**ASSESSMENT REPORT ON
REDUCED SULPHUR COMPOUNDS
FOR DEVELOPING AN
AMBIENT AIR QUALITY GUIDELINE**

Submitted to:

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SUMMARY

Reduced sulphur compounds (RSC) are a complex family of substances. They are defined by the presence of sulphur in a reduced state and are generally characterized by strong odours at relatively low concentrations. One member of the family—hydrogen sulphide (H_2S)—is known to have highly toxic properties, and can cause negative health effects at low concentrations.

Natural sources of RSC in air include volcanoes and sulphur springs, oceans and estuaries, and exposed faces of sulphur-containing oil and coal deposits. The primary anthropogenic sources include oil and gas processing facilities, Kraft pulp mills, chemical manufacturing plants, and livestock operations. Concentrations of RSC in air are generally elevated in the immediate vicinity of the source, and transport and chemical reactions in the air decrease ambient concentrations and alter the chemical composition of air containing reduced sulphur compounds.

Health effects associated with exposure to RSC are diverse, being dependent upon the specific RSC, the conditions of exposure, and the sensitivity of the individual exposed. Health effects of H_2S were not reviewed as a part of this assessment. Health effects levels for other RSC identified are: 17 ppm (methyl mercaptan), 10 ppm (n-butyl mercaptan), 2,770 ppm (ethyl mercaptan), 9 ppm (t-butyl mercaptan), 7,300 ppm (propyl mercaptan), 2 ppm (methyl disulphide), and 1 ppm (carbon disulphide). A human effects level for carbonyl sulphide was not identified in the literature. These health effects levels identified are above the published odour thresholds for human detection.

The literature describing vegetation effects is limited and is focused on the responses of plants to H_2S. The vegetation effects level for H_2S is 140 μg/m^3, three to four times higher than the human odour detection threshold. The vegetation effects levels for methyl mercaptan is substantially above the odour detection threshold, and is higher than the human effects level. Similarly, the vegetation effects level for carbonyl sulphide is higher than that identified for human effects.

Measurement and monitoring of RSC in an air sample is most commonly conducted by first removing SO_2 from the sample, converting all reduced sulphur compounds in the sample to SO_2 and measuring the resulting concentration of SO_2 in the sample. Separation of individual RSC in air can be accomplished by using gas chromatography, with the individual RSC being quantified using a flame photometric, flame ionization or photoionization detector. Detection limits range from 0.4 ppb to 0.25 ppm, depending upon the choice of analytical system.

Several jurisdictions, including Alberta, have established guidelines for RSC, either for individual members of the RSC family or for the total concentration of RSC in air. The majority of these guidelines and similar management tools are based upon the odour threshold. Total RSC in air is generally assumed to be composed H_2S, methyl mercaptan, dimethyl sulphide and dimethyl disulphide, which together account for 95% of the total reduced sulphur compounds in the air sample. Total RSC in air is generally expressed as H_2S equivalents.

Approaches considered for application in Alberta include establishment of guidelines for single RSC in air, development of an index that includes two or more RSC, use of one RSC as a surrogate for one or more other RSC, use of odours as the basis for RSC guideline(s), and an integrated guideline that incorporates an effects-based component and an emissions management component. Due to the scarcity of effects information for RSC other than H_2S, continued use of odour thresholds appears appropriate.

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1.0 INTRODUCTION

Alberta Environment (AENV) is in the process of developing an Ambient Air Quality Guideline (AAQG) for Reduced Sulphur Compounds (RSC). In support of the guideline development, AENV and associated stakeholders require documentation regarding the potential effects of RSC on human health and the environment.

Reduced sulphur compounds (RSC) are a group of inorganic and organic chemicals containing sulphur atoms in their lowest oxidation state (S^{-2}). The group includes hundreds of individual species, of which only a few commonly occur in air. A list of most common RSC is given in Appendix A. Considering toxic properties, commercial use, concentration level in the atmosphere and availability of data the most important substances within the RSC group are hydrogen sulphide (H_2S), methyl mercaptan (CH_3SH), dimethyl sulphide (C_2H_6S), dimethyl disulphide ($C_2H_6S_2$), carbon disulphide (CS_2) and carbonyl sulphide (COS). Of these six, hydrogen sulphide, methyl mercaptan, dimethyl sulphide, and dimethyl disulphide are the four reduced sulphur species most often emitted from industrial operations and are present in air. Even when other RSC are present, these four substances generally account for 95% of the RSC in the air (Nebraska guideline document, Appendix B; Ontario personal communication). The remaining RSC species are generally present in small amounts and/or are present only at specific locations.

The objective of this report is to provide background scientific and technical information on RSC in support of developing an AAQG. The information is presented includes:

- general substance information such as physical and chemical properties of the most common RSC;
- management of RSC in air;
- existing and potential natural and anthropogenic emission sources in Alberta;
- ambient concentrations of RSC in air;
- monitoring techniques for RSC in air; and
- health effects of RSC exposure to humans, animals, and vegetation.

2.0   GENERAL SUBSTANCE INFORMATION

2.1   Hydrogen Sulphide

Physical and chemical properties of hydrogen sulphide are as follows (HSDB, 1998):

Synonym(s):	hydrogen sulfide, dihydrogen sulphide, hydrogen sulphur
Molecular Formula:	H_2S
Molecular Weight:	34.08 g/mol
CAS Number:	7783-06-4
Appearance:	colourless gas, sweetish taste
Odour:	offensive strong odour of rotten eggs
Boiling Point and Melting Point:	212.82 K & 187.66 K
Vapour Pressure:	2.08 MPa @ 298 K
Critical Temperature and Pressure:	373 K & 9.01 MPa
Octanol/Water Partition Coefficient:	no data
Solubility in Water at 293 K:	4.13 g/dm^3
Specific Gravity as Gas (Air = 1):	1.19
Dissociation Constant:	pKa_1 = 7.04; pKa_2 = 11.96
pH:	4.5 in fresh water solution
Heat of Vaporization:	14.08 kJ g/mol @ 298 K
Concentration Conversion Factor:	1 ppm = 1.42 mg/m^3

Hydrogen sulfide does not absorb solar radiation reaching the troposphere and thus is photochemically stable.

The atmospheric lifetime of H_2S is affected by ambient temperature and other ambient conditions including humidity, sunlight, and presence of other pollutants. The decreased temperatures and sunlight as well as decreased levels of hydroxide radicals in northern regions, such as Alberta, in winter increase the atmospheric residence time of H_2S (Bottenheim and Strauss, 1980).

Once released into air, H_2S will behave like many other gaseous pollutants and be dispersed and eventually removed. Residence times in the atmosphere range from about one day to more than 40 days, depending upon season, latitude, and atmospheric conditions.

H_2S released to the atmosphere is oxidized in reactions with OH˙ radicals. The probable mechanism of this hydrogen absorption reaction is as follows:

$$H_2S + OH^\bullet = H_2O + SH^\bullet$$
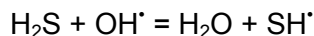
with the reaction rate constant $k_{298} = (4.5 \pm 1.0) \times 10^{-12}$ cm^3 molecule/sec.

Dependency of the rate constant (k) on temperature (T) follows the Arrhenius equation:

$$k = A\, e^{-E/RT}$$

where A = the Arrhenius constant (frequency factor)
E = activation energy
R = ideal gas constant

The $SH^{\bullet}$ radical is oxidized to a transient molecule HSO_3, and then to sulphuric acid (H_2SO_4) as a final product. Other parallel reactions of H_2S with other oxidants such as NO_2, O_2 and O_3 are not important, being much slower than oxidation with $OH^{\bullet}$ radical (NRCC, 1981;Tyndall and Ravishankara, 1991).

2.2 Methyl Mercaptan

Properties of methyl mercaptan are as follows (HSBD, 1998):

Synonym(s):	mercaptomethane, methylmercaptan, methyl sulfhydrate
Molecular Formula:	CH_3SH
Molecular Weight:	48.11 g/mol
CAS Number	74-93-1
Appearance:	colourless gas, water-white liquid when below boiling point, cabbage taste
Odour:	unpleasant rotten cabbage or garlic
Boiling Point and Melting Point:	279.1 K at 101.3 kPa & 150 K
Heat of Vaporization:	24.59 kJ g/mol @ 298 K
Vapour Pressure:	202.6 kPa @ 299.2 K
Critical Temperature and Pressure:	469.9 K & 7.23 MPa
Octanol/Water Partition Coefficient:	log Kow = 0.78
Solubility in Water at 293 K:	23.3 g/dm^3
Specific Gravity as Gas (air = 1):	1.66
Dissociation Constant:	pKa = 10.33 at 298 K
Concentration Conversion Factor:	1 ppm = 2.05 mg/m^3

Methyl mercaptan (CH_3SH) is oxidized by air and in the presence of atmospheric oxidizing agents and metal ions that catalyze the oxidation. If released from land, gaseous CH_3SH will adsorb strongly to the soil and may oxidize. When released in water, CH_3SH is lost through volatilization (estimated half-life 2 hours in a model river) and may be oxidized in the water prior to release.

Concentrations of methyl mercaptan in the troposphere decrease as a result of daytime oxidation by $OH^{\bullet}$ radical and nighttime reaction with the NO_3 molecule:

$$CH_3SH + OH^{\bullet} = CH_3S^{\bullet} + H_2O$$
$$k_{298} = (2.1 \text{ to } 9.04) \times 10^{-11} \text{ cm}^3 \text{ molecule/sec}$$

and

$$CH_3SH + NO_3 = \text{products}$$
$$k_{298} = (0.9 \pm 0.2) \times 10^{-12} \text{ cm}^3 \text{ molecule/sec}$$
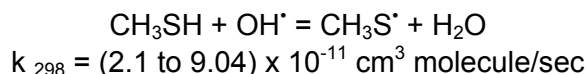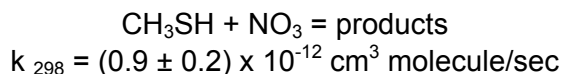
The effective lifetime of CH_3SH in the troposphere is 1 to 26 hours assuming OH˙ concentration equal to 5 x 10^5 radicals/cm^3, depending upon temperature, light and other environmental parameters (Tyndall and Ravishankara, 1991; US EPA, 1985). Under photochemical smog conditions, the half-life is 2 h.

2.3 Dimethyl Sulphide

Following is a summary of physical and chemical properties of dimethyl sulphide (Merck, 1996):

Synonym(s):	dimethyl sulfide, dimethyl monosulfide, dimethyl thioether, thiobismethane, DMS
Molecular Formula:	C_2H_6S
Molecular Weight:	62.14 g/mol
CAS Number:	75-18-3
Appearance:	colourless to straw-colored liquid
Odour:	unpleasant odour
Boiling Point and Melting Point:	310.4 K & 174.8 K
Heat of Vaporization:	28.09 kJ g/mol @ 298 K
Vapour Pressure:	66.9 kPa @ 298 K
Critical Temperature and Pressure:	502 K & 5.68 MPa
Octanol/Water Partition Coefficient:	log Kow = 0.92
Solubility in Water at 298 K:	22 g/dm^3
Specific Gravity as Gas (air = 1):	2.14
Concentration Conversion Factor:	1 ppm = 2.54 mg/m^3

The rate constant for the vapor-phase reaction of dimethyl sulphide (C_2H_6S) with photochemically-produced hydroxyl radicals has been measured as 4.6 x 10^{-12} cm^3 molecule/sec at 298 K. This corresponds to an atmospheric half-life of about 3.5 days at an atmospheric concentration of 5x 10^5 hydroxyl radicals per cm^3 (Atkinson, 1989). A diurnal cycle of C_2H_6S concentration has been reported, which is thought to be consistent with the removal of C_2H_6S during the daytime by hydroxyl radical reaction (Andreae, 1993). The half-life for C_2H_6S reacting with atomic oxygen in the atmosphere has been estimated by Winer (1984) to be 6.2 days based on a reaction rate constant of 5.16 x 10^{-11} cm^3 molecule/sec at 298 K and an average ambient atomic oxygen concentration of 2.5 x 10^4 molecules/cm^3. He also estimated the half-life for nighttime reaction of C_2H_6S with gaseous nitrate radicals in air over the continental US to be on the order of several hours in a clean atmosphere and an hour in a moderately polluted atmosphere. The atmospheric half-life in a presence of nitrate radicals has been estimated to be about 28 minutes at an atmospheric concentration of 5 x 10^8 nitrate radicals per cm^3 (Carlier, 1988).

Major atmospheric sinks for dimethyl sulphide are reactions with OH and NO_3. In marine environments, NO_3 levels are typically low and dimethyl sulphide is removed primarily by OH in the chemical reactions:

$$(1a) \quad OH + CH_3SCH_3 = CH_3SCH_2 + H_2O3$$

and

$$(1b) \quad OH + CH_3SCH_3 = CH_3S(OH)CH_3$$
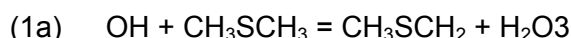
Despite the rather large database, neither the rate constant nor the branching ratio for the above reactions are well defined (Hynes *et al.*, 1986).

The photochemical degradation of C_2H_6S in the atmosphere results in the production of sulfur dioxide and methane sulphonic acid as well as minor products such as dimethyl sulphoxide (DMSO), dimethyl sulphone, and sulphuric acid (Kelly and Smith, 1990). The series of reactions are shown below (Shon *et al.*, 2001):

$$CH_3SCH_2 + O_2 = CH_3SCH_2OO$$
$$CH_3SCH_2OO + NO = CH_3SCH_2O + NO_2$$
$$CH_3SCH_2O = CH_3S\ HCHO$$
$$CH_3S + NO = CH_3SNO$$
$$CH_3S + O_2 = (CH_3SO_2) = CH_3 + SO_2$$
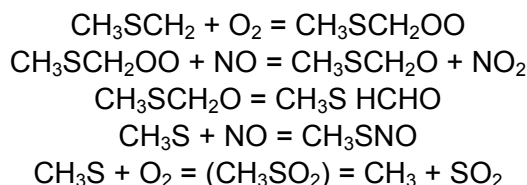
2.4 Dimethyl Disulphide

Basic physical and chemical properties of methyl disulphide ($C_2H_6S_2$) are as follows:

Synonym(s):	Methyl disulphide, methyldithiomethane
Molecular Formula:	$C_2H_6S_2$ or $(CH_3)_2$
Molecular Weight:	94.20 g/mol
CAS Number:	624-92-0
Appearance:	Liquid
Odour:	unpleasant odour of wild radish, cabbage-like
Boiling Point and Melting Point (^oC):	382.9 K & 188.1 K
Heat of Vaporization:	33.78 kJ g/mol @ 382.9 K
Vapour Pressure:	3.82 kPa @ 298 K
Critical Temperature and Pressure:	no data available
Octanol/water Partition Coefficient:	log Kow = 1.77
Solubility in Water:	Insoluble
Specific Gravity as Gas (Air = 1):	3.24
Specific Gravity as Liquid (Water = 1):	1.0625
Concentration Conversion Factor:	1 ppm = 3.92 mg/m^3

In air, $C_2H_6S_2$ will exist at a vapor pressure of 3.83 kPa (28.7 mm Hg) at 298 K (25°C). It will be degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 4 hours (Atkinson, 1994). Direct photolysis (half-life of 3.2-4.6 hours at full sunlight) and reaction with nitrate radicals (half-life of 1.1 hours during night-time hours) will also contribute to its atmospheric transformation (Yin *et al.*, 1990).

The rate constant for the vapor phase reaction of $C_2H_6S_2$ with photochemically-produced hydroxyl radicals has been experimentally determined to be 2.39 x 10^{-10} cm^3 molecule/sec at 298 K. This corresponds to an atmospheric half-life of about 4 hours at an atmospheric concentration of 5 x 10^5 hydroxyl radicals per cm^3; the initial reaction of the hydroxyl radical with $C_2H_6S_2$ is by addition with the final reaction products of this reaction including SO_2, methyl sulphonic acid, sulfuric acid, and methanethiol (Atkinson, 1994).

The rate constant for the vapor phase reaction of $C_2H_6S_2$ with ground-state oxygen atoms has been experimentally determined to be 1.02 x 10^{-10} cm^3 molecule/sec at 298 K, which corresponds to an atmospheric half-life of about 3 days at an atmospheric concentration of 2.5 x 10^3 O$_2$ molecules per cm^3 (Nip, 1981).

Methyl disulphide absorbs UV light in the environmental spectrum between 290 and 400 nm; it has been observed to photolyze at 360 nm. Based upon its absorption spectrum and the calculated actinic flux of solar radiation at the Earth's surface, the rate constant for its direct photolysis is estimated to be roughly 0.0025/min at noon (July 1, clear skies) at a latitude of 40° North; this corresponds to a half-life of about 4.6 hours (Yin *et al*., 1990). Under clean air conditions, the half-life for the direct photolysis of methyl disulphide in natural sunlight was observed to be 3.2 hours (Sickles, 1978).

2.5 Carbon Disulphide

Properties of carbon disulphide (CS_2) are summarized as follows:

Synonym(s):	carbon bisulfide, carbon sulfide, carbon disulphide
Molecular Formula:	CS_2
Molecular Weight:	76.14 g/mol
CAS Number:	75-15-0
Appearance:	Liquid
Odour:	foul smelling commercial grades, sweetish aromatic when pure
Boiling Point and Melting Point:	319 K & 161.6 K
Heat of Vaporization:	26.72 kJ g/mol @ boiling point
Vapour Pressure:	47.9 kPa @ 298 K
Critical Temperature and Pressure:	552 K & 7.9 MPa
Octanol/water Partition Coefficient:	log Kow = 1.94
Solubility in Water:	2860 mg/dm^3
Specific Gravity as Gas (Air = 1):	2.67
Specific Gravity as Liquid (Water = 1):	1.2632
Concentration Conversion Factor:	1 ppm = 3.16 mg/m^3

At the ambient temperature of 298 K (25°C) CS_2 occurs in the gas phase with a vapor pressure of 47.8 kPa (359 mm Hg). Carbon disulphide reacts in the atmosphere with photochemically produced hydroxyl radicals; the half-life for this process is estimated to be 5.5 days. It has a weak UV adsorption band at 317 nm, suggesting a potential for direct photolysis (HSDB, 2001).

