

Draft Screening Assessment

Hydrogen Sulfide (H₂S), Sodium Sulfide (Na(SH)) and Sodium Sulfide (Na₂S)

Chemical Abstracts Service Registry Numbers

7783-06-4

16721-80-5

1313-82-2

**Environment and Climate Change Canada
Health Canada**

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Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA), the Minister of the Environment and the Minister of Health have conducted a screening assessment of hydrogen sulfide (H₂S) (Chemical Abstracts Service Registry Number [CAS RN¹] 7783-06-4), sodium sulfide (Na(SH)), referred to as sodium bisulfide in this assessment (CAS RN 16721-80-5), and sodium sulfide (Na₂S) (CAS RN 1313-82-2). These substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA.

Hydrogen sulfide is a naturally occurring inorganic gas produced from the anaerobic degradation of organic matter and is therefore widely present in anaerobic sediments and water and in biological wastes. It is also found naturally in crude oil petroleum, natural gas, volcanic gases and hot springs and is released from these natural sources primarily to air and to water under specific environmental conditions. It can also be released as a result of anthropogenic activities. Industrial operations that release hydrogen sulfide in Canada include oil and gas facilities, kraft pulp and paper mills, wastewater treatment systems, mining production, and intensive livestock operations.

Sodium bisulfide is reported to be used in Canada as a chemical intermediate for commercial uses in dyes in textiles, paints and coatings, non-pesticidal agricultural products, and building and construction materials (wood and engineered wood). Sodium sulfide is used in Canada in pulp and paper processing, wastewater treatment, mining and smelting, and in food packaging with no direct contact with food. These two substances will dissociate to form bisulfide and sulfide anions and hydrogen sulfide if released to water. Considering that the likely medium of release for these substances is the air and aquatic environments, the environmental assessment is focused on hydrogen sulfide. Similarly, if exposure of the general Canadian population to undissociated sodium bisulfide or sodium sulfide were to occur, either salt would rapidly and completely hydrolyze in bodily fluids to result in the formation of hydrogen sulfide. No specific additional hazard is associated with either salt beyond that associated with hydrogen sulfide. As such, the human health risk characterization is focused on exposure to hydrogen sulfide.

According to an extensive database of measurements in Canada, hydrogen sulfide has been reported in air, surface water, and wastewater effluents in the vicinity of pulp and paper operations, oil and gas facilities, wastewater treatment systems, and livestock operations.

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Hydrogen sulfide has the potential to harm both aquatic organisms and terrestrial plants when exposed at low concentrations. In the case of plants however, low concentrations can also have stimulatory effects.

A risk quotient analysis determined that current hydrogen sulfide concentrations in Canadian air near anthropogenic sources are unlikely to be high enough to cause adverse effects to terrestrial wildlife (mammals or plants) and that concentrations in surface water near such sources are also unlikely to cause adverse effects to aquatic organisms.

Considering all available lines of evidence presented in this draft screening assessment, there is low risk of harm to organisms or the broader integrity of the environment from these substances. It is therefore proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide do not meet the criteria under paragraphs 64(a) or (b) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that 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.

Inhalation is expected to be the predominant route of general population exposure to hydrogen sulfide, and the health effects assessment focused on data examining effects by this route. No genotoxicity or carcinogenicity classifications by other national or international regulatory agencies were identified. Available information does not indicate that hydrogen sulfide is genotoxic or carcinogenic. The upper-bounding concentrations of hydrogen sulfide in ambient air are based on a review of the available Canadian monitoring data. The range of concentrations of 1–31 ppb (1.4–43.4 $\mu\text{g}/\text{m}^3$) is used in the risk characterization. The lowest value of this range represents the overall average concentration measured in an urban area presumed to be away from major anthropogenic sources; the highest value of the range is the highest of all 99th percentile concentrations derived from measurements near point sources in Canada. Margins between upper-bounding concentrations of hydrogen sulfide in ambient air and levels associated with critical health effects (ocular, respiratory, neurological effects) are considered to be adequate to address uncertainties in the health effects and exposure databases. In occupational settings, severe health effects have been reported due to accidental acute exposure to high levels of hydrogen sulfide. These levels, specific to industrial settings, are several orders of magnitude higher than concentrations encountered in a community setting and are not considered relevant for general population risk characterization. Further, requirements are typically in place in occupational settings for the protection of workers, which may include measures to prevent accidental releases of hydrogen sulfide and/or surveillance of air levels to ensure they are below occupational exposure limits. Available toxicity studies conducted specifically with sodium sulfide and sodium bisulfide are summarized in the health effects section.

Based on the information presented in this draft screening assessment, it is proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide do not meet the criteria under paragraph 64(c) of CEPA as they are 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 proposed to conclude that hydrogen sulfide (H_2S), sodium bisulfide ($\text{Na}(\text{SH})$) and sodium sulfide (Na_2S) do not meet any of the criteria set out in section 64 of CEPA.

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1. Introduction

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health have conducted a screening assessment of hydrogen sulfide (Chemical Abstracts Service Registry Number (CAS RN²) 7783-06-4) to determine whether this substance presents or may present a risk to the environment or to human health. A screening assessment of hydrogen sulfide was undertaken because it met the criteria for persistence potential and inherent toxicity to non-human organisms and was identified as a substance to be prioritized on the basis of greatest potential for human exposure. Hydrogen sulfide was identified as a priority for assessment as it met categorization criteria under subsection 73(1) of CEPA (ECCC, HC [modified 2007]).

Two precursors of hydrogen sulfide, sodium bisulfide (CAS RN 16721-80-5) and sodium sulfide (CAS RN 1313-82-2), were also identified as meeting categorization criteria under subsection 73(1) of CEPA and are included in this screening assessment.

This draft screening assessment includes consideration of information on chemical properties, hazards, uses and exposure. Data relevant to the screening assessment of these substances were identified in original literature and review and assessment documents and from recent literature searches was carried out up to May 2017 or ecological effects and up to May 2017 for human health effects and exposure. In addition, an industry survey was conducted in 2000 through a *Canada Gazette* notice issued under the authority of section 71 of CEPA (Canada 2001).

Evaluation of risk to human health involves consideration of data relevant to estimation of 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 conservative estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context

This draft screening assessment was prepared by staff in the Existing Substances Program of Health Canada and Environment and Climate Change Canada in collaboration with the Air Health Science Division at Health 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,

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including Dr. Chris Bevan (CJB Consulting), Dr. John Christopher (California Department of Toxic Substances Control), Dr. Michael Jayjock (The LifeLine Group) and Dr. Pam Williams (E-Risk Sciences). Although external comments were taken into consideration, the final content and outcome of the draft screening assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

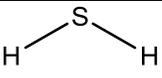
This draft screening assessment focuses on information critical to determining whether substances meet the criteria as set out in section 64 of CEPA, by examining scientific information and incorporating a weight-of-evidence approach and precaution.³ The draft screening assessment presents the critical information and considerations upon which the proposed conclusion is made.

2. Identity of Substances

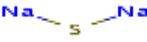
This screening assessment focuses on the substance hydrogen sulfide (CAS RN 7783-06-4) and two of its precursors, sodium bisulfide (CAS RN 16721-80-5) and sodium sulfide (CAS RN 1313-82-2). For this assessment, hydrogen sulfide will also be referred to as H₂S.

Information relevant to the identity of the substances is presented in Table 2-1.

Table 2-1. Substance identity

CAS RN	DSL name (Other names)	Chemical formula	Chemical structure	Molecular mass g/mol
7783-06-4	Hydrogen sulfide (H ₂ S) (Dihydrogen sulfide)	H ₂ S		34.08
16721-80-5	Sodium sulfide (NaSH) (Sodium bisulfide)	Na(SH)		56.06

³A determination of whether one or more of the criteria of section 64 of CEPA 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 products used by consumers. A conclusion under CEPA is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Hazardous Products Regulations*, which are part of the regulatory framework for the Workplace Hazardous Materials Information System for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA does not preclude actions being taken under other sections of CEPA or other Acts.

1313-82-2	Sodium sulfide (Na ₂ S) (Disodium monosulfide)	Na ₂ S		78.046
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Source: NCI (2007); NICNAS 2016.

3. Physical and Chemical Properties

Hydrogen sulfide is a colourless inorganic gas that has a characteristic rotten egg odour (NRCC 1981; Budavari 1996). It is soluble in water as well as in certain polar organic solvents (Budavari 1996). It has a vapour density of 1.19 (air has a vapour density of 1.0), meaning that it will sink in air to ground level under quiet atmospheric conditions or when present in high concentrations.

Pure sodium bisulfide and sodium sulfide are both white, crystalline solids which are readily soluble in water. When exposed to air, both substances also undergo autoxidation and gradually form polysulfur, thiosulfate, and sulfate. Sodium bisulfide also absorbs carbon dioxide, forming sodium carbonate (Bush 2000).

Concentrations in air throughout the assessment are typically presented in units of both ppm or ppb and mg/m³ or µg/m³. When converting between units, a ratio of 1.4 µg/m³ = 1.0 ppb was used. These calculations were done for the purposes of this assessment unless otherwise stated.

Table 3-1. Physical and chemical properties of hydrogen sulfide

Property	Type	Value	Temperature (°C)	Reference
Melting point (°C)	Experimental	-85.49		Budavari 1996
Boiling point (°C)	Experimental	-60.33		Budavari 1996
Vapour density	Experimental	1.19	15	HSDB 2003
Vapour pressure (kPa)	Experimental	102.9	-60	Bush 1980
		562	-20	Bush 1980

Property	Type	Value	Temperature (°C)	Reference
		1049	0	Bush 1980
		1814	20	HSDB 2003
		2026	25.5	Weast 1982
Henry's Law constant (Pa·m ³ /mol)	Experimental	993 (0.0098 atm·m ³ /mol)	25	HSDB 2003
Water solubility (mg/L)	Experimental	7100 5380 3980 5000	0 10 20 20	Bush 1980
pK _a (dimensionless)	Experimental	7.04 pK _a (1) (H ₂ S ↔ HS ⁻) 11.96 pK _a (2) (HS ⁻ ↔ S ²⁻)		ATSDR 2006
Water/air conversion factors		1 ppm = 1.40 mg/m ³ 1 mg/m ³ = 0.71 ppm	20 (101.3 kPa)	ATSDR 2006

Abbreviations: pK_a = acid dissociation constant.

Table 3-2. Physical and chemical properties of sodium bisulfide and sodium sulfide

Property	Sodium bisulfide (NaSH)	Sodium sulfide (Na ₂ S)
Melting point (°C)	350 °C @ 99.7 kPa [a]	950 °C [a]
Boiling point (°C)	123-[b]	NA [c]
Density	1.79 g/cm ³ [c]	1.856 g/cm ³ [a]
Vapour pressure	2266 Pa (17 mm Hg) [b]	NA
log K _{ow}	-3.5 [d]	-3.5 [d]

Water solubility	548 g/L @ 20 °C and pH 11.8[a]	186 000 mg/L (20 °C) [e]
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^a Alfa Aesar

^b TDC MSDS 2004

^c ECHA 2009

^d ILO 2012

^e Chemical Book 2016,

4. Sources

Hydrogen sulfide

It has been estimated that natural sources account for 60 to 90% of the hydrogen sulfide in the atmosphere globally (US EPA 1993; Watts 2000). Hydrogen sulfide is produced naturally through non-specific and anaerobic bacterial reduction of sulfates and sulfur-containing organic compounds, such as proteins and amino acids (Hill 1973). It is also produced endogenously in humans and other mammals as part of normal biological function by the brain, liver, heart and gastrointestinal tract (Kimura 2002; Kamoun 2004; Linden et al. 2010). It is found naturally in crude petroleum, natural gas, volcanic gases and hot springs and is released from these natural sources primarily as a gas. Hydrogen sulfide is found naturally in a variety of environmental media—including anaerobic aquatic sediments and groundwater—owing primarily to the bacterial reduction of other forms of sulfur.

Hydrogen sulfide is also emitted by some plant species as a by-product of sulfite metabolism (Takemoto et al. 1986). Some higher plants produce and release hydrogen sulfide by means of an enzymatic reaction with carbonyl sulfide (Watts 2000). Estimates of the terrestrial emission rate of hydrogen sulfide—including releases from tropical forests and other vegetation sources—can vary widely. Watts (2000), for example, estimated this value to be 0.8 million tonnes of sulfur per year, much lower than the 10 million tonne upper-bound value of Andreae and Jaeschke (1992). Estimates of the emission rate of hydrogen sulfide from oceans, including salt marshes and estuaries, are less variable, ranging from < 1.5 to 2.3 million tonnes of sulfur per year (Watts 2000; Andreae and Jaeschke 1992). Annual global releases of hydrogen sulfide from all natural sources have been estimated by Watts (2000) to be about 4.4 million tonnes, a value that is lower than some previous estimates (e.g., 4.7 to 13 million tonnes; Andreae and Jaeschke 1992).

Hydrogen sulfide can also be released as a result of agricultural activities or industrial processes. These include releases as a by-product from petroleum sector activities (Environment Canada 2004a) since natural gas and gases associated with crude oil contain hydrogen sulfide at levels varying from trace amounts to 70–80% by volume (Pouliquen et al. 1989). Hydrogen sulfide can also be generated during hydraulic fracturing (Marriott et al. 2016; Kahrilas et al. 2015). Other anthropogenic sources include liquid manure storage (Blunden and Aneja 2008; Kim et al. 2008), kraft pulp and paper mills (Teschke et al. 1999; IPCS 2003; ATSDR 2006; Janssen et al. 2009), landfills (IPCS 2003; ATSDR 2006; Kim 2006), decomposition of organic waste from

wastewater treatment (Muezzinoglu 2003), and other industrial processes, such as metal refining (OMOE 2007; NPRI 2013). Releases to the environment are primarily in the form of emissions to ambient air, although sulfides (including hydrogen sulfide) may also be released to water under specific environmental conditions.

According to information submitted in response to a survey conducted under section 71 of CEPA (Environment Canada 2004a), most manufacturing of hydrogen sulfide in Canada occurs as a by-product of the purification of “sour” natural gas and the processing, upgrading, and/or refining of bitumen and “sour” crude oil, as well as through incidental production in the pulp and paper sector (kraft mills). As the companies that submitted information under section 71 were not required to indicate whether the hydrogen sulfide was manufactured intentionally or incidentally, the term “manufacturing” here includes incidental generation of the gas. On the basis of the results of the section 71 survey, the total amount of hydrogen sulfide manufactured in Canada in 2000 was estimated at approximately 8.67 million tonnes (Environment Canada 2004a).

A study of various sulfur compounds in cigarette smoke indicated that, on average, cigarettes produce 31.6 µg of hydrogen sulfide per cigarette (Dong and DeBusk 2010). Exposure to hydrogen sulfide from tobacco smoke is therefore expected to be low.

Sodium bisulfide and sodium sulfide

In response to a survey issued under section 71 of CEPA, a total quantity of 1 000 000 to 10 000 000 kg of sodium bisulfide was reported to have been imported into Canada in the 2011 calendar year. In the same survey, a voluntary DSL IU2 submission was received from a single submitter indicating that the substance was manufactured in an unknown quantity. No consumer products were reported in Canada in that survey (Canada 2012a, 2014). Uses reported in the survey were commercial only and included non-pesticidal agricultural substances, paints and coatings, dyes, intermediates, and building and construction materials (wood and engineered wood). In the 2015 calendar year, a total quantity of 9 217 213 kg of sodium sulfides (which may include sodium bisulfide, sodium sulfide and other sulfides of sodium) was imported into Canada (StatsCan 2015).

Information regarding the import of sodium sulfide was acquired through data obtained from the Canada Border Services Agency (CBSA) (CBSA 2013). In the years 2010 to 2013, a total quantity of 100 000 to 1 000 000 kg of sodium sulfide was imported into Canada (CBSA 2013).

Over 10 000 tonnes of sodium sulfide and sodium bisulfide are reported to be manufactured in or imported into the European Economic Area per year (ECHA 2016).

5. Uses

Hydrogen sulfide

Major uses of hydrogen sulfide internationally involve the manufacturing of elemental sulphur and sulfuric acid (ATSDR 2006). Hydrogen sulfide can also be used as a chemical intermediate in the production of dyes, rubber chemicals, pesticides, polymers, plastic additives, leather and pharmaceuticals. Other reported uses include production of heavy water in the nuclear industry, as an analytical reagent, as a disinfectant in agriculture and as an additive in extreme-pressure lubricants and cutting oils (ATSDR 2006).

No consumer product uses were reported for hydrogen sulfide in the section 71 survey (Environment Canada 2004a).

Over the past decade, endogenously produced hydrogen sulfide has been identified as playing a pivotal role in several physiological and pathophysiological processes, such as neuron synaptic potentiation, vasorelaxation and anti-inflammatory conditions, cardiac contractility, cardioprotection (Mancardi et al. 2009). As a result, several drug therapies have emerged to exploit the benefits of hydrogen sulfide by coupling a hydrogen sulfide-releasing moiety to a conventional drug (Rossoni et al. 2010). Thus, for a particular cohort of the general population, these products may be a future source of exposure coupled with therapeutic benefits (Fiorucci and Santucci 2011).

Sodium bisulfide (NaSH) and sodium sulfide (Na₂S)

Sodium bisulfide is used as a primary reagent for copper-molybdenum mineral separation in Canada. Although hydrogen sulfide gas formation has been reported at a copper-molybdenum plant in British Columbia, technology, scrubbers and exhaust ventilation have eliminated detectable releases of hydrogen sulfide from the facility (Chessor and Johannsen 2006).

In Canada, sodium bisulfide is not used in cosmetics (personal communication, email from the Consumer Product Safety Directorate, Health Canada, to the Existing Substances Risk Assessment Bureau, Health Canada, dated April 18, 2016; unreferenced), food additives (Health Canada [modified 2013]), pesticides (PMRA 2010; PMRA [modified 2013]), drugs (DPD [modified 2015]) or natural health products (LNHPD [modified 2014]; NHPID [modified 2015]). It is not used in food packaging or incidental additives for food (personal communication, email from Risk Management Bureau, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada, dated May 18, 2016; unreferenced).

Sodium bisulfide is reported by the European Union to be used for the manufacture of chemicals, textiles, leather or fur, pulp, paper and paper products, metals, rubber products, and plastic products (ECHA 2016). Over 10 000 tonnes of this substance is manufactured in or imported into the European Economic Area per year.

Sodium sulfide, in combination with sodium hydroxide (NaOH), is used in the production of pulp (Tran and Vakkilainen 2008). Sodium sulfide may be a component of pulping liquors, depending on the process and recovery process. White liquor is used in the first stage of the kraft process, black liquor is a waste product from this process, and green liquor is the dissolved smelt of sodium carbonate, sodium sulfide and other compounds from the recovery boiler in the kraft process.

Under section B.14.062 [S] of the *Food and Drug Regulations*, sodium sulfide is permitted in the manufacture of gelatin or edible gelatin from processing skin, ligaments or bones of animals (Canada [1978]). There is currently no such use of sodium sulfide reported in Canada (personal communication, email from Food Directorate, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada, dated May 18, 2016; unreferenced).

Sodium sulfide is included on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances may contravene either the general prohibition found in section 16 of the *Food and Drugs Act* (FDA) or one or more provisions of the *Cosmetic Regulations*. Section 16 of the FDA states that “No person shall sell any cosmetic that has in or on it any substance that may cause injury to the health of the user.” In addition, the Hotlist includes certain substances that may make it unlikely for a product to be classified as a cosmetic under the FDA (Health Canada [modified 2015]). Under the entry of alkali sulfides (for lithium sulfide, potassium sulfide and sodium sulfide), these three substances are restricted to a maximum permitted concentration of 2% as sulfur in hair removal (depilatory) products (Health Canada [modified 2015]). There are no current cosmetics containing sodium sulfide as an ingredient in Canada (personal communication, email from Consumer Product Safety Directorate, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada, dated April 18, 2016; unreferenced). No consumer products containing sodium bisulfide were found in Canada.

In Canada, sodium sulfide has not been identified as being used in food additives (Health Canada [modified 2013]), pesticides (PMRA 2010; PMRA [modified 2013]), drugs (DPD [modified 2015]) or natural health products (LNHPD [modified 2014]; NHPID [modified 2015]). Sodium sulfide is used in food packaging (with no potential for direct food contact) and is not in incidental additives for food (personal communication, email from Risk Management Bureau, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada, dated May 18, 2016; unreferenced). Sodium sulfide has been identified as being used as a reaction-control agent in the production of synthetic polymers for use in chewing gum base. Dietary exposure to residues of sodium sulfide if present in a finished chewing gum product sold in Canada is expected to be minimal (personal communication, email from Food Directorate, Health Canada, to Existing Substances Risk Assessment Bureau, Health Canada, dated June 16, 2016; unreferenced). No consumer products containing sodium sulfide were found in Canada.

In terms of use information from outside of Canada, sodium bisulfide is used as a flotation agent in mining and metal extraction, in kraft pulping, in dyestuff processing, in hair removal from hides, in rayon and cellophane desulfurizing, in bleaching, in the textile industry, and in photography engraving and lithography, and as an intermediate in the manufacture of other chemicals (SDS 2013; NICNAS 2006, 2016). Sodium sulfide is used as an active constituent in pesticides and veterinary medicines, in the treatment of hides for manufacture of gelatin and collagen, in depilatory personal care products, in the textile industry, in photography engraving and lithography, and in heavy metal removal for wastewater treatment, and as an intermediate in the manufacture of other chemicals (NICNAS 2006, 2016). All of these international uses are industrial or commercial only (often site-limited), except for use in depilatory personal care products (NICNAS 2016). It is also used in the production of rubber chemicals, sulfur dyes and other chemical compounds.

The substance is also used in a number of products, such as pH regulators, water treatment products and water treatment chemicals, and in the manufacture of intermediates.

Sodium sulfide is used in the formulation of mixtures and/or re-packaging and municipal supply (e.g., electricity, steam, gas, and water) and wastewater treatment, including the manufacture of chemicals, textile, leather or fur, pulp, paper and paper products, rubber products, plastic products, and metals (ECHA 2016).

6. Releases to the Environment

Many industrial sectors in Canada, including the oil and gas sector, pulp and paper sector (kraft mills), livestock operations, non-metallic mineral products industries, primary metal industries and other manufacturing industries, and waste and wastewater sector, release quantities of hydrogen sulfide, mostly to air but also to water. According to information submitted under section 71 of CEPA (Environment Canada 2004a), the pattern of release of hydrogen sulfide in Canada is similar to that reported elsewhere (IPCS 1981; Budavari 1996; Canada 2001; IPCS 2003; US EPA 2003).

Data reported to the National Pollutant Release Inventory (NPRI) indicate that the three most significant industries contributing to hydrogen sulfide air emissions in Canada have been the oil and gas, pulp and paper and iron and steel sectors. However, over recent years, emission reductions from the pulp and paper and iron and steel sectors have made the oil and gas sector a relatively larger contributor to the total emissions (NPRI 2016).

According to the NPRI (2016), 146 facilities reported on-site releases of hydrogen sulfide totalling 2154 tonnes in 2014. Of those, 2060 tonnes was released to air, 94 tonnes was released to water and 0.012 tonnes were released to land. The total amounts of hydrogen sulfide disposed of at on-site and off-site locations in 2014 were 132 014 and 32 692 tonnes, respectively. All of the hydrogen sulfide that was reported to the NPRI as disposed of on-site was injected underground. Underground injection is a regulated waste disposal method in which materials are injected into deep underground wells. A total of 226 tonnes was sent to off-site recycling in 2014.

Industrial releases of hydrogen sulfide in Canada have generally decreased since 2000 (when 6301 tonnes were released), although reported releases to water have increased. This is the result of an increased number of reporters rather than an increase in release quantities from individual reporters (NPRI 2013).

The NPRI values reported here likely underestimate total releases from anthropogenic point sources in Canada since some significant sources are not captured by the NPRI (including intensive livestock operations and most smaller upstream oil and gas facilities).

6.1 Sodium Bisulfide and Sodium Sulfide

Considering that under typical surface water conditions (pH ~7) sodium bisulfide and sodium sulfide are expected to dissociate into hydrogen bisulfide anion (HS⁻) and hydrogen sulfide, the focus of release is on hydrogen sulfide. However, the exact formation quantity of hydrogen sulfide generated from the two precursors is unknown and is subject to local conditions. Under very acidic conditions (pH 1.5-3.5), the formation of hydrogen sulfide will predominate. Release of sodium bisulfide to the environment is likely to occur from industrial use as a processing aid, manufacturing of the substance itself, as an intermediate step in further manufacturing of another substance (use of intermediates), in the manufacture of thermoplastics, as a processing aid and in the formulation of mixtures (ECHA 2016).

Considering current uses, the aquatic environment is the likely medium for release of sodium bisulfide and sodium sulfide. Commercial activities involving precursor substances, including sodium bisulfide and sodium sulfide, may also form hydrogen sulfide. In the case of the two sodium salts, the anions of their dissociation, namely the bisulfide and sulfide, can be in equilibrium with hydrogen sulfide and thus indirectly result in its formation.

Sodium bisulfide has a high vapour pressure and is expected to react with oxygen and carbon dioxide gas in air to form sodium thiosulfate, sodium sulfite and sodium carbonate.

6.2 Oil and Gas

The amounts of hydrogen sulfide released to air by the Canadian oil and gas sector as reported to the NPRI for 2009 and 2014 were 1049 and 1140 tonnes, respectively (NPRI 2014). Included in this category are upstream (i.e., exploration and production) activities related to oil and gas, oil sands and heavy oil, as well as oil and gas storage and pipeline transport, and coal manufacturing activities. Increasing trends in releases of hydrogen sulfide are attributed to growth in oil and gas production (Burstyn et al. 2012).

The vast majority of hydrogen sulfide produced in oil sands processes and from the high-concentration sour gas fields of British Columbia, Alberta and Saskatchewan is burned in flare stacks, burned as a fuel, pumped back underground or turned into elemental sulfur and sold as a product. An inventory of greenhouse gases, criteria air contaminants and hydrogen sulfide emissions conducted for the Canadian upstream oil and gas sector for 2000 identified roughly 10 000 tonnes of hydrogen sulfide released (Clearstone 2004). This value, which represents about 250 facilities in Alberta, is much larger than the approximately 1500 tonnes reported to the NPRI (representing only 146 reporting facilities) by the upstream oil and gas sector in 2000 (Clearstone 2004). Most releases from these sources are not reported to the NPRI as these operations typically do not meet the reporting criteria. NPRI reporting depends on a number of criteria, including number of employees, type of facility and quantity of substance that is manufactured, processed or otherwise used (NPRI 2016). An update to the 2005 inventory of air contaminants was conducted in 2011 (Clearstone 2014). The amount of direct hydrogen sulfide emissions in 2011 was estimated to be 3700 tonnes (uncertainty of -10.0 to +28.8 %). Approximately 80% of these emissions were from natural gas production and processing, with the remainder from oil production. Although the release of hydrogen sulfide to the atmosphere is regulated (Clearstone 2014), some emissions occur.

Under Alberta's *Oil Sands Conservation Act*, operators may not release gas containing hydrogen sulfide directly to the atmosphere. Gas from various sources, such as flare lines, relief valves and wells, must be captured and incinerated such that essentially all of the hydrogen sulfide is converted to sulfur dioxide prior to release. Operators must also have emergency response plans in place to effectively deal with any uncontrolled releases of H₂S. The decrease in emissions between 2005 and 2011 is reported to be primarily due to industry reductions of fugitive emissions and compressor seal venting. In 2011, the majority of hydrogen sulfide releases reported were due to fugitive equipment leaks (48%), incomplete combustion of fuels and waste gas streams containing hydrogen sulfide (31%), evaporation losses during product storage and handling (12%) and venting of waste gas streams containing low concentrations of hydrogen sulfide (e.g., less than 10 ppm) (9%). The decrease in emissions between 6000 tonnes in 2005 and 3700 tonnes in 2011 is reported to be primarily due to the implementation of best management practices specified in the Alberta Energy Regulator

(AER) Directive 060, which sets out requirements for flaring, incinerating, and venting in Alberta at all upstream petroleum industry wells and facilities.

A significant issue associated with the disposal of unwanted hydrogen sulfide from oil and gas facilities is the efficiencies of the flares. Clearstone (2004) estimated that in 2000, 898 tonnes of hydrogen sulfide were released as result of flaring during gas processing. Estimates of flare efficiency vary, ranging from 20% to 99% (University of Alberta 2007). In Alberta, the combined volume of flared and vented solution gas is reported to have decreased by 13% from 2008 to 2009 (ERCB 2010). Solution gases are released when crude oil is produced to the surface. It is not currently possible to reliably estimate the amounts of hydrogen sulfide being flared and vented at solution gas batteries in Alberta (Johnson et al. 2011). A qualitative estimate of the proportions of sweet (less than 10 ppm hydrogen sulfide) and sour (>10 ppm hydrogen sulfide) battery sites and gas volumes were estimated using Alberta Energy Utilities Board (AEUB) site inspection data. On the basis of these data, and correlating with the volumes of gas flared at individual sites, it is estimated that 36% of the gas flared and vented in the province is sour. The petroleum industry in Alberta achieved a 95.6% solution gas conservation rate in 2014, compared with 95.3% in 2013. However, since efficiencies vary considerably, the solution gas conservation rate may be considered essentially unchanged from one year to the next. Gas conservation is the recovery of solution gas to use as fuel for production facilities, to sell, to inject for enhanced recovery from oil or condensate pools, or to generate power, among other uses (AER 2016).

No releases of hydrogen sulfide to surface water or land have been reported to the NPRI by any of the oil sands mine operators. It is present in crude oil and may be generated in the process-affected water during open-pit mining and hot water extraction, and then transferred to tailings ponds. Hydrogen sulfide may also be produced in tailings ponds from anaerobic bacterial degradation of organic compounds or by reduction of sulfate ions added to tailings to promote their consolidation (Holowenko et al. 2000). A no-discharge policy exists for the process-affected water from open-pit mining; however, there may still be the potential for hydrogen sulfide in process-affected water to enter waterways through underground seepage from tailings ponds to groundwater aquifers that are connected to surface waters (Timoney and Lee 2009). There is thus the potential for indirect release of contaminants (including hydrogen sulfide) to northern Alberta rivers (RSC 2010).

Several oil sands facilities have reported hydrogen sulfide releases to air from fugitive and other non-point source releases to NPRI (2016). One mine operator has reported that a small portion of hydrogen sulfide in the form of total reduced sulfur (TRS) emissions to air will be emitted from tailings ponds (RSC 2010).

6.3 Pulp and Paper

Hydrogen sulfide may be released to air and water from pulp and paper mills that use a kraft pulping process. Hydrogen sulfide is measured and regulated by the provinces

(Alberta, British Columbia, New Brunswick, Nova Scotia, Ontario and Quebec) as total reduced sulfur (TRS). TRS may include hydrogen sulfide, methyl mercaptan, dimethyl sulphide, dimethyl disulphide, carbon disulfide, carbonyl sulfide, and other organic compounds containing sulfur in a reduced state.

Effluent release of hydrogen sulfide is regulated by all of the provinces. Additionally, in order to comply with the federal *Pulp and Paper Effluent Regulations*, all kraft pulp and paper mills that discharge effluent to the environment have secondary wastewater treatment, which is expected to limit concentrations of hydrogen sulfide in the final effluent released.

NPRI data for 2014 indicate that kraft mills reported total releases of 583 tonnes of hydrogen sulfide from 20 mills in Canada (NPRI 2015). Twelve of those mills reported a total of 47 tonnes released to water (ranging from <0.1 tonnes to 8.4 tonnes each) and 20 mills reported a total of 536 tonnes released to air. These NPRI results can be divided by annual effluent volumes to generate estimates of environmental concentration. While basic corrections for metal-sulfide complexation and representative pH may be applied to these estimates, other aspects of the environmental behaviour of hydrogen sulfide cannot readily be accounted for, namely evaporation and oxidation reactions (as discussed in the Environmental Fate and Behaviour section). These phenomena are inherently addressed by monitoring data and are thus taken into account for the other sectors considered in this assessment. Therefore, the available monitoring data for pulp and paper mills will be used to derive predicted environmental concentrations.

The total quantity of hydrogen sulfide releases reported in 2000 was 1926 tonnes from 34 facilities, the vast majority of which was to air. The amount of hydrogen sulfide released in 2014 was 2060 tonnes to air and 94 tonnes to water, from 146 reporting facilities (NPRI 2013). Declining trends in hydrogen sulfide air emissions from the pulp and paper sector since 2001 are attributed primarily to closing of mills, decreased production levels and changes in estimation methods (NPRI 2013).

6.4 Iron and Steel

Based on data reported to the NPRI (2014), four integrated mills (located in Ontario) belonging to the iron and steel sector released 1855 tonnes of hydrogen sulfide in 2000; 261 tonnes were released in 2004, 200 tonnes in 2008, 118 tonnes in 2014, 130 tonnes in 2015, all to air.

6.5 Livestock Operations

Intensive livestock operations are another source of hydrogen sulfide in Canada. Releases from these sources are not reported to the NPRI, as these operations typically do not meet the reporting criteria. Emission rates of hydrogen sulfide vary depending on

local conditions and methods of manure management. The magnitude of emissions from manures is a function of liquid phase concentration, temperature, and pH. Under anaerobic conditions, livestock and poultry manures will be acidic, with pH values ranging from 5.5 to 6.5 and warm due to bacterial action. This situation creates a considerable amount of hydrogen sulfide that will come rapidly out of solution when the liquid manure is agitated or disturbed. Manure storage tanks, ponds, non-aerated lagoons and land application sites are primary sources of hydrogen sulfide emissions. The factors that increase the emission of hydrogen sulfide include wet manure handling at a manure pH of less than 7.0, a high temperature and a long manure storage time. Under anaerobic conditions, livestock and poultry manures will be acidic, with pH values ranging from 5.5 to 6.5. Under aerobic conditions, any reduced sulfur compounds in manure will be oxidized microbially to nonvolatile sulfate, and there will be minimal emissions of hydrogen sulfide. Hydrogen sulfide generated in dry manure generally will be oxidized as diffusion through aerobic layers occurs. Confinement facilities with manure flushing systems that use fluids from anaerobic lagoons also are sources of hydrogen sulfide emissions. Runoff of hydrogen sulfide from land application of manure does not seem to be problem at ambient temperatures, due to its tendency to evaporate and oxidize rapidly (US EPA 2001).

