 | 96 000 |

[a] Since Cr(III) is not considered to be carcinogenic (i.e. is a threshold toxicant) via the inhalation route, the SoQGs for soil particle inhalation are based on the threshold guideline values for all land uses.

## 5.7 Protection of Groundwater Use as a Source of Raw Water for Drinking

No guideline for the protection of potable groundwater ($SoQG_{PW}$) was derived because the procedure for derivation of $SoQG_{PW}$ is not applicable to inorganic substances (CCME 2006).

## 5.8 Guideline for Consumption of Produce, Meat and Milk

No information was identified suggesting that Cr(VI) or Cr(III) will demonstrate appreciable bioconcentration or biomagnification, and thus a check mechanism for produce, meat and milk was not carried out.

## 5.9 Guideline for Off-site Migration for Commercial and Industrial Land Uses

Exposure scenarios for commercial and industrial sites consider only on-site exposure. Transfers of contaminated soil from one property to another are possible by environmental routes such as wind and water erosion (CCME 2006).

The human health soil quality guideline for off-site migration ($SoQG_{OM-HH}$) refers to the concentration in soil eroded from a site that will raise the contaminant concentration in the receiving soil to the level of the agricultural SoQG within a specific time frame. The $SoQG_{OM-HH}$ was derived as follows:

$$SoQG_{OM-HH} = 14.3 \times SQG_{A-DH} - 13.3 \times BSC$$

Where:

$SoQG_{OM-HH}$ = Human-health-based soil quality guideline for off-site migration (mg/kg)

$SoQG_{A-DH}$ = Direct-contact human-health-based soil quality guideline for agricultural land use = 26 000 mg/kg for Cr(III) and 18 mg/kg or 70 mg/kg for Cr(VI), for an ILCR of $10^{-6}$ and $10^{-5}$, respectively

BSC = Background soil concentration = 42 mg/kg for Cr(III) and 0.84 mg/kg for Cr(VI) (Appendix 2 and Sections 2.5.4 and 3.6.4).

### *5.9.1 Hexavalent Chromium*

For jurisdictions where an ILCR of $1 \times 10^{-6}$ is applied, the Cr(VI) $SoQG_{OM-HH}$ is 250 mg Cr(VI)/kg (rounded up from 246.48 mg/kg), using an $SoQG_{DH-PI}$ of 18 mg Cr(VI)/kg (Section 5.6).

For jurisdictions where an ILCR of $1 \times 10^{-5}$ is applied, the Cr(VI) $SoQG_{OM-HH}$ is 990 mg Cr(VI)/kg (rounded down from 990.08 mg/kg), using an $SoQG_{A-DH}$ of 70 mg Cr(VI)/kg (Section 5.5.1).

### *5.9.2 Trivalent Chromium*

The $SoQG_{OM-HH}$ is 370 000 mg Cr(III)/kg, (rounded down from 371 789 mg Cr(III)/kg), using an $SoQG_{DH-PI}$ of 26 000 mg Cr(III)/kg (Section 5.6).

## 5.10 Final Human Health Soil Quality Guidelines

Human health SoQGs were derived for Cr(VI) and Cr(III) for agricultural, residential and parkland, commercial, and industrial land uses, based on incidental soil ingestion, dermal contact with soil and inhalation of soil particles. Cr(VI) and Cr(III) were assumed to behave as threshold substances via the ingestion, dermal contact and inhalation pathways, and Cr(VI) was also assumed to act as a non-threshold substance via the inhalation pathway. The SoQGs calculated for each land use are presented in Table 8 (Cr(VI)) and Table 9 (Cr(III)).

The overall human health soil quality guidelines ($SoQG_{HH}$) are set as the lowest of the human health guidelines and check values derived for the land use. Based on this, the overall $SoQG_{HH}$ for Cr(VI) for the ingestion and dermal contact pathways are 70 mg/kg for agricultural land use, 70 mg/kg for residential and parkland land use and 110 mg/kg for commercial land use. For

industrial land use, the $SoQG_{HH}$ is 18 mg/kg based on an incremental lifetime cancer risk of $1 \times 10^{-6}$, or 170 mg/kg based on a $1 \times 10^{-5}$ incremental lifetime cancer risk from inhalation exposures. For Cr(III), the overall $SoQG_{HH}$ of 26 000 mg/kg for agricultural and residential and parkland uses and 95 000 mg/kg for commercial and industrial land uses are all based on threshold effects from particle inhalation.

**Table 8. Human health soil quality guidelines: Cr(VI) (mg·kg dw$^{-1}$)**

| Pathway | Agricultural | Residential/parkland | Commercial | Industrial |
|---|---|---|---|---|
| $SoQG_{HH}$ | | | | |
| Threshold or $10^{-5}$ ILCR | 70 | 70 | 110 | 170 |
| Threshold or $10^{-6}$ ILCR | 18 | 18 | 18 | 18 |
| Direct contact | | | | |
| - Ingestion + dermal contact ($SoQG_{DH}$) | 70 | 70 | 106 | 1400 |
| - Inhalation[c] ($SoQG_{DH-PI}$) | | | | |
| Non-threshold | | | | |
| $10^{-5}$ ILCR | 170 | 170 | 170 | 170 |
| $10^{-6}$ ILCR | 18 | 18 | 18 | 18 |
| Threshold | 26 000 | 26 000 | 96 000 | 96 000 |
| Potable groundwater ($SoQG_{PW}$) | NC | NC | NC | NC |
| Consumption of produce, meat and milk ($SoQG_{FI}$) | NC | NC | | |
| Offsite migration ($SoQG_{OM-HH}$) | | | | |
| Threshold or $10^{-6}$ ILCR | | | 250 | 250 |
| Threshold or $10^{-5}$ ILCR | | | 990 | 990 |

**Notes:** NC = not calculated

**Table 9. Human health soil quality guidelines: Cr(III) (mg·kg dw$^{-1}$)**

| Pathway | Agricultural | Residential/parkland | Commercial | Industrial |
|---|---|---|---|---|
| Overall $SoQG_{HH}$ or $PSoQG_{HH}$ | | | | |
| Threshold | 26 000 | 26 000 | 86 000 | 96 000 |
| Direct contact | | | | |
| - Ingestion + dermal contact ($SoQG_{DH}$) | 57 000 | 57 000S | 86 000 | 1 000 000 |
| - Inhalation[c] ($PSoQG_{DH-PI}$) | | | | |
| Threshold | 26 000 | 26 000 | 96 000 | 96 000 |
| Potable groundwater ($SoQG_{PW}$) | NC | NC | NC | NC |
| Consumption of produce, meat and milk ($SoQG_{FI}$) | NC | NC | | |
| Offsite migration ($SoQG_{OM-HH}$) | | | 370 000 | 370 000 |

**Notes:** NC = not calculated

The $SoQG_{HH}$ provided above are considered protective at most sites; however, certain exposure pathways were not evaluated in the development of the $SoQG_{HH}$. Site specific conditions should be assessed to determine whether additional pathways need to be assessed.

In many circumstances, it may be possible to measure total chromium Cr(T) in soil and compare the result to the $SoQG_{HH}$ for Cr(III) because the majority of environmental Cr is expected to be present as Cr(III) compounds (Sections 2.2 and 3.6); however, analytical measurement of Cr(VI)

in soil is strongly recommended for any site potentially contaminated by activities involving Cr(VI). Conversely, where speciated data are available, Cr(III) data may be compared to the $SoQG_E$ for Cr(T) for the same reason.

With the above in mind, the $SoQG_{HHS}$ are considered protective of human health at most sites.

# 6. RECOMMENDED CANADIAN SOIL QUALITY GUIDELINES

According to the soil protocol (CCME 2006), both environmental and human health soil quality guidelines are developed for the following four land uses: agricultural, residential and parkland, commercial, and industrial. The lowest value calculated for both human and ecological receptors, for each of the four land uses, is recommended by CCME as the Canadian Soil Quality Guideline. The environmental soil quality guidelines developed in 1997 (Cr(T)) (CCME 1997) and 1999 (Cr(VI)) (CCME 1999) were considered along with the human health guidelines presented in Chapter 5 in making final recommendations for Canadian Soil Quality Guidelines for the protection of environmental and human health. The recommended Canadian Soil Quality Guidelines for the protection of environmental and human health are presented in Table 9 for Cr(VI) and in Table 10 for Cr(III). The previous soil quality guidelines for Cr(T) for the protection of human health (CCME 1997) are replaced by the soil quality guidelines for Cr(VI) and Cr(III) recommended in this document.

## Table 10. Canadian soil quality guidelines for hexavalent chromium (Cr(VI)) (mg·kg dw$^{-1}$)

| | Land use | | | |
|---|---|---|---|---|
| | **Agricultural** | **Residential/ parkland** | **Commercial** | **Industrial** |
| **Guideline**[a] | **0.4** | **0.4** | **1.4** | **1.4** |
| Human health guidelines and check values[b] ($SoQG_{HH}$) | | | | |
| ILCR $10^{-6}$ | 18[h] | 18[h] | 18[h] | 18[h] |
| ILCR $10^{-5}$ | 70[k] | 70[k] | 110[k] | 170[h] |
| Direct contact guideline | | | | |
| Ingestion and dermal ($SoQG_{DH}$) | 70 | 70 | 110 | 1400 |
| Particulate inhalation ($SoQG_{DH-PI}$)[c] | | | | |
| $10^{-6}$ ILCR | 18 | 18 | 18 | 18 |
| $10^{-5}$ ILCR | 170 | 170 | 170 | 170 |
| Threshold | 26 000 | 26 000 | 96 000 | 96 000 |
| Inhalation of indoor air check ($SoQG_{IAQ}$)[d] | NC | NC | NC | NC |
| Groundwater check (drinking water) ($SoQG_{PW}$)[e] | NC | NC | NC | NC |
| Produce, meat and milk check ($SoQG_{FI}$)[f] | NC | NC | - | - |
| Off-site migration check ($SoQG_{OM-HH}$) | | | | |
| Non-cancer and $10^{-6}$ ILCR | | | 250 | 250 |
| Non-cancer and $10^{-5}$ ILCR | - | - | 990 | 990 |
| Provisional environmental health guidelines and check values ($PSoQG_E$)[g] | 0.4 | 0.4 | 1.4 | 1.4 |
| Soil contact guideline | NC | NC | NC | NC |
| Soil and food ingestion guideline | NC | - | - | - |
| Nutrient and energy cycling check | NC | NC | NC | NC |
| Off-site migration check ($SoQG_{OM-HH}$) | - | - | - | NC |
| Groundwater check (aquatic life) | NC | NC | NC | NC |

**Notes:** NC = not calculated; $SoQG_E$ = soil quality guideline for environmental health; $SoQG_{HH}$ = soil quality guideline for human health. Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

[a] Data are sufficient and adequate to calculate an $SoQG_{HH}$ for this land use but only a provisional $SoQG_E$ ($PSQG_E$). Therefore, the soil quality guideline is the lower of the two (CCME 2006). $PSoQG_E$s are based on the direct contact guideline, as derived in 1999 (CCME 1999 update). The original chromium soil quality guideline derived in 1999 (based on $SoQG_E$ only) and the interim soil quality criteria (CCME 1997) are superseded by the chromium soil quality guideline herein.