The rate constant for the vapor-phase reaction of CS_2 with photochemically-produced hydroxyl radicals is 2.9 x 10^{-12} cm^3 molecule/sec at 297 K. Observed temporal variability and vertical gradients suggest that the tropospheric lifetime of CS_2 is rather short (HSDB, 2001).

2.6 Carbonyl Sulphide

Physical and chemical properties of carbonyl sulphide (Beilstein, 2002):

Synonym(s):	carbon oxysulphide, carbon monoxide monosulfide
Molecular Formula:	COS
Molecular Weight:	60.08 g/mol
CAS Number:	436-58-1
Appearance:	colourless gas
Odour:	typical sulphide when pure
Boiling Point & Melting Point:	223 K & 135 K
Heat of Vaporization:	18.506 kJ g/mol @ 222.9 K
Vapour Pressure:	1.25 MPa @ 298 K
Critical Temperature & Pressure:	378.8 K & 6.35 MPa
Octanol/Water Partition Coefficient:	log Kow = 0.8009 (estimated)
Solubility in Water at 293 K:	1440 mg/dm^3
Specific Gravity as Gas (Air = 1):	2.1
Specific Gravity as Liquid (Water = 1):	1.028
Concentration Conversion Factor:	1 ppm = 2.49 mg/m^3

The atmospheric life-time of carbonyl sulphide (COS), based on removal via the gas-phase reaction with photochemically produced hydroxyl radicals and oxygen, direct photolysis, and unknown removal processes invoked to balance the sulfur cycle, is approximately two years (Baulch, 1984; Khalil and Rasmussen, 1984). Studies conducted by Mihalopoulos et al. (1989), Bremner and Banwart (1976) have shown that COS may be adsorbed from the atmosphere by plants and by moist soil, the latter due to microbial uptake. Experimental rate constants for the gas-phase reaction of COS with photochemically produced hydroxyl radicals ranging from 6.08×10^{-12} cm^3 molecules/sec to 8.8×10^{-15} cm^3 molecules/sec and translate to atmospheric half-lives ranging from 500 days to 6.5 years (Cox, 1980). Carbonyl sulphide is not expected to undergo direct photolysis in the troposphere but it may in the stratosphere.

3.0   REDUCED SULPHUR COMPOUND SOURCES

In heavily industrialized regions such as the eastern United States and western Europe, anthropogenic sulphur emissions exceed natural emissions by about an order of magnitude (Galloway and Whelpade, 1980, Möller, 1984). On a global scale, however, anthropogenic sources emissions are thought to approximately equal those from natural sources (Cullis and Hirschler, 1980, Granat *et al*., 1976).

3.1   Natural Sources of RSC

An understanding of the natural sulphur cycle is required in order to establish a base line with which anthropogenic perturbations can be compared. The global sulphur cycle has been extensively studied over the last few decades, a process that has been accelerated in recent years due to concern over the potential role of sulphate in visibility reduction, acid rain and climate change. Prior to the 1960s, the question of the role of reduced sulphur in the global sulphur cycle was open. However, it has been shown since then that the combined effects of hydrogen sulphide, dimethyl sulphide, carbonyl sulphide, and carbon disulphide are minor in relation to the oxidized forms of sulphur represented in sulphur dioxide and sulphates (Bates *et al*., 1992). Biogenic sulphur emissions are related to either the aerobic generation of methylated sulphur forms during normal metabolism or to H_2S and dimethyl sulphide generation during anaerobic decomposition of organic residues (NRCC, 1977). Most of the global biogenic sulphur emissions have been observed over open oceans and coastal waters.

In addition to oceans, other natural sources of RSC include volcanoes, biomass burning, and soil, oceans, marshes, and tidal flats where anaerobic decay of organic matter is taking place (NRCC, 1981).

Rough estimation of the global atmospheric sulphur budget has been published by Schlesinger (1997) in the unit of Tg S per year (1 Tg = 10^{12} g):

Input   Natural emissions on land (volcanoes, dust, Biogenic) = 17
                  Human activity = 90
                  Sea salt = 144
                  Sea biogenic = 16
                  Sea volcanoes = 5
                  Total input = 272 ± 2

Output   Deposition on land = 90
                  Ocean deposition = 180
                  Total output = 270 ± 2

Schlesinger (1997) quantified annual global natural emissions of volatile sulphur compounds, including RSC, to the atmosphere as shown in Table 1.

**Table 1: Global Natural Emissions of Volatile
Sulphur Compounds to the Atmosphere (Tg/y)**

Source	SO$_2$	H$_2$S	(CH$_3$)$_2$S	CS$_2$	COS	Total
Oceanic	0-15	38-40	0.3	0.4	0.4	38-57
Salt marsh	–	0.8	0.6	0.1	0.1	1.7-1.8
Inland swamps	–	11.7	0.8	2.8	1.8	17
Soil and plants	–	3-41	0.3	0.6-1.5	0.2-1.0	5-48
Burning of biomass	7	0-1	0-1.9	–	0-0.1	7.1-9.1
Volcanoes	8	1	–	–	–-	9

The global sulphur budgets continue to have large uncertainties in their various components. To some extent this is to be expected since enhanced anthropogenic activity leading to increasing sulphur emissions perturbs various components of the sulphur biogeochemical cycle including natural sulphur emissions.

Additional natural emissions information for selected RSC are presented in the following sections.

3.1.1 Hydrogen Sulphide

H$_2$S occurs naturally in and is released to air from sulfur springs, volcanic gas, natural gas, and crude petroleum. High levels of H$_2$S have been found in some water wells that may be responsible for corroding pipes. Volcanic emissions of H$_2$S can be significant. For example, Mount St. Helens emits 65 tons of H$_2$S per day, representing 7.2 percent of global H$_2$S emissions (Lambert, 2000).

3.1.2 Methyl Mercaptan

Methyl mercaptan is released to the atmosphere from both natural and anthropogenic sources. Natural sources include vegetation, animal wastes, microbial degradation, and natural gas (Adams *et al.,* 1979; Farwell *et al.,* 1979; Graedel *et al.,* 1978; Reid 1958). The global flux of methyl mercaptan has not been estimated because it converts to dimethyl disulphide when collected for analysis. At lower concentrations, methyl mercaptan likely contributes not more than 10% to the global natural sulphur budget (Tyndall and Ravishankara, 1991).

Estimation of average methyl mercaptan emission from a saline marsh in North Carolina was 6.56 g S/(m^2 year) (Adams *et al*., 1979). It occurs in small quantities in a variety of vegetables, such as garlic and onion (Clayton and Clayton, 1982).

Methyl mercaptan is formed in the anaerobic fermentation of gelatin and albumin and in the putrefaction of proteins (Reid, 1958). It is formed in the intestinal tract by the action of anaerobic bacteria on albumin (Merck, 1989). West Texas sour gas contains sufficient amounts of methyl mercaptan that commercial quantities are extracted from it (Reid, 1958).

3.1.3 Dimethyl Sulphide

Dimethyl sulphide is the major volatile reduced organic sulfur compound in open ocean(Turner and Liss, 1985, Andreae *et al*., 1985), coastal waters, and marshlands, and its emission from surface water represents a major flux of biogenic reduced sulfur to the atmosphere. Emission rates ranged from 0.02 to 1.87 g S/m^2 y, which translates to the global flux of 39 Tg S/y. Inland soils emitted dimethyl sulfide at rates from 0.0003 to 92.8 g S/m^2 y. Emissions of dimethyl disulphide from five wetland soils in Florida ranged from < 0.02 to 10 µg S/m^2 h (Cooper, 1987).

Values for biogenic emissions of dimethyl sulphide from open oceans and marine environments range from 0.02 to 0.106 g S/m^2 y (Aneja, 1990).

Dimethyl sulphide occurs from the decomposition of plants and animals and from sulfur-containing amino acids during digestion by microorganisms in the rumen of ruminants. In small quantities, it accompanies methyl mercaptan in natural gas (Clayton and Clayton, 1982).

3.1.4   Dimethyl Disulphide

Dimethyl disulphide is released to the atmosphere in emissions from animal waste, food decay, microbes, natural gas, vegetation, oceans and soil (Graedel *et al.*, 1986). Emissions of dimethyl disulfide from five wetland soils in Florida were the result of biogenic formation (Cooper, 1987). Under waterlogged conditions, dimethyl disulfide is formed by microbial degradation of methionine and carbon disulphide from cysteine and cystine (Cooper, 1987).

Plants, including *Spartina alterniflora*, oak and pine trees, emit dimethyl disulfide into the atmosphere (Aneja, 1990). Dimethyl disulfide is also produced by actinomycetes, resulting in concentrations from 0.41 to 6.8 mg m^{-3} (Wilkins, 1996).

3.1.5   Carbon Disulphide

Major global sources of carbon disulphide are seas and oceans (Carroll, 1985, Khalil and Rasmussen, 1984). Existing data suggest that coastal areas and other areas of high biological productivity have greater fluxes of carbon disulphide than the open ocean (Khalil and Rasmussen, 1984). Emissions from the oceans have been estimated to be 0.6 Tg/y and from soil 0.9 Tg/y (Khalil and Rasmussen, 1984). Other natural sources include volcanic emissions, estimated to be 20 Gg/y, and marshlands, estimated emissions 0.1 Tg/y (Khalil and Rasmussen, 1984).

Terrestrial diurnal variation in emissions rates depends on soil temperature and solar irradiation. Emissions rates were highest in the early- to mid-afternoon and lowest during the early morning (Cooper, 1987). Carbon disulphide emissions from a temperate pine forest increased nine-fold when nitrogen fertilizers were added.

3.1.6   Carbonyl Sulphide

Estimated emissions of carbonyl sulfide (COS) from natural sources were estimated by Kelly and Smith (1990) at the total level of 2.7 to 3.5 Tg S/y. Contribution of individual sources, in Tg of sulphur per year, were as follows:

*   Oceanic emissions = 0.4;
*   Salt marshes = 0.12;
*   Inland swamps = 1.85;
*   Soil and plants = 0.2 – 1.0;
*   Burning of biomass = 0.11; and
*   Volcanoes and fumaroles = 0.01.

Emissions of COS have been associated with deciduous trees, coniferous trees, salt marshes and soils. The mean COS emissions from a hardwood forest were 8.66 µg S/day and 19.86 µg S/day from a pine forest (Aneja, 1990). The average emission of COS from five different soil types was found to range from 0.0014 to 6.36 g S/m^3 y (Adams, 1979).

3.2 Anthropogenic Sources of RSC

The primary anthropogenic sources of reduced sulphur compounds include Kraft pulp mills, natural gas wells, processing of natural gas and crude oil at upstream stages and downstream refining, smelting of non-ferrous ores, steel mills, the manufacture of certain abrasives, livestock farming, and sewage treatment facilities.

The most significant discharges of RSC (excluding discharges of H$_2$S by oil and gas industry) are from Kraft pulp mills. There are many sources of RSC in the mill. Some sources emit a small gas volume with high concentrations of RSC, while others have large volumes with low concentrations. Non-condensable gas (NCG) emissions occur at numerous points in a mill. They fall into three types, chiefly according to their RSC concentration:

1) Low volume, high concentration gases (LVHC gases) from continuous digester relief; blow heat recovery; turpentine recovery vent; evaporator hotwell vent; and foul condensate storage tank;

2) Overhead vapors from the foul condensate steam stripping system. These vapors are a mixture of methanol, water and RSC gases; and

3) High volume, low concentration gases (HVLC gases) from brown stock washer filtrate tanks and hood; weak and strong black liquor storage tanks; knotter hood; black liquor oxidation vent; and contaminated condensate tanks.

A typical (average) volume vs. concentration comparison from Banks(1998) is shown in Table 2.

**Table 2: Comparison of Concentrations of RSC in
High and Low Volume Emissions from Kraft Pulp Mills**

Constituent	High Volume (=30)/ Low Concentration (ppm)	Low Volume (=1)/ High Concentration (ppm)
H$_2$S	0.1	500-6,000
CH$_3$SH	0.1	15,000 - 50,000
(CH$_3$)$_2$S	0.2	15,000
(CH$_3$)$_2$S$_2$	0.3	10,000
Air	High	High

Table 3 shows a typical analysis of an NCG gas stream (Santos and Backlund, 1992) from a Kraft pulp mill.

Table 3: Typical Non-Condensable Gas Analysis by Volume

Component	Content (%)
H$_2$S	1.7

CH$_3$SH	2.1
CH$_3$SCH$_3$	2.1
CH$_3$SSCH$_3$	1.7
Turpentine	0.1
Methanol	0.2
Water Vapor	6.0
Nitrogen	77.2
Oxygen	8.9

Reduced sulphur compounds are not included within the Environment Canada Common Air Contaminants inventory. Only recently, H$_2$S and CS$_2$ have been incorporated within the National Pollutant Release Inventory (NPRI) with data collection beginning 1999. In Alberta some Kraft pulp mills have to report RSC emissions, and sour gas plants have to report H$_2$S emissions to Alberta Environment as a requirement their EPEA operating approvals.

3.2.1   Hydrogen Sulphide

Hydrogen sulphide is a by-product of many industrial operations, e.g., gas and oil exploration, coking and crude oil and coal hydrodesulfurization. H$_2$S emission sources associated with gas and oil exploration and production include exploration, well-site preparation, drilling, waste pits, blowouts, well testing, and gas/liquid separation. Drilling operations are significant sources of short-term RSC emissions, which are usually considered to be a temporary source. During drilling, gas may seep into the well bore and become dissolved or entrained in the drilling mud. The gases are separated from the mud in a separator or degasser. Gases removed from the mud are either vented to the atmosphere or routed to a flare for combustion to SO$_2$.

During the gas sweetening/acid gas removal process, the amine unit is a potential source of SO$_2$, H$_2$S, VOC, HAP and CO emissions. As the amine regenerator heats the amine solution, the acid gases are released through the amine still vent. The amine still vent can be vented directly to the atmosphere, to a flare or incinerator, or to a sulfur recovery unit. Amine units designed to remove only CO from the natural gas, generally, vent directly to the atmosphere. Amine units designed for the removal of H$_2$S and CO generally vent to a sulfur recovery unit. During sulfur recovery, emissions sources in the Selexol process include the process vent streams, fugitive emissions from valves, flanges, and compressor seals, exhaust emissions associated with compressor operation and vented emissions due to periodic maintenance activities.

Emission sources associated with the fixed bed sorption process potentially include the sour gas vent from the flash tank associated with molecular sieve bed regeneration, exhaust emissions from process heaters associated with the regeneration cycle, fugitive emissions, and vented emissions from maintenance activities (US EPA 1980).

The Claus sulfur recovery process emission sources include the tail gas stream, which is usually incinerated or which may be passed through a liquid re-dox sulfur recovery unit. Fugitive emissions from equipment leaks, and emissions from maintenance activities are also

associated with the Claus process. Residual H_2S, COS, and CS_2 may also be released to the atmosphere from the recovered molten sulfur.

Emissions from gas sweetening units are influenced by the concentration of acid gases in the waste gas stream as well as the type of control or recovery process that follows the sweetening process. In the amine gas sweetening process, the amine solution absorbs H_2S, CO_2 and VOCs. If the still vent emissions are vented to a sulphur recovery unit, the H_2S will be converted to elemental sulfur and SO_2. The H_2S content of the tail gas, as well as the efficiency of the sulphur recovery unit, will determine H_2S emissions. Emissions resulting from flashing are impacted by the change in pressure to which the entrained gases are subjected as well as the volume, temperature, and composition of the material being transferred. Flash losses occur from tanks, gun barrels, and separators, as the fluid moves from the high-pressure lines to atmospheric pressure. Under high pressure, the fluid can readily dissolve more gases. As pressures are released from the saturated fluid, the dissolved gases will be released. All other factors being equal, the greater the pressure drop, the greater the gas volume released per barrel of oil produced. The composition of the fluid will also impact emissions of RSC compounds (Merck 1986).

The H_2S releases in Alberta reported to NPRI in reporting years 1999 and 2000 (Environment Canada, 1999, 2000) are summarized in Table 4, with the details given in Appendix C.

Table 4: Summary of Hydrogen Sulphide Releases as Reported to NPRI in 1999 and 2000 (tonnes)

Year	Area	No. of Facilities	On Site	Transfer for Disposal	Transfer for Recycling	Total (tonnes)
1999	Canada	144	126,373	66	904,874	1,031,313
	Alberta	75	34,901	66	0	34,967
2000	Canada	155	154,593	314	928,928	1,083,836
	Alberta	78	50,233	116	0	50,349

The highest on-site releases of H_2S in Canada in 2000 were by William Energy (Canada) Inc., Stoddart Gas Processing Plant in Fort St. John, BC, in a quantity of 78,344 tonnes. In Alberta, the largest releases amounting to 17,985 tonnes were by Apache Canada Ltd., Zama Gas Processing Complex.

3.2.2   Methyl Mercaptan

Methyl mercaptan may be released as fugitive emissions and in wastewater during its production or use in the manufacture of methionine, jet fuels, fungicides, pesticides, and plastics. It is released to the environment in emissions from starch manufacturing, petroleum refining, shale oil production, and rendering plants. The lower mercaptans are split off in the cooking of animal and vegetable food and released to the atmosphere (US EPA, 1987).

Methyl mercaptan and other RSC are present in non-condensable gases in the pulp and paper industry (Tables 2 and 3). The primary sources of non-condensable gases (NCGs) in a Kraft mill

are digesters, evaporators and turpentine recovery systems. Kraft mill RSC emissions can be attributed to four reduced sulfur gases namely: H_2S, methyl mercaptan dimethyl sulphide and dimethyl disulphide. Volatile organic compounds (VOC) other than those containing sulphur are also emitted during digester relief. Typical constituents are alcohols, terpenes, and phenols.