The quantity of hydrogen sulfide generated by intensive livestock operations has been estimated on the basis of the number of swine and cattle in Canada and an average emission factor per animal. Hydrogen sulfide production in 2001 was estimated at 126 107 tonnes, of which 121 441 tonnes was from swine and the rest from cattle (Chetner et al. 2001; Statistics Canada 2003). If proper manure management practices are followed at intensive livestock operations, most of this hydrogen sulfide will be incorporated into soil using techniques to avoid evaporative losses.

6.6 Publicly Owned Wastewater Treatment

In 2009, the five wastewater treatment systems that reported to the NPRI (2013)—Greater Vancouver (two systems, Delta and Richmond) and Kamloops, British Columbia; Regina, Saskatchewan; and Burlington, Ontario—indicated total on-site releases of 179 tonnes of hydrogen sulfide. Of this amount, 157 tonnes was released to air and 22 tonnes was released to water. An additional 4.5 tonnes was disposed of off-site. In 2015, there were 156 tonnes released to air and 0 tonnes to water, from three reporting wastewater treatment systems in Canada (Regina, Kamloops and Mississauga) (NPRI 2017). NPRI results from 2014 indicated that four wastewater treatment systems released 153 tonnes to air and 22 tonnes to water.

7. Measured Environmental Concentrations

Hydrogen sulfide has been measured/estimated for Canadian air, surface water, and effluent from publicly owned and industrial wastewater treatment facilities.

Environmental concentrations of hydrogen sulfide are presented in this section, including available monitoring information, focussing on measurements made at or near oil and gas facilities, pulp and paper operations, wastewater treatment systems, and intensive livestock operations.

7.1 Air

A summary of studies measuring levels of hydrogen sulfide in air, including ambient air, is provided in Appendix A.

Ambient air quality objectives for hydrogen sulfide developed by provinces are based on the concentrations at which humans can begin to detect odours (British Columbia 2016). The objectives are reported for total reduced sulfur (TRS) compounds and are measured as hydrogen sulfide.

The reported value of 1 ppb ($1.4 \mu\text{g}/\text{m}^3$) is considered the average concentration found in urban areas away from point sources (Alberta Environment 2000a).

The US EPA (1993) has reported that concentrations of hydrogen sulfide in ambient air from natural sources are typically less than about 0.3 ppb or $0.5 \mu\text{g}/\text{m}^3$.

7.1.1 Oil and gas facilities

Numerous monitoring stations in Alberta measure continuous, hourly hydrogen sulfide concentrations in air, and data are available from the Clean Air Strategic Alliance (CASA) Data Warehouse website. The CASA Data Warehouse is a publicly available database of continuously stored air pollutant concentrations operated by several organizations, including Alberta Environment and Environment and Climate Change Canada. Mean and 99th percentile concentrations of hydrogen sulfide were calculated from review of the CASA data from the 35 monitoring sites near oil sands facilities between May 2007 and May 2017. The Bonnyville station shows the highest 99th percentile concentration of 15 ppb ($20.9 \mu\text{g}/\text{m}^3$) (CASA 2017). The maximum concentration reported across all stations was 113 ppb ($58.2 \mu\text{g}/\text{m}^3$) at the Scotford Station No. 2 on October 2015 (Appendix A, Table A-2). The highest mean of all hourly samples at each station between May 2007 and May 2017 was 0.97 ppb ($1.35 \mu\text{g}/\text{m}^3$).

Hydrogen sulfide was measured in 2015 at an annual average concentration of 0.3 to 0.7 ppb (0.42 to $0.98 \mu\text{g}/\text{m}^3$) at 19 fixed sites in Alberta near oil sands plants (WBEA 2016). The maximum 1-hour and 24-hour average concentrations from this study were 36 ppb ($50.4 \mu\text{g}/\text{m}^3$) and 6 ppb ($8.4 \mu\text{g}/\text{m}^3$), respectively. In 2014, hydrogen sulfide was measured at an average hourly concentration of 0.2 to 0.3 ppb (0.28 to $0.42 \mu\text{g}/\text{m}^3$) in the Western Yellowhead Air Management Zone (AMEC 2014). In the same study, maximum 1-hour and 24-hour average concentrations of 13.5 ppb ($18.9 \mu\text{g}/\text{m}^3$) and 2.3 ppb ($3.22 \mu\text{g}/\text{m}^3$), respectively, were reported. Between August 2013 and August 2016, an average hourly concentration of total reduced sulfur of <1 ppb ($<1.4 \mu\text{g}/\text{m}^3$) was

reported in Saint John, New Brunswick, of which up to 60% may be hydrogen sulfide (New Brunswick 2016; Environment Canada 2004b). In the same study, 99th percentile and maximum hourly concentrations of 0.6 to 1.2 ppb (up to 0.84 to 1.68 $\mu\text{g}/\text{m}^3$) and up to 10.8 ppb (up to 15.12 $\mu\text{g}/\text{m}^3$), respectively, were reported, based on the assumption of up to 60% of total reduced sulfur being hydrogen sulfide.

In 2015, annual average hydrogen sulfide concentrations of 0.6 to 1.4 ppb (0.84 to 1.96 $\mu\text{g}/\text{m}^3$) were reported in southeastern Saskatchewan (SESAA 2015). The maximum 1-hour and 24-hour average concentrations were 118.6 ppb (166.0 $\mu\text{g}/\text{m}^3$) and 14.0 ppb (19.6 $\mu\text{g}/\text{m}^3$), respectively. In 2014, average and maximum hourly hydrogen sulfide concentrations of up to 0.36 ppb (up to 0.504 $\mu\text{g}/\text{m}^3$) and up to 6 ppb (up to 8.4 $\mu\text{g}/\text{m}^3$), respectively, were reported in a First Nations community in southwestern Ontario, based on the assumption of up to 60% of total reduced sulfur being hydrogen sulfide (MOECC 2016; Environment Canada 2004b). In 2014, maximum 1-hour and 24-hour average concentrations of hydrogen sulfide of 2.0 ppb (2.8 $\mu\text{g}/\text{m}^3$) and 1.8 ppb (2.52 $\mu\text{g}/\text{m}^3$), respectively, were reported at a station in the Northwest Territories (Northwest Territories 2014). In 2014, average hourly hydrogen sulfide concentrations of <1 to 1 ppb (<1.4 to 1.4 $\mu\text{g}/\text{m}^3$) were reported in Edmonton, Alberta (ACA 2014). In the same study, maximum 1-hour and 24-hour average concentrations of 22 ppb (30.8 $\mu\text{g}/\text{m}^3$) and 3 ppb (4.2 $\mu\text{g}/\text{m}^3$), respectively, were reported.

There are two air quality objectives for hydrogen sulfide in Alberta. The 1-hour objective is based on odour perception and is set at 10 ppb (14 $\mu\text{g}/\text{m}^3$), whereas the 24-hour average objective is 3 ppb (4.2 $\mu\text{g}/\text{m}^3$) and is set to protect against health effects. In 2009, 1-hour exceedances occurred most frequently in the oil sands region north of Fort McMurray, at the Mildred Lake (571), Mannix (494), Lower Camp (221), and Buffalo Viewpoint (61). One-hour exceedances also occurred less frequently at Calgary East (25) and in Fort Saskatchewan (1) in 2009. A wastewater treatment system is considered to be a potential source of atmospheric emissions of hydrogen sulfide near the Calgary East monitoring station, while potential sources of release in Fort Saskatchewan include nearby oil and gas and fertilizer industries (Alberta Environment 2011).

The Southeast Saskatchewan Airshed Association (SESAA), a non-profit organization of public, industry, government, and non-government members, collects air quality data for the southeast Saskatchewan region. The southeast Saskatchewan airshed encompasses an area of approximately 36 800 km^2 in a region where major economic activities include natural gas and petroleum production, tanneries, wastewater treatment, kraft paper mills, rayon textile manufacturing, and tar and asphalt manufacturing. A review of the 30 monitoring stations indicates monthly average concentrations ranged from 0.2 ppb (0.28 $\mu\text{g}/\text{m}^3$) to 3.2 ppb (4.5 $\mu\text{g}/\text{m}^3$) from January 2010 to July 2013 (SESAA 2013).

Four monitoring stations are located in the Northwest Territories (Yellowknife, Inuvik, Fort Liard and Norman Wells). The maximum hourly hydrogen sulfide concentrations for

2010 to 2011 ranged from 2 to 5 $\mu\text{g}/\text{m}^3$ (NWTENR 2010, 2011, 2014). The maximum recorded 24-hour average ranged from 2 to 4 $\mu\text{g}/\text{m}^3$. The vast majority of readings were less than 1 $\mu\text{g}/\text{m}^3$.

The highest maximum hourly concentrations were reported at the Cameron Hills upstream oil and gas facility in the Northwest Territories, with maximum hourly concentrations of 83 and 81 $\mu\text{g}/\text{m}^3$ (59 and 58 ppb) reported from May 2006 to April 2007 and from May 2008 to April 2009, respectively (Girard 2007; Chepelkevitch 2009). The highest monthly average hydrogen sulfide concentration recorded by passive monitors was 27 $\mu\text{g}/\text{m}^3$ (19 ppb) (Chepelkevitch 2009). The facility also reported 40 to 50 1-hour hydrogen sulfide exceedances of the Alberta Ambient Air Quality Objective (AAQO) of 13.94 $\mu\text{g}/\text{m}^3$ (10 ppb) in each of the 12-month monitoring periods.

WGAQOG (2000) summarized some older data for hydrogen sulfide in air from 93 monitoring stations across Canada for the period January 1989 to December 1998. Fourteen of the sampling sites were at oil and gas facilities and six were at oil sands sites. The 99th percentile hourly concentration associated with oil and gas refineries was 12 ppb (16.8 $\mu\text{g}/\text{m}^3$), while that for oil sands facilities was 7 ppb (9.8 $\mu\text{g}/\text{m}^3$). A maximum hourly concentration of 83 $\mu\text{g}/\text{m}^3$ (59 ppb) was reported for the Cameron Hills upstream oil and gas facility in the Northwest Territories (NWT). Concentrations in areas characterized as “urban” were among the lowest.

Some additional information on concentrations in air in the vicinity of oil and gas facilities is presented in the Exposure Assessment part of the Human Health section of this report, as well as in Appendix A.

7.1.2 Publicly owned wastewater treatment systems (POWWT)

Wastewater treatment systems can release hydrogen sulfide to the air, in addition to water. In late November 2014, hydrogen sulfide was measured at an hourly concentration of 25 ppb (35 $\mu\text{g}/\text{m}^3$) (statistical metric unspecified) near a wastewater treatment system in Alberta (Huffington Post 2016; CBC 2016; Global News 2016).

At an air monitoring station near the Bonnybrook wastewater treatment system in Calgary, Alberta, a maximum monthly average concentration of 2 $\mu\text{g}/\text{m}^3$ (1.4 ppb) and a maximum 1-hour value of 53 $\mu\text{g}/\text{m}^3$ (38 ppb) were reported, based on data from January 1989 to July 2003 (Hoeksema 2004).

7.1.3 Pulp and paper mills

In 2015, hydrogen sulfide was detected in 2 of 12 air samples (each collected in a different month over a 24-hour period) from a smelt-dissolving tank stack at a kraft pulp mill in British Columbia, at concentrations of 47.51 ppb (66.51 $\mu\text{g}/\text{m}^3$) and 121.75 ppb (170.44 $\mu\text{g}/\text{m}^3$) (British Columbia 2015). These values are higher than the total reduced sulfur (TRS measured as hydrogen sulfide) objectives developed by the province to manage air quality in the province. BC TRS (measured as hydrogen sulfide) 1-hour and

24-hour objectives are 7 µg/m³ (5 ppb) and 3 µg/m³ (2 ppb), respectively. It is likely that the elevated concentrations are the result of accidental releases of black liquor spills to the treatment plant (personal communication with Pulp and Paper, Water and Land Issue, Environmental Protection Branch, ECCC, dated September 26, 2016).

Environment Canada (2004b) reported total reduced sulfur concentrations in 1-hour air samples at 50 pulp and paper mills across Canada representing a period from the mid-1990s to 2003. 99th percentile and/or maximum concentrations were estimated for each monitoring site. The highest 99th percentile 1-hour total reduced sulfur concentration was reported to be 63 ppb (88.2 µg/m³) at a mill in Ontario in 2000. The average annual concentration of 3.2 ppb (4.5 µg/m³) estimated for the same Ontario mill was also the highest average annual value reported nationally. Based on the assumption that hydrogen sulfide can make up as much as 60% of total reduced sulfur (Environment Canada 2004b), this corresponds to hydrogen sulfide concentrations of up to 37.8 ppb (52.9 µg/m³) and 1.9 ppb (2.7 µg/m³), respectively.

As mentioned previously, WGAQOG (2000) summarized data for hydrogen sulfide in air from 93 monitoring stations across Canada for the period of January 1989 to December 1998. Sixty-four of the sampling sites were located near pulp and paper mills. The 99th percentile hourly concentration associated with pulp and paper mills was reported as 31 ppb (43.4 µg/m³), the highest for all of the sectors examined. The overall maximum reported hourly concentration for all sectors of 503 ppb (705 µg/m³) was also measured near a pulp and paper mill, as was the highest monthly average concentration of 3.9 ppb (5.5 µg/m³).

Additional information on concentrations of hydrogen sulfide in air in the vicinity of pulp and paper facilities is presented in the Exposure Assessment part of the Human Health section and Appendix A of this report.

7.1.4 Intensive livestock operations

Hydrogen sulfide was measured both upwind and downwind of a beef cattle, a dairy cattle, a poultry and a swine confined feeding operation (CFO) in Alberta over a 14-month period (Alberta Government 2011). The 1-hour and 24-hour average hydrogen sulfide concentrations were compared to the AAQOs, i.e., 10 ppb (14 µg m⁻³) and 3 ppb (4 µg m⁻³), respectively. The mean, minimum, median and maximum 1-hour average hydrogen sulfide concentrations were measured at each mobile station with respect to all wind directions in each measurement period. The 1-hour average concentrations ranged from not detected to 6.59 ppb, and from not detected to 22.8 ppb at mobile stations one and two, respectively. A total of two exceedances of the 1-hour average AAQO for hydrogen sulfide were recorded at mobile station two during the study. A comparison of the average 24-hour concentration measurements to the 24-hour average AAQO for hydrogen sulfide showed that no exceedance of the latter AAQO occurred during the study.

Intensive livestock operations are widespread across Canada. The maximum 1-hour concentration of hydrogen sulfide at two swine feeding operations was 76 and 26 $\mu\text{g}/\text{m}^3$ in the Lethbridge, Alberta area. Hydrogen sulfide levels monitored at all other sites ranged from 1.4 to 11 $\mu\text{g}/\text{m}^3$ (Alberta Environment 2000b).

Air quality surveys conducted near a group of swine rearing facilities south of Girouxville Alberta (Alberta Environment 2007) reported a median 1-hour average concentration of 8.4 $\mu\text{g}/\text{m}^3$ (6 ppb) hydrogen sulfide for the fall 2005 survey, while the median concentration at the same location during the spring 2006 survey was more than double at 21 $\mu\text{g}/\text{m}^3$ (15 ppb). In contrast, 1-hour average hydrogen sulfide concentrations at the background sites ranged from below the detection limit to 1.4 $\mu\text{g}/\text{m}^3$ (1 ppb).

A monitoring study carried out from October 2007 to September 2008 by the Peace Airshed Zone Association (PAZSA 2011) reported a maximum 1-hour concentration of 21 $\mu\text{g}/\text{m}^3$ (14.9 ppb) near Girouxville. During the project, there were no exceedances of the 24-hour hydrogen sulfide AAQO of 4.2 $\mu\text{g}/\text{m}^3$ (3 ppb). For over 90% of the study duration, hydrogen sulfide concentrations were at or below the 1.4 $\mu\text{g}/\text{m}^3$ (1 ppb) detection limit of the instrument at the station. The highest concentrations of hydrogen sulfide were observed during the winter months when conditions favour poor dispersion, particularly during temperature inversions.

7.2 Water

Concentrations of dissolved hydrogen sulfide in water and wastewater are estimated indirectly from measured dissolved sulfide concentrations. As indicated in Table 3-1, the proportion of un-ionized H_2S to dissolved bisulfide ion (HS^-) varies mainly as a function of water pH. When estimating concentrations of hydrogen sulfide from water pH data and measured dissolved sulfide levels, it is typically assumed that most of the dissolved sulfides detected are present as free bisulfide (HS^-) ions or as un-ionized hydrogen sulfide.

Standard methods of quantifying dissolved sulfides typically measure “acid-labile” species (Bowles et al. 2003). These are the sulfides (e.g., HS^-) liberated as gaseous H_2S when acid is added to a water sample. It is only relatively recently however that it has been recognized that oxic surface water typically contains significant amounts of dissolved and colloidal metal sulfides (e.g., $\text{FeS}_{(\text{aq})}$, $\text{ZnS}_{(\text{aq})}$), which can also be liberated by such methods (Adams and Kramer 1999; Bowles et al. 2003; Sukola et al. 2005). Because of the high affinity of dissolved metals for sulfide and the instability of free sulfide in oxic water, it is reasonable to expect on theoretical grounds that the vast majority of the acid-labile sulfide detected in oxic surface water and wastewater is actually in the form of dissolved metal (particularly iron) sulfides, as described in Adams and Kramer (1999) and Sukola et al. (2005).

Only one study was identified that measured concentrations of different dissolved sulfide species in oxic freshwater and wastewater. Rozan et al. (2000) measured total dissolved sulfide concentrations, as well as concentrations of $\text{FeS}_{(\text{aq})}$, $\text{FeSH}^+_{(\text{aq})}$, $\text{CuS}_{(\text{aq})}$,

ZnS_(aq), and polysulfides (S_x²⁻), in water samples from seven rivers and in wastewater samples from two wastewater treatment systems in northeastern United States. They noted that HS⁻ is one of the species that could contribute to their measured total dissolved sulfide results. Assuming, as previously noted, that in addition to free H₂S/HS⁻ it is mostly dissolved iron and zinc sulfides that contribute to measured acid-labile sulfide concentrations, the results of Rozan et al. (2000) suggest that no more than about 15% of dissolved sulfide concentrations determined by standard methods could be present as free H₂S/HS⁻ (Doyle 2013). Considering the likely uncertainties in the concentrations reported by Rozan et al. (2000), this percentage should be considered no more than a rough upper-bound estimate (Tessier 2013). The actual percentage of dissolved sulfide concentrations is expected to be much lower because several different sulfide species in addition to H₂S/HS⁻ (e.g., AgS_(aq), CdS_(aq), HgS_(aq), and PbS_(aq), and S_(aq)⁰) could have contributed to the 15% estimated and, more importantly, because free H₂S/HS⁻ is inherently unstable in oxic water, with losses occurring by both reaction and volatilization (Bowles et al. 2003; Sukola et al. 2005).

The potential to find free bisulfide ions in oxic water is greatly reduced when concentrations of dissolved iron (and other metals) are higher than those of dissolved sulfide (Luther 2013), expressed on a molar basis. In this situation, depending on the relative concentrations of and affinities for other ligands, the metals will tend to react with the available sulfide to form relatively stable dissolved metal-sulfide complexes.

In fact, it is common for dissolved metals (especially iron) to be more abundant than sulfide in oxic surface water and wastewater. For example, in a study of several lakes in Quebec by Sukola et al. (2005), molar concentrations of dissolved iron were reported to be more than two orders of magnitude above molar acid-labile sulfide levels. Similarly, Rozan et al. (2000) reported high concentrations of dissolved iron compared to sulfide in samples of water and wastewater from the northeastern United States. In addition, limited spot-checking of unpublished data on dissolved sulfide and iron levels in the Athabasca and other rivers of northern Alberta (Alberta Environment 2013) indicates that molar dissolved iron concentrations are almost always higher than those of acid-labile dissolved sulfide, typically by two or more orders of magnitude.

It has therefore been assumed in this assessment that at least 85% of dissolved sulfide concentrations measured using standard analytical methods is in the form of iron (and zinc) sulfides, and that no more than about 15% is in the form of free H₂S/HS⁻. However, as explained above, the actual percentage present in the form of H₂S/HS⁻ is in most cases expected to be much lower. In line with this expectation, Luther et al. (2003), using voltametric scans, reported finding significant amounts of FeS_(aq) but only traces of H₂S in water at the oxic-anoxic interface of a stratified man-made lake in Pennsylvania with an excess of dissolved iron relative to sulfide. Similarly, Rozan and Benoit (1999) reported finding no indication of free HS⁻ but significant amounts of dissolved copper-sulfide complexes in oxic relatively metal-rich water samples taken from four rivers in southern New England. Consequently, the concentrations presented

in this section of the report, determined assuming that no more than 15% of measured dissolved sulfide is in the form of free $\text{H}_2\text{S}/\text{HS}^-$, are referred to as “upper-bound” values.

It should be mentioned that unless precautions are taken (e.g., addition of zinc acetate preservative to sampled water), there is a danger that $\text{H}_2\text{S}/\text{HS}^-$ will be lost from water samples by either oxidation or volatilization prior to analysis (Holm et al. 2000). In addition, there is evidence that some dissolved metal sulfides are lost from water by adsorption onto the wall of sample containers and when samples are passed through filters to remove suspended solids (Bowles et al. 2003). This could lead to an underestimation of concentrations. However, results of laboratory tests by Bowles et al. (2003) suggest that such losses may be significantly mitigated by the presence of the organic matter commonly found in natural water and wastewater.

7.2.1 Remote locations

Two published studies were identified that reported concentrations of dissolved sulfides in samples of oxic Canadian freshwater obtained in areas where there is little potential for contamination. Nanogram per litre concentrations of dissolved sulfide (maximum of about 100 ng/L) were reported in three Quebec lakes and three rural Ontario water bodies by Sukola et al. (2005) and Bowles et al. (2003), respectively. Using these reported dissolved sulfide values and water pH data (pH ranged from 5.6 to 7.7), and assuming as explained above that at least 85% of measured sulfide is complexed with iron, upper-bound concentrations of un-ionized hydrogen sulfide in these relatively pristine waters are estimated to range from 0.001 to 0.02 $\mu\text{g}/\text{L}$. Although relevant data are limited, these results suggest that concentrations of un-ionized hydrogen sulfide in uncontaminated oxic fresh water in Canada are unlikely to ever exceed about 0.1 $\mu\text{g}/\text{L}$.

7.2.2 Oil and gas facilities / coal mines

Alberta Environment (2013) has collected data on dissolved sulfide concentrations in over 3000 samples of surface water in the province dating back to the late 1970s. Unfortunately, analytical detection limits for samples obtained prior to about 1990, representing perhaps half of the overall data set, were too high to provide meaningful results. Hydrogen sulfide concentrations in water samples collected more recently (throughout the 1990s and 2000s) were calculated on the basis of available dissolved sulfide data and estimated water pH (typically 8.0), assuming as explained above that at least 85% of measured sulfide is complexed with iron. In fact, the percentage complexed with iron is likely much more than 85% since results of spot-checks indicate that molar concentrations of dissolved iron are almost always higher than those of acid-labile dissolved sulfide, typically by two or more orders of magnitude, in the Athabasca and other rivers of northern Alberta (Alberta Environment 2013).

Upper-bound concentrations of un-ionized hydrogen sulfide so estimated for northern Alberta rivers were generally less than 0.1 $\mu\text{g}/\text{L}$. There were, however, a few exceptions. Hydrogen sulfide concentrations of up to 320 $\mu\text{g}/\text{L}$ were determined for

samples of outflow water collected in 2008 from the bioreactor at an Alberta coal-fired plant. Actual concentrations were likely somewhat lower than this, since in this case sulfide was measured as a “total” concentration (including particulates) as opposed to a dissolved concentration. Unfortunately, no monitoring data were available for surface water in the vicinity of this site. High hydrogen sulfide concentrations (up to 100 µg/L) were also estimated for spring water collected in the early 1990s at an abandoned Alberta oil well. Again, however, no information is available on concentrations in nearby surface water. Somewhat elevated hydrogen sulfide concentrations (up to 0.4 µg/L) were also estimated for water samples collected over a four-year period (2008-2011) from the Athabasca River downstream from an oil sands operation.

7.2.3 Pulp and paper mills

Results from the pulp and paper industry’s National Council for Air and Stream Improvement (NCASI 2012) study of 25 pulp and paper mills—located mostly in the United States but with some in Canada—indicated total dissolved sulfide levels in samples of biologically treated final effluents (the year of sample collection was not specified) ranging from non-detect (< 30 µg/L) to 290 µg/L (0.29 mg/L). The average concentration was 100 µg/L and the median concentration was 70 µg/L. The study also provides estimates for hydrogen sulfide concentrations in receiving waters near the discharge points for the 25 mills. These values are based on a dilution factor that encompasses 80% of mills and takes into account low flow volumes in receiving waters. The estimated hydrogen sulfide values in receiving waters ranged from non-detect to 14 µg/L (0.014 mg/L) with an average of 5 µg/L (0.005 mg/L) and a median of 3.5 µg/L (0.0035 mg/L). However, these concentration estimates do not account for the likely contribution of iron sulfides to their measured dissolved sulfide values. Assuming, as explained above, that the level of metal sulfide complexing is at least 85%, the upper-bound average, median and maximum concentrations in receiving waters would be 0.75 µg/L, 0.53 µg/L and 2.1 µg/L, respectively. In fact, as explained previously, the percentage complexed with iron may be much more than 85%, especially since significant quantities of metals (particularly iron and manganese) are commonly found in pulp and paper wastewater (e.g., Palumbo et al. 2010).

The Meadow Lake mill, located approximately 300 km north of Saskatoon, reported a smelt composition of 6% sodium bisulfide. Through a process known as the green liquor splitter system, 98% sodium bisulfide was removed (Jemaa et al. 2009), thereby reducing the amount of “smelt” containing sodium bisulfide being sent to landfills.

7.2.4 Publicly owned wastewater treatment systems

According to a 2006 survey of publicly owned wastewater treatment systems, approximately 21% of the Canadian population is served by primary treatment or less, and 79% of the population is served by at least secondary treatment (Environment Canada 2010).

Results from a preliminary study of two publicly owned wastewater treatment systems in Canada in November 2012 and winter/spring 2013 indicated that hydrogen sulfide was not present at a detectable concentration (detection limit of 2 µg/L) (ECCC 2015). The wastewater system included a large urban secondary activated sludge process and a facultative lagoon with a retention time of approximately four months. Raw influent, primary effluent and final effluent were collected from one system, and raw influent and final effluent were collected from the second system. Hydrogen sulfide (as S²⁻) was detected in all raw influent and primary effluent samples from both wastewater systems (n=6, and n=3 respectively), and was not present at a detectable concentration in any effluent samples (n=6). It was concluded that any wastewater treatment system that is “secondary or equivalent”, i.e., achieving reductions of biological oxygen demand (BOD) and total suspended solids (TSS) as specified in the *Wastewater Systems Effluent Regulations* (Canada 2012b), will also remove hydrogen sulfide to non-detectable levels.

A monitoring program in Quebec identified relatively high concentrations of dissolved sulfides in the effluents from a wastewater treatment system with primary treatment and non-aerated lagoon systems (MEQ 2001a,b). Samples were collected in the period of 1997 to 1999 at 15 wastewater treatments systems across the province. The highest mean values, i.e., 110 and 140 µg/L, were recorded in the summer at two wastewater treatment systems, both of which discharge to the St. Lawrence River. A mean concentration of 120 µg/L dissolved sulfide was also recorded in a lagoon sample at Martinville (located southeast of Sherbrooke) in the winter. Assuming a water pH of approximately 7.5 (a typical value for St. Lawrence River water; Ramesh 1989, and assuming, as explained above, that at least 85% of the measured dissolved sulfides are complexed with iron, the upper-bound H₂S concentrations in these effluents are estimated to be in the 4.0 to 5.0 µg/L range. In fact, the percentage complexed with iron is likely to be much more than 85% since relatively high concentrations of total extractable iron were reported in system effluents, i.e., from about 200 to 1700 µg/L, depending on whether or not wastewater had been treated with ferric chloride (MEQ 2001a,b). Allowing for 10-fold dilution after release, the resulting upper-bound concentrations in receiving surface waters would be 0.4 to 0.5 µg/L.

The program also included toxicity testing of the effluents. Although it is not possible to establish unambiguous relationships between the presence of a contaminant in an effluent and the observed toxicity, the authors of the MEQ (2001a,b) report did note that hydrogen sulfide is one of the substances that may have been responsible for some of the adverse effects observed.

In an unpublished 2003 study of water quality in the St. Lawrence River, Environment Canada researchers at the Centre St. Laurent in Quebec measured dissolved sulfide at concentrations of 20 µg/L at 0.5, 1.0 and 2.5 km downstream of the outfall of a wastewater treatment system and of 10 µg/L at 7 km downstream of the outfall (Environment Canada 2004c). Assuming a pH of 7.5 (Ramesh 1989) and assuming (as previously explained) that at least 85% of the measured dissolved sulfides are

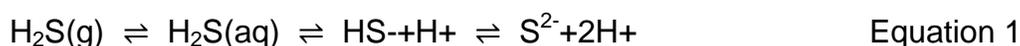
complexed with dissolved iron, which is more abundant than sulfide in these waters (Gagnon and Turcotte 2007), the upper-bound hydrogen sulfide concentrations in these waters are estimated to be 0.75 and 0.40 µg/L, respectively.

Adams and Kramer (1999) measured dissolved sulfides in effluents from wastewater treatment systems in Dundas and Burlington, Ontario, in 1997-1998. Both plants use an activated sludge treatment process with the addition of ferric chloride to enhance floc formation and settling in the clarifier tanks. They reported finding 7.1 µg/L (223 nM) in the effluent from the Dundas plant and 9.0 µg/L (280 nM) in the effluent from the Burlington plant. Samples of surface water were also taken downstream of the Dundas plant in the Desjardin Canal. Measured concentrations ranged from 6.5 µg/L (202 nM) close to the plant outfall, to 5.9 µg/L (184 nM) 500 metres downstream from the outfall. Assuming a pH of 7.4 (Adams and Kramer 1999) and assuming that (as previously explained) at least 85% of the measured dissolved sulfides are complexed with iron, the upper-bound hydrogen sulfide concentrations in these surface waters are estimated to be 0.27 and 0.25 µg/L, respectively.

8. Environmental Fate and Behaviour

A summary of the physical and chemical properties of hydrogen sulfide, sodium bisulfide and sodium sulfide that are relevant to their environmental fate are presented in Tables 3-1 and 3-2. Information on their behaviour in the environment, including their persistence and bioaccumulation potential, are presented below.

Hydrogen sulfide is a weak acid; it equilibrates with its anions HS⁻ and S²⁻ in aqueous solution (second and third equilibria of equation 1) (Li and Lancaster 2013).



Based on equation 1, the leftward equilibrium shift could cause a decrease in hydrogen sulfide concentration but also an increase of the solution pH. Equation 1 is also the basis of the application of hydrogen sulfide gas or inorganic metallic sulfide such as sodium sulfide (Na₂S) and sodium hydrosulfide (NaHS) as hydrogen sulfide sources in solution. An unbuffered stock solution from hydrogen sulfide gas tends to be acidic, whereas that from metallic sulfide is basic (Li and Lancaster 2013).

Sodium bisulfide and sodium sulfide

When exposed to air, sodium bisulfide undergoes autoxidation and gradually forms polysulfur, thiosulfate, and sulfate. It also absorbs carbon dioxide, forming sodium carbonate (Bush 2000). Sodium sulfide, when exposed to air, will oxidize to sodium sulfate (Na₂SO₄), although a number of intermediate sulfur compounds (polysulfides and thiosulfates) will also result (HIGP 1989). Sodium bisulfide is very soluble in water. In this medium, the substance will immediately dissociate: the sulfur will enter the natural sulfur cycle and, depending on the pH, hydrogen sulfide can form. Sodium

sulfide is a solid at environmental conditions with a high boiling point and water solubility. The substance is readily soluble in water.

8.1 Environmental Distribution and Persistence

Hydrogen sulfide is expected to be released to the environment through air and water. Hydrogen sulfide is a gas under environmental conditions and is expected to partition from water or land into the atmosphere. The substance's atmospheric residence time ranges from 0.6 to 29 days. Hydrogen sulfide is soluble in water and is mobile in aquatic environments and moist soil; its aquatic aerobic half-life is short.

8.1.1 Air

Hydrogen sulfide is a gas under typical environmental conditions, so when released to water or land it will tend to partition from these media into the atmosphere. Hydrogen sulfide that is released into the atmosphere may form localized, low-lying clouds that will be rapidly dispersed and consequently diluted by windy conditions and turbulence (NRCC 1981). This dispersion may be accompanied by wet deposition, dry deposition and chemical transformations, which will further decrease ambient concentrations.

Hydrogen sulfide does not absorb solar radiation in the lower atmosphere (troposphere) and thus is photochemically stable (Warnek 1988).

Hydrogen sulfide is removed from the atmosphere mainly by oxidation reactions with hydroxyl radicals (OH•). The exact mechanism of this reaction has not been determined, but it is believed to be initiated via a hydrogen abstraction reaction, as shown below:



The resulting sulfhydryl radical (SH•) does not build up in the atmosphere, and it is thought that it is removed by reaction with formaldehyde and ozone (Iowa 2002).

The rates of reaction of hydrogen sulfide with other oxidants such as O₃, NO₂, O₂ and RO₂ are too slow to compete with the OH• reaction. The rate constant for the reaction of hydroxyl radical with hydrogen sulfide has been experimentally determined to be $(5.2 \pm 0.8) \times 10^{-12} \text{ cm}^3/\text{s}$ (Barnes et al. 1986).

The residence time of hydrogen sulfide in the atmosphere is affected by location, temperature and other atmospheric variables, such as concentrations of radical precursors, sunlight and humidity. The mean tropospheric conversion time of hydrogen sulfide to sulfur dioxide by reaction with hydroxyl radicals in California is about 18 hours (Sprung 1977; NRCC 1981). Jaeschke et al. (1980) found a significant maximum concentration in winter and a minimum concentration in summer. The atmospheric

residence time of hydrogen sulfide has been estimated to range from 0.93 days in summer to 42 days in winter at a latitude equal to that of Edmonton, Alberta (Bottenheim and Strausz 1980), equating to atmospheric half-lives of approximately 0.65 and 29 days, respectively. This variation is due in part to the thermal sensitivity of the chemical transformations of hydrogen sulfide with decreased temperatures and sunlight, as well as decreased levels of hydroxide radicals in northern regions, tending to slow reaction rates. In many places in Canada, the atmospheric half-life of hydrogen sulfide is thus expected to be significantly greater than 2 days for most of the winter months.

