Screening Assessment for the Challenge

Vanadium oxide

(Vanadium pentoxide)

Chemical Abstracts Service Registry Number
1314-62-1

Environment Canada
Health Canada

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Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment of vanadium oxide, Chemical Abstracts Service Registry Number 1314-62-1. This substance was identified in the categorization of the Domestic Substances List (DSL) as a high priority for action under the Challenge. Vanadium oxide, or more specifically vanadium pentoxide, was identified as a high priority as it was considered to pose greatest potential for exposure of individuals in Canada and had been classified by the International Agency for Research on Cancer on the basis of carcinogenicity and by the European Commission on the basis of genotoxicity and developmental toxicity. The substance also met the ecological categorization criteria for persistence and inherent toxicity to aquatic organisms. Therefore, this assessment of vanadium pentoxide considers both environmental and human health risks.

According to information submitted under section 71 of CEPA 1999, between 1 000 000 and 10 000 000 kg of vanadium pentoxide was incidentally produced in Canada in the 2006 calendar year. Some importation activities were reported in the same year, at a total quantity of between 100 000 and 1 000 000 kg. Between 1000 000 and 10 000 000 kg of vanadium pentoxide was used in 2006.

Vanadium pentoxide is naturally-occuring in the environment. Measurements of vanadium in environmental media (ambient air, drinking water, surface water and sediment) and food in Canada and elsewhere capture total vanadium, including any vanadium pentoxide present. In Canada, the major uses of vanadium pentoxide are in the manufacture of ferrovanadium, as a catalyst in the production of sulphuric acid, in catalytic cracking applications and for catalytic reduction of nitrogen oxide and sulphur emissions from power plants. The major anthropogenic sources of vanadium pentoxide are the burning of certain fossil fuels.

Based principally on the weight of evidence–based assessments of international or other national agencies, a critical effect for characterization of risk to human health for vanadium pentoxide is carcinogenicity. Increased incidences of lung tumours were observed in male and female mice and in male rats following inhalation exposure. Genotoxicity was observed in both *in vivo* and *in vitro* assays with vanadium pentoxide in mammalian cells. Based on lung tumours observed in rats and mice for which modes of induction have not been fully elucidated, it cannot be precluded that vanadium pentoxide induces tumours via a mode of action involving direct interaction with genetic material.

Repeated-dose studies with vanadium pentoxide based on oral exposure showed effects in the spleen, liver, kidney and lung as well as on the immune system of rats.

On the basis of the carcinogenic potential of vanadium pentoxide, for which there may be a probability of harm at any level of exposure, it is proposed that vanadium pentoxide is a substance that may be entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

The releases of vanadium pentoxide into the Canadian environment are mainly atmospheric emissions from various industrial activities, in particular fossil fuel combustion. Deposition of the vanadium pentoxide to surrounding terrestrial and aquatic ecosystems then occurs. Because vanadium pentoxide is soluble, it will dissolve in contact with moisture in these ecosystems and yield a variety of dissolved vanadium species, depending on the environmental conditions. Vanadium has been demonstrated to have moderate to high acute and chronic toxicity to aquatic organisms and up to a high chronic toxicity to terrestrial organisms.

Site-specific industrial scenarios based on monitoring data were developed for the most important sources of release of vanadium pentoxide to the environment. Based on risk quotient analyses, harm to aquatic and terrestrial organisms resulting from exposure to vanadium pentoxide is unlikely. Hence, it is concluded that the substance is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends. Vanadium pentoxide meets the criteria for persistence but does not meet the criteria for bioaccumulation potential as set out in the *Persistence and Bioaccumulation Regulations* of CEPA 1999.

Based on the information available, it is concluded that vanadium pentoxide meets one or more of the criteria set out in section 64 of CEPA 1999.

Where relevant, research and monitoring will support verification of assumptions used during the screening assessment and, where appropriate, the performance of potential control measures identified during the risk management phase.

Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999) requires the Minister of the Environment and the Minister of Health to conduct screening assessments of substances that have met the categorization criteria set out in the Act to determine whether these substances present or may present a risk to the environment or to human health.

Based on the information obtained through the categorization process, the Ministers identified a number of substances as high priorities for action. These include substances that

- met all of the ecological categorization criteria, including persistence (P), bioaccumulation potential (B) and inherent toxicity to aquatic organisms (iT), and were believed to be in commerce in Canada; and/or
- met the categorization criteria for greatest potential for exposure (GPE) or presented an intermediate potential for exposure (IPE) and had been identified as posing a high hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity or reproductive toxicity.

The Ministers therefore published a notice of intent in the *Canada Gazette*, Part I, on December 9, 2006 (Canada 2006), which challenged industry and other interested stakeholders to submit, within specified timelines, specific information that may be used to inform risk assessment and to develop and benchmark best practices for the risk management and product stewardship of those substances identified as high priorities.

The substance vanadium oxide, or more specifically vanadium pentoxide, was identified as a high priority for assessment of human health risk because it was considered to present GPE and had been classified by other agencies on the basis of carcinogenicity, genotoxicity and developmental toxicity. The Challenge for this substance was published in the *Canada Gazette* on March 14, 2009 (Canada 2009). A substance profile was released at the same time. The substance profile presented the technical information available prior to December 2005 that formed the basis for categorization of this substance. As a result of the Challenge, submissions of information pertaining to the substance were received.

Although vanadium pentoxide was determined to be a high priority for assessment with respect to human health, it also met the ecological categorization criteria for persistence and inherent toxicity to aquatic organisms. Therefore, this assessment of vanadium pentoxide considers both environmental and human health risks.

Screening assessments focus on information critical to determining whether a substance meets the criteria as set out in section 64 of CEPA 1999. Screening assessments examine

scientific information and develop conclusions by incorporating a weight of evidence approach and precaution[1].

This final screening assessment includes consideration of information on chemical properties, hazards, uses and exposure, including the additional information submitted under the Challenge. Data relevant to the screening assessment of this substance were identified in original literature, review and assessment documents and stakeholder research reports and from recent literature searches, up to June 2010 for the human health and ecological sections of the document. Key studies were critically evaluated; modelling results may have been used to reach conclusions.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure (non-occupational) of the general population, as well as information on health hazards (based principally on the weight of evidence assessments of other agencies that were used for prioritization of the substance). Decisions for human health are based on the nature of the critical effect and/or margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. The final screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the proposed conclusion is based.

This final screening assessment was prepared by staff in the existing substances programs at Health Canada and Environment Canada and incorporates input from other programs within these departments. The ecological and human health portions of this assessment have undergone external written peer review/consultation. Comments on the technical portions relevant to human health were received from Ms. Joan Strawson, Toxicology Excellence for Risk Assessment; Dr. Pam Williams, E Risk Sciences; and Dr. John Christopher, CA OEHA. Additionally, the draft of this screening assessment was subject to a 60-day public comment period. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada. Approaches used in the screening assessment under the Challenge have been reviewed by an independent Challenge Advisory Panel.

The critical information and considerations upon which the final assessment is based are summarized below.

[1] A determination of whether one or more of the criteria of section 64 are met is based upon an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and the use of consumer products. A conclusion under CEPA 1999 on the substances in the Chemicals Management Plan (CMP) Challenge Batches 1-12 is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Controlled Products Regulations*, which is part of regulatory framework for the Workplace Hazardous Materials Information System [WHMIS] for products intended for workplace use.

Substance Identity

Vanadium oxide is a common name for vanadium pentoxide. As the pentoxide, vanadium exists in its +5 oxidation state. For the sake of clarity, the substance will be referred to as vanadium pentoxide in this screening assessment report, even though its name on the DSL is vanadium oxide. Information on the identity of vanadium pentoxide is summarized in Table 1.

Table 1. Substance identity

CAS RN	1314-62-1
DSL name	Vanadium oxide
National Chemical Inventories (NCI) names[1]	Vanadium oxide (AICS, ASIA-PAC, NZIoC, PICCS, SWISS, TSCA) Divanadium pentaoxide (ECL, EINECS) Vanadium pentoxide (ENCS, PICCS)
Other names	C.I. 77938; Divanadium pentoxide; Pentaoxodivanadium; Shcherbinaite[2]; UN 2862; UN 2862 (DOT); Vanadia; Vanadic anhydride; Vanadium oxide (V_4O_{10}); Vanadium(V) oxide
Chemical group (DSL stream)	Discrete inorganics
Major chemical class or use	Vanadium-containing inorganic compounds
Major chemical subclass	Oxides
Chemical formula	V_2O_5
Chemical structure	
SMILES[3]	O=[V](=O)O[V](=O)=O
Molecular mass	181.9 g/mol

[1] National Chemical Inventories (NCI). 2006. AICS, Australian Inventory of Chemical Substances; ASIA-PAC, Asia-Pacific Substances Lists; CAS RN, Chemical Abstracts Service Registry Number; DSL, Domestic Substances List; ECL, Korean Existing Chemicals List; EINECS, European Inventory of Existing Commercial Chemical Substances; ENCS, Japanese Existing and New Chemical Substances; NCI, National Chemical Inventories; NZIoC, New Zealand Inventory of Chemicals; PICCS, Philippine Inventory of Chemicals and Chemical Substances; SWISS, Swiss Giftliste 1 and Inventory of Notified New Substances; SMILES, simplified molecular input line entry specification; TSCA, *Toxic Substances Control Act* Chemical Substance Inventory.

[2] Hughes and Finger (1983).

[3] Simplified Molecular Input Line Entry System.

Physical and Chemical Properties

Table 2 contains experimental and modelled physical and chemical properties of vanadium pentoxide that are relevant to its environmental fate.

Table 2. Physical and chemical properties of vanadium pentoxide

Property	Type	Value	Temperature (°C)	Reference
Melting point (°C)	Experimental	690		O'Neil 2001
Boiling point (°C)	Experimental	1750		Woolery 2005
Density (g/cm^3)	Experimental	3.357	18	Woolery 2005
Vapour pressure (Pa)	Experimental	Negligible	–	NIOSH 2005
Henry's Law constant ($Pa \cdot m^3/mol$)	Estimated	Negligible	–	Expert judgement
Log K_{ow} (dimensionless)	Not applicable			
Log K_{oc} (dimensionless)	Not applicable			
Water solubility (mg/L)	Experimental	8000	20	IPCS 2001
pK_a (dimensionless)	Not applicable			
Log K_{sw} (dimensionless)	Experimental	1.88[1]	–	Harvey et al. 2007
	Experimental	Range:2.58–5.08[1] Median: 3.80[1]	–	Sheppard et al. 2007
Log K_{sdw} (dimensionless)	Experimental	2.28[1]	–	Harvey et al. 2007
Log K_{ssdw} (dimensionless)	Experimental	5.47[1,2]	–	Gobeil et al. 2005
		3.83[1–3]	–	Shiller and Boyle 1987
		3.15[1–4]	–	

Abbreviations: K_{oc}, organic carbon–water partition coefficient; K_{ow}, octanol–water partition coefficient; K_{sw}, soil–water partition coefficient; K_{sdw}, sediment–water partition coefficient; K_{ssdw}, suspended sediment–water partition coefficient; pK_a, acid dissociation constant.

[1] These partition coefficients are reported for dissolved vanadium, not vanadium pentoxide.

[2] Measured for a St. Lawrence River water sample; total suspended solids (TSS) = 12 mg/L; alkaline pH.

[3] Measured for a Mississippi River sample; TSS = 120 mg/L; pH = 7.8.

[4] Measured for a Mississippi River sample; TSS = 103 mg/L; pH = 7.7.

Vanadium is present in vanadium pentoxide in the oxidized pentavalent form. Vanadium pentoxide is very soluble in water. As vanadium pentoxide is a metal-containing inorganic substance, measurement of octanol–water and organic carbon–water partition coefficients (log K_{ow} and log K_{oc}) is not applicable. However, once vanadium pentoxide is dissociated, the fate of dissolved forms of vanadium may in part be characterized by other partition coefficients—namely, coefficients for the partitioning between water and each of soil, sediments and suspended sediments (log K_{sw}, log K_{sdw} and log K_{ssdw}). The magnitude of these partition coefficients, especially the one for soil–water, depends on the method used to estimate them—that is, adsorption of added metal or desorption of "native" metal already contained in the matrix. Lower values are usually expected for adsorption studies. The log K_{ssdw} values from Shiller and Boyle (1987) were calculated based on addition of metals to water samples containing suspended solids, while the value from Gobeil et al. (2005) was based on direct measurement of metals in water and suspended particulate matter (PM) from field samples. The log K_{sw} values from Sheppard et al. (2007) were based on desorption of metals contained in soil. Methodological information is not available for the Harvey et al. (2007) study.

The vapour pressure and Henry's Law constant for vanadium pentoxide are considered negligible. A detailed discussion of how physical and chemical properties influence the environmental fate of vanadium pentoxide, and of vanadium species in general, is presented later in this report.

Sources

Vanadium pentoxide is a naturally occurring substance mainly found at concentrations between 1.5% and 2.5% in titaniferous magnetite deposits worldwide (IPCS 2001). South Africa has the world's largest titaniferous magnetite deposit, with an average grade of 1.5% vanadium pentoxide, and produces nearly half of the global demand for high-purity (≥99.5%) vanadium pentoxide (Perron 2001; IARC 2006). The world's second largest deposit of titaniferous magnetite can be found in Canada, at Lac Doré in Quebec, with an average grade of 0.55% vanadium pentoxide. However, vanadium pentoxide is not mined at the Lac Doré site (Apella Resources 2009). In addition to the mining of titaniferous magnetite deposits, vanadium pentoxide is recovered from spent catalysts, petroleum residues and vanadium-bearing slag, the latter of which can contain up to 24% vanadium pentoxide (IPCS 2001). In 2004, worldwide production of vanadium pentoxide was approximately 86 200 tonnes (Woolery 2005).

Wind-blown continental dust, sea salt sprays and volcanic emissions are also natural sources of vanadium and are estimated to be responsible for releases of between 1.6 and 54.2 tonnes of vanadium into the atmosphere each year (Mamane and Pirrone 1998; Nriagu and Pirrone 1998). The amount of vanadium emitted to air from these natural sources is relatively small compared with global anthropogenic emissions to air, which are considerable at 70 000–210 000 tonnes per year (Hope 1994; Mamane and Pirrone 1998; Nriagu and Pirrone 1998). The contribution of vanadium pentoxide to the total emissions of vanadium is unknown, but it is expected that these natural sources of

vanadium pentoxide are insignificant in comparison with anthropogenic sources. Emission estimates based on data from the late 1990s indicate that the natural vanadium cycle may be heavily disturbed by human activities, with anthropogenic emissions to the atmosphere estimated to be 8.6 times higher than natural emissions of this metal on a global scale (2.4×10^5 vs. 2.8×10^4 tonnes per year: Nriagu and Pirrone 1998; Pacyna and Pacyna 2001).

In Canada, the major anthropogenic source of vanadium pentoxide is the burning of fossil fuels (Environment Canada 2009a). Vanadium is present in fossil fuels such as oil and coal, and the combustion of such fossil fuels leads to the formation of by-products that contain vanadium pentoxide, including solid residues, soot and fly ash. Crude oil and residual fuel oil contain vanadium at concentrations ranging from 3 to 260 µg/g, and from 0.2 to 160 µg/g, respectively (IPCS 2001). Coals from Alberta, British Columbia and Nova Scotia have average vanadium contents of 100 µg/g, 400 µg/g and 800 µg/g, respectively (Spectrum Laboratories Inc. 2009). Petroleum coke, which contains even higher vanadium levels, is increasingly being used as a full or partial replacement for coal in electrical power generation (Scott and Thomas 2007). The content of vanadium pentoxide in fly ash from co-combustion of coal and petroleum coke is approximately 3% (Scott and Thomas 2007). Vanadium pentoxide is also formed as a residue in spent catalysts used in petroleum refinery processes such as cracking and can be recovered. Metallurgy, particularly the production of ferrovanadium, may also contribute to the incidental release of vanadium pentoxide.

Vanadium pentoxide is not currently mined in Canada; therefore, its emission to the environment is mostly an incidental result of anthropogenic activities. According to information submitted in response to a notice published under section 71 of CEPA 1999, between 1 000 000 and 10 000 000 kg of vanadium pentoxide was manufactured in the 2006 calendar year (Environment Canada 2009a). Most submissions reported on vanadium pentoxide; however, there were a few submissions on releases of vanadium for which the respondents did not know whether it was in the form of vanadium pentoxide. The term “manufacture” as defined in the section 71 notice includes the incidental production of a substance at any level of concentration as a result of the manufacturing, processing or other uses of other substances, mixtures or products. The energy sector (i.e., petroleum refineries and electrical power generation plants) collectively incidentally produced about 75% of the vanadium pentoxide that was reported, mostly in combustion by-products such as fly ash, soot and bottom ash, but also as a residual in spent catalysts. The pulp and paper industry reported 15% of the total vanadium pentoxide that was produced incidentally through the burning of fossil fuels and wood fuels. About 9% was in the form of by-products from the production of ferrovanadium alloys and, to a lesser degree, other metals, while the remaining (about 1%) was produced in the manufacture of cement (Environment Canada 2009a). In addition to the vanadium pentoxide that was produced, between 100 000 and 1 000 000 kg of vanadium pentoxide was imported into Canada in the 2006 reporting year.

Uses

The main uses of vanadium pentoxide worldwide are as a formulation component in the production of metal alloys, particularly ferrovanadium, and as a catalyst in the production of sulphuric acid (Perron 2001). Other reported uses include its uses as a catalyst in the production of maleic anhydride for the manufacture of polyester and alkyd resins (Haber 2009), an electrolyte in vanadium redox batteries (Magyar 2003) and a pigment in the production of ceramics and glass (Motolese et al. 1993; Moskalyk and Alfantazi 2003; Vanitec 2009). Vanadium pentoxide is an additive that can be found at concentrations up to 1% in ultraviolet light–resistant tellurite glass (El-Mallawany 2001). It can be mixed with oxides of tungsten, titanium, silicon and other elements in various amounts to give other properties to glass (Blume and Drummond 2002; Kaoua et al. 2007). Vanadium pentoxide may also be employed for the catalytic reduction of industrial nitrogen oxide and sulphur emissions (Hagerman and Faust 1955; Vanitec 2009). Vanadium pentoxide can be found naturally in phosphate fertilizers as a result of phosphate rock processing (Mortvedt and Beaton 1995).

According to information submitted under section 71 of CEPA 1999, between 1 000 000 and 10 000 000 kg of vanadium pentoxide was used in Canada in 2006 (Environment Canada 2009a). The majority (92%) of the vanadium pentoxide was used in the production of ferrovanadium alloys for the manufacture of hardened steel. Vanadium pentoxide was also widely used as a catalyst: for manufacturing sulphuric acid at concentrations ranging between 3% and 9%, for catalytic cracking applications at a concentration of approximately 0.03% and for the selective catalytic reduction of nitrogen oxide and sulphur emissions from power plants at concentrations up to 1% (Environment Canada 2009a). Minor uses of vanadium pentoxide in Canada in the 2006 reporting year included its use as an oxidizing agent and for corrosion protection. A quantity of 38 300 kg of vanadium pentoxide was used in 2006 for chemical fertilizer manufacturing, and a quantity of 24 900 kg (present in alumina) was used for the production of aluminum. In the latter case, some vanadium which was presumed to be in the form of vanadium pentoxide was reported to be naturally present in alumina (Environment Canada 2009a).

Uses of vanadium pentoxide in consumer products were not identified. Vanadium pentoxide is not listed in the Drug Product Database (DPD) or the Therapeutic Product Directorate's internal Non-Medicinal Ingredients Database as a medicinal or non-medicinal ingredient in pharmaceutical drugs (DPD 2010; 2010 personal communication from Therapeutic Products Directorate, Health Canada, to Risk Management Bureau, Health Canada, unreferenced). Vanadium pentoxide was previously identified to be present as a medicinal ingredient in two final pharmaceutical products listed in the DPD, each product contains a total of 5 µg of vanadium pentoxide per tablet (DPD 2010). These products have since been discontinued (DPD 2010). Vanadium pentoxide is listed in the Natural Health Products Ingredients Database without a medicinal or non-medicinal role (NHPID 2010). The NHPID specifies that vanadium pentoxide will not be authorized for use in natural health products. Vanadium pentoxide is not listed in the Licensed Natural Health Products Database, thus is not present in any currently licensed

natural health products (LNHPD 2010). Additionally, the Natural Health Products Directorate multi-vitamin/mineral monograph does not indicate that vanadium pentoxide is a source of vanadium in natural health products (Health Canada 2007).

Releases to the Environment

As mentioned previously, the amount of vanadium pentoxide released from natural sources, such as continental dust, sea salt sprays and volcanic emissions, is expected to be limited in comparison with releases from anthropogenic sources. Vanadium pentoxide is not listed on the National Pollutant Release Inventory (NPRI 2009). However, the reporting of vanadium (CAS RN 7440-62-2) compounds except when in an alloy is required. According to data from the NPRI, 158 tonnes of vanadium compounds was released to the environment in 2006: 156 tonnes was released to air, 1 tonne to water and the remaining amount to land (NPRI 2009). Approximately 1800 tonnes of vanadium compounds was also sent off-site for either disposal or recycling, and a further 1369 tonnes was disposed of on-site. It is noteworthy that the majority of the reported releases were from oil refineries and electrical power generation plants, with minor contributions from the metallurgy and cement industries (NPRI 2009). This is consistent with information submitted under section 71, where oil refineries and electrical power generation plants were identified as the major producers of vanadium pentoxide, albeit through incidental production.

According to information submitted under section 71, the releases of vanadium pentoxide in Canada were mainly from the combustion of fossil fuels and wood fuels (Environment Canada 2009a). Most submissions reported on vanadium pentoxide, however there were a few submissions on releases of vanadium for which the respondents did not know whether it was in the form of vanadium pentoxide. For the purpose of this assessment, it was conservatively assumed that all vanadium reported under section 71 is in the form of vanadium pentoxide. Between 100 000 to 1 000 000 kg of vanadium pentoxide was reportedly released to air in the 2006 calendar year, together with approximately 8000 kg to water and a further 103 000 kg to land. Between 1 000 000 and 10 000 000 kg of vanadium pentoxide was transferred to an off-site waste management facility, about 99% of which was treated as non-hazardous waste. Most of the vanadium released into the atmosphere during combustion is in the form of vanadium pentoxide (i.e., V_2O_5) (Tullar and Suffet 1975), and lower oxides of vanadium will be ultimately oxidized to this compound (US EPA 1985).

A detailed description of each anthropogenic source of vanadium pentoxide releases is given below.

Fossil Fuel Combustion

Vanadium concentrations in fossil fuels depend on the source of the crude feedstock and the refining methods. Up to 50% of the vanadium present in crude oil is in the form of organometallic complexes with porphyrins; the remaining vanadium is in the form of

inorganic vanadium complexes (Crans et al. 1998). Porphyrin structures generally involve a vanadyl ion (VO^{2+}) surrounded by aromatic rings (Amorim et al. 2007); these bonds are weak enough to release the vanadium ion when heated (Rodgers et al. 2001).

In addition to consuming part of its domestic production of oil, Canada also consumes imported oil. Table 3 shows the quantity of crude oil consumed (i.e., refined and eventually burned) as well as its vanadium content, which varies depending on its source. An approximate estimate of the vanadium that could be released to the atmosphere or end up at waste management facilities (after capture by emission control equipment) as a result of crude oil combustion, calculated as the product of the estimated amount of crude oil used and its vanadium content, is provided in Table 3. To simplify the calculation, it was assumed that 100% of the oil produced and imported was consumed during the year.

Table 3. Origin of the crude oil used in Canada in 2006, and corresponding vanadium content

Source location	Vanadium content [mg/kg]	Quantity consumed in Canada[1] (millions of cubic metres)	Maximum quantity of vanadium potentially released or sent to waste management (tonnes)
Crude oil produced domestically			
Eastern Canada	39[2]	7 992	311 688
Western Canada (excluding oil sands)	50[3]	23 717	1 185 860
Canada's oil sands	640[4]	22 962	14 695 616
Crude oil imports			
Middle East	55[4]	20 028	1 101 518
North Sea	62[5]	18 224	1 129 900
Other countries	50[3]	5 998	299 910
Mexico	243[4]	2 109	512 390
Venezuela	1 180[4]	1 768	2 085 886
United States	134[4]	1 177	157 691
Total		103 975	**21 480 459**

[1] Based on data from Statistics Canada (2007a).
[2] Mossman (1999).
[3] IARC (2006) (default value).
[4] Crans et al. (1998).
[5] Fabec and Ruschak (1985).