[b] For an ILCR of 1 in 1 000 000, the $SoQG_{HH}$ is set at the direct contact particulate inhalation value ($SoQG_{DH-PI}$) for non-threshold effects for all land uses because these are the lowest of the human health guidelines and check mechanisms for this land use. For an ILCR of 1 in 100 000, the $SoQG_{HH}$ for agricultural, residential and parkland and commercial land uses is set at the direct contact guideline for ingestion and dermal exposures ($SoQG_{DH}$), while the $SoQG_{HH}$ for industrial land uses is set at the direct contact particulate inhalation value ($SoQG_{DH-PI}$) for non-threshold effects.

[c] The inhalation pathway was developed separately due to the different toxic effects of chromium via the different routes of exposure.

[d] Applies only to volatile compounds and is not calculated for non-volatiles.

[e] Applies to organic compounds and is not calculated for metal substances. Concerns about metal substances should be addressed on a site-specific basis.

[f] Not calculated. Concerns about metal substances should be addressed on a site-specific basis.

[g] Data are insufficient or inadequate to calculate any of the environmental health guidelines or check values. However, there are sufficient and adequate data to calculate provisional $SoQG_E$s. $SoQG_E$ for Cr(VI) taken from CCME (1997; 1999 update).

[h] Based on the $SoQG_{DH-PI}$ for inhalation exposures for non-threshold effects

[k] Based on the $SoQG_{DH}$ for oral and dermal exposures for threshold effects

## Table 11. Canadian soil quality guidelines for trivalent chromium (Cr(III)) or total chromium (Cr(T)) (mg·kg dw$^{-1}$)

| | Land use | | | |
|---|---|---|---|---|
| | **Agricultural** | **Residential/ parkland** | **Commercial** | **Industrial** |
| **Guideline**[a] | **52** | **52** | **87** | **87** |
| Human health guidelines and check values ($SoQG_{HH}$) Cr(III)[b] | 26 000[p] | 26 000[p] | 86 000[q] | 96 000[p] |
| Direct contact guidelines | | | | |
| Ingestion and dermal ($SoQG_{DH}$) | 57 000 | 57 000 | 86 000 | 1 000 000 |
| Particulate inhalation ($SoQG_{DH-PI}$)[c] | 26 000 | 26 000 | 96 000 | 96 000 |
| Inhalation of indoor air check ($SoQG_{IAQ}$)[d] | NC | NC | NC | NC |
| Groundwater check (drinking water) $SoQG_{PW}$[e] | NC | NC | NC | NC |
| Produce, meat and milk check ($SoQG_{FI}$)[f] | NC | NC | - | - |
| Off-site migration check ($SoQG_{OM-HH}$) | - | - | 370 000 | 370 000 |
| Environmental health guidelines and check values | | | | |
| $SoQG_E$ Cr(T)[g] | 64 | 64 | 87 | 87 |
| Soil contact guideline[h] | 64 | 64 | 87 | 87 |
| Soil and food ingestion guideline | NC[j] | - | - | - |
| Nutrient and energy cycling check | 52 | 52 | NC[i] | NC[i] |
| Off-site migration check ($SoQG_{OM-HH}$) | - | - | - | 91 |
| Groundwater check (aquatic life) | NC[m] | NC[m] | NC[m] | NC[m] |
| Canadian Soil Quality Guidelines for the protection of human health Cr(T) (CCME 1997)[n] | 220 | 220 | 630 | 2300 |

**Notes:** NC = not calculated; $SoQG_E$ = soil quality guideline for environmental health; $SoQG_{HH}$ = soil quality guideline for human health. Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

[a] Data are sufficient and adequate to calculate a $SoQG_E$ and $SoQG_{HH}$ for this land use. Therefore, the soil quality guideline is the lower of the two (CCME 2006). $SoQG_{HH}$ are derived for Cr(III), which dominates in most environmental media, except water. Soil concentrations of Cr(T) may be compared to the $SoQG_{HH}$ for Cr(III). $SoQG_E$ are based on the direct contact guideline, as derived in 1997 (CCME 1997). The original Cr(T) soil quality guideline (CCME 1997) (based on $SoQG_E$ only) and the interim soil quality criteria (CCME 1997) are superseded by the chromium soil quality guideline herein, which represents the lowest value between the $SoQG_E$ (Cr(T)) and the $SoQG_{HH}$ (Cr(III)).

[b] The $SoQG_{HH}$ is set at the direct contact ingestion and dermal value ($SoQG_{DH}$) for all land uses because these are the lowest of the of the human health guidelines and check mechanisms for this land use.

[c] The inhalation pathway was developed separately due to the different toxic effects of chromium via the different routes of exposure.

[d] Applies only to volatile compounds and is not calculated for non-volatiles.

[e] Applies to organic compounds and is not calculated for metal substances. Concerns about metal substances should be addressed on a site-specific basis.

[f] Not calculated. Concerns about metal substances should be addressed on a site-specific basis.

[g] $SoQG_E$ for Cr(T) taken from CCME 1997.

[h] The soil contact guideline is the geometric mean of the preliminary soil contact value (TEC or ECL) and the nutrient and energy cycling check for this land use.

[j] Data are insufficient or inadequate to calculate the food and soil ingestion guideline for this land use.

[m] Applies to organic compounds and is not calculated for metal contaminants. Concerns about metal contaminants should be addressed on a site-specific basis.

[n] CCME $SoQG_{HH}$ (Cr(T)) were developed in 1997 and published in 1999 (CCME 1999 update).

[p] Based on the $SoQG_{DH-PI}$ for inhalation exposures for threshold effects

[q] Based on the $SoQG_{DH}$ for oral and dermal exposures for threshold effects

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# APPENDIX 1. SUMMARY TABLES OF BACKGROUND CHROMIUM CONCENTRATIONS IN ENVIRONMENTAL MEDIA

## Ambient air

| Location | Year | Concentration ($ng/m^3$) | Range ($ng/m^3$) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Canada | 2003-2009 | 0.516±0.569<br>0.407 median | 0.0141-15.8 | NAPS monitoring network | EC 2013 |
| Canadian Artic | May-Jun. 1997 | | 0.15-0.32 | | Hoff and Barrie 1986 |
| Northwest | | | 0.6 | | Bowen 1979 |
| **Northwest Territories** | | | | | |
| Twin George | | | <0.6 | | Hoff and Barrie 1986 |
| **Ontario** | | | | | |
| Ontario | | | 3-4<br>3-11 | Rural and non-industrial areas, NAPS $PM_{10}$ data<br>Urban areas with local sources, NAPS $PM_{10}$ data | OMOE 2009 |
| Southern | 1986-1987 | | 3-20 | | EC 1991 |
| Western | | | 5-6 | | |
| Central | | | 3-1 | | |
| Windsor | Jun.-Jul. 1991 + Feb.-Apr. 1992<br>Jul.-Aug. 1992 | 1.6<br>4.8 | nd-41<br>2.4-7.7 | n=46<br>n=6 | Bell *et al.* 1994 |
| Windsor | 1993 | 0.55 geometric mean<br>0.65±0.38 mean | 0.12-1.59 | n=33; Cr(VI) | Bell and Hipfner 1997 |
| Northern | 1986-1987 | | 4-11 | | Bowen 1979 |
| **Alberta** | | | | | |
| Not indicated | 1997 | 1.08 $PM_{10}$<br>0.11 $PM_{2.5}$ | | 10 day study | Alberta Health 1998 |
| Jasper national | | | <0.7 | | Hoff and Barrie 1986 |

| Location | Year | Concentration ($ng/m^3$) | Range ($ng/m^3$) | Comments | Reference |
|---|---|---|---|---|---|
| Park | | | | | |
| **UNITED STATES** | | | | | |
| Hawaii | | 45-67 | | | Bowen 1979 |
| Elizabeth, New Jersey | summer 1999 + spring 2001 | 7.1 mean<br>1.5 median | | XRF analysis in $PM_{2.5}$; n=103 | Turpin *et al.* 2007 |
| Houston, Texas | summer 1999 + spring 2001 | 1.1 mean<br>0.6 median | | XRF analysis in $PM_{2.5}$; n=110 | |
| Los Angeles, California | summer 1999 + spring 2001 | 0.6 mean<br>0.4 median | | XRF analysis in $PM_{2.5}$; n=121 | |
| Hudson County, New Jersey | | 4.5 | 1.5–10 | background Cr(VI) | Scott *et al*. 1997 |
| Arizona | | | 178.7–450.6 | n=116; ICP-AES analysis. 97% <BDL | O'Rourke *et al.* 1999 |
| Hudson County, New Jersey | | 3.7±3.2 Cr(VI)<br>27±23 Cr(T) | | n=25; Industrial sites | Finley *et al*. 1993 |
| **OTHER COUNTRIES** | | | | | |
| Shetland Island and Norway | | 0.7 | | | Bowen 1979 |
| Continental Europe | | 1-140 | | | |
| Japan | | 20-70 | | | |
| Chaoa Chu Kang, Singapore | 2004 | 0.59 | | n=2. Over two-week period | Balasubramanian *et al.* 2007 |
| Antwerp, Belgium | NA | 1.2 | 0-4.85 | n=15. Concentration in $PM_{2.5}$ | Stranger *et al*. 2009 |

Study used to develop EDI

3819 **Indoor air**

| Location | Year | Concentration ($ng/m^3$) | Range ($ng/m^3$) | Comments | Reference |
|---|---|---|---|---|---|
| **Ontario** | | | | | |
| Windsor | summer 1992 | 1.1 | nd-3.2 | n=17; Non-smoking office ICP-MS analysi | Bell *et al.* 1994 |
| | | 4.5 | 2.1-9.1 | n=3; non-smoking & smoking hotel ICP-MS analysis | |
| | | 1.8 | 0.2-6.6 | n=22; non-smoking homes ICP-MS analysis | |
| | | 4.4 | 0.1-41 | n=15; non-smoking homes ICP-MS analysis | |
| | | 2.9 | 0.1-41 | n=37; non-smoking and smoking ICP-MS analysis | |
| Windsor | Jun.-Jul. 1991 + Feb.-Apr. 1992 | 2.5 | nd-41 | Summary (indoor at home, office, and commuting). n=46 | Bell *et al.* 1994 |
| Windsor | 1993 | 0.2 geom. mean 0.23±0.13 | 0.07-0.62 | n=33; pilot study to measure Cr(VI) | Bell and Hipfner 1997 |
| **Alberta** | | | | | |
| n/d | 1997 | 1.63 1.09 | | n=20; 10-day study $PM_{10}$ $PM_{2.5}$ | Alberta Health 1998. |
| **UNITED STATES** | | | | | |
| US | | 6.95±30.35 | 2.62-430.30 | | US EPA 2009 |
| Arizona | | | 223.4-291.3 | n=119; ICP=AES analysis; 99% <BDL | O'Rourke *et al*. 1999 |
| Elizabeth, New Jersey | summer 1999 + spring 2001 | 4 mean 0.8 median | | n=96; XRF analysis in $PM_{2.5}$ | Turpin *et al*. 2007 |
| Houston, Texas | summer 1999 + spring 2001 | 1.2 mean 0.5 median | | n=106; XRF analysis in $PM_{2.5}$ | |
| Los Angeles, California | summer 1999 + spring 2001 | 0.9 mean 0.5 median | | n=124; XRF analysis in $PM_{2.5}$ | |
| Hudson County, New Jersey | | 11.5 6 | 1-17 | Smoking and non-smoking homes near a chromate waste site | Lioy *et al.* 1992 |
| Hudson County, New Jersey | | 3±2.4 Cr(VI) 23±31 Cr (TI) | | n=19; Industrial sites | Finley *et al*. 1993 |
| Hudson County, New Jersey | | 1.2 | 0.2-3.8 | background chromium(VI) | Scott *et al*. 1997 |
| Hudson County, | 1990 | 1.2 | 0.38-3.3 | n=15; Cr(VI) measured indoors in uncontaminated | Falerios *et al*. 1992 |