8.1.2 Surface water and soil

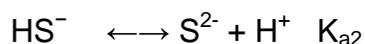
Hydrogen sulfide is a gas that is quite soluble in water, which makes it highly mobile in moist soils and in aquatic environments. Several species of soil and aquatic microorganisms oxidize hydrogen sulfide to elemental sulfur (S^0) under aerobic conditions, and its degradation half-life in these environments usually ranges from 1 hour to several hours (Jørgensen 1982). Volatilization is also an important loss mechanism from soils. However, soils can also act as a sink for airborne hydrogen sulfide being adsorbed onto clay or organic matter, followed by rapid chemical and biological oxidation to elemental sulfur (Cihacek and Bremner 1993). A number of organisms have been found to degrade hydrogen sulfide to elemental sulfur and sulfate, including a heterotrophic bacterium isolated from dimethyldisulfide-acclimated peat (Cho et al. 1992), heterotrophic fungi (Phae and Shoda 1991) and the marine isopod *Saduria (Mesidotea) entomon* (Vismann 1991).

Because of its physical and chemical properties and fate, hydrogen sulfide is very short-lived in water under aerobic (oxic) conditions. Hydrogen sulfide will evaporate relatively rapidly from water, depending on factors such as temperature, humidity and pH (HSDB 2003). The environmental model WVOLWIN uses a Henry's Law constant value to predict an aquatic evaporative half-life of 38 minutes in a river and 56 hours in a lake (Environment Canada 2002). The model does not, however, take into account the fact that a portion of dissolved hydrogen sulfide is ionized. Actual half-lives could therefore be somewhat longer, especially in alkaline water where the dominant species is the HS^- ion. Although the oxidative half-life of hydrogen sulfide in water and wastewater is typically also quite short (i.e., hours to minutes; e.g., Millero et al. 1987; Nielsen et al. 2007; Palumbo et al. 2010), rates are difficult to predict with accuracy due to the complexity of the reactions involved. Sulfides can react chemically with dissolved oxygen, but this is thought to be a slow and complex chain reaction (Millero et al. 1987; Kotronarou and Hoffmann 1991; Nielsen et al. 2003). General rate equations have been developed for wastewater (Wilmot et al. 1988; Nielsen et al. 2004) and pulp and paper mill (Palumbo et al. 2010) effluent, but they do not account for all of the significant factors involved in the oxidation of hydrogen sulfide. In general, in aerobic water oxidation rates can vary (by a factor of up to 100) depending on concentrations of dissolved metals (e.g., nickel, cobalt, manganese and copper), temperature, concentration of other reactants, pH, amount and type of microbial activity and ionic

strength. As well, the presence of some commonly found organic chemicals in wastewater can either increase or decrease the oxidation rate.

However, hydrogen sulfide can exist for relatively long periods in water under anoxic conditions and is often associated with anoxic sediments (Andreae and Jaeschke 1992).

When hydrogen sulfide enters oxygenated water, it dissolves and dissociates according to the reaction:



where K is the equilibrium constant ($K = 9.12 \times 10^{-8}$ at 25 °C).

Dissociation in water depends primarily on the pH of the water, although temperature and ionic strength of the solution have an effect as well (Holm et al. 2000). The acid dissociation constant for the first reaction, pK_{a1} at 20°C and an electrolytic conductivity of 1200 $\mu\text{S}/\text{cm}$ is 7.04, so that at pH 7.04, half of the dissolved sulfide will be hydrogen sulfide and half will be the bisulfide anion (HS^-). Since the pK_{a2} value is high (11.96; ATSDR 2006), S^{2-} will never be a significant sulfide species under normal environmental conditions. The dominant species will be hydrogen sulfide and the bisulfide anion. As the pH increases, the ratio of the concentration of bisulfide ion to aqueous hydrogen sulfide increases. At pH 6 and a temperature of about 20 °C, 91% will be un-ionized hydrogen sulfide, decreasing to about 9% at pH 8 (Pomeroy and Boon 1990) (Table 8-1). Natural variations in water pH can therefore have a significant effect on the proportion of hydrogen sulfide present. Temperature variations have a more limited influence on the extent of ionization, with lower temperatures favouring the un-ionized hydrogen sulfide form. For example, at a pH of 7.0, the proportion present as un-ionized hydrogen sulfide increases from approximately 50% to 60% as the temperature drops from 20 to 10 °C (Australia and New Zealand Environment and Conservation Council 2000).

Table 8-1. Proportions of dissolved sulfide present as un-ionized hydrogen sulfide and as HS^- at environmentally relevant pH and a temperature of approximately 20 °C (from Pomeroy and Boon 1990)

pH	Proportion of un-ionized H_2S	Proportion of HS^-
5.0	0.99	0.01
6.0	0.91	0.09
6.2	0.86	0.14

6.4	0.80	0.20
6.6	0.72	0.28
6.8	0.61	0.39
7.0	0.50	0.50
7.2	0.39	0.61
7.4	0.28	0.72
7.6	0.20	0.80
7.8	0.14	0.86
8.0	0.09	0.91
8.2	0.059	0.941
8.4	0.039	0.961
8.6	0.025	0.975
8.8	0.016	0.986
9.0	0.010	0.99

When free $\text{H}_2\text{S}/\text{HS}^-$ is introduced into either aerobic or anaerobic fresh water containing metals, reactions with dissolved iron and other metals would also be expected, producing dissolved metal (principally iron) sulfide complexes, which may precipitate out of solution if concentrations are high enough. Reactions of this type are expected, for example, in anoxic sediments (Luther et al. 2003). Rozan et al. (2000) have suggested that the relatively large quantities of dissolved iron sulfide complexes that they found in oxic river water in the northeastern United States had diffused upwards into the water column from underlying anoxic sediments.

8.2 Potential for Bioaccumulation

No reliable bioaccumulation data were identified for hydrogen sulfide. However, bioconcentration and food chain biomagnification of hydrogen sulfide are unlikely considering that it is an inorganic gas and that it has a relatively short half-life in water. As hydrogen sulfide is an inorganic gas, it is not expected to bioconcentrate or bioaccumulate.

Sodium bisulfide and sodium sulfide oxidize in air and are soluble in water. Given their respective $\log K_{ow}$ s, neither substance is expected to bioconcentrate in the environment.

Neither sodium sulfide nor sodium bisulfide accumulate in the environment ($\log K_{ow}$ of -4.23 and -3.5, respectively) (ICSC 2008).

9. Potential to Cause Ecological Harm

9.1 Ecological Effects Assessment

Empirical data on the effects of hydrogen sulfide were considered in the weight-of-evidence for assessing the ecological effects of hydrogen sulfide. Some authors identify the mechanism of toxicity as the immediate binding of hydrogen sulfide to the enzyme cytochrome-*c*-oxidase or other metallo- and disulfide-containing proteins (Beauchamp et al. 1983, Doman et al. 2002). Although the three charge states occur naturally in the environment (H_2S , HS^- and S^{2-}), because HS^- is charged, it is unlikely that it will diffuse easily in to the cells. In contrast, hydrogen sulfide is more capable of permeating cell membranes (Powell 1989).

There is limited information identified on the ecological toxicity of sodium sulfide and sodium bisulfide. Available ecological toxicity information for sodium bisulfide and sodium sulfide is reported as read-across from hydrogen sulfide.

9.1.1 Aquatic compartment

Hydrogen sulfide has been demonstrated to have harmful effects on aquatic organisms at low concentrations.

For a given measured concentration of free dissolved sulfide (excluding dissolved metal sulfides), exposure of aquatic organisms to un-ionized hydrogen sulfide is highly dependent on water pH and to a lesser degree on temperature. As indicated earlier in Table 3-1, at a pH of 5 and a temperature of about 20 °C, about 99% of the sulfide is present as un-ionized hydrogen sulfide; at pH of 8, about 91% is in the form of HS^- .

The toxicity of dissolved sulfides is typically believed to derive primarily from exposure to un-ionized hydrogen sulfide rather than to the bisulfide ion, HS^- (US EPA 1976). With increasing temperature, the degree of toxicity also increases, likely due to the increased metabolic demands in ectothermic aquatic organisms (Broderius and Smith 1976). However, Broderius and Smith (1976) reported that the toxicity of un-ionized hydrogen sulfide to fathead minnow appeared to have increased over the pH range of 6.5 to 8.7. Nevertheless, the overall magnitude of this effect was relatively small, resulting in an approximately 2-fold decrease of LC_{50} values (i.e., concentration expected to cause death in 50% of test animals) over the pH range of 7.5 to 8.5. These results suggest that the HS^- ion, concentrations of which increased as the pH of the test waters was increased, was responsible for some of the observed toxicity. An alternative explanation offered by the authors was that, since the actual pH at the gill surfaces of fish is substantially lower than the measured ambient levels, fish would be exposed to a higher proportion of un-ionized hydrogen sulfide than predicted on the basis of measured pHs, especially under more alkaline conditions. The other possibility—that the HS^- ion itself contributed to the toxic response—is supported by evidence of uptake of HS^- by some aquatic organisms (Julian and Arp 1992; Czyzewski and Wang 2012).

Available acute, chronic and early life-stage toxicity data for aquatic organisms are summarized in US EPA (2009). A selection of data from this source is presented in Table 9-1. All data are reported as concentrations of un-ionized hydrogen sulfide. The following brief description of the data is based on information presented in US EPA (2009).

Acute toxicity values for freshwater fish (7 values) and invertebrates (9 values) range from 14.9 to 1070 µg/L, while acute values for estuarine/marine fish (1 value) and invertebrates (6 values) range from 10 to 1,430 µg/L. Chronic toxicity data are mostly available for freshwater organisms, with values for fish (14 values), invertebrates (6 values) and algae (1 value) ranging from 0.5 to 1874.4 µg/L. Early life-stage toxicity data (48 values) are mostly for short-term tests with freshwater organisms, with various measures of effects to fish and invertebrates reported at concentrations ranging from < 2 to 2900 µg/L. The No Observed Effect Concentration (NOEC) in freshwater fish was 0.4 µg/L for swimming endurance in adult bluegill (*Lepomis macrochirus*) over 97 days (US EPA 1976). The difference between the acute and chronic effect concentrations is small and may be due to the mode of action of hydrogen sulfide. Hydrogen sulfide paralyzes the brain and metabolic functions controlling respiration.

Of the 91 toxicity results included in US EPA (2009), effects to 4 different freshwater species and 1 marine species are reported at concentrations of 2 µg/L or below, and over 20 effect values representing 10 different mostly freshwater species are reported in the 2 to 10 µg/L range.

Regarding the lowest effect values, Fung and Berwick (1980) reported 96-hour LC₅₀s for sac fry of whitefish (*Coregonus clupeaformis*) and yellow perch (*Perca flavescens*) of 2.0 µg/L and < 2.0 µg/L, respectively. Smith et al. (1976) reported a 97-day lowest-observed-effect concentration (LOEC; reproduction) of 1 µg/L for bluegill (*Lepomis macrochirus*). Thompson et al. (1991) reported a 49-day LOEC (decreased weight) of 1.1 µg/L for sea urchin (*Lytechinus pictus*). Lastly, Hoque et al. (1998) reported a 6-week LOEC (reduced growth) of 0.5 µg/L for the freshwater tropical fish *Mystus nemurus*.

Results of a recent review of the aquatic toxicity focused on marine biota (Weston Solutions Inc. 2006) suggest that marine organisms generally are less sensitive to hydrogen sulfide than freshwater organisms. The lowest mean species effect values reported in this review were a LOEC of 11.1 µg/L for the mysid shrimp (*Americamysis bahia*) and an EC₅₀ (i.e., concentration expected to cause a specified negative effect in 50% of test animals) of 7.6 µg/L for the larval bay mussel (*Mytilus galoprovincialis*). The authors of this study further concluded that hydrogen sulfide typically acts in an acutely toxic manner regardless of the test exposure period and, as a consequence, that it is appropriate to combine short- and longer-term aquatic effects data when estimating a toxicity threshold.

Given the above evidence, a critical toxicity value (CTV) of 1.0 µg/L has been selected for determining a predicted no-effect concentration (PNEC) for use in the assessment of toxicity to freshwater aquatic organisms. The bluegill (*Lepomis macrochirus*) was selected as the most sensitive organism on the basis of a LOEC of 1 µg/L. Although one lower toxicity value (0.5 µg/L) was reported, since the organism tested was a tropical fish, this result is considered to have limited relevance to conditions in Canada.

Sodium bisulfide and sodium sulfide

There is limited acute and chronic aquatic toxicity data available for the precursors, sodium bisulfide and sodium sulfide. All results utilize read-across data from hydrogen sulfide or sodium sulfide nonahydrate (CAS RN 1313-84-4).

Table 9-1 Selection of aquatic toxicity data for hydrogen sulfide^a

Species	Duration and Test Endpoint	pH During Test ^b	Value (µg/L)	Reference
Microalgae (<i>Scenedesmus vacuolatus</i>)	24-hour EC ₅₀	6.5–6.6	1874.4	Küster et al. 2005
Isopod (<i>Asellus militaris</i>)	96-hour LC ₅₀	7.5	1070	Smith and Oseid 1974; US EPA 1976
Ephemeroptera (<i>Baetis vagans</i>)	96-hour LC ₅₀	7.6	20.0	Smith and Oseid 1974; US EPA 1976
Crustacean (<i>Daphnia magna</i>)	48-hour EC ₅₀	6.4–6.5	122.0	Küster et al. 2005
Fathead minnow (<i>Pimephales promelas</i>)	96-hour LC ₅₀	7.9	16.0	US EPA 1976
White sucker (<i>Catostomus commersonii</i>)	96-hour LC ₅₀	7.8	18.5	US EPA 1976
Bluegill (<i>Lepomis macrochirus</i>)	96-hour LC ₅₀	7.8–8.0	44.8	Smith et al. 1976
Fathead minnow (<i>Pimephelas. promelas</i>) – fry	96-hour LC ₅₀	7.9	6.6	US EPA 1976

Rainbow trout (<i>Salmo gairdneri</i>) – juvenile	96-hour LC ₅₀	8.0	7.0	Fung and Bewick 1980
Whitefish (<i>Coregonus clupeaformis</i>) – sac fry	96-hour LC ₅₀	8.0	2.0	Fung and Bewick 1980
Yellow perch (<i>Perca flavescens</i>) – Sac fry	96-hour LC ₅₀	8.0	<2.0	Fung and Bewick 1980
California killifish (<i>Fundulus parvipinnis</i>)	96-hour LC ₅₀	8.3	1430	Bagarinao and Vetter 1993
Freshwater Crayfish (<i>Procambarus clarkii</i>)	447-day NOEC (survival)	7.69–7.73	4.1	US EPA 1976
Ephemeroptera (<i>Hexagenia limbata</i>) – nymph	138-day NOEC (survival)	7.8–8.2	12.9	US EPA 1976
Bluegill (<i>Lepomis macrochirus</i>)	126-day NOEC (reproduction)	7.6–8.0	0.4	Smith et al. 1976; US EPA 1976
Bluegill (<i>Lepomis macrochirus</i>)	97-day LOEC (reproduction)	7.6–8.0	1.0*	Smith et al. 1976; US EPA 1976
Goldfish (<i>Carassius auratus</i>)	430-day LOEC (final weight)	7.57–7.63	9.0	US EPA 1976
Freshwater tropical fish (<i>Mystus nemurus</i>)	6-week LOEC (decreased growth rate and decreased liver somatic index)	6.9–7.5	0.5	Hoque et al. 1998
Sea urchin (<i>Lytechinus</i>)	49-day LOEC (decreased	8.0	1.12	Thompson et al. 1991

<i>pictus</i>)	wet weight)			
White shrimp (<i>Metapenaeus monoceros</i>)	48-hour LC50	8.0–8.4	8.7	Kang and Matsuda 1994
White shrimp (<i>Metapenaeus monoceros</i>) – juvenile	48-hour LC50	8.0–8.2	18.5	Kang and Matsuda 1994

^a For freshwater tests, water hardness was within the accepted range (50–250 mg/L as Ca-CO₃).

^b Studies did not always indicate whether reported pH values represented mean values.

* Value selected as the critical toxicity value (CTV) for calculating the predicted no-effect concentration (PNEC) for the aquatic exposure scenarios in the ecological risk characterization section

Hydrogen sulfide in sediments has the potential to harm benthic organisms, and it has been suggested that it may be responsible for some unintended toxicity in sediment bioassays (Wang and Chapman 1999). However, when released to water, hydrogen sulfide is expected to be removed rapidly by both oxidation and volatilization.

Consequently, very little exposure of benthos is likely to result from anthropogenic releases to water. Sulfides that are found in sediments are typically produced *in situ* by sulfate-reducing bacteria during the decomposition of organic materials. As a result, micromolar to millimolar levels of sulfide have been measured in natural fresh and marine porewater (Wang and Chapman 1999). Because it is present in sediments primarily due to natural processes, the toxicity of hydrogen sulfide to benthos is not evaluated in this assessment.

9.1.2 Terrestrial compartment

9.1.2.1 Plants

The effects of hydrogen sulfide on terrestrial plants may be beneficial or harmful at low air concentrations. Beneficial effects could in some cases be due to alleviation of a sulfur nutrient deficiency. However, under controlled experimental conditions (with adequate supplies of nutrients), it is more likely an indication of hormesis, i.e., a tendency for certain potentially toxic chemicals to cause stimulatory effects associated with stress induced at low dosages (Taylor and Selvidge 1984).

Available toxicity data for terrestrial plants are reviewed in WGAQOG (2000) and Alberta Environment (2004). These reviews reported approximately 60 individual toxicity results for over 30 horticultural, agricultural and forest species. Although some studies described effects from exposures of short durations (a few hours or less), most involved longer term exposures ranging from several days up to several months.

Most of the identified studies of short duration did not report on relevant effect measures (e.g., reduced growth or survival), and exposure concentrations were unrealistically high

(> 50 000 $\mu\text{g}/\text{m}^3$). One relevant study of acceptable quality by Taylor and Selvidge (1984) examined the effects of exposure concentrations in the 208 to 2788 $\mu\text{g}/\text{m}^3$ range (6.1 $\mu\text{mol}/\text{m}^3$ to 81.8 $\mu\text{mol}/\text{m}^3$) on rates of photosynthesis in bush bean (*Phaseolus vulgaris*). Exposure occurred 5 to 7 weeks after germination in open gaseous exchange systems. Hydrogen sulfide gas was dispensed from cylinders of certified purity and diluted with nitrogen to achieve the desired concentrations. Hydrogen sulfide exposure concentrations were measured using a flame photometric sulfur gas analyzer. Rates of photosynthesis were measured at 30-minute to 1-hour intervals for a total of 6 hours. The initial (1 and 2 hour) effects of the two lowest concentrations (208 to 419 $\mu\text{g}/\text{m}^3$) were stimulatory, with rates of photosynthesis ranging from 109% to 125% of controls. Based on inspection of figure 1 from this study, the lowest concentration associated with evidence of reduced photosynthesis (an approximately 10% reduction) after exposure for 1 to 2 hours was 984 $\mu\text{g}/\text{m}^3$ (32.7 $\mu\text{mol}/\text{m}^3$). The lowest-observed-effect concentration after 6 hours of exposure was 208 $\mu\text{g}/\text{m}^3$, which caused a 15% reduction in photosynthesis relative to controls. Taylor and Selvidge (1984) reported that the relationship between photosynthesis and hydrogen sulfide dosage (defined as concentration multiplied by hours of exposure) was statistically significant, with a second degree polynomial regression accounting for approximately 82% of the observed variation in photosynthesis rates.

The overall lowest adverse effect level in a long-term study of acceptable quality was reported by Thompson and Kats (1978). These authors exposed seven mostly agricultural species to continuous uniform fumigations of hydrogen sulfide in greenhouses over periods of from 2 to about 5 months. Temperatures were maintained near ambient levels (for Duarte, California), and hydrogen sulfide was monitored with a Phillips Model 1900 hydrogen sulfide analyzer. At 100 ppb (140 $\mu\text{g}/\text{m}^3$), grape plants (*Vitis vinifera*) exhibited a statistically significant ($p = 0.05$) 30% decrease in cane dry weight relative to controls. Dry weight is considered a more reliable measure of yield than fresh weight, since the latter is partly a function of water status at the time of harvest (WGAQOG 2000). Although alfalfa (*Medicago sativa* L) exhibited a 10% yield reduction when exposed to the same hydrogen sulfide concentration, the authors reported that this result was not statistically significant. They further note that the 140 $\mu\text{g}/\text{m}^3$ concentration had a stimulatory effect on some other tested species. Exposure at the next highest test concentration of 300 ppb (420 $\mu\text{g}/\text{m}^3$) resulted in harmful effects in six of the seven species tested. The lowest tested concentration of 30 ppb (42 $\mu\text{g}/\text{m}^3$) frequently had a statistically significant stimulatory effect and was never associated with evidence of reduced yield.

Another longer-term study by Maas et al. (1987) reported similar effect levels. These authors exposed three agricultural species for two weeks. They reported a statistically significant ($p < 0.01$) 32% reduction in the fresh weight of clover (*Trifolium pratense*) and a smaller but significant 11% increase in the fresh weight of dwarf French bean (*Phaseolus vulgaris*) at an exposure concentration of 350 $\mu\text{g}/\text{m}^3$.

The available data from long term studies thus suggest that only stimulatory effects are likely in plants exposed to up to approximately 50 $\mu\text{g}/\text{m}^3$ hydrogen sulfide (Thompson and Kats 1978).

The lowest reported long-term adverse effect level of 140 $\mu\text{g}/\text{m}^3$ reported by Thompson and Kats (1978) will be used as a CTV for determination of a PNEC for terrestrial plants exposed for longer periods (i.e., weeks or months). The lowest short-term exposure effect level of 984 $\mu\text{g}/\text{m}^3$ reported by Taylor and Selvidge (1984) will be used as a CTV for determination of a PNEC for plants exposed for short periods (i.e., 1-2 hours).

9.1.2.2 Mammals and birds

Appendix B contains a summary of the health effects information for mammals (including humans) that was reviewed for the human health portion of this assessment. An inhalation-based lowest-observed-effect concentration (LOEC) of 14 mg/m^3 (10 ppm) reported by Lopez et al. (1987) was among the most sensitive laboratory-based acute inhalation results. The effect observed was a significant increase in the cellularity of nasal lavage fluid after exposure for 4 hours, although the levels returned to original levels at 20 hours post-exposure in male Fischer 344 rats. With regard to effects of longer term exposures, Dorman et al. (2000) reported that a LOEC of 14 mg/m^3 (10 ppm) was observed in adult Sprague-Dawley rats exposed for 6 hours per day, 7 days a week, for several weeks, based on decreased absolute and relative adrenal weights in males and decreased relative ovary weights in females.

The Western Interprovincial Scientific Studies Association conducted a study (WISSA 2006) to determine if chronic exposure of cattle (prenatal to 3 months postnatal) to air emissions (including hydrogen sulfide) from activities of the oil and gas industry influenced their health and reproductive behaviour in western Canada. Passive air monitors located on or near all occupied pastures and wintering areas measured hydrogen sulfide at 1100 sites from April 2001 to January 2003. An individual monthly exposure was calculated for each animal based on the air concentration at a given location and on the time that the animal spent near that location. The primary effects endpoints considered were reproductive success and development, and the secondary effects endpoints were immune system pathology and function. The potential for effects (including reduced hatching and fledgling success) to wild European starlings (*Sturnus vulgaris*) that occupied the same areas as cattle was also evaluated (WISSA 2006). Results for the beef cattle and European starlings were negative; that is, no evidence was found of associations between the measured average monthly exposures (arithmetic mean 0.24 $\mu\text{g}/\text{m}^3$ and 95th percentile 0.74 $\mu\text{g}/\text{m}^3$) and most of the health outcomes. Increased exposure to hydrogen sulfide and sulfur dioxide did result in increased heterophil/lymphocyte ratios in starlings during one year of the study. However, it was concluded that this would likely have little effect on nestling immune competence (WISSA 2006).

Additional information on the effects of hydrogen sulfide on livestock and wildlife is summarized in WGAQOG (2000). These authors note that although ambient concentrations of hydrogen sulfide have not been shown to have adverse effects on wildlife, high concentrations (usually hundreds of mg/m^3) due to accidental releases have resulted in deaths of wild animals and birds. In keeping with results from the study by Lopez et al. (1987), non-lethal effects to cattle exposed to concentrations of about $14 \text{ mg}/\text{m}^3$ (10 ppm) resulting from accidental releases (blow-out of Alberta gas wells) were reported to include runny noses and eyes, coughing and decreased feed consumption (WGAQOG 2000).

For inhalation toxicity to wild mammals, a CTV of $14 \text{ mg}/\text{m}^3$ based on the rat LOEC (Dorman et al. 2000) will be used for determination of both acute and chronic (longer-term exposure) PNECs for terrestrial mammals and birds.

9.2 Characterization of Ecological Risk

The approach taken in this ecological screening assessment was to examine scientific and technical information from various sources and to develop conclusions based on a weight-of-evidence approach and using precaution as required under CEPA 1999. Lines of evidence considered include results from conservative risk quotient calculations, as well as information on sources of release of hydrogen sulfide and its overall behaviour in the environment, including its persistence and bioaccumulation potential.

The information collected indicates that large quantities of hydrogen sulfide are released from natural sources, mainly to air. However, release from anthropogenic sources may also be significant. Anthropogenic sources of particular importance in Canada are associated with natural gas and oil production, pulp and paper manufacturing, publicly owned wastewater treatment systems, and intensive livestock operations.

In the environment, hydrogen sulfide is found mainly in the air compartment, where it can persist for relatively long periods (degradation half-life of several weeks) during cold Canadian winters. In the summer; however, it is degraded in air quickly (half-life < 1 day) by reaction with hydroxyl radicals. It is also lost rapidly (half-lives of hours or less) from oxic water by both volatilization and oxidation reactions. However, under anoxic conditions in water, sediment or soil, hydrogen sulfide has the potential to persist for relatively long periods of time.

As an inorganic gas, hydrogen sulfide is not expected to bioaccumulate. Experimental aquatic toxicity information suggests that hydrogen sulfide acts primarily as an acute toxicant and that it has the potential to cause harm to aquatic organisms at low concentrations, i.e., in the low $\mu\text{g}/\text{L}$ range in freshwater.

9.2.1 Risk quotient analysis

A risk quotient analysis that integrates known or potential exposures with known or potential adverse ecological effects was performed for five different scenarios. For these scenarios, conservative predicted environmental concentrations (PECs) were selected from Canadian monitoring data presented in the Measured Environmental Concentrations section. PNECs were determined by dividing a CTV (see Section 9.1) by an assessment factor. PEC, CTV and PNEC values used in this assessment are summarized in Table 9-2. Aquatic PECs are shown with a “less than” symbol (<) because, as explained previously, the percentage of dissolved sulfide present as free H₂S/HS⁻ in oxic water is expected to generally be much less than the 15% value assumed when the concentrations were calculated.

For the short-term air exposure scenario, a PEC of 20.9 µg/m³ corresponding to the highest 99th percentile concentration from all stations reported over the sampling period near oil sands in Alberta, from May 2007 to May 2017 was selected as a conservative estimate of short-term atmospheric exposure near anthropogenic sources (CASA 2017). PNECs were derived from CTVs based on the rat toxicity value (4-hour inhalation-based LOEC; increased cellularity of nasal lavage fluid) of 14 mg/m³ (14 000 µg/m³) from Lopez et al. (1987) and a value of 984 µg/m³ based on a toxicity value for bush bean (1- to 2-hour exposure causing approximately 10% reduced photosynthesis) reported by Taylor and Selvidge (1984). These CTVs were divided by assessment factors of 5 and 10, respectively, to account for limitations in the available effects data and for extrapolation from effects observed in the laboratory under controlled conditions to those in the field. A smaller assessment factor was used to derive the PNEC for mammals in view of the relatively mild and transient nature of the reported adverse effect. The PNECs used were 2800 µg/m³ for mammals and 98.4 µg/m³ for plants. The resulting risk quotients (RQ = PEC/PNEC) are 0.007 and 0.21, respectively, suggesting that harm to terrestrial organisms from short-term exposures to hydrogen sulfide in air is unlikely in Canada.

For the long-term air exposure scenario, a PEC of 27 µg/m³ was selected, corresponding to the highest average 1-month atmospheric concentration reported in air near an anthropogenic source (an oil and gas facility; Chepelkevitch 2009). A monthly average hydrogen sulfide concentration in air is considered to be comparable to experimental exposure periods in plant toxicity studies, i.e., in the range of 14 to 246 days. A PNEC for plants was derived from a CTV based on the chronic toxicity value (LOAEL; growth) of 140 µg/m³ for grape vines (Thompson and Kats 1978). The CTV was divided by an assessment factor of 2 to account for the limitations in the effects data available and recognizing that tested exposure concentrations below this value are more often associated with stimulatory than adverse effects on plants. A PNEC for mammals was derived from a CTV based on the chronic toxicity value (LOAEL; reduced organ weight) of 14 000 µg/m³ (10 ppm) for Sprague-Dawley rats (Dorman et al. 2000). This CTV was divided by an assessment factor of 10 to account for limitations in the available data and for extrapolation from effects observed in the laboratory under controlled conditions to those in the field. The resulting long-term PNECs are 70 µg/m³

for plants and 1400 µg/m³ for mammals. The corresponding risk quotients of 0.38 and 0.02 suggest that there is low likelihood of harm to terrestrial plants or mammals from long-term exposure to hydrogen sulfide in air in Canada.

No distinction was made between short-term and long-term exposure when characterizing either PECs or PNECs for surface water, since available information suggests that hydrogen sulfide generally acts as an acute aquatic toxicant regardless of exposure durations (Westin Solutions Inc. 2006).

Three aquatic scenarios were evaluated representing potential exposures downstream of pulp and paper mills, wastewater treatment plants, and oil sands facilities. PECs of < 0.53 µg/L and < 2.1 µg/L were used for pulp and paper facilities, representing estimated median and maximum concentrations of hydrogen sulfide in the receiving waters of 25 pulp and paper located in the United States and Canada (NCASI 2012). A second PEC of < 0.75 µg/L hydrogen sulfide was chosen on the basis of concentrations estimated downstream of a wastewater treatment plant in Quebec, representing potential exposures in surface waters receiving discharges from plants using only primary wastewater treatment methods (Environment Canada 2004). A third PEC of < 0.40 µg/L was chosen to represent potential concentrations downstream of an oil sands facility. This PEC was the highest estimated concentration in six samples collected over a five-year period (2008–2011). In order to derive a PNEC, a CTV of 1 µg/L was selected on the basis of evidence of harmful chronic effects to aquatic species, as described previously in the Ecological Effects Assessment section. Given the relatively large effects database and recognizing that hydrogen sulfide occurs naturally at concentrations that are likely less than about 0.1 µg/L in Canadian surface waters, an assessment factor of 1 was applied, giving a PNEC of 1.0 µg/L. The determination of the aquatic PNEC considered is reasonably above known naturally occurring levels of hydrogen sulfide in Canadian surface waters (0.067–0.1 µg/L) (Alberta Environment 2004c). The resulting conservative risk quotients are generally less than 1.0, indicating that there is little potential for harm to aquatic organisms downstream of anthropogenic sources releasing dissolved sulfides to surface waters in Canada. Although the maximum risk quotient associated with pulp and paper facilities is above 1.0, the exceedance is relatively small. Because of this, and because actual percentages of sulfide that are in the form of free H₂S/HS⁻ are expected to be much less than the value of 15% assumed when calculating aquatic PECs, it is considered very unlikely that risk quotients based on more realistic estimates of H₂S/HS⁻ percentages would be causing harm at any of the examined pulp and paper mills.

Overall, this information suggests that hydrogen sulfide released to air or water from anthropogenic sources is unlikely to cause harm to aquatic organisms in Canada.

Table 9-2. Risk quotients for hydrogen sulfide*

Scenario	Organism	CTV	AF	PNEC	PEC**	RQ** (PEC/PNEC)
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Short term exposure in air - near a oil sands plant	Mammals	14 000 $\mu\text{g}/\text{m}^3$	5	2800 $\mu\text{g}/\text{m}^3$	20.9 $\mu\text{g}/\text{m}^3$	0.007
	Plants	984 $\mu\text{g}/\text{m}^3$	10	98.4 $\mu\text{g}/\text{m}^3$	20.9 $\mu\text{g}/\text{m}^3$	0.21
Long term exposure in air - near an oil and gas facility	Mammals	14 000 $\mu\text{g}/\text{m}^3$	10	1400 $\mu\text{g}/\text{m}^3$	27 $\mu\text{g}/\text{m}^3$	0.02
	Plants	140 $\mu\text{g}/\text{m}^3$	2	70 $\mu\text{g}/\text{m}^3$	27 $\mu\text{g}/\text{m}^3$	0.38
Exposure to un-ionized hydrogen sulfide in surface water downstream from pulp and paper mills	Freshwater fish and aquatic invertebrates	1.0 $\mu\text{g}/\text{L}$	1	1.0 $\mu\text{g}/\text{L}$	median < 0.53 $\mu\text{g}/\text{L}$ maximum < 2.1 $\mu\text{g}/\text{L}$	median < 0.53 maximum < 2.1
Exposure in surface water downstream from a wastewater treatment system	Freshwater fish and aquatic invertebrates	1.0 $\mu\text{g}/\text{L}$	1	1.0 $\mu\text{g}/\text{L}$	< 0.75 $\mu\text{g}/\text{L}$	< 0.75
Exposure in surface water downstream from an oil sands facility	Freshwater fish and aquatic invertebrates	1.0 $\mu\text{g}/\text{L}$	1	1.0 $\mu\text{g}/\text{L}$	< 0.40 $\mu\text{g}/\text{L}$	< 0.40

* Abbreviations: AF, assessment factor; CTV, critical toxicity value; PEC, predicted environmental concentration; PNEC, predicted no-effect concentration; RQ, risk quotient

** A "less than" symbol (<) is used because the percentage of dissolved sulfide present as free $\text{H}_2\text{S}/\text{HS}^-$ is expected to generally be much less than the 15% value assumed when calculating PECs for water.

