Screening Assessment for the Challenge

Antimony trioxide
(Antimony oxide)

Chemical Abstracts Service Registry Number
1309-64-4

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 antimony trioxide, Chemical Abstracts Service Registry Number 1309-64-4. The substance antimony trioxide was identified in the categorization of the Domestic Substances List (DSL) as a high priority for action under the Challenge. Antimony trioxide was identified as a high priority as it was considered to pose greatest potential for exposure of individuals in Canada and is classified by other agencies on the basis of carcinogenicity. In addition, this substance met the ecological categorization criteria for persistence and inherent toxicity to non-human organisms. The focus of this assessment of antimony trioxide relates to both human health and ecological risks.

According to information reported under section 71 of CEPA 1999, between 1 000 000 and 10 000 000 kg of antimony trioxide was manufactured in Canada in 2006. In addition, Canadian companies reported importing over 1 850 000 kg and using approximately 3 270 000 kg in that year. Between 1 000 to 10 000 kg of antimony trioxide was released into the environment in 2006, with the majority disposed of in landfill sites. In Canada, antimony trioxide is used primarily as a plastic catalyst in manufacturing polyethylene terephthalate and as a synergist with halogenated compounds to provide flame retardancy properties. Flame retardants are used in a variety of household items including furniture upholstery, carpets, mattress covers and other textiles.

Based on available information on concentrations of antimony in environmental media (soil, drinking water, ambient air) and food, as well as results for antimony trioxide from a survey conducted under section 71 of CEPA 1999, the general population is expected to be exposed to antimony trioxide primarily from household items containing flame retardants. However, the total exposure level to antimony trioxide resulting from household products and environmental media identified in this screening assessment is expected to be low.

As antimony trioxide was classified on the basis of carcinogenicity by international regulatory agencies, carcinogenicity was a key focus for this screening assessment. Lung tumours were observed in female but not male rats exposed to the highest concentrations of antimony trioxide tested in 1-year inhalation bioassays. No evidence was available to suggest carcinogenic potential for antimony trioxide via the oral route. Collective evidence from genotoxicity studies suggests that antimony trioxide is not likely to be mutagenic but may exert some clastogenic effects *in vitro*. The mode of action for induction of tumours proposed by other regulatory agencies links to local inflammatory response and pulmonary overload. Although the mode of induction of tumours is not fully elucidated, the tumours observed are not considered to have resulted from direct

interaction with genetic material. Therefore, a threshold approach is used to characterize risk to human health.

There was some evidence for adverse effects on fertility in limited developmental and reproductive toxicity studies in experimental animals, as well as in epidemiological studies. The critical effect concentration for non-cancer effects was based on an increase in lung weight, pulmonary changes and no significant increase in the incidence of lung tumours in female rats exposed by inhalation to antimony trioxide for a year. For the oral route, the critical effect level was based on histopathological changes in the liver and an increase of aspartate transaminase (serum glutamic oxaloacetic transaminase) activity in male rats administered antimony trioxide for 24 weeks. The critical effect levels were also below the levels at which reproductive and developmental toxicity may occur. The margins between upper-bounding estimates of exposure to antimony trioxide from environmental media (based on antimony) and from use of household items and levels associated with effects in experimental animals are considered to be adequately protective to account for uncertainties in the health effects and exposure databases.

On the basis of the adequacy of the margins between conservative estimates of exposure to antimony trioxide and critical effect levels in experimental animals, it is concluded that antimony trioxide is not 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.

Antimony trioxide is released into the Canadian environment mainly as a result of its use as a synergistic flame retardant and as atmospheric emissions from fossil fuel combustion and non-ferrous metal production. Antimony trioxide emitted to air is then deposited onto surrounding terrestrial and aquatic ecosystems. Because antimony trioxide has some solubility in water, it will dissolve in contact with moisture once in these ecosystems and yield a variety of dissolved antimony species, depending on the environmental conditions. Antimony has been demonstrated to have a moderate potential to cause harm to aquatic, soil and sediment organisms.

Site-specific industrial scenarios based on monitoring data were developed for the most important sources of releases of antimony trioxide to the environment. Modelled exposure concentrations in the aquatic environment were also estimated, mainly for the plastics industry from the use of antimony trioxide as a flame retardant. Based on a risk quotient analysis, harm to aquatic and terrestrial organisms resulting from exposure to antimony trioxide 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. Antimony trioxide 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 antimony trioxide does not meet the criteria set out in section 64 of CEPA 1999.

This substance will be considered for inclusion in the DSL inventory update initiative. In addition and where relevant, research and monitoring will support verification of assumptions used during the screening assessment.

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), that 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 antimony oxide, referred to in this assessment as antimony trioxide, 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. 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 (Environment Canada 2009).

Although antimony trioxide was categorized as 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 antimony trioxide considers both human health and ecological risks.

Screening assessments focus on information critical to determining whether a substance meets the criteria 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 October 2009 for the exposure section of the document and up to September 2009 for the human health effects section. 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 (non-occupational) exposure 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 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 conclusion is based.

This 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 scientific experts selected and directed by Toxicology Excellence for Risk Assessment (TERA), including Joan Strawson (TERA), Dr. Michael Jayjock (The Lifeline Group) and Dr. Susan Griffin (US Environmental Protection Agency), as well as an additional professional (non-TERA) reviewer, Herman Gibb (Tetra Tech Sciences). 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 assessments under the Challenge have been reviewed by an independent Challenge Advisory Panel.

[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. Similarly, a conclusion under CEPA 1999 does not preclude actions being undertaken under the Environmental Emergency Regulations based on consideration of the hazard and related properties of the substance.

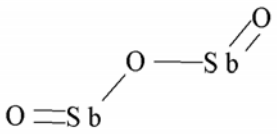
The critical information and considerations upon which the final assessment is based are summarized below.

Substance Identity

Antimony oxide is also commonly known as antimony trioxide. For the purposes of clarity, the substance will be referred to as antimony trioxide in this screening assessment report, even though its name on the DSL is antimony oxide. Information on the identity of antimony trioxide is summarized in Table 1.

Table 1. Substance identity for antimony trioxide

Chemical Abstracts Service Registry Number (CAS RN)	**1309-64-4**
DSL name	**Antimony oxide**
National Chemical Inventories (NCI) names[1]	*Antimony oxide (AICS, ASIA-PAC, ENCS, PICCS, SWISS, TSCA)* *Antimony trioxide (PICCS)* *Diantimony trioxide (ECL, EINECS)*
Other names	*100A; A 120; A 120 (corrosion inhibitor); A 1582; ACC-BS; AN 800; Antimonious oxide; Antimony Bloom 100A; Antimony Bloom 500A; Antimony oxide (O_3Sb_2); Antimony oxide (Sb_20_3); Antimony oxide ($SbO_{1.5}$); Antimony sesquioxide; Antimony trioxide (Sb_2O_3); Antimony white; Antimony(3+) oxide; Antimony(III) oxide; Antox; AO 3; AO 5; AP 50; AP 50 (metal oxide); AT 3; AT 3B; Atox 3CN; Atox B; Atox F; Atox R; Atox S; Bluestar RG; Bluestar Z; C.I. 77052; C.I. Pigment White 11; Chemetron Fire Shield; Dechlorane A-O; Exitelite; F 45; F 45 (oxide); FCP 100; Fire Cut AT 3; Fireshield FSPO 405; FireShield H; FireShield LS-FR; Flame Cut 610; Flame Cut 610R; Flameguard VF 59; Flowers of antimony; FSPO 405; HM 203P; LSB 80; LS-FR; MIC 3; Microfine AO 3; Microfine AO 5; MSA; MSA (flame retardant);MSF; Nyacol A 1510LP; Nyacol A 1530; Octoguard FR 10; P 3; Patox A; Patox C; Patox CF; Patox H; Patox HS; Patox L; Patox M; Patox MK; Patox P; Patox-P; Patox S; Patox U; Performax 401; Poliflam HT 3; Polysafe 60; Polysafe 100T; Pyroguard AN 700; Pyroguard AN 800; Pyroguard AN 800T; Pyroguard AN 900; RAC 1; Sanka Anchimonzol C; Senarmontite[3]; SHLB 80; Stibiox MS; Stibital; Stox W 60; Thermoguard B; Thermoguard L; Thermoguard S; Timonox; Timonox Red Star; Timonox RT; Timonox White Star; TMS; TMS (flame retardant); Trutin 40; TT 88; UF; UF (oxide); Ultrafine II; UN 1549; Valentinite[1]; White Star; White Star N*
Chemical group (DSL stream)	Discrete inorganics
Major chemical class or use	Antimony-containing inorganic compounds

Major chemical subclass	Oxides
Chemical formula	Sb_2O_3
Chemical structure[2]	
SMILES[3]	O=[Sb]O[Sb]=O
Molecular mass	291.5 g/mol

Abbreviations: 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; PICCS, Philippine Inventory of Chemicals and Chemical Substances; SMILES, simplified molecular input line entry specification; SWISS, Swiss Giftliste 1 and Inventory of Notified New Substances; TSCA, *Toxic Substances Control Act* Chemical Substance Inventory. Source: NCI 2007

[1] Reimann and de Caritat 1998.

[2] EURAR 2008.

[3] Simplified Molecular Input Line Entry System

Physical and Chemical Properties

Table 2 contains experimental physical and chemical properties of antimony trioxide that are relevant to its environmental fate. Key studies from which experimental data were reported for some of these properties were critically reviewed for validity.

Table 2. Physical and chemical properties of antimony trioxide

Property	Type	Value	Temperature (°C)	Reference
Physical state	Experimental	Solid (white, odourless and crystalline powder)	~20	EURAR 2008
Particle size (µm)	Experimental	0.12-13.89	-	Weidenfeller 2005
		0.92-5.96 ($D50^2$)	-	Franke 2005
Melting point (ºC)	Experimental	655	-	O'Neil 2006
Boiling point (ºC)	Experimental	1425	-	O'Neil 2006
Density (kg/m^3)	Experimental	5900 ($5.9\ g/cm^3$)	24	Smeykal 2005
		5760 ($5.76\ g/cm^3$)	Not specified	Mindat 2008
Vapour pressure (Pa)	Experimental	Likely negligible << 133 (<< 1 mm Hg)	574	Budavari 1996
	Extrapolated to ~20 (°C) from experimental	Likely negligible	~20	-
Henry's Law constant ($Pa\ m^3/mol$)	Calculated	Likely negligible	-	-
Log K_{ow} (dimensionless)	-	Not applicable	-	-
Water solubility (mg/L) as antimony	Experimental (in distilled water, after 24 h, loading = 100 g/L)	16.5 (pH 5) 22.1 (pH 7) 24.8 (pH 9)	20	Umweltanalytik Gmbh 1993
	Experimental (in reconstituted standard water, after 7 days, loading = 100 mg/L)	2.76 (pH 8)	22.2	LISEC 2002
Log K_{oc} (dimensionless)	-	Not applicable	-	-

Property	Type	Value	Temperature (°C)	Reference
Log K_{sw} (dimensionless)[3]	Experimental	0.1-2.7 (2.4)[3]	-	US EPA 1999
Log K_{sdw} (dimensionless)[3]	Experimental	2.5-4.8 (4.0)[3]	-	US EPA 1999
Log K_{ssw} (dimensionless)[3]	Experimental	4.98[3]	-	Allison and Allison 2005

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.
[1] Values in parentheses represent the original values as reported by the authors.
[2] The average particle size, i.e. the average equivalent diameter, is defined as the diameter where 50 mass-% (of the particles) of the powder have a larger equivalent diameter, and the other 50 mass-% have a smaller equivalent diameter (source: http://www.lactose.com/particle_size/d10_d50_d90.html).
[3] These partition coefficients are reported for dissolved antimony, not antimony trioxide. Range; median in parentheses

As indicated in Table 2, the highest water solubilities were reported in tests using distilled water by Umweltanalytik Gmbh (1993). According to EURAR (2008), the difference between water solubilities in distilled water and in reconstituted standard water (ISO 6341) might be explained by the higher calcium concentration (2 mM) in the reconstituted standard water and precipitation of calcium antimonite ($Ca[Sb(OH)_6]_2$). Upon dissolution in oxic systems, Sb(III) is readily oxidized to Sb(V), which easily hydrolyses to form the anion $Sb(OH)_6^-$. Johnson et al. (2005) presented a solubility product constant ($K_{sp} = [Ca^{2+}][Sb(OH)_6]_2$) of 10-12.55, which predicts a maximal antimony concentration of 0.012 mM or 1.44 mg Sb/L at 2 mM calcium. This corresponds to an antimony trioxide concentration of 1.73 mg/L. Since reconstituted standard water is more relevant for natural water conditions, the value of 2.76 mg/L will be used for the screening assessment of ecological risks.

Further water solubility testing was performed for antimony trioxide (EURAR 2008) at seven pH levels between pH 1 and pH 10 at 20.7 - 20.8°C in reconstituted standard water (ISO 6341). The solutions were agitated for 24 hours at 100 revolutions per minute with an antimony trioxide loading of 100 mg/L. The extent of dissolution of antimony trioxide decreased from a maximum of 4.73 mg/L at pH 1 to a minimum of 0.618 mg/L at pH 7. The solubility of antimony trioxide then increased as the pH increased from 7 to 8, after which a new equilibrium was established. When the pH increased from pH 8 to pH 10, the increase in solubility (from 1.86 to 2.16 mg/L) was proportionally much lower (EURAR 2008). The solubilities measured under acidic conditions (i.e. 4.37 mg/L at pH 1, 2.18 mg/L at pH 3; total dissolved antimony) are used for determining the potential for dissolution at stomach pHs values for the human health risk assessment.

Sources

Antimony trioxide is both naturally occurring and human-made. Although antimony is a metalloid (i.e., possesses electronegativity and ionization energy properties intermediate between those of a metal and a non-metal), it will be referred to as a metal in this assessment. In the environment, antimony trioxide is found in ores such as senarmonite, valentinite and exitelite (ATSDR 1992; Reimann and de Caritat 1998; Scorecard 2009). The majority of these ores are located in China, South Africa, Bolivia, Tajikistan and

Russia (Carlin 1995). Given its negligible vapour pressure and limited water solubility, antimony trioxide will tend to remain in soil rather than migrate into other environmental media, such as air or water. Natural sources of antimony trioxide in air in Canada may be limited to sea salt spray and wind-blown dust. Anthropogenic releases from manufacturing, formulation, processing and use may also introduce antimony trioxide into the environment. Manufacturing can occur via two routes; 1) oxidation of antimony metal (equation 1):

$$4\ Sb + 3\ O_2 \rightarrow 2\ Sb_2O_3 \qquad [1]$$

or, 2) oxidation from crude stibnite (antimony trisulphide) followed by revolatilization (vaporization and condensation) of antimony trioxide (equations 2-3):

$$2\ Sb_2S_3 + 9\ O_2 \rightarrow 2\ Sb_2O_3 + 6\ SO_2 \qquad [2]$$

$$Sb_2O_3\ (solid) + heat \rightarrow Sb_2O_3\ (gas) \qquad [3]$$

Both methods require intense heat from furnaces, while the latter requires a further process to purify the compound (ATSDR 1992; EURAR 2008) (equations 1-3).

Major anthropogenic emissions of antimony (all forms) to the atmosphere were estimated by Lantzy and Mackenzie (1979) to be 39 times higher than natural emissions. More recently, Pacyna and Pacyna (2001) estimated that natural and anthropogenic emissions may be comparable in size, or anthropogenic emissions may even exceed those from natural sources. According to Pacyna and Pacyna (2001), major anthropogenic emissions (in order of importance globally) result from fuel combustion, nonferrous metal production and refuse incineration. On a global basis, incineration activities are estimated to release approximately half as much antimony as smelters (ATSDR 1992). In contrast, non-ferrous metal mining and smelting are important industrial sectors in Canada that are expected to be the main sources of antimony release to the Canadian environment.

Based on information submitted under section 71 of CEPA 1999, 1 000 000 - 10 000 000 kg of antimony trioxide was manufactured in Canada in 2006, and just over 1 850 000 kg was imported during the same year. Canadian companies also reported using approximately 3 270 000 kg of antimony trioxide in 2006 (Environment Canada 2009a). Products containing antimony trioxide may enter the country even if they are not identified as such in the section 71 survey because they may be imported in manufactured items or consumer products or in quantities below the 100 kg reporting threshold for the survey. However, the amount of antimony trioxide entering Canada in this manner cannot be estimated from the limited information that is available.

Uses

Based on information identified in the scientific and technical literature, antimony trioxide is primarily used as a plastic catalyst in the manufacture of polyethylene

terephthalate (PET) plastic as well as in combination with halogenated compounds as a synergist to enhance flame-inhibiting properties (ATSDR 1992; Touval 2004; Haldimann et al. 2007; Kirk-Othmer 2007). Adding antimony trioxide reduces the amount of halogenated flame retardant used to impart a given level of flame resistance (Touval 2004). Polyvinyl chloride (PVC) is an inherently inflammable halogenated polymer to which antimony trioxide may be added to improve flame retardant properties, particularly when flammable plasticizers are added. As a flame retardant, antimony trioxide may be added to PVC and non-PVC plastics, textiles and rubbers. Typically, these products find use in electrical equipment, wires, automotive parts, some building materials and packaging (Wang et al.2006; EURAR 2008). Flame retardants containing antimony trioxide are used in commercial and household items, including furniture, carpets, mattress covers, draperies and textiles, paper and plastic (Jenkins et al. 1998; EFRA 2007). In addition, antimony trioxide is used in enamels for ceramics and plastics, pigments in paint and ceramics, and stabilizers in glass, rubber and adhesives (ATSDR 1992; NTP 2005; EFRA 2007; EURAR 2008; Oorts et al. 2008; i2a 2009).

It should be noted that many of the uses mentioned above also pertain to other antimony compounds. For example, antimony pentoxide and sodium antimonite are also synergistic flame retardants; antimony pentasulphide is used in the production of synthetic rubber; and antimony trisulphate is used in brake linings (EURAR 2008). In the human health related exposures of this assessment, where uses of antimony trioxide are known but the speciation of antimony is not specified, the data are conservatively assumed to be for antimony trioxide.

According to submissions made under section 71 of CEPA 1999, use patterns in Canada include adhesive, binder, sealant, filler; fibre insulator; catalyst, accelerator, initiator, activator; colorant, pigment, stain, dye, ink; flame retardant, fire extinguishing agent; formulation component; polymer additive; polymer, component of a formulation; and component of glass tubing (Environment Canada 2009a).

In some Canadian locations, antimony was used as a replacement for lead in pipe solder, but this was not a significant source of antimony in water (Health Canada 1997). While antimony trioxide has pesticidal uses, it is currently not listed in Canada as an active ingredient or formulant used in pest control products, nor is it used in any fertilizer products in Canada (2009 personal communication from Pest Management Regulatory Agency and Canadian Food Inspection Agency, Health Canada, to Existing Substances, Health Canada; unreferenced). It is also not an approved feed ingredient for animals in Canada (2009 personal communication from Canadian Food Inspection Agency, Health Canada, to Existing Substances, Health Canada; unreferenced).

Although antimony and its compounds are listed on Health Canada's Cosmetic Ingredient Hotlist and are therefore prohibited from being intentionally used as ingredients in cosmetics, antimony trioxide may be present as a manufacturing impurity (Health Canada 2007). Furthermore, antimony trioxide is not listed in the Drug Product Database, the Natural Health Products Ingredients Database or the Licensed Natural Health Products Database as a medicinal or non-medicinal ingredient present in final pharmaceutical

products, natural health products or veterinary drugs manufactured in Canada (2009 personal communications from Natural Health Products Directorate, Therapeutic Products Directorate and Veterinary Drugs Directorate, Health Canada, to Existing Substances, Health Canada; unreferenced). Antimony pentoxide is present in pharmaceutical products used in the treatment of leishmaniasis and schistosomiasis (2009 personal communication from Therapeutic Products Directorate, Health Canada, to Existing Substances, Health Canada; unreferenced). Antimony trisulphide, another antimony salt, is listed as a medicinal ingredient in two homeopathic products in the Licensed Natural Health Products Database (LNHPD 2009).