| Location | Year | Concentration ($ng/m^3$) | Range ($ng/m^3$) | Comments | Reference |
|---|---|---|---|---|---|
| New Jersey | | | | homes | |
| Minneapolis/St. Paul, Minnesota | Apr.-Nov. 1999 | 1.4 | | n=235; ICP-MS analysis in $PM_{2.5}$ | Adgate *et al*. 2007 |
| Townson, Maryland | 1998 | 1.6<br>0.45 | | n=10; INAA analysis<br>Versatile Air Pollutant Sampler VAPS<br>Personal exposure monitors (PEMs), empty room | Graney *et al*. 2004 |
| Chicago, Illinois | Jun. 1994-Apr. 1995 | 1±3 | nd-8 | n=48; $PM_{2.5}$; 11samples >LOD; 10 SE Chicago homes over a 10-month period | Van Winkle and Scheff 2001 |
| **OTHER COUNTRIES** | | | | | |
| Chaoa Chu Kang, Singapore | 2004 | 0.79<br>1.44<br>1.71 | | n=2 in each location; two-week period<br>Living room<br>Master bedroom<br>Bedroom | Balasubramanian *et al*. 2007 |
| Antwerp, Belgium | NA | 0.75 | 0-2.7 | n=15; in $PM_{2.5}$ | Stranger *et al*. 2009 |
| | Study used to develop EDI | | | | |

## Indoor dust

| Location | Year | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Ottawa, Ontario | Winter 1993 | 86.7 mean | 33.5-330.3 | n=48 | Rasmussen *et al*. 2001 |
| Canada, urban centres | Winter, 2007-2010 | 117±112 | 0.5-2930 | n=1025 | Rasmussen *et al*. 2013 |
| **UNITED STATES** | | | | | |
| New Brunswick and Montgomery Township, New Jersey | | 49.5±44.8 (Summer)<br>68.7±86.7 (Fall) | | n=7 | Freeman 1995 |
| Hudson County, New Jersey | 1997 | 111±197 | | n=47; nitric acid digestion; ICP-AES analysis. Control Homes. 94th percentile ≈180 | Freeman *et al*. 1997 |
| Hudson County, New Jersey | Nov. 1996–Feb. 1998 | Wipe sample:<br>73±76: n=23 Visit 1 | | Homes previously classified high and low concentration from remediated, historically | Freeman *et al*. 2000 |

| Location | Year | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|
| | | 47±56: n=21 Visit 2<br>66±82: n=18 Visit 3<br>59±73: n=16 Visit 4<br>Deposition plate:<br>96±90: n=21 Visit 1<br>118±88: n=18 Visit 3<br>179±223: n=16 Visit 4 | | contaminated areas (chromate ore processing facilities). Data not significantly different from each other. | |
| Hudson County, New Jersey | 1992 | Wipe<br>76.8±89.4<br>Vacuum<br>54.1±60 | 3.7-230 | n=8; Control sites. Sulphuric and nitric acid digested, ICP-AES analysis. | Lioy *et al*. 1992 |
| Hudson County, New Jersey | 1992 | 127 | | Control population of study of dust and urine levels of residents living at chromate ore processing sites | Stern *et al*. 1992 |
| New Brunswick, New Jersey | 2008 | 4.6±7.9 µg/g<br>10±27.9 µg/m$^2$ | 0.05-56.6 µg/g<br>0.22-169.3 µg/m$^2$ | n=60; 20 homes (background information) | Stern *et al*. 2010 |
| Region V (Illinois, Indiana, Ohio, Michigan, Minnesota), Wisconsin, Arizona, Maryland | 1995-1997 | 85.7±156.5<br>46±21.2<br>79±160.2 | 0.7498-3050<br>15.88-134.7<br>0.8147-1345 | n=797; All surface<br>n=117; Floor<br>n=270; Indoor dust | Based on US EPA 2009 |
| Hudson County, New Jersey | 1990 | 104 | | Sulphuric and nitric acid digestion; ICP-AES analysis | Freeman *et al*. 1995 |
| Arizona | | 40.3 median | 5.6-134.7 | n=135; ICP-AES analysis; 5% <DL. | O'Rourke *et al*. 1999 |
| **OTHER COUNTRIES** | | | | | |
| Sydney, Australia | 1999 | 83.6 | 4.9-425 | n=82 | Chattopadhyay *et al*. 2003 |
| Sydney, Australia | 1997 and 1999 | 116±34<br>538±1082<br>661±1682 | 63-188<br>60-4290<br>56-5440 | n=9<br>n=16<br>n=10 | Davis and Gulson 2005 |
| Warsaw, Poland | May-Jul. 1997 | 3±49<br>10±37<br>106±37 | 17-268<br>32-68<br>36-202 | 63-125 µm<br>32-63µm<br>0-32 µm | Lisiewicz *et al*. 2000 |
| Bahrain | nd | 11±6.7 | 2-27 | n=76 | Madany *et al*. 1994 |
| East and West Germany | 1990-1992 | 79.9±63 | nd-1330 | n=3893 | Seifert *et al*. 2000 |

| Location | Year | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|
| Kayseri, Turkey | Apr. and Jun. 2002 | 41±19.7 | | n=27 | Turkoglu *et al*. 2004 |
| Muscat, Oman | Nov. 2002-Jan. 2003 | 34±14 | 11-87 | n=119 | Yaghi and Abdul-Wahab 2004 |
| | Study used to develop EDI | | | | |