Based on the calculations shown in Table 3, it is estimated that up to 21 million tonnes of vanadium could have been released or sent to waste management in 2006 as a result of crude oil consumption.

There are three types of coal mined in Canada: bituminous, sub-bituminous and lignite. These are mined mainly (97%) in western Canada (Natural Resources Canada 2009). Sixty-eight megatonnes of coal was mined in Canada in 2004, and the domestic coal

consumption was 59 megatonnes in the same year (Stone 2004). An estimate of the amount of vanadium that could be released or sent to waste management as a result of coal combustion could not be calculated, since the vanadium content of the three types of coal consumed in Canada was not found.

One potential area of uncertainty regarding the emission of vanadium from fossil fuel combustion is the form in which it is emitted. Several lines of evidence gathered from the published literature indicate that regardless of the original form of vanadium, it is mainly emitted in the form of an oxide, likely vanadium pentoxide (i.e., V_2O_5), as described below.

It can be expected that vanadium will be oxidized during fossil fuel combustion due to the relatively high temperatures and oxygen excess in furnaces (Davis 1997). The fly ash emitted from the combustion of residual fuel oil (i.e., heavy oil) may contain the following oxidized forms of vanadium: V_2O_3, V_2O_4, V_2O_5, $Na_2O{\cdot}V_2O_5$, $2Na_2O{\cdot}V_2O_5$, $3Na_2O{\cdot}V_2O_5$, $2NiO{\cdot}V_2O_5$, $3NiO{\cdot}V_2O_5$, $Fe_2O_3{\cdot}V_2O_5$, $Fe_2O_3{\cdot}2V_2O_5$, $Na_2O{\cdot}V_2O_4{\cdot}5V_2O_5$ and $5Na_2O{\cdot}V_2O_4{\cdot}11V_2O_5$ (National Academy of Sciences 1974). It appears that vanadium may be included in simple or complex forms of oxides; however, the relative importance of each form is unknown. In most of the oxides that include the pentoxide, the latter is weakly bound to another oxidized salt (National Academy of Sciences 1974).

Linak et al. (2000) showed that in furnaces where residual fuel oil is burned, the speciation of vanadium depends on the temperature. A temperature range of 720–1220°C in the furnace produces V_2O_5 species; above this range, increasing amounts of VO_2 and V_2O_4 are formed. Similarly, Pavageau et al. (2004) showed that vanadium pentoxide is the form in which vanadium is present in PM produced as a result of fossil fuel combustion. Their findings are based on experiments conducted in a pilot-scale combustion boiler at a high temperature (1100°C) and in excess of air. Combustion of coal occurs at relatively low temperatures (i.e., 760–930°C) (Perry and Green 1984). At these temperatures, Lee and Wu (2002) described a mixture of V_2O_5 and V_2O_4 based on modelled air stack emissions from coal combustion. This mixture contained less than 10% V_2O_4 and over 90% V_2O_5. Lower oxides (V_2O_3 and V_2O_4) are known to undergo further oxidation to V_2O_5 before leaving the stack (US EPA 1985).

On the other hand, a submission received from an oil-fired power plant in response to the section 71 survey indicated that only 10% of the vanadium in oil fly ash emitted by this plant was in the pentoxide form, and it was associated with sodium (NaV_6O_{15} and $Na_4V_2O_7$) (Environment Canada 2009a). Also, Huffman et al. (2000) conducted combustion experiments with four different fuel oils and, using X-ray absorption fine structure (XAFS) spectroscopy on the particulate emission samples, they concluded that most of the vanadium spectra closely resembled that of vanadyl sulfate ($VO{\bullet}SO_4{\bullet}xH_2O$). The combustion conditions in these experiments were not specified (e.g. temperature in the boiler, excess of oxygen).

Most of the vanadium pentoxide released to air as a result of fossil fuel combustion is associated with small-sized PM (0.06 to 3 μm; Mamane and Pirrone 1998; Linak et al.

2000). In addition to oxides, vanadium may also be associated with sulphates when sulphur concentrations in the fuel are high (Mamane and Pirrone 1998).

Overall, the lines of evidence described above suggest that vanadium pentoxide is one of the forms of vanadium emitted upon combustion of fuel oil and coal, depending on combustion conditions. As a worst-case scenario, it will be assumed in this assessment that 100% of the vanadium emitted by fuel oil (i.e., heavy oil) and coal combustion is in the form of vanadium pentoxide. Following deposition of atmospheric emissions containing vanadium pentoxide, the vanadium will enter the soil, surface water and groundwater (through leaching).

Petroleum Refining

There are 20 petroleum refineries in Canada (Gower et al. 2008), 7 of which reported to the NPRI in 2007 (NPRI 2009). Petroleum refining is the physical, thermal and chemical separation of crude oil into major distillation fractions. The distillation process separates hydrocarbons into a series of light to heavy fractions. These distillation fractions are further processed through separation and conversion into finished petroleum products. A petroleum refinery is the facility where this process takes place (US EPA 1995; CCME 2003). Since the boiling points of vanadium compounds are expected to be well above the temperature that exists at the bottom of the refining tank (400°C) vanadium compounds, like other metal-containing compounds, remain in heavier fractions, such as heavy oil and bitumen. Indeed, these fractions are the petroleum products that have the highest vanadium contents. For instance, fuel oil No. 6 (a type of heavy oil) has a vanadium content of 320 ppm mg/kg (National Bureau of Standards 1978), a level that is higher than those in most of the crude oils listed in Table 3.

Petroleum refining processes require fossil fuel combustion (e.g., the combustion of heavy oil) to supply the energy required for these processes. This can result in the production of fly ash. Hence, refineries do release vanadium pentoxide into the atmosphere (2009 e-mail personal communication from ExxonMobil to Ecological Assessment Division, Environment Canada; unreferenced). Based on the NPRI (2009), 101 tonnes of vanadium was released to air by petroleum refineries in Canada in 2007.

Oil Sands Exploitation

Although oil sands are rich in vanadium, the oil extraction process is not expected to result in releases of vanadium pentoxide to the environment. Most vanadium present in oil sands is as VO^{2+} bound to organic porphyrins (Crans et al. 1998). The hydrocarbon fraction is extracted from oil sands by a hot water process involving a temperature much below 700°C (Schramm et al. 2003). These conditions do not favour the formation of vanadium pentoxide, although the subsequent burning of the fuel oil will release vanadium pentoxide.

Much like refinery processes, the coking processes used by two Canadian facilities (Syncrude and Suncor) to upgrade the bitumen to produce synthetic crude oil require the

combustion of fossil fuels (e.g., petroleum coke or heavy oil) to supply the required energy. This will result in fly ash in which vanadium is concentrated to 2–3.5% (3.6–6.2% as vanadium pentoxide) (Holloway et al. 2005). Based on information collected under the section 71 survey, most of these fly ashes are removed from stack gases by electrostatic precipitators (Environment Canada 2009a) and then piled on-site before being recycled. Syncrude operations near Fort McMurray reported release of 6.4 tons of vanadium to air in 2006, while 290 tons where recycled off-site (NPRI 2009). In response to the section 71 survey, another company exploiting oil sands reported 1.6 and 4.5 tons of releases of vanadium to air and water, respectively, for one of their facilities (Environment Canada 2009a). Of the 258 tons of vanadium incidentally produced (fly ash) by this industry in 2006, the majority was piled on site for future recycling.

Overall, releases of vanadium pentoxide resulting from oil sands extraction and refining should be much less than the releases from fossil fuel combustion, as described above.

Metallurgy and Steelmaking

One of the ferrovanadium manufacturers that responded to the section 71 survey reported using vanadium pentoxide to make alloys. Since the chemical reaction to convert vanadium pentpentoxide to ferrovanadium takes place at 1800°C, the vanadium emitted through stacks is likely not in the form of vanadium pentoxide, according to the information presented by Lee and Wu (2002). This manufacturer mentioned that emissions to air are limited by the use of baghouse pollution control devices, the contents of which are then recycled back into the process as a raw material. A quantity of 19 kg of vanadium pentoxide was released to air by this facility in 2006 (Environment Canada 2009a), probably as a result of handling the raw material (vanadium pentoxide pellets). The manufacturer also stated that wastewater effluents are very limited because of the absence of wet cleaning processes and limited handling of the substance (Environment Canada 2009a).

Three steel mills reported releases of vanadium to the NPRI in 2007, mainly as off-site recycling for one of the mills (151 tonnes) (NPRI 2009). Vanadium is contained in steel slags that are a by-product of steel production (Moskalyk and Alfantazi 2003). However, given the very high temperatures that exist in electric arc furnaces (>1200°C; Perry and Green 1984), which is one of the main technologies used by steel mills in Canada, the vanadium contained in this type of slag is likely not in the form of vanadium pentoxide. This would also apply to the vanadium contained in the dust emitted to air during steelmaking. One of the three steel mills listed on the NPRI for 2007 reported 1813 kg of vanadium releases to air (NPRI 2009).

Metal and Mineral Smelters and Ores

Iron, titanium, uranium and phosphorus smelting plants produce vanadium as a by-product due to the presence of this element in these metal ores (IARC 2006). Smelting of iron ore produces a vanadium slag that contains up to 12–24% vanadium pentoxide, which is used for the production of vanadium metal (IPCS 2001). Slag that contains

vanadium can be transformed into construction materials; the use of such materials is not expected to release significant amounts of this metal to the environment (Chaurand et al. 2007). Solvent extraction from uranium ores produces vanadium pentoxide, which will be found mainly in mill effluent, with the remainder being released into air in a particulate form (IPCS 2001). Similarly, phosphate plants that use a salt-roasting process may release vanadium in effluents and in emissions to air (IPCS 2001). Indeed, the temperature reached during this process is high (over 650°C; Zafar et al. 1996); hence, vanadium contained in phosphate rock is expected to be transformed to the pentoxide form.

Although there is a substantial potential for vanadium mining in Canada, there is currently no vanadium production. Most of the vanadium deposits in Canada are located in Chibougamau (Lac Doré deposits), Matagami and Sept-Iles, Quebec, as well as in Manitoba and Alberta (Taner et al. 2000). There are, however, uranium and phosphorus mines for which extraction activities may result in the release of vanadium to the Canadian environment.

Cement Production

Cement plants burn fossil fuel, mainly heavy oil, to feed the high-temperature boilers used for cement production (Lafarge North America 2009). As explained previously in this report, fossil fuel combustion releases vanadium to the atmosphere, likely in the form of vanadium pentoxide. Cement plants may also use waste products (e.g., old tires) as fuel (Lafarge North America 2009), which will result in the production of bottom ash in the boilers and of fly ash emitted through stacks. Bottom ash is recycled into construction materials, such as asphalt and concrete (Environment Canada 2009a). Since these as well as other types of ash are embedded in the materials, releases of vanadium to the environment from these materials are expected to be low. Finally, cement itself can contain high levels of vanadium, although probably not in the pentoxide form, due to its presence in the raw material from which it is made. Again, vanadium is expected to be immobilized to a certain extent within cement, which is thus considered as an inert material for the purpose of this assessment.

Pulp and Paper Mills and Sawmills

Pulp and paper mills and sawmills are the third largest industry in Canada in terms of quantities of heavy oil burned, after thermal power generation plants and marine transportation (McPhie and Caouette 2007). As explained above, the combustion of heavy oil leads to the formation of vanadium pentoxide. The burning of wood in bark boilers in sawmill facilities is also a likely source of vanadium pentoxide emissions. Under the section 71 survey, pulp and paper mills and sawmills reported (unintentionally) manufacturing between 100 000 and 1 000 000 kg of vanadium pentoxide in 2006 (Environment Canada 2009a). According to the same survey, the majority of vanadium pentoxide releases are collected and sent as solids to on-site or off-site landfills as non-hazardous waste, while the rest is released to the air or water. No reports of release were found in the NPRI database for the reporting year 2007 for this sector (NPRI 2009).

Use as a Catalyst

Sulphuric acid manufacturing plants use vanadium pentoxide as a catalyst. Some of these plants responded to the section 71 survey (Environment Canada 2009a). In total, between 10 000 and 100 000 kg of vanadium pentoxide was used as a catalyst in Canada in 2006 (Environment Canada 2009a). According to the information submitted under the section 71 survey, a major portion of the catalysts used is recycled, while the rest is landfilled. Therefore, releases of vanadium pentoxide to the environment resulting from its use as a catalyst should not be significant.

Automobile Catalytic Converters

Catalytic converters used in automobiles contain vanadium pentoxide, which can be emitted to air via the exhaust system. Since emissions are the result of abrasion, it can be reasonably assumed that vanadium will not be transformed and will indeed be released as vanadium pentoxide. Because they contain precious metals such as platinum, the catalytic converters themselves are usually recycled (Puvvada et al. 2002).

Phosphate Fertilizers

Trace amounts of vanadium may be found in fertilizers as residues following phosphate rock purification (Mortvedt and Beaton 1995). The estimated input of vanadium to soil through the application of phosphate fertilizers is 12 g/ha, when applied at a rate equivalent to 20 kg of phosphorus per hectare (Mortvedt and Beaton 1995). As a result of phosphate rock salt roasting, vanadium is expected to be found in the form of vanadium pentoxide in fertilizers. This represents a source of release of this substance to agricultural lands.

Sewage Sludge Application

Based on the uses of vanadium pentoxide, its presence in industrial wastewater in a dissolved form is expected to be low. Indeed, this substance is mainly used to make alloys and as a catalyst, two uses for which losses to process water are expected to be low. During wastewater treatment, dissolved vanadium species could partition to sludge, which could then be spread on agricultural fields, depending on its origin. However, the importance of this source to environmental releases is expected to be low.

Manufactured Items

Vanadium pentoxide is expected to be found in some manufactured items (e.g., batteries, items containing certain pigments). No information is available on the quantity of manufactured items containing vanadium pentoxide that are imported into Canada. It is recognized that vanadium pentoxide contained in manufactured items that are disposed of in landfills may leach into groundwater as dissolved vanadium species, depending on the

presence at the landfill of a liner, a leachate collection system and/or a leachate treatment system (on-site or off-site).

Environmental Fate

Partitioning

As for most of the elements of the periodic table, vanadium may be found in various forms in ambient air, surface water, sediments, soils and groundwater. Typical fugacity modelling is not applicable to vanadium pentoxide or to the metal ions that are released from it upon dissolution, because, as for other non-volatile chemicals, these substances exert zero partial pressure and fugacity in air (Diamond et al. 1992).

Vanadium pentoxide is highly soluble and hence will release pentavalent vanadium ions upon introduction into water. Under conditions commonly found in oxic fresh waters, the vanadate ions ($H_2VO_4^-$ and HVO_4^{2-}) will be the dominant species. Based on experimental studies and speciation modelling, complexation of vanadate anions with dissolved organic matter (DOM) does not seem important. Experimental evidence suggests that vanadate anions have less of a tendency than cationic metals to adsorb to PM in soil and sediment. Because of their anionic character, vanadate ions are generally more mobile in alkaline environments than in neutral to acidic ones. Under reducing conditions (e.g., deep anoxic surface waters), vanadium may be reduced from the pentavalent to the tetravalent form. Tetravalent forms of vanadium may be positively charged (e.g., the vanadyl ion, VO^{2+}) in solution (Irwin et al. 1997). A detailed discussion of these aspects is provided below.

Air

Being a non-gaseous element with a negligible vapour pressure (Table 2), vanadium is emitted to air principally in the form of fine PM. As described in the previous section on releases, there are natural and anthropogenic sources of vanadium to the atmosphere. Anthropogenic sources are mainly related to the combustion of fossil fuel, which will transform endogenous organovanadium complexes into oxides, including the pentoxide form (IPCS 2001). Depending on the size of the PM with which vanadium pentoxide is associated, the PM will travel for a certain distance in air before being deposited to aquatic or terrestrial environments. Its transportation pathway is described in more detail in the Ecological Exposure Assessment section of this report.

Water

Solubility and Dissociation

By virtue of its relatively high aqueous solubility (Table 2), vanadium pentoxide will dissolve and release pentavalent vanadium ions upon introduction into water. Being a first-row transition element, vanadium has the tendency to exist in high oxidation states, and vanadium ions will form oxy complexes in aqueous solutions (Cotton and Wilkinson

1988). However, the aqueous chemistry of the metal is complex and involves a wide range of oxygenated species for which stabilities depend mainly on the acidity and oxygen level of receiving waters. Under conditions commonly found in oxic fresh waters (i.e., pH between 5 and 9; redox potential [E_h] between 0.5 and 1 V), the pentavalent oxyanions $H_2VO_4^-$ and HVO_4^{2-} (also called vanadate ions) will be the dominant species in solution (Brookins 1988; Takeno 2005). Studying the speciation of vanadium in a lakewater sample of pH 7.5, Fan et al. (2005) did not detect vanadium(IV) oxidation states, supporting the idea that pentavalent forms dominate vanadium speciation in neutral surface fresh waters. Finally, it can be noted that polymerization of oxygenated species of vanadium will increase with increases in their concentrations (>10^{-4} M or 18.2 mg/L: Jennette 1981) and will be more prevalent in seawater (Petterson 1993).

Vanadium is expected to be more mobile under oxidizing conditions than under reducing conditions (Garrett 2005), likely in part reflecting the difference in mobilities of the oxidized anionic and reduced cationic forms. Oxidized forms are generally less mobile under acidic conditions than under neutral to alkaline conditions (Reimann and de Caritat 1998). For example, the species $H_2VO_4^-$ and HVO_4^{2-} are among the most mobile forms of vanadium found in natural oxic waters (Crans et al. 1998). One can speculate that, as a possible consequence of this understanding, there is a lack of impetus for researchers to undertake detailed study of the complexation of vanadium in solution; only three studies have been found on the subject. Wanty and Goldhaber (1992) examined the complexation of vanadium(V) by oxalate, an organic acid found in rather low concentrations in most natural waters, and were able to calculate thermodynamic constants. Lu et al. (1998) studied, under laboratory conditions, the chemical interactions between aquatic humic substances (HS) and vanadate ions. Humic substances were extracted from swamp water near a lake in Australia. Nuclear magnetic resonance spectra demonstrated that vanadate monomers (V_1) are able to form complexes with humic substances ($HS–V_1$ complexes). The authors derived a stability constant K of 108 M^{-1} at pH 7.2 based on the formula $[HS–V_1]/[HS][V_1]$, where [ ] are aqueous molar concentrations. Linnik et al. (2003) investigated the distribution of vanadium among its dissolved forms in freshwater samples collected in Ukrainian water bodies. They conducted oxidation and reduction studies at pH values ranging from 2.9 to 8.5 using humic and fulvic acid concentrations ranging from 20 to 200 mg/L. No information was provided on the pH, E_h or dissolved organic carbon (DOC) content of the water samples. Their measurements indicated that 4–49% of dissolved vanadium was in an unbound state in the form of vanadium(V). Most vanadium was part of complexes with natural organic matter (probably in the form of vanadium(IV)). No stability constants were derived.

Modelling Speciation in Solution

Given the great influence of chemical speciation on metal bioavailability in aquatic systems, the inorganic speciation of vanadium in Canadian surface waters with various physical and chemical characteristics was determined (Table 4 and Appendix 1). The characteristics of these surface waters are considered to be representative of the regions for which releases of vanadium pentoxide to the environment are high and for which an exposure scenario was developed (see Ecological Exposure Assessment section).

Table 4. Modelled results for chemical speciation of vanadium in relevant oxic surface waters in Canada

Water type	General physical and chemical characteristics			Proportion of total aqueous vanadium (%)		
	Degree of mineralization	Degree of acidity	DOC content	HVO_4^{2-}	$H_2VO_4^-$	$HS–V_1$
Prairie						
Wabamun Lake (Alberta)	High; conductance ~500 µS/cm	Alkaline; pH ~8	High; >10 mg/L	38.6	61.4	<<1
Canadian Shield						
Allard River	Low; conductance ~60 µS/cm	neutral; pH ~7	High; >10 mg/L	2.9	97.1	<<1
Colombière River (Quebec)	Low; conductance ~30 µS/cm	Slightly acid; pH ~6.5	High; >10 mg/L	<1	99.3	<<1
Seawater						
St. Lawrence Gulf (eastern Canada)	Very high; salinity ~32 ppt	Alkaline; pH ~8	Very low; <1 mg/L	47.7	52.3	<<1

Abbreviations: DOC, dissolved organic carbon; $HS–V_1$, vanadate complex with humic substances; ppt, parts per thousand.

Modelled estimates indicate that the species $H_2VO_4^-$ and HVO_4^{2-} dominate chemical speciation in all types of water considered, with a minor contribution, less than 1%, from $HS–V_1$ complexes (Table 4). These speciation estimates are rough approximations but are consistent with findings by Shiller and Boyle (1987) regarding the behaviour of vanadium in rivers. Manipulations of Mississippi River water by changing the ratios of dissolved vanadium to DOM suggested that complexation with DOM was not important. Furthermore, comparisons of vanadium concentrations in filtered (0.4 µm) and ultrafiltered (10^5 daltons molecular weight cut-off) water samples from three US rivers showed that this metal is not significantly present in the form of colloids or high molecular weight complexes in these waters. Subsequently, using a data set covering 71 rivers in the United States, Shiller and Mao (2000) determined that DOC could play a "secondary" but nevertheless significant role in fluvial dissolved vanadium concentrations.

Methylation of metals is generally known to increase their potential for bioaccumulation. There is no evidence so far of methylated forms of vanadium in the environment.

Sediments

It has been known for a long time that sediments act as sinks for trace metals in aquatic systems (Förstner and Wittmann 1981), even for more mobile elements like vanadium (Johannesson et al. 2000). The suspended particulate flux in surface waters acts as a "conveyor belt" mechanism whereby metals are "scavenged"—adsorbed by or incorporated into particles generated *in situ* or of allochthonous origin. In turn, these

particles fall through the water column and eventually settle to bottom sediments (Santschi 1984).

Vanadium concentrations in the water column are controlled by several factors, including chemical weathering of silicate rocks (Shiller and Mao 2000) and adsorption/desorption equilibria between particulate and dissolved phases, which vary depending on changes in pH (Harita et al. 2005), redox reactions (Wang and Sañudo-Wilhelmy 2009), aqueous concentrations of ferric oxyhydroxides and manganese oxides (Harita et al. 2005) and levels of hydrogen sulphide (Wanty and Goldhaber 1992) and organic matter (Szalay and Szilágyi 1967). Vanadium is generally more soluble in alkaline and oxidizing waters than in acid and reducing waters. For example, when vanadium(V) is reduced to vanadium(IV) because of a decrease in E_h, dissolved vanadium(IV) species are typically cationic and are thus more readily adsorbed onto particles or complexed with organic matter, resulting in deposition into sediments (Wang and Sañudo-Wilhelmy 2009). Harita et al. (2005) showed in the laboratory that removal of dissolved vanadium in oxygenated water increases with an increase in iron oxide level but remains negligible below an iron oxide concentration of 100 nmol/L.

Available coefficients for the partitioning between water and suspended sediment (K_{ssdw}) and sediment (K_{sdw}) are given in Table 2. The values for K_{ssdw} vary over 2 orders of magnitude ($\log K_{ssdw} = 3.15–5.47$), likely due to the methodology used to derive the coefficients as well as the characteristics of the water from which they were determined, including the substrate itself (i.e., suspended sediments). For those water bodies with characteristics that favour a strong tendency of vanadium to sorb to solid particles, a significant proportion of dissolved forms of this metal will end up in sediments, through the settling of suspended particles (Hamilton-Taylor and Willis 1984).

Soils

Similar to sediments, soils are major sinks for metals released from natural and anthropogenic sources. After vanadium pentoxide enters into soils, its transformation processes will involve dissolution, partitioning and aging. The latter designates slow reactions transferring metals from labile pools to insoluble pools (Smolders et al. 2007). In general, metal bioavailability is governed by the mobility and solubility of geochemical forms (Smolders et al. 2007).

The behaviour of vanadium in soils is linked to the physical and chemical properties of both the soil and the vanadium-containing compound entering this compartment. For example, Smolders et al. (2007) amended a sandy soil with vanadium pentoxide at a concentration of 250 mg/kg. Concentrations of vanadium in pore water after 2 and 24 weeks of incubation were, respectively, 19.8 and 7.5 mg/L. This indicates that rapid dissolution led to an initial peak in the concentration of vanadium in pore water, followed by slower aging, which eventually led to a decrease in the vanadium concentration. Experimental evidence suggests that vanadium is generally more mobile and is found in more labile fractions in alkaline soils than in neutral to acidic soils (Table 5). Fiorentino et al. (2007) described a case of natural contamination of groundwater by vanadium

(concentration range of 0.05–2.5 mg/L) in a sandy loess soil of good porosity and permeability and rich in weatherable minerals. The soil pH was not reported, but the groundwater had an average pH of 7.36.