It is possible that antimony trioxide is present as an impurity in the food additive titanium dioxide. In Canada, titanium dioxide is permitted as a food colour (Canada 1978a). The *Food and Drug Regulations* limit the amount of total antimony, expressed as the metal, in titanium dioxide to not more than 50 parts per million (ppm) (Canada 1978b). However, unlike antimony metal, there is no specified limit for the amount of antimony trioxide in titanium dioxide in the *Food and Drug Regulations* (2009 personal communication from Food Directorate, Health Canada, to Existing Substances, Health Canada; unreferenced).

In addition PET plastic, manufactured using antimony trioxide as a catalyst, is used to fabricate water bottles and a variety of food packaging materials (2009 personal communication from Food Directorate, Health Canada, to Existing Substances, Health Canada; unreferenced).

Releases to the Environment

Information reported under section 71 of CEPA 1999 indicated that between 1000 to 10 000 kg of antimony trioxide was released into the environment in 2006. Land received the majority of those releases, with small fractions released to air and water (Environment Canada 2009a). In addition to environmental releases, 100 000 - 1 000 000 kg of antimony trioxide was transferred to hazardous waste facilities, and approximately 14 500 kg was transferred to non-hazardous waste facilities (Environment Canada 2009a).

The National Pollutant Release Inventory (NPRI) collects data only on the combined release of antimony and its compounds. The portion of the total antimony that is in the form of antimony trioxide is unknown. In 2006, the amount of total antimony released to air was 888 kg, with 29 000 kg released to water and 1200 kg released to land (NPRI 2008).

In 2006, the US Toxics Release Inventory (TRI) reported a total of 5240 tonnes of antimony and its compounds released to the environment. Of the total 465 tonnes were antimony, whereas 4775 tonnes consisted of antimony compounds, including antimony trioxide (TRI 2008).

Emissions to Air from Coal Combustion

Alberta and British Colombia are the leading provinces for coal mining and account for 82% of coal produced in Canada. Bedrock in western Canada contains mostly sub-bituminous coal (Stone, 2004) which has higher concentrations of trace metals, including antimony, than many other coals. The antimony content in sub-bituminous coal is 0.722 mg/kg, compared with 1.39 mg/kg in bituminous coal (Bragg et al. 2006). Emission control devices for coal-fired power plants are believed to recover 95-99.9% of antimony released from combustion into stacks (EURAR 2008). Canada's total coal consumption was estimated to be about 59 000 000 tonnes in 2004 (Stone 2004). Total releases of antimony from coal combustion are thus estimated to be between 42 and 4100 kg.

No matter which form of antimony is present in coal, combustion conditions (high temperature and excess oxygen) are expected to lead to the formation of antimony oxides. Whether antimony is released in the trivalent (Sb_2O_3) or pentavalent (Sb_2O_5) form is not well characterized in published reports. In the European Union (EU) risk assessment report (EURAR 2008), it is assumed that coal combustion produces the trivalent form which will be oxidized further to the pentavalent form following contact with moisture and oxygen in air. Conversely, Pavageau et al. (2004) studied coal combustion speciation and observed the formation of pentavalent antimony oxide. Bottom and fly ashes are the main solid waste materials from coal combustion. Bottom ash is generally collected and landfilled in Canada. Fly ash is mostly captured through gas cleanup devices and landfilled or used in the cement/concrete industry in Canada, but a small amount can be emitted to air as particulate matter (PM). Emitted PM will ultimately find its way to soil, surface water and sediments.

Emissions to to Air from Non-Ferrous Metal Production (Smelters)

The form of antimony released to the atmosphere from smelting is antimony trioxide when crude stibnite is oxidised to form antimony trioxide at furnaces temperatures of 850 to 1 000°C (EURAR 2008). The same source it is stated that elemental antimony will oxidize following air contact to form antimony trioxide (Sb_2O_3) and condense after smelting operations on suspended matter of less than 1µm in diameter. Takaoka et al. (2005) reported that the elemental antimony or antimony trioxide emitted from a smelter is converted into Sb (V) compounds in the contaminated soil. Similarly, an atmospheric conversion also occurs to form the pentavalent form from the trivalent form emitted from smelters (EURAR 2008).

Skeaff and Dubreuil (1997) estimated antimony releases associated with non-ferrous metal production in 1993 and earlier years for the Sudbury area in Ontario. The amount of antimony released is a function of the amount of refined metal produced, and varies depending on the metal. In total, 92 934 tonnes of lead, 696 473 tonnes of nickel and copper and 757 307 tonnes of zinc were produced in Canada in the year 2003 (Statistics Canada 2005), which could result in total atmospheric releases of antimony of 1859 kg, 1045 kg and 9845 kg, respectively. An annual total atmospheric release of 12 749 kg is therefore estimated for all non-ferrous metal production industries in 2003. This estimate

was obtained using a calculated emission factor from Skeaff and Dubreuil (1997). Few empirical data were available to assess the accuracy of the emission factor. Also, pollution abatement activities have taken place at some facilities since 1993, which have resulted in lower levels of releases Indeed, 280, 530 and 430 kg of antimony were reported to have been released to air in the NPRI for 2006, 2007 and 2008, respectively, by non-ferrous production and processing industries (NPRI 2008).

Releases to Water from Antimony Mining

Only one antimony mine is currently operating in Canada, and 127 tonnes were extracted in the year 2003 (Statistics Canada 2005). This quantity accounts for a small portion of the total used in Canada. Mine effluents may contain oxidized forms of sulphur-associated antimony minerals (e.g., stibnite). The relative proportions of trivalent and pentavalent antimony compounds released in effluents are unknown.

Releases to Soil and/or Surface Water from Automobiles (Brake Abrasion)

Automobile brake pads contain antimony trisulphide (Ceriotti and Amarasiriwardena 2009). Given the friction coefficient and high temperature from the braking process, Uexküll et al. (2005) suggested that a considerable amount of antimony is oxidized to antimony trioxide. It is expected, therefore, that antimony trioxide will be released to roads due to the wear of break pads and then transported as dust or in runoff from precipitation to adjacent soils, surface waters and sediments.

Releases to Air, Water and Soil from Waste Disposal (Incinerators and Application of Biosolids)

Municipal solid waste incineration accounts for less than 3% of total municipal solid waste disposal in Canada. Only seven municipal incinerators are found across the country.

Effluents from facilities may contain antimony as a result of losses from material handling and washing of equipment. Metals (including antimony) in effluents will end up in industrial or municipal wastewater treatment plants, and as a result of alkalinization processes, a fraction will be deposited in sludge. Sludge spreading on agricultural fields is common in Canada, and this would be a source of antimony release to soils. Antimony in soils may be transported to surface waters, following leaching and runoff. However, there is uncertainty about the original form of antimony in the sludge.

Releases Related to Manufacture, Use and Disposal of Products

The releases of antimony trioxide to the environment depend upon various losses of the substance from its manufacture, industrial use and consumer/commercial use. These losses can be grouped into seven types: 1) discharge to wastewater; 2) emission to air; 3) loss to paved/unpaved land surfaces; 4) chemical transformation; 5) disposal to landfill; 6) disposal by recycling; and 7) disposal by incineration. They are estimated based on

regulatory survey data, industry data and data published by different organizations. To assist in estimating these losses, a spreadsheet (Mass Flow Tool) was used that incorporates all data and assumptions required for the estimation (Environment Canada 2009b).

In the following Mass Flow Tool analysis, only the life cycle for the use as a flame retardant was considered, since: 1) on a mass basis, it is the most important use for the substance; 2) the proportion released to wastewater, as calculated by EURAR (2008), is the highest; and 3) no monitoring data near industrial sites using antimony as a flame retardant were found. On the other hand, empirical data that were available for other anthropogenic sources were considered sufficient for exposure estimation.

In the context of the Mass Flow Tool estimation the discharge to wastewater refers to raw wastewater prior to any treatment, either on-site industrial wastewater treatment or off-site municipal sewage treatment. In a similar manner, the loss via chemical transformation refers to changes in substance identity that occur within the manufacture, industrial use or consumer/commercial use stages, but excludes those during waste management operations, such as incineration and wastewater treatment.

The losses estimated for antimony trioxide over its lifecycle associated with its use as flame retardant are presented in Table 3 (Environment Canada 2009c). The substance is expected to be released to wastewater in proportions ranging from 0.21% to 0.66 % of the total quantity in Canadian commerce. In general, wastewater is a common source for releases to surface water and soil through application of biosolids from wastewater treatment facilities to agricultural soils.

Table 3. Estimated losses of antimony trioxide during its life cycle as a flame retardant.

Type of loss	Proportion (%)	Pertinent life cycle stages
Wastewater	0.21-0.66	Industrial use
Air emission	-	industrial use
Paved/unpaved surfaces	-	Not applicable
Chemical transformation	-	Not applicable
Landfill	96.3-96.8	Industrial use, consumer/commercial use
Incineration	3	Industrial use, consumer/commercial use

Antimony trioxide may also be released to the environment in small amounts via routes other than industrial wastewater. It is recognized that antimony trioxide contained in manufactured items that are disposed of in landfills could have a potential to be released into groundwater, surface water and/or soil, 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).. It is anticipated that these releases (i.e., leaching from the product and the landfill) are relatively small.

Since this substance is present in some manufactured items and although no information is available on the quantity of manufactured items containing antimony trioxide that are imported into Canada, it is anticipated that the mass lost to wastewater would not be significantly different from those estimated in the exposure scenarios section. However, the quantities sent for waste management (e.g., to landfills) would be higher if importation of these items were taken into consideration. Available information is currently not sufficient to estimate these quantities. Although it is anticipated that the potential releases associated with waste disposal could be larger than the amounts considered in this assessment, the total quantities are expected to be relatively small.

Environmental Fate

Partitioning

As antimony trioxide is a metal-containing inorganic substance, a fate analysis based on octanol-water (log K_{ow}) and organic carbon-water partition coefficients (log K_{oc}) is not applicable. Typical mass balance fugacity modelling is also not applicable to antimony trioxide or to the metal ions that are released from it on dissolution, because, as for other non-volatile chemicals, these substances exert zero partial pressure and fugacity in air (Diamond et al. 1992). The fate of the dissociated antimony ions may in part be characterized by partition coefficients, namely soil-water, suspended sediment-water and sediment–water partition coefficients (K_{sw}, K_{ssdw} and K_{sdw}), which are presented in Table 2. Because of the tendency of this metal to sorb to solid particles in aquatic media (Table 2; K_{ssdw}), a proportion of dissolved forms of this metal will end up in sediments (Table 2; K_{sdw}), through adsorption to settling of suspended particles (Hamilton-Taylor and Willis 1984) or through precipitation of calcium antimonate (EURAR 2008). Note that some non-dissolved antimony trioxide (as parent substance) is also expected to be found in sediments and moist soils. When released to dry soils, antimony trioxide will mainly remain there with some of the substance dissolving and leaching locally into groundwater and/or surface water ecosystems (via runoff) when the soil gets soaked by rain or melting snow and ice. The solid parent substance is not expected to be found in significant amounts in the water column, considering that its density is greater than that of water.

Air

Being a non-gaseous element with a negligible vapour pressure at ambient temperatures (Table 2), antimony trioxide is emitted to air principally in the form of fine PM. According to information in the EU risk assessment report (EURAR 2008): 1) anthropogenic activities may result in long-range transport of a portion of the antimony trioxide emitted to air; 2) combustion or incineration processes are likely to transform antimony compounds to antimony trioxide regardless of the pre-incinerated form of antimony; and 3) there are indications that a significant proportion of the fine particulate antimony trioxide in the atmosphere may be water soluble. A study of wet and dry deposition over an 8-week period in an area that was presumably far from sources found 87% of the deposited antimony dissolved in rain, 11% in PM in rain and only 2% in solid form as dry deposition (Stössel and Michaelis 1986).

Water

Antimony trioxide will dissolve and release antimony ions subsequent to its entry into water. The dissolution pattern of the substance will be dependent upon pH (see Table 2). The primary form issued upon dissolution and hydrolysis of the released Sb(III) ions, is expected to be the neutral species $Sb(OH)_3$. The fate analysis of dissolved antimony indicates that it can transform into a variety of species and form dissolved complexes with dissolved ligands present in the aquatic environment (Schecher and McAvoy 1992; Tipping 2002). The aqueous chemistry of the metal is thus complex and involves a wide range of oxygenated species whose stabilities depend mainly on the acidity and oxygen level of the receiving waters. Under conditions commonly found in oxic freshwaters (i.e., pH between 5 and 9; redox potential [E_h] between 0.5 and 1 V), and based on the E_h-pH diagram taken from Filella et al. (2002a), antimony is expected to be present mostly as dissolved $Sb(OH)_6^-$. This species is issued from the oxidation of Sb(III) to Sb(V), which is hydrolysed (EURAR 2008). Under anoxic systems in the same pH range, antimony will be mostly present in the reduced form as dissolved $Sb(OH)_3$. The precipitation of calcium antimonate may limit the concentration of dissolved antimony in the water column (EURAR 2008). The concentration of calcium in natural waters will therefore be an important factor controlling the solubility of antimony. According to thermodynamic data, antimony is generally present as Sb(V) in oxic systems and as Sb(III) in anoxic systems; however, Sb(V) has also been found in anoxic systems and Sb(III) in oxic systems (EURAR 2008).

Antimony is moderately mobile in surface waters (oxidizing conditions, pH 5-8) (Garrett 2004). Filella et al. (2002a, b) reached a similar conclusion in defining antimony as relatively mobile under the same conditions. As noted above, the dominant form of antimony in oxic environments is the anionic $Sb(OH)_6^-$. That form, because of its negative charge, is less likely than the neutral form ($Sb(OH)_3$) to interact with organic material, due to the predominantly negative charge on natural organic material (NOM) at the pH of natural waters (Fillela et al. 2002a, b; EURAR 2008; Strømseng et al. 2009). Tella and Pokrovski (2008) agreed that $Sb(OH)_6^-$ will be the dominant form in organic free aqueous solutions with a near-neutral to basic pH, adding that $Sb(OH)_5$ will be the dominating species at more acidic pH.

There is some experimental evidence for complexation of antimony with NOM. Deng et al. (2001) identified a fraction of antimony associated with dissolved NOM, ranging from 33% to 67% of total antimony in three lakes receiving atmospheric loading from the Sudbury smelters. Measurements were made in surficial oxic waters, so that Sb(V) was dominant. Only 15% of total antimony was in the form of Sb(III) in one of the three lakes, whereas this oxidation state was not detected in the other two lakes. Lake pH was not reported but if it was rather acidic (i.e., below pH 7), the presence of $Sb(OH)_5$ would be favored over $Sb(OH)_6^-$. Buschmann and Sigg (2004) determined conditional distribution coefficients for Sb(III) binding to three commercial humic acids that differed in carbon content and number of functional groups (terrestrial, coal and aquatic) at environmentally relevant Sb(III)/dissolved organic carbon (DOC) ratios and as a function

of pH using an equilibrium dialysis method. The authors proposed that over 30% of the total Sb(III) content may be bound to NOM under environmentally relevant conditions. In their review, Fillela et al. (2002b) reported a few studies that showed little or no interactions between antimony and dissolved NOM. They concluded that information on antimony interactions with NOM is very sparse and does not allow any rigorous conclusion to be drawn regarding its role in antimony fate in natural aquatic systems.

It was not possible to use the Windermere Humic Aqueous Model, version 6.0 (WHAM 2001; Tipping 2002), to obtain further information on the speciation of antimony in natural waters, particularly on the importance of complexation of antimony by colloidal organic matter in a natural context, because of the lack of thermodynamic stability constants for complexes of dissolved forms of antimony with natural organic and inorganic ligands.

Sediments and Soils

It has been known for a long time that sediments act as sinks for trace metals in aquatic systems (Förstner and Wittmann 1981). The suspended particulate flux of surface waters acts as a "conveyor-belt" mechanism whereby metals are "scavenged", adsorbed by or incorporated into particles generated *in situ* or of allochthonous (external) origin. In turn, after being transported some distance, these suspended particles fall through the water column and eventually settle to bottom sediments (Santschi 1984).

The following important conclusions have been drawn from the literature review conducted by the EU (EURAR 2008) regarding the fate of antimony in sediments:

1) The adsorption of antimony in oxic sediments has been correlated with the presence of iron, manganese, and aluminum oxides,
2) The decrease in bioavailable antimony in water by adsorption onto hydrous oxides in oxic sediments is not a permanent decrease, as the hydrous oxides can subsequently dissolve in response to changing pH or E_h conditions. In addition, antimony may become bioavailable to organisms inhabiting the sediment through ingestion of the sediment,
3) In anoxic systems, and in the presence of sulphur, depending on pH, antimony forms soluble or insoluble stibnite, SbS_2^- and $Sb_2S_{3(s)}$, respectively.

Similarly to sediments, soils may be major sinks for metals released by natural sources and anthropogenic activities. Transformation processes will involve dissolution, partitioning (including precipitation) and ageing. The latter designates slow reactions transferring metals from labile pools to insoluble pools (Smolders et al. 2007a). In general, metal bioavailability is governed by the mobility and solubility of geochemical forms (Smolders et al. 2007a). The following most important conclusions have been drawn from the literature review conducted by the EU (EURAR 2008) regarding the fate of antimony in soils:

1) The sorption and precipitation of calcium antimonate seem to be more important than the dissolution processes of antimony trioxide as regards the fate of antimony,
2) The solubility of antimony compounds depends on the soil conditions (mineral and organic matter composition; E_h/pH) and the time given to dissolve,
3) The most important soil characteristic as regards the mobility of antimony in soil (and sediments) appears to be the presence of hydrous oxides of iron, manganese, and aluminum, to which antimony may adsorb. In addition, these hydrous oxides seem to oxidize dissolved trivalent antimonite ($Sb(OH)_3$) to the pentavalent antimonate ($Sb(OH)_6^-$),
4) The largest effect of pH on sorption seems to occur around pH 3–4, at which sorption is maximal, with decreasing sorption at higher pH values. The effect of pH as such is probably less important than the effect of the hydrous oxides. The effect of pH on antimony mobility seems to be mediated by the hydrous oxides, which assume an increasingly negative charge with increasing pH (resulting in weaker sorption of the negatively charged $Sb(OH)_6^-$) pH changes can also however influence the valence of antimony (with higher pH values favouring oxidation) and the solubility of solid antimony trioxide (see discussion in the section on physical/chemical properties),
5) Due to the anionic character of the dissolved species ($Sb(OH)_6^-$), antimony is expected to have a low affinity for organic carbon. However, there are some data indicating that the sorption of Sb(V) by humic acid in acidic soils with high proportions of organic matter may be more important than previously suspected. That being said, the strong Sb(V) scavenging potential of iron(III) hydroxide probably results in a diminished role of organic matter binding in soils with high amounts of noncrystalline hydroxides.

Methylation

There is evidence that antimony may be biomethylated to form volatile species such as trimethylstibnine ($(CH_3)_3Sb$) in the environment (Bentley and Chasteen 2002; Dopp et al. 2004; EURAR 2008). According to Filella et al. (2002a), methylated antimony has been found in natural waters, where it usually accounts for 10% or less of the total dissolved species, and in biota.

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).

The substance antimony trioxide is considered persistent because the trivalent antimony ions that are released into solution when it dissolves cannot be irreversibly degraded. As noted previously, depending upon ambient pH and Eh conditions trivalent antimony can be oxidized to pentavalent antimony, but this transformation is typically reversible.