## Outdoor dust (street dust)

| Location | Year | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Ottawa, Ontario | Winter 1993 | 43.3 mean | 14.7-71.7 | n=45 | Rasmussen *et al*. 2001 |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| **CANADA-WIDE (background)** | | | | | | | |
| | | Natural | Till | 42±45.5 | 1-764 | n=7398; Canada-wide | NRCan 2010*a* (Grunsky 2010a) |
| | | Natural | Till | 64.8±29.7 | 5.9-625 | n=3978; New Brunswick till data | NRCan 2010b (Grunsky 2010b) |
| Appalachian | | Natural | A-Horizon | 23 | | Acid dissolution and AAS | McKeague *et al*. 1979 |
| | | | B-Horizon | 35 | | | |
| | | | C-Horizon | 37 | | | |
| St. Lawrence Lowlands | | | | | | | |
| | | | A-Horizon | 48 | | | |
| | | | B-Horizon | 52 | | | |
| | | | C-Horizon | 51 | | | |
| Interior Plains | | | | | | | |
| | | | A-Horizon | 33 | | | |
| | | | B-Horizon | 48 | | | |
| | | | C-Horizon | 36 | | | |
| Appalachian | | Natural | | 33; n=45 | 10-100 | Geological Survey; $HF/HClO_4/HNO_3$ extraction; AAS analysis | McKeague and Wolnetz 1980 |
| Canadian Shield | | | | 19; n=12 | | | |
| St. Lawrence Lowlands | | | | 51; n=40 | | | |
| Interior Plains | | | | 38; n=34 | | | |
| Cordilleran | | | | 78; n=19 | | | |
| | | Natural | | Median=62<br>Mean=78<br>SD=79 | <0.5-2300 | n=12 477; compiled by GSC. $HClO_4/HNO_3$ extraction; ICP-AES or AAS INAA analysis | Rencz *et al*. 2006 |
| | | Podzolic soil | | <10 | | | |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| | | Basic rocks | | >100 | | | |
| | | Agricultural | | 14.3±8.5 | 10 (sand)-22 (clay) | n=4 | Frank *et al*. 1976 |
| Across Canada | | Natural | C-Horizon | 28±21 | 169 (max) | n=532; aqua regia digestion, ICP-MS or AES | Dodd *et al*. 2017 |
| British Columbia | | | | 17±19 | 69 (max) | n=10 | |
| Alberta | | | | 20±7 | 38 (max) | n=32 | |
| Saskatchewan | | | | 22±14 | 79 (max) | n=65 | |
| Manitoba | | | | 19±14 | 53 (max) | n=20 | |
| Ontario | | | | 24±18 | 145 (max) | n=110 | |
| Québec | | | | 35±25 | 139 (max) | n=39 | |
| New Brunswick | | | | 39±24 | 169 (max) | n=115 | |
| Nova Scotia | | | | 25±14 | 112 (max) | n=67 | |
| Prince Edward Island | | | | 22±5.8 | 31 (max) | n=9 | |
| Newfoundland and Labrador | | | | 34±24 | 166 (max) | n=66 | |
| Across Canada | | Surface Soil | 0-5 | 15±12 | 92 (max) | n=532; aqua regia digestion, ICP-MS or AES | Dodd *et al*. 2017 |
| British Columbia | | | | 14±12.5 | 49 (max) | n=10 | |
| Alberta | | | | 16±5.6 | 31 (max) | n=32 | |
| Saskatchewan | | | | 20±12.8 | 53 (max) | n=65 | |
| Manitoba | | | | 19±11.5 | 42 (max) | n=20 | |
| Ontarop | | | | 13±10.8 | 51 (max) | n=110 | |
| Québec | | | | 20±9.5 | 50 (max) | n=39 | |
| New Brunswick | | | | 11±11.2 | 76 (max) | n=115 | |
| Nova Scotia | | | | 13±12.9 | 92 (max) | n=67 | |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| Prince Edward Island | | | | 9.7±3.7 | 17 (max) | n=9 | |
| Newfoundland and Labrador | | | | 9.3±12.2 | 85 (max) | n=66 | |
| **Nova Scotia** | | | | | | | |
| | | Ortstein Humic Podzol Ae horizon | | 2 | | determined by acid dissolution and atomic absorption | McKeague *et al*. 1979 |
| **Newfoundland and Labrador** | | | | | | | |
| Western | | Enriched serpentine bedrock | | over 1000 | | | Roberts 1980 |
| **Québec** | | | | | | | |
| St Lawrence Lowlands Sector | | Natural | | 49±26 | 7-98 | n=144 | Choinière and Beaumier 1997 |
| Appalachians Sector | | | | 32±42 | 1-500 | n=1821 | |
| Thetford region | | | | 24±9.7 | 1-113 | n=5983 | |
| St Lawrence Lowlands region, Grenville Sector | | | | 13±12 | 1-326 | n=15 352 | |
| Superior and Rae Sectors | | | | 44±28 | 1-388 | n=3892 | |
| Labrador Trough Sector | | | | 44±45 | 1-540 | n=10 428 | |
| 76 major soil types from 12 agricultural regions | | Clay | Surficial soils | 82±17.6 | 7.9-110 | n= 532; Rural samples | Giroux *et al*. 1992 |
| | | Clay Loam | | 52.7±24.7 | | $HNO_3$/HCl extraction; ICP-AES analysis | |
| | | Loam | | 29.7±11.8 | | | |
| | | Sand | | 21.7±13.8 | | | |
| **British Columbia** | | | | | | | |
| Vancouver Island | | Natural | | 65; n=72 | 2.8-77 | 95th percentile values. $HClO_4/HNO_3$ extraction | BC MOE 2005 |
| Greater Vancouver | | | | 50; n=56 | 0.1-73 | | |
| Lower Mainland | | | | 40; n=64 | 0.1-173 | | |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| Southern Interior | | | | 60; n=72 | 7.8-91 | | |
| Kootenay | | | | 20; n=56 | 3.2-27 | | |
| Cariboo | | | | 65; n=24 | 10-78 | | |
| Skeena | | | | 50; n=82 | 13-61 | | |
| Omineca Peace | | | | 65; n=56 | 5.6-89 | | |
| Trail | | Sandbox | | 16.4±5.3 | 7-38 | $HNO_3/HClO_4/HCl$ extraction, ultrasonic nebulization; ICP-AES analysis. Lead smelting contaminated area | Kelly *et al*. 1991 |
| | | Park | | 16.9±4.0 | 13-35 | | |
| | | Residential | | 18.1±4.3 | 12-43 | | |
| Southwest side to Keithley Creek and northeast side to Rabbit Creek | 2003 | | 18-1.3 m | 47±66.8 | 11-105 | n=1560; ICP-AES analysis | Timmins 2005 |
| **Ontario** | | | | | | | |
| Throughout Ontario | | Rural parkland | | Mean=26<br>98th percentile=58 | 8 -78 | n=101 | OMOE 1993 |
| Rural area | | | | Cr(VI) provisional 98th percentile= 0.5 | 0.25-0.92 | | |
| Throughout Ontario | | Old urban parkland | | Mean=27<br>98th percentile=62 | 8 -82 | n=60 | |
| Urban area | | | | Cr(VI) provisional 98th percentile= 0.5 | 0.25–0.51 | | |
| Halton County, Ontario | 1982 | | 15 cm | 24 mean<br>23 median | 12-65 | n=252; composite sample from cultivated land. | Webber *et al*. 1987 |
| Southern Ontario | 1975-1976 | | A-horizon<br>B-horizon<br>C-horizon | 51.5±7.1 mean<br>54.7±17.6 mean<br>49.6±21.9 mean | | 6 agricultural watersheds. $HNO_3/HClO_4/HF$ digestion; AAS analysis | Whitby *et al*. 1978b |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| Sudbury | | | | | 4-75 | Mining and smelting region. n=73 | Dudka *et al*. 1995 |
| Welland | | | | >120 µm/L | | Cr(VI) waste leachate | OMOE 1991 |
| | | | | 8.6 µm/L | | Cr(III) refinery landfill leachate | |
| | | | | | 10-39 | Thermal generation station ash leachate | EC and HC 1994 |
| | | Agricultural | | 67 | | Full depth background site | OMOE 2009 |
| | | All other land uses | | 70 | | | |
| Essex County | | Rural | | 23 | 19-29 | n=18<br>$HNO_3$/HCl digestion; ICP-AES analysis | Gizyn 1994 |
| Southwestern | | Rural soil profiles from 6 watersheds | Ap Horizon | 53±19 | 18-88 | n=26. $HNO_3$/$HClO_4$/HF digestion; AAS analysis using flame atomisation | Whitby *et al*. 1978a |
| | | | B-Horizon | 55±24 | 10-88 | | |
| | | | C-Horizon | 49±25 | 18-95 | | |
| Windsor | | Urban | | 25 | 20-33 | n=12; $HNO_3$/HCl digestion; ICP-AAS analysis | Gizyn 1994 |
| Ottawa | | Urban yard | | 44.8±9 | 28.8-74.5 | n=51; HF/$HClO_4$/HCl digestion; ICP- MS analysis | Rasmussen *et al*. 2001 |
| Port Colborne | | Woodlots | 0-5; n=5 | | 16-50 | $HNO_3$/HF/$HClO_4$ digestion; AAS analysis | Kuja *et al*. 2000 |
| | | Urban area | 5-15; n=5 | | 16-51 | | |
| Sudbury | | Mining and smelting contami-nated area | | 49.3 | 17.9-135.8 | $HNO_3$/$HClO_4$ digestion; ICP-AES analysis | Dudka *et al*. 1995 |
| **New Brunswick** | | | | | | | |
| Belledune | | Contami-nated area | | | 40-120 | Lead smelting operations | Nriagu and Kabir 1995 |
| **Alberta** | | | | | | | |
| Buffalo Head Hills; K4B kimberlite | 2005 | | C-Horizon | 22.1±2.3<br>21.6 median | 18.3-28.3 | n=39. Aqua-Regia digestion; ICP-AES/MS analysis | Fenton *et al*. 2005 |
| | | | C-Horizon | 62±6<br>63 median | 50-75 | n=39. Four Acid digestion; ICP-AES/MS analysis | |
| | 2005 | | B-Horizon | 27.4±2.5<br>27.9 median | 19.7-32.4 | n=41. Aqua-Regia digestion; ICP-AES/MS analysis | |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| | | | B-Horizon | 748<br>75 median | 54-89 | n=41. Four Acid digestion; ICP-AES/MS analysis | |
| | 2005 | | Organic soil | 6.7±5.3<br>4.5 median | 2.6-27.1 | n=42. Aqua-Regia digestion; ICP-AES/MS analysis | |
| Northwestern | | Agricultural site | | | | HF/HClO4/HNO3 digestion; ICP-AES analysis | Soon and Abboud 1990 |
| | | | | 84±16 | 52-112 | n=24; High organic content | |
| | | | | 61±16 | 27-83 | n=28; Low organic content | |
| | | | | 88±14 | 65-113 | n=11; Subsurface | |
| Brenchmark site | | Urban area | 0-15cm | 0.01±0.003 | 0.005-0.027 | DTPA extractable. n=129 | Penney 2004 |
| | | | 15-30cm | 0.008±0.002 | 0.004-0.017 | | |
| **Manitoba** | | | | | | | |
| Manitoba | | Fine clay | | 83 median | | | Haluschak *et al*. 1998 |
| | | Coarse | | 25 median | | | |
| Southern Manitoba | | Lower Pempina escarpment | | | 81-104 | | |
| West Central Manitoba | | Porcupine escarp-ment | | 64 | | | |
| Red River - northern portions | | Heavy clay | | | 75-85<br>means | | |
| Immediately south of Riding Mountain | | | | 70 | | | |
| Southern Interlake region | | | | | 78-110 | | |
| Flin Flon | 2006 | | Surface | 51.6 | 13.7-172 | n=93 | Manitoba Conserva-tion 2007 |
| Flin Flon | | Humus | Underlain by Precambrian bedrock | 10<br>13 | | Smelting region. | McMartin *et al*. 1999 |

| Location | Year | Soil type | Sample depth | Concentration (mg/kg) | Range (mg/kg) | Comments | Reference |
|---|---|---|---|---|---|---|---|
| | | | Underlain by Paleozoic bedrock | | | | |
| Flin Flon | | Garden | | 50 max | | n=33. Smelting region. | Jones and Henderson 2006 |
| Thompson | 2001 | Urban | | 24 | 9-67 | n=62. Smelting region. | Jones and Phillips 2003 |
| **Saskatchewan** | | | | | | | |
| Creighton | 2006 | | Surface | 41 | 14-192 | n=13 | Manitoba Conserva-tion 2007 |
| Southwestern | | Agricultural site | Brown & Dark Brown zones | | 22-97 | n=341. $HNO_3/HF/HClO_4$ digestion; ICP-MS analysis | Mermut *et al*. 1996 |
| | | | Ap-, B-, or C-Horizon | | 22-103 | | |
| **UNITED STATES** | | | | | | | |
| US | | surficial soil former chromite-ore processing facility | 0-15 | 103±20<br>9.3±3.8 | | n=4; Cr(T)<br>n=2; Cr(VI) | Gargas *et al*. 1994 |
| North-East | | Under CCA-treated deck | | 26<br>42<br>59<br>23 | 16-73<br>27-68<br>31-154<br>16-33 | 2-year-old structure<br>5-year-old structure<br>8-year-old structure<br>15-year-old structure | Stilwell and Gorny 1997 |

Study used to develop EDI

3823 **Surface water (fresh)**

| Location | Year | Concentration (µg/L) | Range (µg/L) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Great Lakes | 2003-2015 | 0.08-0.33 | <0.005-3.67 | Unpublished water quality monitoring data from ECCC | ECCC 2017 |
| St Laurence River | | 1.16 | 0.07-0.78 | | |
| New Brunswick, Nova Scotia and Newfoundland and Labrador | | 0.16 | <0.01-14.7 | | |
| Manitoba | | 1.92 | 0.03-24.4 | | |
| Saskatchewan | | 0.61 | <0.005-29.2 | | |
| Alberta | | 0.85 | 0.01-70.4 | | |
| Lake Superior<br>Lake Huron<br>Lake Erie<br>Lake Ontario<br>Lake Michigan | Early 1980 | 0.091 (NF); 0.080 (F)<br>0.13 (NF); 0.11 (F)<br>0.39 (NF); 0.27 (F)<br>0.82 (NF); 0.77 (F)<br>0.68 (NF); 0.68 (F) | | | Rossman and Barres 1988 |
| Lake Ontario | 1993 | 0.58 (F); n=105 | 0.018-3.10 | Laser-exited atomic fluorescence spectrometer analysis. DL <0.1ng/L | Nriagu *et al*. 1996 |
| Lake Erie | 1993 | 0.14 (F); n=31 | 0.06-0.22 | | |
| Lake Superior | 1991 | 0.06 (F); n=47 | 0.03-0.1 | | |
| British Columbia Rivers | 1988 | 6.9 | 0.3-165 | 11 rivers across BC | EC 1989 |
| St. Lawrence River, near Wolfe Island, Ontario | 1977-1996 | | <DL-1 | 22% <DL. Annual median concentrations | EC 1997 |
| St. Lawrence River, Québec | 1986-1988 | 7.1 | 1.5-92 | | EC and HC 1994 |
| Don River, Ontario | 1987 | 11 | | | |
| Humber River, Ontario | 1987 | 8 | | | |
| Oshawa Creek, Ontario | 1987 | 8 | | | |
| Jock River, Ontario | 1987 | 7 | | | |

| Location | Year | Concentration (µg/L) | Range (µg/L) | Comments | Reference |
|---|---|---|---|---|---|
| Kaministiquuia River, Ontario | 1987 | | 10-38 | | |
| Mission River, Ontario | | 18 | | | |
| McKellar River, Ontario | | | 8-14 | | |
| Nova Scotia | 2004-2009 | 2.5 | <MDL-4 | n=12 samples >MDL; MDL=0.6-2 µg/L<br>Raw water samples, groundwater or surface water not specified | Nova Scotia Department of Environment and Labour 2010, cited in HC 2016 |
| Manitoba | 2009-2010 | 3 | <MDL-14 | n=220; 26 >MDL; MDL=1 µg/L<br>Raw water samples, groundwater or surface water not specified | Manitoba Water Stewardship 2010, cited in HC 2016 |
| Rivers and tributaries of the lower Arthabasca River, Alberta | 1997-2015 | 1.4 | 0.03-76.2 | Unpublished water quality monitoring data from the Regional Aquatics Monitoring Program | ECCC 2017 |
| **UNITED STATES** | | | | | |
| Rivers<br>Lakes | | | 1-30<br><5 | | OEHHA 2011 |