Table 5. **Forms and retention of vanadium in soils of various physical and chemical properties**

Soil			Retention and/or forms of vanadium	Reference
Type	**Characteristics**	**pH**		
Acid sulphate soils	–	<4	Vanadium not enriched in river (water and sediments) and its estuary draining these soils; evidence of strong retention in refractory soil fractions	Nordmyr et al. 2008
Sand and gravel (industrial site)	Sand: 65–85% Silt: 13–33% Clay: 2–5% Organic carbon: 0.6–1.5%	4.4–6.8	% vanadium found in fractions: Detrital: 57–93% Iron–manganese oxide: 2.5–38% Undetectable in more labile fractions	Abollino et al. 2006
Fine sand	CEC: 131 mmol/kg Organic carbon: 46 g/kg Extractable iron: 4.6 g/dm^3	<6	70–80% of added vanadium(V) retained by each soil at pH 4; 65–90% of added vanadium(V) retained by each soil at pH 6 Leachability of vanadium increases with pH Results independent of soil composition	Mikkonen and Tummavuori 1994
Fine sand	CEC: 134 mmol/kg Organic carbon: 23 g/kg Extractable iron: 5.1 g/dm^3	6.1		
Clay	CEC: 347 mmol/kg Organic carbon: 42 g/kg Extractable iron: 6.6 g/dm^3	>7		
Sandy loam (industrial site)	SiO_2: 40% Fe_2O_3: 17% CaO: 18% Organic carbon: 0.6%	8.8	21% of total vanadium found in mobilizable fractions (EDTA extraction)	Terzano et al. 2007

Abbreviations: CEC, cation exchange capacity; EDTA, ethylenediaminetetraacetic acid.

Soil–water partition coefficients (log K_{sw}) that were obtained for Canadian soils range from 2.58 to 5.08 (Sheppard et al. 2007; Table 2). These coefficients are based on desorption studies; lower values would be expected for adsorption studies. Generally, the partitioning behaviour of vanadium will vary depending on both the vanadium compound added to the soil and the soil characteristics.

It should be added that non-dissolved vanadium pentoxide (as parent substance) would be expected to be found only in relatively dry soils. When released to these soils, vanadium pentoxide will likely mainly remain there, with some of the substance leaching locally

into groundwater and/or surface water ecosystems when the soil gets soaked by rain or melting snow/ice.

Environmental Persistence

A metal or metalloid ion is considered infinitely persistent because it cannot degrade any further. For most metal-containing compounds, it is the potentially bioavailable metal ion that is liberated (in greater or lesser amounts) upon contact with water that is the moiety of toxicological concern. A parent compound from which persistent metal ions are released is itself considered to meet regulatory persistence criteria (Environment Canada 2003).

Vanadium pentoxide is considered persistent because the pentavalent vanadium ions that are released into solution when it dissolves cannot be irreversibly degraded. As noted previously, depending upon ambient pH and E_h conditions, pentavalent vanadium can be reduced (e.g., to tetravalent vanadium), but this transformation is in principle reversible. Therefore, vanadium pentoxide meets the persistence criteria for all media (i.e., air, water, soil and sediment) as set out in the *Persistence and Bioaccumulation Regulations* of CEPA 1999 (Canada 2000).

Bioavailability

Water Column

A number of authors have noted the similarity of the oxyanion $H_2VO_4^-$ to the structure of the endogenous oxyanion $H_2PO_4^-$ (Jennette 1981; Clarkson 1993; see Figure 1), which suggests a possible mechanism for uptake of vanadium by aquatic organisms. Evidence has accumulated indicating that the vanadium anion is bioavailable, entering living cells via anion exchange systems dedicated to phosphate in a phenomenon called ionic mimicry. The experimental work of Luecke and Quiochio (1990), for example, showed that there is a molecular mechanism that actively transports phosphorus into cells and organelles primarily in the form of monobasic or dibasic orthophosphate (i.e., $H_2PO_4^-$ or HPO_4^{2-}). Furthermore, Cantley et al. (1978) demonstrated that vanadium can enter red blood cells by using Na^+ or K^+ adenosine triphosphatase (ATPase). The vanadate ion binds to a site on the enzyme that is specific to phosphate.

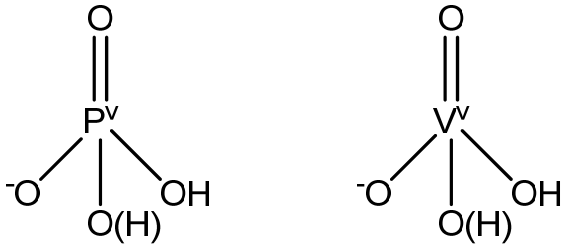

Figure 1. Similar tetrahedral structures of phosphate and vanadate ions. These partially ionized and monovalent ions will be stable in neutral to slightly acidic solutions (adapted from Clarkson 1993).

In line with the ionic mimicry of vanadium described above is the evidence given by the above literature review and modelling pointing to the importance of vanadate species in relevant Canadian surface waters (Table 4).

Couillard et al. (2008) deployed, for a period of 17 days, specimens of the amphipod *Hyalella azteca* along metal contamination gradients in two rivers (the Allard and Colombière rivers in Table 4 and Appendix 1) affected by metal mining in northwestern Quebec. Vanadium was accumulated by the transplanted organisms in a dose-dependent manner (i.e., as a function of vanadium in the dissolved phase) (Figure 2). A similar positive relationship was obtained for the antimony anion in this study. By comparison, no relationship was found between bioaccumulated cadmium and dissolved cadmium in transplanted specimens. Bioavailability of the cadmium cation is well known to be strongly influenced by dissolved calcium, water hardness, inorganic complexation with major ions and pH (Campbell 1995). This result supports the idea that total dissolved vanadium is bioavailable and is a good predictor of bioaccumulated vanadium for organisms that take up most of their metal from the water column (this is the case for *H. azteca*: Borgmann et al. 2007). Vanadate ions ($H_2VO_4^-$ and HVO_4^{2-}) are probably the dominant chemical species of dissolved vanadium in these oxic waters (the Allard and Colombière rivers in Table 4) and are likely important forms in more mineralized oxic waters and in seawater as well (Table 4).

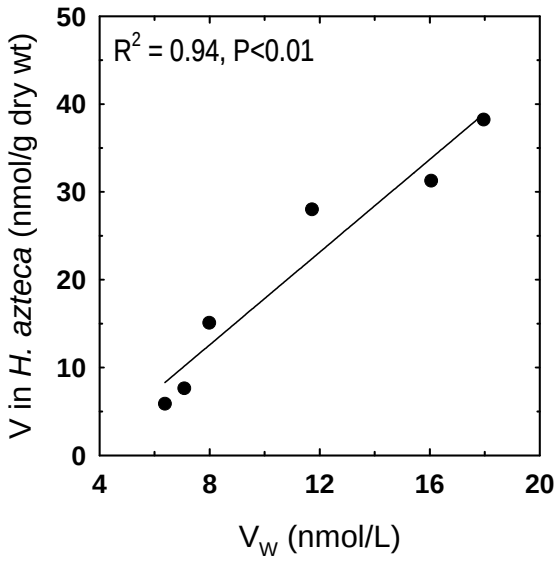

Figure 2. Relationship between vanadium concentrations in the amphipod *H. azteca* and mean dissolved vanadium concentrations after 17 days of deployment in two rivers affected by metal mining in northwestern Quebec. Total body concentrations were corrected for time 0 background concentrations. Dissolved vanadium levels were obtained after filtering water samples on 0.45 µm membranes and correcting for field blanks. Data for both rivers are combined in these relationships (three sites per river and five sampling times per site); their physical and chemical characteristics are roughly similar (adapted from Couillard et al. 2008).

Sediments and Soils

Sediments and soils are much more complex media than surface waters; consequently, determination of bioavailability of metals in these compartments is not straightforward. However, examples below suggest an important role for solubility and labile forms in determining the bioavailability of vanadium in these media.

Snodgrass et al. (2005) exposed, in the laboratory, the larvae of the green frog *Rana clamitans* to sediments contaminated by metals. Sediment treatments included clean sand (vanadium concentration: 2.3 µg/g dry weight [g-dw]), sediment from a coal combustion waste settling basin (69 µg/g-dw) and sediment affected by metal mining (73 µg/g-dw). Concentrations of vanadium were 4 times higher in larvae exposed to coal combustion waste sediments than in those exposed to mine sediments. This result points to differences in chemical speciation favouring increased bioavailability and subsequent bioaccumulation of vanadium in coal combustion waste sediments.

Tyler (2004) evaluated the bioavailability to beech trees of 50 cationic elements measured in the humus horizon of a forest podzol. Root/organic soil concentration ratios (R/S) were calculated, and ionic properties of the elements were considered, including ionic radius (r), ionic charge (z) and ionic potential (IP = z/r). IP was inversely related to R/S (R^2 = 60%; $P < 0.001$). It can be noted that a low IP (e.g., Na^+ or K^+) is related to a weaker tendency to adsorb to the soil matrix, and thus a greater tendency to partition into the soil pore water, presumably favouring root uptake (pers. comm., Peter Campbell, Institut National de la Recherche Scientifique – Centre Eau, Terre et Environnement, as per review comments, dated January 2010, unreferenced). Vanadium was assumed to be in the form of vanadyl ion (VO^{2+}), a cation with an elevated IP, in this acid soil. A low R/S value was obtained, indicating that this ion was not readily accumulated by beech roots, pointing to its low bioavailability.

Martin and Kaplan (1998) conducted a field study to examine the mobility of cadmium, tellurium and vanadium in a loamy-sand soil and the availability of these metals to bean plants. The soil, which had a low cationic exchange capacity but was rich in iron and aluminum oxides, was limed (to improve plant growth) before the addition of soluble metal salts ($VOSO_4$ for vanadium). Extractable vanadium decreased dramatically during the first 18 months after treatment, and little vertical movement in the soil column was noted. In addition, concentrations and amounts of vanadium in roots and aboveground plant tissues remained low and did not change significantly during this period. These results were interpreted as indicating the transformation of vanadium into unavailable forms caused by aging (and not leaching). The authors suggested that because the treated soil was oxic and slightly acidic (pH 6.8), iron and aluminum oxides offered positive charge sites to complex negatively charged vanadate ions also present at this pH (zero point of charge of goethite is pH 7.8), therefore lowering the bioavailability of vanadium.

Bioaccumulation Potential

Bioaccumulation of metals – like that of organic substances – is of potential concern because of the possibility of chronic toxicity to the organisms accumulating these substances in their tissues and the possibility of toxicity to predators eating these organisms. Bioaccumulation potential is typically quantified by determining either a bioconcentration factor (BCF) or a bioaccumulation factor (BAF). However, these ratios are sometimes the object of criticism when applied to metals, because they are considered of little usefulness in predicting metal hazards (Schlekat et al. 2007). For example, some metals naturally may be highly accumulated from the surrounding medium because of their nutritional essentiality. Furthermore, both essential and non-essential metals may be regulated internally within relatively narrow margins by the homeostatic and detoxification mechanisms that many organisms possess. It follows that when ambient concentrations of metals are low, BCF and BAF values are often elevated. Conversely, when ambient metal concentrations are high, BCF and BAF values tend to be lower (BCF: McGeer et al. 2003; BAF: DeForest et al. 2007). Thus, inverse relationships may be observed between BCF and BAF values and metal exposure concentrations, and this complicates the interpretation of these values. Natural background concentrations in organisms may contribute to these negative trends (e.g., Borgmann and Norwood 1995). In addition, inverse relationships can occur for non-essential elements as well because there are a finite number of binding sites for these metals within an organism that could become saturated at higher concentrations (e.g., Borgmann et al. 2004, MacLean et al. 1996).

Water Column

Taking account of these issues, a mechanistically based saturation model for bioaccumulation of metals using the freshwater amphipod *Hyalella azteca* as a test organism has been developed (Borgmann et al. 2004; Norwood et al. 2007). This model can estimate a BCF based on background-corrected metal accumulation at low aqueous concentration, which avoids the above-mentioned concentration dependence. In addition, these researchers showed that 1) lethality occurs when tissue concentrations surpass a critical body concentration and 2) critical body concentrations appear relatively constant for a variety of different non-essential or marginally essential metals in spite of large differences in the waterborne concentrations that result in chronic toxicity (e.g., Schlekat et al. 2007). It can be deduced from these two points that when the uptake of a given metal is more efficient, a lower water concentration is required to reach the chronic toxicity threshold in tissue. Consistent with this statement, these researchers observed a strong negative relationship between estimates of chronic toxicity and BCF and BAF values for non-essential or marginally essential metals and metalloids (in laboratory: Norwood et al. 2007; Schlekat et al. 2007; in field settings: Couillard et al. 2008). This relationship holds because total metal body concentration in *Hyalella* is likely related to the concentration of the metal at the site of toxic action. In principle, animals with metal-handling strategies that do not include important pools of metals stored in detoxified forms may show close relationships between bioaccumulation ratios (BAFs and BCFs) and chronic toxicity (Couillard et al. 2008).

Vanadium is probably essential to enzyme systems that fix nitrogen from the atmosphere (i.e., bacteria). It is concentrated by certain organisms (some algae and marine invertebrates), but its function in these organisms is uncertain (Nielsen 1991; Markert 1994; IPCS 2001). It is also uncertain whether vanadium is needed by other types of organisms.

The selection of studies for assessing the bioaccumulation potential of vanadium builds on the above knowledge and on approved methodologies for deriving BCFs and BAFs (OECD 1993, 1996; Arnot and Gobas 2006). Appendix 2 lists the criteria and considerations used for BCF and BAF data quality assessment. In recognition that these ratios are less meaningful for organisms with large and inert metal pools, bioaccumulation studies with hyperaccumulator plants (e.g., aquatic bryophytes: Samecka-Cymerman et al. 2005) or with animals that accumulate vanadium in large inert metal pools (e.g., marine ascidians: Michibata and Kanamori 1998) were omitted. Metal concentrations in soft tissues only were considered for invertebrates with shells or exoskeletons, when such information was available. It should be noted that other jurisdictions may use different criteria for selecting and assessing studies that pertain to the bioaccumulation of metals. Therefore, the selection and interpretation of BCF and BAF values may differ among the assessments conducted by various jurisdictions.

Table 6 presents the empirical BCF and BAF values found to be in accordance with the criteria and considerations described in Appendix 2. The data presented are for pentavalent forms of vanadium measured as an element and not for the substance vanadium pentoxide. As explained in previous sections of this report, due to its high water solubility, vanadium pentoxide will dissolve and will transform into vanadate ions. These ions are potentially bioavailable and can be taken up by organisms. Unless otherwise stated, all BCF/BAF values reported in Table 6 are based on measured concentrations of the vanadium element.

Table 6. Empirical data selected for estimating bioaccumulation potential of vanadium in aquatic organisms[1]

Test organism	Study type		Evidence of SS	End-point	Mean value (L/kg-ww)	Reference
Freshwater amphipod *Hyalella azteca*	Field	Tr	Yes	BAF	333 (n = 6)	Couillard et al. 2008
Freshwater fish *Jordanella floridae*	Lab	SS	Yes	BCF	27.9[2] (n = 2)	Holdway et al. 1983
European eel (freshwater) *Anguilla anguilla*	Lab	SS	Yes for blood; no for other organs	BCF	5 (blood) 32 (kidney) 15 (liver 13 (bone) 1.6 (carcass) (n = 1)	Bell et al. 1980
Brackish water hydroid *Cordylophora caspia*	Lab[3]	SS	Yes	BCF	13.5 (n = 2)	Ringelband and Helh 2000
Marine clam	Field	Tr	Yes	BAF	25 mm[4]: 30	Gomez-Ariza

Test organism	Study type		Evidence of SS	End-point	Mean value (L/kg-ww)	Reference
Venerupis decussata					36 mm[4]: 24 (n = 4)	et al. 1999
Marine shrimp *Lysmata seticaudata*	Lab	SS	Yes	BCF	11 (n = 8)	Miramand et al. 1981
Marine crab *Carcinus maenas*	Lab	SS	Yes	BCF	12 (n = 8)	Miramand et al. 1981
Marine mussel *Mytilus galloprovincialis*	Lab	SS	Yes	BCF	5[5] (n = 2)	Miramand et al. 1980

Abbreviations: BAF, bioaccumulation factor; BCF, bioconcentration factor; kg-ww, kilogram wet weight; Lab, laboratory; n, number of samples; SS, steady state study; Tr: steady state study coupled with transplantation or deployment.

[1] BCFs and BAFs expressed on a dry weight basis have been converted to a wet weight basis using 0.2 g-dw per 1 g-ww, which is a reasonable conversion factor for invertebrates and fish (e.g., Ikemoto et al. 2008). When published information permitted, body concentrations were corrected for vanadium concentrations in gut contents, and bioaccumulation ratios (BCFs and BAFs) were corrected for background vanadium concentrations in test organisms and water.

[2] Only the BCF determined at the lowest exposure level, 0.041 mg/L, was considered, because this is the only concentration that did not cause an observed effect on growth or reproduction compared with control.

[3] Only nominal concentrations were reported.

[4] Shell length; BAFs based on vanadium in soft tissues only.

[5] Value based on vanadium in soft tissues only.

As shown in Table 6, experimental BCFs and BAFs of acceptable quality reported for fish and aquatic invertebrates vary between 1.6 and 333 L/kg wet weight (kg-ww). The two mean BAFs obtained in field studies were determined in a context of polymetallic contamination, which may have an impact on vanadium bioaccumulation (Gomez-Ariza et al. 1999; Couillard et al. 2008). However, they offer the distinct advantage of being environmentally realistic and of integrating all exposure pathways.

The results obtained by Couillard et al. (2008) with *H. azteca* are of interest for this assessment because this amphipod is a very sensitive organism in regards to metal toxicity (Borgmann et al. 2005). Furthermore, *Hyalella* is a genus that is widely distributed throughout Canada and is often numerically abundant in freshwater habitats (Witt and Hebert 2000). At present, little guidance exists for field measurement of BCFs and BAFs. Weisbrod et al. (2009) indicated that measuring bioaccumulation in nature may rely on natural populations or deployment of sentinel organisms and requires reliable measurements of chemical concentrations in biota and exposure media of interest. The approach taken by Couillard and co-workers (2008) included transplantation of specimens along metal contamination gradients, used reliable methods for analyzing metals in water and tissues, and integrated key characteristics of methodologies for deriving BCF values (OECD 1993, 1996; Borgmann et al. 2004), namely:

- the requirement of having at least three low-exposure (i.e., substantially below acute toxicity) treatment levels per metal for the test species; Couillard et al. (2008) had six deployment sites in total;

- for a given metal, the requirement of obtaining an absorption isotherm with a slope of approximately 1; this isotherm is defined as the log–log relationship between the chemical concentration in the test organism and that in the water (OECD 1993). This condition is equivalent to reaching steady state between organism and water "compartments" for the metal studied.

These two requirements were filled for vanadium. Specimens were gut-cleared before metal analyses. Total body concentrations were corrected for time 0 background concentrations, and dissolved vanadium levels were corrected for field blanks (Figure 2). It should be noted that biouptake of vanadium in nature by *Hyalella* can be mainly attributed to bioconcentration. This is based on the findings of Borgmann et al. (2007), who demonstrated in a field setting that the dissolved phase is the dominant route of accumulation for this metal in the amphipod. Hence, an indication of the relatively low bioaccumulation potential of vanadium may be obtained by comparing the field BAF for vanadium, 333 L/kg-ww, with the laboratory BCF for mercury (generally accepted to be a bioaccumulative element), a value of 9650 L/kg-ww corrected for background mercury levels and obtained with amphipod specimens (*H. azteca*) exposed in test water of 100 mg/L hardness (Schlekat et al. 2007).

Sediments

The toad species *Bufo terrestris* has been found to breed in coal fly ash collection basins from which it accumulates metals in its tissues. Ward et al. (2009) exposed toads for a period of 6 months to control sandy or ash-rich sediments covered with water and placed in mesocosms. Animals were fed weekly with crickets whose vanadium content corresponded to the sediment treatment (e.g., contaminated prey with contaminated sediment). Bioaccumulated vanadium levels increased significantly over time, and steady-state conditions were approached after 4 months of exposure, as suggested by trends of vanadium concentrations in amphibians over time. For this assessment, a biota–sediment accumulation factor (BSAF-sediment) for vanadium of 0.153 g/g-dw was derived from mean values given in the paper and corrected for background vanadium concentration in whole organisms and control sediment. As a basis for comparison, BSAFs for mercury tend to be much higher for infaunal benthos. For example, BSAFs-sediment for mercury ranged from 0.9 to 3.8 g/g-dw in the marine clam *Polymesoda caroliniana* living in sediments of a river estuary (Ruelas-Inzunza et al. 2009). Specimens of the lugworm *Arenicola marina* were reported to have BSAFs-sediment for mercury varying between 0.01 and 1 g/g-dw, depending on sediment composition and contamination by mercury (Casado-Martínez et al. 2008).

Soils

Samecka-Cymerman et al. (2009) measured vanadium concentrations in the terrestrial moss *Brachythecium rutabulum* and the soil on which it grew. Soil and moss samples were taken at a control site and at six sites along a 15 km long metal gradient generated by the atmospheric emissions of a copper smelter. From median values for vanadium in soil and plant given in the paper, a biota–soil accumulation factor (BSAF-soil) corrected

for median background vanadium concentration in control moss and soil was 0.29 g/g-dw. Using the same approach, a BSAF-soil of 1.72 g/g-dw was derived for cadmium. Since terrestrial mosses can accumulate metals directly from atmospheric deposition, without the metal having to reach the soil compartment, the BSAF-soil values derived from this study are likely overestimated.

Biomagnification Potential

Although field-based BAFs can give some indication of the biomagnification potential of a metal (i.e., if very high BAF), a better approach is to derive a trophic transfer factor from prey to predator (TTF: DeForest et al. 2007) (also called biomagnification factor or BMF), or to study changes in metal concentrations in biota making up natural food webs (i.e., trophic magnification). The two studies described below belong to the second category.

Ikemoto et al. (2008) measured vanadium and analyzed stable carbon and nitrogen isotopes in biota found in the Mekong delta, Vietnam, an area experiencing rapid urban and industrial development. Metal concentrations were expressed on a whole body basis. Surface waters were characterized for trace metal concentrations. Phytoplankton, snails, 5 species of crustaceans and 15 species of fish were monitored. Vanadium concentrations in organisms showed no increasing or decreasing trends from lower to higher trophic levels. In contrast, there were clear signs of biomagnification for mercury based on both concentrations measured in whole organisms and nitrogen isotope analysis.

Wren et al. (1983) determined vanadium concentrations in various trophic levels within an undisturbed lake ecosystem and watershed of the Canadian Shield. Organisms studied included a bivalve, five species of fish, three species of birds and three species of mammals. Vanadium was measured in sediments, whole clams and muscle tissues of fish, birds and mammals. Again, vanadium showed no evidence of biomagnification, but mercury clearly did, although no biomagnification factor was calculated.

The food webs in these two studies do not appear to be sufficiently understood to properly evaluate exact predator–prey relationships and associated trophodynamics (e.g., DeForest et al. 2007), because it was not established with certainty that trophic links actually existed between organisms collected. In addition, Wren et al. (1983) did not report metal concentrations on a whole body basis for fish, birds and mammals and this added uncertainty to the metal trends observed in their study. Despite these limitations and in clear contrast with mercury, vanadium has apparently no tendency to biomagnify in the aquatic and terrestrial food webs studied.

Overall, there are several lines of evidence to suggest that the bioaccumulation potential of vanadium in natural ecosystems is low: 1) moderate to low (1.6–333) BCFs and BAFs obtained from six studies conducted at steady state; 2) a BSAF-sediment and a BSAF-soil well below 1; and 3) two field surveys indicating the absence of biomagnification of vanadium in natural food webs. It is therefore proposed that vanadium, and by extension the parent compound from which it may originate, vanadium pentoxide, does not meet

the criteria for bioaccumulation potential (BCF, BAF ≥5000) as set out in the *Persistence and Bioaccumulation Regulations* of CEPA 1999 (Canada 2000).

Potential to Cause Ecological Harm

Ecological Effects Assessment

Aquatic Compartment

There is experimental evidence suggesting that vanadium causes harm to aquatic organisms following short-term (acute) and longer-term (chronic) exposure at relatively low concentrations. Many empirical data are available in the literature for the acute and chronic toxicity of vanadium pentoxide and other vanadium compounds. Because all of these compounds are soluble in water, all data from reliable chronic studies were considered in order to derive a critical toxicity value (CTV), even though different vanadium species may exist in solution following the dissolution of these compounds. Robust study summaries (RSS) were completed for the most sensitive endpoints and are available upon request.