Therefore, antimony trioxide 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 Aspects

Water Compartment

Couillard et al. (2008) deployed, for a period of 17 days, specimens of the amphipod *Hyalella azteca* along metal contamination gradients in two rivers affected by metal mining in northwestern Quebec. Antimony was accumulated by the transplanted organisms in a dose-dependent manner (i.e. as a function of antimony in the dissolved phase; Figure 3). A similar positive relationship was obtained for the vanadium anion in this study. These findings were obtained in a context of important variability, among sampling sites, in concentrations of dissolved major cations, anions and pH. Overall, these results support the idea that total dissolved antimony is a good predictor of the bioavailability of antimony (i.e., bioaccumulated antimony) that can be used for organisms that obtain most of their metal from the water column (this is the case for *Hyalella*: Borgmann et al. 2007). Pentavalent antimony hydroxides (dissolved $Sb(OH)_5$ and $Sb(OH)_6^-$) are probably the dominant chemical species in these northwestern Quebec (and most other) diluted oxic surface waters. A portion of dissolved antimony might also be associated with the dissolved organic matter. Note that although Sb(III) oxidizes to Sb(V) in the environment, there is no evidence (based on a limited number of studies) that the bioavailability (and resulting toxicity) of these two valence states differs significantly.

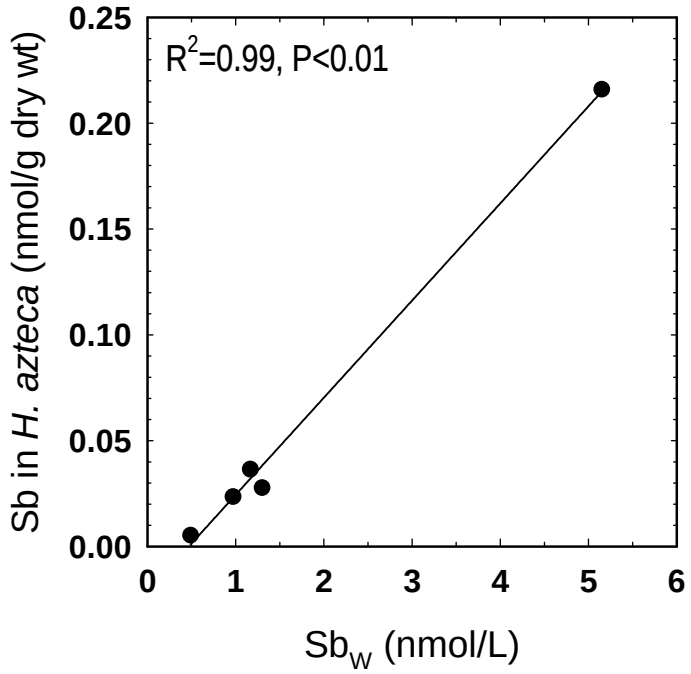


Figure 3. Relationship between antimony concentrations in amphipods and mean dissolved antimony concentrations after 17 days of deployment in two rivers affected by metal mining in northwestern Quebec. Total body concentrations were corrected for time 0, background concentrations. Dissolved antimony levels were obtained after filtering water samples on 0.45 µm membranes and they were corrected for field blanks. Data for both rivers are combined in these relationships (five sites and five sampling times per site) (adapted from Couillard et al. 2008).

The bioavailability of the organo-antimony forms is not well known. However, as mentioned by Farkasovska et al. (1999), methylated species of antimony are less toxic than the inorganic salts. It is therefore possible that they are less bioavailable, but this remains unconfirmed.

Sediment and Soil Compartments

Sediments and soils are much more complex media than surface waters; consequently, determination of the bioavailability of metals in these compartments is not straightforward. According to information in the risk assessment report of the EU (EURAR 2008), bioavailability is, at least to some degree, expected to be reduced in sediment and soil, mostly due to binding to solid-phase aluminum, iron and manganese hydrous oxides. A reduced potential for toxicity of antimony due to sorption may therefore exist in soils and sediments with higher contents of these hydrous oxides. Since sorption has been shown to decrease with increasing pH, antimony toxicity may increase with increasing pH. The examples below support the suggestion that soluble, labile metal forms are associated with elevated bioavailability of antimony in soil.

Tschan et al. (2009) reviewed the available literature on antimony uptake by plants and toxicity risks arising from soil contamination by antimony and found that antimony is generally taken up by terrestrial plants in proportion to the concentration of soluble antimony in soil over a concentration range covering five or more orders of magnitude.

Kuperman et al. (2006) tested antimony, barium and beryllium using reproduction endpoints for *Folsomia candida*, *Eisenia fetida* and *Enchytraeus crypticus* to determine ecological soil screening levels. A sandy loam soil was used because it was expected to represent conditions of relatively high bioavailability. In fact, such soil would normally contain a low proportion of aluminum, iron and manganese hydrous oxides, which can sorb antimony. Therefore, bioavailability of antimony will be higher in this type of soil, and this may be considered as a reasonable worst-case exposure situation.

Potential for Bioaccumulation

Water

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 currently 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 may be highly accumulated from the surrounding medium because of their nutritional essentiality. Furthermore, both essential and non-essential metals may be regulated within relatively narrow margins by the homeostatic and detoxification mechanisms that many organisms possess. It follows that when ambient concentrations of metals are low, BCFs and BAFs often increase. Conversely, when ambient metal concentrations are high, BCFs and BAFs tend to decrease (BAF: DeForest et al. 2007; BCF: McGeer et al. 2003). 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).

Taking account of these issues, a mechanistically based saturation model for bioaccumulation of metals using the freshwater amphipod *Hyalella azteca* as 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, Borgmann et al. (2004) and Norwood et al. (2007) have shown that 1) lethality occurs when tissue concentrations surpass a critical body concentration and 2) critical body concentrations appear relatively constant for a variety of 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/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 the total metal 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 not including 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).

The selection of studies for assessing the bioaccumulation potential of antimony builds on the above knowledge and on accepted methodologies for deriving BCFs and BAFs (OECD 1993, 1996; Arnot and Gobas 2006;). Appendix 1 summarizes 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 compartments, studies with such metal accumulators have been left aside. When information was available, only metal concentrations in soft tissues were considered for invertebrates with shells or important exoskeletons. 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 4 presents the empirical BCF and BAF values found to be in accordance with the criteria and considerations described in Appendix 1. The data presented are for antimony as an element and not for the substance antimony trioxide. As explained in previous sections of this report, antimony trioxide will solubilize in water and transform into antimony ions. These ions are considered potentially bioavailable and can be taken up by organisms. Unless otherwise stated, all BCF/BAF values reported in Table 4 are based on measured concentrations of the antimony element.

Table 4. Experimental data selected for estimating the bioaccumulation potential of antimony from solution[1]

Organism	Study type		Evidence of SS	Water concentration measured	Endpoint	Mean value (L/kg wet weight)	Reliability	Reference
Aquatic organisms								
Freshwater amphipod *Hyalella azteca*	Field	Tr	Y[2]	Y	BAF	5.65 (*n* = 5)	S	Couillard et al. 2008
Freshwater alga *Chlorella vulgaris*	Lab	SS	Y[3]	N[4]	BCF	24	S	Maeda et al. 1997
Freshwater fish *Labeobarbus kimberleyensis*	Field	Su	N	Y	BAF	0.43 Liver only (*n* = 20)	S	Retief et al. 2006
Freshwater insect larva *Hydropsyche* sp.	Field	Su	N	Y	BAF	Range: 0.43–67.9[5] (*n* = 4)	S	Solà and Prat 2006
Terrestrial plants obtaining antimony from soil solution								
Maize *Zea mays*	Lab	SS	N	N[6]	BCF	0.186 (*n* = 2)	S	Tschan et al. 2008
Sunflower *Helianthus annuus*	Lab	SS	N	N[6]	BCF	0.266 (*n* = 2)	S	Tschan et al. 2008

Abbreviations: BAF, bioaccumulation factor; BCF, bioconcentration factor; *n*, number of samples; N, no; S, satisfactory; SS, steady-state study; Su, field survey of organisms, water, sediment, etc.; Tr, steady-state study coupled with transplantation or deployment; Y, yes.

[1] BCFs and BAFs expressed on a dry weight basis have been converted to a wet weight basis using 0.2 g dry weight per 1 g wet weight, which is a reasonable conversion factor for invertebrates and fish (e.g., Ikemoto et al. 2008). For microalgae, the ratio 0.1 g dry weight per 1 g wet weight was used (Maeda et al. 1997). When published information permitted, body concentrations were corrected for antimony concentrations in gut contents, and bioaccumulation ratios (BAFs and BCFs)were corrected for background antimony concentrations in test organism and water.

[2] Explanations provided in text.

[3] Steady state should be reached in a short time for microalgae. This test was multigenerational, and BCF was determined at the maximum antimony concentration reached in log phase.

[4] Only nominal concentrations reported.

[5] BAFs decreased with increases in exposure concentrations; dissolved antimony concentrations at impacted river sites ranged from 0.7 to 3.8 µg/L.

[6] Only measured concentrations in plant shoots reported; aqueous concentrations were nominal; hydroponic studies simulating uptake from soil solution.

Experimental BCFs and BAFs obtained for fish, aquatic invertebrates, plants and algae vary between 0.19 and 67.9 L/kg wet weight (Table 4). The BAFs derived from Couillard et al. (2008) and Solà and Prat (2006) were determined in a context of polymetallic contamination, which may have an influence on antimony bioaccumulation. 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 terms of metal toxicity (Borgmann et al. 2005). Furthermore, *Hyalella* is a species complex that is widely distributed throughout Canada and is often numerically abundant in freshwater habitats (Witt and Hebert 2000). At present, little guidance exists on how to measure BAF in the field. 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 trustworthy methods for analysing metals in water and tissues, and integrated two key characteristics of methodologies for deriving BCF values (OECD 1993,1996; Borgmann et al. 2004):

1) 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;
2) 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 key characteristics were fulfilled for antimony. Many of the antimony concentrations in *Hyalella* and water, corrected for background concentrations, were below detection limits. However, the study was deemed of satisfactory reliability because 1) antimony concentrations (except for one site) were all detectable by the analytical approach used and 2) a strong relationship between antimony levels in amphipods and water was obtained (see Figure 3 above). Specimens were gut-cleared before metal analyses. Total body concentrations were corrected for time 0 background concentrations, and dissolved antimony levels in river waters were corrected for field blanks (see Figure 3). Using these methods, the BAF for antimony was estimated to be 5.65. It can be noted that biouptake of antimony 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. The low bioaccumulation potential of antimony can be clearly seen when the field BAF for antimony (5.65) is compared with the laboratory BCF for bioaccumulative mercury - a value of 9 650 L/kg wet weightt corrected for background mercury levels, and obtained with *Hyalella* specimens exposed in test water of 100 mg/L hardness (Schlekat et al. 2007).

Solà and Prat (2006) monitored the accumulation of seven elements including antimony, in water, sediments and specimens of the genus *Hydropsyche* sp. (Trichoptera) in a river affected by a metal mining effluent. One station was positioned upstream, and four stations were situated downstream of the mine site. The assessor derived BAFs (in L/kg wet weight), calculating concentrations in insect larvae corrected for contribution from gut content and converted to wet weight. In addition, body and aqueous concentrations were corrected for background levels determined at the upstream site. BAF values decreased with increases in dissolved antimony concentrations, the highest concentration being 3.8 µg/L. This result is not unusual as such decreases have been shown to happen for other metals and organisms in field surveys (DeForest et al. 2007). These ratios were probably obtained in a context of acid mine drainage, since very acidic conditions created by the mine prevailed at the collection sites (Solà et al. 2004: pH of 4.5-5.4). BAF may not be the only appropriate metric with which to assess bioaccumulation potential in this insect larva, as metal concentrations in *Hydropsyche* reflect the incorporation of metals taken from sediments as well as from water and diet (Solà and Prat 2006).

Duran et al. (2007) sampled macroinvertebrates at a riverine site 500 m downstream from an antimony mining operation. The following BAFs (in L/kg wet weight) were derived: 43 500 for the isopod *Asellus aquaticus*; 5733 for the amphipod *Gammarus pulex*; 26 600 for the Trichoptera larva *Hydropsyche pellucidula*; and 9250 for the Libellulidae larva *Leucorrhinia dubia*. This study has been rejected, however, because of too limited information with regards to sampling plan, analytical methods, quality assurance and quality control and the absence of physicochemical data for surface waters. Besides, the dissolved antimony concentration reported for the near-field river site, 0.015 µg/L, is suspect because it is below typical background antimony levels for global streamwaters (Reimann and de Caritat 1998).

Soils

Gál et al. (2007) studied the mobility, bioavailability and soil-biota transfer of arsenic and antimony at a former antimony mining and smelting site. Average soil (0-10 cm) pH, E_h and organic matter content were 4.7, 84 mV and 23.2%, respectively. There were significant positive correlations between the antimony concentration in grass and earthworms on one hand, and total antimony concentration in soil on the other land. Biota-soil accumulation factors based on dry weight were low, with mean values reported by the authors of 0.009 for grass, 0.034 for *Lumbricus terrestris* and 0.063 for *Octolasion cyaneum.* These factors were expected to be higher for less antimony-contaminated soils. Indeed, log-log relationships between antimony in soil and biota samples had slopes less than unity which, according to Gál and collaborators (2007), implied that biota–soil accumulation factors are greater when antimony levels in soils are lower.

Potential for Biomagnification

Although field-based BAFs can give some indication of the biomagnification potential of a metal, a better approach is to derive a trophic transfer factor from prey to predator (TTF: DeForest et al. 2007) (also called trophic magnification factor or TMF), 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 antimony and analysed 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 studied. Antimony concentrations in organisms showed no increasing or decreasing trends from lower to higher trophic levels. In contrast, mercury gave clear signs of biomagnification, with mercury concentrations of less than 0.05 µg/g dry weight in phytoplankton increasing to 0.1-1 µg /g dry weight in fish.

Campbell et al. (2005) determined antimony levels and stable carbon and nitrogen isotopes in an Arctic marine food web. Organisms considered included phytoplankton, zooplankton, three species of marine invertebrates, one species of fish, eight species of birds and one species of seal. Antimony was measured in whole organisms, and in muscle and liver tissues of birds and seal. This metal was found in higher concentrations in zooplankton than in liver and muscle of fish, birds and seal; this resulted in negative food web regressions with $\delta^{15}N$ values. On the other hand, mercury concentrations increased with $\delta^{15}N$ values, clearly supporting biomagnification.

The food webs of these two studies appear not 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 exist between organisms collected. The Campbell et al. (2005) investigation suffers from the additional weakness that not reporting metal concentrations on a whole body basis for birds and seal adds uncertainty to the metal trends observed. Despite these limitations and in clear contrast with mercury, antimony apparently does not biomagnify in the aquatic food webs studied.

Overall, there are several lines of evidence to suggest that the bioaccumulation potential of antimony in natural ecosystems is low: very low BCFs and BAFs obtained from three laboratory (steady-state) studies and three field studies, three biota–soil accumulation factors well below 1, and two field investigations indicating the absence of biomagnification of antimony in natural food webs. It is therefore concluded that antimony trioxide does not meet the bioaccumulation criteria (BCF or BAF $\geq$5000) as set out in the *Persistence and Bioaccumulation Regulations* of CEPA1999 (Canada 2000).

Potential to Cause Ecological Harm

Ecological Effects Assessment

Information on the acute and chronic toxicity of dissolved antimony to a variety of aquatic, soil and sediment organisms is reviewed in the risk assessment report prepared recently by the EU (EURAR 2008). The data reviewed indicate that soluble forms of

antimony generally have a moderate potential to cause harm to aquatic, soil and sediment organisms.

The same critical toxicity values (CTVs) and calculated predicted no effect concentrations (PNECs) as those identified in the EU assessment (EURAR 2008) were used in this assessment, given the relevance of the species considered and that there were no more recent published data (up to October 2009) for more sensitive organisms. The PNECs were derived from the lowest acceptable literature value as determined by EURAR (2008). Experimental ecological effects data used as CTVs and corresponding PNECs are summarized in Table 5. Robust study summary (RSS) forms were completed to evaluate the reliability of available key studies. The robust study summary results for the aquatic compartment are presented in Appendix 1. Although robust study summaries were not prepared for the soil and sediment studies as no template is available at this time, the reliability of these studies was judged acceptable in the EURAR (2008). Thus, all of the studies described in Table 5 are deemed to have a satisfactory confidence. Many of the other studies evaluated (notably for aquatic biota) were judged to be unreliable because, among other things, the exposure concentrations were nominal and/or were above the estimated water solubility for antimony (EURAR 2008). The reader should consult the EU risk assessment report (EURAR 2008) for more detailed information.

Table 5. Empirical data for toxicity to aquatic, terrestrial and sediment organisms used to derive CTVs and PNECs (EURAR 2008)

Test organism	Test type	Endpoint	Medium	CTV as total antimony	AF[1]	PNEC as total antimony	Reference
Fish (*Pimephales promelas*)	Chronic 28 days	Growth, NOEC	Surface water	1.13 mg/L	10	0.113 mg/L	Kimball 1978
Invertebrate (*Lumbriculus variegatus*)	Chronic 28 days	Growth, NOEC	Sediment	112 mg/kg dw	10	11.2 mg/kg dw	Heijerick and Vangheluwe 2005a
Invertebrate (*Chironomus riparius*)	Chronic 14 days	Growth, NOEC	Sediment	112 mg/kg dw	10	11.2 mg/kg dw	Heijerick and Vangheluwe 2005b
Plant (*Hordeum vulgare*)	Chronic 5 days	Root elongation NOEC	Soil	999 mg/kg dw	10	37 mg/kg dw[2]	Smolders et al. 2007b

Abbreviations: AF, application factor; CTV, critical toxicity value; dw, dry weight; NOEC, no-observed-effect concentration; PNEC, predicted no-effect concentration.

[1] 10 to account for interspecies and intraspecies variabilities in sensitivity.

[2] PNEC was calculated based on the pore water concentration 9.7 mg/kg dw (not at equilibrium) divided by the AF (10) and multiplied by the K_d (soil–water partition coefficient) used in the study (38 L/kg) to account for equilibrium achievement.

Ecological Exposure Assessment

Background Presence in the Environment

Table 6 presents information on background (baseline) concentrations at Canadian sites thought to have low anthropogenic influence. The background levels are below the PNECs presented in Table 5. These sites were selected as they have physical/chemical properties similar to those of the sites where the highest measured concentrations have been reported (in Table 7 below).

Table 6. Antimony concentrations at sites with low anthropogenic influence

Location	Media	Concentration	Percentile			Method of analysis	Sampling period	Number of samples (N)	Reference
			5th	50th (Median)	90th				
Burnt Island, Egbert, Point Petre, Ontario	Air	0-0.019 µg/m^3	< LOD	< LOD	0.01 µg/m^3	Particulate/ water extraction/ ICP	1988-2004	161	NAtChem 2002
Canada-wide	Glacial till	0-23.8 mg/kg	0.05 mg/kg	0.4 mg/kg	1.9 mg/kg	Total digestion INAA/ICP-MS	1956 to 2006	12629	Rencz et al.2006
Quebec-wide	Sediments and soil	<0.1-4.3 mg/kg	n/a	0.12 mg/kg	0.5 mg/kg (Superior)	Total digestion ICP-MS	1997	1766 (Superior)	Choinière et Beaumier, 1997
Northwest Ontario	Lake sediments	<0.2-60 mg/kg	<0.2 mg/kg	0.2 mg/kg	0.3 mg/kg	Dried, digestion HY-AAS	1976-1989	8661	Friske et al. 1998
Val d'Or, Lac Blouin, Quebec	Lake surface water	0.04-0.17 µg/L	0.046 µg/L	0.1 µg/L	0.14 µg/L	Total digestion ICP-MS	2006	33	MDDEP 2008
Prairies provinces and Northwest Territories	River surface water	0.001-8 µg/L	0.007 µg/L	0.094 µg/L	0.25 µg/L	Total dissolved antimony	2003-2008	1194	NWRI 2003-2008
Abitibi	River surface water	0.0121µg/L	n/a	0.012 µg/L	n/a	Total dissolved antimony	2003	10	Couillard et al. 2008

Abbreviations: HY-AAS, hydride evolution atomic absorption spectrometry; ICP, inductively coupled plasma; ICP-MS, inductively coupled plasma mass spectrometry; INAA, instrumental neutron activation analysis; LOD, limit of detection; na, not available

Exposure Scenarios Based on Monitoring Data and Potential Estimated Releases

The state of the science does not allow a reliable estimation of the speciation of antimony in releases. EURAR (2008) briefly discussed the speciation of antimony combustion products released by coal-fired power plants and metal smelters. It is said that combustion likely leads to antimony trioxide regardless of its form before incineration. Afterwards, trivalent antimony will likely be dissolved in contact with rain or snow, and eventually oxidized to pentavalent forms (Metzger and Braun 1986).