NF = non-filtered; F = filtered

3825 **Groundwater**

| Location | Year | Concentration (µg/L) | Range (µg/L) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Prince Edward Island | 2005-2010 | 6<br>8<br>23.4 | | n=7622; 3 samples >MDL; MDL=5µg/L | PEI Department of Environment, Energy and Forestry 2010, cited in HC 2016 |
| Nova Scotia | 2004-2009 | 2.5 | <MDL-4 | n=12 samples >MDL; MDL=0.6-2 µg/L<br>Raw water samples, groundwater or surface water not specified | Nova Scotia Department of Environment and Labour 2010, cited in HC 2016 |
| Manitoba | 2009-2010 | 3 | <MDL-14 | n=220; 26 >MDL; MDL=1 µg/L<br>Raw water samples, groundwater or surface water not specified | Manitoba Water Stewardship 2010, cited in HC 2016 |
| British Columbia | | | | | BC MOE 2021 |
| Lower Mainland Region1 | | 12 | | 95th percentile; Cr(T) | |
| Lower Mainland Region 2 | | 3.9 | | | |
| Thompson Okanagan | | 19 | | | |
| South Vancouver Island | | 3 | | | |
| Ontario | 2002-2007 | | 0.5-106 | Cr(T) data from Provincial GW Monitoring Information System. 2008 ON background = 11.4 µg/L (97.5%tile) | OMOE 2011 |
| Que.; Superior geological province | | 1.2 (geom. mean)<br>2.6 (geom. mean) | | precambiran granite<br>conglomeratic rocks | Choinière and Beaumier 1997 |
| **UNITED STATES** | | | | | |
| Mojave Desert | | | <0.1-60 µg/L | Total dissolved Cr from observation wells and public water supplies; Almost all was Cr(VI)<br>Highest Cr(VI) =36 µg/L in Sheep Creek, San Gabriel Mountain deposit groundwater. | Ball and Izbicki 2004 |

| Location | Year | Mean concentration (µg/L) | Range (µg/L) | Comments | Reference |
|---|---|---|---|---|---|
| **CANADA** | | | | | |
| Newfoundland and Labrador, Ontario, Saskatchewan | 1998-2009 | 1.49±340 | 0.15-240 | n=14 633 | HC 2012*a* |
| Canada | late 1980s | | 0.3 to 4.3 | | EC and HC 1994 |
| Canada | | <2 | <2 to 9 | Raw, treated and distributed drinking water from 70 Canadian municipalities; concentrations similar in all types of water. AAS analysis | Méranger *et al*. 1979; 1981 |
| Newfoundland and Labrador | 2004-2010 | 2 | <MDL-2.6 | n=1910; Means measured on values >MDL, MDL=1 µg/L | Newfoundland and Labrador Department of Environment and Conservation 2010, cited in HC 2016 |
| | | 2 | <MDL-1.3 | n=3946; Means measured on values >MDL, MDL=1 µg/L | |
| Prince Edward Island | 2005-2010 | | <MDL-23.4 | n=7622; 3 samples >MDL; MDL=5µg/L | PEI Department of Environment, Energy and Forestry 2010, cited in HC 2016 |
| Nova Scotia | 2004-2009 | 2.7 | <MDL-5 | n=118; 9 samples >MDL; MDL=1-2 µg/L | Nova Scotia Department of Environment and Labour 2010, cited in HC 2016 |
| Québec | 2005-2010 | 4 | | 17 005; 14 263 <DL; DL=0.1-30 µg/L; 11 samples >50 µg/L | MDDEP 2012, cited in HC 2016 |
| Ontario | 2009-2014 | 1.2 | <DL-41.3 | n=6101; means measured on samples >DL (n=2063); DL=0.6-5 µgL | OMOE 2014, cited in HC 2016 |
| Manitoba | 2009-2010 | 3 | <DL-1.3 | n=212; means measured on samples >DL (n=19); DL=1 µgL | Manitoba Water Stewardship 2010, cited in HC 2016 |
| Saskatchewan | 2002-2010 | 5.4 | <DL-29 | n=2013; means measured on samples >DL (n=253); DL=0.03-5 µgL | Saskatchewan Ministry of Environment 2010, cited in HC 2016 |
| British Columbia | 2004-2010 | <1 | <DL-5 | 645 facilities rom Abbotsford and Greater Vancouver Regional District | BC Ministry of Health 2010, cited in HC 2016 |
| Yukon | 2007-2010 | 0.7 | <DL-1.2 | n=22; means measured on samples >DL | Government of Yukon |

| Location | Year | Mean concentration (µg/L) | Range (µg/L) | Comments | Reference |
|---|---|---|---|---|---|
| | | | | (n=7); DL=0.02-5 µgL | 2010, cited in HC 2016 |
| Northwest Territories | 2010 | <DL | | n=53; all <DL; RDL=1-20 µg/L | Government of Northwest Territories 2010, cited in HC 2016 |
| **OTHER COUNTRIES** | | | | | |
| US | 1984-1996 | 23<br>17 median | 100-1100 | n=9604; Cr(T) detected in ≈9% of samples; DL=10 µg/L | OEHHA 2011 |
| US | | 1.8 | 0.4-8 | n=3834 tap water | ATSDR 2008 |
| US Arizona | | 0.3 | 0.2-144 | n=73; 40% <BDL; ICP-MS analysis | O'Rourke *et al*. 1999 |
| | | 1.1 | 0.3–65.7 | n=82; 22% <BDL; ICP-MS analysis | |

Study used to develop EDI

## Sediment

| Location | Year | Concentration (mg/kg dw) | Range (mg/kg dw) | Comments | Reference |
|---|---|---|---|---|---|
| Grand Desert Beach, Nova Scotia | | 10.2 | | | Samant *et al*. 1990 |
| Québec | | 31.8±41.9 | 1-500 | n=1821; Apalachians | Choinière and Beaumier 1997 |
| | | 24.4±9.67 | 1-113 | n= 5985; St Lawrence Lowlands Region | |
| | | 48.5±25.5 | 7-98 | n=144; St Lawrence Lowlands –St-Polycarpe Region | |
| | | 12.5±11.5 | 1-326 | n=15 352; Grenville | |
| | | 43.7±27.7 | 1-205 | n=3892; Sperior and Rae | |
| | | 43.7±44.9 | 1-540 | n=10 428; Labrador Trough | |
| | | | 9.3-40.3 | | |
| St. Lawrence River, Québec | 1992 | 62 | | | Carignan *et al*. 1994 |
| Lac St-Pierre, Québec | 2000-2007 | 35.1±31.7 | 4-208 | n=369 | Pelletier 2008 |
| St-Lawrence River, | | | 121-275 | | Loring *et al*. 1979 |

| Location | Year | Concentration (mg/kg dw) | Range (mg/kg dw) | Comments | Reference |
|---|---|---|---|---|---|
| Québec - lower estuary and open gulf | | | | | |
| St-Lawrence River, Québec - lower estuary | | 99±2.3 | | | |
| St-Lawrence River, Québec - upper estuary | | 92±3.1 | | | |
| St-Lawrence River, Québec - open gulf | | 87±18.1 | | | |
| Saguenay Fjord, Québec | | 83±10 | | | |
| St. Lawrence River, Québec | 1999-2002 | 46 | | n=30; sampling conducted in seaway; n=100; Lake Saint-Francois; n=60; between Sorel & Trois-Rivières; LD=1 mg/kg | Saulnier *et al*. 2006 |
| Ontario | 1975-1976 | | 26.9–62 (means) | Bottom sediment; agricultural watersheds in southern Ontario | Whitby *et al.* 1978a; 1978b |
| Ontario | 1975-1976 | | 5–58.3 | Suspended sediments (dissolved and particulate); agricultural watersheds in southern Ontario | |
| Detroit River, Ontario | | 1920 | | Contaminated – Industrial sources | Lum and Gammon 1985 |
| Detroit River, Ontario | 1982 | | 11.1-92.3 (means) | N=62; 31 soft sediment areas, 2 × 50 cm cores from each area. | Fallon and Horvath 1985 |
| Hamilton Harbour, Ontario | | 564 | | Contaminated and industrial sources | Nriagu *et al*. 1983 |
| Marks Bay, Ontario | 1987 | 22.9 | 15-30 | Background data | OMOE and MDNR 1992 |
| Welland River, Ontario | | 5120<br>10 | | Downstream from steel manufacturing plant<br>Upstream control site | Dickman *et al*. 1990 |
| Weaver Creek, British Columbia | 2003 | 198±109 | 124-278 | n=25; collected along upper Weaver Creek above Keithley Road | Timmins 2004 |

## Precipitation

| Location | Year | Precipitation | Concentration | Range (mg/kg dw) | Comments | Reference |
|---|---|---|---|---|---|---|
| Montreal, Québec | | Snow | Site 1: <4 µg/l<br>Site 2: 9±1 µg/l<br>Site 3: 10±2 µg/l | | | Landsberger and Jervis 1985 |
| Eastern Canadian shield | | | | 0.1-0.99 µg/l | | Barrie and Vet 1984 |
| Sudbury, Ontario | 1978-1980 | Rain | 4±2 µg/l | | n=110; Cr(T), background | Chan *et al*. 1984 |
| Great Lakes, US | | Rain | | | IADN stations in the US | Sweet *et al*. 1998 |
| - Lake Superior | | | 0.1±0.1 µg/l | | | |
| - Lake Michigan | | | 0.1±0.2 | | | |
| - Lake Erie | | | <0.1 | | | |
| Warren, Michigan | 1984–1985 | Snow | 0.6 µg/l | 0.1 4.6 µg/l | n=39; U. Michigan Biological Station; field surrounded by deciduous forest | Cadle *et al*. 1990 |
| Wilmington, North Carolina | 1999-2001 | Rain | 4.6±0.5 nM Cr(T)<br>2.2±0.4 nM particulate Cr<br>0.8±0.1 nM Cr(III)<br>1.2±0.2 nM Cr(VI) | | annual average | Keiber *et al*. 2002 |