Table 7 summarizes the data on the acute and chronic toxicity of vanadium to freshwater organisms. It should be noted that concentrations in the cited studies are expressed in milligrams of vanadium per litre. Therefore, the CTV derived from these data is for vanadium as an element rather than for the compounds tested (e.g., vanadium pentoxide).

Table 7. Empirical data for acute and chronic toxicity to freshwater organisms[1]

Test organism	Test compound	Hardness (mg/L as calcium carbonate)	pH	Test type	Endpoint / duration	Value (mg/L as vanadium)	Reference
Fish							
Brook trout *Salvelinus fontinalis*	V_2O_5	37	6.5–7.9	Chronic	EC_{10} / 30 days	0.61*	Ernst and Garside 1987
Rainbow trout *Oncorhynchus mykiss*	V_2O_5	Soft	7.4	Chronic	LC_{50} / 28 days	0.16	Birge 1978
Guppy *Poecilia reticulata*	$VOSO_4$[a]	35	6.0–6.5	Acute	LC_{50} / 6 days	0.37	Knudtson 1979
Fathead minnow *Pimephales promelas*	V_2O_5	n/a	8.1	Chronic	LOEC / 28 days	0.48*	OMOE 1998
Catfish *Clarias batrachus*	NH_4VO_3	n/a	7.5	Chronic	LC_{50} / 28 days	6.31	Chakraborty et al. 1995
Goldfish *Carassius auratus*	$NaVO_3$	35	6.0–6.5	Acute	LC_{50} / 6 days	2.45	Knudtson 1979
Zebrafish *Brachydanio rerio*	$NaVO_3$	223	8.2–8.4	Acute	NOEC (lethality) / 96 h	0.7	Beusen and Neven 1987
Threespine stickleback *Gasterosteus aculeatus*	Na_3VO_4	74–100	7.2–8.3	Acute	NOEC (lethality) / 96 h	0.93	Gravenmier et al. 2005
Chinook salmon *Oncorhynchus tshawytscha*	Na_3VO_4	211	7.4–8.3	Acute	LC_{50} / 96 h	16.5	Hamilton and Buhl 1990
Flannelmouth sucker *Catostomus latipinnis*	Na_3VO_4	144	7.9	Acute	LC_{50} / 96 h	11.5	Hamilton and Buhl 1997
Flagfish *Jordanella floridae*	V_2O_5	347	8.2	Chronic	MATC / 34 days	0.14*	Holdway and Sprague 1979
Colorado squawfish *Ptychocheilus lucius*	Na_3VO_4	196	7.8	Acute	LC_{50} / 96 h	3.8	Hamilton 1995
Razorback sucker *Xyrauchen texanus*	Na_3VO_4	196	7.8	Acute	LC_{50} / 96 h	3.0	Hamilton 1995
Bonytail *Gila elegans*	Na_3VO_4	196	7.8	Acute	LC_{50} / 96 h	2.2	Hamilton 1995

Test organism	Test compound	Hardness (mg/L as calcium carbonate)	pH	Test type	Endpoint / duration	Value (mg/L as vanadium)	Reference
Invertebrates							
Water flea *Daphnia magna*	$NaVO_3$	225	8.1	Chronic	EC_{10} (reproduction) / 21 days	1.0*	van Leeuwen et al. 1987
Naidid oligochaete *Pristina leidyi*	Na_3VO_4	105	7.9	Acute	LC_{50} / 48 h	30.8	Smith et al. 1991
Diptera larva *Chironomus plumosus*	V_2O_5	80	7.8	Acute	LC_{50} / 96 h	0.218	Fragašová 1998
Oligochaete *Tubifex tubifex*	V_2O_5	80	7.8	Acute	LC_{50} / 96 h	0.211	Fragašová 1998
Amphipod *Hyalella azteca*	V and Na_3VO_4	18	6.8–9.0	Acute	LC_{50} / 7 days	0.4	Borgmann et al. 2005
Algae							
Anabaena flos-aquae	Na_3VO_4	n/a	6.8	Chronic	IC_{100} (growth inhibition) / 7 days	0.1	Lee et al. 1979
Chlorella pyrenoidosa	NH_4VO_4	n/a	6.8	Chronic	MATC (growth inhibition) / 7 days	0.32*	Lee et al. 1979
Navicula pelliculosa	Na_3VO_4	n/a	6.8	Chronic	NOEC (growth inhibition) / 7 days	1*	Lee et al. 1979
Scenedesmus obliquus	Na_3VO_4	n/a	6.8	Chronic	MATC (growth inhibition) / 7 days	0.32*	Lee et al. 1979
Scenedesmus quadricauda	V_2O_5	n/a	n/a	Chronic	EC_{50} (growth inhibition) / 12 days	2.23*	Fragašová et al. 1999

Abbreviations: EC_{xx}, the concentration of a substance that is estimated to cause some effect on xx% of the test organisms; LC_{50}, the concentration of a substance that is estimated to be lethal to 50% of the test organisms; IC_{100}, the inhibitory concentration for 100% of test organisms; LOEC, lowest-observed-effect concentration, the lowest concentration in a toxicity test causing a statistically significant effect in comparison with the controls; n/a, not available; NOEC, no-observed-effect concentration, the highest concentration in a toxicity test not causing a statistically significant effect in comparison with the controls; MATC, the maximum acceptable toxicant concentration, the geometric mean of NOEC and LOEC.

[1] The lowest endpoint is reported for individual species.

[a] Vanadium in this compound is in the oxidation state +4 as opposed to +5 for other compounds, including vanadium pentoxide.

* Denotes chronic toxicity data used in SSD (Figure 3) to determine the PNEC.

A species sensitivity distribution (SSD) was developed using the chronic toxicity data shown in Table 7 for three fish, one invertebrate and four algae species (Figure 3). When more than one value for an endpoint was available for a single species, the lowest value was chosen, following guidance from the Canadian Council of Ministers of the Environment (CCME 2007). It is well documented that the toxicity of metals depends on the pH and ionic strength of the external medium (DiToro et al. 2001). As a result, toxicity data used as input in an SSD may be normalized for the effects of pH, ionic strength and DOC (ICMM 2007), depending on assessment needs. However, this was not done for this assessment, as no equations could be found for vanadium to correct the data to account for these toxicity-modifying factors. Also, there is evidence that toxicity-modifying factors may be less important for the vanadate anions (expected to be the dominant species in oxic waters) than for some cationic metals, given results of the speciation modelling (Table 4) and field studies of speciation of dissolved forms of vanadium.

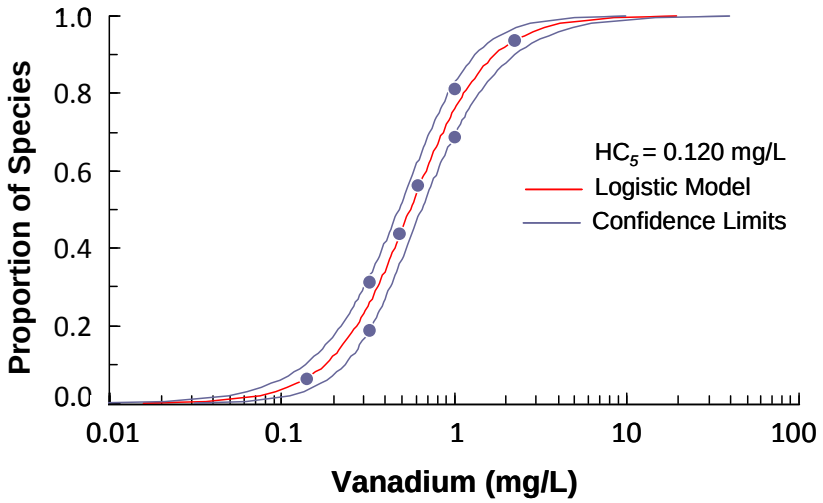

Figure 3. Species sensitivity distribution (SSD) for vanadium based on selected chronic toxicity data for freshwater aquatic organisms. The logistic model fit to data is shown on the graph, along with the 95% confidence interval.

The software SSD Master version 2.0 (SSD Master 2008) was used to plot the SSD. Several cumulative distribution functions (normal, logistic, Gompertz, Weibull and Fisher-Tippett) were fit to the data using regression methods. Model fit was assessed using statistical and graphical techniques. The best model was selected based on consideration of goodness of fit and model feasibility. Model assumptions were verified graphically and with statistical tests. The logistic model provided the best fit of the models tested (Anderson-Darling Statistic [A^2] = 0.178), and the 5th percentile (HC_5), i.e., hazardous concentration to 5% of species, of the SSD plot is 0.12 mg/L, with lower and upper confidence limits of 0.085 and 0.17 mg/L, respectively (Figure 3). The HC_5 of 0.12 mg/L calculated from the SSD is selected as the predicted no-effect concentration (PNEC) for toxicity to freshwater organisms.

Table 8 summarizes the acute and chronic toxicity data for marine organisms. It shows that acute and chronic toxicity values range from 0.05 to 65 mg/L and from 0.25 to 8.0

mg/L, respectively. The lowest acute endpoint (0.05 mg/L) is a 48 h lowest-observed-effect concentration (LOEC) for development in oyster larvae, while the lowest chronic endpoint (0.25 mg/L) is an 8-day LOEC for mortality in brine shrimp, supported by another study on the same species (9-day median lethal concentration [LC_{50}] of 0.2–0.3 mg/L). Even though chronic exposure to vanadium is expected in the environment, the lowest acute value of 0.05 mg/L was chosen as a CTV for marine organisms. The fact that the lowest acute endpoint is lower than the chronic one may be due to a lack of representative chronic data or may indicate that oyster larvae are very sensitive to vanadium. Since the data set for toxicity to marine organisms is relatively small, an assessment factor of 10 was applied to the CTV to obtain a PNEC of 0.005 mg/L, following guidance from the CCME (2007).

Table 8. Empirical data for acute and chronic aquatic toxicity to marine organisms

Test organism	Test compound	Water quality		Test type	Endpoint / duration	Value (mg/L as vanadium)	Reference
		Salinity (‰)	pH				
Marine algae							
Green alga *Dunaliella marina*	$NaVO_3$	38	n/a	Chronic	EC_{10} / 15 days EC_{50} / 15 days	>0.3 0.5	Miramand and Ünsal 1978
Diatom *Asterionella japonica*					EC_{10} / 15 days EC_{50} / 15 days	>0.1 2	
Dinoflagellate *Prorocentrum micans*					EC_{10} / 15 days EC_{50} / 15 days	>0.2 3	
Marine invertebrates							
Oyster *Crassostrea gigas*	V_2O_5	34	8.1	Acute	EC_{50} (D-shape larval development) / 48 h	0.5	Fichet and Miramand 1998
					LOEC (D-shape larval development) / 48 h	0.05[1]	
Urchin *Paracentrotus lividus*					EC_{50} (development of pluteus larva) / 48 h	>0.5 and <0.75	
					NOEC (development of pluteus larva) / 48 h	0.05	
					LOEC (development of pluteus larva) / 48 h	0.1	
Brine shrimp *Artemia salina*					NOEC (development of nauplii larva) / 48 h	0.75[2]	
				Chronic	LOEC (mortality) / 8 days	0.25	
					LC_{65} / 8 days	0.5	
Worm *Nereis diversicolor*	$NaVO_3$	38	n/a	Acute	LC_{50} / 9 days	10	Miramand and Ünsal 1978
Mussel *Mytilus galloprovincialis*					LC_{50} / 9 days	65	
Crab *Carcinus maenus*					LC_{50} / 9 days	35	

Test organism	Test compound	Water quality		Test type	Endpoint / duration	Value (mg/L as vanadium)	Reference
		Salinity (‰)	pH				
Brine shrimp *Artemia salina* (larva)	n/a	n/a	n/a	Chronic	LC_{50} / 9 days	0.2–0.3	Miramand and Fowler 1998
Sea urchin *Arbacia lixula* (pluteus larva)					LC_{100} / 72 h	0.5	
Hydroid *Cordylophora caspia*	NH_4VO_3	10	8	Chronic	LOEC (population growth) / 10 days	2	Ringelband and Karbe 1996
					LC_{50} / 10 days	5.8	
	NH_4VO_3	2 5 10 20	8	Chronic	EC_{50} (population growth inhibition) / 10 days	1.7 4.7 4.5 8.0	Ringelband 2001
Marine fish							
Dab *Limanda limanda*	NH_4VO_3	35	7.7	Acute	LC_{50} / 24 h LC_{50} / 48 h LC_{50} / 72 h LC_{50} / 96 h	44 31 28 28	Taylor et al. 1985

Abbreviations: EC_{xx}, the concentration of a substance that is estimated to cause some effect on xx% of the test organisms; LC_{xx}, the concentration of a substance that is estimated to be lethal to xx% of the test organisms; LOEC, lowest-observed-effect concentration, the lowest concentration in a toxicity test causing a statistically significant effect in comparison with the controls; n/a, not available; NOEC, no-observed-effect concentration, the highest concentration in a toxicity test not causing a statistically significant effect in comparison with the controls.

[1] This was the lowest test concentration.

[2] This was the highest test concentration.

Other Environmental Compartments

When vanadium pentoxide reaches a water body, it dissolves, and some of the vanadium ions partition into suspended PM and to bottom sediments. Thus, sediment-dwelling organisms will be exposed to vanadium. No toxicity data specific to sediment-dwelling organisms are available for this metal. In the absence of such data, the CTV for benthic organisms was conservatively chosen as being 90 mg/kg—that is, the 95th percentile of available background concentrations of vanadium in sediments from a large area in northern Saskatchewan – with the expectation that these data are more-or-less representative of sediment in Canada (see Table 10 in the Ecological Exposure Assessment section below). The values for northern Saskatchewan are in line with values available for Europe. Indeed, the European FOREGS Geochemical Baseline Mapping Programme indicates that the 10th and 95th percentiles for concentrations of vanadium in sediments are 10 and 80 mg/kg, respectively (EuroGeoSurveys 2009). No assessment factor was applied to this CTV; hence, the PNEC for benthic organisms is 90 mg/kg. It should be mentioned that a lowest effect level (LEL) of 35 µg/g-dw for vanadium was calculated by Thompson et al. (2005) based on biological field surveys in relation to uranium mining and milling activities in Canada. However, because of uncertainties associated with this LEL – related in part to the fact that the LEL is much below the 95th percentile of sediment background concentrations in the region studied – it was deemed inappropriate for use in the exposure scenarios described in this assessment.

Vanadium pentoxide enters terrestrial ecosystems as a result of atmospheric deposition. Vanadium has a low toxicity to terrestrial organisms, including plants, earthworms and birds, as shown in Table 9. The no-observed-effect concentration (NOEC) for plants ranges from 55 to 200 mg/kg as vanadium, while it is 207 mg/kg as vanadium for earthworms. Acute and chronic toxicity values for birds range from 37 to 250 mg/kg body weight (kg-bw) as vanadium.

The soil quality guideline for the protection of environmental health published by the CCME for vanadium is 130 mg/kg and is considered to be a “no- to low-” effect level (or threshold level) concentration (CCME 1999; Environment Canada 1999). It is based on the plant and invertebrate toxicity data shown in Table 9, including the data for cabbage in sandy soil; more specifically, the 25th percentile of the effects and no-effects data distribution was used to calculate the guideline, and no uncertainty factor was applied. More details on these calculations are provided in Environment Canada (1999).

The CTV chosen for chronic toxicity to terrestrial organisms for the purpose of this assessment was 130 mg/kg, based on the soil quality guideline. No assessment factor was applied to this CTV; hence, the PNEC for soil organisms is 130 mg/kg.

Table 9. Empirical acute and chronic toxicity data for terrestrial organisms

Test organism	Test compound	Test type	Soil type	Endpoint	Value (mg/kg or mg/kg-bw as vanadium)	Reference
Plants						
Cabbage *Brassica oleacera*	Soils spiked with a solution of $VOSO_4$[1]	Chronic (98 days)	Sandy soil; pH 5.35, OM 1.1%	NOEC (reduction in biomass)	60	Kaplan et al. 1990
				LOEC	80	
		Chronic (133 days)	Loamy-sand soil (pH 5.1; OM 1.9%)	NOEC (reduction in biomass)	100	
				LOEC	>100	
Radish *Raphanus sativus*	V_2O_5	Acute (3 days)	Artificial soil (68% sand, 10% silt and 22% clay; pH 4.2–4.3; OM 5.6%)	NOEC (seedling emergence)	200	Environment Canada 1995
				EC_{25}	330	
				EC_{50}	577	
				LOEC	410	
Lettuce *Lactuca sativa*		Acute (5 days)		NOEC (seedling emergence)	55	
				EC_{25}	134	
				EC_{50}	251	
				LOEC	127	
Invertebrates						
Earthworm *Eisenia foetida*	V_2O_5	Acute (14 days)	Artificial soil (68% sand, 10% silt and 22% clay; pH 4.2–4.3; OM 5.6%)	NOEC (mortality)	207	Environment Canada 1995
				LC_{25}	287	
				LC_{50}	370	
				LOEC	417	

Test organism	Test compound	Test type	Soil type	Endpoint	Value (mg/kg or mg/kg-bw as vanadium)	Reference
Birds						
Mallard *Anas platyrhynchos*	V_2O_5	Acute oral (single dose followed by a 7-day observation period)	n/a	NOEC (mortality)	62	Rattner et al. 2006
				LD_{50}	113	
	$NaVO_3$			NOEC (mortality)	62	
				LD_{50}	76	
		Chronic (dietary exposure for 4 weeks)	n/a	NOEC (sublethal effects)	250[2]	
Canada goose *Branta canadensis*	$NaVO_3$	Acute oral (single dose followed by a 7-day observation period)	n/a	LD_{50}	37	
Mammals						
See Appendix 4						

Abbreviations: EC_{xx}, the concentration of a substance that is estimated to cause some effect on xx% of the test organisms; LC_{xx}, the concentration of a substance that is estimated to be lethal to xx% of the test organisms; LD_{50}, the dose of a substance that is estimated to be lethal to 50% of the test organisms; n/a, not available; LOEC, lowest-observed-effect concentration, the lowest concentration in a toxicity test causing a statistically significant effect in comparison with the controls; n/a, not available; NOEC, no-observed-effect concentration, the highest concentration in a toxicity test not causing a statistically significant effect in comparison with the controls; OM, organic matter.

[1] Vanadium in this compound is in the oxidation state +4 as opposed to +5 for other compounds, including vanadium pentoxide.

[2] 250 mg/kg; concentration in diet.

Ecological Exposure Assessment

The exposure of ecological receptors to various forms of vanadium derived from vanadium pentoxide in the environmental compartments of concern – namely soil, water and sediments – has been assessed using a conservative, yet realistic, approach. Exposure scenarios focused on the major emission sources identified for each of the main industrial sectors described in the Releases to the Environment section of this report. When available, representative monitoring data were used to derive predicted environmental concentrations (PECs); otherwise, modelling was conducted to estimate these concentrations.

Presence in the Environment

Vanadium has been measured in a variety of locations and environmental media in Canada. As is the case with other metals, monitoring data are reported as total vanadium, without further details about its speciation. Therefore, it is not possible to say which form of vanadium was measured in samples or whether it originated from the pentoxide compound. Even though there are many sources of vanadium releases to the environment (natural and anthropogenic), the conservative assumption was made that all the anthropogenic vanadium present in environmental samples in contaminated areas was derived from vanadium pentoxide.

Worldwide, the concentrations of vanadium in relatively pristine areas are as follows: usually less than 3 µg/L in surface fresh water, 130 mg/kg in soil, 1–3 µg/L in the open ocean, 20–200 mg/kg-dw in marine sediments and 0.001–14 ng/m^3 in the atmosphere in remote areas (IPCS 2001).

Levels measured in various environmental media in certain areas of Canada are summarized in Table 10; these areas were chosen for their similarity to the areas for which exposure scenarios were developed. Although these areas are subjected to some level of anthropogenic activity, concentrations up to the 95th percentile values are generally expected to be minimally influenced by anthropogenic inputs.

Table 10. Concentrations of vanadium in selected areas[1] in Canada

Location	Medium	Concentration range	Percentiles				Sampling period	N	Analytical method	Reference
			5th	50th	90th	95th				
Northern Saskatchewan	Lake sediments	5–730 mg/kg	5 mg/kg	20 mg/kg	55 mg/kg	90 mg/kg	1978	965	AAS / aqua regia	Natural Resources Canada 1978
	Soil (till)	6–96 mg/kg	14 mg/kg	25 mg/kg	45 mg/kg	55 mg/kg	1993–1994	327	ICP-AES / aqua regia	Bednarski 1994
Southwestern Prairies	River water	0.001–10.5 µg/L	0.001 µg/L	0.223 µg/L	1.40 µg/L	3.04 µg/L	2002–2008	1445	n/a	NWRI 2009
Canadian Shield	River and lake water	0–16.1 µg/L	n/a	1.11 µg/L	2.7 µg/L	8.6 µg/L	2003–2007	3231	ICP-MS / n/a	OMOE 2009
St. Lawrence lowlands	River and lake sediments	4.5–50.9 mg/kg	7.9 mg/kg	15.3 mg/kg	n/a	33.3 mg/kg	1999–2004	97	ICP-OES / aqua regia	Szalinska et al. 2007
Atlantic shore (Nova Scotia)	Bay water	1.02–1.34 µg/L	n/a	1.16 µg/L	n/a	n/a	1998	n/a	ICP-MS / nitric acid pH 1.6	NRCC 1999
	Bay sediments	8–115 mg/kg	20 mg/kg	56 mg/kg	n/a	101 mg/kg	1988–1994	188	AAS / HF and aqua regia	Loring et al. 1996
Canada-wide	Soil (till)	5–270 mg/kg	17 mg/kg	49 mg/kg	102 mg/kg	125 mg/kg	1956–2006	7301	AAS, ICP-MS / aqua regia	Rencz et al. 2006
	Surface soil	10–90 mg/kg	n/a	38–42 mg/kg (mean)	n/a	n/a	n/a	n/a	n/a	Environment Canada 1999

Location	Medium	Concentration range	Percentiles				Sampling period	N	Analytical method	Reference
			5th	50th	90th	95th				
Manitoba, Saskatchewan, Alberta	Surface soil	5–304 mg/kg	44 mg/kg	93 mg/kg	154 mg/kg	178 mg/kg	1992	1077	AAS / HF and aqua regia	2010 personal communication from Natural Resources Canada to Ecological Assessment Division, Environment Canada; unreferenced
	Soil (till)	25–358 mg/kg	56 mg/kg	103 mg/kg	142 mg/kg	155 mg/kg	1992	672	AAS / HF and aqua regia	
	Lake sediments	21–309 mg/kg	44 mg/kg	118 mg/kg	201 mg/kg	230 mg/kg	1992	270	n/a	
	River sediments	21–189 mg/kg	36 mg/kg	77 mg/kg	121 mg/kg	135 mg/kg	1992	84	n/a	
Northern Manitoba	Surface soil	4.0–42.5 mg/kg	n/a	8.7 mg/kg (mean)	n/a	n/a	1995–2003	37	n/a	2009 personal communication from Contaminated Sites Division, Health Canad to Existing Substances Risk Assessment Burea Health Canada; unreferenced[2]
Southern Ontario	Surface soil	32–344 mg/kg	n/a	68 mg/kg	n/a	n/a	1995	294	n/a	

Abbreviations: AAS, atomic absorption spectrometry; AES, atomic emission spectrometry; ICP, inductively coupled plasma; MS, mass spectrometry; N, number of samples; n/a: not available; OES, optical emission spectrometry.

[1] These areas were chosen for their similarity with the areas for which exposure scenarios were developed.

[2] The data shown relate to soil background concentrations, not contaminated sites.

Monitoring data that were available for areas that are believed to be directly impacted by releases of vanadium pentoxide (i.e., in the surroundings of known sources of releases) were not included in Table 10 but rather are presented in the section below, along with corresponding exposure scenarios.

Exposure Scenarios

Fossil Fuel Combustion

As previously indicated in the Releases to the Environment section, the major anthropogenic source of vanadium releases to the Canadian environment is fossil fuel combustion. This source emits vanadium to air in small-sized PM, which can be expected to be deposited to terrestrial and aquatic ecosystems. As mentioned previously, in spite of the uncertainty regarding the form in which vanadium is emitted as a result of fossil fuel combustion, there is evidence from the literature that the pentoxide is one of the forms emitted.