However, it seems very likely that a fraction of the antimony in stack releases to air will be present in pentavalent (Sb_2O_5) form (EURAR 2008). In this assessment, it is assumed that antimony is oxidized by combustion; as a conservative estimation of potential exposures, antimony trioxide (Sb_2O_3) is assumed to be released unless otherwise stated. Table 7 illustrates the main exposure scenarios considered for which monitoring data are available. The concentrations represent the most elevated antimony concentrations monitored in Canada and other countries, and these are used as reasonable worst-case predicted environmental concentrations (PECs). Highest environmental concentrations from other countries were thought to be relevant to potential worst-case Canadian scenarios. Details about each of these exposure scenarios are given below.

Table 7. Anthropogenic sources of antimony trioxide and PECs from exposure scenarios based on antimony monitoring data.

Location	Zonage	Industry	Media	Concentration (PECs)	Sampling period	Reference
Port Colborne, Ontario	Urban	Nickel refinery / iron smelter	Soil	0.10-91.1 mg/kg	2000-2001	MOE 2002
Japan	Rural/ Urban	Roadside/brake abrasion	Soil	0.645-7.27 mg/kg	1999-2001	Ozaki et al. 2004
Texas, USA	Rural	Roadside/brake abrasion	Soil	0.6-9.8 mg/kg	2004	Turer 2005
Saudi Arabia	Urban	Roadside/brake abrasion	Soil	0.48-2.76 mg/kg	2008	Kadi 2009
California, USA	Rural	Roadside/brake abrasion	Soil	0.46-0.6 mg/kg		McKenzie et al. 2009
Abitibi-Temiscamingue, Quebec	Rural	Nickel/Copper smelter (stack deposition from air)	Surface water Bottom water Surface sediments Deep sediment Surface porewater Deep porewater	0.01-1.08 µg/L 0.02-0.89 µg/L 0.2-6.2 mg/kg ND[1]-3.1 mg/kg 0.02-5.50 µg/L 0.01-2.64 µg/L	1997-1998	Kliza and Telmer 2001
One facility in Canada	Rural	Gold smelter	Air	62.4 µg/m3	2004	2009 personal communication from Air Quality Research Division, Environment Canada
Sudbury, Ontario	Rural	Nickel/copper/iron smelter	Till	ND[1]-13 mg/kg	1992-1993	Bajc and Hall 2000
			Soil and Humus	ND[1]-16 mg/kg		
			Surface porewater	< 0.25 µg/L	2000	Chen et al. 2003
Wabamun Lake, Alberta	Rural	Coal-fired power plant	Surface water	0.17-0.25 µg/L	2002	Alberta Environment 2006
			Sediments	0.3-3.4 mg/kg	2002	Alberta Environment 2003

[1] not detected

Coal Combustion

Canada's annual coal consumption is 59 000 000 tonnes (Stone 2004). Coal-fired power plants are responsible for 93% of coal consumption in the country (Stone 2004). Therefore, no other industrial applications are explored. The average antimony concentration in sub-bituminous coal is reported as 0.722 mg/kg by the US Geological Survey (Bragg et al. 2006). The emission factor for coal-fired power plants has been estimated to be 31 mg of antimony per tonne of coal used (Hasanen et al. 1986). Nriagu and Pacyna (1988) estimated the emission factors for release to the atmosphere for coal combustion to be 0.2-1.5 mg/kg as antimony. On the basis of the higher antimony emission factor value, and on a 95% efficiency for pollution control devices estimated by EURAR (2008), it is estimated that 2.1 tonnes of antimony may be released by this industrial sector, based on an antimony concentration of 0.722 mg/kg.

Coal-fired Power Generation Plants

The site considered is located in Alberta, near the large, shallow Lake Wabamun, 65 km west of Edmonton. The lake, surrounded by three coal-fired power plants within a 10 km radius, is believed to provide an ecologically realistic worst case for coal-fired power generation facilities. On this site, power plants are built near coal mines, and emissions resulting from coal mining are consequently reflected in the monitoring data. There is no other major industrial sector present on site, so the monitoring data essentially reflect the influence of the coal sector. Two facilities with their respective coal mines, including the biggest coal-powered electricity plant in Canada (Statistics Canada 2000), are located near the lake and discharge wastewaters into cooling ponds upstream of the lake (Stantec Consulting Ltd. 2003). A third facility is situated a few kilometres southeast from the studied lake and may not affect local metal deposition from the air as dominants winds blow from the northwest (Windfinder 2009). Lake sediments are rich in silt, fine sand, organic matter and coal detritus (Alberta Environment 2002).

Water samples taken by Alberta Environment (2002) were analysed for a variety of elements. Antimony was measured only in the largest coal-fired power plant effluent, at 100 m from an ash lagoon discharge and was below the detection limit (5 µg/L). Another study done in the area measured antimony concentrations in water before and after an oil spill (Alberta Environment 2006). Extractable antimony concentrations, measured by inductively coupled plasma mass spectrometry, ranged between 0.17 and 0.25µg/L. The spill did not have any significant influence on the antimony concentrations.

A paleolimnology study from Donahue et al. (2006) in the same area mentions significant differences in the antimony concentrations in sediments in lakes near coal-fired power plants compared with lakes not perturbed by anthropogenic activities. The mean antimony concentration out of 69 samples collected at various points in Lake Wabamun is 1.5 ± 0.9 mg/kg in sediments (Alberta Environment 2003).

Non-ferrous Metal Production

Nickel and Copper Smelters

Non-ferrous metal smelting releases antimony as a byproduct from air stacks and in bottom ashes. Antimony is often found in igneous rock from the Canadian Shield, along with gold, copper, silver, nickel, iron and lead ores. Antimony releases to air from non-ferrous metal smelters are estimated to be as high as 12 749 kg/year (calculation based on emission factors from Skeaff and Dubreuil 1997). This estimate is higher than what is reported by the NPRI (see the section on releases to the environment). Because of the lower temperature of the stack gas releases from smelters, antimony releases to the atmosphere are mostly associated with PM (EURAR 2008).

The Abitibi-Temiscamingue region in northeastern Quebec is characterized by a high level of non-ferrous metal-producing activities. The biggest smelter in the area is located in Rouyn-Noranda. Many lakes and rivers surround this town. Natural Resources Canada (Kliza and Telmer 2001) ran a 2-year sampling survey on 100 lakes within a 100 km radius of the smelter. This initiative of the Geological Survey of Canada and Metals In The Environment (MITE) program has contributed to a better understanding of smelter air stack PM release and deposition in local lakes.

Surface (0-1 cm) and deep (19-20 cm) sediment samples were collected, and pore water was sampled at the same depths. Surface and bottom water samples were taken from the lakes. The dissolved pore water concentration of antimony is representative of antimony exposure where benthic organisms live. Differences between antimony concentrations in sediments at the two depths may reflect the influence of anthropogenic activities. Concentration ranges for each sample type are described in Table 7. In some cases, antimony concentrations are 2-3 times higher in surface sediments than in deeper sediments. A clear correlation is observed between distance from the smelter and the antimony sediment concentrations, with concentrations decreasing as distance from the source increases.

The Sudbury smelter in Ontario is similar to the Rouyn-Noranda one in that the nickel and copper smelter is located in a town that is at the centre of an active mineral-producing area. Chen et al. (2003) studied metal concentrations from air stack dispersion in sediments and porewaters in lakes surrounding the town. They attempted to examine the antimony speciation in porewaters and at the water-sediment interface. The antimony concentration in water is below the detection limit of 0.25 µg/L. Bajc and Hall (2000) in association with the Ontario Geological Survey, published a study on antimony concentrations in the Sudbury region. Till, soil and humus were sampled at over 370 sites in the area. Most values obtained were below the detection limit; concentration ranges are listed in Table 7. The average concentrations were 0.76 mg/kg in humus, 2.46 mg/kg in soil and 0.56 mg/kg in till samples.

Vehicles Brake Abrasion

Antimony trioxide is produced via vehicle brake abrasion. Brake pads contain antimony trisulphide, which oxidizes with friction and the high temperatures reached during the braking process (EURAR 2008). Assuming that all antimony emitted by cars is issued from this process, we can conservatively assume that the total release is in the form of antimony trioxide.

Concentrations of antimony of between 0.6 and 9.8 mg/kg have been found in soil along roads in other counties (Table 7). It has been estimated by Sternbeck et al. (2002) that emission factors for antimony from vehicle brake abrasion reach a maximum at 65 µg of antimony per vehicle per kilometre (per vehicle-kilometre). A total distance of 332.2 billion vehicle-kilometres is driven in Canada each year (Statistics Canada 2009) and roughly 21.6 tonnes of antimony could therefore be released in this way onto land surfaces and surface waters near roads across the country. The road network in Canada is comprosed of 1 042 000 km of road (Transport Canada 2009), which means that an average of 20.7 g of antimony could be released every year along each kilometre of road in Canada.

Incinerators, Wastewater Treatment Plant Sludge and Waste Disposal

Wastewater treatment plant sludge may be applied to agricultural soils. Antimony speciation in sludge may vary considerably however (Fjallborg and Dave 2004). There is no Canadian information available on the matter, which is complicated by the fact that wastewater and sludge are issued from many different treatment plants.

Most municipal solid waste disposed of in Canada is landfilled (97%): therefore, there are very few large-scale municipal incinerators in Canada. The total municipal waste generated in Canada is estimated at 27 250 000 tonnes for the year 2006 (Statistics Canada 2008b). The total quantity of landfill leachate generated in Canada is estimated to be 2 138 000 m^3/year (Conestoga-Rovers & Associates 2008. concentration of total antimony in leachate is, however, below the detection limit of 0.05 mg/L for all 10 landfill sites monitored by the Chemical Management Plan (Conestoga-Rovers & Associates 2008).

Stack emissions to air from hospital incinerators have been monitored by Environment Canada (2009 personal communication from, Air Quality Research Division, Environment Canada to Ecological Assessment Division, Environment Canada; unreferenced). Most antimony is found in the PM fraction. Antimony is found in air stack emissions at concentrations up to 33 µg/m^3. Facilities are typically equipped with an afterburner air pollution control device, which does little to prevent emissions of metals associated with PM.

Industrial Release from Mining

This scenario was not evaluated as no clear connection could be made with antimony trioxide. This source could warrant further consideration in a more general assessment of antimony and its compounds.

Realistic Worst Case Modeled Aquatic Exposure from Industrial Releases

As antimony trioxide is used industrially and is expected to be released to water, seven site-specific industrial release scenarios for the use of the substance as a flame retardant mainly in plastic products were developed to estimate aquatic concentrations of antimony. The scenarios are based on the information specific to the sites of plastic manufacturing facilities where the highest releases or exposures are expected to occur. The loss of antimony trioxide to sewer water is estimated to be from 0.21% to 0.66% of the total quantity used, resulting from the chemical container handling operations and the compounding process (see section on releases to the environment, Table 3). The scenarios also assume that the releases occur 250 days/year, typical for small and medium-sized facilities. For four of the facilities, the releases are known to be sent to local sewage treatment plants (STPs) which were estimated to have 50% removal rates, based on information from Swedish STPs (EURAR 2008). For the three remaining facilities, the removal rate is assumed to be 0% since there are no data on STPs and the presence of antimony trioxide could not be determined. The specific receiving waters at the sites have a 1.2- to 10-fold dilution capacity for the STP effluent, flows for which vary from approximately 1 000 to 300 000 m^3/day, depending upon the size of the treatment operation. Based on the above assumptions and additional specific data for the sites, and conservatively assuming that antimony trioxide is used at the highest quantity in the reported range for each facility, aquatic concentrations (near the point of discharge) that vary from 0.5 to 70.1 µg/L can be calculated (see Table 8 below) (Environment Canada 2009d).

A consumer release scenario was not performed, as releases linked to consumer products or manufactured items are mainly indoors and releases are quite low, about 0.05% per year. Therefore ecological exposures from this source will likely be limited and not significant. However, scenarios concerning household products were considered in the human health section.

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 risk quotient calculations as well as information on persistence, bioaccumulation, toxicity, sources and fate of the substance.

Antimony is expected to be persistent in water, soil and sediment; it is also expected to have a low bioaccumulation potential. The high importation volumes of antimony trioxide into Canada, along with information on its uses and from monitoring, indicate the likely release from anthropogenic sources and the presence of the metal at levels higher than local background concentrations in some areas of the Canadian environment. Once released into the environment, it will contribute to exposure in water, soil, sediment and air. It has also been demonstrated to generally have moderate potential for toxicity to aquatic, sediment and soil organisms. Table 8 presents the results of risk quotient calculations based on the most elevated concentrations monitored in the Canadian environment and - in cases where Canadian data are missing – in other countries associated with different exposure scenarios. The table also presents quotients for the industrial exposure scenarios for the plastics industry.

Table 8. Risk quotients (RQs) calculated from the different exposure scenarios[1]

Location	Zonage	Industry	Media	PECs (as total antimony)	PNECs[1]	RQs (PEC/PNEC)
Port Colborne, Ontario	Urban	Nickel refinery/iron smelter	Soil	0.10–91.1[2] mg/kg	37 mg/kg dw	0.003–2.46
Texas, USA	Rural	Roadside/brake abrasion	Soil	0.6–9.8 mg/kg	37 mg/kg dw	0.016–0.26
California, USA	Rural	Roadside/brake abrasion	Soil	0.46–0.6 mg/kg	37 mg/kg dw	0.012–0.016
Abitibi-Temiscamingue, Quebec	Rural	Nickel/copper smelter (stack deposition from air)	Surface water	0.01–1.08 µg/L	113 µg/L	8.9e-05 – 9.6e-03
			Bottom water	0.02–0.89 µg/L	113 µg/L	1.8e-04 – 7.9e-03
			Surface sediments	0.2–6.2 mg/kg	11.2 mg/kg dw	0.018–0.55
			Deep sediment	ND[3]–3.1 mg/kg	11.2 mg/kg dw	0–0.28
			Surface pore water[2]	0.02–5.50 µg/L	113 µg/L	1.8e-04 – 0.049
			Deep pore water[2]	0.01–2.64 µg/L	113 µg/L	8.9e-05 – 0.023
One facility in Camada	Rural	Gold smelter	Air	62.4 µg/m^3	–	–
Sudbury, Ontario	Rural	Nickel/copper/iron smelter	Till	ND[3]–13 mg/kg	37 mg/kg dw	0–0.35
			Soil and humus	ND[3]–16 mg/kg		0–0.43
Wabamun Lake, Alberta	Rural	Coal-fired power plant	Air	0.5–2.1 µg/m^3	–	–
			Surface water	0.17–0.25 µg/L	113 µg/L	0.0015–0.002
			Sediments	0.3–3.4 mg/kg	11.2 mg/kg dw	0.027–0.30
Site 1	Industrial	Plastics	Surface water	64.1[4] µg/L	113 µg/L	0.57
Site 2	Industrial	Plastics	Surface water	0.5[4] µg/L	113 µg/L	0.0044
Site 3	Industrial	Plastics	Surface water	17.1[4] µg/L	113 µg/L	0.15
Site 4	Industrial	Plastics	Surface water	55.1[4] µg/L	113 µg/L	0.49
Site 5	Industrial	Plastics	Surface water	70.1[4] µg/L	113 µg/L	0.62
Site 6	Industrial	Plastics	Surface water	32.1[4] µg/L	113 µg/L	0.28

[1] PNECs are those developed in Table 5. The aquatic PNEC was used for calculating the risk quotient for risk from exposure to sediment pore water.
[2] On approximately 1700 samples. Only this value, 91.1, exceeded the PNEC; all other values were always below the PNEC.
[3] Not Detected
[4] Include the maximum background aquatic concentration (0.1 µg/L - Table 6) a negligible addition to the modelled aquatic concentrations (except for site 2).

With one exception, RQs derived from monitoring data are below 1, indicating that measured antimony concentrations in the aquatic, sediment and soil compartments in Canada do not likely reach levels that could cause harmful effects to organisms inhabiting these media. The exception is for Port Colborne soil, where one measurement out of approximately 1700 resulted in a risk quotient of 2.46. The measured environmental concentration of 91.1 mg/kg appears to be an "outlier", with the average being 2.2 mg/kg; the 10^{th}-percentile concentration 0.4mg/kg; the 90^{th}-percentile concentration 5.5 mg/kg and the highest values being 91.1, 34.5, 23.6, 19.1, 18.7, 13.6, 13.3 and 11.3 mg/kg.

For the water compartment, PECs and PNECs for total dissolved antimony were compared, since the limited data available did not allow for bioavailability corrections. For soils and sediment, a conservative approach was taken, since the total concentration monitored was assumed to be as bioavailable as in the toxicity studies. In some cases for sediment, the PNEC was also based on dissolved pore water concentrations.Using a better measurement of the bioavailable concentration in the monitoring sample would likely have lowered some of the risk quotients for soils and sediments. Regardless, risk quotients calculated for all of these reasonable worst-case scenarios were below 1.

Risk quotient analyses based on modelled exposure concentrations were also performed in this assessment for seven industrial facilities (sites 1-7) (Table 8). Site-specific estimates of exposure were made for the aquatic medium near the facilities to determine whether there is potential for ecological harm at these specific sites from the manufacture of plastic products containing antimony trioxide as a flame retardant (the most important use). The site-specific industrial scenarios (considering the actual receiving water bodies) presented above yielded PECs varying from 0.5 to 70.1 µg/L (Environment Canada 2009d). Using the PNEC of 0.113 µg/L (see Table 5), the resulting risk quotients (PEC/PNEC) vary from 0.0044 to 0.62 (Table 8). Therefore, harm to aquatic organisms is not likely at these sites.

No ecological PNECs were developed for the air compartment. However, exposure values for air are considered in the human health assessment below. Long-Range Transport Potential (LRTP) was not quantified in this screening assessment as this source is not expected to contribute significantly to the Predicted Environmental Concentrations (PECs) presented above that represent reasonable worst-case scenarios.

The risk quotients calculated from monitoring and from modelled PECs suggest that antimony trioxide is unlikely to cause ecological harm in Canada.

Uncertainties in Evaluation of Ecological Risk

Information on the influence of abiotic factors (e.g., pH, hardness) on antimony ecotoxicity was limited and could have been used to evaluate potential aquatic toxicity in different types of water across Canada. However, total dissolved antimony is expected to be a good predictor of potential for toxicity in this assessment (see the section on bioavailability aspects section).

No monitoring values in water and sediments were available at industrial sites where antimony trioxide is used as a flame retardant (e.g., plastic manufacturers). Considering that aquatic concentrations were modelled and high uncertainties are associated with extrapolating sediment concentrations from water concentrations, no risk quotients were presented for sediment from these sites.

Accumulation of persistent antimony in sediments over time may increase exposure and associated risks, as sediment acts as a sink for metals (see section on environmental fate). Whether this occurs depends on the rate of accumulation of "clean" sediments (which will tend to reduce the concentration of antimony over time) and the rate of sediment burial that eventually makes the contaminated layer inaccessible to most biota.

Canadian monitoring levels of antimony in soil near roadways were also missing, and this data gap was addressed by considering monitoring information from other countries.

The quantity of antimony trioxide imported in consumer products was unknown, but this source is not expected to contribute significantly to exposures.