## Biota used as human food

| Location | Year | Concentration (µg/g dw) | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|---|
| Yukon | 1994-2001 | 0.22±0.15 | | Moose kidney; n=384 | Gamberg *et al*. 2005 |
| | | 0.52±0.24 | | Moose liver; n=56 | |
| | | 0.256±0.09 | | Moose muscle; n=37 | |
| | 2002-2003 | | 0.90-2.81 (means) | Caribou kidney; n=89 | Gamberg 2004 |
| | 2002 | 0.80 | | Elk kidney; n=1 | |
| | 2002 | 0.79±0.87 | | Moose kidney; n=53 | |

| Location | Year | Concentration (µg/g dw) | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|---|
| | 2003 | 0.93±0.59 | | Moose kidney; n=43 | |
| | 2002 | 0.87±0.12 | | Mule deer kidney; n=3 | |
| Spain | 2000-2001 | 0.243 | 0.052-4.641 | Bovin liver; n=120 | Lopez Alonzo *et al.* 2004 |
| | | 0.054 | nd-1.583 | Bovin kidney; n=120 | |
| | | 0.076 | nd-5.657 | Bovin muscle; n=120 | |
| US; Northeastern | | 0.19 | 0.03-1.46 | Fish from 167 Lakes | Yeardley *et al.* 1998 |
| US; Calcasieu River/Lake, Louisiana | | | <0.1-6.8 | Oysters, mussels, clams and mollusks | Ramelow *et al.* 1989 |

## Commercial food

| Food type | Concentration (µg/g dw) | | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|---|
| CANADA | | | | | |
| | | | | | |
| UNITED STATES | | | | | |
| Beverage | 22.9±103.1 | | 22.9-2618 | n= 684 | US EPA 2009 |
| Food | 63.9±98 | | 0.5-1988 | n=715 | |
| Food; Arizona | 49 | | 22-462 | n=159; 28% <BDL; Cr(T) | O'Rourke *et al*. 1999 |
| Beverage; Arizona | 7.4 | | 5.8-140 | n=154; 40% <BDL; Cr(T) | |
| | Cr(III) w.w. | Cr(VI) w.w. | | Estimated with diphenylcarbazide by permanganate oxidation. Some samples cooked in stainless steel. | Shroeder *et al*. 1962 |
| Beef liver | 0.1 | 0.12 | | | |
| Chicken breast | 0.06 | 0.10 | | | |
| Eggs | 0.19 | 0.03 | | | |
| Thyme | 3.38 | 0.41 | | | |
| Black pepper | 1.02 | 1.24 | | | |
| Tomatoes, stewed | 0.13 | 0.02 | | | |
| Corn oil | 0.41 | 0.61 | | | |
| Corn oil margarine | 0.14 | 0.0 | | | |

| Food type | Concentration (µg/g dw) | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|
| Dairy products | | <0.05-20 | Ranges of means of foods in the various categories; Cr(T) | Anderson *et al*. 1992 |
| Meat, poultry and fish | | 6-122 | | |
| Grain products | | 2-89 | | |
| Fruits and vegetables | | 2-118 | | |
| Condiments | | 2-145 | | |
| Miscellaneous prepared foods | | 3-82 | | |
| EUROPE | | | | |
| UNITED KINGDOM | | | | |
| Green vegetables | <LOQ | | Not indicated if Cr(T) or speciated | U.K. Food Standard Agency 2009 |
| Carcass meat | <LOQ | | | |
| Offal | <0.01 | | | |
| Meat product | 0.037 | | | |
| Poultry | <0.01 | | | |
| Fish | 0.04 | | | |
| Oil and fats | 0.02 | | | |
| Eggs | 0.01 | | | |
| Sugar and preserves | 0.08 | | | |
| Potatoes | 0.031 | | | |
| Other vegetables | 0.024 | | | |
| Canned vegetables | 0.039 | | | |
| Fruit products | 0.017 | | | |
| Beverages | <0.003 | | | |
| Milk | <0.003 | | | |
| Dairy products | <LOQ | | | |
| Nuts | <LOQ | | | |
| Bread | <0.02 | | | |
| Miscellaneous cereal | <LOQ | | | |
| SPAIN; Southern Tarragona Province | | | | |
| Radish root | 0.15±0.19 | | Cr(T) | Schuhmacher *et al*. 1993 |
| Potatoes | 0.03±0.01 | | | |
| Celery | 0.10±0.07 | | | |

| Food type | Concentration (µg/g dw) | | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|---|
| Onion | 0.02±0.01 | | | | |
| Leek | 0.05±0.05 | | | | |
| Chard | 0.04±0.04 | | | | |
| Spinach | 0.22±0.05 | | | | |
| Lettuce | 0.06±0.03 | | | | |
| Endive | 0.03±0.02 | | | | |
| Cabbage | 0.05±0.07 | | | | |
| Cauliflower | 0.08±0.07 | | | | |
| Tomato | 0.06±0.12 | | | | |
| Green pepper | 0.03±0.01 | | | | |
| Artichoke | 0.03±0.01 | | | | |
| Green bean | 0.06±0.07 | | | | |
| Eggplant | 0.01±0.01 | | | | |
| FRANCE | | | | | |
| | Cr(VI) µg/kg | Cr(III) µg/kg | | All Cr(VI) non-detect | Vacchina 2015 |
| Milk; cow, goat, soya | <1 | 6.2 | 4.0-8.3 | | |
| Milk, powdered | <10 | 14 | | | |
| Baby formula | <1 | 8.6 | | | |
| Yogurt | <10 | 13 | | | |
| Cheese | <10 | 45.5 | 29-62 | | |
| Tofu | <10 | 68 | | | |
| Flour; wheat, corn, rye, spelt | <10 | 31.9 | 16-63 | | |
| Bread | <10 | 74 | | | |
| Pasta | <10 | 52 | | | |
| Yeast | <10 | 167 | | | |
| Chocolate; dark, milk, white | <10 | 141 | 18-333 | | |
| Tea | <10 | 1199 | | | |
| Coffee | <10 | 27 | | | |
| Root vegetables | <10 | 194 | 22-516 | | |
| Fruit | <10 | 25 | 23-27 | | |
| Juice and wine | <10 | 42.4 | 14-65.5 | | |
| Eggs | <10 | 47 | | | |

| Food type | Concentration (µg/g dw) | | Range (µg/g dw) | Comments | Reference |
|---|---|---|---|---|---|
| Meat | <10 | 67.1 | 15-185 | | |
| Fish and seafood | <10 | 722 | 114-1857 | | |
| PORTUGAL | | | | | |
| | Cr(VI) | Cr(T) | 5.0-111.0; Cr(T) | Electrothermal atomization atomic absorption spectrometry (ETAAS) analysis. n=76 | Soares *et al*. 2010 |
| White bread | 5.65±5.44 | 47.3±20.0 | <5.60-18.80; Cr(VI) | | |
| Whole bread | 6.82±4.88 | 50.8±22.2 | 15.1-126.0; Cr(T) 0.16-0.14; Cr(VI) | | |
| | Cr(VI) µg/kg | Cr(III) µg/kg | | | Sykula-Zajac & Pawlak 2012 |
| White bread | 5.65±5.44 | 47.3±20.0 | | n=76 | |
| Whole bread | 6.82±4.88 | 50.8±22.2 | | n=76 | |
| | Study used to develop EDI | | | | |

## Human tissues and biological fluids

| Tissue/fluid | Population | Concentration | Range | Comment | Reference |
|---|---|---|---|---|---|
| Serum | Unexposed | 0.06±0.02 µg/L | 0.01-0.17 µg/L | | Sunderman *et al*. 1989 |
| Whole blood | Unexposed; n=5 | | 2.8-45 µg/L | Normal Cr in the general population (no documented exposure to non-dietary sources) | Iyengar & Woittiez 1988 |
| Serum | Unexposed; n=8 | 0.19 µg/L | 0.12-2.1 µg/L | | |
| Urine | Unexposed; n=7 | 0.4 µg/L | 0.24-1.8 µg/L | | |
| Milk | Unexposed; n=7 | 1.4 µg/L | 0.4-5.1 µg/L | | |
| Hair | Unexposed; n=20 | 460 µg/kg | 60-4100 µg/kg | | |
| Liver | Unexposed; n=8 | | 8-72 µg/kg | | |
| Lung | Unexposed | | 18-16 656 | 15 deceased individuals in Germany, including 8 smokers. No occupational exposure. (ng/g dw) | Raithel *et al*. 1988 |
| Lung | Unexposed | | | | Raithiel *et al*. 1989 |
| Lower lobes | | L=1275; R=1306 | 357-3172 (n=19) | Deceased individuals without lung cancer. n=7 individuals without exposure; n=19 individuals with potential | |
| Middles lobes | | L=nd; R=777 | 632-3616 (n=19) | | |
| Upper lobes | | L=1761; R=1248 | 632-5495 (n=19) | | |

| Tissue/fluid | Population | Concentration | Range | Comment | Reference |
|---|---|---|---|---|---|
| Hilar tissues | | L=12684; R=8342 | 2466-30 796 (n=19) | exposure (ng/g dw). | |
| Lung | Unexposed | | | n=30 non-occupationally exposed people. Smoking habits not documented (ng/g dw). | Raithel *et al*. 1993 |
| Lower lobes | | L=791; R=851 | | | |
| Middles lobes | | L=nd; R=742.4 | | | |
| Upper lobes | | L=1375; R=1078 | | | |
| Hilar tissues | | L=4611; R=3375; n=10 | | | |
| Serum | Unexposed | 0.15 | 0.12-0.20 | n=52 unexposed individuals from Ontario. GFAAS analysis. | Randall & Gibson 1987 |
| Serum | | 0.160±0.083 µg/L | 0.0382-0.351 | n=20; INAA analysis | Versieck *et al*. 1978 |
| Serum | Unexposed | <0.15 µg/L | | Non-industrially exposed | O'Flaherty *et al*. 2001 |
| Whole blood | Unexposed | | 0.2-0.3 µg/L | Unexposed individuals | ATSDR 2008 |
| Maternal blood | | 0.199 µg/L | 0.12-0.33 µg/L | High-resolution ICP-MS analysis. Control group. | Ziaee *et al*. 2007 |
| Umbilical cord blood | | 0.194 µg/L | 0.11-0.56 µg/L | | |
| Hair | Unexposed | | 50-100 ppm | | ATSDR 2008 |
| Urine | Unexposed | <10 µg/L | | Normal individuals; assuming no source of excessive exposure, urinary Cr 24-hour period. | |
| Urine | Exposed | | 40-50 µg/L | Sampled immediately after work shift to reflect exposure to 50 µg/m$^3$ soluble Cr(VI) compounds | |
| Blood | Exposed | | 387-4160 nmol/L | English strontium and lead chromate pigment factory workers | McAughey *et al*. 1988 |
| | Unexposed | | <20 nmol/L | | |
| Urine | Exposed | | 41-1250 (nmol/nmol creatinine) | | |
| | Unexposed | | <1 | | |
| Urine | Exposed mothers | <0.19 mg/L | <0.19-2.06 µg/L | Residents near a steel production factory in North Rhine-Westphalia, Germany. Ambient air range: 8.0-81.6 ng /m$^3$; n=210. Borken (control site) average=5 ng/m$^3$; | Wilhelm *et al*. 2007 |
| | Exposed children | 0.36 µg/L | <0.19-2.09 µg/L | | |
| | Unexposed children | | <0.19-1.38 µg/L | | |