In Canada, 17% of the total electricity generated is produced from the combustion of coal and oil by power generation plants (Environment Canada 2009b). There were 23 coal-fired electrical power generation plants in Canada in 2004, and they were responsible for 93% of the coal consumption in the country (Stone 2004). In 2000, there were nine oil-fired power generation plants (Statistics Canada 2000). These include eight heavy fuel oil–fired facilities and one petroleum coke-fired facility; other petroleum-based power generation, such as diesel generators and light fuel oil combustion turbines, are not included, as they are not expected to emit as much vanadium pentoxide. Power generation is among the most important activities in terms of fossil fuel combustion in the country; hence, it likely represents a large source of releases of vanadium pentoxide to the environment. As such, exposure scenarios for both coal-fired and oil-fired power plants were developed and are described below.

Coal-fired Power Generation Plants

An area comprising a large shallow lake located 65 km west of Edmonton, Alberta, the Wabamun Lake area, was chosen to represent a realistic worst-case exposure scenario. This area contains four coal-fired power generation plants that are located within a 20 km radius of the lake. Two of these plants, along with their respective coal mines, are located by the lakeshore and discharge their wastewater into cooling ponds connected to the lake (Stantec Consulting Ltd. 2003). One of these two plants is the biggest coal-fired power plant in Canada (Statistics Canada 2000). The third and fourth plants are located about 10 and 20 km southeast of Wabamun Lake, respectively. These plants may not necessarily affect the lake through atmospheric fallout, since the dominant winds in this area blow from the northwest direction (Windfinder 2009). However, they can affect surrounding terrestrial ecosystems.

Releases resulting from coal mining activities as well as atmospheric fallout from the power plants are expected to affect the surrounding environment and be reflected in monitoring data. There are no other major industries along or close to the lake; hence, above-background levels of vanadium measured in the various environmental media in this area can reasonably be attributed to the activities of the power plants. Natural Resources Canada (Goodarzi and Sanei 2002) and Alberta Environment (2002, 2003) have conducted monitoring studies in the Wabamun Lake area; consequently, there are many monitoring data available with which to characterize the exposure to vanadium in this ecosystem.

An air deposition survey was conducted in the Wabamun Lake area between 1994 and 1997 by Goodarzi and Sanei (2002) in order to monitor the impacts of the power plants. This survey indicated an average concentration of 2.59 mg/kg in a moss metal biomonitor following a 4-month exposure period during the winter. The mean concentration in the moss just before the exposure period was 1.34 mg/kg. These numbers indicate that vanadium was deposited to soil during the exposure period. Atmospheric deposition of vanadium over a 4-month period was a maximum of 0.82 g/ha, with an average value of 0.37 g/ha. During summertime, the deposition rate dropped to a mean of 0.20 g/ha. The influence of wind was reflected in the data, as the highest values for atmospheric deposition of vanadium were found southeast of the main two power plants, in the direction of dominant winds. Soil samples collected in various locations in the area surrounding the lake showed a mean vanadium concentration of 63 mg/kg. The highest concentration, 116 mg/kg, was measured at a sampling station located downwind from all four power plants. The analytical method used an extraction with aqua regia.

The Alberta Ministry of the Environment conducted a water quality and sediment survey of Wabamun Lake in 2002 (Alberta Environment 2002, 2003). The highest measured concentration of vanadium in water was 20 μg/L at the ash pond effluent discharge (filtered water). The mean concentration for the rest of the lake (i.e., more than 100 m away from the effluent) was 1 μg/L (Alberta Environment 2002). Based on the speciation modelling conducted for Wabamun Lake (Table 4 and Appendix 1), nearly all dissolved vanadium is expected to be in the form of $H_2VO_4^-$ and HVO_4^{2-}, with a very minor contribution from $HS–V_1$ complexes (<1%). The sediments in Wabamun Lake are rich in silt, fine sand and organic matter, and they also contain coal flakes (Alberta Environment 2002). The highest vanadium concentration measured in sediments during a survey conducted in 2002 was 105 μg/g. The mean sediment concentration in the whole lake was 70 μg/g (Alberta Environment 2003).

Based on the monitoring data presented above, the following realistic worst-case PECs were chosen to quantify the exposure of ecological receptors to vanadium for a coal-fired power plant scenario: 116 mg/kg for soil, 2 μg/L for surface water and 105 μg/g for sediments (all concentrations as total vanadium). The value of 2 μg/L for surface water was obtained by dividing the concentration monitored at the ash pond effluent (20 μg/L) by a factor of 10 to account for dilution. This calculation is intended to represent a water concentration at a location close to the point of effluent discharge to the lake; the value

obtained is actually very similar to the mean concentration measured in the whole lake (i.e. 2 μg/L vs 1 μg/L).

Oil-fired Power Generation Plants

Most oil-fired power plants used to burn heavy oil in their furnaces. However, over the last two decades, these power plants have started to use lighter fuels such as natural gas in order to reduce air pollution (Statistics Canada 2007b). As mentioned previously in this report, vanadium is typically found in heavy fuel oils. Therefore, present releases of vanadium by oil-fired power plants are likely less than they were two decades ago. Nevertheless, because vanadium is persistent and because some plants stopped burning heavy oil only recently, historical contamination due to emissions from oil-fired power plants is considered.

In order to choose an appropriate exposure scenario, the eight heavy fuel oil-fired power plants surveyed by Statistics Canada in 1999 were all considered (Statistics Canada 2000). Based on both the data collected under the section 71 survey and the data available from the NPRI, the power plant for which the highest releases of vanadium were reported for 2006 was chosen for the exposure scenario (Environment Canada 2009a, NPRI 2009). It should be noted that for the power plant chosen, as well for other power plants, releases were reported in terms of total vanadium and not vanadium pentoxide specifically since the portion of the later in the combustion products is unknown. Hence, the releases reported overestimate the actual quantity of vanadium pentoxide emitted to the environment.

The power plant chosen for the exposure scenario is Tufts Cove Generating Station, a public power plant owned by Nova Scotia Power and located in Halifax Harbour, close to Dartmouth. The oil that this plant purchases and consumes contains less than 300 ppm of vanadium (Environment Canada 2009c). It can be noted that due to an increasing shift from heavy fuel oil to natural gas as a combustible, the emissions of vanadium by this plant have decreased since 2006, as illustrated by NPRI data (NPRI 2009).

The presence of the power plant may result in two types of releases to the harbour: direct releases of treated effluent and cooling water, and atmospheric deposition from stack emissions (Nova Scotia Power Inc. 2009). While fly ash contains forms of vanadium pentoxide (Environment Canada 2009a), it is unknown whether effluents also contain them. A wind rose that was developed close to the power plant indicates that dominant winds blow away from the harbour (Nova Scotia Power Inc. 2009), so atmospheric deposition is expected to occur mainly in surrounding terrestrial environments. Unfortunately, no monitoring data for concentrations of vanadium in soil could be found for these environments. Although the extent to which atmospheric deposition could also affect the harbour has not been quantified, data for this habitat were available and are thus considered. The mean concentration of vanadium in sediments collected in the immediate vicinity of the power plant in 2007 was 139 μg/g, while the highest concentration was 270 μg/g (Nova Scotia Power Inc. 2009). All the water samples collected in the area were below the detection limit of 2 μg/L (Jacques Whitford

Environment Ltd. 2001). Again, speciation modelling conducted for seawater (Table 4 and Appendix 1) indicates that nearly all dissolved vanadium should be in the form of the inorganic species $H_2VO_4^-$ and HVO_4^{2-}.

Based on these monitoring data, the PECs for exposure of marine organisms to vanadium were chosen to be 2 µg/L for water and 270 µg/g for sediment (as vanadium). It is recognized that these values are very conservative since they represent total vanadium and since they likely result from other sources of pollution such as untreated municipal wastewater and heavy maritime transport. Regarding the latter, since heavy oil is the main fuel burned by marine vessels, these vessels could probably be expected to release vanadium in the pentoxide form, depending on the combustion conditions prevailing in engines.

Other Fossil Fuel Combustion Facilities

Petroleum refineries and oil sands projects also burn fossil fuels for their energy supply; however, no exposure scenario was developed for these facilities because releases of vanadium from them are expected to be similar to those from power generation plants.

Metallurgy

As mentioned previously in this report, vanadium pentoxide is used by one metallurgical plant in Canada to make ferrovanadium alloys (Environment Canada 2009a). Except for a minor portion that may be lost while handling the raw material (vanadium pentoxide pellets), all of the substance is transformed into alloys and is therefore considered inert at that point for the purpose of the ecological assessment. Indeed, alloys are not expected to release significant quantities of vanadium into the environment, and the vanadium present in the alloys is likely not in the pentoxide form due to the metallurgical process used. Under the section 71 survey, the metallurgical plant reported atmospheric emissions of 19 kg of vanadium pentoxide in 2006. Because the quantity released is low compared with releases from other sources, no exposure scenario has been developed for metallurgical works.

Metal and Mineral Smelters and Ores

Uranium Mining and Milling

Uranium ores can contain vanadium as a trace element in an oxide form (Golightly et al. 1983). There are three uranium mills operating in Canada, all located in northern Saskatchewan. Together they produce about 11 000 tonnes of uranium per year (Natural Resources Canada 2007). As a first step following mining, the uranium ore is crushed and processed in mills that are located close to the mine. During the milling process, most of the impurities are removed from the ore to produce a uranium concentrate called "yellowcake" (Natural Resources Canada 2007; UPA 2010). Milling involves a series of physical and chemical treatment steps, including leaching of the ores and solvent extraction, to extract the uranium (IAEA 1998; UPA 2010). Given the conditions and

eluting solutions used during the solvent extraction phase of the milling process, vanadium pentoxide is expected to be produced in uranium mills (IPCS 2001; Atomix 2003). However, other forms of vanadium can also be released in the vicinity of uranium mines due to extraction operations, transportation and mineral waste disposal. Hence, there is uncertainty about the extent to which the concentrations of vanadium measured in uranium mining and milling areas are related to vanadium pentoxide formed during milling or to other forms of vanadium present in the ore and waste rock. Due to this major uncertainty, uranium mining and milling activities will not be considered further in this assessment. However, these sources would be important in any future moiety-based assessment that could be conducted for all vanadium compounds.

Phosphate Rock Mining and Smelting

Depending on its provenance, phosphate rock contains vanadium at concentrations between 3 and 300 mg/kg (Mortvedt and Beaton 1995). Only one company extracts phosphate rock to produce fertilizers in Canada. According to data reported to the NPRI, this facility did not release vanadium in 2006 (NPRI 2009). From northern Ontario, where it is extracted, the phosphate rock is transported to Alberta, where it is processed. Phosphorus is extracted from phosphate rock by a salt-roasting process that also removes impurities; this process is likely to result in vanadium pentoxide emissions. The NPRI data indicate that 3 kg of vanadium were released to water in 2006, and 667 kg were disposed of by underground injection (NPRI 2009). Because these releases are low compared with those from other sources, no exposure scenario has been developed for this sector.

Other Mining and Smelting Activities

Given the importance of other sectors of the (base) metal mining industry in Canada, vanadium may also be released as a by-product from mining and processing of these metals. Borgmann et al. (2007) measured vanadium concentrations ranging from 0.36 to 0.82 µg/L (filtered water) in two rivers impacted by metal mining (copper, zinc and gold) in the Abitibi–James Bay region, Quebec; the concentrations for control sites located in the same rivers ranged from 0.33 to 0.92 µg/L. This suggests that vanadium is not significantly released as a by-product of the mining of these metals in this region.

Pulp and Paper Mills and Sawmills

To supply their energy needs, pulp and paper mills and sawmills burn a variety of fuels that emit vanadium pentoxide to the atmosphere. No monitoring data for vanadium concentrations in the vicinity of such mills could be found. Therefore, these concentrations were modelled. To do this, the atmospheric dispersion model AERMOD (2009) was run to describe the atmospheric dispersion and deposition of vanadium-associated particulate matter from air stacks. The model was run to estimate emissions over 5 years of industrial activity. The following values were used as input to the model:

- plant operating days: 320 days/year;

- stack activity: 18 hours/day;
- stack height: 68.7 m;
- stack diameter: 3.01 m;
- air velocity in stack: 12.75 m/s
- air stack temperature: 463.5 K
- vanadium emission rate: 0.426 g/s

The first two parameters listed above are default values for a generic industrial facility (Environment Canada 2008). The other parameters, except emission rate, are mean values that were calculated from 57 pulp mills registered to the NPRI (2009). Finally, the emission rate was based on the highest release reported among all the pulp mills that responded to the section 71 survey (Environment Canada 2009a). The particle size distribution estimated by Linak and al. (2000) for emissions of vanadium resulting from fuel oil combustion was also entered in the model. The model was run to estimate deposition rates up to a radial distance of 5000 meters from the stack.

The model estimates indicate that peak deposition of vanadium on ground will occur at a point located at about 2000 meters from the stack. At this point, the cumulative deposition on soil after a 5-year long emission period is 0.0486 g/m^2. The range of deposition values for this 5-year period for the whole area modeled (i.e. a circle having a radius of 5000 meters, with modeled stack in the centre) is 0.012 to 0.0486 g/m^2.

The pulp mill that reported the highest releases of vanadium to air under the section 71 survey has been active for the past 102 years. Assuming a soil density of 1.5 g/cm^3 and a soil depth of 10 cm, soil vanadium concentrations ranging from 1.6 to 6.6 mg/kg were estimated to result from 102 years of deposition. The PEC for soil based on this modelled worst-case scenario was calculated to be 49 mg/kg (rounded value). To obtain this number, the mean surface soil concentration background value of 42 mg/kg (upper value of the range) across Canada (Environment Canada, 1999; Table 10) was added to the highest concentration expected (6.6 mg/kg) based on modelled input of vanadium pentoxide to soil.

Leaching from Landfills

It is recognized that the vanadium present in manufactured items that contain vanadium pentoxide and that are disposed of in landfills have the potential to leach into groundwater in the form of dissolved vanadium species. This could constitute a source of exposure for this environmental medium. Monitoring data collected under the Chemicals Management Plan monitoring program indicate that total vanadium concentrations in leachate from landfills are <0.01–0.03 mg/L. These data are based on a single sampling episode conducted at 10 different landfills in Canada. Knowing that the sum of all landfill leachate generated in Canada is estimated to be 8 428 000 m^3/year (Conestoga-Rovers & Associates 2008), 253 kg of vanadium is potentially released annually from landfills through leaching. Because the highest concentration measured in leachates from landfills (which will likely be diluted after reaching and mixing with groundwater) is four times less than the PNEC value for chronic toxicity to aquatic organisms (0.120 mg/L), a more

detailed exposure scenario for groundwater has not been developed, as no concern is expected for this environmental compartment.

Emissions from Automobile Catalytic Converters along Roadways

Vanadium pentoxide can be emitted from automobile catalytic converters due to abrasion, which may represent a highly dispersive source of vanadium to the urban environment. Conservatively assuming that all vanadium emitted by cars is from this source, monitoring data for vanadium concentrations in soil along roadways were gathered from the published literature. Tsukatani et al. (2002) measured the mutagenic activity of urban roadside soils in Japan in relation to heavy metals. The road studied was a busy one, with about 50 000 vehicles travelling on it every day. The concentrations of vanadium in the soils sampled from the median strip to about 150 m away from the road ranged from 6 to 53 mg/kg and tended to decrease with distance from the road. A study conducted in Germany showed a similar vanadium concentration (51 mg/kg) measured at the edge of an urban road (3200 vehicles per day; Munch 1993). The background concentration for a forest soil sampled in this area was 27 mg/kg. No monitoring data were found for Canada. The data from the study conducted in Japan were chosen as a worst-case exposure scenario, even though there are areas in Canada where the traffic volume is higher (e.g., > 400 000 cars daily on Highway 401 near Toronto, Ontario). Therefore, a PEC for soil of 53 mg/kg was chosen for this exposure scenario.

Even though the scenario above was selected for the exposure assessment, generic calculations were done for Canada to estimate releases of vanadium from vehicles. Schmid et al. (2001) estimated that emission factors for vehicle-derived PM can reach a maximum of 107.9 mg/vehicle-km. The vanadium content in vehicle-derived PM is between 0.15 and 15.14 mg/kg PM (Furusjö et al. 2007). Using the latter value along with the one estimated by Schmid et al. (2001), a high-end emission factor for vanadium in vehicle-derived PM was calculated as being 1.63 µg/vehicle-km. Knowing that 332.2 billion vehicle-kilometres are driven in Canada each year (Statistics Canada 2008), it was estimated that 541.5 kg of vanadium could be released to soil surfaces adjacent to roads across the country. Given that there are 1 042 000 km of roads in Canada (Transport Canada 2008), it was estimated that an average of 520 mg of vanadium is released every year along each kilometre of road in Canada. Arbitrarily considering that PM will deposit over a 10 cm soil depth over a 1-m wide strip away from a road, a resulting added vanadium soil concentration of 3.5×10^{-3} mg/kg is obtained. This number is based on the assumption that the traffic is evenly spread across the country. A more realistic calculation was made for a hypothetical 1 km section of Highway 401 near Toronto, Ontario (>400 000 cars daily). The resulting numbers are a vanadium emission of 652 mg/km per year and an added vanadium soil concentration of 4.3×10^{-3} mg/kg. These numbers are very small in relation to typical background values for surface soils (mean of 38-42 mg/kg; Table 10) and suggest that emissions from automobile catalytic converters are likely not of environmental concern.

Industries manufacturing catalytic converters for cars are likely to release vanadium pentoxide as a result of handling the substance during the manufacturing process; however, no such industry has reported to the section 71 survey.

Phosphate Fertilizers

As mentioned above, vanadium pentoxide is found in phosphate fertilizers as a result of salt roasting of phosphate rock. Therefore, vanadium pentoxide will be introduced in agricultural soils through the application of fertilizers. Mortvedt and Beaton (1995) estimated the input of vanadium to soil as being 12 g/ha when applied at a rate equivalent to 20 kg of phosphorus per hectare. Assuming that a high rate of 75 kg/ha of phosphate fertilizers is applied during one growing season for highly demanding crops (e.g., cereals; OMAFRA 2009), this would translate into an added vanadium concentration in soil of 0.015 mg/kg, assuming a soil density of 1.5 g/cm^3 and a mixing depth of 0.2 m (plough depth) (Environment Canada 2005). If a period of 50 years of consecutive applications of fertilizers to an agricultural soil is arbitrarily chosen, this would result in an added concentration of vanadium of 0.75 mg/kg. This value was added to the mean background concentration for soil of 42 mg/kg (upper value of the range of means; Table 10) to give a total value of 43 mg/kg, which is used to represent a conservative soil PEC for this scenario.

Characterization of Ecological Risk

The approach taken in this ecological screening assessment was to examine various supporting information and develop conclusions based on a weight of evidence approach and using precaution as required under CEPA 1999. Lines of evidence considered include results from a conservative, but realistic, risk quotient calculation, as well as information on persistence, bioaccumulation, toxicity, sources and fate of vanadium pentoxide and associated moieties of concern.

The dispersive releases of vanadium pentoxide into the Canadian environment are due mainly to atmospheric emissions from various industrial activities, in particular fossil fuel combustion. Deposition of vanadium pentoxide to surrounding terrestrial and aquatic ecosystems then occurs. Because vanadium pentoxide is soluble, it will dissolve in contact with moisture in these ecosystems and yield a variety of dissolved species, depending on the environmental conditions, such as pH and E_h. Overall, vanadium will be found in water but will eventually partition to sediments. It is also expected to be mobile to some extent in soil.

Pentavalent vanadium (and, by extension, vanadium pentoxide) is considered infinitely persistent. Experimental evidence shows that pentavalent vanadium has a low bioaccumulation potential in both aquatic and terrestrial ecosystems. In addition, vanadium seems to have a low potential for biomagnification in natural food webs. Pentavalent vanadium has been demonstrated to have moderate to high acute and chronic toxicity to aquatic organisms and low chronic toxicity to terrestrial organisms.

A risk quotient analysis, integrating conservative, yet realistic, estimates of exposure with toxicity information, was performed for both soil and water compartments to determine whether there is potential for ecological harm in Canada. Site-specific industrial scenarios based on monitoring data were developed for the most important sources of releases of vanadium pentoxide to the environment. Other exposure scenarios were developed for emissions from automobile catalytic converters as well as phosphate fertilizer application. The PEC values derived for each scenario for each medium of concern are summarized in Table 11. It should be noted that in aquatic ecosystems, the bioavailability of metals may be modified by certain factors, such as pH, hardness and DOM. For this screening assessment, it was assumed that the bioavailability of vanadium in surface water is approximately equal to that in toxicity tests (i.e., typically relatively high). Thus, the PEC values for water were based on total dissolved vanadium. This assumption is not overly conservative because, based on realistic speciation modelling conducted for representative water bodies in Canada, the vanadate ions are the dominant forms in water, and these forms are highly bioavailable due to ion mimicry with the phosphate anion.

As described previously, PNEC values were derived for aquatic pelagic and terrestrial organisms based on the CTVs, which in turn were based on the numerous empirical toxicity data available. Because of the lack of data on toxicity to sediment-dwelling biota, the PNEC for benthic organisms was based on a high-end (95th percentile) background concentration of vanadium in sediments. PNEC values are summarized in Table 11.

Table 11. Risk quotients (RQs) calculated for the different exposure scenarios, based on highest measured or predicted exposure concentrations

Location	Industry	Medium	PEC[1]	PNEC	RQs
Wabamun Lake (Alberta)	Coal-fired power plants	Soil	116 mg/kg	130 mg/kg	0.89
		Surface fresh water	2 µg/L	120 µg/L	0.02
		Freshwater sediments	105 mg/kg	90 mg/kg	1.2
Halifax Harbour (Nova Scotia)	Oil-fired power plants	Soil	n/a	n/a	–
		Surface salt water	2 µg/L	5 µg/L	0.4
		Saltwater sediments	270 mg/kg	90 mg/kg	3.0
Pulp mill (modelled scenario; location of the mill assumed to be on Canadian Shield)	Pulp and paper mills and sawmills	Soil	49 mg/kg	130 mg/kg	0.38
Japan, on the side of a busy road	Automobile catalytic converters	Soil	53 mg/kg	130 mg/kg	0.41
Canada-wide, agricultural	Phosphate fertilizer	Soil	43 mg/kg	130 mg/kg	0.33

Location	Industry	Medium	PEC[1]	PNEC	RQs
areas	application				

Abbreviation: n/a, not applicable.

[1] As a worst case, the background vanadium concentration was not subtracted from the PEC values in those cases where the PEC values were based on monitoring data.

The resulting risk quotients (PEC/PNEC) shown in Table 11 range from 0.02 to 3.0.

For all scenarios considered, the PEC for soil is lower than the PNEC. It is also lower than the 95th percentile of background concentrations in till Canada-wide (125 mg/kg; Table 10). Even though till is not a horizon that usually supports life, and even if its composition may differ from that of topsoil, this layer is often used to establish geochemical backgrounds for metals. These background values are relevant because one can expect that organisms in the wild are adapted to natural levels of metals present in their environment.

The risk quotient analysis suggests that the concentrations of vanadium in freshwater and saltwater sediments for the fossil fuel combustion scenarios (Wabamun Lake and Tufts Cove) are high enough to potentially cause harm to benthic communities (RQs of 1.2 and 3.0, respectively). However, the PEC values for these scenarios are based on maximum total vanadium concentrations, and account for all sources of release. Given additionally that the PNEC for sediments is expected to be quite conservative being based on the upper end of background concentrations rather than on low-effect endpoints from toxicity tests (which are expected to be higher than background concentrations), the risk for ecological harm at these sites is considered overestimated.

Overall, harm to aquatic and terrestrial organisms resulting from exposure to vanadium pentoxide is unlikely based on an analysis of sites receiving atmospheric fallout or liquid effluent containing vanadium derived from vanadium pentoxide. This information suggests that releases of vanadium pentoxide are unlikely to be causing ecological harm in Canada.

Uncertainties in Evaluation of Ecological Risk

There is uncertainty regarding the quantity of vanadium pentoxide released by various industrial activities in Canada, especially for incidental releases. Indeed, some facilities may not be aware that they release this substance (e.g., upon combustion of fossil fuel). In addition, there is uncertainty about the form in which vanadium is released to the environment. For instance, submissions were received under the section 71 survey from respondents who provided data on their releases of vanadium; however, they did not know whether it was in the form of vanadium pentoxide. Even though much evidence found in the literature indicates that vanadium pentoxide is presumably the major form released from most of the industrial processes addressed in this assessment (i.e., fossil fuel combustion, petroleum refining, phosphate mining), there is still uncertainty about the contribution of vanadium pentoxide to PECs in some cases. This uncertainty was addressed by conservatively assuming that most of the vanadium released by these processes is vanadium pentoxide. However, this was not done for uranium mining and

milling because the uncertainty regarding the relative importance of the forms of vanadium released by this industrial activity was deemed too high.