There is also uncertainty about the extent to which releases of antimony trioxide were responsible for the antimony present in environmental samples analysed. In the context of this Challenge assessment, all sources of bioavailable antimony attributable to the dissolution of antimony trioxide were considered. The other sources of this metal in the environment were to some extent covered in this assessment through use of monitoring data to estimate PECs. However, although antimony trioxide likely represents most of the total quantity of antimony currently in use in Canada (94% based on the 1984-1986 DSL survey: Environment Canada 1988), the total quantity of antimony released from all possible antimony-containing substances was not systematically considered in this assessment. The conclusion reached in this assessment, that antimony trioxide has a relatively low potential to cause ecological harm in Canada, does not preclude the possible consideration of this substance in a future moiety-based assessment of antimony-containing substances.

Potential to Cause Harm to Human Health

Exposure Assessment

Environmental Media and Food

While antimony trioxide occurs naturally and is known to be released to the environment by human activities, there is not a large source of environmental monitoring data for this compound. Accordingly, it was conservatively assumed that all antimony detected in environmental media was antimony trioxide.

Environmental monitoring of antimony has been ongoing since the late 1960s in both North America and Europe (ATSDR 1992, FOREGS 2005). Studies from both Canada and the United States show detectable levels of antimony in ambient air ranging between

0.0005 and 0.055 µg/m^3 (Brar et al. 1970; ATSDR 1992). Canadian data from an ongoing monitoring site in Port Colborne, Ontario, were used in estimating the intake from ambient air. The maximum antimony trioxide concentration in total suspended PM from the 179 sites was 0.0026 µg/m^3 (MOE 2002). While other reports may have found higher levels of antimony (ATSDR 1992; Dietl et al.1997), the Canadian data from Port Colborne, which were consistent over time, were considered suitable for the intake estimate.

No data were identified regarding the concentrations of antimony in indoor air data from non-occupational settings. Antimony has been identified in the indoor air of industrial facilities (Kentner et al. 1995; Kim et al. 1997; Kim and Jo 2006; EURAR 2008). In one study from South Korea, antimony was also detected at low levels in residences in close proximity to industrial facilities (Kim and Jo 2006). However, due to the low concentrations found in those residences, indoor air is not considered to be a significant source of exposure to antimony trioxide. In the absence of indoor air data, the value of 0.0026 µg/m^3 for antimony trioxide in total suspended PM in outdoor air from the above Canadian study was used as a surrogate for the level of antimony in indoor air.

Antimony metal occurs naturally in surface water and is detected at low levels in drinking water; however antimony and its related compounds are not highly soluble in water. Antimony has been detected in Canadian surface water at concentrations that range from 0.001 to 9.1 mg/L, however, concentrations are typically less then 10 µg/L and often closer to 1 µg/L (Health Canada 1997). ATSDR (1992) reported a study conducted by Eckel and Jacob in 1989 that found that only 70 of 1077 samples contained antimony at a concentration higher than 5 µg/L in surface water in the United States. Drinking water in Canada is usually found to have a lower concentration of antimony than surface water (Health Canada 1997). There are currently no guidelines for antimony trioxide in drinking water in Canada, as there are insufficient data for its presence (2009 personal communication from Water, Air and Climate Change Bureau, Health Canada, to Existing Substances, Health Canada ; unreferenced). Health Canada has established a maximum acceptable concentration of 6 µg /L for antimony in drinking water, and there are no studies in Canada that identified higher concentrations in drinking water (Health Canada 1997). A study of tap water in eight Port Colborne, Ontario homes over a 4-year period found antimony concentrations ranging between 0.45 and 0.97 µg/L (MOE 2002).

Antimony trioxide may also be used as a catalyst in the synthesis of PET, which is used to manufacture plastic water bottles. In several studies conducted globally, antimony has been found to leach from plastic bottles into drinking water at concentrations ranging from 0.095 to 1.31 µg/L (Dabeka et al. 2002, Shotyk, Krachler & Chen 2006, Westerhoff et al. 2008, 2009 personal communication from Food Directorate, Health Canada, to Existing Substances, Health Canada; unreferenced). Dabeka et al. (2002) tested several varieties of bottled water (mineral, soda, spring and distilled) that are commercially available to Canadians and found concentrations of antimony ranging from 0.03 to 1.31 µg/L in 199 samples. Another study found concentrations of antimony ranging from 0.112 to 0.375 µg/L in 12 brands of water packaged in PET bottles purchased in Canada (Shotyk et al. 2006). The maximum concentrations of antimony found in water bottled in

PET plastic are higher than those found in tap water. Therefore, to derive the general population's intake of antimony trioxide from drinking water, the intake of antimony from drinking water was estimated by conservatively assuming that all drinking water contains antimony at the maximum concentration reported from the above studies for antimony in bottled water (1.31 µg/L) and this intake was converted to an intake of antimony trioxide.

Several studies have investigated levels of antimony in soil in Canada and the United States. The presence of antimony is expected as it is naturally found in ores and does not easily migrate from soil to other environmental media. Concentrations of antimony have ranged from 0.15 to 8.8 mg/kg (ATSDR 1992; MOE 1994). In a 50-home study in Ottawa, Ontario, antimony levels were tested in garden soil and residential street dust (Rasmussen et al 2001). Concentrations in garden soil were found to range from 0.11 to 1.98 mg/kg (mean concentration, 0.36 mg/kg; 95th-percentile concentration, 1.00 mg/kg). Concentrations of antimony in street dust ranged from 0.09 to 15.88 mg/kg (mean concentration, 0.89 mg/kg; 95th-percentile concentration, 1.62 mg/kg). Over 1000 samples of soil collected in Port Colborne, Ontario, had antimony at concentrations ranging from 0.10 to 91.1 mg/kg, with a mean concentration of 2.2 mg/kg (MOE 2002). The present screening assessment used the maximum concentration of antimony in soil, assumed to be present as antimony trioxide for this assessment from the Port Colborne study (91.1 mg/kg) to estimate exposures to antimony from soil.

Antimony has also been measured in indoor dust from 48 homes in locations around Ottawa, Ontario (Rasmussen et al. 2001). Concentrations were found to range from 1.16 to 57.41 mg/kg, with a mean concentration of 7.28 mg/kg and a 95th-percentile concentration of 15.38 mg/kg. The authors acknowledged that further work is needed to determine different dust particle sizes, which would allow for more accurate estimation of human absorption of dust and, consequently, antimony (Rasmussen et al 2001). The mean concentration of antimony in the house dust was found to be greater than the mean concentration in soil collected from the same homes, which suggests that there could be one or more additional sources of antimony within the home. As antimony trioxide is used in plastic manufacturing, in flame retardants and polyester fabrics used in household items, as well as in paint enamels for ceramics and glassware, and in paint pigments in plastic material, the presence of these materials in homes may lead to the presence of antimony trioxide in household dust. In a study conducted in Sydney, Australia, Davis and Gulson (2005) found that homes near industrial areas recorded antimony concentrations greater than 30 mg/kg dust, while concentrations of antimony in non-industrial residential sites were approximately 7 mg /kg dust. To estimate the dermal exposure of the general population of Canada to antimony trioxide from house dust, the conservative 95th-percentile value of 15.38 mg/kg for antimony in indoor dust from homes in Ottawa was used. This exposure assessment is summarized in Appendix 4.

No data were found on the concentrations of antimony trioxide in foods as consumed and there are few studies on the concentrations of antimony in food products. Older data from the United States suggest an antimony level below 10 µg Sb/kg in raw coffee beans and processed consumable coffee (Kuennen et al. 1982). The Ontario Ministry of the

Environment (MOE 2002) studied the level of antimony in produce grown in backyard domestic gardens around homes in Port Colborne, Ontario, assuming the antimony may be taken up by root vegetables or other edible plants. Antimony was not detected in celery, tomato or radish (detection limit <0.05 µg/g), but was measured in beet root (7.8 µg/kg), pepper (18 µg/kg) and lettuce (21 µg/kg).

Antimony was also detected in various food items in a total food basket study of food products from grocery chains in Windsor, Ontario (Enviro-Test Laboratories 1992). Antimony concentrations in the 35 items sampled ranged from <20 µg/kg (detection limit) to 160 µg/kg, with antimony detected in shellfish, pasta, vine vegetables, cooking oil, peanut butter and freshwater fish. Canadian data from the Port Colborne and Windsor food studies were used to calculate the estimated daily exposure to antimony from food. This assessment did not adopt the EURAR (2008) proposed oral absorption of 1% for antimony trioxide, because of the high uncertainty associated with the data, as described in the report - "Although all these studies have been performed with different study protocols which do not meet current standards they indicate an average intestinal absorption of 3-8 % (0.15-40 %)…" Instead the exposure assessment incorporates the conservative assumption that 100% of the antimony trioxide ingested with food is absorbed, which may overestimate the contribution from foods.

Antimony is also found in tobacco smoke with smokers and those exposed to second-hand smoke showing increased levels of exposure (Richter et al 2009).

The upper-bounding estimates of daily intake of antimony from air, food, soil and drinking water were calculated and are summarized for all age groups in Appendix 3. When converted to intake levels of antimony trioxide from antimony, using the molar ratio 1.2, the total upper-bounding estimates ranged from 0.4 to 4.5 µg/kg-bw per day. Dermal exposure to antimony trioxide in household dust was estimated in Appendix 4, with values ranging from 0.31 to 0.45 ng/kg-bw per day. With the molar ratio, dermal exposure to antimony trioxide from household dust is estimated to range from 0.37 to 0.54 ng/kg-bw per day.

Consumer Products

Antimony trioxide is used as a plastic catalyst and in flame retardants, commonly in combination with brominated compounds. It is also found in paint enamels for ceramics and glassware, and in paint pigments in plastic material. Concentrations of antimony trioxide in products that may be found in homes are presented in Table 9.

Table 9**:** Concentrations of antimony trioxide in different products

Product type	**Antimony trioxide concentration (mg/kg)**	**Reference**
Plastics	<80000-250000 (polymers) 180-200 (PET) 20000 (PP) 35000-100000 (HDPE)	EURAR 2008 EURAR 2008 Vasile 2000 Vasile 2000

Product type	Antimony trioxide concentration (mg/kg)	Reference
Fabrics (flame retardants)	2000-5000 40000- 60000	HealthyStuff.org 2009 EURAR 2008
Fabrics (polyester)	160-700 >0.6- 25[1]	HealthyStuff.org 2009 Sørensen et al. 2005
Fabrics (poly-cotton blend)	27000- 38000	CPSC 2006a
Enamel/ paints	20000-100000	IPCS 1997
Glass	<10000 8000	EURAR 2008 EURAR 2008
PVC mattress covers	0.2 – 220.6 µg/g	Jenkins 2000
Coated foam (flame retardants)	41000	CPSC 2006a

Abbreviations: HDPE, high-density polyethylene; PET, polyethylene terephthalate; PP, polypropylene; PVC, polyvinyl chloride.
[1] Corrected for antimony exposure.

Migration of antimony trioxide from fabric and upholstery has been evaluated and ranged from 0.0032 to 2.5 mg/cm^2 (NRC 2000; CPSC 2006a).

The Danish Environmental Protection Agency (EPA) of the Danish Ministry of Environment has published three studies containing information on concentrations of antimony (not antimony trioxide) in consumer products. In two of the studies, children's toys made from leather materials were assessed and found to contain antimony at levels ranging from <0.5 to 10 mg/kg (Sørensen et al. 2005). If bromine was also present (a common combination in flame retardants with antimony trioxide), the levels were between 7 and 24 mg/kg (Sørensen et al. 2005). The Danish EPA also considered the dermal migration rate of antimony from polyethylene and cotton fabric napkins to skin. Antimony concentrations in these fabrics ranged from 0.63 to 200 mg/kg (Abildgaard et al. 2003). These scenarios are not considered further in this assessment, as these were not uses identified in Canada (Environment Canada 2009a).

The primary source of exposure to antimony trioxide for the general population in Canada is flame retardants used in furniture upholstery and mattress covers. Flame retardant polyester fabrics used in children's stuffed toys may be another source of exposure. Estimates of exposure to antimony trioxide from mattress covers, furniture upholstery and polyester fabrics used in children's stuffed toys are presented in Appendix 5.

Exposure estimates were derived using algorithms reported by the US National Research Council (NRC 2000) and the US Consumer Product Safety Commission (CPSC 2006b). Dermal exposures to antimony trioxide while lying on a mattress cover or sitting on a couch were considered. The highest consumer product exposure estimates are for infants (aged 0-6 months) from lying on a mattress cover containing antimony trioxide, mediated

by perspiration (45 µg/kg-bw per day). Children up to age 4 were determined to have potential dermal exposure, equal to 2.2 µg/kg-bw per day from mattresses covers from urine mediated transfer. Dermal exposure to young children from sitting on couches was determined to be 30 µg/kg-bw per day.

Due to the low volatility of antimony trioxide, exposure to antimony trioxide vapour is considered to be negligible. Rather, antimony trioxide is released as dust by abrasion or wear. The concentration of antimony trioxide present in particulates emitted from sitting on furniture was estimated to be 0.24 µg/m^3. This resulted in exposure estimates ranging from 0.05 to 0.14 µg/kg-bw per day, with the highest exposures being for children 6 months to 4 years of age. The intake from inhalation when sitting on furniture is considerably higher than exposure from indoor air (see Appendix 3); however, this is considered very conservative since antimony trioxide levels in furniture decrease over time (NRC 2000, CPSC 2006a,b). Inhalation exposure due to dust in the home was estimated, but is considered negligible, with values of 10^{-7} µg/kg-bw per day.

Oral exposure from mouthing polyester fabrics used in children's stuffed toys was considered for children, with an exposure of 0.7 µg/kg-bw per day for infants aged 0-6 months. In addition, sleeping studies for children suggest mouthing of mattress covers during sleeping hours (Howard and Wong 2001; CPSC 2006b). Infants between 0 and 6 months of age are estimated to have an oral exposure of 0.2 µg/kg-bw per day based on this exposure scenario.

Uncertainty in Exposure Assesssment

Confidence in the exposure estimates for environmental media is considered moderate. All soil, water and ambient air data are Canadian and considered adequate to permit quantification of exposure to antimony metal. Data for indoor air are unavailable and ambient air data were used as surrogates. Confidence in data from food sources is moderate to low, as the data are from a combined food basket study and a harvested produce study from Canada; in addition, the detection limit of the analytical method was used as the maximum concentration in each food category in which antimony was not detected. However, due to the lack of environmental data for antimony trioxide, the estimated intakes were calculated based on concentrations of antimony metal, which would, in all likelihood, overestimate any actual exposure to antimony trioxide from foods.

Confidence in the exposure estimates for consumer products is considered moderate. While there is sufficient information regarding the presence of antimony trioxide in products around the home, many of the assumptions for estimating the exposure may be conservative. For example, 100% dermal absorption is assumed. This is considered very conservative, as a study in the United States investigated exposure to antimony trioxide from fabric used in firefighter uniforms and determined that there was no difference in urinary antimony levels before and after the treated uniforms were worn (de Perio et al 20010), suggesting low potential for migration and/or absorption.

Health Effects Assessment

A summary of the available health effects information for antimony trioxide is available in Appendix 6.

The International Agency for Research on Cancer (IARC) has classified antimony trioxide as a Group 2B carcinogen (possibly carcinogenic to humans) based on "sufficient evidence" for its carcinogenicity in experimental animals and "inadequate evidence" for its carcinogenicity in humans (IARC 1989). The European Commission has classified antimony trioxide as a Category 3 carcinogenic substance (causes concern for humans owing to possible carcinogenic effects) with "limited evidence for a carcinogenic effect" (EURAR 2008; ESIS 2009). Under the new European Commission regulation on classification, labelling and packaging (CLP-Regulation (CE) No 1272/2008) entered into force in January 2009, antimony trioxide was classified as a Category 2 carcinogen (suspected human carcinogen) (European Commission 2009).

In experimental animal toxicity studies, an increased incidence of lung tumours was observed in two strains of female rats exposed to antimony trioxide. Exposure of female Charles River Fischer rats to antimony trioxide at 0, 1.9 or 5.0 mg/m^3 for 6 h/day, 5 days/week, for 12 months resulted in a significantly increased incidence of lung tumours (scirrhous carcinomas) in 44% of the exposed rats at 5.0 mg/m^3 (Watt 1983). Tumours were localized in the bronchoalveolar region (scirrhous carcinomas: 0/28, 0/31, 15/34; squamous cell carcinomas: 0/28, 0/31, 2/34; and bronchiolar adenomas: 1/28, 1/31, 3/34; for 0, 1.9 and 5.0 mg/m^3, respectively). When male and female Wistar rats were exposed to antimony trioxide at 0 or 45 mg/m^3 for 7 h/day, 5 days/week, for 12 months, 27% of the exposed female rats developed lung tumours, but no lung tumours were observed in controls in either sex or in the exposed males (Groth et al. 1986). Lung neoplasms (19/70) included 5/19 scirrhous carcinomas, 9/19 squamous cell carcinomas and 11/19 bronchoalveolar adenomas and carcinomas. In another study in which male and female Fischer 344 rats were exposed to antimony trioxide at 0, 0.06, 0.51 or 4.50 mg/m^3 for 6 h/day, 5 days/week, for 12 months, neoplastic effects were not observed (Newton et al. 1994). According to Newton et al. (1994), a pathologist who compared the degree of lung damage in the pathological slides from the three studies (Watt 1983; Groth et al. 1986; Newton et al. 1994) suggested that the rats in the Watt (1983) study might have been exposed to higher exposure concentrations than stated. Furthermore, the severity of lung damage could also be related to strain sensitivity and particle size. The US National Toxicology Program (NTP) (2007a, b) began 2-year toxicology and carcinogenicity inhalation studies on antimony trioxide in both rats and mice in 2008, but only the 14-days preliminary results are currently available. No long-term oral or dermal studies were identified.

Two comparative mortality studies suggested an association of lung, liver, biliary tract and gall bladder cancer development in humans who were occupationally exposed to antimony trioxide (Jones 1994; Schnorr et al. 1995). However, antimony plant workers were generally co-exposed to other chemicals.

No classifications by international regulatory agencies for the mutagenicity of antimony trioxide in somatic cells or germ cells were identified. The EU risk assessment (EURAR 2008) proposed that there is no systemic mutagenicity by the oral route of exposure and expected no concern for local mutagenicity of antimony trioxide. Two expert groups, the Swedish Critical Group for Occupational Standards (SCG 2000) and the European Food Safety Authority (EFSA 2004), both noted the clastogenic effect *in vitro* and independently decided that no conclusive *in vivo* evidence of genotoxicity was available for antimony trioxide.

Limited mutagenic potential was observed in *in vitro* mutation assays. Negative results were obtained from Ames tests in different strains of *Salmonella typhimurium* and *Escherichia coli* (Kanematsu et al. 1980; Kuroda et al. 1991; Elliott et al. 1998). Positive results were obtained from deoxyribonucleic acid (DNA) repair *Rec* assays (Kanematsu et al. 1980; Kuroda et al. 1991). In mammalian cells, no effects on gene mutation were observed in mouse lymphoma L5178Y $TK^{+/-}$ cells (Elliott et al. 1998). In terms of clastogenic effects, sister chromatid exchange (SCE) was positive in V79 Chinese hamster cells and human lymphocytes in culture (Kuroda et al. 1991; Gebel et al. 1997). Induction of chromosomal aberration was observed in cultured human lymphocytes at high concentrations (Elliott et al. 1998). In *in vivo* assays, no induction of micronucleus in bone marrow was observed in rats or mice by the oral route (Elliott et al. 1998; Kirkland et al. 2007). For the inhalation route, induction of micronucleus in pheripheral blood was observed in mice but not in rats (NTP 2010). There was no induction of micronucleus or SCE in workers occupationally exposed to antimony trioxide at 0.000 062 or 0.000 14 mg/m^3 (Cavallo et al. 2002). Generally, negative results were obtained for chromosomal aberration, including a standard study conducted in rats (Gurnani et al. 1992, 1993; Kirkland et al. 2007). With respect to DNA damage or repair, antimony trioxide had no effect on unscheduled DNA synthesis in the hepatocytes of rats orally administered antimony trioxide (Elliott et al. 1998). Formamido-pyrimidine-glycosylate enzyme-modified comet assay using blood obtained from workers occupationally exposed to antimony trioxide at 0.000 14 mg/m^3 showed a higher level of oxidative DNA damage compared with control, but workers had possibly been exposed to other chemicals (Cavallo et al. 2002). In germ cells, limited data were available for assessing the mutagenicity potential. No sperm head abnormalities were observed in mice orally administered antimony trioxide for 21 days (Gurnani et al. 1992).