3.2.3 Dimethyl Sulphide

Dimethyl sulphide is released to the environment from wood pulp mills, petroleum refineries, sewage treatment plants, fish processing industries, food decay, leather manufacturing industries, paint, rendering plants, sulfur dioxide scrubbing, and starch manufacturing plants. It is formed in some food items (e.g., beer) during fermentation of yeast (Graedel *et al.,* 1986). Dimethyl sulfide is found in natural gas, crude oil and emissions from some types of vegetation. It also occurs in natural, unrefined oil of peppermint used in flavoring chewing gum and mouthwash.

3.2.4 Dimethyl Disulphide

Dimethyl disulfide emissions to the atmosphere result from fish processing, rendering, sewage treatment, SO_2 scrubbing, starch manufacture, whiskey manufacture and wood pulping (Graedel *et al.,* 1986). Dimethyl disulfide has been detected in the air of refuse waste from a food center (Koe and Ng, 1987), in exhaust gases from pulp mills (Himberg, 1987) and gasoline engines (Hampton, 1982).

Effluent treatment ponds at pulp and paper mills emit dimethyl disulphide and other RSC compounds. The release rate as measured by flux isolation chambers by Tang (1999) is given in Table 5.

**Table 5: Emission Rate of RSC by Wastewater
Treatment Ponds at Pulp and Paper Mills**

Emission ($\mu g\ m^{-2}\ s^{-1}$)	Settling Pond	Primary Pond	Secondary Pond
Dimethyl disulphide	5.15	0.71	0.24
Dimethyl sulphide	2.63	0.62	0.19
Methyl mercaptan	0.98	0.38	0.11
Hydrogen sulphide	0.38	0.11	0.05
TRC (as H_2S)	14.29	2.53	0.83

3.2.5 Carbon Disulphide

Carbon disulphide (CS_2) production and use in the manufacture of rayon, carbon tetrachloride, xanthogenates, soil disinfectants, electronic vacuum tubes, and as a solvent for phosphorus, sulfur, selenium, bromine, iodine, fats, resins, and rubbers may result in its release to the environment through various waste streams.

The CS_2 is emitted from oil refineries, timber processing facilities, gas plants, oil sands operations and sulphides manufacturing plants. Releases of CS_2 have been reported to NRRI. Summary of CS_2 releases in Canada and comparison with Alberta are shown in Table 6 (Environment Canada, 1999, 2000).

**Table 6: Summary of Carbon Disulphide Releases
as Reported to NPRI in 1999 and 2000 (tonnes)**

Year	Area	No. of Facilities	On Site	Transfer for Disposal	Transfer for Recycling	Total (tonnes)
1999	Canada	30	2,875	<1	0	2,876
	Alberta	26	2,762	<1	0	2,762
2000	Canada	35	3,164	1	0	3,165
	Alberta	30	3,028	1	0	3,029

The highest on-site releases of CS_2 in Canada in 2000 were by the Shell Canada Limited Waterton Complex in Pincher Creek, Alberta, in a quantity of 443.6 tonnes. Detailed releases of CS_2 reported to NPRI in Alberta in 1999 and 2000 are given in Appendix D. Alberta contributed almost in 96% to total Canadian CS_2 releases.

3.2.6   Carbonyl Sulphide

The commercial importance of COS is limited; it is not produced in large quantities and its used is only for small-scale synthesis and experiments (Lay, 1986). It may be released to the environment from automobiles, coal-fired power plants, biomass combustion, fish processing, combustion of refuse, and plastics, petroleum manufacture and manufacture of synthetic fibers, starch and rubber (Khalil and Rasmussen, 1984). Carbonyl sulphide may be formed in the atmosphere by the gas-phase reaction of carbon disulphide and photochemically produced hydroxyl radicals.

Carbonyl sulphide is emitted from flue gas desulphurization sludge field storage sites. Adams *et al.*, (1979) estimated emission less than 0.01 to nearly 0.3 kg of sulfur per day for an equivalent 0.4 km^2 (100-acre) sludge impoundment surface.

4.0 AMBIENT AIR CONCENTRATIONS OF REDUCED SULPHUR COMPOUNDS

4.1 RSC Concentrations Near Natural Sources

Background concentrations of RSC result mainly from biological activity within soils, vegetation and water ecosystems. As a result, ambient concentrations vary with seasons and climatic condition, primarily temperature and humidity. During the ice-free growing season (April to October) greater biological activity is taking place and higher gaseous exchange between soil and water and the atmosphere occurs. This results in a large seasonal variability of ambient air concentrations.

Only limited literature data is available for ambient air concentrations of RSC from natural sources. Very few measurements of natural RSC over land have been made. Of all reduced sulphur compounds the most commonly reported are hydrogen sulphide (H_2S) and dimethyl sulphide (C_2H_6S). The first is a commonly occurring RSC that is relatively stable in the atmosphere, and the second has been studied due to its importance from a climate change perspective.

H_2S occurs in air at higher concentrations near sulfur springs, volcanic gas vents, and near areas where crude petroleum sources are exposed (Merck, 1996). Ambient concentrations of H_2S near natural sources tend to be low, in the range of 0.1 to 1 µg m^{-3} (OEHA, 2001). However, these concentrations can be much higher in areas of sulphur springs, unstratified lakes, and geothermal areas. For example, in a geothermal area of New Zealand, continuous monitoring of H_2S revealed concentrations in excess of 80 µg m^{-3} (>50 ppb) during winter months (WHO, 1987).

One of the most interesting ambient measurement studies was conducted at a bird sanctuary on Lake Rotorua in New Zealand. It is also adjacent to the town of Rotorua. The area has significant geothermal activity and sulphur emissions. The analytical method (lead acetate type method) was not very sophisticated and had a high limit of detection (7 ng m^{-3}). Average concentrations of up to 146 ng m^{-3} of reduced sulphur were measured in the town itself. Average concentrations at some locations in the bird sanctuary were as high as 5.4 ng m^{-3} (Siegel *et al*., 1986).

Dimethyl sulphide concentrations over the Canadian Boreal Shield during a time period from July to October 1995 ranged from 5 to 67 ng m^{-3} (Sharma, 1997).

Measurements of H_2S in the remote ocean atmosphere showed concentrations ranging from 42 ng m^{-3} (30 pptv) up to 278 ng m^{-3} (200 pptv). Detection limits for the methods cited were as low as 0.55 ng m^{-3} (US EPA, 1986). Concentrations of H_2S and CH_3SCH_3 in the atmosphere over oceans are given in Table 7.

Table 7: Atmospheric Concentrations of Certain RSC Over Oceans

Region	RSC	Concentration (ng m^{-3})	Date	Reference
Arctic Ocean	CH_3SCH_3	46 to 1,551	July to Aug 1994	Sharma *et al.,* 1999
Gulf of Alaska	CH_3SCH_3	127 to 261	June 1992	Ferek *et al.*, 1995
North Atlantic	CH_3SCH_3	52 to 258	July to Sept 1989	Andreae *et al.*, 1993
North Pacific	H_2S	0.6 to 20.3	March 1990	Yvon *et al.*, 1993
South Pacific (coastal)	CH_3SCH_3	65 to 646	Nov 1988 to May 1990	Ayers *et al.*, 1991

4.2   RSC Concentrations Near Anthropogenic Sources

In the following discussion of ambient air RSC concentrations resulting from anthropogenic sources, RSC and TRS measurements are generally reported. RSC and TRS are primarily composed of H_2S, methyl mercaptan, dimethyl sulphide, and dimethyl disulphide. Where possible, these substances are identified and discussed individually, however, much of the available data and information do not allow separation and discussion of individual RSC from the total (TRS) or family (RSC) of substances discussed.

Concentration measurements of reduced sulphur compounds are usually conducted at locations surrounding industrial sources of RSC, which includes pulp and paper mills that use the Kraft pulping process, steel industry facilities, refineries, tank farms for unrefined petroleum products, some chemical manufacturing plants, and animal feedlots. The monitoring sites also include neighboring communities that are potentially impacted by industry emissions.

In Ontario, a significant drop in annual mean total reduced sulphur concentrations at 12 monitoring sites began in 1991 with further drop in 1992 to a level of 0.8 ppb (1 µg m^{-3}). Mean average ambient TRS (primarily H_2S, methyl mercaptan, dimethyl sulphide, and dimethyl disulphide) levels in 1997 were 50% lower than they were in 1988. This is shown in Figure 1 (OME, 1999). In the year 2000, monitoring for TRS compounds was performed at 10 ambient locations (OME, 2000). The highest annual TRS mean (1.2 ppb) was recorded in Oakville. The maximum 1-hour concentration (101 ppb) was measured at the Windsor West site where the greatest number of hours (26) above the Ambient Air Quality Standard (AAQS) was recorded as well. Elevated TRS levels in Windsor are mainly attributed to transboundary impact from nearby sources in Michigan. At other locations, elevated TRS levels are mainly attributed to the pulp and paper industry.
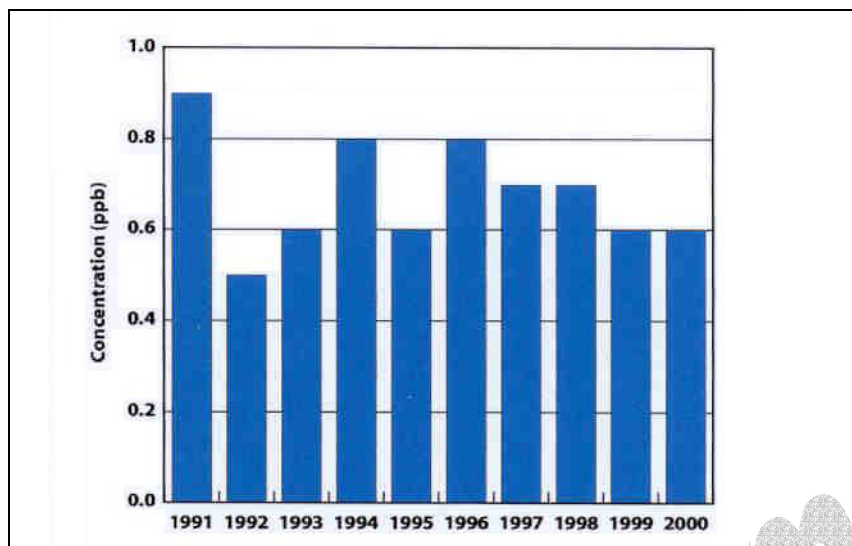
**Figure 1: A Ten-Year Trend for
Annual Mean TRS Levels (ppb) at Ten Sites**

Annual mean concentrations distribution of TRS compounds (ppb) at nine locations in Ontario from 1990 to 1999 showing ten-year trend are given in Table 8.

Table 8: Concentrations of TRS Compounds in Ontario from 1990 to 1999 (ppb)

City	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
Cornwall	3.4	2.9	2.3	1.8	2.5	1.3	1.2	2.0	1.2	0.9
Fort Frances	5.5	4.3	4.2	4.2	2.4	2.5	2.7	2.6	1.4	1.2
Hamilton	0.9	0.8	0.6	0.7	1.1	1.2	1.4	1.0	0.7	0.6
Marathon	1.1	0.9	0.5	0.7	0.9	0.5	0.4	0.2	0.1	0.1
Oakville	1.3	1.6	0.5	1.0	1.3	0.3	1.3	1.3	1.1	1.4
Red Rock	1.7	1.4	2.0	1.9	3.2	1.1	1.2	1.6	2.8	3.8
Terrace Bay	1.3	1.3	1.7	1.7	1.1	1.7	1.3	1.0	1.0	0.9
Tiverton	0.0	0.0	0.1	0.1	0.1	0.2	0.3	0.1	0.2	n/a
Thunder Bay	0.2	0.2	0.3	0.2	0.3	0.3	0.8	1.1	1.3	1.2
Composite Mean	**1.7**	**1.5**	**1.1**	**1.3**	**1.4**	**1.0**	**1.2**	**1.2**	**1.1**	**1.3**

Continuous monitoring of TRS compounds in Alberta has been performed at five permanent stations located near Kraft pulp mills, except Fort McMurray station which is located in the vicinity of oil sand mining and processing operations. Monthly monitoring results for the past three years (1999-2001) showing maximum 1-hour and 24-hour values in ppm for each station at reported months are given in Appendix E (AENV monitoring data). Most of the stations were 100% operational. Only two stations operated at less than 90% of the time during two months of the overall monitoring period. The highest 1-hour concentration (72 ppb) was measured at Station #2 in November 2001 at Grande Prairie near Weyerhaeuser Canada. The annual

average of maximum 1-hour and 24-hour monthly concentrations for all stations are summarized in Table 9.

Table 9: Concentrations of TRS Compounds in Alberta from 1999 to 2001 (ppb)

Location	Company	No. of Stations	Year	No. of Months Monitored	Max. 1-hour (ppb)	Max. 24-hour (ppb)
Grande Prairie	Weyerhaeuser Canada	2	1999 2000 2001	12 12 11	3.2 5.5 8.6	1.3 – –
Hinton	Welwood of Canada	1	1999 2000 2001	12 12 10	1.2 8.9 7.5	– – –
Peace River	Daishowa-Marubeni Int.	2	1999 2000 2001	12 12 10	1.9 2.1 4.6	1.1 1.3 –
Boyle	Alberta-Pacific Forest Industries Inc.	2	1999 2000 2001	12 12 11	4.5 2.2 2.8	– – –
Fort McMurray	Southern Wood Buffalo Zone	3	1999 2000	1 5	2.3 2.2	1.0 0.7
Average					**4.7**	**1.3**

Concentrations shown in Table 9 were calculated from the detailed monitoring results given in Appendix E.

Statistical analysis of 1-hour maximum TRS concentrations expressed in ppm of H_2S (1 ppm = 10^3 ppb) gives the following results:

- Mean concentration   0.004704 ppm
- Median   0.0025 ppm
- Standard deviation   0.008648
- Sample variance 7.479×10^{-5}
- Range   0.09946 ppm
- Minimum   0.000 ppm
- Maximum   0.09946 ppm
- 99% results at concentrations   <0.072 ppm
- 90% results at concentrations   <0.011 ppm
- 50% results at concentrations   <0.003 ppm

Ambient concentrations of H_2S for the past three years has been analyzed for 80 monitoring stations operating in Alberta. The stations are located in areas affected by the oil and gas exploration and production facilities such as sour gas wells, gas and crude oil batteries, gas and oil processing plants, oil refineries, and tar sands mining and processing. Alberta Environment (AENV) monthly results for the January 1999 to November 2001 period contains 1,653 entries of H_2S maximum concentrations (1-hour averaging time). A full record of the AENV monitoring data is given in Appendix E.

The highest ambient concentrations of H_2S (1-hour average) were recorded in May, June and July 2001 at 2.0 ppm, 1.0 ppm and 3.0 ppm, respectively, at Christina Lake Thermal Project operated by PanCanadian Petroleum Ltd. These were exceptionally high concentrations by comparison with the average concentration of 0.00964 ppm. Statistical analysis of 1-hour H_2S concentrations (ppm) gives the following results:

- Mean concentration   0.009644 ppm
- Median   0.003 ppm
- Standard deviation   0.092873
- Sample variance   0.008625
- Range   3 ppm
- Minimum   0.000 ppm
- Maximum   3.000 ppm
- 99% results at concentrations   <0.068 ppm
- 90% results at concentrations   <0.012 ppm
- 50% results at concentrations   <0.003 ppm

When the highest three concentrations are excluded, the average concentration of H_2S (1-hour highest) from anthropogenic sources in Alberta is 0.00602 ppm (6.02 ppb or 8.5 μg/m³) based on a three year monitoring period.

Several reduced sulphur compounds are formed and emitted from anaerobically stored manure. Data specific to this activity were provided for 1999, and are summarized in Table 10 (Clanton and Morey, 1999).

Table 10: Measurement Results of RSC in Air Samples at Manure Storage Site

RSC	No. of Samples	Minimum (ppb)	Maximum (ppb)	Mean (ppb)
Carbonyl sulphide	36	2.9	35.1	10.9
Methyl mercaptan	13	1.9	26.9	8.5
Dimethyl sulphide	8	2.2	44.4	8.6
Carbon disulphide	49	1.9	405	32.3
Dimethyl disulphide	7	1.2	6.5	2.7
Hydrogen sulphide	48	4	2820	445

5.0 HEALTH EFFECTS OF REDUCED SULPHUR COMPOUNDS

5.1 Approach

A review of the toxicological databases was conducted to identify and obtain information associated with the effects following inhalation exposures to RSC compounds. A broad search was conducted, such that information on all reduced sulphur compounds would be identified, if available. Databases that were reviewed were U.S. Environmental Protection Agency Integrated Risk Information System (IRIS), National Library of Medicine Hazardous Substances Database (HSDB), and Agency of Toxic Substances and Diseases Registry (ATSDR) Toxicological Profiles. In addition, searches were conducted in the Toxline database for primary scientific literature that could be sourced and reviewed.

Publications describing health effects following exposure to methyl mercaptan, n-butyl mercaptan, propyl mercaptan, ethyl mercaptan, t-butyl mercaptan, dimethyl sulphide, dimethyl disulphide, carbon disulphide, carbonyl sulphide, methyl disulphide, and mixtures of two or more RSC were identified, reviewed, and are summarized in the following sections.

5.2 Hydrogen Sulphide

In discussions with Alberta Environment, the evaluation of health effects associated with exposures to hydrogen sulphide (H_2S) was deemed outside the scope of the current investigation. Alberta Health and Wellness will be conducting a separate assessment of the health effects of H_2S. The assessment of H_2S in this report is limited to only its odour concerns. The odour threshold of hydrogen sulphide is 30-50 µg/m^3 (NLM, 2001).

5.3 Mercaptans

5.3.1 Human Health Effects

There is very little information associated with health effects following inhalation of methyl mercaptan. Most of the information is related to occupational exposures and interpretation of results may be confounded by exposures to other sulphur containing compounds. Table 11 summarizes some of the human studies associated with the toxicity of the mercaptans.