Regarding the exposure scenario for the pulp mill, there is uncertainty in the PEC calculations that are based on deposition modelling. Even though this uncertainty errs on the very conservative side, the risk quotient analysis still indicates that no harm would be caused to terrestrial ecosystems.

Another area of uncertainty pertains to the effects of vanadium on benthic organisms. Indeed, the PNEC for these organisms was based on the 95th percentile of background concentrations of vanadium in sediments rather than on ecotoxicological data. However, the 95th percentile is expected to be a conservative estimate of an effect threshold for both marine and freshwater organisms.

Finally, there is uncertainty about reaching a conclusion outside the context of a more general moiety-based assessment for vanadium, considering all potential sources of vanadium. Indeed, many vanadium-containing substances besides vanadium pentoxide can contribute to the total release of vanadium moieties to the environment. In the context of this Challenge assessment, only sources of vanadium that are believed to be related to vanadium pentoxide were considered. The other sources of vanadium to the environment were covered in some cases by using monitoring data to characterize exposure. The conclusion reached in this assessment for vanadium pentoxide does not preclude the inclusion of this substance in any future moiety-based assessment that could be conducted for vanadium and its compounds.

Potential to Cause Harm to Human Health

Exposure Assessment

Environmental Media and Food

Since the combustion of fossil fuels such as oil and coal is the primary source of vanadium pentoxide, it is expected that inhalation of ambient air and particulate matters (PM) would be a predominant route of exposure vanadium pentoxide for the general population of Canada. Unfortunately, no data were found for levels of vanadium pentoxide in air, other environmental media, or in food. Therefore, for the purpose of this screening assessment, estimates of exposure to vanadium pentoxide were derived using data for levels of vanadium in these sources. This approach likely overestimates actual exposures to vanadium pentoxide from environmental media and food.

Air monitoring was conducted by Environment Canada at 53 locations across Canada at various time points between 1986 and 2008. Vanadium was measured in PM with an aerodynamic diameter of $\leq$2.5 μm (i.e., the respirable fraction or $PM_{2.5}$ fraction). The air monitoring data indicate ambient air concentrations of vanadium to be in the range of <0.1–525 ng/m^3. For data obtained between 2004 and 2008 from nine locations across

Canada, the maximum vanadium concentration in the $PM_{2.5}$ fraction was 59.5 ng/m^3 and was found in Montreal, Québec (2009 personal communication from Environment Canada to Health Canada; unreferenced). Similar values were reported in other cities. For example, in New York City, a mean vanadium concentration of 14.5 ± 15.1 ng/m^3 was reported in lower Manhattan (Peltier and Lippmann 2009); and for Mexico City, a heavily industrialised city, a mean personal exposure vanadium concentration of 23 ± 12 ng/m^3 was reported in the heavily industrialized city (Riveros-Rosas et al. 1997). The vanadium concentration in Montreal, 59.5 ng/m^3 is used to derive exposure estimates from air, because this is the most recent Canadian data. Emissions from use of vanadium pentoxide to produce ferrovanadium are expected to be minimal since, as previously noted the furnaces in the facility operate at 1800 ^{o}C, the vanadium emitted through stacks is likely not vanadium pentoxide and any emissions to air are limited by the use of baghouse pollution control devices.

Recent data on the concentrations of vanadium in drinking water are very limited. In Canada, measured levels of vanadium in 2004 samples of drinking water from the province of Saskatchewan between the years 1976 and 2007 were available from Saskatchewan Environment (2007 personal communication from Saskatchewan Environment to Health Canada; unreferenced). According to the information provided, a typical vanadium concentration of 1 µg/L was observed in the 2001–2007 calendar years, together with a minimum vanadium concentration of 0.4 µg/L in one sample from the Wakaw-Humboldt regional water supply and a maximum concentration of 13 µg/L vanadium in one sample from the Hafford distribution system. In the United States, a study of municipal waters from 26 locations nationwide in 1999 identified vanadium at concentrations between 0.5 and 6.3 µg/L (Miller-Ihli and Baker 2001). The study by Soldi et al. (1996) found no significant fallout of vanadium from the oil refinery to nearby groundwater and reported concentrations of vanadium in the groundwater ranging between 400 and 500 µg/L. More recently, a study of drinking water in Argentina found the presence of vanadium at concentrations in the range of 5.52–8.11 µg/L (Wuilloud et al. 2005). The vanadium concentration of 1 µg/L in drinking water in Saskatchewan was used to derive the exposure estimate for vanadium pentoxide in drinking water, because this is the typical concentration observed in recent years.

Vanadium is naturally found in soil and rocks, but it is expected that vanadium from anthropogenic sources, such as the combustion of fossil fuels and wood fuels, will deposit to soil, particularly in areas close to point sources, and be taken up by plants (Preda and Cox 2002; Ambrozini et al. 2009; Bañuelos and Ajwa 1999). Studies of vanadium uptake as a function of pH and redox potential of the nutrient solution suggested that under normal conditions, VO^{3-} and VO^{2+} were the predominant forms of vanadium taken up by plant roots from soil through passive diffusion (Welch 1973; Tyler 2004). The application of sewage sludge and fly ash to agricultural soils is also likely to further increase the vanadium content of soils (Dungan and Dees 2007; Kuzmick et al. 2007). Concentrations of vanadium in surface soil samples collected in 1992 from Manitoba, Saskatchewan and Alberta ranged from 5 to 304 µg/g, with the 95th percentile concentrations of 178 µg/g, respectively; soil concentrations of vanadium in long-term monitoring sites (1995–2003) established across the province of Manitoba ranged from 4.0 to 42.5 µg/g, with a mean

concentration of 8.7 µg/g; in Ontario, vanadium concentrations in surface soils collected in 1995 ranged from 32 to 344 µg/g, with mean and median vanadium concentrations of 70 ± 27 and 68 µg/g, respectively (Table 10; 2009 personal communication from Contaminated Sites Division, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada; unreferenced).

In the city of Ottawa, the maximum vanadium concentrations in house dust and garden soil samples collected in 2001 were 43.6 µg/g and 85.6 µg/g, respectively (Rasmusen et al. 2001). Recently, an extensive study of trace metals in sediments and mangroves in Queensland, Australia, noted vanadium concentrations of 6–171 µg/g in old, preindustrial sediments, which were consistent with the levels observed in bedrock where vanadium is expected to occur naturally (Preda and Cox 2002). The vanadium concentrations of more recent estuarine sediments and soils were lower, at 2–65 µg/g and 4–80 µg/g, respectively. However, upon closer examination of the data using factor analysis and a correlation matrix, it was found that iron had the greatest influence in controlling the distribution of trace elements in soil and sediment and, more importantly, that trace metal enrichment (including vanadium) had in fact occurred in areas with increased human activity. The study by Colina and co-workers (2005) of sediment from Lake Maracaibo, Venezuela, identified vanadium(IV) as the predominant species in sediment and a total vanadium concentration of 1.7–113.5 µg/g. The levels of vanadium in sediment were considered high and attributed to the petroleum industry (Colina et al. 2005). More recently, a study of soil from a rural forest in Denmark found accumulation of heavy metals, including vanadium, in soil, but little difference between the concentrations of vanadium in topsoil and subsoil, where average concentrations were 5.9 µg/g and 5.0 µg/g, respectively (Hovmand et al. 2008). An Italian study of the environmental and health impacts of emissions from a thermoelectric company and oil refinery on the surrounding area reported that the level of vanadium in soil ranged between 64 and 122 µg/g-dw (Triolo et al. 2008). The maximum vanadium concentration in Canadian surface soil samples collected in 1995 of 344 µg/g was used to derive an estimate of exposure from soil.

The presence of vanadium in soil and food resulting from both the metal's natural occurrence and its release as a result of anthropogenic activities is expected to contribute to the general population's exposure to vanadium. However, no Canadian data on the levels of vanadium in food were identified. Several international studies reported levels of vanadium in a variety of food items and food groups, including milk, beverages, shellfish, nuts and vegetables. Some of these studies also included levels of vanadium in food grown in industrialized areas with significant anthropogenic activity. The concentrations of vanadium in foods potentially contaminated from such activity are not considered representative of vanadium concentrations that are likely typical in Canadian foods. Survey data from the United States (Pennington and Jones 1987) and the UK (Evans et al. 1985) were also examined to present the range of intake from various diets.

In Spain, there were no significant differences in the levels of vanadium in different types of cows' milk (Lopez-Garcia et al. 2009); the highest level was observed in whole milk, which had a mean vanadium concentration of 0.35 ± 0.05 µg/L was observed, followed

by slightly lower levels of 0.32 ± 0.04 µg/L and 0.25 ± 0.04 µg/L were measured in semi-skimmed and skim milk, respectively. The maximum level of vanadium in infant formula (6.2 ± 0.4 ng/g) was in a prebiotic formulation (Lopez-Garcia et al. 2009).

Beer, white wine and tea have been studied for their vanadium content, including beer, white wine and tea (Wryzykowska et al. 2001; Moreda-Pineiro et al. 2003; del Mar Castiñeira Gomez et al. 2004). Concentrations of vanadium in beer in Poland ranged between 8 and 55 µg/L (Wyrzykowska et al. 2001). The authors suggested that the high levels of vanadum might be attributed to notable historical use of the substance in the manufacture of battery systems and the subsequent accumulation of vanadium in environmental media (Wyrzykowska et al. 2001).

The concentration range of vanadium in 127 white wine samples from four regions in Germany was 1.2–364 µg/L (del Mar Castiñeira Gomez et al. 2004).

Concentrations of vanadium in tea leaves (i.e. not the beverage as consumed) were highly variable. The highest mean concentration was in tea leaves from China (.44 ± 0.31 µg/g; Moreda-Pineiro et al. 2003). The lowest mean concentration was for tea leaves in Lipton (London, UK) Yellow Label tea bags (0.18 ± 0.04 µg/g; Ødgård and Lund 1997). For these Lipton tea bags the extraction efficiency of vanadium to a tea infusion was reported to be low (5%). However, an extraction efficiency of 40.5% was observed for an infusion of nettle (*Urticae folium*) tea bags (Stanowice, Poland; Lozak et al. 2002).

With regard to individual food items, Gundersen and co-workers (2001) found vanadium in tomatoes grown in Demark at concentrations ranging from 0.09 to 0.126 ng/g fresh weight (g-fresh weight). They noted no significant difference between the concentrations of vanadium in tomatoes grown in soil and in those grown in greenhouses on rockwool. A study of wild berries (lingonberries or *Vaccinium vitis-idaea* L.) from northern Finland, reported that the maximum level of vanadium (0.036 µg/g-wet weight) was found in lingonberries grown near ferrochrome and steel manufacturing sites (Pöykiö et al. 2005). Svoboda and Chrastný (2008) recently performed an extensive study of 22 varieties of edible mushrooms grown in a rural area in the Czech Republic and noted vanadium concentrations between 0.04 and 1.66 µg/g dry matter. They also observed that vanadium was accumulating in the *Amanita muscaria* species. The mean concentrations of vanadium in the leaves and roots of parsley from Araraquara, Brazil, were identified as 2.25 µg/g and 3.56 µg/g, respectively (Ambrozini et al. 2009). Interestingly, the levels of vanadium in raw pistachios, as in tea, depended upon their country of origin. A study of pistachios from Turkey, Iran and California revealed vanadium concentrations between 4.1 and 20.8 µg/g-dry weight, the highest concentration being found in pistachios from the north Fandoghi region of Iran (Anderson and Smith 2005).

Two recent studies were identified that looked at the level of vanadium in a number of foods (Triolo et al. 2008; Nardi et al. 2009). The Italian study looked at the environmental and health impacts of emissions from a thermoelectric company and oil refinery on the surrounding area (Triolo et al. 2008). The level of vanadium in soil was found to range between 64 and 122 µg/g-dry weight, and vanadium was detected in

lettuce, potato, courgette, aubergine, tomato and lemon at levels between 0.003 and 0.02 µg/g-fresh weight (the highest levels were in lettuce and potato). Eight samples of olives were also studied, but vanadium was detected only in those sampled closest to the thermoelectric and oil refineries (0.032 µg/g-fresh weight).

The Brazilian study by Nardi and co-workers (2009) tested a variety of food groups (including eggs, meat, fish, dairy products, vegetables and nuts) for a number of essential elements and vanadium was identified in rice (9.6 ± 0.09 ng/g), eggs (35.3 ± 0.2 ng/g), bread (2.5 ± 0.2 ng/g), cheese (20.3 ± 1.0 ng/g) and powdered milk (34.4 ± 1.2 ng/g).

No data on the level of vanadium in breast-milk was identified

In this screening assessment, the upper-bounding estimates of daily intake of vanadium pentoxide from environmental media and food were derived by converting estimated intakes of vanadium to intakes of vanadium pentoxide. The conversion process, as well as the intakes for all age groups, are summarized in Appendix 3.

The upper-bounding daily estimates of intake of vanadium pentoxide for the general population in Canada ranged from 1.01 µg/kg-bw per day for adults 60 years old and older to 5.58 µg/kg-bw per day for children 0.5–4 years of age. As a comparison, a total diet study conducted in the United States determined that the average consumer ingested 11–32 µg of vanadium pentoxide per day (converted from 6–18 µg of vanadium per day) (Pennington and Jones 1987), while a similar study in the UK calculated the average consumer ingestion of vanadium from common diet to be in the range of 11–48 µg of vanadium pentoxide per day (converted from 6–27 µg of vanadium per day) (Evans et al. 1985). Pennington and Jones (1987) also presented the data from a number of other studies of vanadium intakes, which ranged from 11 to 2080 µg/day (converted from 6–1165 µg of vanadium per day) or the equivalent of 0.15–30 µg/kg-bw per day for an average adult. These data suggest that geography and the spectrum of foods that constitute the staple of the diet can greatly influence the average intake of vanadium.

For infants and children the highest upper-bounding estimate of exposure to vanadium pentoxide results from ingestion of soil. For adults, the highest upper-bounding estimate of exposure is from food. However, the bioavailability of vanadium pentoxide from oral ingestion of soil or food is low (Tyler 2004). Furthermore, the actual proportion of vanadium in environmental media and foods that exists as vanadium pentoxide is unknown. While it is likely to be much lower than 100% as assumed in this screening assessment, a conservative approach has been taken given the absence of adequate information about the forms of vanadium commonly found in environmental media and food.

Consumer Products

Based on available information, vanadium pentoxide is not found in consumer products used by the general population of Canada. The section 71 submission did not identify consumer products containing vanadium pentoxide (Environment Canada 2009a).

Vanadium pentoxide is not listed in the Drug Product Database (DPD) or the Therapeutic Product Directorate's internal Non-Medicinal Ingredients Database as a medicinal or non-medicinal ingredient in pharmaceutical drugs (DPD 2010; 2010 personal communication from Therapeutic Products Directorate, Health Canada, to Risk Management Bureau, Health Canada, unreferenced). Vanadium pentoxide is listed in the Natural Health Products Ingredients Database (NHPID) without a medicinal or non-medicinal role. The NHPID specifies that vanadium pentoxide will not be authorized for use in natural health products. Vanadium pentoxide is not listed in the Licensed Natural Health Products Database, thus is not present in any currently licensed natural health products (LNHPD 2010). Additionally, the Natural Health Products Directorate multi-vitamin/mineral monograph does not indicate that vanadium pentoxide is a source of vanadium in natural health products (Health Canada 2007).

The confidence in exposure estimates is low. Intakes of vanadium pentoxide were derived from estimated intakes of vanadium, which essentially assumes that all vanadium in environmental media and food is present as vanadium pentoxide. In addition, ambient air data were used as a surrogate for indoor air concentration, and data on vanadium in foods were from other countries were used as a surrogate for Canada. Some of the vanadium concentrations used for deriving dietary intake are from sites where there could be point sources of vanadium release into the environment, which could result in overestimates of exposure of the general population.

Health Effects Assessment

Appendix 4 contains a summary of the available health effects information for vanadium pentoxide. Information on other vanadium compounds has also been taken into consideration.

The International Agency for Research on Cancer (IARC 2006) has classified vanadium pentoxide as a Group 2B carcinogen (possibly carcinogenic to humans) based on sufficient evidence of carcinogenicity in experimental animals. The European Commission (1996a) has classified vanadium pentoxide as Category 3 for mutagenicity (causes concern for humans owing to possible mutagenic effects) based on positive results in a range of *in vivo* and *in vitro* assays for different vanadium compounds. The European Commission (2006) has also proposed to classify vanadium pentoxide as Category 2 for carcinogenicity (should be regarded as if carcinogenic to humans) and as Category 2 for mutagenicity (should be regarded as if mutagenic to humans), but no decision has been taken yet as the European Commission has been waiting for additional information on these two endpoints. The European Commission (1996b) has also classified vanadium pentoxide as Category 3 for developmental toxicity (causes concern for humans owing to possible developmental toxic effects). The European Commission expert panel justified the developmental toxicity classification based on a number of studies that showed that vanadium compounds have effects on the developing fetus via oral, intraperitoneal, subcutaneous and intravenous routes.

In chronic lifetime studies in both rats and mice exposed by inhalation to vanadium pentoxide, there was an increased incidence of lung tumours (adenomas and carcinomas) in both species at all tested exposure concentrations (rat: 0.5, 1 or 2 mg/m^3; mice: 1, 2 or 4 mg/m^3). However, statistically significant increases in incidence were observed only in mice. In both sexes of mice, the incidences of carcinomas and combined adenomas and carcinomas were significantly increased in all treated groups (males: lung adenoma 13/50, 16/50, 26/50 and 15/50 at 0, 1, 2 or 4 mg/m^3, respectively; lung carcinoma 12/50, 29/50, 30/50 and 35/50, respectively; lung adenoma/carcinoma combined 22/50, 42/50, 43/50 and 43/50, respectively; females: lung adenoma 1/50, 17/50, 23/50 and 19/50, respectively; lung carcinoma 0/50, 23/50, 18/50 and 22/50, respectively; lung adenoma/carcinoma combined 1/50, 32/50, 35/50 and 32/50, respectively). Although the increased incidence of lung tumours in both sexes of rats was not statistically significant, the incidence of carcinomas and combined adenomas and carcinomas in males exceeded historical control ranges at 0.5 and 2 mg/m^3 (lung adenoma 4/50, 8/49, 5/48 and 6/50 at 0, 0.5, 1 or 2 mg/m^3, respectively; lung carcinoma 0/50, 3/49, 1/48 and 3/50, respectively; lung adenoma/carcinoma combined 4/50, 10/49, 6/48 and 9/50, respectively), and the incidence of adenomas and combined adenomas and carcinomas in females was at the upper end of the range for historical controls at 0.5 mg/m^3 (lung adenoma 0/49, 3/49, 1/50 and 0/50 at 0, 0.5, 1 or 2 mg/m^3, respectively; lung carcinoma 0/50, 0/49, 0/50 and 1/50, respectively; lung adenoma/carcinoma combined 0/49, 3/49, 1/50 and 1/50, respectively) (NTP 2002; Ress et al. 2003). The NTP (2002) concluded that clear evidence of lung tumours was seen in mice in both sexes, while some evidence of carcinogenicity was seen in male rats and an equivocal response was seen in female rats.The basis for the observed interspecies differences and differences between responses in male and female rats has not been articulated.

Non-neoplastic effects observed in mice included dose-related increased incidences of inflammation, fibrosis, hyperplasia and squamous metaplasia in the respiratory tract (lungs, larynx and nose) of males and females at all dose levels, hyperplasia of the bronchial lymph nodes in females at all dose levels, decreased body weight gain in females at all dose levels and in males at 2 mg/m^3 and above, abnormal breathing in males and females at 2 mg/m^3 and above and a significant decrease in survival in males in the highest exposure group. Non-neoplastic effects observed in rats included a dose-related increased incidence of inflammation, fibrosis and hyperplasia in the respiratory tract (lungs, larynx and nose) of males and females at all dose levels, nephropathy in males exposed to 1 mg/m^3 and above and decreased mean body weights in females at 2 mg/m^3 (highest concentration tested). The lowest lowest-observed-adverse-effect concentration (LOAEC) for non-neoplastic effects was 0.5 mg/m^3, based on effects on the respiratory tract of rats (NTP 2002; Ress et al. 2003). However, at this LOAEC, some evidence of increased tumour incidence was also observed in male rats. Long-term oral or dermal studies using vanadium pentoxide were not identified.

Vanadium pentoxide was found to be genotoxic in a number of *in vivo* assays in experimental animals and humans and *in vitro* assays in hamster and human cells. In *in vivo* bioassays, a significant increase in the frequency of deoxyribonucleic acid (DNA) damage in different tissues (testes, liver, kidney, lung, spleen, heart) was observed in

mice administered vanadium pentoxide by intraperitoneal injection (Altamirano-Lozano et al. 1993, 1996, 1999). However, negative results were reported in pulmonary and bronchio-alveolar lavage (BAL) cells of female mice administered vanadium pentoxide by nose-only inhalation (Schuler 2010). Vanadium pentoxide also produced positive results in a dominant lethal assay in mice injected intraperitoneally and caused a high frequency of K-*ras* mutations in lung neoplasm cells of male and female mice exposed by inhalation (Altamirano-Lozano et al. 1996; NTP 2002). *In vivo* assays for micronuclei were positive for bone marrow cells in mice dosed intraperitoneally, subcutaneously and by inhalation, but were negative for peripheral blood cells in mice exposed by inhalation and for bone marrow cells in mice dosed orally (Si et al. 1982; Yang et al. 1986b, c; NTP 2002; Sun et al. undated). In *in vivo* assays in humans exposed occupationally to vanadium pentoxide by inhalation, mixed results were reported for DNA damage (comet assays) in leukocytes, and negative results were observed for DNA adducts in leukocytes and sister chromatid exchange in lymphocytes. Also, positive results were reported in a chromosomal instability assay in lymphocytes (Ivancsits et al. 2002; Ehrlich et al. 2008).

In *in vitro* assays, vanadium pentoxide was not mutagenic in the majority of bacterial mutation assays using *Salmonella typhimurium* and *Escherichia coli*, with and without metabolic activation, and in a gene mutation assay in Chinese hamster V79 cells, without metabolic activation (Kada et al. 1980; Si et al. 1982; Zhong et al. 1994; NTP 2002). Similarly, assays for chromosomal aberration in human lymphocytes and for sister chromatid exchange in Chinese hamster V79 cells and human lymphocytes all showed negative responses (Roldan and Altamirano 1990; Zhong et al. 1994; Sun et al. undated). However, both positive and negative results were reported in DNA damage assays (positive in human lymphocytes and negative in human mucosal epithelial cells) (Rojas et al. 1996; Kleinsasser et al. 2003). Also, vanadium pentoxide produced positive results for aneuploidy induction and inhibition of microtubule polymerization in human lymphocytes and micronucleus induction in Chinese hamster V79 cells (Zhong et al. 1994).

Fully elucidated modes of action for induction of tumours have not been developed. Vanadium pentoxide demonstrates genotoxic activity, as it causes mutation in germ cells in mice (positive dominant lethal mutation assay), induces DNA damage in humans and shows evidence for K-*ras* gene mutation in lung neoplasms of mice exposed to vanadium pentoxide by inhalation. IARC (2006) reported that different mechanisms could be responsible for the genotoxicity of vanadium pentoxide other than direct interaction of the compound with DNA. These possible mechanisms include the induction of oxidative damage leading to DNA alkali-labile sites and DNA strand breakage, the inhibition of microtubule polymerization and the inhibition of enzymes involved in DNA synthesis and DNA repair (IARC 2006). IARC (2006) did not comment on whether the mode of action for lung tumours in mice could be due to one or a combination of these genotoxic mechanisms. In the carcinogenicity studies conducted by the US National Toxicology Program (NTP 2002), the authors reported that the K-*ras* mutations identified in vanadium pentoxide–induced alveolar/bronchiolar carcinomas could be an important player in the induction of lung carcinogenesis and that the random pattern of these mutations suggests a mode of action of vanadium pentoxide that implicates oxidative damage. Further evidence to support an oxidative mode of action for V_2O_5

carcinogenicity was provided by Duffus (2007). However, based on the available evidence (no fully elucidated mode of action; possibility of one or a combination of genotoxic modes of action), genotoxicity caused by oxidative stress may not be the only mechanism involved in gene mutation and the carcinogenicity of vanadium pentoxide; therefore, carcinogenicity resulting from direct interaction with genetic material cannot be precluded.