The mode of induction of tumours has not been fully elucidated. ATSDR (1992) suggested that the carcinogenicity of inhaled antimony and related compounds was related to pulmonary deposition and the consequent induction of reactive processes, including macrophage infiltration and fibrosis. Recently, the EU risk assessment (EURAR 2008), which was reviewed by the Organisation for Economic Co-operation and Development Screening Information Dataset Initial Assessment Meeting in 2008 (OECD 2008), noted that "despite the lack of conclusive data on local genotoxicity in the lung, the overall expert judgement by the Technical Committee of New and Existing Chemical Substances is that the most likely mechanism for carcinogenicity appears to be impaired lung clearance and particle overload followed by an inflammatory response,

fibrosis and tumours. Consequently, diantimony trioxide can be regarded as a threshold carcinogen." This is supported by preliminary data from a cellular toxicity study conducted by Health Canada indicating that antimony trioxide treatment strongly induced phagocytic oxidative burst in rat bronchoalveolar macrophages *in vitro* (2010 emails from Environmental Health Science and Research Bureau, Health Canada, to Existing Substances Division, Health Canada; unreferenced). Oxidative burst generally leads to the release of reactive species and the production of inflammatory mediators (Iles and Forman 2002; Gwinn and Vallyathan 2006). The association of pulmonary overload with inhalation of poorly soluble particles with characteristics and behaviour similar to those of antimony trioxide was common where lung tumours were developed only in rats, while lung inflammations were observed in mice or primates (Mossman 2000; Borm et al. 2004; Knaapen et al. 2004).

No classifications for reproductive or developmental toxicity were available from international regulatory agencies. In oral gavage studies, no testicular toxicity was seen in both male rats and mice repeatedly exposed to antimony trioxide doses up to 1200 mg/kg-bw per day for 4 weeks (Omura et al. 2002). In a limited inhalation study in which female rats were repeatedly exposed to antimony trioxide at 250 mg/m^3 for 1.5–2 months premating and then throughout mating and gestation, some adverse effects on fertility were observed (Belyaeva 1967). In a limited study, embryotoxic effects were observed in female rats (albino, 6–7 per group) exposed to 0.082 mg/m^3 24 h/day throughout gestation (Grin' et al. 1987). However, a more thorough study conducted in Sprague-Dawley rats exposed to antimony trioxide at concentrations up to 6.3 mg/m^3 throughout gestation showed no significant effects on reproductive or developmental indices (MPI Research, Inc. 2003). Women occupationally exposed to antimony, antimony trioxide and antimony pentasulphide dust for 2 years had a higher incidence of various sexual disturbances, and their babies had lower weights 3–12 months after birth (Belyaeva 1967). However, information regarding the control subjects and the exposure levels was not clear. No dermal studies were identified.

Limited effects were observed in oral repeated-dose toxicity studies with antimony trioxide. According to a limited study, the lowest-observed-effect level (LOEL) identified was 500 mg/kg-bw per day based on histopathological changes in liver and an increase in aspartate transaminase (serum glutamic oxaloacetic transaminase) activity in male Wistar rats (5 per group) fed 0%, 1% or 2% antimony trioxide in the diet (corresponding to 0, 500 and 1000 mg/kg-bw per day, respectively) for 24 weeks (Sunagawa 1981). A more thorough study with a higher LOEL was available, but as a conservative approach, the critical LOEL was based on the lowest LOEL identified in the Sunagawa (1981) study. In inhalation assays with antimony trioxide, the major effects were pulmonary related. The critical lowest-observed-effect concentration (LOEC) was 1.9 mg/m^3 based on an increase in lung weight and pulmonary changes in female rats exposed to antimony trioxide for 6 h/day, 5 days/week, for 12 months (Watt 1983). Other than pulmonary effects, there was some evidence for eye irritation, corneal irregularities, chromodacryorrhea (shedding of bloody tears) and cataract development in animals whole body exposed to antimony trioxide (Newton et al. 1994; NTP 2007a). No repeated-dose dermal studies were identified.

In human volunteers, patch tests with fibre containing antimony trioxide did not induce skin reaction (Stevenson 1965; Haskell Laboratory for Toxicology and Industrial Medicine 1970; Motolese et al. 1993). Accidental oral intake of antimony trioxide–contaminated beverages resulted in gastrointestinal symptoms requiring hospitalization, followed by recovery within days (Dunn 1928; Werrin 1963). Although occupational exposure to antimony trioxide dust for a long period of time resulted in signs of adverse effects, the workers generally were also exposed to other antimony-related compounds and traces of arsenic, lead or other metals. Major symptoms included antimony pneumoconiosis, respiratory and pulmonary inflammations, conjunctivitis and dermatitis (Renes 1953; Karajovic 1957; Klucik et al. 1962; McCallum 1963, 1967; Stevenson 1965; Cooper et al. 1968; Potkonjak and Pavlovich 1983; White et al. 1993).

Toxicokinetic studies showed that oral absorption and elimination of antimony trioxide, which contains the trivalent state of antimony (Sb(III)), was slow, and excretion was primarily via feces and to a lesser extent in urine (Gross et al. 1955a; TNO 2005). EURAR (2008) proposed an oral absorption of 1%. Antimony trioxide binds to red blood cells and undergoes significant distribution to most tissues and organs, with the highest concentrations of antimony found in bone marrow and thyroid (Gross et al. 1955a; Sunagawa 1981; Hiraoka 1986). Repeated inhalation exposure of experimental animals and humans to antimony trioxide revealed accumulation of antimony in lung tissues (McCallum et al. 1970; Gerhardsson et al. 1982; Leffler et al. 1984; Newton et al. 1994; Garg et al. 2003). EURAR (2008) proposed a 6.82% inhalation absorption rate based on data on physical particle size and density, a multiple-path particle deposition model and gastrointestinal tract absorption rate. Biological elimination half-times were estimated to be 600–1100 days for non-smokers and 1700–3700 days for smokers based on seven male workers who were accidentally exposed to radioactive antimony trioxide ($^{125}Sb_2O_3$) aerosols (Garg et al. 2003). Excretion of antimony from the lungs occurs rapidly by mucociliary transport, followed by a slower phase. In humans, antimony was detected in blood, urine, breast milk, placenta, amniotic fluid, umbilical cord blood and fetal liver (Belyaeva 1967; Clemente et al. 1982; Iyengar et al. 1982; Shand et al. 1985; Wappelhorst et al. 2002). No toxicokinetic data after dermal exposure were identified. However, EURAR (2008) proposed a 0.26% dermal absorption rate based on an *in vitro* study using human skin (Roper and Stupart 2006).

The confidence in the toxicity database for antimony trioxide is considered to be moderate. Data are identified for carcinogenicity, genotoxicity, reproductive and developmental toxicity, acute and repeated–dose toxicity as well as epidemiology. However, long-term toxicity studies had been conducted only in rats. Standard chronic studies and repeated-dose dermal studies were lacking. Epidemiological studies identified were generally limited by potential cofounding factors. Results from NTP 2-year inhalation carcinogenicity and genetic toxicology studies (micronucleus assay) currently under way in rats and mice on antimony trioxide are expected to address uncertainties in the health effects database.

Characterization of Risk to Human Health

Based on the classifications by other national and international agencies, a critical effect for characterization of risk to human health for antimony trioxide is carcinogenicity by the inhalation route. Lung neoplasms were observed in females of two strains of rats in two of three 1-year inhalation toxicity studies at the highest concentrations tested. No lung tumours were observed in male rats in two 1-year studies in which male rats were tested. In one of the 1-year studies, no significant increased incidence of lung tumours was identified in either sex. The collective evidence from genotoxicity data indicates that antimony trioxide is not likely to be mutagenic but may exhibit some clastogenicity in *in vitro* assays. Although the mode of induction of tumours is not fully elucidated, ATSDR (1992) and EU risk assessment (EURAR 2008) suggested that lung tumour formation is likely related to local inflammatory response and pulmonary overload. Therefore, a threshold approach is used to characterize risk to human health.

Although no classifications for reproductive or developmental toxicity by international regulatory agencies were identified, there was some evidence for adverse effects on fertility in limited developmental and reproductive toxicity studies in experimental animals, as well as in epidemiological studies. The margins of exposure are based on conservative upper-bounding estimates of general population exposure and the critical LOEC and LOEL for cancer and non-cancer effects. The critical LOEC for inhalation is 1.9 mg/m^3 based on an increase in lung weight, pulmonary changes and no significant increased incidence of lung tumours in rats. The critical LOEL for the oral route is 500 mg/kg-bw per day based on minimal hepatic changes observed in the rat subchronic study.

The principal routes of exposure to antimony trioxide for the general population are expected to be from proximity to or contact with certain household items (furniture upholstery, mattress coverings, polyester fabric used for children's stuffed toys).

Based on consumer product scenario modelling, the air concentration of antimony trioxide resulting from sitting on furniture with upholstery containing antimony trioxide was 0.24 µg/m^3. Comparison of this upper-bounding estimate with the critical effect concentration for inhalation of 1.9 mg/m^3 results in a margin of exposure of 7900. The comparison of the oral critical effect level of 500 mg/kg-bw per day with the scenario of a child mouthing the polyester fabric on a plush toy or upholstery (i.e. estimated exposure of 0.7 µg/kg-bw per day) yields a margin of exposure of 710 000. Since no repeated-dose dermal toxicity studies were identified, the oral subchronic study (500 mg/kg-bw per day) is used as a surrogate to compare with exposure from lying on a mattress cover (45 µg/kg-bw per day), which results in a margin of exposure of 11 000. These margins of exposure are considered to be representative of margins for similar exposure scenarios and are considered adequate to account for uncertainties in the health effects and exposure databases.

Comparison of the upper-bounding estimates of total daily intake from environmental media and food (0.44 to 4.45 µg/kg-bw per day) with the critical effect level for repeated exposure via the oral route of 500 mg/kg-bw per day results in margins of exposure ranging from 110 000 to 1 100 000. Leblanc et al. (2005) reported that European daily

antimony exposure from food only was 4 µg/day; in the UK, the upper-range antimony intake from food was 4 µg/day (Ysart et al. 1999), whereas the 97.5th-percentile exposure to antimony from food in France was less than 2 µg/day (Leblanc et al. 2005). Considering that these data are reflective of different dietary patterns, it is likely that the actual margins of exposure for the general Canadian population are greater than those estimated since it is also conservatively assumed that all of the antimony present in environmental media and food originates from antimony trioxide.

Uncertainties in Evaluation of Risk to Human Health

The determination of margins of exposure within the scope of this screening assessment does not take into account possible differences between humans and experimental animals in terms of sensitivity to effects induced by antimony trioxide. Two-year chronic toxicity studies were lacking. Carcinogenicity was observed in two of three 1-year inhalation toxicity studies in rats. As lung tumours were observed in female rats but not in male rats, sex differences might also determine the severity of the health impact. Currently, the purity of antimony trioxide is generally within 99.3–99.5% according to the International Antimony Oxide Industry Association (EURAR 2008). Common impurities include arsenic (0.1–0.2%), a group 1 human carcinogen (carcinogenic to humans), and lead (<0.25%), a group 2A carcinogen (possibly carcinogenic to humans), according to IARC classifications (IARC 1987a, b, 2004, 2006). Impurities could have confounded the results of the experimental animal toxicity studies, although systemic toxicity was generally not observed. Epidemiological studies were also limited because of the lack of exposure data and information with which to characterize co-exposures to other chemicals.

There is uncertainty regarding the accuracy of the estimated exposures to antimony trioxide from environmental media, foods as consumed and consumer products due to the lack of data on the concentrations of antimony trioxide in these sources. However, the estimates of exposure to antimony from environmental media and food are sufficiently conservative for this screening assessment as the maximum concentrations of antimony that were used were Canadian data and the estimates were based on the worst-case assumption that all of the antimony present in these sources was antimony trioxide. There is additional uncertainty arising from the assumption that antimony trioxide in consumer products coming into contact with skin solubilizes, migrates and is then absorbed at a fractional rate. The information regarding the types of products containing antimony trioxide and the release of antimony trioxide from products in the home is considered adequate in this assessment.

Specific to food is the significant absence of robust Canadian survey data documenting concentrations of antimony in foods across all provinces and territories. Contemporary Canadian estimates of antimony trioxide exposure in this screening assessment, based on limited data, are, on a microgram per day basis, approximately 8-13 times as great as those reported in the United States 23 years ago in a preliminary report (Iyengar et al.1987). It is also pertinent that foods from one of the two Canadian datasets used in this

assessment may have been influenced by a point source of contamination. Thus estimates of the actual contribution of antimony trioxide to total antimony from food are unknown.

Conclusion

Based on the information presented in this final screening assessment, it is concluded that antimony trioxide 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, antimony trioxide 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 (Canada 2000).

On the basis of the adequacy of the margins between estimated exposures to antimony trioxide and critical effect levels, it is concluded that antimony trioxide is not 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 antimony trioxide does not meet any of the criteria set out in section 64 of CEPA 1999.

This substance will be considered for inclusion in the DSL inventory update initiative. In addition and where relevant, research and monitoring will support verification of assumptions used during the screening assessment.

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Appendix 1 - Robust Study Summaries

Surface water (aquatic):

Robust Study Summaries Form and Instructions: Aquatic iT				
No	**Item**	**Weight**	**Yes/No**	**Specify**
1	Reference:The effects of lesser known metals and one organic to fathead minnows (Pimephales promelas) and Daphnia magna. Kimball, G.L. 1978. Department of Entomology, Fisheries and Wildlife, University of Minnesota.			
2	Substance identity: CAS RN	n/a		10025919
3	Substance identity: chemical name(s)	n/a		Antimony chloride
4	Chemical composition of the substance	2	Y	
5	Chemical purity	1	Y	Reagent grade
6	Persistence/stability of test substance in aquatic solution reported?	1		N/A
Method				
7	Reference	1	N	
8	OECD, EU, national, or other standard method?	3	N	
9	Justification of the method/protocol if not a standard method was used	2	Y	
10	GLP (Good Laboratory Practice)	3		N/A
Test organism				
11	Organism identity: name	n/a		*Pimephales promelas*
12	Latin or both Latin & common names reported?	1	Y	
13	Life cycle age / stage of test organism	1	Y	fry (embryo larval test)
14	Length and/or weight	1	Y	not spec.
15	Sex	1		n/a
16	Number of organisms per replicate	1	Y	1

17	Organism loading rate	1	N	
18	Food type and feeding periods during the acclimation period	1	N	
Test design / conditions				
19	Test type (acute or chronic	n/a		Chronic
20	Experiment type (laboratory or field	n/a		Lab
21	Exposure pathways (food, water, both)	n/a		Water
22	Exposure duration	n/a		28d
23	Negative or positive controls (specify)	1	Y	Negative
24	Number of replicates (including controls)	1	Y	4
25	Nominal concentrations reported?	1	Y	6 + Control
26	Measured concentrations reported?	3	Y	
27	Food type and feeding periods during the long-term tests	1	Y	
28	Were concentrations measured periodically (especially in the chronic test)?	1	Y	
29	Were the exposure media conditions relevant to the particular chemical reported? (e.g., for the metal toxicity - pH, DOC/TOC, water hardness, temperature)	3	Y	Temperature: 25.1 C, pH:7.97, Dissolved oxygen: 6.87 mg/L, Alkalinity: 234 mg/L
30	Photoperiod and light intensity	1	N	
31	Stock and test solution preparation	1	Y	
32	Was solubilizer/emulsifier used, if the chemical was poorly soluble or unstable?	1		N/A
33	If solubilizer/emulsifier was used, was its concentration reported?	1		N/A
34	If solubilizer/emulsifier was used, was its ecotoxicity reported?	1		N/A
35	Analytical monitoring intervals	1	Y	
36	Statistical methods used	1	Y	
Information relevant to the data quality				
37	Was the endpoint directly caused by the chemical's toxicity, not by organism's health (e.g. when mortality in the control >10%) or physical effects (e.g. 'shading effect')?	n/a	N	Did not report control organisms health/reponse
38	Was the test organism relevant to the Canadian environment?	3	Y	
39	Were the test conditions (pH, temperature, DO, etc.) typical for the test organism?	1	Y	
40	Does system type and design (static, semi-static, flow-through; sealed or open; etc.) correspond to the substance's properties and organism's nature/habits?	2	Y	
41	Was pH of the test water within the range typical for the Canadian environment (6 to 9)?	1	Y	Mean 7.97

42	Was temperature of the test water within the range typical for the Canadian environment (5 to 27°C)?	1	Y	Mean 25.1
43	Was toxicity value below the chemical's water solubility?	3	Y	
Results				
44	Toxicity values (specify endpoint and value)	n/a	n/a	28 day NOEC (lenght): 1.13 mg Sb/L
45	Other endpoints reported - e.g. BCF/BAF, LOEC/NOEC (specify)?	n/a		28 day NOEC (weight): 2.31 mg Sb/L; MATC (Length): 1.62 (Weight): 3.22 mg Sb/L
46	Other adverse effects (e.g. carcinogenicity, mutagenicity) reported?	n/a	N	
47	***Score*: ... %**		***82.9***	
48	***EC Reliability code*:**		***1***	
49	***Reliability category (high, satisfactory, low)*:**		***High Confidence***	
50	***Comments***			

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 should be obtained at SS). Calculation methods may be based on kinetic rate constants or on concentrations obtained at SS (the latter called "Steady State studies").
2. BAFs measured under field exposures defined in time 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 similar advantages 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 low in order to (i) minimize BCF/BAF decreases with increases in exposure concentrations, (ii) be well below levels causing chronic toxicity (e.g., OECD 1996; 1993) and (iii) water concentrations and tissue concentrations must 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 which 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, in these times, brought about notably by sources of inadvertent contamination, poor reproducibility, and problems associated with filtration and separation of dissolved and particulate forms of metals in water (e.g., Batley and Gardner 1977; Beneš and Steinnes 1974; Hume 1973; Stevenson 1985).

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

12. All the criteria above, except No. 8, can be transposed to the evaluation of the present bioaccumulation ratios. The criterion No. 8 is replaced by the criteria below.
13. 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.
14. To the extent possible, gut cleared tissue concentrations are needed for organism exposed to contaminated sediments.
15. 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 criteria above 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 Studies Summaries developed for bioaccumulation data. These RSS are available upon request.

Appendix 3. Upper bounding estimates of daily intake of antimony (and antimony trioxide) by the general population of Canada

Route of exposure	Estimated intake (µg/kg-bw per day) of antimony trioxide by various age groups							
	0–6 months[1]			0.5–4 years[4]	5–11 years[5]	12–19 years[6]	20–59 years[7]	60+ years[8]
	Breast milk fed[2]	Formula fed[3]	Not formula fed					
Air[9]	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (<0.0005)	0.00 (0.0005)
Drinking water[10]	0.00	0.14	0.03	0.02	0.02	0.01	0.01	0.01
Food and beverages[11]	0.00	0.00	3.32	2.64	1.75	1.05	0.94	0.76
Soil[12]	0.36	0.36	0.36	0.59	0.19	0.05	0.04	0.04
Total intake (Sb)	**0.37**	**0.50**	**3.72**	**3.24**	**1.96**	**1.11**	**0.99**	**0.81**
Estimated total intake[13] (Sb_2O_3)	**0.44**	**0.60**	**4.46**	**3.89**	**2.35**	**1.33**	**1.19**	**0.97**

1 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).

2 No data were identified on concentrations of antimony trioxide in breast milk.

3 For exclusively formula fed infants, intake from water is synonymous with intake from food. No data were identified on concentrations of antimony trioxide in formula in Canada or elsewhere. The concentration of antimony in water (0.97 µg/L) used to reconstitute formula was based on a Canadian drinking water study (MOE 2002).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).