A mortality was reported for a male worker involved in emptying methyl mercaptan tanks for one week (Shults *et al.,* 1970). No details were available regarding exposure levels, but the individual was hospitalized in a coma and had developed hemolytic anemia and methemoglobinemia. These effects were reversed by blood transfusions, but the person died as the result of an embolism in the pulmonary arteries. Shults *et al.* (1970) theorized that the hemolytic anemia may have been the result of methyl mercaptan acting as an oxidant on the red blood cells and the effects were exacerbated by a deficiency in the enzyme, glucose-6-phosphate dehydrogenase.

Table 11: Summary of Health Effects Due to Exposure to Mercaptans

Chemical	Concentration	Exposure Time	Subjects	Effects	Reference
Methyl mercaptan	Unknown	Unknown	Human	Hemolytic anemia, methemoglobinemia	Shults *et al.*, 1970
n-Butyl mercaptan	0.0001 to 0.001 ppm		Human	Odour threshold	NLM, 2001
Propyl mercaptan	0.00075 ppm		Human	Odour threshold	NLM, 2001
Ethyl mercaptan	0.001 ppm		Human	Odour threshold	NLM, 2001
Methyl mercaptan, hydrogen sulphide (8 μg/m^3)	0.0010-0.0025 ppm	Unknown	Human	Ocular and nasal symptoms, coughs	Jaakkola *et al.*, 1990
Methyl mercaptan	0.016 ppm		Human	Odour threshold	NLM, 2001
Methyl mercaptan, hydrogen sulphide (0.006 ppm)	1.0-2.5 ppm	Unknown	Human	Nasal, respiratory, and ocular symptoms, incidence of headaches	Marttila *et al.,* 1994
Methyl mercaptan, sulphur dioxide (0.5 ppm)	1.4 ppm	Unknown	Human	Incidence of chest illnesses	Enerson *et al.,* 1984
t-Butyl mercaptan	9 ppm (LOAEL)	6 hours/day, 5 days/week, 13 weeks	Rats	Inflammatory lesions	NLM, 2001
n-Butyl mercaptan	10 ppm (NOAEL)	6 hours/day,	Mice	Maternal effects, embryotoxic effects	Thomas *et al.,* 1987
Methyl mercaptan	17 ppm (NOAEL)	7 hours/day, 5 days/week, 3 months	Rats	Decreased body weights	Tansy *et al.,* 1981
n-Butyl mercaptan	50-500 ppm	1 hour	Human	Muscular weakness, malaise, sweating, nausea, drowsiness, vomiting, headache	Gobbato and Terribile, 1968
Methyl mercaptan	675 ppm (LC$_{50}$)	4 hours	Rats	Mortality	Tansy *et al.,* 1981
Methyl mercaptan	1,200 ppm	15 minutes	Rats	Lethargy, coma	Zeive *et al.,* 1974
n-Butyl mercaptan	2,500 ppm (LC$_{50}$)	4 hours	Mice	Mortality	Fairchild and Stokinger, 1958
Ethyl mercaptan	2,770 ppm (LC$_{50}$)	4 hours	Rats	Mortality	Fairchild and Stokinger, 1958
n-Butyl mercaptan	4,020 ppm (LC$_{50}$)	4 hours	Rats	Mortality	Fairchild and Stokinger, 1958
Ethyl mercaptan	4,420 ppm (LC$_{50}$)	4 hours	Mice	Mortality	Fairchild and Stokinger, 1958
Propyl mercaptan	7,300 ppm (LC$_{50}$)	4 hours	Rats	Mortality	NLM, 2001

NOAEL – No observable adverse effect level
LOAEL – Lowest observable adverse effect level
LC$_{50}$ – Lethal concentration (50% mortality)

Workers in a pulp mill where woodchips are digested into carbohydrates, had a higher incidence of chest illness than a control group of railroad workers. The 8-h time weighted average (TWA) concentration of methyl mercaptan was 1.4 ppm (2.8 mg/m^3), with a maximum of 15.0 ppm (30 mg/m^3). Sulphur dioxide was also present (mean 0.5 ppm) in the pulp mill. Spirometric values of the pulp mill workers were not different from those of the control group (Enarson *et al.,* 1984).

As part of the South Karelia Air Pollution Study in Finland (Jaakkola *et al.,* 1990), residents in the vicinity of paper mills were studied for respiratory and other symptoms. The highly polluted areas had concentrations of 0.0010 ppm - 0.0025 ppm (2 - 5 µg/m^3) methyl mercaptan as an annual mean; the highest daily average concentration was 0.025 ppm (50 µg/m^3) as estimated from a computer simulation model. Exposures to hydrogen sulphide (annual mean of 8 µg/m^3 and highest daily average of 100 µg/m^3) and low concentrations of sulphur dioxide were also present. The complaints of the residents from severely polluted areas were compared with those from moderately polluted and nonpolluted areas. A dose-related increase in eye and nasal symptoms and for cough during the previous 12 months were observed in the severely polluted areas, compared with the control group.

Marttila *et al.* (1994) also assessed the effects of long-term exposure to malodourous sulphur compounds released into the ambient air from the same pulp mills as a determinant of eye and respiratory symptoms and headaches in children. In the severely polluted area, the annual mean concentrations of hydrogen sulphide and methyl mercaptan were estimated to be 6 ppb (8 mg/m^3) and 1.0 - 2.5 ppm (2 - 5 mg/m^3), respectively. The highest daily average concentrations were 71 ppm (100 mg/m^3) and 25 ppm (50 mg/m^3), respectively. The incidence of nasal symptoms, cough, eye symptoms, and headache were increased in the severely polluted community, but were not statistically significant. Marttila *et al.* (1994) concluded that exposure to malodourous sulphur compounds may affect the health of children.

For other mercaptans, workers accidentally exposed to estimated concentrations of 50 to 500 ppm n-butyl mercaptan (5,742 mg/m^3 to 57,420 mg/m^3) for one hour have complained of muscular weakness, malaise, sweating, nausea, drowsiness, vomiting, headache, mild depression and confusion (Gobbato and Terribile, 1968). NLM (2001) suggested that the estimated air concentration for propyl mercaptan and ethyl mercaptan that may be hazardous to humans would be 500 ppm while exposures to 4 ppm would cause minimal symptoms. No effects would be observed below 0.5 ppm.

Odour thresholds for methyl mercaptan, ethyl mercaptan, propyl mercaptan and n-butyl mercaptan range from 0.0016 ppm (0.0032 mg/m^3), 0.001 ppm (0.0025 mg/m^3), 0.00075 ppm (0.0023 mg/m^3) and about 0.0001 to 0.001 ppm (0.0004 mg/m^3 to 0.004 mg/m^3), respectively (Amoore and Hautala, 1983, NLM, 2001).

5.3.2   Animal Toxicity Studies

A summary of the effects of mercaptan exposures in animals is presented in Table 11.

Tansy *et al.* (1981) exposed male rats to concentrations of methyl mercaptan of 0, 2, 17, or 57 ppm (0, 4, 34, and 114 mg/m^3) for 7 hours per day, 5 days per week, for 3 months. No histopathological effects were observed in the respiratory, cardiovascular, or renal systems. Decreased body weights were associated with the highest exposed group of rats while no such decreases were observed at concentrations less than 17 ppm (34 mg/m^3). There was an increase in the total protein content of the blood and associated decrease in serum albumin, indicative of liver damage. However, the liver damage may also have been caused by dehydration, rather than chemical-related. It can be concluded that the no-observable-adverse-effect level (NOAEL) for subchronic exposure in rats is 17 ppm. However, the nose was not examined for possible local effects. In addition, Tansy *et al.* (1981) reported an LC$_{50}$ of 675 ppm (1,328 mg/m^3) for male and female rats exposed for 4 hours to methyl mercaptan. Zero mortality was observed at 400 ppm (787 mg/m^3) and 100% mortality for the rats exposed to concentrations greater than 700 ppm (1,377 mg/m^3) for 4 hours.

Zeive *et al.* (1974) found that rats exposed to 1,400 ppm (2,755 mg/m^3) of methyl mercaptan for 15 minutes exhibited lethargic responses and even comas. Exposures to concentrations less than 1,200 ppm (2,361 mg/m^3) methyl mercaptan did not result in such effects.

Life Systems Inc. (1992) conducted a review of the toxicological information associated with methyl mercaptan in support of the development of a Minimal Risk Level (MRL) by the Agency for Toxic Substances and Disease Registry (ATSDR). They concluded that there was insufficient information available for the derivation of an MRL.

Fairchild and Stokinger (1958) reported an LC$_{50}$ of 4,020 ppm (14,829 mg/m^3) for rats and 2,500 ppm (9,222 mg/m^3) for mice exposed to n-butyl mercaptan. NLM (2001) lists an LC$_{50}$ for rats of 7,300 ppm (22,739 mg/m^3) for propyl mercaptan for 4-hour exposures. Fairchild and Stokinger (1958) also listed an LC$_{50}$ of 2,770 ppm (6,925 mg/m^3) for 4-hour exposures to ethyl mercaptan in rats and 4,420 ppm (11,050 mg/m^3) for 4 hours in mice.

Thomas *et al.* (1987) studied the teratogenic effects of inhalation exposures to n-butyl mercaptan. Female mice were exposed for 6 hours per day during days 6 to 16 of gestation while rats were similarly exposed for the same duration, except on days 6 to 19 of gestations. The average daily concentrations of n-butyl mercaptan were 10, 68 and 152 ppm (36.9, 251, and 561 mg/m^3). Significant effects were noted in the mice exposed to 68 or 152 ppm (251 and 561 mg/m^3) n-butyl mercaptan. These include emaciation, unkempt appearance, red or brown staining of the perivaginal region, lethargy and staining of the hair. No mortalities were observed in any of the rats. Decreased weight gains and in some cases, even weight losses were observed on days 12 through 17 of gestation. Early resorptions per litter and embryotoxic effects were also found at concentrations greater than 68 ppm (251 mg/m^3).

NLM (2001) cited a study where rats were exposed to 0, 9, 97 or 196 ppm (0, 33.2, 358, or 723 mg/m^3) t-butyl mercaptan for 6 hours per day, 5 days per week, for 13 weeks. Inflammatory

lesions were observed in rats exposed to the lowest concentration (i.e., 9 ppm or 33.2 mg/m^3). At the next highest concentration, minimal to mild interstitial fibrosis was observed in the lung while interstitial fibrosis was found in the lungs at the highest concentration. Nephrosis was also observed in the male rats exposed to 196 ppm (723 mg/m^3).

5.4   Dimethyl Sulphide

5.4.1   Human Health Effects

Odour thresholds for dimethyl sulphide are reported to range from 0.001 to 0.063 ppm (0.0025 mg/m^3 to 0.16 mg/m^3) (NLM, 2001).

No studies were located for the effects of human exposure to dimethyl sulphide; those observed for rats are summarized in Table 12.

Table 12: Summary of Health Effects Due to Exposure to Dimethyl Sulphide

Concentration	Exposure Time	Subjects	Effects	Reference
0.001-0.063 ppm		Human	Odour threshold	NLM, 2001
3,000-140,000 mg/m^3	30-35 minutes	Rats	Mucous membrane irritation	NLM, 2001
40,250 ppm (LC$_{50}$)	24 hours	Rats	Mortality	Tansy *et al.,* 1981

LC$_{50}$ – Lethal concentration (50% mortality)

5.4.2   Animal Toxicity Studies

Rats exposed to 3 to 140 mg/L dimethyl sulphide (3,000 mg/m^3 to 140,000 mg/m^3) for 30 to 35 minutes developed irritation of mucous membranes while those exposed to 5% (v/v) died within 15 minutes (NLM, 2001). Tansy *et al.* (1981) determined an LC$_{50}$ of 40,250 ppm (102,296 mg/m^3) for rats exposed to dimethyl sulphide for 24 hours.

Fowles *et al.* (1999) developed a series of acute benchmark concentrations for dimethyl sulphide based on Tansy *et al.* (1981). A benchmark concentration (BMC) is the exposure concentration of a chemical that corresponds to a prescribed increase in a health effect endpoint relative to a background level. The derivation of the BMC is the currently favoured method in the establishment of health-based criteria. Using the probit model for the fitting of the mortality data, the BMC for a 1%, 5%, and 10% increase in toxicological effects were 21,843, 25,895, and 28,328 ppm (55,514, 65,812, and 71,996 mg/m^3), respectively. The use of a different model (i.e., Weibull) yielded 1%, 5%, and 10% BMC of 16,003, 22,638, and 26,358 ppm dimethyl sulphide (40,672, 57,535, and 66,989 mg/m^3), respectively.

5.5 Dimethyl Disulphide

5.5.1 Human Health Effects

Table 13 summarizes the human health effects of inhalation exposure to dimethyl disulphide.

Table 13: Summary of Health Effects Due to Exposure to Dimethyl Disulphide

Concentration	Exposure Time	Subjects	Effects	Reference
0.0008-0.004 ppm		Human	Odour threshold	NLM, 2001
0.05-31 ppm	Unknown	Human	Hematological effects	Klingberg *et al.,* 1988
2 ppm	3 months	Rats	Microscopic liver damage	NLM, 2001
805 ppm	4 hours	Rats	Slight liver damage	NLM, 2001
805 ppm (LC_{50})	24 hours	Rats	Mortality	Tansy *et al.,* 1981
1,843–6,748 ppm	30-35 minutes	Rats	Pulmonary irritation	NLM, 2001

LC_{50} – Lethal concentration (50% mortality)

Klingberg *et al.* (1988) investigated the hematological effects of exposure to organic sulphides. In the study, 18 pulp and paper workers that were occupationally exposed to methyl mercaptan, dimethyl sulphide (0.05 - 0.31 ppm) and dimethyl disulphide were found to have elevated levels of iron and transferrin and decreased concentrations of ferritin. Five of the highly exposed workers had decreased activities of δ-aminolevulinic acid synthase and heme synthase. Klingberg *et al.* (1988) concluded that low level exposures to organic sulphides may inhibit the intracellular uptake of iron in the reticuloendothelial system.

The odour threshold for dimethyl disulphide was 0.0008 - 0.004 ppm (0.003 - 0.014 mg/m^3) for detecting the chemical in the air and 0.0075 ppm (0.029 mg/m^3) for recognition in the air (Verschueren, 1983).

5.5.2 Animal Toxicity Studies

Tansy *et al.* (1981) determined an LC_{50} of 805 ppm (30,071 mg/m^3) for dimethyl disulphide. Like dimethyl sulphide, Fowles *et al.* (1999) also derived a series of acute BMCs for dimethyl disulphide based on Tansy *et al.* (1981). Using the probit model for the fitting of the data, the BMC for a 1%, 5%, and 10% increase in toxicological effects were 638, 683, and 709 ppm (2,458, 2,631, and 2,732 mg/m^3), respectively. The use of a different model (I.e.*,* Weibull) yielded 1%, 5%, and 10% BMC of 553, 641, and 684 ppm (2,131, 2,470, and 2,635 mg/m^3), respectively.

NLM (2001) reported that rats exposed to 1,843 - 6,748 ppm (7.1 - 26 mg/L) of dimethyl disulphide for 30 - 35 minutes experienced pulmonary irritation with lung ecchymoses and convulsions. In addition, rats exposed to 805 ppm dimethyl disulphide for 4 hours demonstrated zero mortality and only slight histopathological liver damage but at a lower concentration of 2 ppm for 3 months, microscopic liver damage was observed (NLM, 2001).

5.6   Carbon Disulphide

There are numerous studies on the effects of carbon disulphide inhalation exposures. Rather than cover all of the carbon disulphide studies in this report, only recent relevant studies are discussed to provide a general assessment of its toxicity. The reader is directed to several good recent reviews of carbon disulphide toxicity for more detailed discussion. Recent reviews of carbon disulphide toxicology have been published by Beauchamp *et al.* (1983), ATSDR (1996) and Environment Canada and Health Canada (2000). A summary of the effects of the inhalation of carbon disulphide by humans is presented in Table 14.

5.6.1   Human Health Effects

Chronic exposures to low concentrations of carbon disulphide result in central nervous system (CNS) and peripheral nervous system (PNS) effects that are different than those observed for acute effects. Epidemiological studies have demonstrated that carbon disulphide is not carcinogenic (Wilcosky *et al.,* 1984, Nurminen and Hernberg, 1985, MacMahon and Monson, 1988, Swaen *et al.,* 1994, Liss and Finkelstein, 1996, Peplonska *et al.*, 1996).