9.2.2 Uncertainties in evaluation of ecological risk

The principal sources of uncertainty in this evaluation relate to the assessment of exposures of and effects to aquatic organisms.

One source of uncertainty relates to the measurement of hydrogen sulfide and involves the method for determining concentrations of un-ionized hydrogen sulfide in freshwater. Concentrations of hydrogen sulfide are normally estimated on the basis of measured concentrations of dissolved sulfide and on information on pH and sometimes temperature of the receiving waters. In so doing, it is normally assumed that most of the

measured dissolved sulfide is in the form of the free bisulfide (HS^-) ion or un-ionized hydrogen sulfide. However, results of a recent study of sulfide speciation in oxic surface water suggest that at least 85% of dissolved sulfide concentrations measured using standard methods is present as dissolved iron sulfides—principally FeS and FeSH^+ —and that no more than 15% could be in the form of free $\text{H}_2\text{S}/\text{HS}^-$. In this assessment, reported dissolved sulfide levels have consequently been multiplied by a factor of 0.15 when estimating concentrations of un-ionized hydrogen sulfide. The 15% value for un-ionized hydrogen sulfide should, however, be considered only a rough upper-bound estimate. In fact, given the inherent instability of hydrogen sulfide in oxic water and since available data suggest that dissolved iron concentrations in oxic surface water and wastewater are typically higher than those of dissolved sulfide, the actual percentages of dissolved sulfide present as $\text{H}_2\text{S}/\text{HS}^-$ are expected to generally be much less than 15%. This means that the measured $\text{H}_2\text{S}/\text{HS}^-$ concentrations reported are likely overestimations of un-ionized hydrogen sulfide.

Another potential source of uncertainty relates to the technical difficulties associated with sampling and accurately measuring aqueous concentrations of dissolved sulfides. For example, much of the older data (late 1970s to early 1990s) available for dissolved sulfides in Alberta rivers was unusable because of the high detection limits of the analytical methods employed. In addition, when method detection limits are acceptable, there is the challenge of avoiding loss of sulfide from sampled water prior to chemical analysis (e.g., by volatilization, oxidation or adsorption to container walls). As a consequence, although there is no evidence that such losses have occurred in the data reviewed, there is a chance that concentrations of hydrogen sulfide in Canadian surface water may in some cases have been underestimated. However, this uncertainty is counter-balanced by the approach used to estimate $\text{H}_2\text{S}/\text{HS}^-$ described above.

While there are sufficient environmental concentration data for hydrogen sulfide (measured as dissolved sulfide) in Canadian surface waters and effluents, there are still some limitations in the available data set. For example, although very high sulfide concentrations were measured in the outflow from a bioreactor at an Alberta coal mine, no information was available on concentrations in nearby surface water. Furthermore, data on dissolved sulfide concentrations in surface water downstream of oil sands operations were only identified at one location in Alberta. It is not clear how representative this location is, or how sulfides are released to water from the oil sands facility. In addition, most of the data for surface water associated with pulp and paper mills were for facilities located in the United States.

Regarding the assessment of exposure of terrestrial organisms, there is a relatively large amount of credible short-term (1 hour) data available on concentrations in Canadian air near anthropogenic sources of release. Longer term (one month) exposure information is less abundant and consequently there is greater uncertainty associated with estimation of the maximum long-term terrestrial PEC. The assessment factors used to derive the terrestrial PNECs from the CTVs are considered adequate to address uncertainties related to the limitations of the toxicity data available. The lowest

assessment factor of 2, used to derive a long-term PNEC for plants of $70 \mu\text{g}/\text{m}^3$, is considered justified in view of the stimulatory effects of hydrogen sulfide observed in many plant species when exposed at low levels.

10. Potential to Cause Harm to Human Health

10.1 Exposure Assessment

10.1.1 Environmental media

Hydrogen sulfide is a diprotic acid in equilibrium with two anionic forms, namely the bisulfide (HS^-) and sulfide (S^{2-}) ions. At pH values relevant to environmental waters (e.g., pH of 6 to 9), hydrogen sulfide and the bisulfide anion will be the predominant species. Alkali metal salts, such as sodium bisulfide ($\text{Na}(\text{SH})$) and sodium sulfide (Na_2S), are readily soluble in water and have the potential for release to environmental waters from industrial processes. If released to water, sodium bisulfide and sodium sulfide would dissociate to form the anions HS^- and S^{2-} , respectively. These anions would enter the natural sulfur cycle and, depending on the pH, result in the formation of hydrogen sulfide via the equilibrium mentioned above. Therefore, the focus of this assessment of environmental exposure is on hydrogen sulfide (H_2S).

Also, as noted earlier, 60 to 90% of hydrogen sulfide in the atmosphere is estimated to be the result of natural sources (US EPA 1993; Watts 2000). In addition, a comparison of the total import volume of sodium sulfides in Canada for the 2015 calendar year, namely 9 217 213 kg, with the total volume of hydrogen sulfide manufactured (including incidentally) in the 2000 calendar year, namely 8 670 000 000 kg, suggests that any releases resulting from the commercial use of either of the two sulfides of sodium would contribute only a minor fraction to total hydrogen sulfide levels in the environment (Environment Canada 2004a; StatsCan 2015).

Hydrogen sulfide is part of the natural sulfur cycle and has many natural sources, including volcanoes, sulfur springs and petroleum deposits. It has been detected in well water in Canada and in groundwater in other countries within close proximity to pulp and paper mills and petroleum refineries (US EPA 2003; ATSDR 2006). While not quantified, hydrogen sulfide has been detected in hot tap water in some homes, resulting in a musty or rotten egg smell. Although hydrogen sulfide can be found in water under specific environmental conditions, the majority is released to air, and inhalation of ambient air is expected to be the primary route of human exposure.

Empirical data on measured concentrations of hydrogen sulfide in ambient air identified from the published literature are presented in section 7, Measured Environmental Concentrations, and in Appendix A.

The reported value of 1 ppb ($1.4 \mu\text{g}/\text{m}^3$) as the average concentration found in urban areas away from point sources (Alberta Environment 2000a) is considered to be a

conservative representation of the potential concentration to which the general population could be exposed. Numerous measurements of hydrogen sulfide concentrations in ambient air near point sources have been reported, including pulp and paper mills, oil and gas fields, natural gas processing facilities, petroleum refineries, and livestock farms, constituting a large data set of millions of samples collected over several decades. The highest 99th percentile ambient air concentration of 31 ppb (43.4 $\mu\text{g}/\text{m}^3$) measured near a Canadian pulp and paper mill (WGAQOG 2000) is based on samples collected hourly, continuously over 4 years between 1994 and 1998. The range of all reported 99th percentile concentrations measured near point sources in Canada falls within the range of concentrations measured near this mill at that time; this value is considered to be an upper-bound conservative representation of the potential hydrogen sulfide concentration to which the general population could be exposed while living near a point source. The range of concentrations of 1–31 ppb (1.4–43.4 $\mu\text{g}/\text{m}^3$) is used in the risk characterization.

10.1.2 Consumer products

No consumer products containing sodium bisulfide or sodium sulfide were identified in Canada.

10.2 Health Effects Assessment

Limited information on the toxicity of sodium sulfide and sodium bisulfide has been identified in the literature. However, soluble sulfides (which include sodium sulfide and sodium bisulfide) are reported to be rapidly and completely hydrolyzed in body fluids to produce hydrogen sulfide. As a result, there are no toxicological distinctions between them and hydrogen sulfide in terms of their systemic effects and toxicokinetic profile (Health Canada 1987), and this section will focus on the health effects of hydrogen sulfide. The available toxicity studies conducted specifically with sodium sulfide and sodium bisulfide are summarized at the end of this section.

Appendix B contains additional details on the key health effects studies for hydrogen sulfide reviewed for this assessment.

Hydrogen sulfide is produced endogenously as part of normal biological function, playing a role in regulating blood pressure, body temperature, vascular smooth muscle, cardiac function and cerebral ischemia and in modulating the hypothalamic–pituitary–adrenal axis. It is produced by the brain, liver, heart and gastrointestinal tract (Kimura 2002; Kamoun 2004; Linden et al. 2010). Endogenous hydrogen sulfide is produced from cysteine by cystathionine β -synthase and cystathionine γ -lyase (Abe and Kimura 1996; Lu et al. 2008). In the brain, the endogenous levels of hydrogen sulfide detected range from 50 to 160 μM in humans, rats and bovines (Abe and Kimura 1996; Lu et al. 2008). Hydrogen sulfide in the gastrointestinal system has also been attributed to the metabolism of sulfhydryl-containing amino acids by bacteria present in the intestinal tract and the mouth (Abe and Kimura 1996).

10.2.1 Genotoxicity and carcinogenicity

No genotoxicity or carcinogenicity classifications by other national or international regulatory agencies were identified. No long-term or carcinogenicity studies with hydrogen sulfide were identified. Ames tests in *Salmonella typhimurium* strains TA97, TA98 and TA100, with and without metabolic activation, indicated no mutagenic potential (Hughes et al. 1984). Mixed results were observed in *in vitro* comet assays depending on cell types and on whether DNA repair system is active. Positive results were observed in human small intestine cells (Attene-Ramos et al. 2010) but not in Chinese hamster ovary (CHO) cells nor human colonic cancer cells when DNA repair system was active (Attene-Ramos et al. 2006). When DNA repair was inhibited, positive results were observed in both CHO cells and human colonic cancer cells (Attene-Ramos et al. 2006).

In vitro toxicogenomic analysis in human intestinal epithelial cells showed that hydrogen sulfide can modulate gene expression involved in cell cycle and can trigger inflammatory, DNA damage and DNA repair responses (Attene-Ramos et al. 2010). In rats exposed nose-only to 200 ppm (280 mg/m³) hydrogen sulfide for 3 hours per day for 1 day or for 5 consecutive days, both nasal pathology and gene expression profiles of the nasal respiratory epithelial cells were examined (Roberts et al. 2008). Nasal pathology showed an initial mild respiratory epithelial injury with mild inflammation and loss of the basal cellular structure. One day post-exposure, respiratory epithelial regeneration occurred. The effect was reversible as complete recovery was observed in all animals after exposed for 5 consecutive days. In terms of gene expression profiles, early changes were involved with cellular defence/inflammation, and later changes in gene expression were associated with cellular proliferation and microtubule-based movement. Together, results from genomic and pathology suggest that exposure to hydrogen sulfide may cause acute injury to the nasal respiratory epithelium; however, the effect is reversible as the respiratory epithelium can rapidly be repaired and become resistant to further damage.

The predictive quantitative structure–activity model DEREK (2009) did not identify any chemical structural features of concern for hydrogen sulfide. Some other quantitative structure–activity models could not be performed on the substance because of its inorganic nature (TOPKAT 2004; CASETOX 2009; Toxtree 2009; Model Applier 2010).

10.2.2 Odour threshold

Hydrogen sulfide is very odorous, with a low olfactory threshold, from less than 0.01 to 0.3 ppm (0.014 to 0.42 mg/m³). There is uncertainty associated with determination of a specific odour threshold, as it varies with individual sensitivity (WHO 2000; Greenberg et al. 2013). The median odour detection threshold for hydrogen sulfide reported by Amoore and Hautala (1983), based on a compilation of 25 published reports of odour threshold, is 0.008 ppm (0.011 mg/m³). Amoore and Hautala (1983) also reported that a 250-fold range in odour sensitivities would be likely in a group of 100 observers. Although hydrogen sulfide can be perceived as a nuisance in a community setting

because of its smell, there is insufficient evidence that it causes adverse health effects at those low levels (Logue et al. 2001; Horton et al. 2009).

After prolonged exposure, olfactory fatigue can occur, where the sensory system is adapted to the smell of hydrogen sulfide. At high concentrations (100–200 ppm [140–280 mg/m³]), hydrogen sulfide paralyzes the olfactory nerve, preventing odour detection (Reiffenstein et al. 1992; Guidotti 1994).

10.2.3 Ocular effects

A threshold for human eye irritation was observed at 10–20 ppm (15–30 mg/m³) and serious eye damage was observed at 50–100 ppm (70–140 mg/m³) in a study by Savolainen (1982). The WHO air quality guideline value for hydrogen sulfide is 0.15 mg/m³ (0.11 ppm) for an average concentration over 24 hours based on eye irritation (WHO 2000; IPCS 2003). In occupational settings, workers exposed to 10.71–20.71 ppm (15–29 mg/m³) hydrogen sulfide for 6 to 7 hours have reported eye irritation (IPCS 1981). Riffat et al. (1999) reported that exposure to hydrogen sulfide at concentrations greater than 50 ppm (70 mg/m³) for 1 hour can severely damage eye tissue. In a community around a paper mill where an annual mean concentration of 6 µg/m³ hydrogen sulfide, with daily peak concentrations up to 100 µg/m³, was recorded, eye irritation was reported 12 times more frequently than in communities without exposure (Jaakkola et al. 1990). However, co-exposure to methyl mercaptan and methyl sulfide also occurred. In an occupational survey, Vanhoorne et al. (1995) noted that a significantly higher number of eye irritation complaints were reported by workers exposed to hydrogen sulfide concentrations greater than 5 mg/m³. However, the workers were also exposed to carbon disulfide.

10.2.4 Respiratory effects

In laboratory animals following acute inhalation exposure, the lowest LOEC was 10 ppm (14 mg/m³), identified in Dorman et al. (2002) and Lopez et al. (1987). In the Dorman et al. (2002) study, the LOEC was based on a significant decrease in cytochrome oxidase activity in the liver of Sprague-Dawley rats exposed to 0, 10, 30, 80, 200 or 400 ppm (0, 14, 42, 110, 280 or 560 mg/m³) hydrogen sulfide for 3 hours. In the Lopez et al. (1987) study, the LOEC was based on a significant transient increase in the cellularity of nasal lavage fluid, which returned to original levels at 20 hours post-exposure, in male Fischer 344 rats exposed to 0, 10, 200 or 400 ppm (0, 14, 280 or 560 mg/m³) hydrogen sulfide for 4 hours.

With regard to short-term repeated inhalation exposure, the lowest LOEC was 10 ppm (14 mg/m³) (no-observed-effect-level [NOEC] = 1 ppm or 1.4 mg/m³), based on significantly reduced cytochrome oxidase activity in lung mitochondria in male Fischer 344 rats exposed to 0, 1, 10 or 100 ppm (0, 1.4, 14 or 140 mg/m³) hydrogen sulfide 8 hours/day, 5 days/week, for 5 weeks (Khan et al. 1998).

For longer-term inhalation exposure, a no-observed-adverse-effect concentration (NOAEC) of 10 ppm (14 mg/m³) was identified with a lowest-observed-adverse-effect

concentration (LOAEC) of 30 ppm (42 mg/m³) in Brenneman et al. (2000) and Dorman et al. (2004). Brenneman et al. (2000) exposed male Sprague-Dawley rats to 0, 10, 30 or 80 ppm (0, 14, 42 or 110 mg/m³) hydrogen sulfide for 6 hours/day, 7 days/week, for 10 weeks. The LOAEC was based on mild to moderate olfactory neuron loss and basal cell hyperplasia in the olfactory mucosa. The Dorman et al. (2004) study was a reassessment of the nasal and lung histopathology from the Chemical Industry Institute of Toxicology (CIIT 1983a,b,c) studies. In the CIIT (1983a,b,c) studies, Fischer 344 rats, Sprague-Dawley rats and B6C3F1 mice were exposed to 0, 10.1, 30.5 or 80 ppm (0, 14, 42 or 110 mg/m³) hydrogen sulfide for 6 hours/day, 5 days/week, for 90 days. A LOEC of 80 ppm was identified primarily on the basis of decreased feed consumption and body weights. Dorman et al. (2004) reassessed the nasal and lung histopathology from the CIIT (1983a,b,c) studies and identified a significant increase in the incidence of olfactory neuronal loss at 30 ppm and higher in all animals except male Sprague-Dawley rats. For male Sprague-Dawley rats, a significant olfactory neuronal loss was observed at 80 ppm. No chronic inhalation studies were identified. IPCS (2003), US EPA (2003) and ATSDR (2006) used the Brenneman et al. (2000) study to derive a medium-term tolerable concentration, an inhalation reference concentration and an intermediate-duration inhalation minimal risk level, respectively.

Dorman et al. (2004) noted that there are significant differences between the breathing styles and nasal anatomy of rodents and humans. Rodents such as mice and rats are obligatory nasal breathers. A large portion of the rodent nasal cavity is lined by olfactory mucosa (50%) relative to humans (10%). In addition, the structure of the nasal cavity of rodents allows a greater surface area to be exposed to inhaled chemicals in a slower speed of air flow for more efficient chemical uptake than in humans. Combining these factors increases the probability that a chemical inhaled in the rodent nose will deposit in the olfactory mucosa for a longer duration that is sufficient to cause toxicity and, with time, could result in irreversible lesions.

Respiratory effects in human subjects have also been studied.

The Bhambhani research group conducted several studies on the effects of hydrogen sulfide on healthy human subjects and demonstrated that exposure for 15 to 30 minutes via "oral inhalation" during various exercise levels to concentrations up to 10 ppm (14 mg/m³) did not result in adverse effects. In two studies, healthy volunteers were exposed to 0, 0.5, 2 or 5 ppm (0, 0.7, 2.8 or 7 mg/m³) hydrogen sulfide via oral inhalation during graded cycle exercise until exhaustion (Bhambhani and Singh 1985, 1991). In the 1985 study, 16 male subjects were ranked on the basis of their relative maximum oxygen uptake and categorized into "high fit" and "low fit" groups. A significant decrease in respiratory exchange ratio (RER) was observed at 0.5 ppm in the "low fit" group and at 5 ppm in the "high fit" group at the maximum exercise level. Female subjects were not classified into fitness groups due to small group size, and a significant reduction in RER was observed at 5 ppm at all exercise levels. In Bhambhani and Singh (1991), a significant decrease in RER was observed at 2 ppm in 16 healthy male volunteers exercising at the maximum level. Although statistically significant

changes in RER were observed at 2 ppm, no significant pulmonary effects were observed by the authors. The authors concluded that healthy individuals can safely exercise at their maximum metabolic rates while exposed to 5 ppm hydrogen sulfide.

Bhambhani and co-workers later focused on health effects at 50% of a predetermined maximal aerobic exercise level. No significant physiological or pulmonary effects were observed when healthy volunteers were exposed to 0 or 5 ppm (0 or 7 mg/m³) hydrogen sulfide while exercising at 50% of a predetermined maximal aerobic level for 30 minutes (Bhambhani et al. 1994, 1996b). A significant decrease in muscle citrate synthase levels was observed in men but not in women at 5 ppm. Bhambhani et al. (1996a, 1997) further tested the effect of hydrogen sulfide at 10 ppm (14 mg/m³) in healthy volunteers exercising at 50% of a predetermined maximal aerobic level after 15 or 30 minutes of exposure. In the group exposed for 15 minutes, no significant effects on pulmonary functions were observed (Bhambhani et al. 1996a). In the group exposed for 30 minutes, a significant decrease in oxygen uptake and a significant increase in the RER and blood lactate levels were observed (Bhambhani et al. 1997). A non-statistically significant increase in muscle lactate levels and a non-statistically significant decrease in muscle citrate synthase activity were also observed. From these studies, the authors concluded that oral inhalation of hydrogen sulfide up to 10 ppm (14 mg/m³) did not significantly alter pulmonary function in healthy individuals.

Participants in the above-mentioned studies were exposed while exercising to exhaustion, a scenario that is representative of an occupational setting but not of the typical activity levels for the general population. It does, however, indicate that at typical activity levels, exposure to hydrogen sulfide up to 10 ppm (14 mg/m³) would not compromise pulmonary function in healthy individuals.

In an earlier study, Jappinen et al. (1990) examined possible respiratory effects associated with hydrogen sulfide in 26 male pulp mill workers and in 10 asthmatic volunteers. In the cohort of non-asthmatic mill workers exposed to hydrogen sulfide in the workplace with exposure levels ranging from 1 to 11 ppm, standard histamine challenges were performed after a holiday or one day away from work and at the end of the work day. There were no statistically significant effects on respiratory function or bronchial reactivity observed when comparing responses obtained after a holiday or one day away from work with responses obtained at the end of the work day. In the volunteer study, the asthmatic volunteers were exposed to 2 ppm (2.8 mg/m³) hydrogen sulfide, which represented one fifth of the Finnish maximum allowable workplace concentration of 10 ppm, for 30 minutes in an exposure chamber. In this part of the study, respiratory measurements were compared before and after exposure. When the asthmatic subjects were exposed to 2 ppm hydrogen sulfide, 3 out of 10 hydrogen sulfide-exposed subjects reported headaches, and measurement of airway resistance increased. As a group, airway resistance increased 26.3% on average, and specific airway conductance decreased by an average of 8.4%, which while not statistically significant, is considered biologically significant. Thus, the effect level for increased airway resistance in asthmatics was 2 ppm (2.8 mg/m³) hydrogen sulfide.

Both the ATSDR (2006) and IPCS (2003) used the Jappinen et al. (1990) study to derive the acute-duration inhalation minimal risk level and the short-term tolerable concentration, respectively, while acknowledging the limitations of the study. In February 2010, the American Conference of Governmental Industrial Hygienists (ACGIH) lowered the recommended workplace threshold limit value (TLV, 8 h/day) for hydrogen sulfide from 10 ppm to 1 ppm (ACGIH 2010).

More recently, Bates et al. (2013) examined any potential association of self-reported asthma and asthma symptoms in a population living in Rotorua, New Zealand, with chronic exposure to hydrogen sulfide from geothermal sources. A total of 1637 men and women (aged 18 to 65) who had resided in Rotorua for at least 3 years participated. Participants filled out a questionnaire with questions related to residential and workplace histories, doctor-diagnosed medical conditions, including asthma, and respiratory symptoms in the last 12 months. Hydrogen sulfide exposure levels of the participants at homes and workplaces were estimated from hydrogen sulfide levels collected from more than 50 city-wide sampling sites over 2 weeks in the summer and winter of 2010 and range from 0 to 64 ppb. Participants were grouped into 4 quartiles (0-10 ppb, 11-20 ppb, 21-30 ppb and 31-64 ppb), and no increased asthma risk was found with hydrogen sulfide exposure.

10.2.5 Neurological effects

Effects of hydrogen sulfide exposure on the behaviour of experimental animals were examined in several studies. In rats exposed to 100 or 200 ppm (139 or 280 mg/m³) hydrogen sulfide for 1 to 2 hours, a LOAEC of 200 ppm was observed based on a significant decrease in discriminated avoidance response (Higuchi and Fukamachi 1977). The lowest NOAEC following short-term inhalation exposure of test animals identified in the literature was 30 ppm (42 mg/m³), with a LOAEC of 80 ppm (110 mg/m³) based on a significant reduction in spontaneous motor activity and body temperature in rats exposed (nose-only) to 0, 30, 80, 200 or 400 ppm (0, 42, 110, 280 or 560 mg/m³) hydrogen sulfide for 3 hours/day for 5 consecutive days (Struve et al. 2001). Struve et al. (2001) analyzed the brains for catecholamines and found that there were no exposure-related decreases in brain catecholamine levels in either the striatum, hindbrain or hippocampus following exposures up to 400 ppm. In addition, learning and memory were not impaired following exposures (whole-body) up to 80 ppm hydrogen sulfide for 5 days as evaluated using a modification of the Morris water maze protocol.

Fiedler et al. (2008) conducted a human exposure study with 74 healthy subjects (35 females, 39 males) exposed to 0.05, 0.5 and 5 ppm (0.07, 0.7 and 7 mg/m³) hydrogen sulfide in a random order for 2 hours over 3 weeks in an exposure chamber. The authors reported that although some symptoms, such as decreased odour detection and increased irritation and anxiety, were statistically different with exposure over time, the magnitude of these changes were minor. A significant decline in cognitive recall through auditory verbal learning was observed at all exposure levels over time and the authors suggested that the decline in verbal learning could be due to fatigue. However,

the authors noted that a threshold effect was not consistently observed for other neurobehavioural measures, as no significant effects on other sensory or cognitive measures, such as complex reaction time, were observed in a dose-response manner. Thus, Fiedler et al. (2008) reported that up to 5 ppm hydrogen sulfide had statistically significant effects of minor magnitude in healthy individuals. However, according to the authors the exposure dose range was within the range of anticipated general population exposures; therefore, they could not identify a no-adverse-effect level.

Of note, this acute 2-hour inhalation effect level of 5 ppm for neurological effects in healthy human volunteers (Fiedler et al. 2008) is of the same order of magnitude as the acute inhalation effect level of 2 ppm based on bronchial reactivity in asthmatic human volunteers (Jappinen et al. 1990).

In epidemiological studies, the Kilburn research group studied the neurological effects of humans exposed to hydrogen sulfide (Kilburn and Warshaw 1995; Kilburn 1997, 1999, 2003). Neurobehavioural effects were evaluated in individuals who had been exposed for various lengths of time to low-level environmental hydrogen sulfide concentrations. A number of neurobehavioural effects, possibly associated with hydrogen sulfide exposure, were identified, which included alterations in balance, visual fields, choice reaction time, colour discrimination, grip strength and delayed verbal recall. In some cases, exposure levels were not reported; in others, exposure levels were estimated. Some subjects were also concurrently exposed to other substances.

The same subjects who live in the city of Rotorua, New Zealand, with chronic exposure to hydrogen sulfide from geothermal sources described in Bates et al. (2013), were also administered a series of neuropsychological tests (Reed et al. 2014) to examine any association between chronic, low-level exposures to hydrogen sulfide and cognitive function. The neuropsychological tests evaluate attention, memory, psychomotor speed, fine motor function and mood. Two sets of metrics for hydrogen sulfide exposure were used for comparison. One set was based on current exposure, with the 4 quartiles (0-10 ppb, 11-20 ppb, 21-30 ppb and 31-64 ppb) of hydrogen sulfide exposure estimates used in Bates et al. (2013). Another set was a long-term exposure estimate where hydrogen sulfide exposure over the last 30 years was modeled on the basis of self-reported residential, workplace and school locations and assuming little change in hydrogen sulfide sources over the last 30 years. Overall, no association was identified between hydrogen sulfide exposure and cognitive function in this population residing in Rotorua city with chronic, low-level exposure to hydrogen sulfide.

In human case studies, which were usually occupational or accident-related, concentrations and durations of exposure to hydrogen sulfide were usually not quantified, and co-exposure to other chemicals was often the case. Some neurological effects observed included coma, seizures, dizziness, dementia, decreased ability to communicate, decreased attention and concentration, memory impairment, impaired visual perception and coordination, impaired motor function, ataxia, cerebral atrophy, and irritability (Allyn 1931; Ahlborg 1951; McDonald and McIntosh 1951; Spolyar 1951;

Breyse 1961; Milby 1962; Krekel 1964; Adelson and Sunshine 1966; Thoman 1969; Simson and Simpson 1971; Burnett et al. 1977; Osbern and Crapo 1981; Hagley and South 1983; Beauchamp et al. 1984; Arnold et al. 1985; Audeau et al. 1985; Deng and Chang 1987; Luck and Kaye 1989; Wasch et al. 1989; NIOSH 1991; Parra et al. 1991; Tvedt et al. 1991a,b; Kilburn 1993; Snyder et al. 1995; Hall and Rumack 1997; Watt et al. 1997).

10.2.6. Reproductive and developmental effects

No reproductive or developmental classifications by other national or international regulatory agencies were identified. A no-observed-effect concentration (NOEC) of 80 ppm (110 mg/m³) was identified in male and female Sprague-Dawley rats exposed to 0, 10, 30 or 80 ppm (0, 14, 42 or 110 mg/m³) hydrogen sulfide for 6 hours/day, 7 days/week (Dorman et al. 2000). Exposure began 2 weeks prior to mating. For pregnant rats, exposure continued during a 2-week mating period and then from gestation days 0 through 19. Exposure of dams and pups resumed between postnatal days 5 and 18. For male rats, exposure started from 2 weeks prior to mating and continued for 70 consecutive days. No reproductive toxicity was observed in the exposed male and female F₀ rats. No developmental toxicity was observed in the pups. There were no significant effects observed in pup growth, development, behavioural performance or neuropathology. Behavioural tests included motor activity, passive avoidance, functional observation battery and acoustic startle response.

In terms of neurodevelopmental effects, Hannah and Roth (1991) examined the perinatal effect of hydrogen sulfide on developing cerebellar Purkinje cells in rat pups. A LOEC of 20 ppm (28 mg/m³) was identified on the basis of significant alterations in the architecture and growth characteristics of the Purkinje cell dendritic fields in pups when pregnant Sprague-Dawley rats were exposed to 0, 20 or 50 ppm (0, 28 or 70 mg/m³) hydrogen sulfide for 7 hours/day from gestation day 5 to postnatal day 21. However, the US EPA (2003) questioned whether these alterations could be seen as adverse, as “the effects reported are highly selective and could be due to environmental factors not directly related to exposure including variability resulting from the restricted sampling technique (i.e., one Purkinje cell per pup).” Developmental neurochemical changes were examined by Skrajny et al. (1992). A LOEC of 20 ppm (28 mg/m³) was identified on the basis of significantly increased serotonin levels in the frontal cortex in exposed pups when pregnant rats were exposed to 0, 20 or 75 ppm (0, 28 or 105 mg/m³) hydrogen sulfide for 7 hours/day from gestation day 5 to postnatal day 21 (Skrajny et al. 1992). No evidence is available to suggest that changes in Purkinje cell dendritic fields or neurotransmitter levels would lead to toxicological alterations in neurobehavioural performance.

10.2.7 Toxicokinetics of hydrogen sulfide

Hydrogen sulfide is absorbed rapidly by the lungs and is widely distributed in the body (US EPA 2003; ATSDR 2006). Sulfide levels were detected in the liver, blood, brain, lungs, spleen and kidneys in humans who had been accidentally exposed (Kimura et al.

1994; Imamura et al. 1996) and in experimentally exposed animals (Nagata et al. 1990; Kohno et al. 1991). Similar distribution patterns were observed in humans and in experimental animals. The concentration of sulfide was highest in the heart. The level in the brain was comparable to the levels in the lung, liver, kidney and spleen. Hydrogen sulfide can be metabolized by oxidation (US EPA 2003; ATSDR 2006). Additionally, methylation and conjugation with metalloproteins are two postulated pathways. The major metabolic pathway is oxidation of the sulfide, firstly to thiosulfate and then to sulfate (Bartholomew et al. 1980; Beauchamp et al. 1984). The major oxidation site is the liver, and excretion of the metabolites is primarily through the kidneys. One of the postulated metabolic pathways is methylation. Weisiger et al. (1980) found that hydrogen sulfide can be methylated by the intestinal mucosa of Sprague-Dawley rats *in vitro* (Weisiger et al. 1980). This process is catalyzed by thiol S-methyltransferase. This pathway is thought to be a minor metabolic pathway, as the methylation rate is expected to be significantly slower than the oxidation rate (Levitt et al. 1999). Another postulated metabolic pathway is reaction of hydrogen sulfide with metalloproteins. This pathway was postulated primarily on the basis of limited evidence (Smith and Abbanat 1966; Beauchamp et al. 1984).

10.2.8 Toxicodynamics of hydrogen sulfide

Several mechanisms of hydrogen sulfide toxicity have been proposed for exposure at high concentrations. Some investigators suggested the involvement of a neurotoxic pathway. Others suggested that toxicity is triggered in the lung at the site of contact. One of the postulated mechanisms involved the inhibition of cytochrome oxidase, which is a critical enzyme for cellular mitochondrial respiration (Chance and Schoener 1965; Nicholls 1975; Smith et al. 1977). Inhibition of cytochrome oxidase would lead to blockage of oxidative metabolism. As the brain and the nervous system have a high oxygen demand, blockage of oxidative metabolism can lead to respiratory arrest (Warenycia et al. 1989). Other investigators have suggested that direct inhibition of cytochrome oxidase in the lung tissues is the primary pathway leading to respiratory arrest (Khan et al. 1990). Another postulated mechanism is related to the effects on nerve endings. Based on studies in rats, Almeida and Guidotti (1999) suggested that the hydrosulfide anion can act on nerve endings of the pulmonary vagi, paralyzing the ventilatory centre in the brain. The hydrosulfide anion can also directly act on neurons in the ventilatory centre in the brain *in vitro*, interfering with neurotransmission (Kombian et al. 1993).

10.2.9 Sodium sulfide and sodium bisulfide

10.2.9.1 Mutagenicity and carcinogenicity

Limited data is available for sodium sulfide and sodium bisulfide. Sodium sulfide was not mutagenic in bacterial mutation assays using *Salmonella typhimurium* strains TA98, TA100, TA1535 and TA1537, with and without metabolic activation (conducted according to the Organisation for Economic Co-operation and Development Test Guideline [OECD TG] 471). Negative results were also observed in a mammalian

lymphoma cell mutation assay (OECD TG 476) using mouse lymphoma L5178Y cells exposed to sodium sulfide at doses up to 781 µg/ml, with and without metabolic activation (ECHA 2016a; NICNAS 2016).

One *in vivo* assay was identified for sodium sulfide. Negative results were noted for micronucleus induction in bone marrow cells of NMRI mice administered sodium sulfide intraperitoneally at doses of 24, 48 or 96 mg/kg bw (ECHA 2016a; NICNAS 2016).

No carcinogenicity studies are available except for one limited study conducted with sodium sulfide. In this study, Charles River CD rats (n=26/sex/dose) were administered sodium sulfide in water by gavage at doses of 9 and 18 mg/kg bw/day, in presence or absence of 1 percent thyroid extract, twice a week for 56 weeks and two to three times a week for an additional 22 weeks (for a total of 78 weeks). A significant association between mortality and dose was noted in treated males without the 1 percent thyroid extract. However, this association was not found in the other group treated with sodium sulfide and the thyroid extract. While females treated with sodium sulfide and thyroid extract exhibited a higher mortality, the significance was not specified. The results were termed “ambiguous” by the authors. No evidence for the carcinogenicity of sodium sulfide was found in this study (Weisburger et al. 1981; Health Canada 1987).