4 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).

5 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).

6 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).

7 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).

8 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).

9 No data were identified regarding concentrations of antimony trioxide in air; therefore concentrations of antimony were used. The maximum concentration of antimony identified in ambient air in Ontario was 0.0026 µg/m^3 from ongoing monitoring in 2001 (MOE 2002). As concentrations in indoor air were not available, the concentrations in ambient air were used as a surrogate

10 No data were identified regarding concentrations of antimony trioxide in water; therefore concentrations of antimony were used. The maximum concentration of antimony identified in Canadian drinking water was 0.97 µg/L from a study conducted in Port Colborne, Ontario (MOE 2002). The maximum concentration of antimony found to be leaching from PET plastic bottles into drinking water was 1.31 µg/L (Dabeka et al. 2002). As this concentration is higher than that identified in tap water, this value was used to calculate exposure.

11 No data were identified regarding concentrations of antimony trioxide in foods as consumed; therefore concentrations of antimony in food products were used. Estimates of intake from food are based upon concentrations in foods that are selected to represent the 12 food groups addressed in calculating intake (maximum concentration found in cooking oils at 160 µg/kg). In cases where antimony was not detected, the detection limit of 20 µg/kg was used (Health Canada 1998):

- Dairy products: not detected in milk or cheese/butter, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)
- Fats: detected at concentration of 160 µg/kg in cooking oils (Enviro-Test Laboratories 1992)
- Fruits and fruit products: not detected in fruit, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)
- Vegetables (including legumes): detected at concentration of 21 µg/kg in root vegetables grown in domestic gardens (MOE 2002)
- Cereal products: not detected in cereal products, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)
- Meat and poultry: not detected in meat products, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)
- Fish: detected at concentration of 80 µg/kg (Enviro-Test Laboratories 1992)
- Eggs: not detected in eggs, detection limit of <20 µg/kg (Enviro-Test Laboratories 1992)
- Foods, primarily sugar: not detected in food/sugar products, detection limit of <20 µg/kg (Enviro-Test Laboratories 1992)
- Mixed dishes: not detected in mixed dishes/soups, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)
- Beverages: : not detected in alcoholic beverages or soft drinks, detection limit of 20 µg/kg (Enviro-Test Laboratories 1992)

[12] No data were identified regarding the concentration of antimony trioxide in soil or house dust, therefore concentrations of antimony were used. The maximum concentration of antimony measured in soil around homes in Ottawa, Ontario, was 91.1 µg/g (MOE 2002). The 95th-percentile antimony concentration measured in house dust in 50 homes from Ottawa, Ontario, was 15.38 µg/g (Rasmussen et al. 2001). The maximum value for antimony in soil (91.1 µg/g) (rather then the lower concentration from house dust) was used to calculate estimates of antimony intake, and those estimates were converted to estimated intakes of antimony trioxide from soil.

[13] All concentrations used were from antimony measurements in the environment, as there are no documented data on antimony trioxide. To estimate the daily intake of antimony trioxide, the molar ratio of 1.2 was used; multiplying the total antimony intake by the value 1.2 to give the estimated total antimony trioxide intake.

Appendix 4. Upper-bounding estimate of dermal exposure to antimony trioxide in house dust using method from Environ (2003a, b)

Route of exposure	Estimated intake (ng/kg-bw per day) of antimony trioxide by various age groups					
	0–6 months[1]	**0.5–4 years[2]**	**5–11 years[3]**	**12–19 years[4]**	**20–59 years[5]**	**60+ years[6]**
Dermal exposure to dust[7]	0.54	0.41	0.41	0.37	0.50	0.49

[1] Assumed to weigh 7.5 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 1695 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.05 mg/cm^2 per day and exposure frequency of 24 h/day (Environ 2003a, b), absorption factor is 0.26%.

[2] Assumed to weigh 15.5 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 2890 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.05 mg/cm^2 per day and exposure frequency of 22 h/day (average of 1 to 2 year-old child and 3 to 5 year-old child) (Environ 2003a, b), absorption factor is 0.26%.

[3] Assumed to weigh 31.0 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 5120 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.07 mg/cm^2 per day (average of 3 to 5 year-old child, 6 to 8 year-old child and 9 to 11 year-old child) and exposure frequency of 18 h/day (average of 3 to 5 year-old child, 6 to 8 year-old child and 9 to 11 year-old child) (Environ 2003a, b), absorption factor is 0.26%.

[4] Assumed to weigh 59.4 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 9390 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.07 mg/cm^2 per day and exposure frequency of 17 h/day (Environ 2003a, b), absorption factor is 0.26%.

[5] Assumed to weigh 70.9 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 10 555 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.07 mg/cm^2 per day and exposure frequency of 24 h/day (Environ 2003a, b), absorption factor is 0.26%.

[6] Assumed to weigh 72.0 kg (Health Canada 1998), surface area of hands, arms, legs and feet of 10 555 cm^2 (Health Canada 1995), adherence rate of dust to skin of 0.07 mg/cm^2 per day and exposure frequency of 24 h/day (Environ 2003a, b), absorption factor is 0.26%.

[7] Concentrations of antimony were measured in indoor dust in studies in Canada and Australia. The maximum concentration of antimony measured in dust from homes was 15.38 mg/kg. This value is from a Canadian study of dust collected in homes around Ottawa, Ontario (Rasmussen et al. 2001). All estimated intakes were converted by molar weight to give dermal exposure to antimony trioxide. See below for an example calculation.

Example Calculation

Scenario	**Assumptions**	**Estimated exposure**
Exposure to dust	**Dermal – Child (0.5–4 years of age)** Suggested by Environ (2003a, b) for a child less than 1 year old: Concentration of antimony in house dust (C_{dust}) is 15.38 µg/g (Rasmussen et al. 2001). Units conversion factor of 1×10^{-6} g/µg (CF_1), adherence rate of dust to skin (AR_{dust}) is 0.05 mg/cm^2 per day, exposed skin surface area (S_{at}) (hands, arms, legs and feet) of 2890 cm^2 (Health Canada 1995), exposure frequency at home (EF_h) is 22 h/day, units conversion factor day/hr (CF_2) of 0.0417 day/h, body weight of 15.5 kg (Health Canada 1998), and absorption factor for the dermal route (AF_d) is 0.0026 (EURAR 2008). Intake= $\frac{C_{dust} \times CF_1 \times AR_{dust} \times S_{at} \times AF_d \times EF_h \times CF_2}{BW}$ = 15.38 µg/g × 1×10^{-6} g/µg × 0.05 mg/cm^2 per day × 2890 cm^2 × 0.0026 × 22 h/day × 0.0417 day/h / 15.5 kg = 3.42×10^{-7} mg Sb/kg-bw per day × 1.2 Sb_2O_3/Sb = 4.10×10^{-7} mg Sb_2O_3/kg-bw per day	0.41 ng/kg-bw per day (Sb_2O_3)

Appendix 5: Upper -bounding estimates of potential exposure to antimony trioxide from consumer products[1]

Consumer product scenarios	Assumptions	Estimated exposure
Sitting on a couch	**Dermal – Child (aged 0.5-4 years)** Use scenario for antimony trioxide from NRC (2000). Scenario assumes that a person spends 25% of his/her day, exposing 25% of his/her body sitting on antimony trioxide treated upholstery where clothing presents no barrier to migration and there would be sufficient water present (i.e., from sweat) to allow dissolution. To calculate the intake of antimony trioxide from dermal exposure, the following assumptions are made: area density (S_a: application rate to the fabric or back-coating) is 2.5 mg/cm^2 (EURAR 2008), area of body in contact (A_b: 25% upper torso [trunk, arms and neck]) is 758.5 cm^2 (Health Canada 1995), fractional rate of extraction by sweat (μ_w) is 0.001/24 hr (NRC 2000), exposure frequency (f_E) is 6 h/day,and body weight (bw) is 15.5 kg (Health Canada 1998). Average exposure $= \frac{S_a \times A_b \times \mu_w \times f_E}{bw}$ $= \frac{2.5\ mg/cm^2 \times 758.5\ cm^2 \times 0.001/24\ h \times 6\ h/day}{15.5\ kg}$ = 0.031 mg /kg-bw per day	0.03 mg/kg-bw per day (as antimony trioxide)
Lying on a mattress cover (sweat mediated)	**Dermal – Infants (aged 0-6 months)** Use scenario and default values for antimony trioxide from CPSC (2006b). Scenario assumes that a person sleeping will roll and sweat during sleep, exposing almost the entire body surface area to the mattress cover and allowing antimony to migrate to skin. Bed sheets or clothing presents no barrier. To calculate the dermal exposure dose of antimony trioxide, the following assumptions are made: area density of antimony trioxide (S_a) is 3.24 µg/cm^2, area of body in contact (A_b) is 3460 cm^2 (Health Canada 1995), absorption rate (f_a) is 5% per day (or 0.002 h^{-1} = 0.05/24h), exposure duration (t) is 15 h (Howard and Wong 2001) and body weight (bw) is 7.5 kg (Health Canada 1998). Average exposure $= \frac{S_a \times A_b \times \mu_w \times f_E}{bw}$ $= \frac{3.24\ \mu g/cm^2 \times 3460\ cm^2 \times 0.002\ h^{-1} \times 15\ h}{7.5\ kg}$ = 44.8 µg/kg-bw per day	45 µg/kg-bw per day (as antimony trioxide)
Lying on a mattress cover (urine mediated)[2]	**Dermal – Child (aged 0.5-4 years)** Use scenario and default values for antimony trioxide from CPSC (2006b). Scenario assumes that the event of urinating in bed will occur intermittently and that a person sleeping will roll	2.2 µg/kg-bw per day (as antimony

Consumer product scenarios	Assumptions	Estimated exposure
	during sleep exposing almost the entire body surface area to the mattress cover. Bed sheets or clothing presents no barrier. To calculate the dermal exposure dose of antimony trioxide, the following assumptions are made: area density of antimony trioxide (S_a) is 3.24 µg/cm^2, area of body in contact (A_b) is 3980 cm^2 (Health Canada 1995), absorption rate (f_a) is 5% per day, exposure duration (t) is 13 h (Howard and Wong 2001), number of exposures per day (N) is 1, number of days on which exposure occurs during averaging period (N_A) is 3, averaging period (t_A) is 30 days and body weight (bw) is 15.5 kg (Health Canada 1998). Average exposure $= \frac{S_a \times A_b \times f_a \times t \times N \times N_A}{bw \times t_A}$ $= \frac{3.24\ \mu g/cm^2 \times 3980\ cm^2 \times 0.002\ h^{-1} \times 13\ h \times 1\ day^{-1} \times 3\ day}{15.5\ kg \times 30\ day}$ = 2.16 µg/kg-bw per day	trioxide)
Sitting on upholstery	**Inhalation – Particulate** Use scenario and default values for antimony trioxide from NRC (2000). Scenario assumes that a person spends a 25% of his/her life in a 30 m^3 room containing 30 m^2 of antimony trioxide-treated fabric. To calculate the average concentration of antimony trioxide attached to respirable particles in a room, the following assumptions are made: area density (S_a: application rate to the fabric or back-coating) is 2.5 mg/cm^2, units conversion factor of 1×10^3 µg/mg (CF_1), the area of antimony trioxide-treated fabric within the room (A_c) is 30 m^2, units conversion factor of 1×10^4 cm^2/m^2 (CF_2), the release rate of antimony trioxide (μ_r) is calculated using equation 5 in Chapter 3 from NRC (2000) and is 2.3×10^{-7}/day, room volume (V_r) is 30 m^3 and air exchange rate (R_v) is 0.25 air changes per hour (6 air changes/day) (NRC 2000). Average concentration $= \frac{S_a \times CF_1 \times A_c \times CF_2 \times \mu_r}{V_r \times R_v}$ $= 2.5\ mg/cm^2 \times 1000\ \mu g/mg \times 30\ m^2 \times 1\times10^4\ cm^2/m^2 \times 2.3\times 10^{-7}/day / 30\ m^3 \times 6$ air changes/day = 0.958 µg/m^3 × 0.25/day (length of time spent in room) = 0.24 µg/m^3 per day	0.24 µg/m^3 per day (as antimony trioxide)
Mouthing polyester fabric used to plush toys/ upholstery	**Oral – Infants (aged 0-6 months)** Use scenario and default values for antimony trioxide from NRC (2000). Scenario assumes that a child sucks on a 50 cm^2 piece of fabric 1 h daily for 2 years. To calculate the average oral dose rate of antimony trioxide, the following assumptions	0.7 µg/kg-bw per day (as antimony trioxide)

Consumer product scenarios	Assumptions	Estimated exposure
	are made: area density (S_a: application rate to the fabric or back-coating) is 2.5 mg/cm^2, the area of fabric wetted on each occasion (A_f) is 50 cm^2, fraction of time spent mouthing fabric (t_s) is 1 hr/day, fractional rate of extraction by saliva (μ_a) is 0.001 (NRC 2000) and body weight (bw) is 7.5 kg (Health Canada 1998). Assumes all antimony dissolved is consumed. Average exposure $= \frac{S_a \times A_c \times \mu_a \times t_c}{bw}$ $= \frac{2.5\ mg/cm^2 \times 50\ cm^2 \times 0.001 \times 0.0417}{7.5\ kg}$ = 0.000695 mg/kg-bw per day	
Mouthing mattress cover	**Oral – Infants (aged 0–6 months)** Default values from CPSC (2006a) for ingestion from mouthing. Scenario assumes migration from mattress cover to skin surface, to facilitate hand-to-mouth oral exposure in addition to licking lips and mouthing directly. Also assumes 100% oral absorption. To calculate the oral exposure, the following values are used: dermal application (L_D; from surface migration tests) is 3.24 µg/cm^2, mouthing area (A_m; addition of mouth area, lip area and half hand area) is 11 cm^2, extraction efficiency (E) is 0.038 (Environ 2003), number of exposures per day (N) is 1 and body weight (bw) is 7.5 kg. Average exposure $= \frac{L_D \times A_m \times E \times N}{bw}$ $= \frac{3.24 \mu g/cm^2 \times 11\ cm^2 \times 0.038 \times 1}{7.5\ kg}$ = 0.18 µg/kg-bw per day	0.2 µg/kg-bw per day (as antimony trioxide)

[1]All calculations shown in this appendix demonstrate the worst-case scenario. Other age groups were also considered where applicable.
[2]Based on the assumptions that infants wear diapers and that chronic bedwetting ceases in most children under the age of 11 (Howard and Wong 2001).

Appendix 6: Summary of health effects information for antimony trioxide

Endpoint	Lowest effect levels[1]/results
Experimental animals and *in vitro*	
Acute toxicity	**Oral LD_{50}** (rat) = >600 mg/kg-bw (Fleming 1982). **Other oral LD_{50}** (rat) = >16000–34600 mg/kg-bw (Smyth and Thompson 1945; Smyth and Carpenter 1948; Gross et al. 1955a; Ebbens 1972; Myers et al. 1983). **Inhalation LC_{50}** (rat) = >5200 mg/m^3 (nose-only) in a 4 h exposure (LPT and IAOIA 2006). **Dermal LD_{50}** (rabbit) = >2000 mg/kg-bw in a 24 h exposure (Ebbens 1972). **Other dermal LD_{50}** (rabbit) = >8000–8300 mg/kg-bw (Gross et al. 1955a; Myers et al. 1983).
Short-term repeated-dose toxicity	**Lowest oral LOEL:** 1000 mg/kg-bw per day was identified based on reduced body weight gain in Sprague-Dawley Crl:CD rats (six per sex per group) orally administered antimony trioxide at 0, 250, 500 or 1000 mg/kg-bw per day for 21 days. No clinical signs of toxicity were identified (Kirkland et al. 2007). [additional studies: Smyth and Thompson 1945; Smyth and Carpenter 1948; Fleming 1982; Hiraoka 1986] **Lowest inhalation LOEC:** 2.6 mg/m^3 was identified based on an increase in lung weights and pulmonary changes in female Sprague-Dawley rats (26 per group) treated with antimony trioxide at 0, 2.6, 4.4 or 6.3 mg/m^3 (nose-only, 99.87% purity) 6 h/day during gestation days 0-19. An increase in absolute and relative lung weights relative to brain weights was observed in all exposed groups. Pulmonary changes included accumulation of pigmented macrophages, scattered foci of acute inflammation (0/10, 7/10, 4/10, 6/10) and type II cell hyperplasia (0/10, 5/10, 4/10, 5/10) for 0, 2.6, 4.4 and 6.3 mg/m^3, respectively (MPI Research, Inc. 2003). [additional studies: NTP 2007a, b] No dermal studies were identified.
Subchronic toxicity	**Lowest oral LOEL:** 500 mg/kg-bw per day based on histopathological changes in liver and an increase in aspartate transaminase (serum glutamic oxaloacetic transaminase, or SGOT) activity in male Wistar rats (five per group) treated with antimony trioxide at 0%, 1% or 2% in their diet (corresponding to 0, 500, 1000 mg/kg-bw per day, respectively) for 24 weeks. Cloudy swelling of the hepatic cords was observed in 3/5 animals at 1% diet and 2/5 animals at 2% diet. A significant increase in SGOT was seen at 1% and 2% diet and a significant increase in alkaline phosphatase level was observed at 2% diet. A significant decrease in red blood cell count observed at 1% and 2% diet but was within the normal range. This study was not used in EURAR (2008) and NRC (2000) risk assessments because of the small group size and the fact that only the abstract and data tables were available in English (Sunagawa 1981).