Table 14: Summary of Carbon Disulphide Effects in Humans and Animals

Concentration	Exposure Time	Subjects	Effects	Reference
Unknown	30 years	Human	Cerebral atrophy	Frumkin, 1998
Unknown	> 1 year	Human	Mortalities associated with arteriosclerotic heart disease	MacMahon and Monson, 1988
0.016 ppm		Human	Odour threshold	NLM, 2001
1.0 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Johnson *et al.,* 1983
2.6 ppm (LOAEL)	15 years	Human	Neurological	Cassitto *et al.,* 1993
3.2 ppm (NOAEL)	Unknown	Human	Neural conductance, blood pressure, serum chemistry	Cirla and Graziano, 1981
4 ppm	Unknown	Human	Higher blood lipid levels	Krstev *et al.,* 1992
4.2 ppm (NOAEL)	4 years	Human	Blood chemistry	Drexler *et al.,* 1995
4.2 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Reinhardt *et al.,* 1997a
4.8 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Vasilescu and Florescu, 1980
4.8 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Sandrini *et al.,* 1983
4.8 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Hirata *et al.,* 1996
4.8 ppm (LOAEL)	Unknown	Human	Neural conductance changes	Takebayashi *et al.,* 1998
4.8 ppm (LOAEL)	Unknown	Human	Vestibular alterations	Merluzzi *et al.,* 1981
4.8 ppm (LOAEL)	Unknown	Human	Changes in wave pattern of brainstem auditory evoked potentials	Hirata *et al.,* 1992
4.8 ppm (LOAEL)	Unknown	Human	Changes in dopaminergic system	Wasilewska *et al.,* 1989; Stanosz *et al.,* 1994b; Yang *et al.,* 1996
> 7 ppm	Various	Human	Mortalities associated with circulatory diseases	Swaen *et al.,* 1994
5.1-7.1 ppm	≥ 10 years	Human	Increased total serum cholesterol and low density lipoprotein cholesterol levels	Stanosz *et al.,* 1994
7.7 ppm	Unknown	Human	Increased serum cholesterol levels, diastolic blood pressure increases	Egeland *et al.,* 1992
9.6-48 ppm	Unknown	Human	Mortalities	Balcarova and Halik, 1991
> 10 ppm	Unknown	Human	Changes in cholesterol levels and blood pressure	Vanhoorne *et al.,* 1992
> 10 ppm	Unknown	Human	Retinal hemorrhaging	Sugimoto *et al.,* 1976, 1977, 1978; Tolonen *et al.,* 1976; Karai *et al.,* 1983; Vanhoorne *et al.,* 1996
10-30 ppm	Unknown	Human	Neural conductance changes	Seppalainen and Tolonen, 1974

Concentration	Exposure Time	Subjects	Effects	Reference
10-30 ppm	Unknown	Human	Mortalities associated with coronary disease	Hernberg *et al.,* 1970, 1971, 1973; Tolonen *et al.,* 1975
15-30 ppm (NOAEL)	Unknown	Human	Angina, blood pressure changes	Sugimoto *et al.,* 1978
16 ppm	6 months	Rats	Histopathological changes in cardiovascular system	Antov *et al.,* 1985
> 20 ppm	> 10 years	Human	Mortalities associated with coronary disease	Tiller *et al.,* 1968
20 ppm (NOAEL)	7 hours/day, 1-5 days	Rats	Chromosomal aberrations, dominant lethal mutations, sperm abnormalities	Belisle *et al.,* 1980
20-45 ppm	4-29 years	Human	ECG changes	Kamal *et al.,* 1991
> 20.3 ppm	Unknown	Human	Mortalities	Sweetnam, 1987
40-67 ppm	Unknown	Human	Peripheral neuropathy	Chu *et al.,* 1996
50 ppm (LOAEL)	Unknown	Mice	Increased fatty deposit formation in aortic valves associated with high fat diet	Lewis *et al.,* 1999
50 ppm (LOAEL)	6 hours/day, 5 days/week, up to 13 weeks	Rats	Neurofilament crosslinking in spinal cord	Erve *et al.,* 1998a, 1998b
51 ppm (LOAEL)	6 hours/day, 5 days/week, 13 weeks	Rats	Neurofilament crosslinking in spinal cord	Valentine *et al.,* 1997, 1998
220 ppm (LC$_{50}$)	1 hour	Mice	Mortality	Gibson and Roberts, 1972
321 ppm	5 hours/day, 6 days/week, 15 months	Rats	Histopathological changes in coronary arteries	Wronska-Nofer and Parke, 1978; Wronska-Nofer *et al.,* 1980
600 ppm	5 hours/day, 5 days/week, 13 weeks	Rats	Reproductive behaviour	Tepe and Zenick, 1984; Zenick *et al.,* 1984
640 ppm	Days 7-15 of gestation	Rats	Maternal and pup mortality	Lehotzky *et al.,* 1985
790 ppm	15 hours	Rats	Neurological changes	Environment Canada and Health Canada, 2000
803 ppm	18 hours	Rats	Reduced respiration and cardiac rates	Tarkowski and Sobczak, 1971

NOAEL – No observable adverse effect level
LOAEL – Lowest observable adverse effect level
LC$_{50}$ – Lethal concentration (50% mortality)

Several studies have demonstrated that occupational exposures to carbon disulphide result in increased mortalities due to coronary and circulatory diseases (Hernberg *et al.,* 1970, 1971, 1973; MacMahon and Monson, 1988; Swaen *et al.,* 1994; Tiller *et al.,* 1968; Tolonen *et al.,* 1979). However, dose-response relationships could not be established due to the lack of reliable exposure data. As well, confounding factors may exist in the occupational exposures, preventing determination of a causal relationship. Hernberg *et al.* (1970, 1971, 1973) and Tolonen *et al.* (1975) observed an increased incidence in mortalities associated with coronary heart disease for workers exposed to carbon disulphide in a Finnish viscose rayon plant. The control group consisted of workers in a paper mill. Workers in the viscose rayon plant had been exposed to concentrations of 10 - 30 ppm (31 - 94 mg/m^3) during the initial period of the study. There were also increases in reports of chest pain and non-fatal myocardial infarction as well as increased number of individuals with increased blood pressures. Follow-up surveys of this group of workers still found a higher incidence of deaths due to coronary heart disease (Tolonen *et al.,* 1979, Hernberg and Tolonen, 1981, Nurminen *et al.,* 1982).

Tiller *et al.* (1968) evaluated the incidence of cardiac related mortalities in viscose rayon workers employed for more than 10 years and exposed to concentrations greater than 20 ppm (62.5 mg/m^3) carbon disulphide. There was a 2½ times greater incidence of mortalities due to coronary heart disease in the rayon process workers (42%) compared to nonprocess workers (17%) in the same plant. The proportion of deaths for other rayon workers not directly exposed to carbon disulphide was 24%. However, interpretation of this investigation is limited because of the selection of inappropriate controls, possible simultaneous exposures to other chemicals in the plant, and failure to account for other coronary heart disease risk factors.

In another cohort study, the incidence of mortalities due to all causes, ischemic diseases, and other circulatory diseases combined were all higher in male viscose rayon workers in the U.K. compared to the general population (Sweetnam *et al.*, 1987). Concentrations of carbon disulphide that these workers were exposed to often exceeded 20.3 ppm or 63 mg/m^3 (Tiller *et al.,* 1968). However, exposures were confounded by a simultaneous exposure to H$_2$S.

MacMahon and Monson (1988) observed similar findings in a study of U.S. workers who had been employed in a viscose rayon plant for at least one year. Workers with the greatest exposure based on their job titles had a higher incidence of mortalities due to arteriosclerotic heart disease compared to the general population. Workers with 15 years of exposure or more were principally susceptible. However, the study did not quantify the carbon disulphide exposures nor account for other confounding factors contributing to coronary diseases.

Dutch viscose rayon workers exposed to carbon disulphide concentrations at least 7 ppm (21.9 mg/m^3) and higher were also found to have a 15% higher mortality rates due to circulatory diseases compared to the general population (Swaen *et al.,* 1994). Workers who were continuously exposed to carbon disulphide in the bleaching and spinning departments had a higher risk of mortality from cardiovascular disease and an insignificant increase in mortality risk from ischemic heart disease. These mortality risks were inversely related to cumulative exposure as measured by personal air sampling in the latter part of the study. It is possible that higher exposures may have been experienced in the past. Swaen *et al.* (1994) concluded that the risks associated with cardiovascular mortality did not decrease after exposures were

stopped. The risks of mortality due to coronary disease were the greatest 20 - 30 years after beginning exposures. Confounding factors, however were not considered in this study but risks were compared with an unexposed population that had similar lifestyles.

In a Polish viscose rayon plant, workers who had experienced chronic carbon disulphide poisoning had a higher incidence of circulatory system-related mortalities including ischemic heart disease and cerebrovascular disease (Peplonska *et al.* 1996). Female workers exhibited similar results but were based on smaller sample sizes and in some cases, the increased mortality rates were not statistically significant.

A ten-year epidemiological study of Czechoslovakian viscose rayon workers found higher total mortalities and mortalities due to cardiovascular mortalities in spinners that were exposed to higher concentrations of carbon disulphide (Balcarova and Halik, 1991). The concentrations of carbon disulphide were not measured but Balcarova and Halik (1991) estimated that they ranged from 9.6 - 48 ppm (30 - 150 mg/m^3). There was an increased incidence of myocardial infarction with the spinners compared to the controls.

Kamal *et al.* (1991) found a higher incidence of pathological changes as evidenced by electrocardiograms (ECG) in Egyptian workers at a viscose rayon factory. Workers had been exposed to 20 - 45 ppm (62.5 - 141 mg/m^3) of carbon disulphide for 4 - 29 years. There was however no correlation between the duration of exposure and ECG activities.

In a Japanese study (Sugimoto *et al.,* 1978), viscose rayon workers were exposed to concentrations below 20 ppm (62.5 mg/m^3) carbon disulphide, although historical concentrations may have ranged from 15 - 30 ppm (46.9 - 93.8 mg/m^3). Blood pressure changes and the incidence of angina were not different between the exposed and control groups. No differences were also observed in total serum cholesterol, triglycerides, and β-lipoprotein compared to the controls. It is suggested that ethnic or other demographic factors such as dietary habits may contribute to the toxicity of carbon disulphide.

Increases in serum low-density lipoprotein cholesterol (LDL-C) levels and diastolic blood pressure were related to increased exposures to carbon disulphide (Egeland *et al.,* 1992). Male workers in this study were exposed to median concentrations of 0.96 - 7.7 ppm (3 - 24 mg/m^3) carbon disulphide in the viscose rayon plant. The control group was an unexposed group of workers in synthetic textile mills. Egeland *et al.* (1992) concluded that the higher LDL-C in the highest exposure group corresponded to a 26% increased risk of coronary heart disease. Triglyceride and fasting glucose levels were not correlated with carbon disulphide exposures.

Stanosz *et al.* (1994a) found similar results in female workers (i.e.*,* aged 40 - 55) exposed to 5.1 - 7.1 ppm (16 - 22 mg/m^3) carbon disulphide for at least ten years in the viscose fibre production plant. These workers had higher total cholesterol and LDL-C compared to the controls. There was also a significantly lower concentration of high-density lipoprotein cholesterol (HDL-C) concentrations in the women exposed to carbon disulphide. Stanosz *et al.* (1994) suggest that the observed effects were the result of altered lipid metabolism caused by hormone production by the ovaries.

Similar increases in LDL-C and decreases in HDL-C were also observed in workers exposed to concentrations greater than 10 ppm (31 mg/m^3) in a Belgian viscose rayon factory (Vanhoorne *et al.,* 1992). They also noted an increase in the blood pressure and levels of apolipoprotein A1, a protective factor for coronary risk, in the exposed population. However, there were no significant increases in the incidence of angina, myocardial infarction, or ischemia as diagnosed by ECG changes. Despite observing such responses, the results may have been confounded by concomitant exposures to hydrogen sulphide (Vanhoorne *et al.,* 1992).

Workers in a Yugoslavian viscose rayon plant were exposed to time-weighted average concentrations of 4 ppm (12.5 mg/m^3) or 18.5 ppm (57.8 mg/m^3) carbon disulphide. Compared to non-exposed workers, the total lipids and triglycerides concentrations in the serum of the exposed workers were higher. However, there was no established dose-response (Krstev *et al.,* 1992).

Exposure to lower concentrations of carbon disulphide (i.e., 4.2 ppm or 13 mg/m^3) for four years found no correlation between exposure and blood pressure, blood coagulation, or serum concentrations of cholesterol, LDL-C, HDL-C, triglycerides, apolipoproteins, electrolytes or glucose (Drexler *et al.,* 1995). Similarly, Hernberg *et al.* (1971) did not observe any effects on serum cholesterol levels of workers chronically exposed to 10 - 30 ppm (31 - 94 mg/m^3) carbon disulphide. Franco *et al.* (1982) found no differences in blood lipid chemistry of workers exposed to carbon disulphide in a viscose rayon plant compared unexposed workers in another part of the same plant.

The primary effect of carbon disulphide exposure has been neurological. Environment Canada and Health Canada (2000) report that exposures to 500 - 1,000 ppm (1,563 - 3,125 mg/m^3) carbon disulphide resulted in a range of psychiatric disturbances while exposure to 5,000 ppm (15,625 mg/m^3) resulted in CNS depression, coma, respiratory paralysis and death.

Neurophysiological effects stemming from long-term exposures to carbon disulphide have been noted in a number of occupational studies. Seppalainen and Tolonen (1974) found deficits in the motor nerve conduction and slow motor fibre conduction of male Finnish viscose rayon workers who had been exposed to 10 - 30 ppm (31 - 94 mg/m^3) carbon disulphide and hydrogen sulphide over the long-term. These included the deep peroneal, posterior tibial and ulnar nerves.

Johnson *et al.* (1983) studied white male workers in a U.S. viscose rayon plant and found that workers with high exposures of 7.7 ppm (24 mg/m^3) had reduced maximum motor nerve conduction velocities and the amplitude ratio of muscle action potentials following peroneal nerve stimulations. Reduced sensory nerve conduction velocity and increased sural nerve action potential were also observed in this exposed group. Moderate exposures (median 4.2 ppm or 13 mg/m^3) and low exposures (1 ppm or 3 mg/m^3) also resulted in slightly lower conduction velocities of both nerves. The highest group also reported neurobehavioural ailments more frequently than the moderate or low exposures groups.

Similar reductions in peroneal and sural nerve conduction velocities were observed by Reinhardt *et al.* (1997a) in workers exposed to median concentrations of 4.2 ppm (13 mg/m^3) of

carbon disulphide. Other studies have shown similar reductions in the conduction velocities of the motor and sensory nerves after exposures to mean concentrations of 4.8 - 9.6 ppm or 15 - 30 mg/m^3 (Vasilescu and Florescu, 1980; Sandrini *et al.,* 1983, Hirata *et al.,* 1996; Takebayashi *et al.,* 1998). Exposures to concentrations in this range also resulted in vestibular alterations (Merluzzi *et al.,* 1981), changes in the wave pattern of brainstem auditory evoked potentials (Hirata *et al.*, 1992) and alterations of the dopaminergic system (Wasilewska *et al.,* 1989, Stanosz *et al.,* 1994b, Yang *et al.,* 1996).

Cirla and Graziano (1981) indicated that exposures to concentrations less than 3.2 ppm or 10 mg/m^3 (mean concentrations ranged from 1.6 - 6.4 ppm or 5 - 20 mg/m^3) resulted in no significant reductions in nerve conduction, no increases in peripheral nerve impairment and no differences in psychological testing. There were also no significant differences in blood pressure or serum levels of blood lipid or lipoproteins. The neurological effects were more pronounced following exposures to higher concentrations of carbon disulphide (Gilioli *et al.,* 1978, Ruijten *et al.,* 1993, Chu *et al.,* 1995, Vanhoorne *et al.,* 1995).

Neurobehavioural studies have shown to be contradictory. In some studies, workers exposed to 9.6 - 28.8 ppm (30 - 90 mg/m^3) for a long-term had lower performances compared to unexposed workers. This was especially true in the tests for motor speed or dexterity (Hanninen, 1971, Cassitto *et al.,* 1978, Hanninen *et al.,* 1978, De Fruyt *et al.,* 1998). Other studies have demonstrated no significant effects in the battery of neuropsychological tests (Cirla and Graziano, 1981, Putz-Anderson *et al.,* 1983, Reinhardt *et al.,* 1997b, Takebayashi *et al.,* 1998).

Cassitto *et al.* (1993) investigated the neurobehavioural performance of workers in a viscose rayon plant over a 15-year period. Monitoring of the plant indicated that prior to 1971, the concentrations averaged approximately 19.2 ppm (60 mg/m^3) with peaks up to 38.4 ppm (120 mg/m^3). Improvements in the indoor air management decreased concentrations to approximately 0.64 ppm or 2 mg/m^3 (peaks up to 5.1 ppm or 16 mg/m^3) in the preparation room, 1.6 ppm or 5 mg/m^3 (peaks ranged from 9.6 - 11.2 ppm or 30 - 35 mg/m^3) in the spinning department, and 0.32 - 1.28 ppm or 1 - 4 mg/m^3 (peaks up to 4.2 ppm or 13 mg/m^3) in the washing department. They observed significant differences in memory and perceptive abilities in workers in the early years of the investigation with the current ones. They concluded that even exposures to concentrations not exceeding 2.6 ppm or 8 mg/m^3 could result in absentmindedness and difficulties in perceptive abilities.

Exposures to higher concentrations resulted in axonal degeneration and disorganized neurofilaments in the sural nerve. Chu *et al.* (1996) observed such histopathological changes in a male viscose rayon worker who had been exposed to 40 - 67 ppm or 125 - 209 mg/m^3 carbon disulphide. The individual displayed symptoms of peripheral neuropathy.

Hemorrhaging of the retinal capillaries have been noted in exposures to concentrations greater than 10 ppm (31 mg/m^3) carbon disulphide (Sugimoto *et al.* 1976, 1977, 1978, Tolonen *et al.*, 1976, Karai *et al.,* 1983, Vanhoorne *et al.,* 1996). Exposures to lower concentrations, however have not demonstrated a clear association with retinal damage (Sugimoto *et al.,* 1984, 1992, Omae *et al.,* 1998). Other effects on the eyes have included impaired colour vision in viscose rayon workers exposed to concentrations greater than 10 ppm (31 mg/m^3) (Raitta *et al.,* 1981, Vanhoorne *et al.,* 1996). However, the effects on the eyes are not consistent within a population of individuals and therefore, some people may be more susceptible than others.

Frumkin (1998) presented a case study of a 60-year old male worker who had been exposed to carbon disulphide in a viscose rayon plant for 30 years. No measured carbon disulphide measurements were available, but the individual was employed in the spinning room which typically has the highest concentrations of carbon disulphide in a viscose rayon manufacturing plant. The patient was diagnosed with olivopontocerebellular atrophy, characterized by autonomic dysfunction, parkinsonism, and ataxia.

ATSDR (1996) lists an odour threshold of 0.016 ppm (0.05 mg/m^3) for carbon disulphide while ACGIH (1991) suggests a threshold of 0.1 - 0.2 ppm (0.3 - 0.6 mg/m^3).

5.6.2 Animal Toxicity Studies

Gibson and Roberts (1972) determined an LC$_{50}$ of 220 ppm or 688 mg/m^3 for 1-hour exposures in male mice.