| Tissue/fluid | Population | Concentration | Range | Comment | Reference |
|---|---|---|---|---|---|
| | | | | n=215. | |
| Urine | Unexposed postpartum women | 4.81±0.76 nmol/d<br>7.10±1 nmol/d | | | Anderson *et al*. 1993 |
| Breast milk | | 0.27 µg Cr/l | 0.12-0.53 | n=109; US mothers at various stages of lactation (0-28 days) | Casey *et al*. 1985 |
| Breast milk | United Arab Emirates | 0.689±0.517 µg/Ll<br>0.59 median | 0-2.527 | n=205 | Abdulrazzaq *et al*. 2008 |
| Breast milk | | 0.3±0.17 µg/L<br>0.27 median | 0.06-1.56 | n=255; Mature milk (2-48 wk postpartum) from Denver, CO | Casey & Hambidge 1984 |
| Breast milk | | 1.73 | 0.05-18.67 | n=79 | Yoshida *et al*. 2008 |
| Breast milk | | 3.43±0.39 nmol/L | | n=17 | Anderson *et al*. 1993 |
| Hair | Unexposed | 0.234 | | US; n=27M; 28F; 30-60 yr | Takagi *et al*. 1986 |
| | | 0.35 | | Canada; n=58M; 34F; 30-60 yr | |
| | | 0.27 | | Poland; n=24M; 22F; 30-60 yr | |
| | | 1.02 | | India; n=100M; 155F; 30-60 yr | |
| | | 0.23 | | Japan; n=228M; 229; 2-80 yr | |
| Fingernails | Unexposed | 0.52 | | US; n=34M; 37F; 30-60 yr | Takagi *et al*. 1988 |
| | | 0.82 | | Canada; n=21M; 19F; 30-60 yr | |
| | | 0.52 | | Poland; n=25M; 24F; 30-60 yr | |
| | | 1.4 | | Japan; n=125M; 127F; 2-80 yr | |
| | | 1.3 | | India; n=73M; 27F; 30-60 yr | |

Study used to develop EDI

# APPENDIX 2. TYPICAL ENVIRONMENTAL CONCENTRATIONS OF TOTAL CHROMIUM USED IN EDI CALCULATIONS[1]

| Media | Units | Distribution | Statistics | Cr(T) |
|---|---|---|---|---|
| Drinking water[2] | µg/L | Lognormal | Arithmetic mean (SD) | 1.49 (3.4) |
| | | | Minimum-maximum | 0-34 |
| Outdoor air[3] | µg /$m^3$ | Lognormal | Arithmetic mean (SD) | 0.000516 (0.000569) |
| | | | Minimum-maximum | 0-0.01 |
| Indoor air[4] | µg /$m^3$ | Lognormal | Arithmetic mean (SD) | 0.00243 (0.01535) |
| | | | Minimum-maximum | 0-0.12 |
| Surface soil[5] | µg /g | Lognormal | Arithmetic mean (SD) | 42 (45.5) |
| | | | Minimum-maximum | 0-401 |
| Settled dust[6] | µg /g | Lognormal | Arithmetic mean (SD) | 81.14 (136.33) |
| | | | Minimum-maximum | 0-1339 |
| Breast milk[7] | µg /L | TRI | Arithmetic mean (SD) | 0.59 (1.05) |
| | | | Minimum-maximum | 0-10 |
| Food[8] | µg /kg | Lognormal | Arithmetic mean (SD) | 63.9 (98) |
| | | | Minimum-maximum | 0-946 |

[1] See Section 3.6 for assumed fractionation of Cr(VI) and Section 5.2 for details on the methodology for estimating EDI.
[2] Estimated from average Cr concentrations in drinking water from Ontario (1998-2007), Saskatchewan (2000-2009), and Newfoundland and Labrador (2000-2009) (HC 2012a).
3 Outdoor air $PM_{2.5}$ concentrations from EC 2013 (HC 2012a).
[4] Weighted estimate, based on Adgate *et al*. 2007; Alberta Health 1998, Balasubramanian and Lee 2007; Bell *et al*. 1994; Finley *et al*. 1993; Graney *et al*. 2004, Stranger *et al*. 2009; US EPA 2009; Van Winkle and Scheff 2001 (HC 2012a).
[5] Estimated from Geological Survey of Canada data Grunsky 2010, (HC 2012a).
[6] Weighted estimate, based on Rasmussen *et al*. 2001, Chattopadhyay *et al*. 2003, Davis and Gulson 2005; Freeman *et al*. 1997; 2000; Lioy *et al*. 1992; Listewicz *et al*. 2000; Madany *et al*. 1994; Seifert *et al*. 2000; Stern *et al*. 1992; Turkoglu *et al*. 2004; US EPA 2009; Yaghi and Abdul-Wahab 2004 (HC 2012a).
[7] Weighted estimate, based on Abdulrazzaq *et al*. 2008; Anderson 1993; Casey *et al*. 1985; Casey and Hambridge 1984; Yoshida *et al*. 2008 (HC 2012a).
[8] Estimated from data from NHEXAS (US EPA 2009).

# APPENDIX 3. RECEPTOR CHARACTERISTICS OF THE CANADIAN GENERAL POPULATION[1]

| | Statistic | Breastfed Infant (0-6 m) | Non-breastfed infant (0-6 m) | Toddler (7 m-4 yr) | Child (5-11 yr) | Teen (12-19 yr) | Adult (20+ yr) |
|---|---|---|---|---|---|---|---|
| Body weight (kg) | Min-max | 2.8-21.5 | 2.8-21.5 | 7.1-35.9 | 14.2-71.5 | 30.0-112.2 | 38.1-126.5 |
| | Mean (SD) | 8.2 (2.9) | 8.2 (2.9) | 16.5 (4.5) | 32.9 (8.9) | 59.7 (13.5) | 70.7 (14.5) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Skin surface area; hands ($cm^2$) | Min-max | 242-416 | 242-416 | 299-614 | 396-863 | 556-1142 | 614-1262 |
| | Mean (SD) | 320 (30) | 320 (30) | 430 (50) | 590 (80) | 800 (100) | 890 (110) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Skin surface area: arms ($cm^2$) | Min-max | 200-1367 | 200-1367 | 396-1882 | 797-2645 | 1409-3465 | 1588-3906 |
| | Mean (SD) | 550 (180) | 550 (180) | 890 (240) | 1480 (300) | 2230 (340) | 2510 (360) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Skin surface area: legs ($cm^2$) | Min-max | 539-1496 | 539-1496 | 907-3012 | 1604-5655 | 3042-7945 | 3753-8694 |
| | Mean (SD) | 910 (160) | 910 (160) | 1690 (340) | 3070 (660) | 4970 (810) | 5720 (760) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Soil loading to exposed skin[2] ($kg/cm^2$/event) | Mean | | | | | | |
| Hands | | $1.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ |
| Surfaces other than hands | | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-8}$ |
| Time spent[3] outdoors (hr/d) | Min-max | 0-3 | 0-3 | 0-3 | 0-4 | 0.13-9.45 | 0.11-10.76 |
| | Mean/ mode (SD) | 1.25 | 1.25 | 1.25 | 2.2 | 1.42 (1.17) | 1.43m(1.28) |
| | Distribution | Triangular | Triangular | Triangular | Triangular | Lognormal | Lognormal |

[1] Mean receptor characteristics from Richardson (1997) and CCME (2006) unless otherwise stated.
[2] Soil loadings from Kissel *et al.* 1996; 1998 as referenced in CCME (2006).
[3] Time spent outdoors by infant, toddler or child is assumed to be equivalent to that of an adult if child or infant is assumed to be accompanied by an adult.

# APPENDIX 4. TYPICAL VALUES FOR INTAKES OF AIR, WATER AND SOIL BY THE CANADIAN GENERAL POPULATION[1]

| Intake rates[1] | Statistic | Breastfed Infant (0-6 m) | Non-breastfed Infant (0-6 m) | Toddler (7 m-4 yr) | Child (5-11 yr) | Teen (12-19 yr) | Adult (20+ yr) |
|---|---|---|---|---|---|---|---|
| Air inhalation ($m^3$/d) | Min-max | 1.1-4.4 | 1.1-4.4 | 4.6-15.6 | 8.3-25 | 9-28.9 | 9.5-33 |
| | Mean (SD) | 2.18 (0.59) | 2.18 (0.59) | 8.31 (2.19) | 14.52 (3.38) | 15.57 (4.0) | 16.57 (4.05) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Water ingestion[2] (L/d) | Min-max | - | 0.1-0.7 | 0.2-0.9 | 0.2-1.1 | 0.2-2.0 | 0.2-2.7 |
| | Mean (SD) | - | 0.3 (0.2) | 0.6 (0.4) | 0.8 (0.4) | 1 (0.6) | 1.5 (0.8) |
| | Distribution | N/A | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Soil ingestion[3] (kg/d) | | 2.00E-05 | 2.00E-05 | 8.00E-05 | 2.00E-05 | 2.00E-05 | 2.00E-05 |
| Soil inhalation[4] ($m^3$/d) | | 1.66E-09 | 1.66E-09 | 6.32E-09 | 1.10E-08 | 1.18E-08 | 1.26E-08 |
| Indoor settled dust ingestion (kg/d) | Min-max | 8.00E-08-1.77E-03 | 8.00E-08-1.77E-03 | 0.00-9.40E-04 | 0.00-8.33E-04 | 0.00-3.39E-05 | 0.00-6.20E-05 |
| | Mean (SD) | 3.74E-05 (8.33E-05) | 3.74E-05 (8.33E-05) | 4.06E-05 (5.22E-05) | 3.17E-05 (4.58E-05) | 2.07E-06 (2.32E-06) | 2.51E-06 (3.06E-06) |
| | Distribution | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |
| Food[5] (g/d) | Min-max | 0.5 - 1 L/d | 1.91E+02-3.01E+03 | 4.37E+02-4.02E+03 | 6.14E+02-4.72E+03 | 5.96E+02-6.18E+03 | 7.13E+02-6.57E+03 |
| | Mean/mode (SD) | 0.7 (Mode) | 8.38E+02 (4.07E+02) | 1.41E+03 (5.38E+02) | 1.81E+03 (6.34E+02) | 2.08E+03 (8.46E+02) | 2.32E+03 (8.86E+02) |
| | Distribution | Triangular | Lognormal | Lognormal | Lognormal | Lognormal | Lognormal |

[1] Probability distribution function curves for receptor intake rates from HC (2012a) unless otherwise stated.
[2] Breastfed infants assumed to be exclusively breastfed (6 m), no drinking water. Non-breastfed infants assumed to consume 0.3 L of drinking water, based on HC (2012c).
[3] Soil ingestion rates from CCME (2006).
[4] Soil inhalation rates based on Allan *et al*. 2008 and a $PM_{10}$ concentration of 0.76 µg/$m^3$ (CCME 2006).
[5] Breastfed infants are assumed to be exclusively breastfed for six months and non-breastfed infants are assumed to be fed a mixture of milk, formula and food.