10.2.9.2 Repeated-dose toxicity

Limited information is available for Na₂S and NaSH. In one study, Yorkshire pigs (n=144) were fed diets containing 0, 225, or 450 ppm sodium sulfide (equivalent to 0, 6.75 or 13.5 mg/kg bw/day using a dose conversion by Health Canada 1994) for 104 days. No signs of toxicity were observed (Cromwell et al. 1978).

Under physiological pH, these substances will dissociate into hydrogen sulfide anions (HS⁻) and hydrogen sulfide. Under acidic conditions (such as in the stomach), the formation of hydrogen sulfide is more important (Meyer et al. 1983; NICNAS 2016). Adverse health effects observed after repeated oral exposure cannot be ruled out (NICNAS 2016).

No dermal or inhalation studies are available for Na₂S or NaSH. Repeated exposure to these chemicals could lead to lung effects and nasal damage due to the release of hydrogen sulfide (NICNAS 2016).

10.2.9.3 Reproductive and developmental effects

No data is available for sodium sulfide or sodium bisulfide. Based on the available data for hydrogen sulfide, no reproductive and developmental toxicity is expected for these two substances.

10.2.9.4 Skin and eye irritation

Considering the high basicity of the sulfide anions, sulfides such as sodium sulfide and sodium bisulfide are expected to have severe irritation/corrosive properties (NICNAS 2016). Sodium bisulfide is reported as a strong irritant to skin and mucous membranes

(HSDB, 2003). A 30% sodium bisulfide solution applied into the conjunctival sac of the eyes of six Himalayan rabbits caused irreversible eye damage (ECHA 2016b).

10.3 Characterization of Risk to Human Health

Sodium bisulfide and sodium sulfide, if released to the environment via commercial activities, are anticipated to result in the formation of the sodium cation and the bisulfide or sulfide anions, respectively. Since such anions are in equilibrium with hydrogen sulfide, sodium bisulfide and sodium sulfide are expected to contribute to total hydrogen sulfide levels in the environment (albeit to a minor extent relative to other sources). If exposures to humans were to occur to undissociated sodium bisulfide or sodium sulfide, either salt would rapidly and completely hydrolyze in bodily fluids to result in the formation of hydrogen sulfide. No specific additional hazard is associated with either salt beyond that of hydrogen sulfide (Health Canada 1987). This section will therefore focus on characterizing the human health risk associated with exposure to hydrogen sulfide.

Hydrogen sulfide is produced endogenously at low concentrations as part of normal biological function, playing a regulatory role in mammalian physiology, and it is thus normally present in mammals, including humans (Mancardi et al. 2009). No mutagenicity or carcinogenicity classifications by other national or international regulatory agencies were identified, and no data were identified to suggest that hydrogen sulfide is mutagenic or carcinogenic.

The predominant route of exposure to hydrogen sulfide for Canadians is through inhalation of ambient air. Hydrogen sulfide in ambient air comes from both natural and anthropogenic sources, and a review of the available monitoring data reveals that a representative upper-bounding range of ambient air concentrations to which the general population would be exposed is 0.001–0.031 ppm (0.0014–0.0434 mg/m³). The lowest value of this range represents the overall average concentration measured in an urban area presumed to be away from major anthropogenic sources (Alberta Environment 2000a); the highest value of the range is the highest of all 99th percentile concentrations derived for each of 64 sites monitored near Canadian pulp and paper mills (WGAQOG 2000).

Cigarette smoke is a source of hydrogen sulfide. Although smoking does not provide an appropriate basis on which to assess the risk to the general population, given its use by some individuals, the additional intake of hydrogen sulfide from cigarette smoke would reduce the margin of exposure.

10.3.1 Odour threshold

Hydrogen sulfide is very odorous, with a low olfactory threshold, from less than 0.01 to 0.3 ppm (0.014 to 0.42 mg/m³). Although hydrogen sulfide can be perceived as a nuisance in a community setting because of its smell, there is insufficient evidence that it causes adverse health effects at those low levels (Logue et al. 2001; Horton et al. 2009).

Olfactory nuisance is not considered to be adverse for the purpose of this screening assessment and therefore is not taken into account in terms of calculating a margin of exposure for hydrogen sulfide.

10.3.2 Ocular effects

The critical LOEC for ocular effects is 10–20 ppm (15–30 mg/m³) based on the human eye irritation threshold reported in Savolainen (1982). The WHO air quality guideline value for hydrogen sulfide was derived on the basis of eye irritation observed in the Savolainen (1982) study. Comparison of this LOEC with the upper-bounding range of ambient air concentrations of 0.001–0.031 ppm (Alberta Environment 2000a; WGAQOG 2000) results in a margin of exposure of 320–20 000. The margins of exposure for ocular effects are considered adequate to address uncertainties in the health effects and exposure databases for the general population.

10.3.2 Respiratory effects

An acute respiratory effect level of 2 ppm (2.8 mg/m³) was identified on the basis of biologically significant increases in airway resistance observed in asthmatic volunteers exposed to hydrogen sulfide for 30 minutes in an exposure chamber (Jappinen et al. 1990). Comparison of the respiratory effect level of 2 ppm with the upper-bounding range of ambient air concentrations of 0.001–0.031 ppm (Alberta Environment 2000a; WGAQOG 2000) results in margins of exposure ranging from 60 to 2000.

The upper-bounding ambient air concentration of 0.031 ppm is considered to be very conservative, as it is the highest 99th percentile concentration obtained from a set of numerous 1-hour measurements taken near point sources across Canada spanning several years, and this upper-bounding ambient air concentration encompasses the range of 99th percentile air levels for all point sources in the air monitoring database. This biologically significant increase in airway resistance in asthmatics in response to hydrogen sulfide exposure is not confounded by uncertainty associated with either variability between species or toxicokinetic differences between sexes. On the basis of these considerations, the margins of exposure derived, using a biologically relevant respiratory endpoint in human asthmatics (a susceptible subgroup), are considered adequate to address uncertainties in the health effects and exposure databases for both healthy and susceptible asthmatic individuals.

For longer-term inhalation exposure risk characterization, the inhalation NOAEC of 10 ppm (14 mg/m³) identified on the basis of nasal (portal of entry) olfactory neuronal loss observed in rats and mice exposed to 30 ppm (42 mg/m³) or higher hydrogen sulfide for 10 weeks (Brenneman et al. 2000) or for 90 days (Dorman et al. 2004) was selected as the point of departure. The comparison of the subchronic NOAEC of 10 ppm for olfactory neuronal loss with the upper-bounding range of ambient air concentrations of 0.001–0.031 ppm (Alberta Environment 2000b; WGAQOG 2000) results in margins of exposure ranging from 320 to 10 000. The margins of exposure for respiratory effects

are considered adequate to address uncertainties in the health effects and exposure databases for the general population.

10.3.3 Neurological effects

Historically, neurotoxic effects have been documented in humans as a result of exposure to high levels of hydrogen sulfide via inhalation in occupational settings. Many case studies of acute human exposure to hydrogen sulfide reported neurological effects, including nausea, headaches, delirium, disturbed equilibrium, poor memory, neurobehavioural changes, olfactory paralysis, loss of consciousness, or “knockdown”, tremors and convulsions. Reports of concentrations causing these effects are limited, but it is estimated that levels in the range of 100–200 ppm (140–280 mg/m³) can cause a loss of smell, and 500–1000 ppm (700–1400 mg/m³) can cause loss of consciousness (US EPA 2003). Acute exposure to high levels (>500–1000 ppm [$>700\text{--}1400\text{ mg/m}^3$]) of hydrogen sulfide can be fatal. These levels, specific to industrial workplace settings, are several orders of magnitude higher than concentrations encountered in a community setting (0.0014–0.0434 mg/m³) and are consequently not considered relevant for general population risk characterization.

In a rat inhalation study in which whole body exposures were 0, 10, 30, 80 ppm or 400 ppm for 5 days, the NOAEC was 30 ppm and the LOAEC was 80 ppm (110 mg/m³) as derived on the basis of significant reductions in spontaneous motor activity (ambulations and total movements) (Struve et al. 2001). In addition, cognitive function was not impaired following exposures (nose only) to up to 80 ppm hydrogen sulfide for 5 days, evaluated as learning and memory using a modified Morris water maze protocol. Following motor activity testing, Struve et al. (2001) analyzed the brains for catecholamines and found that there were no hydrogen sulfide-related decreases in brain catecholamine levels in the striatum, hindbrain or hippocampus following exposures up to 400 ppm. The authors concluded, on the basis of these data, that cognitive dysfunction is not anticipated to occur following short-term, repeated hydrogen sulfide exposures to the lowest tested concentration of 10 ppm (14 mg/m³), a level identical to the US occupational limit (8-hour TLV) in effect in 2001.

Comparison of the cognitive function inhalation NOAEC of 30 ppm with the upper-bounding range of ambient air concentrations of 0.001–0.031 ppm (Alberta Environment 2000b; WGAQOG 2000) results in margins of exposure ranging from 970 to 30 000. The margins of exposure for neurological effects are considered adequate to address uncertainties in the health effects and exposure databases for the general population.

10.3.4 Reproductive and developmental effects

No reproductive or developmental classifications by other national or international regulatory agencies were identified. The limited data identified in the literature did not show any evidence of reproductive or developmental effects of hydrogen sulfide in experimental animals. No reproductive or developmental effects, including neurodevelopmental effects, were observed in rats exposed to concentrations up to 80 ppm (110 mg/m³) in a study by Dorman et al. (2000). Neurodevelopmental effects, such

as changes in Purkinje cell dendritic fields or neurotransmitter levels, were observed (Hannah and Roth 1991; Skrajny et al. 1992). However, no significant effects on behavioural performance or neuropathology were observed, and the evidence is considered insufficient to suggest adverse neurodevelopmental effects.

10.3.5 Overall

Inhalation is expected to be the predominant route of general population exposure to hydrogen sulfide, and the health effects assessment focused on data examining effects by this route. Margins between upper-bounding concentrations of hydrogen sulfide in ambient air and levels associated with critical health effects are considered adequate to address uncertainties in the health effects and exposure databases for the general population.

While exposure of the general population to hydrogen sulfide is not of concern at current levels, this substance can be associated with health effects of concern (pulmonary edema and severe neurological effects) at higher concentrations. Therefore, there may be a concern to human health if exposures of the general population were to increase.

10.4 Uncertainties in Evaluation of Risk to Human Health

Given that hydrogen sulfide is a gas, the primary route of exposure examined is the inhalation route; other routes of exposure are of limited significance. The health effects database for hydrogen sulfide is limited to the inhalation route. Relevant information was identified for acute, short-term and subchronic toxicity, genetic toxicity and reproductive and developmental toxicity, with a number of consistent effects observed across acute, short-term and subchronic studies. Long-term chronic experimental studies (i.e., with exposures greater than 90 days) were not identified for either hydrogen sulfide or its two precursors considered in this assessment except for one limited oral study conducted with sodium sulfide.

Data on hydrogen sulfide releases for certain sectors, e.g., metal and metal refining, located in proximity to human populations, were limited. However, confidence in the general population exposure assessment is high because a large set of ambient air level measurements, representative of most geographical locations in Canada, was available for several industries.

The availability of human data, especially data complementing experimental observations in animal models, increases confidence in the evaluation. The studies used to determine the lowest concentrations at which either adverse effects or no adverse effects were obtained included both experimental animal and human studies, with the human studies indicating biologically relevant effects at inhalation concentrations lower than those reported in the animal studies. The use of the human effect as the point of departure in the risk characterization (margins of exposure) increases confidence in the overall analysis.

11. Conclusion

Considering all available lines of evidence presented in this draft screening assessment, there is low risk of harm to organisms and the broader integrity of the environment from hydrogen sulfide, sodium bisulfide and sodium sulfide. It is proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide do not meet the criteria under paragraphs 64(a) or (b) of CEPA as they are 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.

On the basis of the adequacy of the margins between estimated exposures to hydrogen sulfide and critical health effect levels, it is proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide are 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 proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide do not meet the criteria in paragraph 64(c) of CEPA.

Therefore, it is proposed to conclude that hydrogen sulfide, sodium bisulfide and sodium sulfide do not meet any of the criteria set out in section 64 of CEPA.

References

- Abe K, Kimura H. 1996. The possible role of hydrogen sulfide as an endogenous neuromodulator. *J Neurosci*. 16:1066–1071.
- [ACA] Alberta Capital Airshed. 2014. Ambient Air Quality Monitoring Report 2014. Alberta Capital Airshed.
- [ACGIH] American Conference of Governmental Industrial Hygienists. 1992. Documentation of the threshold limit values and biological exposure indices. 6th ed. Cincinnati (OH): ACGIH. p. 786–788. In: Hydrogen Sulfide (HAZARDTEXT® Hazard Management). In: Heitland G, editor. TOMES® System. MICROMEDEX, Englewood (CO).
- [ACGIH] American Conference of Governmental Industrial Hygienists. 2010. Hydrogen sulfide: TLV chemical substances 7th edition documentation. <http://www.acgih.org/forms/store/ProductFormPublic/hydrogen-sulfide-tlv-r-chemical-substances-7th-edition-documentation>.
- Adams N, Kramer J. 1999. Silver speciation in wastewater effluent, surface water and pore waters. *Environ Toxicol Chem*. 18(12):2667–2673.
- Adelson L, Sunshine I. 1966. Fatal hydrogen sulfide intoxication report of three cases occurring in sewer. *Arch Pathol*. 81:375–380. [cited in IPCS 2003].
- Admiraal W, Peletier H. 1979. Sulphide tolerance of benthic diatoms in relation to their distribution in an estuary. *British Phycological Journal* 14:185-196.
- [AEP] Alberta Environment and Parks. 2016. Alberta Ambient Air Quality Objectives and Guidelines Summary. <http://aep.alberta.ca/air/legislation/ambient-air-quality-objectives/documents/AAQO-Summary-Jun2016.pdf>.
- [AER] Alberta Energy Regulator. 2016. Upstream Petroleum Industry Flaring and Venting Report Industry Performance for Year Ending December 31 2014. ST60B - 2015. <https://www.aer.ca/documents/sts/ST60B-2015.pdf>.
- Ahlborg G. 1951. Hydrogen sulfide poisoning in the shale oil industry. *Arch Ind Hyg Occup Med*. 3:247–266. [cited in IPCS 2003].
- [AIHA] American Industrial Hygiene Association. 1991. Emergency response planning guideline for hydrogen sulfide. Set 6. Akron (Ohio): AIHA.
- [AER] Alberta Energy Regulator. 2016. Directive 060. Upstream Petroleum Industry Flaring, Incinerating, and Venting. Effective date: March 22, 2016. <http://www.aer.ca/documents/directives/Directive060.pdf>.

Alberta Environment. 2000a. Air quality monitoring in the City of Lethbridge—September 1998 to July 1999. [cited 2011 Feb 25].
<http://www.environment.gov.ab.ca/info/library/6411.pdf>.

Alberta Environment. 2000b. Air quality monitoring near livestock feeding operations in the Lethbridge area—September 1998 to July 1999. [cited 2011 Feb 25].
<http://www.environment.gov.ab.ca/info/library/6390.pdf>.

Alberta Environment. 2004a. Assessment report on reduced sulphur compounds for developing ambient air quality objectives. Prepared by AMEC Earth and Environmental Limited and University of Calgary, November 2004. 152 p.

Alberta Environment. 2004b. Water Quality Data for Dissolved Sulfide and Dissolved Oxygen. Personal communication from Ron Tchir, October 2004.

Alberta Environment. 2007. Air Quality Monitoring Girouxville Area Confined Feeding Operations Fall 2004, Spring 2005 and Spring 2006 Final Report. 978-0-7785-6282-5 (online).

Alberta Environment. 2011. Hydrogen sulfide levels. Updated September 2011.
<http://www.environment.alberta.ca/01646.html>.

Alberta Environment. 2013. Dissolved sulphide and iron levels (spreadsheet). Personal communication (July 2013) with Mary Raven, Water Quality Technologist, Water Policy Branch, Alberta Environment and Sustainable Resource Development, Edmonton, AB.

Alberta Government. 2011. Managing air emissions from confined feeding operations in Alberta. Ambient air quality measurement around confined feeding operations in Alberta. Environmental Stewardship Division, Alberta Agriculture and Rural Development, Edmonton, AB.

Allyn LB. 1931. Notes on hydrogen sulfide poisoning. *Ind Eng Chem*. 23:234. [cited in US EPA 2003].

Almeida AF, Guidotti TL. 1999. Differential sensitivity of lung and brain to sulfide exposure: a peripheral mechanism for apnea. *Toxicol Sci*. 50:287–293. [cited in IPCS 2003; US EPA 2003].

[AMEC] Amec Foster Wheeler. 2014. 2014 Annual Report: Western Yellowhead Air Management Zone. Amec Foster Wheeler, Environment & Infrastructure: Edmonton (AB). http://www.wyamz.ca/documents/WYAMZ_AnnualReport_2014.pdf.

Amoore JE, Hautala E. 1983. Odor as an aid to chemical safety: odor thresholds compared with threshold limit values and volatilities for 214 industrial chemicals in air and water dilution. *J Appl Toxicol.* 3:272–290.

Anderson RC. 1987. 90-day subchronic oral toxicity in rats. Test material: hydrogen sulfide (H₂S). Arthur D. Little, Inc. Report to Dynamac Corporation. Work Assignment No. 11148-008. Reference 55461. [cited in US EPA 2003].

Andreae MO, Jaeschke WA. 1992. Exchange of Sulphur between Biosphere and Atmosphere over Temperate and Tropical Regions. In: Howarth RW, Stewart JWB, Ivanov MV, editors. *Sulphur Cycling on the Continents: Wetlands, Terrestrial Ecosystems and Associated Water Bodies (Scope 48)*. MS Swaminathan Research Foundation: Chennai, India. Chapter 3.

Andrew FD, Renne RA, Cannon WC. 1980. Reproductive toxicity testing for effects of hydrogen sulfide in rats. Pacific Northwest Laboratory annual report for 1979 to the DOE Assistant Secretary for Environment: Part 1. Biomedical sciences. Contract No. EY-76-C-06-1830. [cited in US EPA 2003].

[APHA] American Public Health Association, American Water Works Association and Water Environment Federation. 2000. *Standard Methods for the Examination of Water and Wastewater*. 14th edition, Washington, DC.

Arnold IMF, Dufresne RM, Alleyne BC, Stuart PJW. 1985. Health implication of occupational exposures to hydrogen sulfide. *J Occup Med.* 27(5):373–376. [cited in IPCS 2003; US EPA 2003].

[ATSDR] Agency for Toxic Substances and Disease Registry. 2006. Toxicological profile for hydrogen sulfide. Atlanta (GA): US Department of Health and Human Services, Public Health Service, ATSDR.
<http://www.atsdr.cdc.gov/ToxProfiles/tp114.pdf>.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008. Health consultation, exposure investigation report: Penntex Resources, Illinois/Lawrence Oil Field Greater Bridgeport Area, Lawrence County, Illinois. March 20, 2008. Atlanta (GA): US Department of Health and Human Services, Public Health Service, ATSDR.

Attene-Ramos MS, Nava GM, Muellner MG, Wagner ED, Plewa MJ, Gaskins HR. 2010. DNA damage and toxicogenomic analyses of hydrogen sulfide in human intestinal epithelial FHs 74 Int cells. *Environ Mol Mutagen.* 51(4):304–14.

Attene-Ramos MS, Wagner ED, Plewa MJ, Gaskins HR. 2006. Evidence that hydrogen sulfide is a genotoxic agent. *Mol Cancer Res.* 4(1):9–14.

Audeau FM, Gnanaharan C, Davey L. 1985. Hydrogen sulfide poisoning: associated with pelt processing. *N Z Med J.* 98:145–147. [cited in IPCS 2003].

Australia and New Zealand Environment and Conservation Council. 2000. Paper No. 4. Australia and New Zealand Guidelines for Fresh and Marine Water Quality, Volume 2, Aquatic Ecosystems - Rationale and Background Information. p. 8.3-169–8.3-175.

[BC EMS]. 2015. British Columbia Environmental Monitoring Databases. Environmental Monitoring System (EMS). Available at:
<http://www2.gov.bc.ca/gov/content/environment/research-monitoring-reporting/monitoring/environmental-monitoring-databases>.

Bagarinao T, Vetter RD. 1993. Sulphide tolerance and adaptation in the California killifish, *Fundulus parvipinnis*, a salt marsh resident. *J Fish Biol.* 42:729–748.

Barnes I, Bastian V, Becker K, Fink E, Nelson W. 1986. Oxidation of sulphur compounds in the atmosphere: I. Rate constants of OH radical reactions with sulphur dioxide, hydrogen sulphide, aliphatic thiols and thiophenol. *J Atmos Chem.* 4:445–466.

Bartholomew TC, Powell GM, Dodgson KS, Curtis CG. 1980. Oxidation of sodium sulfide by rat liver, lungs and kidney. *Biochem Pharmacol.* 29:2431–2437. [cited in US EPA 2003; ATSDR 2006].

Bates MN, Garrett N, Graham B, Read D. 1997. Air pollution and mortality in the Rotorua geothermal area. *Aust N Z J Public Health* 21:581–586. [cited in IPCS 2003].

Bates MN, Garrett N, Graham B, Read D. 1998. Cancer incidence, morbidity and geothermal air pollution in Rotorua, New Zealand. *Int J Epidemiol.* 27:10–14. [cited in IPCS 2003].

Bates MN, Garrett N, Shoemack P. 2002. Investigation of health effects of hydrogen sulfide from a geothermal source. *Arch Environ Health* 57(5):405–411.

Bates MN, Garrett N, Crane J, Balmes JR. 2013. Associations of ambient hydrogen sulfide exposure with self-reported asthma and asthma symptoms. *Environ Res.* 122:81–87.

Beauchamp RO Jr, Bus JS, Popp JA, Boreiko CJ, Andjelkovich DA. 1984. A critical review of the literature on hydrogen sulphide toxicity. *CRC Crit Rev Toxicol.* 13(1):25–97. [cited in IPCS 2003; US EPA 2003].

Beck JF, Cormier F, Donini JC. 1979. The combined toxicity of ethanol and hydrogen sulfide. *Toxicol Lett* 3:311–313. [cited in IPCS 2003].

Beck JF, Bradbury CM, Connors AJ, Donini JC. 1981. Nitrite as an antidote for acute hydrogen sulfide intoxication? *Am Ind Hyg Assoc J.* 42(11):805–809.

Bhambhani Y, Singh M. 1985. Effects of hydrogen sulfide on selected metabolic and cardiorespiratory variables during rest and exercise: a year end report. Report submitted to Alberta Worker's Health and Safety and Compensation, June 1985.

Bhambhani Y, Singh M. 1991. Physiological effects of hydrogen sulfide inhalation during exercise in healthy men. *J Appl Physiol.* 71(5):1872–1877.

Bhambhani Y, Burnham R, Snyder G, MacLean I, Martin T. 1994. Comparative physiological responses of exercising men and women to 5 ppm hydrogen sulfide exposure. *Am Ind Hyg Assoc J.* 55(11):1030–1035.

Bhambhani Y, Burnham R, Snyder G, MacLean I, Lovlin R. 1996a. Effects of 10-ppm hydrogen sulfide inhalation on pulmonary function in healthy men and women. *J Occup Environ Med.* 38(10):1012–1017.

Bhambhani Y, Burnham R, Snyder G, MacLean I, Martin T. 1996b. Effects of 5 ppm hydrogen sulfide inhalation on biochemical properties of skeletal muscle in exercising men and women. *Am Ind Hyg Assoc J.* 57:464–468.

Bhambhani Y, Burnham R, Snyder G, MacLean I. 1997. Effects of 10-ppm hydrogen sulfide inhalation in exercising men and women. Cardiovascular, metabolic and biochemical responses. *J Occup Environ Med.* 39(2):122–129.

Blunden J, Aneja VP. 2008. Characterizing ammonia and hydrogen sulfide emissions from a swine waste treatment lagoon in North Carolina. *Atmos Environ.* 42:3277–3290.

Bottenheim J, Strausz O. 1980. Gas-phase chemistry of clean air at 55° N latitude. *Environ Sci Technol.* 14:709–718. [cited in US EPA 1980].

Bowles K, Ernste M, Kramer J. 2003. Trace sulfide determination in oxic freshwaters. *Analytica Chimica Acta* 477:113–124.

Brenneman KA, James RA, Gross EA, Dorman DC. 2000. Olfactory neuron loss in adult male CD rats following subchronic inhalation exposure to hydrogen sulfide. *Toxicol Pathol.* 28(2):326–333.

Brenneman KA, Meleason DF, Sar M, Marshall MW, James RA, Gross EA, Martin JT, Dorman DC. 2002. Olfactory mucosal necrosis in male CD rats following acute inhalation exposure to hydrogen sulfide: reversibility and the possible role of regional metabolism. *Toxicol Pathol.* 30(2):200–208.

Breyse PA. 1961. Hydrogen sulfide fatality in poultry feather fertilizer plant. *Am Ind Hyg Assoc J.* 22:220–222. [cited in IPCS 2003; US EPA 2003].

[British Columbia] Government of British Columbia. 2015. Environmental Monitoring System Web Reporting (EMS WR). [results for 2015 for HSulfide]. Ministry of Environment, Government of British Columbia. <http://www.env.gov.bc.ca/emswr/>.

British Columbia. 2016. B.C. Ambient Air Quality Objectives. <http://www.bcairquality.ca/reports/pdfs/aqotable.pdf>.

Broderius SJ, Smith LL. 1976. Effect of hydrogen sulfide on fish and invertebrates. Part II – hydrogen sulfide determination and relationship between pH and sulfide toxicity. US Environmental Protection Agency, Office of Research and Development, Environmental Research Laboratory, Duluth, Minnesota. EPA-600/3-76-062b.

Budavari S. 1996. The Merck index: an encyclopedia of chemicals, drugs and biologicals. 12th ed. Whitehouse Station (NJ): Merck Research Laboratories. p. 4845.

Buick JB, Lowry RC, Magee TR. 2000. Is a reduction in residual volume a sub-clinical manifestation of hydrogen sulfide intoxication? *Am J Ind Med.* 37:296–299. [cited in IPCS 2003].

Burnett WW, King EG, Grace M, Hall WF. 1977. Hydrogen sulfide poisoning: review of 5 years' experience. *Can Med Assoc J.* 117:1277–1280. [cited in IPCS 2003; US EPA 2003].

Burstyn I, Senthilselvan A, Kim H-M, Cherry NM, Pietroniro E, Waldner C. 2012. Industrial Sources Influence Air Concentrations of Hydrogen Sulfide and Sulfur Dioxide in Rural Areas of Western Canada. *J Air & Waste Management Assoc.* 57:10:1241–1250.

Bush DR. 1980. Sodium Sulfides. In: Mark HF, Grayson M, Eckroth D. *Kirk-Othmer Encyclopedia of Chemical Technology*, vol. 11. New York (NY): John Wiley & Sons, p. 275–284.

Bush DR. 2000. Sodium Sulfides. In: Mark HF, Grayson M, Eckroth D. *Kirk-Othmer Encyclopedia of Chemical Technology*, vol. 11. New York (NY): John Wiley & Sons. p. 275–284.
<http://onlinelibrary.wiley.com/doi/10.1002/0471238961.1915040902211908.a01/full>

[CBSA] Canada Border Services Agency. 2013. Information gathered on the import of commodities corresponding to the codes HS 283010. Confidential information.

Campagna D, Kathman SJ, Pierson R, Inserra SG, Phifer BL, Middleton DC, Zarus GM, White MC. 2004. Ambient hydrogen sulfide, total reduced sulphur, and hospital visits for

respiratory diseases in northeast Nebraska, 1998–2000. *J Expo Anal Environ Epidemiol.* 14:180–187.

Canada. [1978]. *Food and Drug Regulations*. C.R.C., c. 870. http://laws-lois.justice.gc.ca/eng/regulations/c.r.c.,_c._870/index.html.

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C., 1999, c. 33. *Canada Gazette*, Part III, vol. 22, no. 3. <http://www.gazette.gc.ca/archives/p3/1999/g3-02203.pdf>

Canada. 2000. *Canadian Environmental Protection Act, 1999: Persistence and Bioaccumulation Regulations*, P.C. 2000-348, 29 March 2000, SOR/2000-107. <http://gazette.gc.ca/archives/p2/2000/2000-03-29/pdf/g2-13407.pdf>

Canada. 2001. *Canadian Environmental Protection Act, 1999: Notice with respect to certain substances on the Domestic Substances List (DSL)*. *Canada Gazette*, Part I, vol. 143, no. 40, <http://publications.gc.ca/gazette/archives/p1/2009/2009-10-03/pdf/g1-14340.pdf?file=.pdf>.

Canada. 2012a. *Canadian Environmental Protection Act, 1999: Notice with respect to certain inanimate substances (chemicals) on the Domestic Substances List*. *Canada Gazette*, Part I, vol. 146, no. 48. http://publications.gc.ca/collections/collection_2012/gazette/SP2-1-146-48.pdf.

Canada. 2012b. *Fisheries Act. Wastewater Systems Effluent Regulations*. P.C. 2012-942, 2012 June 28, SOR/2012-139. <http://laws-lois.justice.gc.ca/eng/regulations/sor-2012-139/index.html>.

Canada. 2014. Draft results from Phase Two of the Domestic Substances List Inventory Update. Environment Canada: Gatineau (QC).

[CASA] Clean Air Strategic Alliance. 2007-2016. Data warehouse. Alberta Environment. [cited 2017]. <http://airdata.albe.ca>.

CASETOX [Prediction module]. 2009. Version 2.1.0.6. Beachwood (OH): MultiCASE. [cited 2011 Jan 24]. <http://www.multicase.com/products/prod03.htm> [restricted access].

[CBC] Canada Broadcasting Corporation. 2016. Professor raises health concerns about the Gold Bar gas smell. *CBC News: Edmonton*. <http://www.cbc.ca/news/canada/edmonton/professor-raises-health-concerns-about-gold-bar-gas-smell-1.3549653>.

[CESPA] Connaissance et la Surveillance de la pollution atmosphérique. 2010. Banque de données sur la Connaissance et la Surveillance de la pollution atmosphérique—

données préliminaires. Québec (QC): Ministère du Développement durable, de l'Environnement et des Parcs, Direction du suivi de l'état de l'environnement.

Chance B, Schoener B. 1965. High and low energy states of cytochromes. I. In mitochondria. *J Biol Chem.* 241:4567–4573. [cited in IPCS 2003].

Chemical Book. Sodium sulfide. 2016.

http://www.chemicalbook.com/chemicalproductproperty_en_cb3429046.htm#Chemical%20Properties

Chepelkevitch L. 2009. Cameron Hills annual air quality report 2008/2009. Report prepared by Paramount Resources for Water, Air and Climate Change Bureau, Health Canada, Ottawa, ON.

Chessor A, Johannsen K. 2006. Mineral flotation cell exhaust system upgrade. www.aiha.org/aihce06/handouts/d3chessor.pdf.

Chetner S, Dzikowski P, Arendt A, Hoskinson G. 2001. Literature review and intensive livestock emission inventory for Alberta. Edmonton (AB): Alberta Agriculture, Food and Rural Development, Conservation and Development Branch.

Cho KS, Hirai M, Shoda M. 1992. Degradation of hydrogen sulfide by *Xanthomonas* sp. strain DY44 isolated from peat. *Appl Environ Microbiol.* 58:1183–1189.

Cihacek LJ, Bremner JM. 1993. Characterization of the sulfur retained by soils exposed to hydrogen sulfide. *Commun Soil Sci Plant Anal.* 24:85–92.

[CIIT] Chemical Industry Institute of Toxicology. 1983a. Ninety day vapour inhalation toxicity study of hydrogen sulfide in B6C3F1 mice. Report to the Chemical Industry Institute of Toxicology, Research Triangle Park, NC, by ToxiGenics, Inc. CIIT Docket No. 42063. EPA/OTS Document No. 0883-0255.

[CIIT] Chemical Industry Institute of Toxicology. 1983b. Ninety day vapour inhalation toxicity study of hydrogen sulfide in Fischer-344 rats. Report to the Chemical Industry Institute of Toxicology, Research Triangle Park, NC, by ToxiGenics, Inc. CIIT Docket No. 22063. EPA/OTS Document No. 0883-0255.

[CIIT] Chemical Industry Institute of Toxicology. 1983c. Ninety day vapour inhalation toxicity study of hydrogen sulfide in Sprague-Dawley rats. Report to the Chemical Industry Institute of Toxicology, Research Triangle Park, NC, by ToxiGenics, Inc. CIIT Docket No. 32063. EPA/OTS Document No. 0883-0255.

[Clearstone] Clearstone Engineering Ltd. 2014. A national inventory of greenhouse gas (GHG), criteria air contaminant (CAC) and hydrogen sulfide (H₂S) emissions by the upstream oil and gas industry. Prepared for Environment Canada.

[Clearstone] Clearstone Engineering Ltd. 2004. A national inventory of greenhouse gas (GHG), criteria air contaminant (CAC) and hydrogen sulfide (H₂S) emissions by the upstream oil and gas industry. Volume 2—Overview of the CAC inventory. Draft report by Clearstone Engineering Ltd. for the Canadian Association of Petroleum Producers.

Cromwell GL, Hays VW, Clark TL. 1978. Effects of copper sulfate, copper sulfide and sodium sulfide on performance and copper stores of pigs. *J Anim Sci.* 46(3):692–8.

[CSB] US Chemical Safety and Hazard Investigation Board. 2003. Investigation report: Hydrogen sulfide poisoning (2 dead, 8 injured), Georgia-Pacific Naheola Mill, Pennington, Alabama, January 16, 2002. Report No.: 2002-01-I-AL. http://www.csb.gov/assets/document/GP_Report.pdf.

[CTUMS] Canadian Tobacco Use Monitoring Survey. 2008. Supplementary tables, CTUMS annual 2008 [Internet]. Ottawa (ON): Health Canada. http://www.hc-sc.gc.ca/hc-ps/tobac-tabac/research-recherche/stat/ctums-esutc_2008/ann-table1-eng.php.

[CUM] Communauté Urbaine de Montréal. 2005. La qualité des eaux autour de l'île de Montréal. Réseau de suivi de milieu aquatique. Annexe RUISSO 2004. Montréal (QC): Communauté Urbaine de Montréal, Service de l'environnement et Environnement Québec.