Inhalation of 0, 3.2, 224, or 640 ppm (0, 10, 700, or 2,000 mg/m^3) carbon disulphide from day 7 through 15 of gestation (Lehotzky *et al.,* 1985) by pregnant rats induced 35% perinatal mortality of pups. Thirty-three percent of pregnant rats exposed during gestation to 640 ppm (2,000 mg/m^3) carbon disulphide died. Neurobehavioural indices such as eye opening and the auditory startle reflex were reduced. Immature gait, motor uncoordination, diminished open field activity, and altered behavioral patterns were observed on day 21 and 36, but by day 90, they had returned to normal. At the concentrations administered, disturbed learning ability was shown by diminished performance and lengthened latency of the conditioned avoidance response.

Rats exposed to 790 ppm (2,469 mg/m^3) for 15 hours did not result in any mortalities although neurological problems were observed (Environment Canada and Health Canada, 2000).

Tarkowski and Sobczak (1971) exposed male rats to 803 ppm (2,509 mg/m^3) carbon disulphide for 18 hours. The rats exhibited reduced respiratory and cardiac rates and also, severe narcosis. A limitation for evaluation of this study was that only seven animals and one concentration were tested.

Antov *et al,* (1985) exposed rats to 16 ppm (50 mg/m^3) carbon disulphide and higher for six months. They had histopathological changes to the cardiovascular system including distention of the lumen, attenuation of myocardial vessels, and irregular thickening of the aorta wall. A dose-response was observed related to the histopathological changes. When rats were fed an atherogenic diet and exposed to the same concentrations of carbon disulphide, increased

mortalities and serum globulin, decreases in serum albumin, and histopathological changes in the myocardium and aorta were observed.

In another study where rats were exposed to 321 ppm (1,003 mg/m^3) carbon disulphide for 5 hours per day, 6 days per week for 15 months (Wronska-Nofer and Parke, 1978, Wronska-Nofer *et al.,* 1980), no gross or histological lesions in the aorta were exhibited, but lipid droplets were noted in the histological preparations of the coronary arteries. When fed an *ad libitum* chow diet, there was an increased rate of cholesterol influx from the serum into the aorta wall. If the rats were fed an atherogenic diet (i.e.*,* 2% cholesterol, 0.15% thiouracil) during the exposures, there was more aggressive infiltration of the coronary arteries by lipid deposits.

Female mice were exposed to 0 - 800 ppm (0 - 2,500 mg/m^3) carbon disulphide by inhalation. Half the animals were placed on an atherogenic high fat diet while the other were on a control diet (Lewis *et al.,* 1999). Mice that were exposed to 500 ppm (1,563 mg/m^3) or 800 ppm (2,500 mg/m^3) carbon disulphide and fed a control diet had a slight increase in the rate of fatty deposit formation in the aortic valve leaflets. However, for mice fed a high fat diet, there was a marked increase in the fatty deposit formation in the mice exposed to 500 ppm (1,563 mg/m^3) or 800 ppm (2,500 mg/m^3) compared to the unexposed group. A slight increase was also observed in the mice exposed to 50 ppm (156 mg/m^3) and fed a high fat diet compared to the control high fat diet group.

Like the human epidemiological investigations, exposures to carbon disulphide resulted in decreases in the nerve conduction velocities of the peripheral nerves and spinal cord.

In a series of studies, rats were exposed to 51.2, 512, or 800 ppm (160, 1,600, or 2,500 mg/m^3) of carbon disulphide for 6 hours per day, 5 days per week for 13 weeks. Neurofilament protein crosslinking in the spinal cord was observed in the histopathology of the rats of all three exposure groups (Valentine *et al.,* 1997, 1998). This crosslinking had developed during weeks 2 to 4 of exposure. In addition, there were gait abnormalities, reductions in the grip strength of the hind and fore limbs, and increased expression of nerve growth factor receptor mRNA in the sciatic nerve (Moser *et al.,* 1998, Toews *et al.,* 1998). During the later weeks of the studies, axonal swelling and degeneration and electrophysiological alterations in the peripheral nerves and/or spinal cord was found in the rats exposed to 512 or 800 ppm (1,600 or 2,500 mg/m^3) carbon disulphide (Herr *et al.,* 1998, Sills *et al.,* 1998). Aside from the peripheral nervous system and the spinal cord, there were no other histopathological lesions of the brain, heart, aorta, lung and female reproductive tract (Sills *et al.,* 1998).

Sills *et al.* (2000) exposed female mice to 0 or 800 ppm (2,500 mg/m^3) carbon disulphide for 6 hours per day, 5 days per week for 20 weeks. Neurobehavioural and histopathological endpoints were examined at the end of the exposure period. In general, the mice exposed to 800 ppm (2,500 mg/m^3) carbon disulphide had decreased body weights, piloerection, ptosis, and hunched postures. Neurological effects included an altered gait, decreased rearing and locomotor movement and neuromuscular weakness. Focal to multifocal axonal swelling was observed in the muscular branch of the posterior tibial nerve, with an occasional axonal swelling

in the lumbar segment of spinal cord. Electron micrographs of the swollen axons showed an accumulation of neurofilament proteins within the axoplasm.

The crosslinking of proteins was also observed in rats exposed to 50, 500 or 800 ppm (156, 1563, or 2,500 mg/m^3) carbon disulphide for 6 hours per day, 5 days per week for 2, 4, 8 or 13 weeks (Erve *et al.,* 1998a, 1998b). The authors theorized that the protein cross-linking may be used as biomarkers for assessing exposures to carbon disulphide.

Exposures to 20.2 or 40 ppm (63 or 125 mg/m^3) for 7 hours per day for 1 or 5 days did not result in an increase in the frequency of chromosomal aberrations in bone marrow cells, dominant lethal mutations, or sperm abnormalities in rats (Belisle *et al.,* 1980). However, there was no observed increase in sperm abnormalities in the positive control rats, suggesting that there may have been a problem with the testing.

Tepe and Zenick (1984) and Zenick *et al.* (1984) exposed male rats to either 349 or 600 ppm (1,090 or 1,875 mg/m^3) carbon disulphide for 5 hours per day, 5 days per week for several weeks. At the highest concentration, copulatory behaviour was affected as evidenced by the reducing times to mount and ejaculate. Sperm counts, reproductive hormonal levels in the blood and testicular histopathology were not affected by the exposures.

Price *et al.* (1996) determined a benchmark concentration (BMC) for carbon disulphide based on the effects on the peripheral nervous system and ischemic heart disease risk factors. A benchmark concentration is the exposure concentration of a chemical that corresponds to a prescribed increase in a health effect endpoint relative to a background level. The derivation of the BMC is the current favourable methodology in the establishment of health-based criteria. A BMC of 16.2 and 18.5 ppm (60.6 and 57.8 mg/m^3) for decreased in motor conduction velocity and amplitude ratios in the peroneal nerve, respectively were derived based on the Johnson *et al.* (1983) study.

Environment Canada and Health Canada (2000) has established a tolerable concentration of 0.032 ppm (0.1 mg/m^3) for carbon disulphide. This was based on the derivation of a BMC for reductions in peroneal motor nerve conduction velocity observed by Johnson *et al.* (1983).

5.7   Carbonyl Sulphide

5.7.1   Human Health Effects

The toxicological effects of carbonyl sulphide may be attributed to its hydrolyzation with water to form carbon dioxide and hydrogen sulphide. Most of its effects result from exposure to the resulting hydrogen sulphide. Table 15 summarizes the human health effects from inhalation exposures to carbonyl sulphide.

NLM (2001) indicates that exposures to approximate concentrations of 250 ppm (615 mg/m^3) carbonyl sulphide resulted in irritation of mucous membranes, conjunctivitis, photophobia, lacrimation, corneal opacity, rhinitis, bronchitis, cyanosis and pulmonary edema. At concentrations between 250 and 500 ppm (615 mg/m^3 and 1,230 mg/m^3), headaches, nausea, vomiting, diarrhea, vertigo, amnesia, dizziness, apnea, palpitations, tachycardia, hypotension, muscle cramps, weakness, disorientation and coma may result. Exposure to concentrations greater than 750 ppm (1,845 mg/m^3) may result in abrupt physical collapse (i.e., "knockdown") accompanied by respiratory paralysis, asphyxial seizures, and death.

5.7.2 Animal Toxicity Studies

Table 15 presents a summary of some of the toxicological investigations of carbonyl sulphide in animals.

Table 15: Summary of Carbonyl Sulphide Effects in Humans and Animals

Concentration	Exposure Time	Subjects	Effects	Reference
50 ppm	7 weeks	Rabbits	Blood chemistry	Kamstrup and Hugod, 1979
50 ppm (NOAEL)	1-7 weeks	Rabbits	Histopathological effect	Hugod, 1981
250 ppm (LOAEL)	Unknown	Humans	Irritation, pulmonary edema	NLM, 2001

NOAEL – No observable adverse effect level
LOAEL – Lowest observable adverse effect level

Kamstrup and Hugod (1979) continuously exposed rabbits to approximately 50 ppm (123 mg/m^3) carbonyl sulphide for 7 weeks. Slightly elevated mean serum cholesterol concentrations were observed. However, the mean serum triglyceride concentration was largely unaffected by the exposures, although an insignificant increase was present. There was also a small increase in the concentration of free cholesterol in the aorta. This was likely attributed to the difference in serum cholesterol concentrations between experimental and control animals. No significant difference in arterial uptake of labeled cholesterol between exposed animals and controls was observed. No histopathological changes in lungs or atherosclerosis-like changes in coronary arteries, aorta or pulmonary arteries were found.

Hugod (1981) exposed rabbits to 50 ppm (123 mg/m^3) carbonyl sulphide for 1 - 7 weeks. No ultrastructural effects were observed in the rabbits.

The toxicity of carbonyl sulphide may be related to its metabolic conversion to hydrogen sulphide. This was demonstrated in an intraperitoneal injection study where rats were exposed to 20 - 30 mg/kg via intraperitoneal injection of gaseous carbony sulphide (Chenglis and Neal, 1980). Ataxia with loss of righting reflex, cyanosis, dyspnea, and on occasion convulsions resulted, similar to the effects observed following hydrogen sulphide exposures. Pretreatment of rats with acetazolamide, an inhibitor of carbonic anhydrase, reduced the blood levels of hydrogen sulphide and, therefore, decreased the toxicity of carbonyl sulphide. Sodium nitrite pretreatment also protected animals against carbonyl sulphide toxicity. Chenglis and Neal (1980) concluded that carbonyl sulphide is metabolized by carbonic anhydrase to hydrogen

sulphide and it is this byproduct that is responsible for its toxicity. The value of this study in terms of explanation of toxicity via inhalation is questionable.

5.8   Mixtures of Reduced Sulphur Compounds

Studies that evaluated inhalation exposures to a mixture of reduced sulphur compounds are summarized in Table 17.

**Table 16: Summary of Effects of Exposure to
Mixtures of Reduced Sulphur Compounds**

Chemical	Concentration	Subject	Effects	Reference
Thiophenes, disulphides	Unknown	Human	Delayed airways disease, hypothalamic function effects	Watt, 1997

Workers in a pulp mill were exposed to a mixture of methyl mercaptan (8-h Time Weighted Average [TWA] concentration of 0.07 - 2.0 ppm), hydrogen sulphide (8-h TWA of 0.05 - 5.2 ppm), and dimethyl sulphide (8-h TWA of 0.03 - 3.2 ppm). Although the number of complaints among the workers was comparable to that of a control group, the average annual number of days on sick leave of the workers tended to be higher for the group of exposed workers (12.9 versus 7.1 days). Delta-aminolaevulinic acid synthase and heme synthase activity levels in blood were decreased in 8 and 6 cases, respectively. No statistical calculation was performed to determine if these effects were significant (Tenhunen *et al.,* 1983).

Tansy *et al.* (1981) determined a rat LC_{50} of 550 ppm for an equimolar mixture of methyl mercaptan, dimethyl sulphide, and dimethyl disulphide for a 4-hour exposure.

6.0 EFFECTS OF REDUCED SULPHUR COMPOUNDS ON VEGETATION

6.1 Introduction

Sulphur is essential for plant growth. Generally, plants acquire sulphur by absorbing sulphate through their roots. However, when exposed to sulphur-containing air pollutants, sulphur can also enter into plants via stomates of the leaves and be assimilated into organic sulphur. When uptake of gaseous sulphur exceeds the demands from sulphur metabolism and the rate of detoxification, accumulation of sulphur-containing compounds in vegetative tissues can occur (Maas, 1987; Herschbach *et al*., 1995). In addition, de Kok *et al*. (1997) demonstrated that there is a strong interaction between atmospheric and soil sulphur nutrition as plants are able to grow in the absence of sulphate with H_2S in air as the sole source of sulphur.

Plants exposed to atmospheric sulphur-containing compounds may respond in a number of ways (Noggle *et al*., 1986):

- the sulphur may act as a fertilizer, enhancing growth and yield;
- there may be no response by the plant;
- biochemical and/or physiological changes may be triggered that can range from easily measured effects to those that are not detectable;
- visible injury may occur, which may or may not be accompanied by a decrease in growth and/or yield; and/or
- the plant's growth and/or yield may decrease.

The effects of reduced sulphur compounds on vegetation were most intensively studied from the 1980's through to the early 1990's (e.g., Kord *et al*., 1993b; de Kok *et al*., 1997; Chen and Paull, 1998). Studies have focused on the effects of H_2S on plant growth and physiology, with a few publications reporting the impacts of other RSC (e.g., Ren *et al*., 1996; Obenland *et al*., 1998). Very few studies have examined the response of plants to a variety of concentrations under controlled environmental conditions, which are necessary to evaluate injury threshold concentration levels or to derive dose-response relationships.

Injury to plants by any potentially toxic gaseous compound is controlled by three processes (Tingey and Taylor, 1982):

1. the resistance of the plant to uptake of the compound through the stomates (stomatal resistance);
2. the biochemical resistance of the plant or its ability to detoxify the compound and its metabolites; and
3. the capability of the plant to compensate for and repair any injury caused by the compound and its metabolites.

Thus, the type and extent of injury to plants by sulphur compounds is dependent upon the rate of entry of the pollutant into the plant, and the capacity of physiological processes within the plant to prevent the accumulation of toxic compounds (Noggle *et al*., 1986). The rate of entry or uptake is dependent upon the physiology of the plant and environmental factors.

Stomata are epidermal pores on plant leaves and sometimes on plant stems that have the ability to open and close in order to regulate gas exchange from air into the leaf, and from the leaf into the air. The distribution and anatomy of stomata are dependent upon the plant species, cultivar and genotype, and the stage of development of the plant. The stomatal aperture is dependent upon a variety of environmental factors such as water availability, humidity, air temperature, light, ambient and internal leaf CO_2 concentration, and plant nutrient status.

Taylor *et al*. (1983) compared the relative fluxes of SO_2, H_2S, COS, CS and methyl mercaptan (CH_3SH) into bush bean and soybean. The ranking of the five gases for internal flux via the stomata was $SO_2 > H_2S > COS > CS_2 > CH_3SH$. The greater the flux, the greater the potential for toxic effect. The authors suggest that this trend implies potential ranking for the relative toxicity of the compounds examined, although biological effects were not directly measured.

The response of plants to air pollutants, including those containing reduced sulphur, is dependent upon a variety of genetic, physiological and environmental factors, and the specific conditions of exposure; a wide range of responses to RSC exposure is therefore possible.

6.2   Approach

A literature review was conducted in 1999 on the effects of reduced sulphur compounds on vegetation, with a specific focus on hydrogen sulphide (H_2S), carbonyl sulphide (COS), carbon disulphide (CS_2), sulphides, disulphides and mercaptans or thiols. The review included publications examining vegetation as a source of biogenic sulphur emissions and as a contributor to the global sulphur cycle (e.g., Goldan *et al*., 1988; Mihalopoulos *et al*., 1989; Brown and Bell, 1986; Morrison and Hines, 1990; Bartell *et al*., 1993; Kesselmeier and Merk, 1993) as well as studies focusing on the utilization of H_2S for investigating plant biochemical reactions (e.g., Buwalda *et al*., 1988, 1990, 1993; Schutz *et al*., 1991; Youssefian *et al*., 1993). The 1999 review culminated with the submission of a draft document to Alberta Environment in 2000. The draft report presented a synthesis of the scientific literature published to 1999 describing the effects of reduced sulphur compounds on vegetation.

To bring the review up to date, a search of the scientific literature published since 1999 was conducted to identify any papers published since the preparation of the draft report. A range of search terms was utilized to ensure that any publication describing the effects of RSC on vegetation was identified. The databases searched were Current Contents, Biological Abstracts, CISTI Source, Environment Abstracts, Web of Science, Wildlife Worldwide, Zoological Record, Agricola, Toxline, ArticleFirst, Chemical Abstracts, GEOBASE, and Pollution Abstracts. Despite the diversity of databases searched, there were no new publications containing the results of original research into the effects of RSC on vegetation. Occasional references to early work were found in peripherally relevant papers, however, as no new information was presented, these publications were not included in this assessment.

The effects of RSC may be differentially expressed among horticultural, agricultural and forest species, with different economic consequences. Effects on appearance of leaves and other plant parts would result in a negative economic effect on horticultural species (e.g., lettuce), and may have no economic consequences for agricultural or forest species. Effects on yield are the key concern for agricultural species, while effects on plant growth are the primary concern for

forest species. For this reason, the effects of each of the RSC are assessed for each of these three plant groups in the following review and assessment.

6.2.1   Hydrogen Sulphide (H₂S)

6.2.1.1   *Physiological Responses*

The effects of exposure to H$_2$S of horticultural, agricultural and forest plant species are presented in Tables 18, 19 and 20, respectively. These tables provide a summary of the species exposed, the concentration and duration of exposure, and the response to this exposure. The comment section in the tables provides additional information regarding the results of the experiments as well as comments regarding the reliability of the data.

Several studies presented in Tables 18 to 20 have shown that exposure to H$_2$S at low levels can increase plant growth and/or the rate of physiological processes in a variety of species. Positive growth and physiological responses to pollutant exposure has sometimes been termed a "fertilizer" effect. However, increases in plant photosynthetic rates in the absence of corresponding enhanced dry weight accumulations can be a stress response to a toxic compound. Plants may increase their rate of photosynthesis in order to keep up with the required rate of injury compensation and repair. Once plants are unable to keep up with the required rate of repair, detectable reductions in plant dry weight accumulation may occur. Fertilizer or positive growth effects from exposure to sulphur-containing pollutants would be most pronounced when plants were grown in conditions of limited sulphur availability in the soil or growing medium (Khalil *et al*., 1996; de Kok *et al*., 1997).