In a rat inhalation study with exposures to vanadium pentoxide for up to 16 days, hyperplasia of the alveolar epithelium, interstitial inflammation, increases in alveolar macrophage numbers and alveolar cell proliferation were observed in the respiratory tract of female rats exposed to 1 mg/m^3 and above (NTP 2002). In a similar study in mice, a dose related increase in lung weight was observed in female mice at 1 mg/m^3 and above. This increase was accompagnied with lung effects such as a dose-dependant increased in cell proliferation, alveolar histiocytosis, subacute alveolitis and/or granulocytic infiltration (Schuler 2010). In a 3-month inhalation study in rats, increased lung weight and inflammation were noted in males, and adverse effects on the blood and epithelial hyperplasia of the lungs were observed in males and females at 2 mg/m^3 and above. Some of these effects (inflammation and epithelial hyperplasia of the lungs) were also observed at identical concentrations in a 3-month study in mice (NTP 2002). The lowest LOAEC for repeated-dose inhalation exposure was 1 mg/m^3, based on hyperplasia of the alveolar epithelium, interstitial inflammation, increases in alveolar macrophage numbers and alveolar cell proliferation in the respiratory tract of rats and lung effects in mice.

In a study in which rats were exposed orally to vanadium pentoxide in the diet at 3.7 mg/kg-bw per day for 3 weeks, histological and enzymatic alterations, including inhibition of biosynthesis, enhanced catabolism and increased use of L-ascorbic acid, were observed in the liver and kidney tissues of male rats (Chakraborty et al. 1977). Mice administered a vanadium pentoxide dose of 6 mg/kg-bw per day by gavage on 5 days/week for 6 weeks exhibited signs of immunotoxicity, as demonstrated by decreased spleen cellularity and changes in white blood cell production and function (Mravcova et al. 1993). In a 6-month rat study, in which vanadium pentoxide was administered via drinking water, dose-related immunotoxicity was observed, as demonstrated by decreased phagocytic ability of immune cells in male rats (the only sex tested) at 0.25 mg/kg-bw per day and above (Mravcova et al. 1993). No other oral studies were identified for vanadium pentoxide. In a study with exposure to sodium metavanadate via drinking water for 180 or 210 days, no effect on cardiovascular function and no changes in the brain, liver, lungs, heart and blood vessels were noted in male rats (Boscolo et al. 1994). However, in another study in which male rats were exposed to sodium metavanadate in drinking water for 3 months, dose-dependent histological changes in the spleen, kidneys and lungs were noted at 0.3 mg/kg-bw per day and above (Domingo et al. 1985). The lowest lowest-observed-adverse-effect level (LOAEL) for repeated-dose oral exposure was 0.25–0.30 mg/kg-bw per day as vanadium pentoxide or sodium metavanadate based on immunotoxicity and histological changes in the spleen, kidney and lung in male rats.

In a dominant lethal assay, the LOAEL for reproductive toxicity was 2.8 mg/kg-bw per day, the only dose tested, based on a decreased pregnancy rate (52%) in untreated

females mated with male mice exposed intraperitoneally to vanadium pentoxide (Altamirano-Lozano et al. 1996). Other effects observed in treated males at this dose were a reduction in sperm motility with treatment for 20 days or longer, a marked reduction in sperm counts with the advancement of treatment and a significant increase in the percentage of morphological abnormalities in spermatozoa after 50–60 days of treatment. No parental LOAEL was reported. Although the dominant lethal assay provides limited information on reproductive toxicity, it is not a guideline reproductive toxicity study. No other reproductive studies were identified.

The lowest oral LOAEL for developmental toxicity was 9 mg/kg-bw per day based on skeletal abnormalities in fetuses of pregnant rats dosed by gavage with vanadium pentoxide during days 6–15 of gestation (Yang et al. 1986a). The lowest oral LOAEL for maternal toxicity was also 9 mg/kg-bw per day, based on decreased maternal body weight gain (Yang et al. 1986a). No other developmental toxicity studies were identified for vanadium pentoxide.

In an acute inhalation study with exposure to vanadium pentoxide for 6 hours, the lowest LOAEC was 3 mg/m^3, based on a statistically significant reduction in lung function characterized by airway obstructive changes (increased resistance and decreased flow) and significant influx of inflammatory cells (neutrophils, eosinophils) into the lung of male monkeys (Knecht et al. 1992). Acute oral or dermal studies using vanadium pentoxide were identified, but none reported a LOAEL.

Respiratory tract effects and allergic response in humans have been investigated in several studies, predominantly in occupationally exposed workers. However, for most of these studies, concomitant exposure to other chemicals may have occurred. The more robust studies are described in Appendix 4. From these studies, no LOAEC was determined, but the findings support the evidence that inhalation to vanadium pentoxide can cause effects on the respiratory tract, asthmatic response and contact allergic dermatitis in humans.

Toxicokinetic studies on vanadium pentoxide show that it is rapidly absorbed following inhalation, but poorly absorbed through the skin or following ingestion (in rats, approximately 3% of the ingested amount of vanadium pentoxide was absorbed from the gastrointestinal tract 3 days after exposure) (IPCS 2001; IARC 2006). Elimination from the lung is initially fast, but complete only after several days. Vanadium pentoxide distribution following inhalation exposure was mainly to the bone and kidney (IARC 2006). Similarly, oral rat studies in which vanadium pentoxide was given via drinking water have shown that vanadium is distributed mainly to the kidneys, spleen, tibia and testes (IPCS 2001). Excretion of ingested vanadium pentoxide occurs mostly through the feces, while urine is the main route of excretion for absorbed vanadium (ACGIH 2001).

The confidence in the toxicity database for vanadium pentoxide is considered to be low to moderate, as adequate information is available to identify critical endpoints based on repeated-dose inhalation exposures of acute to long-term duration, with the exception of reproductive and developmental toxicity studies for that route of exposure. However,

there were limited data for effects induced by oral or dermal routes of exposure (reproductive toxicity and chronic toxicity/carcinogenicity studies).

Characterization of Risk to Human Health

Based principally on the weight of evidence–based assessments of international or other national agencies (NTP 2002; Ress et al. 2003; IARC 2006), a critical effect for characterization of risk to human health for vanadium pentoxide from inhalation exposure is carcinogenicity. Significantly increased incidences of lung tumours were observed in male and female mice in all groups following inhalation exposure. In rats, although not statistically significant, the incidence of lung carcinomas and combined adenomas and carcinomas in males exceeded historical control ranges at 0.5 and 2 mg/m^3. Genotoxicity was observed in both *in vivo* and *in vitro* assays with vanadium pentoxide in mammalian cells. Although the mode of induction of tumours in mice has not been fully elucidated, based on the weight of evidence of carcinogenicity and the genotoxicity of vanadium pentoxide, it cannot be precluded that the tumours observed may have resulted from direct interaction with genetic material.

With respect to non-cancer effects, respiratory tract effects were among the primary adverse effects seen following inhalation exposure. The LOAEC for chronic exposure in rats (0.5 mg/m^3) was selected as the critical non-cancer effect level for repeated-dose inhalation exposure, based on an increased incidence of inflammation, fibrosis and hyperplasia in the respiratory tract (lungs, larynx and nose) of male and female rats exposed for 2 years. This LOAEC has been selected as a critical effect level by the US Agency for Toxic Substances and Disease Registry (ATSDR 2009) as well, which led to the selection by ATSDR of the NTP (2002) chronic rat study as the basis for the determination of a chronic-duration inhalation estimate of exposure levels posing minimal risk to humans for vanadium. It is likely that the irritating effects of inhaled vanadium pentoxide will serve to limit human exposures subsequent to an acutely irritating episode.

From oral exposure studies, the lowest oral LOAEL for subchronic exposure was a dose range of 0.25–0.30 mg/kg-bw per day as vanadium pentoxide or sodium metavanadate. In this dose range, reductions in phagocytosis were noted in male rats in a 6-month study, while histological changes in the spleen, kidney and lung were noted in male rats after 3 months of exposure. For short-term exposure, the lowest oral LOAEL was 3.7 mg/kg-bw per day based on histological and enzymatic alterations in the liver and kidney tissues of male rats exposed for 3 weeks. Long-term oral studies were not identified in the literature.

Estimates of daily intake of vanadium pentoxide for the general population were derived from measured concentrations of vanadium in environmental media and food. Based on the reported concentrations of vanadium in environmental media and the smaller contributions from food, upper-bounding estimates of daily intake of vanadium pentoxide range from 1.01 to 5.58 µg/kg-bw per day for the general population of Canada (Appendix 3). Soil ingestion represented a principal source of exposure for most age

groups. For example, for children aged 0.5–4 years, soil ingestion represented about 71% of total vanadium exposure. Given that a significant proportion of the vanadium in environmental media (soil) and food is considered to be naturally occurring and that the speciation is unknown, it was not considered meaningful to derive margins of exposure between the exposure estimates and the critical effect level obtained following oral administration to rats.

The main source of vanadium pentoxide in air is considered to be the combustion of fossil fuels. As there was some evidence of induction of tumours at the critical non-neoplastic effect level for inhalation exposure in rats (0.5 mg/m^3), margins of exposure were not derived, as there is considered to be a probability of harm at any level of exposure.

Uncertainties in Evaluation of Risk to Human Health

This screening assessment does not include a full analysis of the mode of induction of effects, including cancer, of vanadium pentoxide, nor does it take into account possible differences in sensitivity between humans and experimental species. Also, only limited information was available concerning the potential toxicity of vanadium pentoxide following exposure via oral and dermal routes.

Since there were no data and in particular Canadian data on the levels of vanadium pentoxide in environmental media and food, confidence in the upper-bounding exposure estimates is very low. As a result of the conservative approach used in this screening assessment, these estimates are likely higher than actual exposures to vanadium pentoxide from environmental media and food.The potential exposure to vanadium via ingestion of soil was found to be substantially higher than that noted for food ingestion.

Conclusion

Based on the information presented in this final screening assessment, it is concluded that vanadium pentoxide is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends. Additionally, vanadium pentoxide meets the criteria for persistence but not the criteria for bioaccumulation potential as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

On the basis of the carcinogenicity of vanadium pentoxide, for which there may be a probability of harm at any level of exposure, and applying a precautionary approach, it is concluded that vanadium pentoxide may be entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that vanadium pentoxide meets one or more criteria under section 64 of CEPA 1999.

Where relevant, research and monitoring will support verification of assumptions used during the screening assessment and, where appropriate, the performance of potential control measures identified during the risk management phase.

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Appendix 1: Description of the speciation modelling conducted with WHAM VI, and physical and chemical characteristics of the surface waters used

Speciation of vanadium in the dissolved phase was determined using the Windermere Humic Aqueous Model (WHAM 2001; Tipping 2002). Even though this model is designed to model the interactions of natural organic matter with cations, and not with oxyanions such as vanadate, WHAM was used as a tool to describe simultaneous chemical equilibria of vanadium species in solution. The conditions for running the model are described below:

- Vanadium complexes most likely to occur in selected water types were determined from E_h–pH diagrams (Brookins 1988).
- Thermodynamic constants for vanadium–inorganic ligand interactions were obtained from National Institute of Standards and Technology (NIST) Standard Reference Database 46 (Smith and Martell 2004) and the International Union of Pure and Applied Chemistry (IUPAC) Stability Constants Database (IUPAC 2001), assuming that the metal ion and ligand in the environment are at equilibrium (Stumm and Morgan 1996).
- The constants were corrected for an ionic strength of 0 using the Dubye–Huckel equation in order to produce a thermodynamic database usable by WHAM.
- In the absence of thermodynamic stability constants for humic acid (HA) and fulvic acid (FA) with vanadate ions built into the model, the constant for the humic substance (HS)–vanadate complex determined by Lu et al. (1998) was used. In other words, a new component "HS" was defined as being a monomeric anion in the solution, and the chemical reaction of HS with vanadate, together with its log K, were "user-entered" in the WHAM thermodynamic database.
- Wanty and Goldhaber (1992) determined the stability constant for the complex vanadium(V)–oxalate. However, this constant was not used for modelling because it is assumed that oxalate is a negligible entity in most natural waters.
- All chemical concentrations were converted into moles per litre before entering them in WHAM (including HS concentrations).
- To convert dissolved organic carbon (DOC) concentrations (milligrams of carbon per litre) to HS concentrations (moles per litre), it was assumed that 1) the ratio of dissolved organic matter (DOM) to DOC is 2:1 (Buffle 1988) and 2) 60% of DOM is composed of HS (i.e., HA and FA) (Perdue and Ritchie 2003). A molecular weight of 800 g/mol was used for HS (Tipping 2002).
- Dissolved vanadium concentrations were entered in the model spreadsheet as VO_4^{3-} concentrations, a format usable by WHAM. For the same reason, dissolved inorganic carbon or HCO_3^- concentrations were entered in the spreadsheet as CO_3^{2-} concentrations.

Table A1.1. Physical and chemical characteristics of surface waters used to model speciation of vanadium in solution[1]

(a)

Water type	n	HCO_3^-	CO_3^{2-}	P_{TOT}	NO_3^{2-}	Cl^-	F^-	SO_4^{2-}	pH	Ca
Prairie Wabamun Lake (Alberta)[2]	4	**Mean concentrations (mg/L, raw data from the report) and pH values**								
		250.75	5.125	0.01	0.05	7	0.4	56.875	8.24	22.85
		Mean concentrations (mol/L)								
		4.11×10^{-3}	8.54×10^{-5}	3.2×10^{-7}	8.07×10^{-7}	1.97×10^{-4}	2.11×10^{-5}	5.92×10^{-4}	–	5.70×10^{-4}
Canadian Shield Allard River, Station 12 (Quebec)[3]	3	**Mean concentrations (mol/L, raw data from the report) and pH values**								
		DIC		**P_{TOT}**	**NO_3^{2-}**	**Cl^-**	**F^-**	**SO_4^{2-}**	**pH**	**Ca**
		5.28×10^{-4}		–	5.27×10^{-6}	2.77×10^{-5}	–	5.99×10^{-5}	6.97	2.33×10^{-4}
Colombière River, Station 1 (Quebec)[3]		2.13×10^{-4}		–	1.08×10^{-7}	1.30×10^{-5}	–	2.41×10^{-5}	6.36	1.17×10^{-4}
Seawater St. Lawrence Gulf (eastern Canada)[4]	–	**HCO_3^- [4]**	**Br^- [4]**	**PO_4^{3-} [5]**	**NO_3^{2-} [6]**	**Cl^- [4]**	**F^- [4]**	**SO_4^{2-} [4]**	**pH [4]**	**Ca [4]**
		Mean concentration (mol/L) and pH values								
		2.35×10^{-3}	9.61×10^{-4}	1.16×10^{-6}	6.50×10^{-6}	5.98×10^{-1}	5.26×10^{-5}	3.08×10^{-2}	8.1	1.15×10^{-2}

(b)

Water type	Mg	Na	K	Al	Fe	Mn	DOC (mg/L as carbon)	TSS	Conduc-tivity (µS/cm)	V
Prairie Wabamun Lake (Alberta)[2]	**Mean concentrations (mg/L, raw data from the report)**									
	16.775	63.25	9.7	0.0475	0.0275	0.0278	12.5	2.33	502	0.00125
	Mean concentrations (mol/L)									
	6.90×10^{-4}	2.75×10^{-3}	2.48×10^{-4}	1.76×10^{-6}	4.92×10^{-7}	5.06×10^{-7}	–	–	–	2.45×10^{-8}
Canadian Shield Allard River, Station 12 (Quebec)[3]	**Mean concentrations (mol/L, raw data from the report)**									
	1.36×10^{-4}	1.04×10^{-4}	4.14×10^{-5}	7.23×10^{-6}	3.93×10^{-6}	2.68×10^{-7}	18.17	–	60	1.80×10^{-8}
Colombière River, Station 1	2.65×10^{-5}	3.78×10^{-5}	8.78×10^{-6}	4.91×10^{-6}	4.79×10^{-6}	3.98×10^{-7}	14.33	–	27	6.38×10^{-9}

(Quebec)[3]										
Seawater St. Lawrence Gulf (eastern Canada)[4]	**Mg** [3]	**Na** [3]	**K** [3]	**Al** [7]	**Fe** [7]	**Mn** [7]	**DOC** [5] **(mg/L as carbon)**	**TSS**	**Salinity** [7–9] **(‰)**	**V** [8]
	Mean concentrations (mol/L)									
	5.84×10^{-2}	5.13×10^{-1}	1.08×10^{-2}	9.18×10^{-8}	3.53×10^{-8}	9.19×10^{-9}	0.942	–	31.76	2.19×10^{-8}

Abbreviations: DIC, dissolved inorganic carbon; DOC, dissolved organic carbon; n, number of water samples; TSS, total suspended solids.

[1] All values are for the dissolved phase, except Wabanum Lake study reporting total (unfiltered) aqueous concentrations.

[2] Alberta Environment (2002). Only stations not directly under the influence (i.e., more than 100 m from ash pond effluent) of point source discharges considered: WS1, 134, 136 and 137. Unfiltered water samples. Sampling period: May 2002.

[3] Couillard et al. (2008). Abitibi-James Bay area in Quebec. Only stations without perturbations (i.e., mining effluents) considered. Filtered water samples (0.45 µm pore size). Sampling performed in June 2003.

[4] Poisson et al. (1979). Average seawater of 38‰ salinity.

[5] Packard et al. (2000). Mean of four water samples obtained in surface waters (Stations 4 and 5 at <30 m depth). Sampling performed in April 1994.

[6] Plourde and Therriault (2004). Modelled estimates for surface waters (<30 m depth).

[7] Yeats (1993). Mean of 62 samples (water column integrated) obtained during ice-free seasons 1979–1984. Filtered water samples (0.4 µm pore size).

[8] Yeats (1992). Surface water samples obtained during ice-free season 1979. Filtered water samples (0.4 µm pore size). Average salinity of 31.76‰.

[9] Salinity is not entered in WHAM.

Appendix 2: Criteria and considerations for determining the quality of BCF and BAF values, and other bioaccumulation ratios, for metals and elements

The following criteria and considerations were used to determine the reliability of BCF and BAF studies used in this screening assessment:

1. Evidence is provided to the effect that steady state (SS) is reached between chemical concentrations in the test organism and those of its surrounding medium (BCFs and BAFs are required to be obtained at SS). Calculation methods may be based on kinetic rate constants or on concentrations obtained at SS.
2. BAFs measured under field exposures defined in time (e.g., transplantation of organisms) are preferred over laboratory-derived BCFs because they provide information about the element's actual bioaccumulation behaviour in the environment and because they comprise a bioaccumulation measure that includes all routes of chemical uptake and elimination.
3. Reports of samplings in natural environments offer advantages similar to the above, but assumption of SS is to be judged on a case-by-case basis.
4. Metal concentrations in test organism and water are measured simultaneously.
5. Metal concentrations in water are: (1) low in order to minimize BCF/BAF decreases with increases in exposure concentrations; (2) well below levels causing chronic toxicity (e.g., OECD 1993, 1996); and (3) significantly above detection limits (tissue levels must also be significantly above detection limits).
6. Methodological details are provided (e.g., organism weights, replication, use of controls, method of chemical analysis, water quality).
7. Quality assurance and quality control (QA/QC) checks are reported, allowing one to judge whether or not good laboratory practices were followed.
8. To the extent possible, BCFs and BAFs are expressed on a wet weight basis. When published information permits, body concentrations are corrected for metal concentrations in gut contents, and bioaccumulation ratios are corrected for background metal concentrations in test organism and water.
9. Consideration is given to degree of essentiality of the metal entity. For example, BCFs and BAFs are expected to be of little usefulness for macroelements. Elements known by science to be macronutrients include H, C, N, O, P, S, Cl, Ca, Mg, Na, K, and Fe (Markert 1994). In this context, a micronutrient can be defined as any non-macronutrient element for which there is some evidence of nutritional essentiality.
10. Consideration is given to detoxification mechanisms. For example, BCFs and BAFs are less meaningful for organisms that store large quantities of metals in inert forms or for organisms that regulate metals to a constant tissue level regardless of exposure concentration.
11. Studies reporting metal concentrations in water and tissues measured before 1977–1978 are generally considered of low reliability because of numerous analytical difficulties, at that time, brought about notably by sources of inadvertent contamination, poor reproducibility and problems associated with filtration and separation of metals in water (e.g., Hume 1973; Beneš and Steinnes 1974; Batley and Gardner 1977; Stevenson 1985).

Considerations in the evaluation of BSAF-soil, BSAF-sediment, BMF and TTF

1. All the criteria above, except number 8, are directly relevant for the evaluation of BSAFs (soil and sediment), BMF and TTF. Criterion number 8 is replaced by the considerations (2-4) identified below.
2. To the extent possible, BSAF-soil, BSAF-sediment, BMF and TTF are expressed on a wet weight basis. Bioaccumulation ratios are corrected for background metal concentrations in test organism and in abiotic compartment.
3. To the extent possible, gut cleared tissue concentrations are needed for organism exposed to contaminated sediments.
4. To the extent possible, non gut cleared tissue concentrations are needed for trophic transfer calculations.

Studies selected in the present context may not meet all of the above criteria and may be attributed high to moderate confidence scores; those with low confidence scores are not retained. These critical evaluations are made with the help of robust study summaries developed for bioaccumulation data. These robust study summaries are available upon request.

Appendix 3a. Upper-bounding estimates of daily intake of vanadium pentoxide by the general population in Canada (derived from vanadium intake[1])

Route of exposure	Estimated intake (µg/kg-bw per day) of vanadium pentoxide by various age groups							
	0–6 months[2–4]			0.5–4 years[5]	5–11 years[6]	12–19 years[7]	20–59 years[8]	60+ years[9]
	Breast fed	Formula fed	Not formula fed					
Ambient air[10]	0.00			0.01	0.01	0.00	0.00	0.00
Indoor air[11]	0.03			0.06	0.04	0.02	0.02	0.02
Drinking water[12]	0.00	1.90	0.71	0.81	0.63	0.36	0.38	0.40
Food and beverages[13]	0.00	0.15	0.30–0.31	0.44–0.74	0.54–0.93	0.39–1.14	0.68–3.10	0.34–3.31
Soil[14]	2.46	2.46	2.46	3.96	1.29	0.31	0.26	0.26
Estimated total intake	2.49	4.54	3.50–3.51	5.28–5.58	2.51–2.90	1.09–1.84	1.34–3.76	1.01–3.97

1 The upper-bounding estimates of daily intake for vanadium from environmental media and food were converted to molar-equivalent vanadium pentoxide mass by applying the ratio of the molecular weights of vanadium pentoxide and vanadium (181.88/101.88).

2 No data on the levels of vanadium in breast milk were identified.

3 Assumed to weigh 7.5 kg, to breathe 2.1 m^3 of air per day, to drink 0.8 L of water per day (formula fed) or 0.3 L/day (not formula fed) and to ingest 30 mg of soil per day (Health Canada 1998).

4 For exclusively formula-fed infants, intake from water is synonymous with intake from food. The concentration of vanadium in water used to reconstitute formula (6.6 ng/g) was based on the level reported for a prebiotic formulation (6.2 ± 0.4 ng/g) (Lopez-Garcia et al. 2009). Approximately 50% of non-formula-fed infants are introduced to solid foods by 4 months of age, and 90% by 6 months of age (NHW 1990). This was the only data point identified for this medium.

5 Assumed to weigh 15.5 kg, to breathe 9.3 m^3 of air per day, to drink 0.7 L of water per day and to ingest 100 mg of soil per day (Health Canada 1998).

6 Assumed to weigh 31.0 kg, to breathe 14.5 m^3 of air per day, to drink 1.1 L of water per day and to ingest 65 mg of soil per day (Health Canada 1998).

7 Assumed to weigh 59.4 kg, to breathe 15.8 m^3 of air per day, to drink 1.2 L of water per day and to ingest 30 mg of soil per day (Health Canada 1998).

8 Assumed to weigh 70.9 kg, to breathe 16.2 m^3 of air per day, to drink 1.5 L of water per day and to ingest 30 mg of soil per day (Health Canada 1998).

9 Assumed to weigh 72.0 kg, to breathe 14.3 m^3 of air per day, to drink 1.6 L of water per day and to ingest 30 mg of soil per day (Health Canada 1998).

10 Maximum vanadium concentration of 59.5 ng/m^3 in ambient air $PM_{2.5}$ fraction was used to calculate the upper-bounding exposure estimates. The study consisted of eight samples across Canada, and the maximum value was detected in Montreal, Quebec (2009 personal communication from Environment Canada to Health Canada; unreferenced). Canadians are assumed to spend 3 h outdoors each day (Health Canada 1998).

11 No data on the levels of vanadium in indoor air were identified. The maximum concentration of vanadium measured in ambient air $PM_{2.5}$ fraction, 59.5 ng/m^3, was used to calculate upper-bounding exposure estimates (2009 personal communication from Environment Canada to Health Canada; unreferenced).