Curtis E, Anderson CR, Simon J, Jensen AH, Day DL, Kelley KW. 1975. Effects of aerial ammonia, hydrogen sulfide and swine-house dust on rate of gain and respiratory-tract structure in swine. *J Anim Sci.* 41:735–739. [cited in IPCS 2003].

Czyzewski B, Wang DN. 2012. Identification and characterization of a bacterial hydrosulphide ion channel. *Nature* 483:494–498.

[DPD] Drug Product Database [database]. [modified 2015 Jul 17]. Ottawa (ON): Health Canada. [updated 2015 Jul 17; accessed 2016 Apr 6]. <http://webprod5.hc-sc.gc.ca/dpd-bdpp/index-eng.jsp>.

Deng JF, Chang SC. 1987. Hydrogen sulfide poisonings in hot spring reservoir cleaning: two case reports. *Am J Ind Med.* 11:447–451. [cited in IPCS 2003].

[DEREK] Deductive Estimation of Risk from Existing Knowledge [Prediction module on CD-ROM]. 2009. Version 12.0.0. Cambridge (MA): Harvard University, LHASA Group. [cited 2011 Jan 24]. <http://lhasa.harvard.edu/?page=toxicology.htm> [restricted access].

Dong J-Z, DeBusk SM. 2010. GC-MS analysis of hydrogen sulfide, carbonyl sulfide, methanethiol, carbon disulfide, methyl thiocyanate and methyl disulfide in mainstream vapor phase cigarette smoke. *Chromatographia* 71:259–265.

Dorman DC, Brenneman KA, Struve MF, Miller KL, James RA, Marshall MW, Foster PMD. 2000. Fertility and developmental neurotoxicity effects of inhaled hydrogen sulfide in Sprague-Dawley rats. *Neurotoxicol Teratol.* 22:71–84.

Dorman DC, Moulin FJM, McManus BE, Mahle KC, James RA, Struve MF. 2002. Cytochrome oxidase inhibition induced by acute hydrogen sulfide inhalation: correlation with tissue sulfide concentrations in the rat brain, liver, lung, and nasal epithelium. *Toxicol Sci.* 65:18–25.

Dorman DC, Struve MF, Gross EA, Brenneman KA. 2004. Respiratory tract toxicity of inhaled hydrogen sulfide in Fischer-344 rats, Sprague-Dawley rats, and B6C3F1 mice following subchronic (90-day) exposure. *Toxicol Appl Pharmacol.* 198:29–39.

Doyle PJ. 2013. Estimating concentrations of H₂S in oxic fresh water and wastewater. Unpublished document, prepared for Ecological Assessment Division, Environment Canada, Gatineau, Quebec. 5 p.

[ECHA]. European Chemicals Agency. 2016a. Disodium sulfide. <https://echa.europa.eu/registration-dossier/-/registered-dossier/16038/1>.

[ECHA]. European Chemicals Agency. 2016b. Sodium hydrogensulfide. <https://echa.europa.eu/registration-dossier/-/registered-dossier/15390/4/5>.

Elovaara E, Tossavinen A, Savolainen H. 1978. Effects of subclinical hydrogen sulfide intoxication on mouse brain protein metabolism. *Exp Neurol.* 62(1):18–25. [cited in US EPA 2003].

Environment Canada. 1996. Investigation of flare gas emissions in Alberta. Calgary (AB): Environment Canada, Alberta Energy and Utilities Board and Canadian Association of Petroleum Producers.

Environment Canada. 2002. Model predictions run on WVOLWIN. Existing Substances Branch, Environment Canada, Ottawa, ON.

Environment Canada. 2004a. Hydrogen sulfide (CAS RN 7783-06-4). Data collected pursuant to section 71 (CEPA 1999) and in accordance with the published notice “*Notice with Respect to Certain Substances on the Domestic Substances List (DSL)*”, Canada Gazette, Vol. 135 no. 46”. Data prepared by: Environment Canada, Health Canada, Existing Substances Program.

Environment Canada. 2004b. ClO₂ and H₂S from the forest products industry. Draft report prepared by Jacques Whitford Environment Ltd. for Pulp and Paper Division, Environment Canada, Gatineau, QC.

Environment Canada. 2004c. Personal communication from from Dr. Christian Gagnon, St. Lawrence Centre, Environment Canada, to Miles Constable, Prairie and Northern Region, Environment Canada. November 2004.

Environment Canada. 2010. Municipal water and wastewater survey, Municipal Water Use 2006 summary tables. http://www.ec.gc.ca/Water-apps/MWWS/pdf/MWWS_2006_WaterUse_May2010.pdf.

Environment Canada (2012 unpubl). Hydrogen sulfide (CAS No. 7783-06-4) in Canadian municipal wastewater. CMP Research and Monitoring Section. Science and Risk Assessment Directorate.

[ECCC, HC] Environment and Climate Change Canada, Health Canada. [modified 2007 Apr 20]. Categorization. Ottawa (ON): Government of Canada. [accessed 2016 Dec 1]. <http://www.chemicalsubstanceschimiques.gc.ca/approach-proche/categor-eng.php>.

[ECCC] Environment and Climate Change Canada. 2015. Hydrogen sulfide (CAS No. 7783-06-4) in Canadian Municipal Wastewater. CMP Research and Monitoring Section, Science and Risk Assessment Directorate, Environment Canada.

[ERCB] Energy Resources Conservation Board. 2010. Upstream Petroleum Industry. Flaring and Venting Report. Industry Performance for Year Ending December 31, 2009. November 2010. Calgary Alberta. <http://www.ercb.ca/docs/products/STs/st60B-2010.pdf>.

Farahat SA and Kishk N. 2010. Cognitive functions changes among Egyptian sewage network workers. *Toxicol Ind Health* 26(4):229–238.

Fenga C, Cacciola A, Micali E. 2002. Cognitive sequelae of acute hydrogen sulphide poisoning. A case report. *Med Lav*. 93(4):322–328. [in Italian with English abstract].

Fiedler N, Kipen H, Ohman-Strickland P, Zhang J, Weisel C, Laumbach R, Kelly-McNeil K, Olejeme K, Liou P. 2008. Sensory and cognitive effects of acute exposure to hydrogen sulfide. *Environ Health Perspect*. 116(1):78–85.

Finnbjornsdottir RG, Oudin A, Elvarsson BT, Gislason T, Rafnsson V. 2015. Hydrogen sulfide and traffic-related air pollutants in association with increased mortality: a case-crossover study in Reykjavik, Iceland. *BMJ Open* 5:e007272.

Fiorucci S, Santucci L. 2011. Hydrogen sulfide-based therapies: focus on H₂S releasing NSAIDs. *Inflamm Allergy Drug Targets* 10(2):133–40.

Fung DK, Bewick PH. 1980. Short-term toxicity of aqueous hydrogen sulfide to representative fish species of Lake Huron. In: Eaton JG, Parrish PR and Hendricks AC,

eds. Aquatic Toxicology. American Society for Testing and Materials, ASTM STP 707. p. 377–396.

Gagnon C, Turcotte P. 2007. Role des colloïdes dans la spéciation physique des métaux dans un panache majeur de dispersion d'eaux usées municipales. *Revue des Sciences de l'Eau* 20(3):275–285.

Gaitonde UB, Sellar RJ, O'Hare AE. 1987. Long term exposure to hydrogen sulfide producing subacute encephalopathy in a child. *Br Med J.* 294(6572):614.

Gmelin Database. 2009. Gesellschaft Deutscher Chemiker, licensed to Elsevier Information Systems GmbH; © 1988-1999, Gmelin Institut fuer Anorganische Chemie und Grenzgebiete der Max-Planck-Gesellschaft zur Foerderung der Wissenschaften.

Girard D. 2007. Cameron Hills annual air quality report. 2006/2007. Report prepared by Paramount Resources for Water, Air and Climate Change Bureau, Health Canada, Ottawa, ON.

Golder Associates. 2007. Appendix A. Air quality report. Report on air quality assessment of the refinery—upgrader complex expansion project section V and revamps. Submitted to Consumers' Co-operative Refineries Ltd., Regina, SK. [cited 2011 April]. <http://www.environment.gov.sk.ca/2007-184EIA%28ProjectProposalAppendixA1%29>.

Global News. 2016. Golba Bar Park users cause a stink over H₂S concerns. Global News, a division of Corus Entertainment Inc. Corus News. <http://globalnews.ca/news/2529293/gold-bar-park-users-cause-a-stink-over-h2s-concerns/>.

Grant WM, Schuman JS. 1993. Toxicology of the eye: effects on the eyes and visual systems from chemicals, drugs, metals, Vol. 1, 4th ed. Springfield (IL): Charles C. Thomas.

Green FHY, Schurch S, De Sanctis GT, Wallace JA, Cheng S, Prior M. 1991. Effects of hydrogen sulfide exposure on surface properties of lung surfactant. *J Appl Physiol.* 70:1943–1949. [cited in IPCS 2003; US EPA 2003].

Greenberg MI, Curtis JA, Vearrier D. 2013. The perception of odor is not a surrogate marker for chemical exposure: a review of factors influencing human odor perception. *Clin Toxicol.* 51(2):70–6.

Guidotti TL. 1994. Occupational exposure to hydrogen sulfide in the sour gas industry: some unresolved issues. *Int Arch Occup Environ Health* 66(3):153–160.

Haahtela T, Marttila O, Vilkkä V, Jappinen P, Jaakkola JJ. 1992. The South Karelia air pollution study: acute health effects of malodorous sulfur air pollutants released by a pulp mill. *Am J Public Health* 82(4):603–605.

Hagley SR, South DL. 1983. Fatal inhalation of liquid manure gas. *Med J Aust.* 2:459–460. [cited in US EPA 2003].

Haider SS, Hasan M, Islam F. 1980. Effect of air pollutant hydrogen sulfide on the levels of total lipids, phospholipids and cholesterol in different regions of the guinea-pig brain. *Indian J Exp Biol* 18:418–420. [cited in IPCS 2003].

Hall AH, Rumack BH. 1997. Hydrogen sulfide poisoning: an antidotal role for sodium nitrite? *Vet Hum Toxicol.* 39(3):152–154. [cited in US EPA 2003].

Hannah RS, Roth SH. 1991. Chronic exposure to low concentrations of hydrogen sulfide produces abnormal growth in developing cerebellar Purkinje cells. *Neurosci Lett.* 122:225–228.

Hannah RS, Hayden LJ, Roth SH. 1989. Hydrogen sulfide exposure alters the amino acid content in developing rat CNS. *Neurosci Lett.* 99:323–327. [cited in IPCS 2003; US EPA 2003].

Hannah RS, Bennington R, Roth SH. 1990. A relationship between hydrogen sulfide exposure and taurine levels in maternal rats. *Proc West Pharmacol Soc.* 33:177–179. [cited in IPCS 2003].

Hayden LJ, Goeden H, Roth SH. 1990a. Exposure to low levels of hydrogen sulfide elevates circulating glucose in maternal rats. *J Toxicol Environ Health* 31(1):45–52. [cited in IPCS 2003; US EPA 2003].

Hayden LJ, Goeden H, Roth SH. 1990b. Growth and development in the rat during subchronic exposure to low levels of hydrogen sulfide. *Toxicol Ind Health* 6(3/4):389–401. [cited in IPCS 2003; US EPA 2003].

Health Canada. 1987. Sulphide (as H₂S). http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/sulphide-sulfure/sulphide-sulfure-eng.pdf.

Health Canada. 1994. Human Health Risk Assessment for Priority Substances. <http://hc-sc.gc.ca/ewh-semt/pubs/contaminants/approach/index-eng.php>.

Health Canada. 1998. Exposure factors for assessing total daily intake of priority substances by the general population of Canada. Unpublished report. Ottawa (ON): Health Canada, Environmental Health Directorate.

Health Canada. [modified 2013 Jun 27]. Food additives permitted for use in Canada. Ottawa (ON): Health Canada. [accessed 2016 Apr 7]. <http://www.hc-sc.gc.ca/fn-an/securit/addit/list/index-eng.php>.

Health Canada. [modified 2015 Dec 14]. Cosmetic ingredient hotlist: list of ingredients that are prohibited for use in cosmetic products. Ottawa (ON): Health Canada, Consumer Product Safety Directorate. [accessed 2016 Apr 6]. <http://www.hc-sc.gc.ca/cps-spc/cosmet-person/hot-list-critique/hotlist-liste-eng.php>.

Hemminki K, Niemi ML. 1982. Community study of spontaneous abortions: relation to occupation and air pollution by sulphur dioxide, hydrogen sulfide, and carbon disulfide. *Int Arch Occup Environ Health* 51:55–63. [cited in IPCS 2003].

Hendrickson RG, Chang A, Hamilton RJ. 2004. Co-worker fatalities from hydrogen sulfide. *Am J Ind Med*. 45(4):346–350.

Hessel PA, Herbert FA, Melenka LS, Yoshida K, Nakaza M. 1997. Lung health in relation to hydrogen sulfide exposure in oil and gas workers in Alberta, Canada. *Am J Ind Med*. 31:554–557. [cited in IPCS 2003; US EPA 2003].

Higashi T, Toyoma T, Sakurai H, Nakaza M, Omae K, Nakadate T, Yamaguchi N. 1983. Cross sectional study of respiratory symptoms and pulmonary functions in rayon textile workers with special reference to hydrogen sulfide exposure. *Ind Health* 21:281–292. [cited in US EPA 2003].

[HIGP] Hawai'i Institute of Geophysics & Planetology. 1989. Fate of Sodium sulfide and Sodium in Steam muffler effluent. State of Hawaii, Division of Water & Land Development.
<http://scholarspace.manoa.hawaii.edu/bitstream/handle/10125/26752/Fate%20of%20Sodium%20Sulfide.pdf>.

Higuchi Y, Fukamachi M. 1977. Behavioral studies on toxicity of hydrogen sulfide by means of conditioned avoidance responses in rats. *Folia Pharmacol*. 73:307–319. [in Japanese]. [cited in IPCS 2003; US EPA 2003].

Hill FB. 1973. Atmospheric sulphur and its links to the biota. *Brookhaven Symp Biol*. 30:159–181. [cited in IPCS 2003].

Hirsch AR. 2002. Hydrogen sulfide exposure without loss of consciousness: chronic effects in four cases. *Toxicol Ind Health* 18(2):51–61.

Hirsch AR, Zavala G. 1999. Long term effects on the olfactory system of exposure to hydrogen sulfide. *Occup Environ Med*. 56:284–287. [cited in IPCS 2003; US EPA 2003].

Hoeksema GC. 2004. Ambient atmospheric hydrogen sulfide and total reduced sulphur compounds in western Canada: a summary. Unpublished data report. Edmonton (AB): Environment Canada.

Hoque MT, Yusoff FM, Law T, Syed MA. 1998. Effect of hydrogen sulfide on liver somatic index and Fulton's condition factor in *Mystus nemurus*. J Fish Biol. 52:23–30. <http://dx.doi.org/10.1111/j.1095-8649.1998.tb01549.x>.

Holm TR, Fisher RP, Frant MS, Gagnon C, Goodwin LR. 2000. 4500-S²⁻ (Sulfide). In: American Public Health Association, American Water Works Association, Water Environment Federation. 2000. Standard Methods for the Examination of Water and Wastewater. 20th ed. Washington (DC).

Holowenko FM, MacKinnon MD, Fedorak PM. 2000. Methanogens and sulfate-reducing bacteria in oil sands fine tailings waste', Can J Microbiol. 46(10):927–937.

Horton RA, Wing S, Marshall SW, Brownley KA. 2009. Malodor as a trigger of stress and negative mood in neighbors of industrial hog operations. Am J Public Health 99(Suppl 3):S610–S615.

[HSDB] Hazardous Substances Data Bank [database on the Internet]. 2003. Hydrogen sulfide. Bethesda (MD): National Library of Medicine (US). [revised 2003 Feb 14; cited 2003 Sept 10]. <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>.

[Huffington Post] The Huffington Post Alberta. 2016. Edmonton park's stink is silent, but could be deadly. http://www.huffingtonpost.ca/2016/04/26/edmonton-gold-bar-park_n_9781448.html.

Hughes TJ, Sparacino C, Frazier S. 1984. Validation of chemical and biological techniques for evaluation of vapors in ambient air testing of twelve (12) vapour-phase compounds (project summary). Research Triangle Park (NC): US Environmental Protection Agency, Health Effects Research Laboratory 3: EPA-6001. [cited in US EPA 2003].

Imamura T, Kage S, Kudo K, Jitsufuchi N, Nagata T. 1996. A case of drowning linked to ingested sulfides: a report with animal experiments. Int J Legal Med. 109(1):42–44. [cited in US EPA 2003].

Inserra SG, Phifer BL, Anger WK, Lewin M, Hilsdon R, White MC. 2004. Neurobehavioral evaluation for a community with chronic exposure to hydrogen sulfide gas. Environ Res. 95(1):53–61.

[

Iowa. 2002. Iowa concentrated animal feeding operations, air quality study. Final report. Iowa State University and the University of Iowa Study Group.

[IPCS] International Programme on Chemical Safety. 1981. Hydrogen sulfide. Geneva (CH): World Health Organization. (Environmental Health Criteria 19). Jointly sponsored by the United Nations Environment Programme, the International Labour Organization and the World Health Organization.

<http://www.inchem.org/documents/ehc/ehc/ehc019.htm>.

[IPCS] International Programme on Chemical Safety. 2003. Hydrogen sulfide: human health aspects. Geneva (CH): World Health Organization. (Concise International Chemical Assessment Document 53). Jointly sponsored by the United Nations Environment Programme, the International Labour Organization and the World Health Organization, and produced within the framework of the Inter-Organization Programme for the Sound Management of Chemicals.

<http://www.inchem.org/documents/cicads/cicads/cicad53.htm>.

[IPCS] International Programme on Chemical Safety. 2005. Sodium sulfide (anhydrous). ICSC: 1047. Prepared in the context of cooperation between the International Programme on Chemical Safety and the European Commission.

http://www.ilo.org/dyn/icsc/showcard.display?p_lang=en&p_card_id=1047&p_version=1

Jaakkola JJ, Vikka V, Marttila O, Jappinen P, Haahtela T. 1990. The South Karelia air pollution study: the effects of malodorous sulphur compounds from pulp mills on respiratory and other symptoms. *Am Rev Respir Dis* 142:1344–1350. [cited in IPCS 2003; ATSDR 2006].

Jaeschke W, Claude H, Herrmann J. 1980. Sources and sinks of atmospheric H₂S. *J Geophys Res Ser C* 85(C10):5639–5644. [cited in NRCC 1981].

Janssen AJ, Lens PN, Stams AJ, Plugge CM, Sorokin DY, Muyzer G, Dijkman H, Van Zessen E, Luimes P, Buisman CJ. 2009. Application of bacteria involved in the biological sulfur cycle for paper mill effluent purification. *Sci Total Environ*. 407:1333–1343.

Jappinen P, Tola S. 1990. Cardiovascular mortality among pulp mill workers. *Br J Ind Med*. 47(4):259–262. [cited in US EPA 2003].

Jappinen P, Vilkkka O, Marttila O, Haahtela T. 1990. Exposure to hydrogen sulfide and respiratory function. *Br J Ind Med*. 47:824–828.

Jemaa N, Thompson R, Paleologou M, Cormier M, Dokis R, Brown C, Sheedy M. 2009. Caustic Soda Production from Green Liquor Using the Green Liquor Splitting (GLS) System. *Eco-Tec* 172. http://eco-tec.com/techpapers/TP_172.

Jørgensen BB. 1982. Ecology of the bacteria of the sulphur cycle with special reference to anoxic–oxic interface environments. *Philos Trans R Soc Lond B Biol Sci.* 298:543–561.

Julian D, Arp A. 1992. Sulfide permeability in the marine invertebrate *Urechis caupo*. *J Comp Physiol. B* 162:59–67.

Kage S, Nagata T, Kimura K, Kudo K, Imamura T. 1992. Usefulness of thiosulfate as an indicator of hydrogen sulfide poisoning in forensic toxicological examination: a study with animal experiments. *Jpn J Forensic Toxicol.* 298:543–561. [cited in IPCS 2003; US EPA 2003].

Kage S, Kashimura S, Ikeda H, Kudo K, Ikeda N. 2002. Fatal and nonfatal poisoning by hydrogen sulfide at an industrial waste site. *J Forensic Sci.* 47(3):652–655.

Kage S, Ikeda H, Ikeda N, Tsujita A, Kudo K. 2004. Fatal hydrogen sulfide poisoning at a dye works. *Leg Med (Tokyo)* 6(3):182–186.

Kahrilas GA, Blotevogel J, Stewart SP, Borch T. 2015. Biocides in hydraulic fracturing fluids: a critical review of their usage, mobility degradation, and toxicity. *Environ Sci Technol.* 49(1):16–32.

Kamoun P. 2004. Endogenous production of hydrogen sulfide in mammals. *Amino Acids* 26(3):243–254.

Kang JC, Matsuda O. 1994. Combined effects of hypoxia and hydrogen sulfide on early developmental stages of white shrimp *Metapenaeus monoceros*. *J Facul Appl Biol Sci, Hiroshima Univ.* 33:21-27.

Khan AA, Schuler MM, Prior MG, Yong S, Coppock RW, Florence LZ, Lillie LE. 1990. Effects of hydrogen sulfide exposure on lung mitochondrial respiratory chain enzymes in rats. *Toxicol Appl Pharmacol.* 103(3):482–490. [cited in US EPA 2003].

Khan AA, Yong S, Prior MG, Lillie LE. 1991. Cytotoxic effects of hydrogen sulfide on pulmonary alveolar macrophages in rats. *J Toxicol Environ Health* 33:57–64. [cited in IPCS 2003; US EPA 2003].

Khan AA, Coppock RW, Schuler MM, Prior MG. 1998. Biochemical effects of subchronic repeated exposure to low and moderate concentrations of hydrogen sulfide in Fischer 344 rats. *Inhal Toxicol.* 10:1037–1044.

Kilburn KH. 1993. Case report: profound neurobehavioral deficits in an oil field worker overcome by hydrogen sulfide. *Am J Med Sci.* 306(5):301–305. [cited in US EPA 2003].

Kilburn KH. 1997. Exposure to reduced sulphur gases impairs neurobehavioral function. *South Med J.* 90(10):997–1006.

Kilburn KH. 1999. Evaluating health effects from exposure to hydrogen sulfide: central nervous system dysfunction. *Environ Epidemiol Toxicol.* 1:207–216. [cited in US EPA 2003].

Kilburn KH. 2003. Effects of hydrogen sulfide on neurobehavioral function. *South Med J.* 96(7):639–646.

Kilburn KH, Warshaw RH. 1995. Hydrogen sulfide and reduced-sulfur gases adversely affect neurophysiological functions. *Toxicol Environ Health* 11(2):185–197.

Kim KH. 2006. Emissions of reduced sulphur compounds (RSC) as a landfill gas (LFG): a comparative study of young and old landfill facilities. *Atmos Environ.* 40:6567–6578.

Kim KY, Ko HJ, Kim HT, Kim YS, Young MR, Lee CM, Kim CN. 2008. Quantification of ammonia and hydrogen sulphide emitted from pig buildings in Korea. *J Environ Manage.* 88(2):195–202.

Kimura H. 2002. Hydrogen sulfide as a neuromodulator. *Mol Neurobiol.* 26(1):13–19.

Kimura K, Hasegawa M, Matsubara K, Maseda C, Kagawa M, Takahashi S, Tanabe K. 1994. A fatal disaster case based on exposure to hydrogen sulfide—an estimation of the hydrogen sulfide concentration at the scene. *Forensic Sci Int.* 66:111–116. [cited in US EPA 2003; ATSDR 2006].

Kohno M, Tanaka E, Nakamura T, Shimojo N, Misawa S. 1991. [Influence of short-term inhalation of hydrogen sulfide in rats]. *Eisei Kagaku (Jpn J Toxicol Environ Health)* 37:103–106. [in Japanese]. [cited in IPCS 2003; US EPA 2003].

Kombian SB, Warenycia MW, Mele FG, Reiffenstein RJ. 1988. Effects of acute intoxication with hydrogen sulfide on central amino acid transmitter systems. *Neurotoxicology* 9:587–596. [cited in IPCS 2003; US EPA 2003].

Kombian SB, Reiffenstein RJ, Colmers WF. 1993. The actions of hydrogen sulfide on dorsal raphe serotonergic neurons in vitro. *J Neurophysiol.* 70:81–96. [cited in ATSDR 2006].

Kosmider S, Rogala E, Pacholek A. 1967. Electrocardiographic and histochemical studies of the heart muscle in acute experimental hydrogen sulfide poisoning. *Arch Immunol Ther Exp.* 15:731–740. [cited in IPCS 2003; US EPA 2003].

Kotronarou A, Hoffmann M. 1991. Catalytic autoxidation of hydrogen sulfide in wastewater. *Environ Sci Technol.* 25:1153.

Krekel K. 1964. [Electrocardiographic (ECG) changes in two workers after hydrogen sulfide poisoning.] *Zentralbl Arbeitsmed Arbeitsschutz* 14:159–163. [in German]. [cited in IPCS 2003].

Küster E, Dorusch F, Altenburger R. 2005. Effects of hydrogen sulfide on *Vibrio fischeri*, *Scenedesmus vacuolatus*, and *Daphnia magna*. *Environ Toxicol Chem.* 24(10):2621–2629.

Lefebvre M, Yee D, Fritz D, Prior MG. 1991. Objective measures of ocular irritation as a consequence of hydrogen sulfide exposure. *Vet Hum Toxicol.* 33(6):564–566. [cited in US EPA 2003].

Legator MS, Singleton CR, Morris DL, Philips DL. 2001. Health effects from chronic low-level exposure to hydrogen sulfide. *Arch Environ Health* 56(2):123–131. [cited in US EPA 2003].

Levitt MD, Furne J, Springfield J, Suarez F, DeMaster E. 1999. Detoxification of hydrogen sulfide and methanethiol in the cecal mucosa. *J Clin Invest.* 104(8):1107–1114. [cited in US EPA 2003].

Lewis RJ, Schnatter AR, Drummond I, Murray N, Thompson FS, Katz AM, Jorgensen G, Nicolich MJ, Dahlman D, Thériault G. 2003. Mortality and cancer morbidity in a cohort of Canadian petroleum workers. *Occup Environ Med.* 60(12):918–928.

Li Q, Lancaster JR Jr. 2013. Chemical foundations of hydrogen sulfide biology. *Nitric Oxide* 35:21–34.

Linden DR, Levitt MD, Farrugia G, Szurezewski JH. 2010. Endogenous production of H₂S in the gastrointestinal tract: still in search of a physiologic function. *Antioxid Redox Signal.* 12(9):1135–1146.

[LNHPD] Licensed Natural Health Products Database [database]. [modified 2014 Feb 27]. Ottawa (ON): Health Canada. [accessed 2016 Apr 6]. <http://webprod5.hc-sc.gc.ca/lnhpd-bdpsnh/index-eng.jsp>.

Logue JN, Ramaswamy K, Hersh JH. 2001. Investigation of illness associated with exposure to hydrogen sulfide among Pennsylvania school students. *J Environ Health* 63(6):9–13.

Lopez A, Prior M, Yong S, Albassam M, Lillie LE. 1987. Biochemical and cytological alterations in the respiratory tract of rats exposed for 4 hours to hydrogen sulfide. *Fundam Appl Toxicol.* 9(4):753–762.

Lopez A, Prior M, Lillie LE, Gulayets C, Atwal OS. 1988a. Histologic and ultrastructural alterations in lungs of rats exposed to sub-lethal concentrations of hydrogen sulfide. *Vet Pathol.* 25(5):376–384.

Lopez A, Prior M, Lillie LE, Lefebvre M. 1988b. Nasal lesions in rats exposed to hydrogen sulfide for four hours. *J Vet Res.* 49:1107–1111. [cited in IPCS 2003; US EPA 2003].

Lopez A, Prior MG, Reiffenstein RJ, Goodwin LR. 1989. Peracute toxic effects of inhaled hydrogen sulfide and injected sodium hydrosulfide on the lungs of rats. *Fundam Appl Toxicol.* 12:367–373. [cited in IPCS 2003].

Lu M, Hu LF, Hu G, Bian JS. 2008. Hydrogen sulfide protects astrocytes against H₂O₂-induced neural injury via enhancing glutamate uptake. *Free Radic Biol Med.* 45(12):1705–1713.

Luck J, Kaye SB. 1989. An unrecognized form of hydrogen sulfide keratoconjunctivitis. *Br J Ind Med.* 46:748–749. [cited in IPCS 2003].

Luther G III. 2013. Email from Dr. George Luther III (University of Delaware) to Patrick Doyle July 5, 2013.

Luther G III, Glazer B, Ma S, Trouwborst R, Shultz B, Druschel G, Kraiyya C. 2003. Iron and sulfur chemistry in a stratified lake: evidence for iron-rich sulfide complexes. *Aquatic Geochemistry* 9(2):87–110.

Maas F, DeKok LJ, Peters JL, Kuiper PJ. 1987. A comparative study of the effects of H₂S and SO₂ fumigation on the growth and accumulation of sulphate and sulphhydryl compounds in *Trifolium pratense* L., *Glycine max* Merr. and *Phaseolus vulgaris* L. *J Exp Bot.* 38(194):1459–1469.

Mancardi D, Penna C, Merlino A, Del Soldato P, Wink DA, Pagliaro P. 2009. Physiological and pharmacological features of the novel gasotransmitter: hydrogen sulfide. *Biochim Biophys Acta* 1787(7):864–72.

Marriott RA, Payman P, Marrugo-Hernandez JJ, Raval S. 2016. Hydrogen sulfide formation in oil and gas. *Can J Chem.* 94(4): 406–413.

Marttila O, Haahtela T, Silakoski I, Vattinen S, Suominen O. 1994a. The South Karelia air pollution study: relationship of outdoor and indoor concentrations of malodorous sulphur compounds released by pulp mills. *J Air Waste Manage Assoc.* 44:1093–1096. [cited in IPCS 2003].

Martilla O, Jaakola JK, Vikka V, Jappinen P, Haahtela T. 1994b. The South Karelia air pollution study: the effects of malodorous sulphur compounds from pulp mills on

respiratory and other symptoms in children. *Environ Res.* 66:152–159. [cited in IPCS 2003].

Martilla O, Jaakola JK, Partti-Pellinen K, Vikka V, Haahtela T. 1995. South Karelia air pollution study: daily symptom intensity in relation to exposure levels of malodorous sulphur compounds from pulp mills. *Environ Res.* 71:122–127. [cited in IPCS 2003].

McDonald JM, McIntosh AP. 1951. Fatalities from hydrogen sulfide in wells. *Arch Ind Hyg Occup Med.* 3:445–447. [cited in IPCS 2003].

[MEQ] Ministère de l'Environnement du Québec. 1997. Air quality in Québec (1975–1994). Ministère de l'environnement et de la faune, Direction du milieu atmosphérique and Direction des politiques du secteur industriel. [cited 2011 March 2].
<http://www.mddelcc.gouv.qc.ca/air/qualite1975-1994/air.pdf>

[MEQ] Ministère de l'Environnement du Québec. 2001a. Toxic potential assessment of municipal wastewater treatment plant effluent in Quebec—Appendices. St. Lawrence Vision 2000, Phase III—Industrial and Urban Component. Report, vol. A. Ministère de l'Environnement du Québec and Environment Canada. 136 p.

[MEQ]. Ministère de l'Environnement du Québec. 2001b. Toxic potential assessment of municipal wastewater treatment plant effluent in Quebec—Appendices. St. Lawrence Vision 2000, Phase III—Industrial and Urban Component. Appendices, vol. B. Ministère de l'Environnement du Québec and Environment Canada. 222 p.

Meyer B, Ward K, Koshlap K, Peter L. 1983. Second dissociation constant of hydrogen sulfide. *Inorg Chem.* 22(16):2345–2346.

Milby TH. 1962. Hydrogen sulfide intoxication: review of the literature and report of an unusual accident resulting in two cases of nonfatal poisoning. *J Occup Med.* 4:431–437. [cited in IPCS 2003; US EPA 2003].

Millero FJ, Hubinger S, Fernandez M, Garnett S. 1987. Oxidation of H₂S in seawater as a function of temperature, pH, and ionic strength. *Environ Sci Technol.* 21:439–443.

Miyazato T, Ishikawa T, Michiue T, Oritani S, Maeda H. 2013. Pathological and toxicological findings in four cases of fatal hydrogen sulfide inhalation. *Forensic Toxicol.* 31:172–170.

[Model Applier] Leadscope FDA Model Applier [Prediction module on CD ROM]. 2010. Version 1.3.3. Columbus (OH): Leadscope, Inc. [cited 2011 Jan 24].
http://www.leadscope.com/model_appliers/ [restricted access].

[MOECC] Ontario Ministry of the Environment and Climate Change. 2016. Technical Memorandum: Aamjiwnaang First Nation Community Air Monitoring Station. 2014

Report. Ontario Ministry of the Environment and Climate Change, Southwestern Region Technical Support Section, Air, Pesticides, and Environmental Planning Unit: London (ON). <http://www.aamjiwnaang.ca/wp-content/uploads/2016/04/2014-Aamjiwnaang-First-Nation-Air-Monitoring-Report.pdf>.

Moulin FJ, Brenneman KA, Kimbell JS, Dorman DC. 2002. Predicted regional flux of hydrogen sulfide correlates with distribution of nasal olfactory lesions in rats. *Toxicol Sci.* 66:7–15. [cited in US EPA 2003].

Mousa HAL. 2015. Short-term effects of subchronic low-level hydrogen sulfide exposure on oil field workers. *Environ Health Prev Med.* 20:12–17.

Muezzinoglu A. 2003. A study of volatile organic sulfur emissions causing urban odors. *Chemosphere* 51(4):245–52.

Nagata T, Kage S, Kimura K, Kudo K, Noda M. 1990. Sulfide concentrations in postmortem mammalian tissues. *J Forensic Sci.* 35(3):706–712. [cited in ATSDR 2006].

Nagpal NK, Pommen LW, Swain LG. 2001. A compendium of working water quality guidelines for British Columbia. Victoria (BC): British Columbia Ministry of Environment, Environmental Protection Division, Water, Air and Climate Change Branch. http://www.for.gov.bc.ca/hfd/library/ffip/Nagpal_NK2001.pdf.