Measurements of an increase or decrease in plant fresh weight are not reliable indicators or toxic effects of contaminants unless accompanied by measurements of dry weight. Fresh weight measurements are partially a function of the water status of the plant at the time of harvest, and are not a reliable indication of the true stress response of the plant. Nevertheless, fresh weight measurements may be useful indicators of the economic impact of exposure to toxic compounds for plant species such as lettuce and spinach, since to be marketable the leaves of these plants must not be wilted.

An increase in lettuce (*Lactuca sativa* L., cv. Dark Green Boston) fresh and dry weight was noted in greenhouse grown plants exposed to 100 ppb H$_2$S for 56 days (Table 18; Thompson *et al*., 1979). Thompson *et al*. (1979) also noted an increase in the fresh weight of sugarbeets exposed to 100 ppb H$_2$S for 131 days (Table 19). Coyne and Bingham (1978) observed a significant increase in photosynthesis and stomatal conductance in field grown snap beans (*Phaseolus vulgaris* L. cv. GV50) exposed to 100 ppb H$_2$S for four hours per day for 18 days (Table 19). Measurements of dry weight of exposed plants were not presented in the paper, so it is unclear whether the enhanced rate of photosynthesis was a true fertilizer effect or if it represented a change in plant water status.

Table 17: Effects of H₂S on the Growth, Yield and Physiology of Horticultural Plant Species

Reference	Horticultural Species Examined	H₂S (ppb)	Exposure Duration	Measured Response	Comments
Bosma *et al.* (1990)	Spinach (*Spinacia oleracea* L. cv. Estivato)	Average <150	3 or 4 days (field)	• Increase in water soluble non-protein sulfhydryl compounds and cysteine content in leaves	• Sulfhydryl and cysteine content expressed on a fresh weight basis
Buwalda *et al.* (1993)	Spinach cv. Estivato leaves	250	24 hours (fumigation chamber)	• Rapid accumulation of thiols in leaves of plants grown in both light and dark conditions	
de Kok *et al.* (1983a)	Spinach cv. Melody Hybrid	30	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • No effect on fresh weight of shoots	
		100	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • No effect on fresh weight of shoots	
de Kok *et al.* (1986)	Spinach cv. Estivato	250	3 days (stainless steel cabinet)	• No effect on *in vitro* nitrate reductase activity	
		250	24 hours (stainless steel cabinet)	• No effect on *in vitro* anaerobic nitrate reductase activity • Effect on *in vivo* aerobic nitrate reductase activity	
de Kok *et al.* (1989)	Spinach cv. Estivato	750	12 days (stainless steel cabinet)	• 69% reduction in shoot yield	• Shoot yield measured as fresh weight • Flux of H₂S into the shoots exhibited a linear relationship with H₂S concentration up to 300 ppb H₂S; flux of H₂S into the shoot leveled off and remained constant above 300 ppb • No effect on shoot yield observed at H₂S concentrations below 750 ppb
	Pumpkin (*Curcurbita pepo* L. cv. small sugar pumpkin)	750	12 days (stainless steel cabinet)	• 36% reduction in shoot yield	• Shoot yield measured as fresh weight
de Kok *et al.* (1997)	Curly kale (*Brassica oleracea* L.)	400	2 weeks	• Reduction in shoot growth at and above 400 ppb • Accumulation of water-soluble non-protein thiols was greater in shoots than in roots	• *Brassica* has a high sulphur requirement compared to other species
Herschbach *et al.* (1995)	Spinach (cv. Estavito)	250	48 hours (fumigation chamber)	• Inhibition of net sulphur uptake (S deficient plants only) • High accumulation of thiols • Inhibition of sulphate transport to shoot	
Khalil *et al.* (1996)	Radish (*Raphinus sativus* L.) and tomato (*Lycopersicon esculentum* L.)	5000	30 minutes (fumigation chamber), repeated on days 30, 45 and 60	• Increased glutamate-oxalate (GOT) and glutamate-pyruvate transaminase (GPT) activity during first part of experiment only in tomato plants	• H₂S applied in a mixture with SO₂
		15,000	30 minutes (fumigation chamber), repeated on days 30, 45 and 60	• Increased GPT activity at 17 and 47 days of age in radish plants	• H₂S applied in a mixture with SO₂
		25,000	30 minutes (fumigation chamber), repeated on days 30, 45 and 60	• Inhibited GOT activity in tomato plants • Enhanced GPT activity in tomato plants	• H₂S applied in a mixture with SO₂

Reference	Horticultural Species Examined	H$_2$S (ppb)	Exposure Duration	Measured Response	Comments
Kord and Abbass (1993)	Tomato (*Lycopersicon esculentum*)	50,000	4 hours (2 groups of plants; group 1 fumigated once, group 2 fumigated two times, 7 days apart; field conditions)	• Decrease in shoot height and root length • Decrease in fresh and dry weight • Decrease in chlorophyll a • Decrease in carbohydrase • Reduced activity of nitrate reductase and glutamate dehydrogenase • Increase in peroxidase activity	
		10,0000	4 hours (2 groups of plants; group 1 fumigated once, group 2 fumigated two times, 7 days apart; field conditions)	• Decrease in shoot height and root length • Decrease in fresh and dry weight • Decrease in chlorophyll a • Decrease in carbohydrase • Increase in cysteine • Decrease in serine • Increase in amino-N • Reduced activity of peroxidase, nitrate reductase and glutamate dehydrogenase	
Kord *et al.* (1993a)	Rocket (*Eruca sativa*) and radish (*Raphanus sativus*)	50,000	4 hours (2 groups of plants; group 1 fumigated once, group 2 fumigated two times, 7 days apart; fumigation chamber)	• Decrease in root and stem length • Reduced stem fresh and dry weight • Decrease in photosynthetic activity • Decrease in plant pigments • Decrease in carbohydrase activity • No effect on ammonium • Increase in total soluble proteins • Decrease in enzymatic activity except peroxidase • Increase in net free amino acids for plants fumigated once	
		10,0000	4 hours (2 groups of plants; group 1 fumigated once, group 2 fumigated two times, 7 days apart; fumigation chamber)	• Decrease in root and stem length • Reduced stem fresh and dry weight • Decrease in photosynthetic activity • Decrease in plant pigments • Decrease in carbohydrase activity • No effect on ammonium • Increase in total soluble proteins • Decrease in enzymatic activity except peroxidase • Decrease in serine • Increase in cysteine • Increase in net free amino acids for plants fumigated once	

Reference	Horticultural Species Examined	H₂S (ppb)	Exposure Duration	Measured Response	Comments
Kord *et al*. (1993b)	Rocket, radish and tomato seedlings	50,000	7 days (Petri-plates)	• Inhibition of radicle and plumule elongation after 1 day exposure • Decrease in radicle and plumule dry weight • Inhibition of accumulation of soluble sugars and biosynthesis of polysaccharides • Decrease in photosynthetic activity • Decrease in total soluble nitrogen	
		100,000	7 days (Petri-plates)	• Maximum inhibition of accumulation of sugars • Maximum inhibition of chlorophyll a formation • Decrease in photosynthetic activity	
Maas *et al*. (1987a)	Spinach (cv. Monosa)	250	3, 5, 7, 10 and 14 days (stainless steel cabinet)	• 26% reduction in relative growth rate at 15 °C after 11 days exposure	
				• 46% reduction in relative growth rate at 18 °C after 14 days exposure	
				• 60% reduction in relative growth rate at 25 °C after 11 days exposure	
				• Yield reductions at all temperatures	• Yields calculated on a fresh weight basis
				• Decrease in shoot/root ratio after 14 days at 18 °C	
				• Decrease in shoot/root ratio after 11 days at 25 °C	
				• Increase in content of water-soluble non-protein sulfhydryl and sulphate content	• Sulfhydryl and sulphate content expressed on a fresh weight basis
		150	14 days (stainless steel cabinet)	• Increase in sulphate content in shoots but not in roots	
		250	14 days (stainless steel cabinet)	• Significant increase in transpiration rate but not water uptake rate	• Transpiration rates and water uptake rates calculated on a fresh weight basis
Maas *et al*. (1988)	Spinach (cv. Monosa)	250	18 days (stainless steel cabinet)	• No effect on dark respiration rate • 23% reduction in CO₂ fixation • Reduced efficiency of photosynthetic electron transport	
		100	28 days (stainless steel cabinet)	• No effect on shoot dry matter content • No effect on fresh weight per shoot	
		300	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • 59% reduction in fresh weight per shoot	
Thompson and Kats (1978)	Grapes (*Vitis vinifera* cv. Thompson seedless)	30	117 days (greenhouse)	• No effect on total cane length • No effect on dead length • No effect on percent dead • No effect on total dry weight	

Reference	Horticultural Species Examined	H$_2$S (ppb)	Exposure Duration	Measured Response	Comments
		300	117 days (greenhouse)	• No effect on total cane length • Increase in dead length • Increase in percent dead • Decrease in total dry weight	
		3,000	117 days (greenhouse)	• Decrease in total cane length • Increase in dead length • Increase in percent dead • Decrease in total dry weight	
		30	145 days (greenhouse)	• No effect on leaf and cane dry weight	• Five plants per treatment only
		100	145 days (greenhouse)	• No effect on leaf dry weight • Decrease in cane dry weight	• Five plants per treatment only
		300	145 days (greenhouse)	• Decrease in leaf dry weight • Decrease in cane dry weight	• Five plants per treatment only
Thompson and Kats (1978)	Lettuce (cv. Dark Green Boston)	30	59 days (greenhouse)	• Increase in fresh weight • Increase in head diameter	
		100	59 days (greenhouse)	• No effect on fresh weight • No effect on head diameter	
		300	59 days (greenhouse)	• Reduction in fresh weight • Reduction in head diameter	
Thompson *et al.* (1979)	Lettuce (*Lactuca sativa* L. cv. Dark Green Boston)	30	77 days (greenhouse)	• No effect on total plant fresh and dry weight • Increase in head fresh weight	
			56 days (greenhouse)	• Increase in total plant fresh and dry weight • No effect on head fresh weight	
		100	77 days (greenhouse)	• Increase in total plant fresh and dry weight • No effect on head fresh weight	
			56 days (greenhouse)	• Increase in total plant fresh and dry weight • Increase in head fresh weight	
		300	77 days (greenhouse)	• Decrease in total plant fresh and dry weight • No head formed	
			56 days (greenhouse)	• Decrease in total plant fresh and dry weight • Decrease in head fresh weight	• Associated loss in appearance economically significant
Thornton and Setterstrom (1940)	Tomato (*Lycopersicon esculentum* Mill.)	1,000,000	960 minutes	• 30 minutes until 50% injury to leaves • 45 minutes until 50% injury to stems	

**Table 18: Effects of H₂S on the Growth, Yield and Physiology of Agricultural Plant Species**

Reference	Agricultural Species Examined	H₂S (ppb)	Exposure Duration	Measured Response	Comments
Bosma *et al*. (1990)	Clover (*Trifolium pratense* cv. Mekra)	50	5 weeks (climate controlled rooms)	• Increase in water-soluble non-protein sulfhydryl content in leaves after 6 hours	• Sulfhydryl content expressed on a fresh weight basis
		Average < 150	3 or 4 days (field)	• Increase in water-soluble non-protein sulfhydryl content of leaves	• Sulfhydryl content expressed on a fresh weight basis
	Sugar Beet (*Beta vulgaris* cv. Monohill)	50	4 weeks (climate controlled rooms)	• Increase in water-soluble non-protein sulfhydryl content of leaves after 6 hours	• Sulfhydryl content expressed on a fresh weight basis
de Kok *et al*. (1989)	Maize (*Zea mays* L. cv. Gelber badischer Landmais)	750	12 days (stainless steel cabinet)	• No effect on shoot yield	• Shoot yield measured as fresh weight • Flux to the shoots also monitored; no direct relationships between flux to the shoots and species sensitivity
Maas *et al*. (1987b)	Clover (cv. Marino)	250	14 days (stainless steel cabinet)	• 32% reduction in yield • Increase in water soluble non-protein sulfhydryl content of leaves and shoot • Increase in sulphate content of shoots	• Yield reduction and sulphate and sulfhydryl content expressed on a fresh weight basis
	Bean (*Phaseolus vulgaris* L. cv. Rubio)	250	14 days (stainless steel cabinet)	• 11% increase in yield • Increase in water soluble non-protein sulfhydryl content of leaves and shoot • Increase in sulphate content of shoots	• Yield increase and sulphate and sulfhydryl content expressed on a fresh weight basis
	Soybean (*Glycine max* Merr. Cv. White Hilium)	250	14 days (stainless steel cabinet)	• No effect on yield • Increase in water soluble non-protein sulfhydryl content of leaves and shoot • Increase in sulphate content of shoots	• Yield and sulphate and sulfhydryl content expressed on a fresh weight basis
Taylor and Sevidge (1984)	Bush bean (*Phaseolus vulgaris* cv. Blue Lake 274)	6,100 12,300 20,500 32,700 40,900 81,800	6 hours (Teflon film chambers)	• All exposure concentrations impaired photosynthesis; effects more pronounced with increasing concentration and duration • No effect on transpiration rate at any of the concentrations tested • Leave necrosis at 81.8 ppb	
de Kok *et al*. (1983b)	Sugar beet (cv. Holly Hybrid US10)	30	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • No effect on fresh weight per shoot	
		100	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • No effect on fresh weight per shoot	
		300	28 days (stainless steel cabinet)	• No effect on dry matter content of shoots • 27% reduction in fresh weight per shoot	

| Reference	Agricultural Species Examined	H$_2$S (ppb)	Exposure Duration	Measured Response	Comments
Bennett *et al*. (1980)	Snap bean (*Phaseolus vulgaris* L. cv. GV50)	300 to 7,000	4 hours per day for 40 days (field conditions)	• Reduction in pod fresh weight by 15% • Reduction in pod dry weight by 34% • Reduction in top dry weight by 46% • Reduction in total dry weight by 29% • Reduction in leaf area by 22%	• Variable delivery of H$_2$S
Thompson *et al*. (1979)	Sugar Beet (cv. Holly Hybrid)	30	131 days (greenhouse)	• Increase in fresh weight of leaves • Increase in dry weight of leaves • Increase in fresh weight of roots	• Results variable in 3 experiments
		100	131 days (greenhouse)	• Increase in fresh weight of leaves • Increase in dry weight of leaves • Increase in fresh weight of roots	• Results variable in 3 experiments
		300	131 days (greenhouse)	• Increase in fresh weight of leaves • No effect on dry weight of leaves • No effect on fresh weight of leaves	• Results variable in 3 experiments
Thompson and Kats (1978)	Sugar Beet (cv. Holly Hybrid)	30	134 days (greenhouse)	• No effect on leaf dry weight • Increase in leaf fresh weight • Increase in root fresh weight • No effect on percent sugar in root	
		100	134 days (greenhouse)	• No effect on leaf dry weight • No effect on leaf fresh weight • No effect on root fresh weight • No effect on percent sugar in root	
		300	134 days (greenhouse)	• Reduction in leaf dry weight • No effect on leaf fresh weight • No effect on root fresh weight • Reduction in percent sugar in root	
	Alfalfa (*Medicago sativa* L. cv. Eldorado)	30	28 to 35 days (greenhouse)	• No effect on average dry weight per pot after first and second cuttings	
		300	28 to 35 days (greenhouse)	• No effect on average dry weight per pot after first cutting • Reduction in average dry weight per pot after second cutting	
		3,000	28 to 35 days (greenhouse)	• Reduction in average dry weight per pot after first cutting • Plant death before second cutting	
Thompson and Kats (1978) [*cont*]	Alfalfa (cv. Hayden)	30	28 to 35 days (greenhouse)	• No effect on average dry weight per pot after first and second cutting	

Reference	Agricultural Species Examined	H$_2$S (ppb)	Exposure Duration	Measured Response	Comments
		300	28 to 35 days (greenhouse)	• Reduction in average dry weight per pot after first and second cutting	
		3,000	28 to 35 days (greenhouse)	• Reduction in average dry weight per pot after first cutting • Plant death before second cutting	
Coyne and Bingham (1978)	Snap beans (*Phaseolus vulgaris* L. cv. GV50)	740	4 hours per day for 18±2 days (field conditions)	• 25% increase in maximum stomatal conductance • 10% increase in maximum apparent photosynthesis	
		3,250	4 hours per day for 18 days (field)	• Decrease in stomatal conductance • Decrease in maximum apparent photosynthesis	
		5,030	4 hours per day for 18 days (field)	• Decrease in stomatal conductance • Decrease in maximum apparent photosynthesis	
Thornton and Setterstrom (1940)	Buckwheat (*Fagopyrum esculentum* Moench)	1,000,000	960 minutes	• 60 minutes until 50% injury to leaves • 120 minutes until 50% injury to stems	
	Tobacco (*Nicotiana glutinosa* L.)	1,000,000	960 minutes	• 100 minutes until 50% injury to leaves • 480 minutes until 50% injury to stems	

Table 19: Effects of H$_2$S on the Growth, Yield and Physiology of Forest Plant Species

Reference	Forest Species Examined	H$_2$S (ppb)	Exposure Duration	Measured Response	Comments
Thompson and Kats (1978)	Douglas fir (*Pseudotsuga meziesii*) seedlings	30	246 days (greenhouse)	• No visible injury	• Data not shown in paper
		100	246 days (greenhouse)	• Slight burn observed • No reduction in growth and dry weight	• Data not shown in paper
		300	246 days (greenhouse)	• Extensive foliar injury • Reduction in growth and dry weight	• Data not shown in paper
	Ponderosa pine (*Pinus ponderosa*)	30	76 days (greenhouse)	• No observed effects	• Data not shown in paper
		300	76 days (greenhouse)	• Tip burn observed	• Data not shown in paper
		3,000	76 days (greenhouse)	• Tip burn and defoliation observed	• Data not shown in paper

It has also been shown that exposure to various levels of H_2S can result in an increase in the levels of water-soluble sulphydryl compounds (i.e., glutathione), cysteine, and sulphate in both roots and shoots (Maas *et al*., 1987a, 1987b; Bosma *et al*., 1990; Buwalda *et al*., 1994; de Kok *et al*., 1997). Although a direct relationship was not found between the degree of accumulation of sulphur compounds and growth reduction, it is generally accepted that the increase interferes with normal sulphur metabolism. de Kok *et al*. (1983a) suggest that high concentrations of accumulated sulphide have an inhibitory effect on photosynthetic electron transport. Maas and de Kok (1988) noted that exposure of spinach to H_2S resulted in the inactivation of NADH-oxidizing enzymes. The authors suggest that the interference of H_2S in plant physiological and biochemical processes may eventually result in impaired growth and yield.