Endpoint	Lowest effect levels[1]/results
	Other LOEL: 1686 mg/kg-bw per day (male) and 1879 mg/kg-bw per day (female) based on a 10% increase in relative and absolute liver weights in Wistar rats (12 per sex per group) administered antimony trioxide (99% purity) at 0, 84, 421 or 1686 mg/kg-bw per day (male) and 0, 97, 494 or 1879 mg/kg-bw per day (female) for 90 days. In males, a significant increase in serum triglycerides and red blood cell count and a significant decrease in alkaline phosphatase activity were observed at 1686 mg/kg-bw per day. In females, a significant increase in serum cholesterol and aspartate aminotransferase activity were observed at 1879 mg/kg-bw per day, and a dose-related decrease in alkaline phosphatase activity was observed at 494 and 1879 mg/kg-bw per day. Pituitary cysts at the top dose (4/12 in males, 3/13 in females) compared with controls (1/12 males and females) were considered a common spontaneous event in this strain of rat, and values were within the historical control range (Hext et al. 1999). [additional studies: Gross et al. 1955a] **Lowest inhalation LOEC:** 23.46 mg/m^3 based on an increase in lung weight and pulmonary changes in Fischer 344 rats (50 per sex per group) exposed (whole body) to antimony trioxide (99.68% purity) at 0, 0.25, 1.08, 4.92 or 23.46 mg/m^3 for 6 h/day, 5 days/week for 13 weeks followed by a recovery period for up to 27 weeks. Five rats per sex per group were terminated after 1, 2, 4, 8 and 13 weeks of exposure and at 1, 3, 9, 18 and 27 weeks post-exposure. Significant increases in mean absolute and relative lung weights were observed at 4.92 and 23.46 mg/m^3,with no recovery in the 23.46 mg/m^3 exposed group 27 weeks post-exposure. Alveolar macrophages, pneumonocyte hyperplasia and alveolar wall thickening were observed at 4.92 and 23.46 mg/m^3 during and after 27 weeks post-exposure. A significant increased incidence of corneal irregularities (30%) was observed in all exposed groups, with no recovery. Lung fibrosis and inflammatory changes were observed in all groups, including controls (Newton et al. 1994). [additional studies: Dernehl et al. 1945; Gross et al. 1952, 1955b; Cooper et al. 1968] No dermal studies were identified.
Chronic toxicity/ carcinogenicity	**Inhalation carcinogenicity in rats:** A total of 148 female Charles River Fischer rats were separated into three groups (48 control, 50 per exposed group) and exposed (whole body) to antimony trioxide (99.4% purity, 0.02% arsenic, and 0.2% lead) at 0, 1.9 or 5.0 mg/m^3 for 6 h/day, 5 days/week for 12 months followed by a 2- to 15-month recovery period. Exposure concentrations were reported as 1.6 and 4.2 mg/m^3 as antimony, respectively, and corresponding values as antimony trioxide were calculated. Two to eight animals per group were sacrificed pre-exposure, after 3, 6, 9 and 12 months of exposure and 1 year post-exposure. A significant increased incidence of lung tumours was observed at 5.0 mg/m^3 in the bronchioloalveolar region (scirrhous carcinomas: 0/28, 0/31, 15/34; squamous cell carcinomas: 0/28, 0/31, 2/34; bronchiolar adenomas: 1/28, 1/31,

Endpoint	Lowest effect levels[1]/results
	3/34; for 0, 1.9 or 5.0 mg/m^3, respectively). The LOEC was 1.9 mg/m^3 based on an increase in lung weight and pulmonary changes (lung fibrosis, adenomatous hyperplasia, pneumoncyte hyperplasia, inflammatory changes, multinucleated giant cells, pigmented macrophages) observed at 1.9 and 5.0 mg/m^3. An increase in focal subacute-chronic interstitial inflammation and granulomatous inflammation was observed at 5.0 mg/m^3 (Watt 1983). In the Watt (1983) studies, swine (Sinclair S-1 miniature, females, two per control, three per exposed group) were co-exposed to antimony trioxide with the rats in the same chamber for 12 months with no recovery period. The NOEC was 5.0 mg/m^3. **Other inhalation carcinogenicity in rats:** Wistar rats (90 per sex per group, 8 months old) were exposed (whole body) to 0 or time-weighted average (TWA) concentration of antimony trioxide (purity not specified; major contaminants: lead, 0.23%; tin, 0.21%; arsenic, 0.004%) of 45 mg/m^3, 7 h/day, 5 days/week, for 12 months, followed by a recovery period of up to 5 months. Serial sacrifices were performed in five rats per sex per group at 6, 9 and 12 months of exposure. A significant 27% increase in the incidence of lung tumours was observed in the exposed females 5 months post-exposure (19/70) compared with the control females (0/69). Lung neoplasms included 5/19 scirrhous carcinomas, 9/19 squamous cell carcinomas and 11/19 bronchoalveolar adenomas and carcinomas. No lung tumours were identified in control and exposed males. Non-neoplastic effects included pulmonary changes (interstitial fibrosis, alveolar-wall cell hypertrophy and hyperplasia and columnar cell metaplasia) in both sexes and a significant decrease in body weight in males (Groth et al. 1986). Fischer 344 (CDF F344 Crl Br) rats (65 per sex per group) were exposed (whole body) to antimony trioxide (99.68 ± 0.10% purity) at 0, 0.06, 0.51 or 4.50 mg/m^3, 6 h/day, 5 days/week, for 12 months, followed by a 12-month recovery period. Five animals per sex per group were terminated after 6 and 12 months of exposure and 6 months post-exposure. The incidence of neoplasms was within the historical range for controls. Two pulmonary carcinomas were identified in males (1/50 each from controls and at 4.50 mg/m^3) and 1/50 in females at 0.51 mg/m^3. The LOAEC was 4.50 mg/m^3 based on impaired pulmonary clearance and pulmonary changes. A significant reduction in the rate of pulmonary clearance by 80% and a minimal to moderate severity of chronic interstitial inflammation, interstitial fibrosis, lymphoid aggregates, granulomatous inflammation and bronchiolar/alveolar hyperplasia of the lungs were observed at 4.50 mg/m^3 in both sexes. A significant increase in incidence of cataract was observed in females (13%, 40%, 36%, 47%) but not in males (11%, 15%, 21%, 18%), for the 0, 0.06, 0.51 and 4.50 mg/m^3 exposure groups, respectively. No differences in body weight gain, absolute and relative lung weights, clinical chemistry, hematology or survival were observed. A high frequency of chronic interstitial inflammation in the lungs of all exposed groups, including controls, was noted (Newton et al. 1994). No oral or dermal studies were identified.

Endpoint	Lowest effect levels[1]/results
Reproductive toxicity	**Oral reproductive toxicity NOEL:** 1200 mg/kg-bw per day based on male rats (Crj:Wistar, 7–8 per group) and mice (Crj:CD-1, 8–10 per group) administered antimony trioxide (purity>99.999%) at 0, 12 or 1200 mg/kg-bw per day, 3 days/week (rats) or 5 days/week (mice) for 4 weeks. No effects on body weight gain, testes, auxiliary sex organ weights or sperm parameters (count, morphology and motility) were observed. The duration of treatment was less than the time for complete spermatogenesis (Omura et al. 2002). **Inhalation reproductive toxicity LOAEL:** 250 mg/m^3 based on a decrease in fertility in female rats (10 controls, 24 in treatment, strain not specified) exposed to antimony trioxide (purity not specified) at 0 or 250 mg/m^3, 4 h/day for 1.5–2 months premating and then during mating and gestation until 3–5 days prior to delivery. While all controls conceived, 16/24 animals in the exposed group conceived, which was considered significantly different by EURAR (2008). An average of 6.2 offspring per litter in the exposed group was observed compared with an average of 8.3 offspring per litter in the control group. No morphological changes were observed in the fetuses. Determination of pregnancy and data on the incidence of resorption or fetal deaths were not reported (Belyaeva 1967). No dermal studies were identified.
Developmental toxicity	**Lowest inhalation LOAEC for developmental toxicity in rats:** 0.082 mg/m^3 based on embryotoxic effects in female rats (albino, 6–7 per group) exposed to antimony trioxide at 0, 0.027, 0.082 or 0.27 mg/m^3, 24 h/day for 21 days throughout gestation. Reduction in maternal body weight gain and a significant decrease in placenta weights were observed at 0.27 mg/m^3. A dose-related decrease in overall embryo mortality and an increase in pre-implantation deaths were observed at 0.082 and 0.27 mg/m^3. An increase in post-implantation death was observed at 0.27 mg/m^3. Dose-dependent macroscopic embryotoxic effects were observed at 0.082 and 0.27 mg/m^3: haemorrhage into the fetal cerebral membrane and liver and enlargement of the kidney cavity and the cerebral ventricles. This study was not used in the EURAR (2008) and NRC (2000) risk assessments because of the lack of information on the purity of antimony trioxide and on how the antimony trioxide was generated and measured (Grin' et al. 1987). **Other inhalation NOEC for developmental toxicity in rats:** 6.3 mg/m^3 was identified in female Sprague-Dawley rats (26 per group) treated with antimony trioxide (99.87% purity) at 0, 2.6, 4.4 or 6.3 mg/m^3 (nose-only) 6 h/day during gestation days 0–19 with no significant effects on the number of corpora lutea, implantation loss, viable fetuses or resorption. No effects on fetal body weights, crown-rump distance, mean fetal sex ratio, skeletal information or ossifications were observed (MPI Research, Inc. 2003). No oral or dermal studies were identified.
Genotoxicity and related endpoints: *in vivo*	**Micronucleus induction:** **Negative:** CD-1 mice (five per sex per group) orally treated with 0 or 5000 mg/kg-bw (acute), 400, 477 or 1000 mg/kg-bw per day for 7, 14 or 21 days (Elliott et al. 1998). **Negative:** Sprague-Dawley Crl:CD (SD) rats (six per sex per group) orally

Endpoint	Lowest effect levels[1]/results
	treated with 0, 250, 500 or 1000 mg/kg-bw per day for 21 days (Kirkland et al. 2007). **Negative:** Wistar Han rats (five per sex per group) exposed to antimony trioxide at 0, 3, 10, 30 mg/m^3 6h/day, 5 days/week for 12 months (250 exposures in total) (NTP 2010). **Positive:** B6C3F1 mice (five per sex per group) exposed to antimony trioxide at 0, 3, 10, 30 mg/m^3 6h/day, 5 days/week for 12 months (250 exposures in total) (NTP 2010). **Chromosomal aberrations:** **Negative:** Sprague-Dawley Crl:CD rats (six per sex per group) orally administered antimony trioxide at 0, 250, 500 or 1000 mg/kg-bw per day for 21 days (Kirkland et al. 2007). **Negative:** Swiss mice (five per sex per group) orally treated with 0, 400, 677 or 1000 mg/kg-bw (acute) (Gurnani et al. 1992). **Positive:** Male swiss mice (five per group) orally administered antimony trioxide at 0, 400, 677 or 1000 mg/kg-bw per day for 7, 14 or 21 days. Dose-related increase frequency of chromosomal aberrations at 400 and 677 mg/kg-bw per day and unexplained lethality at 1000 mg/kg-bw per day on day 20 were observed. This study was not used in the EURAR (2008) risk assessment as the same data were published in another journal, with data discrepancy (Gurnani et al. 1992, 1993). **DNA damage:** **Negative:** Unscheduled DNA synthesis (liver) in male Alderley Park rats (five per group) orally treated with antimony trioxide at 0, 3200 or 6000 mg/kg-bw acutely (Elliott et al. 1998). **Sperm head abnormalities:** **Negative:** Male swiss mice (five per group) orally treated with 0, 400, 667 or 1000 mg/kg-bw (acute) or daily for 7, 14 or 21 days with unexplained lethality observed at 1000 mg/kg-bw on day 20 (Gurnani et al. 1992).
Genotoxicity and related endpoints: *in vitro*	**Mutagenicity:** **Negative:** Ames test**,** *Salmonella typhimurium* TA98, TA100, TA1535 and TA1537; *Escherichia coli* WP2PuvrA or WP2P in the presence or absence of metabolic activation (S9) (Kuroda et al. 1991; Elliott et al. 1998). **Negative:** Ames test**,** *S. typhimurium* TA98, TA100, TA1535, TA1537 and TA1538; *E. coli* B/r WP2 try$^-$, WP2 hcr$^-$ try$^-$ in the absence of metabolic activation (S9) (Kanematsu et al. 1980). **Positive:** DNA repair-Rec assay, *Bacillus subtilis* H17(Rec$^+$), M45(Rec$^-$) (Kanematsu et al. 1980; Kuroda et al. 1991). **Negative:** TK$^{+/-}$ mutation assay, mouse lymphoma L5178Y TK$^{+/-}$ cells with dose ranged 0–50 µg/mL without any significant cytotoxicity observed (Elliott et al. 1998). **Sister chromatid exchange:** **Positive:** V79 Chinese hamster cells with dose ranged 0–0.34 µg/mL with no cytotoxicity observed (Kuroda et al. 1991).

Endpoint	Lowest effect levels[1]/results
Sensitization	No sensitization responses observed when tested in guinea pigs (Ebbens 1972; LPT and IAOIA 2005a).
Irritation	**Skin irritation:** No irritation to intact skin and minimal irritation (erythema) in abraded skin when tested in rabbits (Ebbens 1972).
	Eye irritation: Mild irritation (Wil Research Laboratories 1979) and mild conjunctival redness (LPT and IAOIA 2005b) have been reported. Minimal to mild irritation in the washed eye and minimal irritation to extreme irritation 1h to 7 days post-exposure in the unwashed eye were reported (Ebbens 1972). No irritation was observed in some other studies (Smyth and Carpenter 1948; Gross et al. 1955a; Myers et al. 1983). All studies were tested in rabbits.
Humans studies	
Volunteer studies	Healthy volunteers (10 per sex) were patch tested with Dacron fibre containing 1% antimony trioxide on arms (men and women) or legs (women) where occluded areas were held with adhesive tape for 6 days. Two weeks after removal, new patches were applied for 48 hrs: no skin reaction was observed (Haskell Laboratory for Toxicology and Industrial Medicine 1970). Forty-six healthy men and 127 healthy women were patch tested with Dacron fibre containing 1% antimony trioxide and 6% octabromobiphenyl using the same method described above. No skin reaction was observed, except one subject developed papules along the edge of the patch area and under the adhesive tape (Haskell Laboratory for Toxicology and Industrial Medicine 1970). Thirty subjects were patch tested with antimony trioxide suspended in water or in soft yellow paraffin; no skin reaction was observed (Stevenson 1965). One hundred and ninety workers (enamellers and decorators) from five ceramics factories (119 female and 71 males) who were occupationally exposed to antimony trioxide underwent patch tests with 100% antimony trioxide applied to the healthy skin of the back for 2 days. One day later, 2/190 workers showed sensitization responses to antimony trioxide, compared with 0/92 controls. In these workers, dermatitis was present (22/190), and 44/190 claimed to have had lesions in the past due to occupational exposure, whereas no dermatitis was reported from the control group (Motolese et al. 1993).

Endpoint	Lowest effect levels[1]/results
Genotoxicity studies and related endpoints: *in vivo*	**Micronucleus induction:** **Negative:** Lymphocytes from male workers (6–17/group) exposed to antimony trioxide at 0.000 062 or 0.000 14 mg/m^3 compared with 23 healthy non-exposed men (Cavallo et al. 2002). **Sister chromatid exchange:** **Negative:** Lymphocytes from male workers (6–17/group) exposed to antimony trioxide at 0.000 062 or 0.000 14 mg/m^3 compared with 23 healthy non-exposed men (Cavallo et al. 2002). **Oxidative DNA damage:** **Positive:** Formamido-pyrimidine-glycosylase (fpg) enzyme-modified comet assay using lymphocytes from male workers (6–17/group) exposed to antimony trioxide at 0.000 062 or 0.000 14 mg/m^3 compared with 23 healthy non-exposed men (Cavallo et al. 2002).
Genotoxicity studies and related endpoints: *in vitro*	**Sister chromatid exchange:** **Positive:** Lymphocytes from healthy non-smoking donors (number not specified), dose range 0–1.5 µg/mL with cytotoxicity observed at 1.5 µg/mL (Gebel et al. 1997). **Chromosomal aberration:** **Positive:** Peripheral lymphocytes from two donors (no additional information about donors stated) treated with 0, 10, 50 or 100 µg/mL with or without S9 showed increased chromosomal aberration frequency in one donors at 100 µg/mL with and without S9 and dose-dependent increase in another donor at 50 or 100 µg/mL with S9 (Elliott et al. 1998).
Epidemiological studies	
Case studies	Seventy people in Newcastle, UK, accidentally drank lemonade contaminated with 0.015% antimony trioxide and 0.12% boron trioxide (amount drunk by each individual not specified, but one glass contained 43 mg antimony trioxide). Fifty-six people became ill and required hospitalization, with symptoms including burning stomach pain, colic, nausea and vomiting. They recovered in days (Dunn 1928). More than 150 children had acute poisoning from beverages contaminated with antimony at 30 mg/L (type of antimony compound not specified in Health Canada 1997). Symptoms included nausea, vomiting and diarrhea (Werrin 1963).
Cohort studies	In a cohort study in England, 1420 male antimony plant workers who worked between 1961 to 1992 with at least 3 months of employment were occupationally exposed to 60% antimony content (antimony trioxide, metallic antimony), up to 0.5% arsenic (metallic arsenic, arsenic trioxide) and traces of lead and polycyclic aromatic hydrocarbons. Workers on the site were subdivided into four occupational groups for comparison: antimony workers, maintenance workers, zircon sand workers and others (office workers and management staff). Antimony workers and maintenance workers who worked

Endpoint	Lowest effect levels[1]/results
	for more than 20 years had a significant increase in mortality from lung cancer compared with the regional mortality rate. Smoking was a potential confounding factor, as the prevalence of smoking among workers in 1961 was 72% (Jones 1994). In another cohort study in Texas, 1014 male antimony smelter workers (91.5% Spanish ancestry) employed between 1937 and 1971 for a minimum of 3 months were exposed to antimony ore (antimony trioxide, antimony sulphide) containing traces of arsenic and lead. Twelve air samples taken in 1975 and 50 samples taken in 1976 showed mean antimony levels of 0.551 and 0.747 mg/m^3, respectively (corresponding to antimony trioxide concentrations of 0.660 and 0.894 mg/m^3, respectively). Arsenic contaminant levels in 1975 and 1976 were 0.002 and 0.005 mg/m^3, respectively. A significant positive trend in mortality with increasing duration of employment was observed. A significant increase in mortality from lung cancer was observed compared with Texas ethnic-specified lung cancer death rates, and a significant increase in mortality from cancers of the liver, biliary tract and gall bladder was observed compared with both Texas ethnic-specified rates and the rates in US white males. The exposed group also showed a significant increase in incidence of ischemic heart disease compared with rates in Mexican-American (Schnorr et al. 1995). In another cohort study, 28 workers in an antimony sulphide smelter (25-61 years old) worked for 1-15 years converting stibnite into antimony trioxide in a rotational basis. The antimony concentration ranged from 0.081-138 mg/m^3 (corresponding to 0.097-165 mg/m^3 as antimony trioxide) at various locations. Chest x-rays revealed 3/13 cases of antimony pneumoconiosis and 5/13 suspicious cases. Fourteen subjects showed abnormal lung functions, but no consistent pattern of abnormalities was observed (Cooper et al. 1968). One hundred male workers in a sulphide smelter producing antimony trioxide and antimony metal in Newcastle, UK, were exposed to an average antimony trioxide concentration of 0.63-6.3 mg/m^3 (max 43.9 mg/m^3). Two men developed tuberculosis lesions, and one man had pneumoconiosis and chronic bronchitis with respiratory obstruction. Dermatitis with rashes (number not specified), especially in warm weather, was also reported (McCallum 1963). Seventy-eight male mining workers in an antimony smelter who worked for 2 weeks to 5 months were exposed to two working zones with average antimony concentrations of 10.07 and 11.81 mg/m^3, respectively (antimony trioxide levels of approximately 12.05 and 14.14 mg/m^3, respectively) (concentrations ranged from 0.40 to 70.7 mg/m^3 as antimony, approximately 0.48-84.6 mg/m^3 as antimony trioxide), average arsenic concentrations of 0.36 and 1.10 mg/m^3 respectively, and traces of selenium, lead and copper. Effects to both working zones combined on the respiratory tract included 11% laryngitis, 8% pharyngitis, 20% rhinitis, 8.5% septal perforation, 1.5% secondary sinusitis, 10% tracheitis, 7% bronchitis and 5.5% pneumonitis, effects on the gastrointestinal tract included 3% gastritis and 5.5% gastroenteritis; other effects included 20% dermatitis, 4% conjunctivitis and 1% neuritis. Some heavily exposed workers (number and concentration not specified) reported systemic toxicity with symptoms such as abdominal cramps, diarrhea,

Endpoint	Lowest effect levels[1]/results
	vomiting, dizziness, nerve tenderness and tingling, severe headache and prostration (Renes 1953).
	Three brazing rod manufacturing plant workers (28–33 years old) with no history of skin disease were exposed to antimony trioxide fumes and traces of antimony metal dust under hot conditions and developed dermatitis. The 8 h TWA concentration of antimony from one worker was 0.39 mg/m^3 (approximately 0.47 mg/m^3 as antimony trioxide) (White et al. 1993). Women (number not specified) occupationally exposed to dust containing antimony trioxide, metallic antimony and antimony pentasulphide (concentration not specified) over a period of 2 years between 1962 and 1964 had 12–16 times higher antimony levels in the blood compared with controls. The exposed group had a higher incidence of various sexual disturbances compared with controls (77.5% vs. 56% control). Effects included disturbances of the menstrual cycle, infection of sex organs, incidence of late spontaneous abortions and incidence of premature births. Babies born from the exposed women had normal body weights at birth but lower weights after 3–12 months (Belyaeva 1967). [additional studies: Karajovic 1957; Klucik et al. 1962; Stevenson 1965; McCallum 1967; Potkonjak and Pavlovich 1983]

[1] LC_{50}, median lethal concentration; LD_{50}, median lethal dose; LOAEC, lowest-observed-adverse-effect concentration; LOEC, lowest-observed-effect concentration; LOEL, lowest-observed-effect level; NOEC, no-observed-effect concentration.