Nam B, Kim H, Choi Y, Lee H, Hong ES, Park JK, Lee KM, Kim Y. 2004. Neurological sequela of hydrogen sulfide poisoning. *Ind Health* 42(1):83–87.

[NCASI] National Council for Air and Stream Improvement. 2012. The presence, fate, and ecological significance of hydrogen sulfide in pulp and paper mill effluents. Technical Bulletin No. 997. August 2012.

[NCI] National Chemical Inventories [database on a CD-ROM]. 2007. Issue 1. Columbus (OH): American Chemical Society, Chemical Abstracts Service. [cited 2010 Jun 20]. <http://www.cas.org/products/cd/nci/require.html>

Nelson K, Robinson DL. 2002. A case review: near fatal residential hydrogen sulfide exposure. *Air Med J.* 21(3):46–48.

[New Brunswick] New Brunswick Environment and Local Government. 2016. Air Quality Data Portal. <http://www.elgegl.gnb.ca/airnb/>.

[NHPID] Natural Health Products Ingredients Database [database]. [modified 2015 Nov 23]. Ottawa (ON): Health Canada. [accessed 2016 Apr 6]. <http://webprod.hc-sc.gc.ca/nhp-id-bdipsn/search-rechercheReq.do>.

Nicholls P. 1975. The effect of sulphide on cytochrome aa3. Isoteric and allosteric shifts of the reduced a-peak. *Biochim Biophys Acta* 396:24–35. [cited in IPCS 2003].

[NICNAS] National Industrial Chemicals Notification and Assessment Scheme. 2006. High Volume Industrial Chemicals List (HVICL). [cited in NICNAS 2016]. Available at http://www.nicnas.gov.au/data/assets/pdf_file/0019/6661/NICNAS_AHVICL_2006_PDF.pdf.

[NICNAS] National Industrial Chemicals Notification and Assessment Scheme. 2016. Inventory Multi-tiered Assessment and Prioritisation (IMAP). Human Health Tier II assessment for soluble sulfide salts. Available at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1636#cas-A_1313-82-2.

Nielsen AH, Vollertsen J, Hvitved-Jacobsen T. 2003. Determination of kinetics and stoichiometry of chemical sulfide oxidation in wastewater of sewer networks. *Environ Sci Technol.* 37:3853–3858.

Nielsen AH, Vollertsen J, Hvitved-Jacobson T. 2004. Chemical sulfide oxidation of wastewater—effects of pH and temperature. *Water Sci Technol.* 50(4):185–192.

Nielsen AH, Hvitved-Jacobsen T, Vollertsen J. 2007. Effects of iron on chemical sulfide oxidation in wastewater from sewer networks. *J Environ Eng.* 133(6):655-658.

Nikkanen HE, Burns MM. 2004. Severe hydrogen sulfide exposure in a working adolescent. *Pediatrics* 113(4):927–929.

[NIOSH] National Institute for Occupational Safety and Health. 1991. Fatal accident circumstances and epidemiology (FACE) report: two maintenance workers die after inhaling hydrogen sulfide in manhole, January 31, 1989. NTIS Publication No. PB91212761. Morgantown (WV): US Department of Health and Human Services, NIOSH. [cited in IPCS 2003].

[NIOSH] National Institute for Occupational Safety and Health. 2002. Pocket Guide to Chemical Hazards. National Institute for Occupational Health and Safety, U.S. Dept. of Health and Human Services.

[Northwest Territories] Government of the Northwest Territories. 2014. Northwest Territories Air Quality Report 2014. Environment Division, Department of Environment and Natural Resources, Government of the Northwest Territories: Yellowknife (NT). http://www.enr.gov.nt.ca/sites/default/files/128-air_quality_report_2014-proof.pdf.

[NPRI] National Pollutant Release Inventory. 2016. Reporting to the National Pollutant Release Inventory. Environment and Climate Change Canada. <https://www.ec.gc.ca/inrp-npri/default.asp?lang=En&n=F6300E68-1>.

[NPRI] National Pollutant Release Inventory [database on the Internet]. 2013, 2014, 2015, 2016, 2017. H₂S data for various reporting years. Gatineau (QC): Environment Canada. [cited 2015/09/23]. <http://www.ec.gc.ca/inrp-npri/>.

[NRCC] National Research Council of Canada. 1981. Hydrogen sulphide in the atmospheric environment: scientific criteria for assessing its effects on environmental quality. Ottawa (ON): National Research Council of Canada, Associate Committee on Scientific Criteria for Environmental Quality. Report No.: NRCC 18467.

[NWTENR] Northwest Territories Environment and Natural Resources. 2010. Northwest Territory Air Quality Report. 2010.

[NWTENR] Northwest Territories Environment and Natural Resources. 2011. Northwest Territory Air Quality Report. 2011.

[OECD] Organisation for Economic Cooperation and Development. 2009. Manual for the Assessment of Chemicals. Annex 1: Guidance for Completing a SIDS Dossier. [Internet]. Paris (FR): OECD, Environment Directorate. [cited July, 2011]. <http://www.oecd.org/dataoecd/13/17/36045066.pdf>.

[OMEE] Ontario Ministry of Environment and Energy. 1994. Water management policies, guidelines, provincial water quality objectives. Toronto (ON): OMEE.

[OMOE] Ontario Ministry of the Environment. 2007. Ontario air standard for total reduced sulphur. Standards Development Branch. Ontario Ministry of the Environment.

O'Neil MJ, Smith A, Heckelman PE. 2001. Hydrogen sulfide. The Merck index. An encyclopedia of chemicals, drugs, and biologicals. Whitehouse Station, NJ: Merck & Co., Inc., p. 859.

Oseid DM, Smith LL. 1974. Chronic toxicity of hydrogen sulfide to *Gammarus pseudolimnaeus*. Trans Am Fish Soc. 103(4):819-822.

Osbern LN, Crapo RO. 1981. Dung lung: a report of toxic exposure to liquid manure. Ann Intern Med. 95:312–314. [cited in US EPA 2003].

Palumbo JE, Brown LC, Stratton SC. 2010. Modeling the oxidation of sulfide in pulp and paper wastewaters. J Environ Eng. 136(10):1171-1179.

Parra O, Monso E, Gallego M, Morera J. 1991. Inhalation of hydrogen sulfide: a case of subacute manifestations and long term sequelae. Br J Ind Med. 48:286–287. [cited in IPCS 2003].

Partlo LA, Sainsbury RS, Roth SH. 2001. Effects of repeated hydrogen sulfide exposure on learning and memory in the adult rat. *Neurotoxicology* 22:177–189.

Partti-Pellinen K, Marttila O, Vikka V, Jaakola JK, Jappinen P, Haahtela T. 1996. The South Karelia air pollution study: effects of low-level exposure to malodorous sulphur compounds on symptoms. *Arch Environ Health* 51:315–320. [cited in IPCS 2003].

[PASZA] Peace AirShed Zone Association. 2011. Girouxville Area Ambient Air Quality Monitoring Summary Report. 27 p.

Phae CG, Shoda M. 1991. A new fungus which degrades hydrogen sulfide, methanethiol, dimethyl sulfide and dimethyl disulfide. *Biotechnol Lett.* 13:375–380.

[PMRA] Pest Management Regulatory Agency. 2010. PMRA list of formulants. Ottawa (ON): Health Canada, PMRA. HC Pub. No.: 100460, Cat. No.: H114-22/2010E. [accessed 2016 Apr 6]. http://publications.gc.ca/collections/collection_2010/arla-pmra/H114-22-2010-eng.pdf.

[PMRA] Pest Management Regulatory Agency. [modified 2013 Jul 27]. Pesticide Product Information Database [database]. Ottawa (ON): Pest Management Regulatory Agency, Health Canada. [accessed 2016 Apr 6]. <http://pr-rp.hc-sc.gc.ca/pi-ip/index-eng.php>.

Pomeroy RD, Boon AG. 1990. The problem of hydrogen sulphide in sewers. 2nd ed. Chesham (GB): Clay Pipe Development Association.

Pouliquen F, Blanc C, Arretz E. 1989. Hydrogen sulfide. In: Elvers B, Hawkins S, Ravenscroft M, Schultz G, editors. *Ullmann's encyclopedia of industrial chemistry*, vol. A13. Deerfield Beach (FL): VCH Publishers. p. 467–485.

Prior MP, Sharma AK, Yong S, Lopez A. 1988. Concentration–time dependent interactions in hydrogen sulfide toxicity in rats. *Can J Vet Res.* 52:375–379.

Prior M, Green F, Lopez A, Balu A, De Sanctis GT, Fick G. 1990. Capsaicin pre-treatment modifies hydrogen sulphide-induced pulmonary injury in rats. *Toxicol Pathol.* 18:279–288. [cited in IPCS 2003].

PubChem. 2016. Sodium monosulfide. Open Chemistry Database. US National Library of Medicine. <https://pubchem.ncbi.nlm.nih.gov/compound/237873#section=Top>.

Ramesh R. 1989. Chemical composition of the St. Lawrence River and its controlling factors. *Regional Characterization of Water Quality: Proceedings of the Baltimore Symposium*, May 1989. p. 275–281.

Reed BR, Carne J, Garrett N, Woods DL, Bates MN. 2014. Chronic ambient hydrogen sulfide exposure and cognitive function. *Neurotoxicol Teratol.* 42:68–76.

Reiffenstein RJ, Hulbert WC, Roth SH. 1992. Toxicology of hydrogen sulfide. *Annu Rev Pharmacol Toxicol.* 32:109–134. [cited in IPCS 2003; US EPA 2003].

Richardson DB. 1995. Respiratory effects of chronic hydrogen sulfide exposure. *Am J Ind Med.* 28:99–108. [cited in IPCS 2003; US EPA 2003].

Riffat R, Weeks JL, Brady P. 1999. Safety alert. Research indicates that even long-term, low-level exposure to hydrogen sulfide emissions can cause significant health effects. *Industrial Wastewater*, May/June, p. 31–34. [cited in IPCS 2003].

Roberts ES, Thomas RS, Dorman DC. 2008. Gene expression changes following acute hydrogen sulfide (H₂S)-induced nasal respiratory epithelial injury. *Toxicol Pathol* 36(4):560–7.

Rogers RE, Ferin J. 1981. Effect of hydrogen sulfide on bacterial inactivation in the rat lung. *Arch Environ Health* 36(5):261–264. [cited in US EPA 2003].

Rossoni G, Manfredi B, Tazzari V, Sparatore A, Trivulzio S, Del Soldato P, Berti F. 2010. Activity of a new hydrogen sulfide-releasing aspirin (ACS14) on pathological cardiovascular alterations induced by glutathione depletion in rats. *Eur J Pharmacol.* 648(1-3):139–45.

Roth SH, Skrajny B, Reiffenstein RJ. 1995. Alteration of the morphology and neurochemistry of the developing mammalian nervous system by hydrogen sulfide. *Clin Exp Pharmacol Physiol.* 22:379–380. [cited in IPCS 2003].

Rozan T, Benoit G. 1999. Geochemical factors controlling free Cu ion concentrations in river water. *Geochimica et Cosmochimica Acta* 63(19/20):3311–3319.

Rozan T, Lassmann M, Ridge D, Luther G III. 2000. Evidence of iron, copper and zinc complexation as multinuclear sulphide clusters in oxic waters. *Nature* 406:879–882.

[RSC] Royal Society of Canada. 2010. Environmental and Health Impacts of Canada's Oil Sands Industry. Gosselin, P, Hrudehy SE, Naeth A, Plourde A, Therrien R, van der Kraak G, Xu Z. Ottawa (ON). 440 p.

Saeedi A, Najibi A, Mohammadi-Bardbori A. 2015. Effects of long-term exposure to hydrogen sulfide on human red blood cells. *Int J Occup Environ Med.* 6:20–25.

Saillenfait AM, Bonnet P, de Ceaurriz J. 1989. Effects of inhalation exposure to carbon disulfide and its combination with hydrogen sulfide on embryonal and fetal development in rats. *Toxicol Lett.* 48:57–66. [cited in IPCS 2003; US EPA 2003].

Sastre C, Baillif-Couniou V, Kintz P, Cirimele V, Bartoli C, Christia-Lotter MA, Piercecchi-Marti MD, Leonetti G, Pelissier-Alicot AL. 2013. Fatal accidental hydrogen sulfide poisoning: a domestic case. *J Forensic Sci.* 58 (Suppl 1): S280–4.

Savolainen H. 1982. [Nordic expert group for TLV evaluation. 40. Hydrogen sulfide]. *Arbete och hälsa* 31:1–27. [in Finnish]. [cited in WHO 2000; IPCS 2003].

Schechter MT, Spitzer WO, Hutcheon ME, Dales RE, Eastridge LM, Steinmetz M, Tousignant P, Hobbs C. 1989. Cancer downwind from sour gas refineries: the perception and the reality of an epidemic. *Environ Health Perspect.* 79:283–290. [cited in IPCS 2003].

[SDS] Safety Data Sheet. 2013. Sodium Hydrosulphide. Orica Chemicals. Issued 06/06/2013. Version 6. [cited in NICNAS 2016].

[SESAA] Southeast Saskatchewan Airshed Association. 2013. Air quality monitoring program. Hydrogen sulphide. <http://sesaa.ca/programs/#H2S>.

[SESAA] Southeast Saskatchewan Airshed Association. 2015. 2015 Annual Report. Prepared by: Air & Climate Business Unit – Environment Division, Saskatchewan Research Council: Saskatoon (SK).
http://sesaa.ca/documents/2015_SESAA_AnnualReport.pdf.

Siegel SM, Penny P, Siegel BZ, Penny D. 1986. Atmospheric hydrogen sulfide levels at the Sulphur Bay Wildlife Area, Lake Rotorua, New Zealand. *Water Air Soil Pollut* 28:385–391.

Simson RE, Simpson GR. 1971. Fatal hydrogen sulfide poisoning associated with industrial waste exposure. *Med J Aust.* 1:331–334. [cited in US EPA 2003].

Sittig M. 1991. *Handbook of toxic and hazardous chemicals and carcinogens*, vols. 1–2. 3rd ed. Bracknell (GB): Noyes Publications. p. 914–916.

Skrajny B, Hannah RS, Roth SH. 1992. Low concentrations of hydrogen sulfide alter monoamine levels in the developing rat central nervous system. *Can J Physiol Pharmacol.* 70:1515–1518.

Skrajny B, Reiffenstein RJ, Sainsbury RS, Roth SH. 1996. Effects of repeated exposures of hydrogen sulfide on rat hippocampal EEG. *Toxicol Lett.* 84:43–53. [cited in US EPA 2003].

Skrtic L. 2006. *Hydrogen sulfide, oil and gas, and people's health*. Berkeley (CA): University of California, Energy and Resources Group.
<http://www.earthworksaction.org/publications.cfm?pubID=168>.

Smith B, Cummins K. 2004. Hydrogen sulfide exposure at a waste treatment facility. *J Occup Environ Hyg.* 1(3):D23–D25.

Smith L, Kruszyna H, Smith RP. 1977. The effect of methemoglobin on the inhibition of cytochrome c oxidase by cyanide, sulfide or azide. *Biochem Pharmacol* 26:2247–2250. [cited in IPCS 2003].

Smith LL, Oseid DM, Kimball GL, El-Kandelgy SM. 1976. Toxicity of hydrogen sulfide to various life stages of bluegill (*Lepomis macrochirus*). *Trans Am Fish Soc.* 105(3):442–449.

Smith LL, Oseid DM. 1974 Effect of hydrogen sulphide on development and survival of eight freshwater species. In: Blaxter JHS, editor. *The early life history of fish*. Berlin (Germany): Springer-Verlag.

Smith RP, Abbanat RA. 1966. Protective effect of oxidized glutathione in acute sulfide poisoning. *Toxicol Appl Pharmacol* 9:209–217. [cited in US EPA 2003].

Smith RP, Gosselin RE. 1964. The influence of methemoglobinemia on the lethality of some toxic anions. *Biochem Pharmacol* 26:2247–2250. [cited in IPCS 2003; US EPA 2003].

Snyder JF, Safir EF, Summerville GP, Middleberg RA. 1995. Occupational fatality and persistent neurological sequelae after mass exposure to hydrogen sulfide. *Am J Emerg Med* 13:199–203. [cited in US EPA 2003].

Solvay America Inc. 2011-2013. <http://www.solvay.us/en/binaries/PSS-Sodium-Sulfide-164363.pdf>.

Spolyar LW. 1951. Three men overcome by hydrogen sulfide in starch plant. *Ind Health Mon* 11:116–117. [cited in IPCS 2003].

Sprung JL. 1977. Tropospheric oxidation of H₂S. In: Pitts JN, Metcalfe RL, editors. *Advances in environmental science and technology*, vol. 7. New York (NY): John Wiley & Sons. p. 263–278. [cited in NRCC 1981].

Statistics Canada. 2003. Census of Agriculture. [cited 2003 Aug 19]. <http://www.statcan.gc.ca/daily-quotidien/031202/dq031202a-eng.htm>.

StatsCan] Statistics Canada. 2015. Canadian International Merchandise Trade Database. Table 990-0028. 28. Imports – Inorganic chemicals; organic or inorganic compounds of precious metals, of rare-earth metals, of radioactive elements or of isotopes. Top 9 countries for 2015 from which we imported trade commodity “283010

Sodium sulphides” to “Canada”, custom basis, 6 digit commodity level. Summary.
<http://www5.statcan.gc.ca/cimt-cicm/home-accueil?lang=eng>.

Struve MF, Brisbois JN, James RA, Marshall MW, Dorman DC. 2001. Neurotoxicological effects associated with short-term exposure of Sprague-Dawley rats to hydrogen sulfide. *Neurotoxicology* 22(3):375–385.

Sukola K, Wang F, Tessier A. 2005. Metal-sulfide species in oxic waters. *Analitica Chimica Acta* 528:183–195.

Svensden K. 2001. The Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals and The Dutch Expert Committee on Occupational Standards: Hydrogen sulfide. *Arbete och hälsa* 127:1–310.
https://gupea.ub.gu.se/dspace/bitstream/2077/4260/1/ah2001_14.pdf].

Swistock B, Sharpe W, Robillard PD. 2001. Hydrogen sulfide (Rotten egg odor) in Pennsylvania groundwater wells. Penn State University, Agricultural Analytical Services Lab. <http://www.aasl.psu.edu/HydrogenSulfide.pdf>.

Takemoto BK, Noble RD, Harrington HM. 1986. Differential sensitivity of duckweeds (Lemnaceae) to sulfite: II. Thiol production and hydrogen sulphide emission as factors influencing sulphite phytotoxicity under low and high irradiance. *New Phytol.* 103:541–548.

Taylor GE Jr, Selvidge WJ. 1984. Phytotoxicity in bush bean (*Phaseolus vulgaris*) of 5 sulfur-containing gases released from advanced fossil energy technologies. *J Environ Qual.* 13(2):224-230.

TDC. 2004. Material Safety Data Sheet Sodium hydrosulfide solution.
http://www.sfm.state.or.us/CR2K_SubDB/MSDS/SODIUM_HYDROSULFIDE_45.PDF.

Teschke K, Ahrens W, Andersen A, Boffetta P, Fincham S, Finkelstein M, Henneberger P, Kauppinen T, Kogevinas M, Korhonen K, Liss G, Liukkonen T, Osvoll P, Savela A, Szadkowska-Stanczyk I, Westberg H, Widerkiewicz K. 1999. Occupational exposure to chemical and biological agents in the nonproduction departments of pulp, paper, and paper product mills: an international study. *Am Ind Hyg Assoc J.* 60:73–83.

Tessier A. 2013. Email from Dr. Andre Tessier (INRS-Eau, University of Quebec) to Patrick Doyle, August 28, 2013.

Thoman M. 1969. Sewer gas: hydrogen sulfide intoxication. *Clin Toxicol* 2:383–386. [cited in IPCS 2003].

Thompson B, Bay S, Greenstein D, Laughlin J. 1991. Sublethal effects of hydrogen

sulfide in sediments on the urchin *Lytechinus pictus*. Marine Environmental Research 31:309–321.

Thompson CR, Kats G. 1978. Effects of continuous H₂S fumigation on crop and forest plants. Environ Sci Technol. 12:550–553.

Thorn J, Beiger L. 2004. Work-related symptoms and inflammation among sewage plant operatives. Int J Occup Environ Health 10(1):84–89.

Timoney KP, Lee P. 2009. Does the Alberta tar sands industry pollute? The scientific evidence. Open Conserv Biol J. 3:65–81.

[TOPKAT] Toxicity Prediction by Komputer Assisted Technology [Internet]. 2004. Version 6.2. San Diego (CA): Accelrys Software Inc. [cited 2011 Jan 24]. <http://accelrys.com/products/discovery-studio/predictive-toxicology.html>.

[Toxtree] Toxic hazard estimation by decision tree approach. 2009. Version 1.60. Sofia (BG): Ideacon Ltd. [cited 2011 Jan 24]. <http://toxtree.sourceforge.net/>.

Tran H, Vakkilainen EK. 2008. The Kraft Chemical Recovery Process. Manuscript 1.1-1–1.1-8. <http://www.tappi.org/content/events/08kros/manuscripts/1-1.pdf>.

Tvedt B, Edland A, Skyberg L, Forberg O. 1991a. Delayed neuro-psychiatric sequelae after acute hydrogen sulfide poisoning: affection of motor function, memory, vision and hearing. Acta Neurol Scand 84:348–351. [cited in IPCS 2003; US EPA 2003].

Tvedt B, Skyberg K, Aaserud O, Hobblesland A, Mathiesen T. 1991b. Brain damage caused by hydrogen sulfide: a follow-up study of six patients. Am J Ind Med. 20:91–101. [cited in IPCS 2003; US EPA 2003].

University of Alberta. 2007. Flare research project. Edmonton (AB): University of Alberta, Dept. of Mechanical Engineering. [cited 2007 Feb]. <http://www.mece.ualberta.ca/groups/combustion/flare/index.html>.

[US EPA] US Environmental Protection Agency. 1976. Effects of hydrogen sulfide on fish and invertebrates. Part 1—Acute and chronic toxicity studies. EPA-600/3-76-062a. Washington (DC): US EPA.

[US EPA] US Environmental Protection Agency. 1986. Sulfide—hydrogen sulfide. In: Quality criteria for water: 1986. Washington (DC): US EPA. Report No.: EPA 440/5-86-001.

[US EPA] US Environmental Protection Agency. 1993. Health assessment document for hydrogen sulfide. Washington (DC): US EPA, Office of Research and Development.

[US EPA] US Environmental Protection Agency. 2001. Emissions from animal feeding operations. Research Triangle Park, North Carolina: US EPA. Emission Standards Division, Office of Air Quality Planning and Standards.

[US EPA] US Environmental Protection Agency. 2003. Toxicological review of hydrogen sulfide (CAS No. 7783-06-4). Washington (DC): US EPA, Integrated Risk Information System (IRIS). [cited 2011 Jan 26].
<http://www.epa.gov/IRIS/toxreviews/0061tr.pdf>.

[US EPA] US Environmental Protection Agency. 2009. Technical review of hydrogen sulfide: chemistry, environmental fate and ecological toxicity. Washington (DC): US EPA, Office of Environmental Information, Office of Information Access and Analysis, Environmental Analysis Division, Analytical Support Branch. October 5, 2009. 25 p.

Vanhoorne M, De Rouck A, De Bacquer D. 1995. Epidemiological study of eye irritation by hydrogen sulfide and/or carbon disulphide exposure in viscose rayon workers. *Ann Occup Hyg*. 3:307–315. [cited in IPCS 2003].

Vismann B. 1991. Physiology of sulfide detoxification in the isopod *Saduria (Mesidotea) entomon*. *Mar Ecol Prog Ser*. 76:283–293.

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

Warencya MW, Smith KA, Blashko CS, Kombian SB, Reiffenstein RJ. 1989. Monoamine oxidase inhibition as a sequel of hydrogen sulfide intoxication increases in brain catecholamine and 5-hydroxytryptamine levels. *Arch Toxicol*. 63(2):131–136. [cited in US EPA 2003].

Warnek P. 1988. *Chemistry of the Natural Atmosphere*, Academic Press, London. [cited in Iowa, 2002].

Wasch HH, Estrin WJ, Yip P, Bowler R, Cone JE. 1989. Prolongation of the P-300 latency associated with hydrogen sulfide exposure. *Arch Neurol*. 46:902–904. [cited in US EPA 2003].

Watt MW, Watt SJ, Seaton A. 1997. Episode of toxic gas exposure in sewer workers. *Occup Environ Med*. 54:277–280. [cited in US EPA 2003].

Watts SF. 2000. The mass budgets of carbonyl sulfide, dimethyl sulfide, carbon disulfide and hydrogen sulfide. *Atmos Environ*. 34:761–779.

[WBEA] Wood Buffalo Environmental Association. 2016. Annual 2015 Report: Continuous Data. Revision: March 17, 2016. Wood Buffalo Environmental Association:

Fort McMurray (AB). <http://www.wbea.org/news/whats-new-archives/2015-ambient-annual-report>.

Weast RC, editor. 1982. Handbook of chemistry and physics. 63rd ed. Cleveland (OH): CRC Press.

Weisburger EK, Ulland BM, Nam J, Gart JJ, Weisburger JH. 1981. Carcinogenicity tests of certain environmental and industrial chemicals. *J Natl Cancer Inst.* 67(1):78.

Weisiger RA, Pinkus LM, Jakoby WB. 1980. Thiol S-methyltransferase: suggested role in detoxication of intestinal hydrogen sulfide. *Biochem Pharmacol.* 29:2885–2887. [cited in IPCS 2003; US EPA 2003; ATSDR 2006].

Weston Solutions Inc. 2006. Site-specific sulfide criterion for produced water discharges at five California OCS platforms. Technical Report 427-272. Weston Solutions Inc., Port Gamble Environmental Laboratories, Port Gamble, WA. 80 p.

Wetterau H, Oerket W, Knappe WG. 1964. Tests for the application of dried green fodder with higher H₂S content (experiments with poultry and fattened pigs). *Fetterung* 5:383–393. [cited in US EPA 2003].

[WGAQOG] Working Group on Air Quality Objectives and Guidelines. 2000. TRS science assessment document: post peer review draft. Unpublished report. August 2000. *Canadian Environmental Protection Act National Advisory Committee*.

[WHO] World Health Organization. 2000. Hydrogen sulfide. In: Air quality guidelines: Part II. Evaluation of human health risks. Geneva (CH): WHO. Available from: http://www.euro.who.int/_data/assets/pdf_file/0019/123076/AQG2ndEd_6_6Hydrogensulfide.PDF.

Wilmot PD, Cadee K, Katinic JJ, Kavanagh BV. 1988. Kinetics of sulfide oxidation by dissolved oxygen. *J Water Pollut Control Fed.* 60(7):1264–1270.

Wing S, Horton RA, Rose KM. 2013. Air pollution from industrial swine operations and blood pressure of neighbouring residents. *Environ Health Perspect.* 121(1):92–96.

[WISSA] Western Interprovincial Scientific Studies Association. 2006. Western Canada study of animal health effects associated with exposure to emissions from oil and natural gas field facilities. Interpretive overview by the Science Advisory Panel. [http://www1.agric.gov.ab.ca/\\$Department/deptdocs.nsf/all/webdoc12577/\\$FILE/advisor-y-panel-report-may18-2006.pdf](http://www1.agric.gov.ab.ca/$Department/deptdocs.nsf/all/webdoc12577/$FILE/advisor-y-panel-report-may18-2006.pdf).

[WSER] Waste Wastewater Systems Effluent Regulations. SOR/2012-139. Fisheries Act. Registration 2012-06-29

Xu X, Cho SI, Sammel M, You L, Cui S, Huang Y, Ma G, Padungtod C, Pothier L, Niu T, Christiani D, Smith T, Ryan L, Wang L. 1998. Association of petrochemical exposure with spontaneous abortion. *Occup Environ Med.* 55:31–36.

Appendices

Appendix A: Summary of hydrogen sulfide ambient air monitoring data

Table A1. Hydrogen sulfide concentrations near pulp and paper industry

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Nova Scotia, New Brunswick, British Columbia, Quebec	1989–1998	Air sampled continuously for various durations at 64 sites (~21 000 samples per site)	2.7 ppb (3.78 µg/m ³) (in New Brunswick between Jan 89 and Jul 94)	31 ppb (43.4 µg/m ³ , (at Nova Scotia site, between 1994 and 1998)	503 ppb (714 µg/m ³) (Nova Scotia site, between 94 and 98)	Distances to mill were not provided;	WGAQOG 2000
Nova Scotia, New Brunswick, British Columbia, Quebec	1999–2003	Air sampled continuously every hour (for various durations ranging from 2 to 5 years) at 54 sites	1.44 ppb (2 µg/m ³) (Prince George site, BC, 2 km from mill)	26 ppb (36 µg/m ³) (Fort Frances Que, located 1450 m from mill, over year 2001)	114 ppb (158 µg/m ³) (Fort Frances Que, over year 2001)	Based on TRS measurements; (H ₂ S concentration calculated based on the assumption that TRS contains up to 60% H ₂ S)	Environment Canada 2004b
Cap-de-la-Madeleine, Quebec	1979–1994	Air sampled continuously every hour for the length of the sampling period at 1 location	NA	NA	0.5 ppb (0.7 µg/m ³)	Distances between monitoring station and potential source were not provided	MEFQ 1997

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in study is provided;

Table A2. Hydrogen sulfide concentrations near oil and gas operations

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum* **	Notes	Reference
Alberta	May 2007 – May 2017	Air sampled continuously every hour at 35 stations	0.97 ppb (1.35 µg/m ³)	15 ppb (20.9 µg/m ³) (from Bonnyville Station)	113 ppb (158 µg/m ³) (Scotford Station No. 2 on October 15 2015, 9am)	Data extracted from the CASA website, a collection of air pollutant concentrations near oilsands in Alberta	CASA 2017
Alberta	May 2007- May 2012	Air sampled continuously every hour at 35 stations	1.4 ppb (1.95 µg/m ³)	21 ppb (29.3 µg/m ³) (from Midred Lake, Alberta)	100 ppb (140 µg/m ³) (Reported at Mildred Lake station on October 25 2009, 8pm)	Data extracted from the CASA website, a collection of air pollutant concentrations near oilsands in Alberta	CASA 2017
Alberta	May 2012- May 2017	Air sampled continuously every hour at 35 stations	1.0 ppb (1.39 µg/m ³) ()	17 ppb (23.7 µg/m ³) (, reported from Scotford Station No. 2)	113 ppb (158 µg/m ³) (Reported from Scotford Station No. 2 on October 15 2015, 9am)	Data extracted from the CASA website, a collection of air pollutant concentrations near oilsands in Alberta	CASA 2017

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Alberta (Wood Buffalo regional air shed)	2015	Continuous 1 hour sampling at 8 sites	0.7 ppb (0.98 µg/m ³) (annual)	NA	36 ppb (50.4 µg/m ³) Max 24-hour average: 6 ppb (8.4 µg/m ³) (across all sites)	Most monitoring sites are located at or near oil sands plants.	WBEA 2016
Montréal, Quebec	1979–1994	Air sampled continuously every hour at 1 location	NA	NA	0.3–0.5 ppb (0.42–0.7 µg/m ³)	Distance from sampling site to industry not known	MEFQ 1997
Alberta, Saskatchewan, northern British Columbia	April 2001 – January 2003	Air sampled continuously every hour for 3 years at 1100 sites	0.2 ppb (0.28 µg/m ³)	0.53 ppb (0.74 µg/m ³) (95th percentile across all samples at all sites)	NA	Passive sampling in cattle pastures near refinery; no information was provided on distances from oil refinery to pasture	WISSA 2006
Saskatchewan (Regina)	2002–2006	Continuous hourly sampling at 2 sites	NA	NA	< 10.8–30.2 ppb (< 15.1–42.3 µg/m ³)	Sites located both at the refinery and 1.9 km away	Golder Associates 2007
Northwest Territories	2008–2009	2 sites	0.7 ppb (hourly measurements averaged over a month)	NA	58 ppb (4.2–81.2 µg/m ³)	Data not collected for July and August	Chepelkevitch 2009

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Northwest Territories	2014	Continuous 1 hour sampling at 1 station (Norman Wells)	NA	NA	2.0 ppb (2.8 µg/m ³) Max 24-hour average: 1.8 ppb (2.52 µg/m ³)	NA	Northwest Territories 2014
Nova Scotia, New Brunswick, British Columbia, Quebec	1989–1998	Air sampled continuously every hour for various durations at 18 sites	1.37 ppb (2 µg/m ³) (BC site, over 5 years (94-98))	12ppb (16.6 µg/m ³ , (Manitoba site, between May 99 Jan 00)	113 ppb (157 µg/m ³) (Quebec site, between 90 and 98)	Distances to mill were not provided;	WGAQOG 2000

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study/database; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in the study is provided;

Table A3. Hydrogen sulfide concentrations near wastewater treatment systems

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Alberta (Bonnybrook / Calgary)	1989–2003	Air sampled continuously every hour at 1 location	1.2 ppb (168 µg/m ³)	NA	38 ppb (53 µg/m ³) Max 24-hr average: 7.2 ppb (10 µg/m ³)	This report is an analysis of data from the CASA database	Hoeksema 2004

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in study is provided;

Table A4. Hydrogen sulfide concentrations in urban areas

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Alberta (Lethbridge)	September 1998 – July 1999	Air measured hourly, sampled for 78 hours over 8-day period for 4 seasons at 5 locations	1 ppb (1.4 $\mu\text{g}/\text{m}^3$)	NA	0.6–3 ppb (0.84–4.2 $\mu\text{g}/\text{m}^3$)	NA	Alberta Environment 2000a
Quebec (urban areas)	2002–2009	Air sampled continuously every hour at 4 sampling stations	0.9 ppb (1.25 $\mu\text{g}/\text{m}^3$)	NA	22 ppb (30.8 $\mu\text{g}/\text{m}^3$)	NA	CESPA 2010
New Brunswick (Saint John)	Aug 4, 2013 – Aug 11, 2016	Continuous 1 hour sampling at 2 sites, reporting for total reduced sulfur (Forest Hills & West Side)	NA	0.6 and 1.2 ppb (0.84 and 1.68 $\mu\text{g}/\text{m}^3$) (for West Side and Forest Hills, respectively)	10.8 ppb (15.12 $\mu\text{g}/\text{m}^3$)	Concentrations were converted assuming hydrogen sulfide accounts for up to 60% of total reduced sulfur (Environment Canada 2004b)	New Brunswick 2016

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Southwestern Ontario (Sarnia)	2014	Continuous 1 hour sampling at 1 station (for total reduced sulfur);	0.36 ppb (0.504 µg/m ³) (annual avg)	NA	6 ppb (8.4 µg/m ³)	Aamjiwnaang First Nation community, located within a heavily industrialized area south of Sarnia, ON. Concentrations were converted assuming hydrogen sulfide accounts for up to 60% of total reduced sulfur (Environment Canada 2004b)	MOECC 2016

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in study is provided;

Table A5. Hydrogen sulfide concentrations near livestock operations

Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
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Sampling location	Time of sampling	Sampling regime	Average*	99th percentile**	Maximum**	Notes	Reference
Alberta (Livestock operations near Lethbridge)	September 1998 – July 1999	Air sampled during a 10-day period over 4 seasons, sampled continuously every hour at 17 sites located downwind of livestock operations: Lethbridge and Warner	26 ppb (36.4 $\mu\text{g}/\text{m}^3$), (at site #12, 15 of the 17 sites averaged 5 ppb or lower)	NA	54 ppb (76 $\mu\text{g}/\text{m}^3$) (within 30 meters of source, near a hogfarm)	Measurements by Alberta Environment's Mobile Air Monitoring Lab; distance from livestock operations not provided for most sites	Alberta Environment 2000b

Abbreviations: NA, not available; NS, not stated; TRS, total reduced sulfur

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in study is provided;

Table A6. Hydrogen sulfide concentrations near mixed-use sources of exposure

Sampling location	Time of sampling	Sampling regime	Average*	99 th percentile**	Maximum***	Addition al Notes	Referen ce
Alberta (Edmonton)	2014	Continuous 1 hour sampling at 5 stations	<1-1 ppb (<1.4-1.4 µg/m ³) (max annual average)	NA	22 ppb (30.8 µg/m ³) Max 24-hour average : 3 ppb (4.2 µg/m ³)	NA	ACA 2014
Saskatchewan (southeastern)	2015	Continuous 1 hour sampling at 5 sites	1.4 ppb (1.96 µg/m ³) (annual)	NA	118.6 ppb (166.0 µg/m ³) (Wauchope station August 1 st 2015, at 5:00 am); Max 24-hour average: 14.0 ppb (19.6 µg/m ³) (Wauchope station)	The region monitored encompasses activities from agriculture, oil/gas, mining, power generation and transportation.	SESAA 2015
Saskatchewan (Western Yellowhead Air Management Zone, WYAMZ)	2014	Air sampled continuously every hour at 2 locations (Maidstone and Kindersley)	0.2 and 0.3 ppb (0.28 and 0.42 µg/m ³) (annual average for Kindersley and Maidstone stations, respectively)	NA	13.5 ppb (18.9 µg/m ³) (Maidstone), Max 24hr average: 2.3 ppb (3.22 µg/m ³) (Maidstone)	The region of the WYAMZ contains agricultural, oil/gas, mining, power generation and transportation activities.	AMEC 2014

* Typically, for each station in the study or database, an average of all hourly samples over the sampling period is calculated; the highest average amongst stations is provided here.