# APPENDIX 5. ESTIMATED TOTAL DAILY TOTAL AND HEXAVALENT CHROMIUM INTAKE BY AGE CLASS FOR THE CANADIAN GENERAL POPULATION[1]

| Medium of exposure | Daily total and hexavalent chromium intake (µg/kg bw/day) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | BF[2]-infant (0-6 m) | | NBF[2]-infant (0-6 m) | | Toddler (7 m-4 yr) | | Child (5-11 yr) | | Teen (12-19 yr) | | Adult (20 yr+) | |
| **Air** | | | | | | | | | | | | |
| | Cr | Cr(VI) | Cr | Cr(VI) | Cr | Cr(VI) | Cr | Cr(VI) | Cr | Cr(VI) | Cr | Cr(VI) |
| Ambient air (inhalation) | 5.07E-06 | 9.99E-07 | 5.07E-06 | 9.99E-07 | 9.27E-06 | 1.86E-06 | 1.24E-05 | 2.46E-06 | 4.14E-06 | 8.15E-07 | 3.63E-06 | 7.22E-07 |
| Indoor air (inhalation) | 9.64E-05 | 1.93E-05 | 9.64E-05 | 1.93E-05 | 1.80E-04 | 3.63E-05 | 1.52E-04 | 3.07E-05 | 9.14E-05 | 1.80E-05 | 8.29E-05 | 1.66E-05 |
| **Drinking water** | | | | | | | | | | | | |
| Drinking water (ingestion) | n/a | n/a | 1.89E-02 | 1.88E-02 | 1.7E-02 | 1.67E-02 | 1.18E-02 | 1.19E-02 | 8.34E-03 | 8.4E-03 | 1.07E-02 | 1.07E-02 |
| **Indoor settled dust** | | | | | | | | | | | | |
| Settled dust (ingestion) | 8.27E-02 | 8.19E-03 | 8.27E-02 | 8.19E-03 | 6.66E-02 | 6.51E-03 | 2.36E-02 | 2.35E-03 | 9.78E-04 | 9.81E-05 | 9.66E-04 | 9.51E-05 |
| Settled dust (dermal) | 2.51E-02 | 2.48E-03 | 2.51E-02 | 2.48E-03 | 1.78E-02 | 1.77E-03 | 1.35E-02 | 1.35E-03 | 7.18E-03 | 7.30E-04 | 6.8E-03 | 6.78E-04 |
| **Soil** | | | | | | | | | | | | |
| Soil (ingestion) | 7.43E-02 | 1.4E-03 | 7.43E-02 | 1.48E-03 | 1.43E-01 | 2.86E-03 | 1.79E-02 | 3.59E-04 | 9.78E-03 | 1.94E-04 | 8.28E-03 | 1.64E-04 |
| Soil (inhalation) | 3.18E-07 | 6.44E-11 | 3.18E-07 | 6.44E-11 | 5.74E-07 | 1.39E-10 | 7.69E-07 | 2.82E-10 | 2.60E-07 | 1.54E-10 | 2.23E-07 | 1.44E-10 |
| Soil (dermal) | 1.72E-02 | 3.42E-04 | 1.72E-02 | 3.42E-04 | 1.22E-02 | 2.45E-04 | 9.32E-03 | 1.87E-04 | 4.97E-03 | 9.88E-05 | 4.69E-03 | 9.27E-05 |
| **Food** | | | | | | | | | | | | |
| Food, breastmilk, formula[3] (ingestion) | 2.71E-02 | 0 | 3.40 | 3.39E-01 | 2.89 | 2.91E-01 | 1.88 | 1.87E-01 | 1.15 | 1.15E-01 | 1.09 | 1.09E-01 |
| **Total** | | | | | | | | | | | | |
| **TOTAL EDI[4]** | **3.24E-01** | **1.50E-02** | **4.04** | **4.43E-01** | **3.45** | **3.68E-01** | **2.06** | **2.31E-01** | **1.21** | **1.40E-01** | **1.16** | **1.40E-01** |

[1] Median EDI values for each age class were modeled based on receptor characteristic details (see appendices 3, 4 and 6). Probability distribution functions of typical concentrations of air (indoor and outdoor), drinking water, indoor settled dust, soil, and food, based on details in Appendix 2. EDI probabilistic modeling completed as described in HC (2011 Draft). Median chosen as the EDI for the Canadian population.

[2] BF = breastfed; NBF = non-breastfed

[3] For the breastfed infant, Cr(VI) is assumed to be zero (see Section 3.6.6).

[4] Since a probabilistic method was used to develop the EDIs, the total EDI is not the sum of all sub-EDIs for each age category. The total EDI and each sub-EDI have individual probability distribution functions. The 50th percentile (median) for each distribution is displayed in the above table.

# APPENDIX 6. TYPICAL VALUES FOR AVERAGE BODY WEIGHTS AND INTAKES OF AIR, WATER AND SOIL BY THE CANADIAN GENERAL POPULATION USED IN SQG CALCULATION

| Age (years) | Body weight[1] (kg) | Air intake[2] ($m^3$/d) | Water intake[1] (L/d) | Soil intake[1] (g/d) | Soil inhalation[3,4] (g/d) | Settled indoor dust ingestion[5] (g/d) |
|---|---|---|---|---|---|---|
| **0-6 months** | 8.2 | 2.2 | 0.3 | 0.02 | 0.0000017 | 0.037 |
| **7 months to 4 years** | 16.5 | 8.3 | 0.6 | 0.08 | 0.0000063 | 0.041 |
| **5-11** | 32.9 | 14.5 | 0.8 | 0.02 | 0.000011 | 0.032 |
| **12-19** | 59.7 | 15.6 | 1.0 | 0.02 | 0.000012 | 0.0021 |
| **20+** | 70.7 | 16.6 | 1.5 | 0.02 | 0.000013 | 0.0025 |

[1] HC (2012c) and CCME (2006)
[2] Allan *et al.* (2008)
[3] HC (2012c)
[4] Air intake ($m^3$/d) x average airborne concentration of respirable particulate (0.00076 g/$m^3$)
[5] Wilson *et al.* (2013).

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| Industrial use | Chromium (III) | Chromium (VI) |
|---|---|---|
| Corrosion inhibitor | | Barium potassium chromate<br>Lithium chromate<br>Lithium dichromate<br>Magnesium chromate<br>Strontium chromate<br>Zinc chromate<br>Zinc sodium chromate<br>Zinc tetroxychromate |
| Metal finishing and lathing | Chromic chloride<br>Chromic fluoborate<br>Chromic fluoride | Cadmium dichromate<br>Calcium dichromate<br>Strontium chromate |
| Refractories | Magnesium chromite | Magnesium chromate<br>Magnesium dichromate |
| Catalysts | Chromic acetylacetonate<br>Chromic fluoride<br>Chromic nitrate<br>Cobalt chromite<br>Copper chromite<br>Zinc chromite | Cadmium chromate<br>Chromic chromate<br>Copper chromate<br>Magnesium dichromate<br>Nickel chromate<br>Silver chromate<br>Tetramino copper chromate |
| Paints and pigments | Chromic phosphate<br>Cobalt chromite | Barium potassium chromate<br>Cadmium chromate<br>Copper sodium chromate<br>Strontium chromate<br>Zinc sodium chromate |
| Leather tanning | Chromic chloride | |
| Timber industry<br>(Wood preservatives) | | Chrome copper arsenate (CCA)<br>Chrome zinc chloride<br>Copper dichromate |

| Industrial use | Chromium (III) | Chromium (VI) |
|---|---|---|
| Textiles, mordants and dyes | Chromic acetate<br>Chromic chloride<br>Chromic fluoride<br>Chromic lactate<br>Chromic naphthenate<br>Chromic nitrate<br>Chromic potassium oxalate | Chromic chromate |

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# APPENDIX 8. BIOACCUMULATION AND BIOCONCENTRATION OF CHROMIUM IN FRESHWATER BIOTA AND COMMON PRODUCE

| | Species | BCF/BTF | Reference |
|---|---|---|---|
| **Freshwater biota** | | | |
| Chromium (VI) | Fish muscle<br>Rainbow trout (*Oncorhynchus mykiss*)<br>American oysters (*Crassostrea vlrglnlca)*<br>Blue mussel (*Mytilus edulis*)<br>Polychaete worm (*Neanthes arenaceodentata*) | >1 L/Kg<br>1 L/Kg<br>125<br>192<br>200 | USEPA 1985 |
| | Rainbow trout (*Oncorhynchus mykiss*) | 1 | ATSDR 2000 in UKTAG 2007 |
| | Bivalve molluscs and polychaetes | 125-236 | US EPA 1980 in UKTAG 2007 |
| Chromium (III) | American oysters (*Crassostrea vlrglnlca)*<br>Soft shell crab (*Callinectes sapidus*)<br>Blue mussel (*Mytilus edulis*) | 116<br>153<br>86 | US EPA 1985 |
| | Oyster | 116 | US EPA 1985 in UKTAG 2007 |
| | Soft shell clam (*Mya arenaria*)<br>Blue mussel (*Mytilus edulis*) | 153<br>86 | US EPA 1980 inUKTAG 2007 |
| Total chromium | Terrestrial plant vegetative functions (leaves, stems, straw, etc.)<br>Terrestrial plant reproductive/storage functions (fruits, seeds, tubers, etc.) | 0.0075 kg soil/kg plant at 200 ppm soil Cr<br>0.0045 kg soil/kg plant at 200 ppm soil Cr | Baes *et al*. 1984 |
| | Wood mouse (*Apodemus sylvaticus*)<br>White-toothed shrew (liver) (*Crocidura russula*) | 0.71±0.05 µg/g (n=23)<br>2.31±0.16 µg/g (n=34) | Sanchez-Chardi and Nadal 2007*a*; *b* |
| | Tilapia (*Tilapia Zilli*)<br>Catfish (*Clarias gariepinus*) | 224<br>232 | Eneji *et al*. 2011 |
| | Rainbow trout (*Oncorhynchus mykiss*) | 3.1-21.3 (muscle, liver, kidney) | Calamari *et al*. 1982 |
| | Molluscs | 440 | ATSDR 2000 in UKTAG 2007 |

# APPENDIX 9. CR(III) ADEQUATE INTAKES

| Age group (sex) | Adequate intake (µg/day) |
|---|---|
| 0-6 months[a] | 0.2 |
| 7-12 months | 5.5 |
| 1-3 years | 11 |
| 4-8 years | 15 |
| 9-13 years (female) | 21 |
| 9-13 years (male) | 25 |
| 14-18 years (female) | 24 |
| 14-18 years (male) | 35 |
| 19-50 years (female) | 25 |
| 19-50 years (male) | 35 |
| >51 years (female) | 20 |
| >51 years (male) | 30 |
| Pregnant adult woman | 30 |
| Lactating woman[b] | 45 |

Source: IOM (2006)
[a] Based on 0.25 µg/L average Cr concentration in breast milk.
[b] Considered sufficient to replace the amount of Cr secreted in milk.