[12] The typical concentration of vanadium in drinking water in Saskatchewan, 1 µg/L, was used to calculate the upper-bounding exposure estimates (2007 personal communication from Saskatchewan Environment to Health Canada; unreferenced).

[13] Maximum concentrations of vanadium measured in food items are listed in Appendix 3b. Amounts of foods consumed on a daily basis by each age group are described in Health Canada (1998).

[14] The maximum concentration of vanadium measured in soil from Southern Ontario, 344 µg/g, was used to calculate the upper-bounding exposure estimates (2009 personal communication from Contaminated Sites Division, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada; unreferenced)

Appendix 3b: Maximum concentrations of vanadium measured in food items

Food/beverage	Concentration (µg/g)[1]	Reference
Tomatoes	1.1×10^{-2}	Triolo et al. 2008
Lettuce	2×10^{-2}	
Potato	2×10^{-2}	
Squash	3×10^{-3}	
Lemon	3×10^{-3}	
Olive	3.2×10^{-2}	
Lingonberries	3.6×10^{-2}	Pöykiö et al. 2005
Mushrooms	1.66	Svoboda and Chrastný 2008
Parsley	3.56	Ambrozini et al. 2009
Pistachios	20.8	Anderson and Smith 2005
Whole milk	4.0×10^{-4}	Lopez-Garcia et al. 2009
Milk 2%	3.6×10^{-4}	
Skim milk	2.9×10^{-4}	
Infant formula	6.6×10^{-3}	
Beer	5.5×10^{-2}	Wyrzykowska et al. 2001
Wine	0.364	del Mar Castiñeira Gomez et al. 2004
Rice	9.69×10^{-3}	Nardi et al. 2009
Eggs	3.55×10^{-2}	
Bread	2.7×10^{-3}	
Cheese	2.13×10^{-2}	
Tea	0.000 11[2]–0.30[3]	Ødegárd and Lund 1997 Moreda-Pineiro et al. 2003

[1] While these data are presented as a representation of potential content of vanadium pentoxide in Canadian food, previous research in the United States and the UK has determined lower values for many of these food commodities (Evans et al. 1985; Pennington and Jones 1987). The combined information should be used solely to demonstrate the high variability in the vanadium pentoxide content in food items, which most likely reflects regional soil content and differences in uptake rates in plants.

[2] Lipton tea with 0.6% extraction efficiency (Ødegárd and Lund 1997).

[3] Chinese tea leaves with 40.5% extraction efficiency (Lozak et al. 2002).

Appendix 4. Summary of health effects information for vanadium pentoxide

Endpoints	Lowest effect levels[1]/Results
Acute toxicity (vanadium pentoxide)	**Lowest oral LD_{50}** (rat) = 10 mg/kg-bw (MAK Commission 1992). **Other oral LD_{50}s** (rat, mice, rabbit) = 64–137 mg/kg-bw (Yao et al. 1986b). **Lowest inhalation LC_{50}** (rat, 6 h) = 126 mg/m^3 (NTIS undated). **Other inhalation LC_{50}** (rabbit, 2 h) = 205 mg/m^3 (MAK Commission 1992). **Inhalation LC_{67}** (rat, 1 h) = 1440 mg/m^3 (US EPA 1992). **LOAEC** = 3 mg/m³ based on statistically significant reduction in lung function characterized by airway obstructive changes (increased resistance and decreased flow) and significant influx of inflammatory cells (neutrophils, eosinophils) into the lung after the pre-exposure challenges in a group of 24 male monkeys exposed to 0.5 and 3 mg/m^3 for 6 h separated by 2-week intervals, followed after another 2 weeks by challenge with methacholine to assess for non-specific bronchial reactivity (Knecht et al. 1992). **Lowest dermal LD_{50}** (rabbit) = 50 mg/kg-bw (NTIS undated). **Other dermal LD_{50}** (rat) = >2500 mg/kg-bw (Leuschner et al. 1994).
Short-term repeated-dose toxicity (vanadium pentoxide)	**Lowest inhalation LOAEC =** 1 mg/m^3 based on hyperplasia of the alveolar epithelium, interstitial inflammation, increases in alveolar macrophage numbers and alveolar cell proliferation in the respiratory tract of female F344/N rats (groups of 40 or 60) exposed to 0, 1, 2 or 4 mg/m^3, 6 h/day, 5 days/week, for up to 16 days and based on dose related increase in lung weight in female B6C3F1 mice (groups of 48) exposed to 0, 0.25, 1 or 4 mg/m^3, 6 h/day for 16 days. This increase was accompagnied with lung effects such as a dose-dependant increased in cell proliferation, alveolar histiocytosis, subacute alveolitis and/or granulocytic infiltration (NTP 2002; Schuler 2010).. **Other inhalation LOAECs** = 2–5 mg/m^3 in rats, mice and monkeys (Knecht et al. 1985; NTP 2002; Avila-Costa et al. 2004a, b, 2005a, b, 2006; Colin-Barenque et al. 2004, 2008; Gonzalez-Villalva et al. 2004, 2006; Nino-Cabrera et al. 2004; Mussali-Galante et al. 2005; Fortoul et al. 2007, 2008; Pinon-Zarate et al. 2008). **Lowest oral LOAEL =** 3.7 mg/kg-bw per day based on histological and enzymatic alterations, including inhibition of biosynthesis, enhanced catabolism and increased use of L-ascorbic acid in the liver and kidney tissues of male albino rats dosed (five males per dose) at 3 mg/kg-bw per day 5 times a week for the first week and at 4 mg/kg-bw per day 5 times a week for the following 2 weeks (Chakraborty et al. 1977). **Other oral LOAEL** = 6 mg/kg-bw per day based on spleen enlargement, diminished spleen cellularity, increased leukocyte count in the peripheral blood, decreased phagocytosis, signs of intense response to mitogens, high stimulation to B-cells and potential vanadium-related hypersensitivity in male and female ICR mice dosed by gavage (10 per sex per dose) at 0 or 6 mg/kg-bw per day, 5 days/week for 6 weeks (Mravcova et al. 1993). No dermal studies were identified.

Endpoints	Lowest effect levels[1]/Results
Subchronic toxicity (vanadium pentoxide)	**Lowest inhalation LOAEC =** 2 mg/m^3 based on adverse effects on the blood and epithelial hyperplasia of the lungs in males and females and an increase in lung weight and inflammation in males only in a 3-month rat study and inflammation and epithelial hyperplasia of the lungs in males and females in a 3-month mouse study (NTP 2002: male and female B6C3F1 mice and F344/N rats exposed to 0, 1, 2, 4, 8 or 16 mg/m^3, 6 h/day, 5 days/week, for 3 months). No other inhalation studies were identified. **Lowest oral LOAEL** = 0.25 mg/kg-bw per day based on a dose-related decrease in phagocytosis in peritoneal cells of male Wistar rats (10 per group) exposed via drinking water to 0, 1 or 100 mg/L as vanadium (equivalent to 0, 0.14 or 14 mg/kg-bw per day as vanadium or 0, 0.25 or 25 mg/kg-bw per day as vanadium pentoxide using a dose conversion from Health Canada 1994) for 6 months (Mravcova et al. 1993). No other oral studies were identified. No dermal studies were identified.
Subchronic toxicity (other vanadium compounds)	**Sodium metavanadate** **Oral NOAEL** = 2.4 mg/kg-bw per day based on no effect on cardiovascular function and no changes in the brain, liver, lungs, heart or blood vessels in male Sprague-Dawley rats (six per group) exposed via drinking water to sodium metavanadate at 0, 0.06, 0.6 or 2.4 mg/kg-bw per day for 180 or 210 days. Also, no effect on urinary excretion of creatinine, total nitrogen, protein or sodium was noted (Boscolo et al. 1994). **Other oral study: LOAEL** = 0.3 mg/kg-bw per day based on dose-dependent histological changes in the spleen, kidneys and lungs of male Sprague-Dawley rats (10 per group) exposed via drinking water to sodium metavanadate at 0, 0.3, 0.6 or 3 mg/kg-bw per day for 3 months. A significant increase in the concentrations of urea and uric acid in plasma was also observed at the highest dose (Domingo et al. 1985).
Chronic toxicity/ carcinogenicity (vanadium pentoxide)	**Inhalation study in mice:** Groups of 50 B6C3F1 mice per sex were exposed to vanadium pentoxide by inhalation (whole body) at 0, 1, 2 or 4 mg/m^3 (equivalent to 0, 1.33, 2.66 or 5.32 mg/kg-bw per day, using a dose conversion by Health Canada 1994), 6 h/day, 5 days/week, for 2 years (104 weeks). There was an increased incidence of lung tumours in both sexes at all doses (males: lung adenoma 13/50, 16/50, 26/50 and 15/50 at 0, 1, 2 or 4 mg/m^3, respectively; lung carcinoma 12/50, 29/50, 30/50 and 35/50, respectively; lung adenoma/carcinoma combined 22/50, 42/50, 43/50 and 43/50, respectively; females: lung adenoma 1/50, 17/50, 23/50 and 19/50, respectively; lung carcinoma 0/50, 23/50, 18/50 and 22/50, respectively; lung adenoma/carcinoma combined 1/50, 32/50, 35/50 and 32/50, respectively). In both sexes, the incidences of carcinoma and combined tumours were significantly increased in all treated groups. **Non-neoplastic LOAEC** = 1 mg/m^3 based on significant increased incidence of inflammation, fibrosis, hyperplasia and squamous metaplasia in the

Endpoints	Lowest effect levels[1]/Results
	respiratory tract (lungs, larynx and nose) of males and females. Some of these local irritant effects were seen at all dose levels, and the severity increased with exposure concentration. Other non-neoplastic effects included a significant increase in the incidence of hyperplasia of the bronchial lymph node in females at all dose levels, with a non-significant positive trend in males, a decrease in mean body weights in females at all dose levels and in males at 2 mg/m^3 and above, abnormal breathing at 2 mg/m^3 and above and a significant decrease in survival in males in the highest exposure group (NTP 2002; Ress et al. 2003). **Inhalation study in rats:** Groups of 50 F-344 rats of each sex were exposed to vanadium pentoxide by inhalation (whole body) at 0, 0.5, 1 or 2 mg/m^3 (equivalent to 0, 0.16, 0.31 or 0.62 mg/kg-bw per day, using a dose conversion by Health Canada 1994), 6 h/day, 5 days/week, for 2 years (104 weeks). There was an increased incidence of lung tumours in males at all doses (lung adenoma 4/50, 8/49, 5/48 and 6/50 at 0, 0.5, 1 or 2 mg/m^3, respectively; lung carcinoma 0/50, 3/49, 1/48 and 3/50, respectively; lung adenoma/carcinoma combined 4/50, 10/49, 6/48 and 9/50, respectively). Although not statistically significant, the incidence of carcinomas and combined adenomas/carcinomas at 0.5 and 2 mg/m^3 exceeded the NTP historical ranges in controls (0–4% and 0–14%, respectively). The marginally increased incidence of lung tumours in females (lung adenoma 0/49, 3/49, 1/50 and 0/50 at 0, 0.5, 1 or 2 mg/m^3, respectively; lung carcinoma 0/50, 0/49, 0/50 and 1/50, respectively; lung adenoma/carcinoma combined 0/49, 3/49, 1/50 and 1/50, respectively) was not statistically significant and did not occur in a concentration-related fashion, but the incidences of adenomas and combined adenomas/carcinomas at 0.5 mg/m^3 was at the high end of the range for historical controls (0–6%). **Non-neoplastic LOAEC** = 0.5 mg/m^3 based on significantly increased incidence of inflammation, fibrosis and hyperplasia in the respiratory tract (lungs, larynx and nose) of males and females. Some of these local irritant effects were seen at all dose levels, and the severity increased with exposure concentration. Other non-neoplastic effects included a significant increase in the incidence of nephropathy in males exposed to 1 mg/m^3 and above and marginally decreased mean body weights in females at 2 mg/m^3 (NTP 2002; Ress et al. 2003). **Other inhalation study:** Groups of 62–84 male and female Kunmig albino mice were exposed to vanadium pentoxide by inhalation at 0, 0.5, 2 or 8 mg/m^3 (equivalent to 0, 0.67, 2.66 or 10.64 mg/kg-bw per day, using a dose conversion by Health Canada 1994), 4 h/day for 1 year. No tumours were seen at 0 or 0.5 mg/m^3. Papillomatous and adenomatous tumours were reported in the lungs of 2/79 and 3/62 mice exposed at 2 and 8 mg/m^3, respectively (Yao et al. 1986a). No oral or dermal studies were identified.

Endpoints	Lowest effect levels[1]/Results
Reproductive toxicity (vanadium pentoxide)	**Lowest LOAEL for reproductive toxicity:** 2.8 mg/kg-bw per day based on decreased pregnancy rate by 52% in untreated females mated with male CD-1 mice (15–20 per group) exposed to 0 or 8.5 mg/kg-bw intraperitoneally (males treated every third day for 60 days prior to mating) compared with untreated females mated with control males. A reduction in sperm counts was noted in males treated for 20 days or longer. A marked reduction in sperm motility was observed with the advancement of treatment, and a significant increase in the percentage of morphological abnormalities in spermatozoa after 50–60 days of treatment was also observed (Altamirano-Lozano et al. 1996). No other oral studies were identified. No inhalation or dermal studies were identified.
Developmental toxicity (vanadium pentoxide)	**Lowest oral LOAEL** = 9 mg/kg-bw per day based on skeletal abnormalities in fetuses of pregnant Wistar rats (18–21 per group) dosed by gavage during days 6–15 of gestation with 0, 1, 3, 9 or 18 mg/kg-bw per day (none of the results were reported on a per litter basis, so interpreting the results is difficult). LOAEL for maternal toxicity = 9 mg/kg-bw per day based on decreased maternal body weight gain (Yang et al. 1986a). **Other oral study:** Altamirano-Lozano et al. 1996. No inhalation or dermal studies were identified.
Genotoxicity and related endpoints: *in vivo* (vanadium pentoxide)	**DNA damage (comet assay)** **Negative:** Bone marrow; male CD-1 mice; intraperitoneal (0, 5.75, 11.5 and 23 mg/kg-bw, single injection) (Altamirano-Lozano et al. 1993, 1996, 1999). **Negative:** Bronchio-alveolar lavage (BAL) and pulmonary cells; female B6C3F1 mice; nose only inhalation (0, 0.25, 1 and 4 mg/m^3, 6 hours per day for 16 days) (Schuler 2010). **Positive:** Testes, liver, kidney, lung, spleen, heart; male CD-1 mice; intraperitoneal (0, 5.75, 11.5 and 23 mg/kg-bw, single injection) (Altamirano-Lozano et al. 1993, 1996, 1999). **Micronuclei** **Negative:** Peripheral blood cells; male and female B6C3F1 mice; inhalation (0, 1, 2, 4, 8 or 16 mg/m^3, 6 h/day, 5 days/week, for 3 months) (NTP 2002). **Negative:** Bone marrow: 615 and Kunming albino mice; oral (0, 1, 3, 6 or 11 mg/kg-bw per day, 6 weeks) (Si et al. 1982; Yang et al. 1986b, c; Sun et al. undated). **Positive:** Bone marrow: 615 and Kunming albino mice; inhalation (0, 0.5, 2 or 8 mg/m^3, duration not specified), intraperitoneal (0, 0.2, 2 or 6 mg/kg-bw per day, 5 days), subcutaneous (0, 0.25, 1 or 4 mg/kg-bw per day, 6 days/week for 5 weeks) (Si et al. 1982; Yang et al. 1986b, c; Sun et al. undated). **Dominant lethal assay** **Positive:** Male CD-1 mice; intraperitoneal (0 or 8.5 mg/kg-bw every third day for 60 days; 0 or 2.8 mg/kg-bw per day) (Altamirano-Lozano et al. 1996).

Endpoints	Lowest effect levels[1]/Results
	Sister chromatid exchanges (SCE) **Negative:** Bone marrow cells; male CD-1 mice; intraperitoneal (0, 5.75, 11.5 and 23 mg/kg-bw, single injection) (Altamirano-Lozano et al. 1993, 1996, 1999). **K-*ras* mutation and allelic losses on chromosome 6** **Positive:** Lung neoplasms; male and female B6C3F1 mice; inhalation (0, 1, 2 or 4 mg/m^3, 6 h/day, 5 days/week, for 2 years) (NTP 2002).
Genotoxicity and related endpoints: *in vitro* (vanadium pentoxide)	**Mutagenicity in bacteria** **Negative:** *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538, with and without activation (Si et al. 1982). **Negative:** *S. typhimurium* strains TA97, TA98, TA100, TA102 and TA1535, with and without rat or hamster S9 activation (NTP 2002). **Negative:** *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538, without metabolic activation (Kada et al. 1980). **Negative:** *Escherichia coli* strains WP2 and WP2hcr, without metabolic activation (Kada et al. 1980). **Negative:** *E. coli* strains ND-160 and MR 102, with and without activation (Si et al. 1982). **Positive:** *E. coli* strains WP2, WP2uvrA and CM891, with and without activation (Si et al. 1982). **Positive:** *Bacillus subtilis* strains H17 and M45, without metabolic activation (Kada et al. 1980). **Micronucleus induction** **Positive:** Chinese hamster V79 cells in absence of metabolic activation (Zhong et al. 1994). **Aneuploidy induction** **Positive:** Dose-related increases of hyperdiploid human lymphocytes in absence of metabolic activation (Ramirez et al. 1997). **Microtubule polymerization and depolymerization assays** **Positive:** Inhibition of tubulin polymerization and induction of tubulin depolymerization in human lymphocytes in absence of metabolic activation (Ramirez et al. 1997). **DNA damage (comet assay)** **Negative:** Human mucosal epithelial cells (from inferior nasal turbinates) (Kleinsasser et al. 2003). **Positive:** Human lymphocytes (from peripheral blood) (Kleinsasser et al. 2003). **Positive:** Human lymphocytes (Rojas et al. 1996). **Chromosome aberration assay** **Negative:** Human lymphocytes in absence of metabolic activation (Roldan and Altamirano 1990). **Gene mutation assay** **Negative:** Chinese hamster V79 cells in absence of metabolic activation

Endpoints	Lowest effect levels[1]/Results
	(Zhong et al. 1994). **SCE assay** **Negative:** Chinese hamster V79 cells (Zhong et al. 1994). **Negative:** Human lymphocytes (Sun et al. undated). **Negative:** Human lymphocytes (Roldan and Altamirano 1990).
Human studies (vanadium pentoxide)	
	The most robust human studies are described below. Several other inhalation epidemiological studies are described in IPCS (2001), but concomitant exposures to other chemicals may have occurred in those studies.
Clinical human study	Nine healthy volunteers were exposed to vanadium pentoxide in an exposure chamber. Volunteers were separated into three groups: two volunteers were exposed to the vanadium pentoxide dust at 0.1 mg/m^3 (particle size <5 μm) for 8 h, five volunteers were exposed to 0.25 mg/m^3 for 8 h and two volunteers were exposed to 1 mg/m^3 for 8 h. Volunteers exposed to 0.1 mg/m^3 produced considerable amounts of mucus within 24 h after exposure. This was easily cleared by slight coughing, increased after 48 h, subsided within 72 h and completely disappeared after 4 days. Dose-dependent coughing was induced at 0.25 mg/m^3 and above. Accidental exposure to a "heavy cloud" of vanadium pentoxide dust (concentration unknown) for 5 min resulted in the production of sputum, rales, expiratory wheezing and coughing (Zenz and Berg 1967).
Observational studies	A study investigated 24 male workers occupationally exposed to vanadium pentoxide at 0.2–0.9 mg/m^3 for at least 6 months (time period of measurement not stated) from two different centres. These were age-matched with 45 control subjects from the same areas. In the exposed group, eye, nose and throat irritation was reported in 62.5% (compared with 6.6% of controls), coughing in 83.4% (33.3% of controls), sputum production in 41.5% (13.3% of controls) and wheezing in 16.6% (0% of controls). Physical findings included wheezes, rales or rhonchi in 20.8% (0% of controls), hyperemia of the pharynx and nasal mucosa in 41.5% (4.4% in controls) and "green tongue" in 37.5% (0% of controls). It is not clear what levels or duration of exposure caused the effects. However, the findings reinforce the picture of exposure to vanadium pentoxide causing respiratory tract effects (Lewis 1959). Workers (18 males) were examined after being exposed to varying degrees to vanadium pentoxide dust in excess of 0.5 mg/m^3 (measured over a 24-h period) during a pelletizing process. Three of the most heavily exposed men developed respiratory irritation (dry and persistent cough, sore throat, inflamed throat) and eye irritation (slight conjunctivitis). Upon resumption of work after a 3-day exposure-free period, the symptoms returned within 0.5–4 h, with greater intensity than before, despite the use of respiratory protective equipment. After 2 weeks, all workers, even those primarily assigned to office and laboratory duties, developed symptoms, including nasopharyngitis, hacking cough and wheezing. Based on these data, it has been concluded that vanadium pentoxide exposure can produce respiratory and eye irritation (Zenz et al. 1962).

Endpoints	Lowest effect levels[1]/Results
	Forty male workers from a South African plant exposed to vanadium pentoxide for variable periods were examined when presenting with persistent symptoms (cough, breathlessness) over a 24-month study period. Exposure was assessed by measurement of ambient vanadium pentoxide over 7 days during the 24-month study period. Exposure to vanadium pentoxide was noted to vary from less than 0.05 mg/m^3 to 1.53 mg/m^3. Workers were investigated by 1) blood count and serum immunoglobulin E, 2) intracutaneous allergen skin tests, 3) spirometry and 4) bronchoprovocation by histamine inhalation or exercise challenge. Twelve of the 40 workers had bronchial hyperreactivity (BHR), while BHR was normal in the 12 age-matched companion subjects. In 10 workers, BHR was diagnosed by histamine inhalation, and in six of the workers, the abnormality was severe. A further two workers had BHR by exercise challenge. After removal from exposure, 9 of the 12 subjects returned for follow-up 5–23 months later. BHR was worse in one subject, still present although less severe in five and no longer found in one. Baseline spirometry measurements were normal in 7 subjects and only mildly impaired in the remaining 5 subjects with BHR. Based on these data, the authors of the study concluded that inhaled vanadium pentoxide induces BHR and asthma in subjects previously free of lung disease and that this abnormality may persist for up to 23 months following exposure (Irsigler et al. 1999). Three cases of vanadium pentoxide–induced skin allergy have been reported and confirmed by patch tests (Proctor et al. 1988). Barth et al. (2002) conducted a neurobehavioural study in 49 male workers occupationally exposed to vanadium pentoxide for a mean duration of 12.2 years (range 0.5–31 years). Forty-nine controls employed at a steel production plant with no exposure to vanadium pentoxide or other known neurotoxins were included in the study. No exposure data were given, but the exposed group had a mean urinary vanadium concentration of 14.4 µg/L (range 2.1–95.3 µg/L) and a mean serum vanadium concentration of 7.5 µg/L (range 2.2–46.4 µg/L). In controls, mean values were <1 µg/L for both parameters. In six neurobehavioural tests, the exposed group had lower scores in two (for visuospatial ability and attention). These defects were correlated with urine and serum levels of vanadium pentoxide.
Genotoxicity *in vivo*	No incidence of DNA damage (comet assay) and DNA adducts in leukocytes and no increase in the frequency of SCEs in lymphocytes were observed in 49 male workers occupationally exposed by inhalation and/or the dermal route to vanadium pentoxide (12 unexposed controls from same factory) for 12.4 years (range 0.5–31 years); exposure levels were not given, but the median serum concentration of elemental vanadium in the exposed group was 5.4 µg/L (range 2.2–46.4 µg/L) compared with 2.54 µg/L (range 1.01–12.5 µg/L) in controls (Ivancsits et al. 2002). An increased incidence of DNA damage (comet assay) in leukocytes and a statistically significant increase in the incidence of different parameters of chromosomal instability, such as micronuclei, nucleoplasmic bridges and nuclear buds in lymphocytes, were observed in 52 male workers occupationally exposed by inhalation to vanadium pentoxide. Fifty-two unexposed jail wardens were used as controls. Exposure levels were not

Endpoints	Lowest effect levels[1]/Results
	given, but the median serum concentration of elemental vanadium in the exposed group was 2.2 µg/L (range 1.54–3.89 µg/L) compared with 0.3 µg/L (range 0.24–0.39 µg/L) in controls. The mean exposure duration was unknown (Ehrlich et al. 2008).

[1] LD_{50} = median lethal dose; LOAEC/LOAEL = lowest-observed-adverse-effect concentration/level; LOEC/LOEL = lowest-observed-effect concentration/level; NOAEC/NOAEL = no-observed-adverse-effect concentration/level.