** 99th percentile of hourly samples over the sampling period are calculated for each monitoring station in the study; the highest 99th percentile is provided here;

*** the highest 1-hour concentration amongst all 1-hour samples measured in study is provided;

Appendix B: Summary of health effects information for hydrogen sulfide**Table B1. Experimental animals and in vitro**

Endpoint	Lowest effect levels/results
Acute toxicity	<p>Inhalation LC₅₀ (rat) = 470–820 mg/m³ (Prior et al. 1988).</p> <p>Inhalation LC₁₀₀ (rat) = 700–2300 mg/m³ (Beck et al. 1979; Lopez et al. 1989; Khan et al. 1990).</p> <p>Inhalation LC₁₀₀ (mouse) = 1000 mg/m³ (Smith and Gosselin 1964).</p> <p>Inhalation LC₁₀₀ (rabbit) = 700–1400 mg/m³ (Kage et al. 1992).</p> <p><u>Respiratory effects</u> Lowest LOEC: 10 ppm (14 mg/m³) based on a significant decrease in cytochrome oxidase activity in the liver of male Sprague-Dawley rats (6 per group) exposed to 0, 10, 30, 80, 200 or 400 ppm (0, 14, 42, 110, 280 or 560 mg/m³) hydrogen sulfide for 3 h. A significant decrease in oxidase activity in the liver was observed at 14 mg/m³ and higher. A significant decrease in cytochrome oxidase activity in the lung was observed at 42 mg/m³ and higher. A significant increase in sulfide concentration was observed at 110 mg/m³ and higher in the lung and at 280 mg/m³ and higher in the liver (Dorman et al. 2002).</p> <p>Other lowest LOEC: 10 ppm (14 mg/m³) based on a significant transient increase in the cellularity of nasal lavage fluid in male Fischer 344 rats (12 per group) exposed to 0, 10, 200 or 400 ppm (0, 14, 280 or 560 mg/m³) hydrogen sulfide for 4 h. Four animals per exposed group were sacrificed at 1, 20 and 44 h post-exposure. A significant increase in cellularity of nasal lavage fluid was observed at 14 mg/m³ and higher. In the 14 and 280 mg/m³ exposed groups, changes were restored to original levels 20 h post-exposure. A significant increase in lactate dehydrogenase activity was observed at 280 mg/m³ and higher, and a significant increase in alkaline phosphatase activity was observed at 560 mg/m³ in bronchoalveolar lavage fluid.</p>

Endpoint	Lowest effect levels/results
	<p>Changes in nasal lavage fluid might be an early marker in detecting mild lesions, but histopathological evaluation is a more common practice (Lopez et al. 1987).</p> <p>Other LOEC: 615 mg/m³ based on transient histological changes in the lung and edematogenic effect in male Fischer 344 rats (12 per group) exposed to 0, 83 or 440 ppm (0, 116 or 615 mg/m³) hydrogen sulfide for 4 h. Four animals per exposed group were sacrificed 1, 18 and 42 h post-exposure (Lopez et al. 1988a).</p> <p><u>Neurological effects</u> Male Wistar rats (number of animals was not stated in secondary reference) were exposed to 100–500 ppm (139–695 mg/m³, values cited from US EPA 2003) hydrogen sulfide for 2 h. At 200 ppm (280 mg/m³) and higher, a significant decrease in discriminated avoidance response was observed. At 300 ppm (417 mg/m³) and higher, Sidman-type conditioned avoidance response was suppressed. After exposure to 139–280 mg/m³ for 1 h, increased blood pressure and respiratory rates, histological and biochemical changes in respiratory tissues and fluid were observed (Higuchi and Fukamachi 1977).</p> <p><u>Other studies</u> Elovaara et al. 1978; Rogers and Ferin 1981; Kombian et al. 1988; Lopez et al. 1988b; Khan et al. 1990, 1991; Prior et al. 1990; Green et al. 1991; Kohno et al. 1991; Lefebvre et al. 1991; Brenneman et al. 2002.</p>
Short-term toxicity	<p><u>Respiratory effects</u></p> <p>Lowest LOEC: 10 ppm (14 mg/m³) based on a significant decrease in cytochrome oxidase activity in lung mitochondria in male Fischer 344 rats (number of animals used was not stated) exposed to 0, 1, 10 or 100 ppm (0, 1.4, 14 or 140 mg/m³) hydrogen sulfide 8 h/day, 5 days/week, for 5 weeks. No effect on the enzymes in liver mitochondria was observed. Non-significant decreasing trend in the brain mitochondria for cytochrome oxidase activity was observed. In erythrocytes, a significant decrease in superoxide</p>

Endpoint	Lowest effect levels/results
	<p>dismutase activity was observed at 140 mg/m³. No histopathological examinations were performed (Khan et al. 1998).</p> <p><u>Neurological effects</u> Lowest NOAEC: 30 ppm (42 mg/m³) and LOAEC: 80 ppm (110 mg/m³) based on a significant reduction in motor activity and body temperature in male CD rats exposed to nose-only inhalation of hydrogen sulfide at 0, 30, 80, 200 or 400 ppm (0, 42, 110, 280 or 560 mg/m³) for 3 h/day for 5 consecutive days. One group of rats (10 per exposed group) was tested for spatial learning with a Morris water maze daily immediately after exposure. Another group of animals (10 per exposed group) was tested for spontaneous motor activity after the fifth exposure. Significant reductions in motor activity was observed at 80 ppm (110 mg/m³) and higher. In the Morris water maze testing, animals exposed to 400 ppm had significantly increased latencies in both the acquisition phase (day 1-4) and the probe trial (day 5) compared to control animals. No effects on catecholamine levels in the striatum, hindbrain or hippocampus from the animals tested for motor activity were observed (Struve et al. 2001).</p> <p>Male CD rats (5–7 per group) were exposed by whole-body inhalation of hydrogen sulfide at 0, 10, 30 or 80 ppm (0, 14, 42 or 110 mg/m³) for 3 h/day for 5 consecutive days. Multiple fixed-interval schedule operant performance was assessed daily and compared with the week pre-exposure and the week post-exposure. No significant effect on fixed-interval schedule performance was observed. Learning and memory were not impaired with hydrogen sulfide exposure up to 80 ppm based on Morris water maze testing. Motor activity was not affected by hydrogen sulfide exposure (Struve et al. 2001).</p> <p>Other LOAEC: 125 ppm (174 mg/m³) based on mild impaired performance during reacquisition of a reversed contingency radial arm maze task in male Sprague-Dawley rats (10–12 per group) exposed to 0 or 125 ppm (0 or 174 mg/m³) hydrogen sulfide 4 h/day, 5 days/week, for 5 weeks. No effects on memory retention or</p>

Endpoint	Lowest effect levels/results
	<p>acquisition were observed (Partlo et al. 2001).</p> <p><u>Other studies</u> Kosmider et al. 1967; Curtis et al. 1975; Haider et al. 1980; Skrajny et al. 1996; Brenneman et al. 2002; Dorman et al. 2002.</p>
Subchronic toxicity	<p><u>Respiratory effects</u></p> <p>Lowest NOAEC: 10 ppm (14 mg/m³) and LOAEC: 30 ppm (42 mg/m³) based on mild to moderate olfactory neuron loss and basal cell hyperplasia in the olfactory mucosa in male Sprague-Dawley rats (12 per group) exposed to 0, 10, 30 or 80 ppm (0, 14, 42 or 110 mg/m³) hydrogen sulfide for 6 h/day, 7 days/week, for 10 weeks. Only the nasal cavity and the olfactory system were examined. No effects were observed in the control animals or in animals exposed to 10 ppm (14 mg/m³) hydrogen sulfide (Brenneman et al. 2000).</p> <p>Other lowest NOAEC: 10 ppm (14 mg/m³) and LOAEC: 30 ppm (42 mg/m³) based on a significant increase in olfactory neuron loss in male and female Fischer 344 rats and B6C3F1 mice and in female Sprague-Dawley rats. A significant olfactory neuron loss was observed at 80 ppm (110 mg/m³) in male Sprague-Dawley rats. A 100% incidence in rhinitis was observed in B6C3F1 mice at 110 mg/m³ (Dorman et al. 2004). Dorman et al. (2004) is a reassessment of the nasal and lung histopathology from CIIT (1983a,b,c) as described below.</p> <p>Other LOEC: 80 ppm (110 mg/m³) in Fischer 344 rats, Sprague-Dawley rats and B6C3F1 mice exposed to 0, 10.1, 30.5 or 80 ppm (0, 14, 42 or 112 mg/m³) hydrogen sulfide for 6 h/day, 5 days/week, for 90 days (CIIT 1983a,b,c).</p> <p>In Sprague-Dawley rats (10 of each sex per group), the LOEC of 110 mg/m³ was based on a decrease of body weight in females and a decrease of absolute brain weight in males. At 110 mg/m³, feed consumption and body weights were reduced in both sexes. No significant differences in haematology, serum chemistry, urinalysis, ophthalmology, neuropathology or histopathology were</p>

Endpoint	Lowest effect levels/results
	<p>observed (CIIT 1983c).</p> <p>In Fischer 344 rats (10 of each sex per group), the LOEC of 110 mg/m³ was based on a decrease in feed consumption and body weight in both sexes and an increase in relative brain weight in males. A significant elevated sulfhemoglobin level was observed in males at 110 mg/m³. No significant differences in haematology, serum chemistry, urinalysis, ophthalmology, neuropathology or histopathology were observed (CIIT 1983b).</p> <p>In B6C3F1 mice (10 of each sex per group), the LOEC of 110 mg/m³ was based on reduced feed consumption and body weight and inflammation of the nasal mucosa in both sexes. Significant decrease of absolute, but not relative heart, liver and spleen weights in males and a significant decrease in kidney weight in females were observed at 110 mg/m³. No compound-related gross lesions or significant differences in haematology, serum chemistry, urinalysis, ophthalmology or neuropathology were observed (CIIT 1983a).</p> <p><u>Other studies</u> Wetterau et al. 1964; Anderson 1987; Dorman et al. 2002; Moulin et al. 2002.</p>
Chronic toxicity/carcinogenicity	No studies were identified.
Reproductive/developmental toxicity	<p>Reproductive and developmental NOEC = 80 ppm (110 mg/m³). Sprague-Dawley rats (12 of each sex per group) were exposed to 0, 10, 30 or 80 ppm (0, 14, 42 or 110 mg/m³) hydrogen sulfide for 6 h/day, 7 days/week, for 2 weeks prior to mating and continued during the 2-week mating period. For pregnant females, exposure continued from GD 0 to GD 19. The dams and their pups were exposed from PND 5 to PND 18. For males, exposure continued for 70 days. For reproductive effects: no statistically significant effects on reproductive performance, mating index, fertility index, post-implantation loss per litter, number of late resorptions or stillbirths were observed. No effects upon the number of live pups, litter size, average length of gestation or</p>

<p>Endpoint</p>	<p>Lowest effect levels/results</p> <p>average number of implants were observed. Testicular tubular degeneration was 42% in 112 mg/m³ males compared with 17% in control males, which was not statistically significant. Relative ovary weights were significantly decreased in low-exposure group females only. For developmental effects: no significant differences were observed in pup weight gain or development, behavioural performance or neuropathology. Behavioural assessment included motor activity (PNDs 13, 17, 21, 60 ± 2), passive avoidance (PNDs 22 ± 1, 62 ± 3), functional observational battery (PND 60 ± 2), acoustic startle response (PNDs 21, 62 ± 3) and neuropathology (PNDs 23 ± 2, 61 ± 2). Systemic toxicity with a LOEC of 10 ppm (14 mg/m³) was observed in the F₀ parents based on decreased relative and absolute adrenal weights in males and decreased relative ovary weights in females (Dorman et al. 2000).</p> <p>Other studies: Andrew et al. 1980; Saillenfait et al. 1989; Hayden et al. 1990a,b.</p>
<p>Neurodevelopmental effects</p>	<p>Lowest LOEC = 20 ppm (28 mg/m³) based on significant alterations in the architecture and growth characteristics of the Purkinje cell dendritic fields in pups when pregnant Sprague-Dawley rats (10 per group) were exposed to 0, 20 or 50 ppm (0, 28 or 70 mg/m³) hydrogen sulfide for 7 h/day from GD 5 to PND 21. Only developing cerebellar Purkinje cells were examined (one Purkinje cell from each pup). In pups exposed to 28 or 70 mg/m³, there was a significantly increased segment length over the low and middle branching orders, and the mean vertex path length was also significantly increased in the Purkinje cells. The authors concluded that these effects were indicative of significant alterations in the architecture and growth characteristics of the Purkinje cell dendritic fields (Hannah and Roth 1991). The US EPA (2003) questioned whether these alterations could be seen as adverse, as “the effects reported are highly selective and could be due to environmental factors not directly related to exposure including variability resulting from the restricted sampling technique (i.e., one Purkinje cell per pup).”</p> <p>Other lowest LOEC = 20 ppm (28 mg/m³) based on a</p>

Endpoint	Lowest effect levels/results
	<p>significant increase in serotonin levels in the frontal cortex in exposed pups when 20 pregnant Sprague-Dawley rats were exposed to 0, 20 or 75 ppm (0, 28 or 105 mg/m³) hydrogen sulfide for 7 h/day from GD 5 to PND 21. At 28 mg/m³, a significant increase in serotonin levels in the frontal cortex on PND 21 was observed. At 105 mg/m³, a significant increase in serotonin levels was observed in the cerebellum and frontal cortex on PNDs 14 and 21. At 105 mg/m³, a significant increase in norepinephrine levels in the cerebellum was observed on PNDs 7, 14 and 21. At 28 mg/m³, a significant decrease in norepinephrine levels was observed in the frontal cortex on PNDs 14 and 21 (Skrajny et al. 1992).</p> <p>Other studies: Hannah et al. 1989, 1990; Roth et al. 1995.</p>
Genotoxicity and related endpoints	<p><i>In vitro</i></p> <p>Mutagenicity: Negative: Ames tests, <i>Salmonella typhimurium</i> TA97, TA98, TA100 in the presence or absence of metabolic activation S9 at dose levels of 0, 17, 57, 175, 582 or 1750 µg/plate (Hughes et al. 1984).</p> <p>Comet assays [using sodium sulfide (Na₂S) as hydrogen sulfide is released when Na₂S is dissolved in aqueous solution]: Positive: Nontransformed human small intestine FHs 74 Int cells were treated with Na₂S·9H₂O ranging from 250 to 2000 µM for 2 hours. Dose-dependent responses were observed. Cytotoxicity was not observed (Attene-Ramos et al. 2010).</p> <p>Negative: Chinese hamster ovary (CHO) cells were treated with Na₂S for 4 hours at concentrations ranging from 25 to 5000 µM. Acute cytotoxicity was observed at ≥ 7500 µM (Attene-Ramos et al. 2006).</p> <p>Positive: In a modified comet assay, DNA repair was inhibited using hydroxyurea and 1-β-D-arabinofuranosylcytosine (AraC). CHO cells were treated with Na₂S for 2 hours at concentrations ranging from 250 to 3000 µM (Attene-Ramos et al. 2006).</p>

Endpoint	Lowest effect levels/results
	<p>Negative: Cl.16E subclone of the human colonic cancer cells HT29 were treated with Na₂S at concentration of 2000 µM (Attene-Ramos et al. 2006).</p> <p>Positive: In a modified comet assay, DNA repair was inhibited using hydroxyurea and 1-β-D-arabinofuranosylcytosine (AraC).Cl.16E subclone of the human colonic cancer cells HT29 were treated with Na₂S at concentrations ranging from 500 to 2000 µM (Attene-Ramos et al. 2006).</p> <p><i>In vivo</i> Male Sprague-Dawley rats (3-4 animals per time point) were exposed nose-only to 0 or 200 ppm (280 mg/m³) hydrogen sulfide for 3 hours per day for 1 day or for 5 consecutive days. Nasal pathology and gene expression profiles of the nasal respiratory epithelial cells were examined. In terms of nasal pathology, mild respiratory epithelial injury was observed in animals after an acute 3-hour exposure to hydrogen sulfide. Infiltration with inflammatory cells was observed 3 hours post-exposure. By 24-hour post-exposure, respiratory epithelial regeneration occurred. Complete recovery from initial respiratory epithelial injury was observed in all animals after 5 consecutive days of exposure. Gene expression profiling by microarray was performed in the nasal respiratory epithelium of the rats. Gene expression profiles were generated at 3, 6 and 24 hour after the initial 3-hour exposure and at 24-hour after the last exposure. Initial gene expression changes were involved with cellular defense/ inflammation followed by alteration of gene expression involved in cellular proliferation and microtubule-based movement. Overall, hydrogen sulfide was found to alter gene expression involved with cell cycle regulation, cellular division, DNA metabolism and repair, protein kinase regulation and cytoskeletal organization and biogenesis (Roberts et al. 2008).</p> <p>No other studies were identified.</p>

Table B2. Human studies

Endpoint	Lowest effect levels/results
Ocular effects	<p>WHO (2000) reported a threshold for eye irritation at 10–20 ppm (15–30 mg/m³) and serious eye damage at 50–100 ppm (70–140 mg/m³) based on the study of Savolainen (1982). Detail information was not reported in WHO (2000).</p> <p>In a community around a paper mill with an environmental exposure to an annual mean concentration of 6 µg/m³ of hydrogen sulfide (daily peaks of hydrogen sulfide concentrations as high as 100 µg/m³) and co-exposure to methyl mercaptan and methyl sulfides, eye irritation was reported 12 times more frequently than in communities without exposure (Jaakkola et al. 1990).</p> <p>A group of viscose rayon workers (123, males) exposed to hydrogen sulfide and/or carbon disulfide for at least a year and 67 referents were given self-administered questionnaires with questions concerning eye complaints. For the viscose rayon workers, personal exposure levels for hydrogen sulfide and carbon disulfide were measured and varied from 0.2-8.9 mg/m³ and 4-112 mg/m³, respectively. The referents were not exposed to hydrogen sulfide, carbon disulfide or any other irritant substances occupationally. After adjusting for age and smoking, viscose rayon workers exposed to > 5 mg/m³ hydrogen sulfide had significantly more eye complaints than the referents (Vanhoorne et al. 1995).</p> <p>Other studies: Riffat et al. 1999.</p>
Respiratory effects	<p>Lowest LOEC: 0.5 ppm (0.7 mg/m³) was based on a significant decrease in RER in the “low fit” males exercising at maximum level. Sixteen healthy male volunteers and 5 healthy female volunteers were exposed to 0, 0.5, 2 or 5 ppm (0, 0.7, 2.8 and 7 mg/m³) hydrogen sulfide via oral inhalation during graded exercise until exhaustion. Exercise duration ranged from 13 to 16 min. The male subjects were ranked in serial order on the basis of their relative maximum oxygen uptake (VO₂ max) and classified into “high fit” (mean age 24 ± 5.1 SD) and “low fit” (mean age 26.3 ± 5.9 SD) groups, whereas females (mean age 23.8 ± 4.7 SD) were not classified. The mean VO₂ max values for “high fit” and “low fit” groups were 46.9 ± 3.9 SD ml/kg per minute and 36.9 ± 3.2 SD ml/kg per minute, respectively. A number of physiological and pulmonary parameters were measured at two submaximal and maximal exercise levels. In the “high fit” group, exposure at 5 ppm</p>

Endpoint	Lowest effect levels/results
	<p>resulted in a significant increase in absolute oxygen consumption and blood lactate concentration and a significant reduction in RER during submaximal and maximal exercise. In the “low fit” group, a significant decrease in RER was observed at 0.5, 2 and 5 ppm at the maximal exercise level and at 2 and 5 ppm at the submaximal exercise level. At 5 ppm, a significant increase in blood lactate concentration was observed during all exercise levels and a significant increase in absolute oxygen consumption was observed at the maximal exercise level. In the female group, significant increases in absolute oxygen consumption and relative oxygen consumption were observed at 5 ppm during maximal exercise. RER was significantly decreased at 5 ppm during all exercise levels (Bhambhani and Singh 1985).</p> <p>Other effect level: 2 ppm (2.8 mg/m³) in 10 asthmatic volunteers [7 women with mean age 44.1 (ranging from 31 to 61) and 3 men with mean age 40.7 (ranging from 33 to 50)] exposed to 2.8 mg/m³ hydrogen sulfide for 30 min in an exposure chamber. The subjects had had bronchial asthma for 1–13 years (mean 3.7 years) and had been taking medications. They did not take medications for 2 days prior to the study. Severe asthmatics were not included in the study. Airway resistance increased 26.3% on average, and specific airway conductance (SGaw) decreased 8.4% on average. These effects were not statistically significant. The SGaw decreased in six and increased in four subjects. In two of the four subjects with decreased SGaw, changes were greater than 30% for both airway resistance and SGaw indicating possible bronchial obstruction in the two subjects. Three of the 10 subjects reported headaches after exposure. No significant differences in forced vital capacity, forced expiratory volume in 1 s and forced expiratory flow were observed. It should be noted that the investigators compared pre- and post-exposure results only and did not expose subjects to both treatment and control conditions; nor were non-asthmatic control subjects used for comparison. The authors also examined possible respiratory effects associated with hydrogen sulfide in 26 male pulp mill workers (mean age 40.3, range 22-60 years old). Amongst the 26 workers, 6 were smokers, 4 had previous allergies and 5 were atopic subjects. These workers were exposed to hydrogen sulfide in the workplace with exposure levels ranging from 1-11 ppm. Respiratory effects were compared based on responses to standard histamine challenges performed after a holiday or one day away from work with responses obtained at the end of the work day. No statistically</p>

Endpoint	Lowest effect levels/results
	<p>significant changes in respiratory function (forced vital capacity [FVC], forced expiratory volume in one second [FEV₁] and bronchial responsiveness) were observed (Jappinen et al. 1990).</p> <p>Other LOEC: 2 ppm (2.8 mg/m³) based on significantly reduced RER in healthy male volunteers (16 in total, mean age 25.2 ± 5.5 SD) exposed to 0, 0.5, 2 or 5 ppm (0, 0.7, 2.8 or 7 mg/m³) hydrogen sulfide via oral inhalation during graded cycle exercise until exhaustion. Exposure duration was at least 16 min. Physiological parameters were measured at three levels, two submaximal and maximal levels. RER was significantly reduced at both 2 and 5 ppm during maximal exercise and at 5 ppm during submaximal exercise. Maximum oxygen uptake (VO₂ max) was significantly increased during maximal exercise at 5 ppm. A significant increase in blood lactate levels was observed at all exercise levels in subjects exposed to 5 ppm hydrogen sulfide. Heart rate, expired ventilation and maximal power output were not affected (Bhambhani and Singh 1991).</p> <p>A group of healthy subjects (13 men with mean age 24.7 ± 4.6 SD, 12 women with mean age 22.0 ± 2.1 SD) were exposed to 0 or 5 ppm (7 mg/m³) hydrogen sulfide for 30 min via oral inhalation during exercise at 50% of their predetermined maximal aerobic power. No significant effects were observed on physiological, perceptual or arterial blood parameters. Biochemical properties of skeletal muscle were analyzed immediately following exercise. Muscle lactate, lactate dehydrogenase and cytochrome oxidase were non-significantly decreased. In men, citrate synthase was significantly decreased at 5 ppm, which the authors suggested might be an indication of aerobic metabolism inhibition (Bhambhani et al. 1994, 1996b).</p> <p>A group of healthy subjects (9 men with mean age 24.7 ± 6.4 SD, 10 women with mean age 21.8 ± 3.0 SD) were exposed to 0 or 10 ppm (14 mg/m³) hydrogen sulfide for 15 min via oral inhalation during exercise at 50% of their predetermined maximal aerobic power. No significant effects on pulmonary function were observed with variables derived from the flow volume loop, maximum ventilation volume and diffusion capacity of the lung for carbon monoxide (Bhambhani et al. 1996a).</p> <p>A group of healthy subjects (15 men with mean age 23.4 ± 5.2 SD, 13 women with mean age 21.8 ± 3.0 SD) were exposed to 0</p>

Endpoint	Lowest effect levels/results
	<p>or 10 ppm (14 mg/m³) hydrogen sulfide for 30 min via oral inhalation during exercise at 50% of their predetermined maximal aerobic power. A significant decrease in oxygen uptake and a significant increase in both the RER and blood lactate levels were observed in both men and women at 10 ppm. A non-statistically significant increase in muscle lactate levels and a non-statistically significant decrease in muscle citrate synthase activity were observed (Bhambhani et al. 1997).</p> <p>Other studies: Higashi et al. 1983; Jappinen et al. 1990; Richardson 1995; Hessel et al. 1997; Buick et al. 2000; Campagna et al. 2004.</p>
Neurological effects	<p>A group of 74 healthy subjects (35 females and 39 males, mean age = 24.7 ± 4.2 SD; mean years of education = 16.5 ± 2.4 SD) were exposed to 0.05, 0.5 and 5 ppm (0.07, 0.7 and 7 mg/m³) hydrogen sulfide in a random order for 2 hours over 3 weeks in an exposure chamber. It should be noted that effects were compared before and after exposure for each subject, and a non-exposed control group was not included in the study. Odour ratings, sensory function (postural sway, visual acuity and visual contrast sensitivity), cognitive tests (simple reaction time and continuous performance tests, finger tapping test, symbol–digit substitution test, auditory verbal learning test) were examined. Significant effects were observed for odour detection, irritation and anxiety following hydrogen sulfide exposure over time at all exposure levels. A significant decline in cognitive recall through auditory verbal learning was observed at all exposure levels. As no significant effects on other sensory or cognitive measures were observed, the authors suggested that the decline in verbal learning could be due to fatigue (Fiedler et al. 2008).</p> <p>In a cohort study, 103 subjects were exposed to various durations of low-level environmental exposure of hydrogen sulfide from 1 to 22 years prior to the assessment. In some cases, exposure levels were not reported; in others, they were estimated based on various measured levels. Some subjects were concurrently exposed to other substances. A number of neurobehavioural deficits were identified in the subjects, including alterations in balance, visual fields, choice reaction time, colour discrimination, grip strength and delayed verbal recall (Kilburn 1997, 1999).</p> <p>In another cohort study, neurobehavioural assessment was conducted in 19 subjects who had been environmentally exposed</p>

Endpoint	Lowest effect levels/results
	<p>to hydrogen sulfide for 20 min to 9 years (exposure levels were not quantified). The assessment occurred 1.7–22 years after exposure, and none of the subjects had been unconscious from the exposure. A referent population was used for comparison. Exposed subjects exhibited deficits in reaction time, balance, blink reflex, colour error scores, visual performance, grip strength, hearing and a number of cognitive parameters (Kilburn 2003).</p> <p>Other studies: Kilburn and Warshaw 1995; Hirsch 2002; Inserra 2004; Farahat and Kishk 2010.</p>
Case studies	<p>A number of case studies of hydrogen sulfide exposure were reported where concentrations and durations of exposure were usually unquantified and co-exposure to other chemicals was frequent. Some of the effects reported include loss of consciousness, death, pulmonary, intracranial and cerebral edema, hemorrhagic bronchitis, chest pain, respiratory distress, bradycardia, cardiac arrhythmias and irregularities, increase in blood pressure, cyanosis, nausea, vomiting, headache, dyspnea, eye irritation and other ocular effects, such as visual impairment, photophobia and corneal erosion, nasal irritation, reduced hearing, hemoptysis, neurological effects such as coma, seizures, dizziness, dementia, decreased ability to communicate, decreased attention and concentration, memory impairment, impaired visual perception and coordination, impaired motor function, ataxia, cerebral atrophy and irritability (Allyn 1931; Ahlborg 1951; McDonald and McIntosh 1951; Spolyar 1951; Breyse 1961; Milby 1962; Krekel 1964; Adelson and Sunshine 1966; Thoman 1969; Simson and Simpson 1971; Burnett et al. 1977; Osbern and Crapo 1981; Hagley and South 1983; Beauchamp et al. 1984; Arnold et al. 1985; Audeau et al. 1985; Deng and Chang 1987; Luck and Kaye 1989; Wasch et al. 1989; NIOSH 1991; Parra et al. 1991; Tvedt et al. 1991a,b; Kilburn 1993; Snyder et al. 1995; Hall and Rumack 1997; Watt et al. 1997; Fenga et al. 2002; Kage et al. 2002, 2004; Nelson and Robinson 2002; CSB 2003; Hendrickson et al. 2004; Nam et al. 2004; Nikkanen and Burns 2004; Smith and Cummins 2004; Miyazato et al. 2013; Sastre et al. 2013).</p>
Reproductive and developmental effects	<p>In a retrospective study of petrochemical workers in China, 2853 female workers (from oil refinery, chemical, polyester, resin, carpet and non-chemical plants) including 1620 women reported exposure to petrochemicals. A significantly increased risk of</p>

Endpoint	Lowest effect levels/results
	<p>spontaneous abortions was identified in workers with frequent petrochemical exposures (OR = 2.7, 95% CI = 1.8–3.9). The possible confounders of age, education level, plant, shift of work, standing and kneeling at work, noise level, dust level, passive smoking and diets were adjusted for. When the risk associated with hydrogen sulfide exposure was examined, the OR was 2.3 with 95% CI = 1.2–4.4. Also, elevated ORs were observed for benzene (OR = 2.5 with 95% CI = 1.7–3.7) and gasoline (OR = 1.8 with 95% CI = 1.1–2.9). However, no exposure information regarding the first trimester was available. Odds ratios for other effects were not investigated (Xu et al. 1998).</p> <p>Neurodevelopmental effects In a case study, a 20-month-old child was exposed to at least 0.6 ppm (0.84 mg/m³) hydrogen sulfide and other unspecified chemicals emitted from a coal mine for nearly 1 year. The child was admitted to the hospital with ataxia, choreoathetosis, dystonia and inability to stand, where brain scan suggested toxic encephalopathy. Shortly after admission, the child recovered spontaneously. After 10 weeks of admission, ataxia had resolved, and choreoathetoid movements were reduced. Repeated brain scan was normal (Gaitonde et al. 1987).</p>
Epidemiological study	<p>A number of ecological epidemiological, community-based and sulfate mill studies were available. Exposure levels were usually unquantified, and co-exposure to a number of other chemicals was common. In most cases, inadequate data were available to draw conclusions of possible correlations between hydrogen sulfide exposure and health effects. Some of the health effects assessed include mortality, cataracts, conjunctiva disorders, orbit disorders, nervous system disorders, sense organ disorders, respiratory system-related disorders and cancers (Burnett et al. 1977; Hemminki and Niemi 1982; Arnold et al. 1985; Schechter et al. 1989; Jaakkola et al. 1990; Jappinen and Tola 1990; Haahtela et al. 1992; Marttila et al. 1994a,b, 1995; Kilburn and Warshaw 1995; Partti-Pellinen et al. 1996; Bates et al. 1997, 1998, 2002; Legator et al. 2001; Lewis et al. 2003; Thorn and Beiger 2004).</p>

Abbreviations: CI, confidence interval; GD, gestation day; OR, odds ratio; PM₁₀, particulate matter less than or equal to 10 µm in diameter; PND, postnatal day; RER, respiratory exchange ratio; SD, standard deviation; LC₅₀, median lethal concentration; LOAEC, lowest-observed-adverse-effect concentration; LOEC, lowest-observed-effect concentration; NOEC, no-observed-effect concentration