The National Research Council of Canada (NRCC, 1981) listed a variety of biochemical changes that occur in plants as a result of exposure to H_2S, including decrease in sugar, starch and chlorophyll levels, and stimulation or depression of several enzymatic activities and inhibition of NADH oxidation by mitochondria. It was suggested that these biochemical impacts occur due to the influence of H_2S on enzyme inhibition.

6.2.1.2   Relative Sensitivities of Plant Species to Acute Exposures of H_2S

Early research on the impacts of H_2S on plants exposed to short durations of high concentrations was conducted by McCallan *et al*. (1936). They exposed 29 plant species to concentrations of H_2S ranging from 20,000 to 400,000 ppb. Plants were exposed for five days "out of doors" (conditions were not described) between June and September. Visible impacts were rated qualitatively from none to very severe. Typical symptoms included the scorching of young shoots and leaves, basal and marginal scorching of older leaves, with mature leaves remaining visually unaffected.

Plant species studied by McCallan *et al*. (1936) exhibited a wide variation in sensitivity and response. Species exhibiting only **very slight injury** at 400,000 ppb H_2S included:

- carnation (*Dianthus caryolphyllus* L.);
- purslane (*Portulaca oleracea* L.);
- Boston fern (*Nephrolepisis exaltata* Shott var. *bostoniensis*);
- apple (*Pyrus malus* L.);
- cherry (*Prunus avium* L.);
- peach (*Prunus persica* L.);
- strawberry (*Fragaria chiloensis*); and
- coleus (*Coleus blumei*).

Plant species displaying **slight to moderate** injury at concentrations between 40,000 and 400,000 ppb H_2S included (McCallan *et al*., 1936):

- pepper (*Caspium frutescens* L.);
- rose (*Rosa* sp.);
- nasturtium (*Tropaeolum minus* L.);
- castor bean (*Ricinus communis* L.);
- gladiolus (*Gladiolus* sp.);
- sunflower (*Helianthus debilis* Nutt.);

- buckwheat (*Fagopyrum esculentum* Moench.); and
- cornflower (*Centaurea cyanus*).

Plant species deemed sensitive to H$_2$S due to **severe injury** at 50,000 ppb (McCallan *et al*., 1936) included:

- soybean (*Glycine max* Merr.);
- tobacco (*Nicotiana glauca*);
- aster (*Callistephus chinensis* Nees.);
- kidney bean (*Phaseolus vulgaris*)
- cucumber (*Cucumis sativus* L.);
- salvia (*Salvia spendens* Ker.);
- poppy (*Eschscholzia californica* Chamb.);
- tomato (*Lycopersicon esculentum* Mill.);
- clover (*Melilotus alba* Desr.);
- radish (*Raphanus sativus* L.);
- calliopsis (*Coreopsis* sp.); and
- cosmos (*Cosmos* sp.).

Although the pioneering work of McCallan *et al*. (1936) provides interesting observations regarding relative sensitivities or resistance of plant species to acute exposure to H$_2$S, it is not useful in the identification of effects levels or establishment of air quality guidelines for the protection of vegetation. There is insufficient information presented (concentrations, duration of exposure, exposure conditions), making the interpretation and application of these observations in an air quality guideline context impossible.

6.2.1.3   Horticultural Species

The effects of exposure of horticultural species to H$_2$S are presented in Table 18. No significant effects were detected in the growth and yield of spinach exposed to 30 and 100 ppb H$_2$S for 28 days (de Kok *et al*., 1983a). Lettuce exposed to 30 and 100 ppb H$_2$S for periods of 56, 59 and 77 days showed an increase or no effect on growth and yield parameters (Thompson and Kats, 1978; Thompson *et al*., 1979). Exposure of Thompson seedless grapes to 30 ppb H$_2$S for 117 days did not result in a significant effect on total cane length, the number of dead canes, or the total dry weight of plants (Thompson and Kats, 1978).

Significant effects on a variety of growth, yield and physiological parameters occurred in spinach exposed to 250 or 300 ppb H$_2$S for 11, 14 or 28 days (de Kok *et al*., 1983b, 1986; Maas *et al*., 1987a, 1988). Lettuce exposed to 300 ppb H$_2$S for 56, 59 or 77 days resulted in significant decreases in whole plant fresh and dry weight and in head diameter (Thompson *et al*., 1979).

Thompson seedless grapes exposed to H$_2$S concentrations of 300 ppb and higher H$_2$S displayed an increase in the number of dead canes and a decrease in total dry weight, leaf dry weight and cane dry weight (Thompson and Kats, 1978). Exposure of Thompson seedless grapes to 100 ppb H$_2$S for 145 days resulted in a decrease in cane dry weight (Thompson and Kats, 1978). However, only five plants per treatment were measured in this experiment, a relatively low number.

Spinach exposed to 250 ppb H_2S for 24 or 48 hours, and curly kale exposed to the same concentrations over 2 weeks rapidly accumulated water-soluble, non-protein thiols (Buwalda *et al.*, 1993; Hershbach *et al.*, 1995; de Kok *et al.*, 1997). Kord *et al.* (1993a) observed an increase in total soluble proteins in rocket and radish plants exposed to 50,000 and 100,000 ppb H_2S for four hours, and regardless of whether plants were fumigated once or twice. H_2S-induced thiol accumulation may be ascribed to enhanced glutathione levels in plant shoots and roots (de Kok *et al.*, 1997).

Rocket and radish plants fumigated for 30 minutes with 100,000 ppb H_2S showed an increase in cysteine as did tomato plants fumigated with the same concentration of H_2S over four hours (Kord *et al.*, 1993a; Kord and Abbass, 1993). Again, the cysteine increase occurred in groups of tomato plants fumigated once or twice. Kord and Abbass (1993) also found that tomato plants exposed to 50,000 ppb H_2S over the four-hour period showed an increase in peroxidase enzyme activity.

Vegetation exposed to H_2S also exhibited inhibitory effects to growth, yield and physiology. Rocket, radish and tomato plants fumigated with 50,000 to 100,000 ppb H_2S decreased shoot and root length as well as in stem fresh and dry weight (Kord *et al.*, 1993a; Kord and Abbass, 1993). In addition, the decrease in growth and yield was enhanced with increasing time and concentration of exposure. Kord *et al.* (1993b) also noted that the elongation of rocket, radish and tomato seedling radicles and plumules was inhibited with 50,000 ppb H_2S exposure for seven days. Shoot growth in curly kale exposed to H_2S for two weeks was significantly reduced at and above a level of 400 ppb H_2S (de Kok *et al.*, 1997).

Exposure to H_2S also inhibited the productivity of some vegetation. Rocket, radish, and tomato plants exposed to 50,000 to 100,000 ppb H_2S decreased in pigments (e.g., chlorophyll a and b and carotenoids), photosynthetic activity and carbohydrase activity (Kord *et al.*, 1993; Kord and Abbass, 1993). Again, increasing the duration of exposure and concentration enhanced the inhibitory effects. Rocket, radish and tomato seedlings also decreased in photosynthetic activity with exposure to 50,000 or 100,000 ppb H_2S, and the higher concentration produced the maximum inhibitory effect (Kord *et al.*, 1993b).

From the literature available, the lowest exposure concentration that produced a measurable effect (growth stimulation) in this group (horticultural species) was 30 ppb H_2S over 77 days. The lowest exposure concentration to produce a negative effect was 100 ppb H_2S for 145 days, which caused a decrease in cane dry weight in grapes (Thompson and Kats, 1978). However, this observation was based on the measurement of only five plants. Negative effects on growth, yield and physiological parameters were noted in spinach, lettuce, grapes, kale, tomato, rocket and/or radish at 250, 300, 15,000, 25,000, 50,000, and/or 100,000 ppb H_2S at exposure durations ranging from 4 hours to 177 days (Table 18).

6.2.1.4   Agricultural Species

The effects of exposure of agricultural plant species to H_2S are summarized in Table 19. de Kok *et al.* (1989) reported no significant effect on shoot fresh weight in maize (*Zea mays*) exposed to

750 ppb H_2S for 12 days. Beans (*Phaseolus vulgaris*) exposed to 250 ppb H_2S for 14 days showed a significant increase in yield (Maas *et al*., 1987b). Coyne and Bingham (1978) exposed field grown bean plants to 740 ppb H_2S for four hours per day for 18 days, and found a significant increase in stomatal conductance (reduced stomatal resistance) compared with control plants. No effect was observed on soybean yield of plants exposed to 250 ppb H_2S for 14 days (Maas *et al*., 1987b).

Field grown snap bean subjected to varying concentrations (300 to 700 ppb) of H_2S for 4 hours per day for 40 days showed a decrease in a wide variety of growth and yield measurements compared with control plants (Bennett *et al*., 1980). Coyne and Bingham (1978) exposed field grown snap bean to 3,250 ppb H_2S for four hours per day for 18 days and found a significant decrease in stomatal conductance and photosynthesis. It is unknown whether these decreases were accompanied by decreases in growth and yield. Taylor and Sevidge (1984) exposed bush beans to concentrations of H_2S ranging from 6,100 to 81,800 ppb and found that photosynthesis was impaired at all concentrations. The degree of impairment increased with increasing H_2S concentration; however, it is unknown how the impairment impacted growth and yield of the plants.

Impairment to the growth and yield of sugar beets was not observed in plants exposed to 30 or 100 ppb H_2S for 131 or 134 days (Thompson and Kats, 1978; Thompson *et al*., 1979; de Kok *et al*., 1983b). Sugar beet exposed to 300 ppb H_2S showed a decrease in fresh weight per shoot after 28 days (de Kok *et al*., 1983b) and a decrease in leaf dry weight and percent sugar content of the root after 134 days (Thompson and Kats, 1978).

Clover (*Trifolium pratense*) exposed to 250 ppb H_2S for 14 days showed a significant decrease in yield compared with control plants. Thompson and Kats (1978) exposed alfalfa to 300 ppb H_2S for 28 to 35 days and found a significant decrease in average dry weight per pot after each of two cuttings.

From these studies it appears that concentrations as low as 50 ppb H_2S for four or five weeks will induce some biochemical responses in different crop species (Table 19). However, exposure to 250 ppb H_2S for 14 days is the lowest exposure concentration causing reduced plant growth or crop yield for clover, while exposure to 300 ppb H_2S for 134 and 35 days reduced growth or yield for sugar beets and alfalfa, respectively.

6.2.1.5   Forest Species

Only one study reviewed examined the effect of H_2S on forest species (Table 20). Douglas fir and ponderosa pine seedlings were exposed to continual fumigation of H_2S at various concentrations, however no data are available for review in the paper (Thompson and Kats, 1978). Douglas fir seedlings were exposed to 30, 100 and 300 ppb H_2S for 246 days in a greenhouse. Although a slight burn on the needles was observed in plants exposed to 100 ppb H_2S, there was no observed effect on growth and dry weight accumulation. Extensive foliar injury was observed in plants exposed to 300 ppb H_2S, which was accompanied by a significant reduction in growth and dry weight.

Ponderosa pine seedlings were exposed to continuous fumigation of H₂S concentrations of 30, 300 and 3,000 ppb H₂S for 76 days (Thompson and Kats, 1978). Tip burn was observed at 300 ppb H₂S, and both tip burn and defoliation were observed at 3,000 ppb H₂S. No observed effects occurred in plants exposed to 30 ppb H₂S.

Only two forest species were examined in this study. The lack of available data for review within the paper make it difficult to identify the potential H₂S effects level for forest species.

6.2.2 Dimethyl Sulphide

No information on the response of plants to dimethyl sulphide could be located in the literature.

6.2.3 Carbonyl Sulphide (COS)

Various studies have concluded that approximately half of the current 500 pptv atmospheric concentration of carbonyl sulphide is of anthropogenic origin, with a seriously imbalanced budget of sources being twice the size of sinks (Chin and Davis, 1993; Johnson *et al.*, 1993). Soil and plants have been identified as both sources (Aneja *et al.*, 1979a, b; Adams *et al.*, 1981; Steudler and Pewterson, 1984, 1985; Carroll *et al.*, 1986; Rennenberg *et al.*, 1990; Kesselmeier and Merk, 1993) and as sinks for this stable gas (Taylor *et al.*, 1983; Brown and Bell, 1986; Hofmann *et al.*, 1992; Bartell *et al.*, 1993; Kesselmeier and Merk, 1993). Protoschill-Krebs *et al.* (1996) demonstrated in hydroponic pea cultures that the key enzyme in the consumption of carbonyl sulphide is carbonic anhydrase.

The effects of exposure to carbonyl sulphide in horticultural and agricultural species are tabulated in Tables 21 and 22, respectively.

6.2.3.1 Horticultural Species

Chen and Paull (1998) fumigated banana with 10,000 to 60,000 ppm carbonyl sulphide and exposed avocado, mango, papaya, and red ginger to 10,000 and 20,000 ppm carbonyl sulphide for 24 hours. While exposure slowed coloration and flesh softening in papaya, the fumigations increased softening in bananas, mangoes and avocados. The authors note that red ginger inflorescences were less tolerant to carbonyl sulphide than fruit, being able to withstand 20,000 ppm carbonyl sulphide for only 0.75 hours. Lemons fumigated with 70 ppm carbonyl sulphide for 20 hours showed a slight amount of peel injury after 12 hours, but increased in offensive juice odours and rind injury with increasing exposure duration (Obenland *et al.*, 1998).

Table 20: Effects of COS on the Growth, Yield, and Physiology of Horticultural Plant Species

Reference	Horticultural Species Examined	COS (ppm)	Exposure Duration	Measured Response	Comments
Chen and Paull (1998)	Banana (*Musal* sp.), Avocado (*Pensea Americana*), Mango (*Mangifera indica*), Papaya (*Carica papaya*), Red Ginger (*Alpinia purpuratra*)	10,000 to 60,000 (1 to 6%) for bananas and 10,000 to 20,000 (1 to 2%) for all others	24 hours (fumigation chamber)	• Increase in softening for bananas and mangoes • Increase in severity of skin injury for bananas and mangoes • Slowed skin colorations and flesh softening in papaya • Increase in softening of avocado	
Obenland *et al.* (1998)	Lemons (*Citrus limon* Burm.)	70	20 hours (fumigation chamber)	• Slight amount of peel injury after first 12 hours • Increase in offensive odour in juice • Increase in rind injury	

Table 21: Effects of COS on the Growth, Yield and Physiology of Agricultural Plant Species

Reference	Horticultural Species Examined	COS (ppm)	Exposure Duration	Measured Response	Comments
Ren *et al.* (1996)	Wheat (Australian Standard White)	25, 50, 100, 250, 500	24, 48, 96 hours (fumigation chamber)	• Reduced germination rates • No effect on plumule length	

6.2.3.2   Agricultural Species

Ren *et al*. (1996) exposed wheat to 24, 50, 100, 250, and 500 ppm carbonyl sulphide for 24 to 96 hours and observed reduced germination rates at longer exposures and lower moisture conditions, but noted no effect on the plumule length of the plants.

6.2.4   Dimethyl Disulphide

No information on the response of plants to dimethyl disulphide was identified in the literature.

6.2.5   Methyl Mercaptan

Only one scientific publication that studied the response of plants to methyl mercaptan was found in the literature. Taylor and Selvidge (1984) exposed bush bean plants to methyl mercaptan concentrations ranging from 6,100 – 81,800 ppb for 6 hours (Table 21). No effect on the rates of photosynthesis and transpiration were noted for any of the concentrations. Effects on other physiological or morphological (i.e., seed yield) aspects were not evaluated.

6.2.6   Mixtures of Reduced Sulphur Compounds and Other Air Pollutants

Plants are unlikely to experience exposure to only one toxic compound in polluted air. Therefore, some studies have examined the effects of RSC compounds when mixed with other gaseous compounds. Coyne and Bingham (1978) observed apparent photosynthesis and leaf stomatal conductance in snap beans exposed to various levels of H_2S alone and in combination with a single concentration (740 ppb) of ozone (O_3). The presence of O_3 increased the severity of the negative impact on photosynthesis and leaf stomatal conductance from plant exposure to various concentrations of H_2S. The effect of exposing plants to both compounds was synergistic, that is, the toxicity to the plant of both compounds simultaneously was greater than the additive effect of the plants being exposed to the compounds separately.

Shinn *et al*. (1976) experimented with a geothermal gas mixture consisting of 15 CO_2 : 1 H_2S : 1 CH_4 : 2 N_2 parts per volume added to air. An increase in net photosynthesis and a decrease in stomatal resistances of lettuce plants were noted at low concentrations. A significant depression in net photosynthesis of lettuce did not occur until exposure concentrations approached 75,000 ppb CO_2 : 5,000 ppb H_2S added to air. These results would suggest that the other compounds in the gas mixture (e.g., CO_2) would enhance plant physiological processes to provide the plants with protection against the potentially negative effect from exposure to H_2S alone.

Kercher (1982) modeled the impact on sugar beets from expected ground-level concentrations of H_2S and CO_2 from geothermal energy development in Imperial Valley, California. The model incorporated meteorological variables measured within the valley and determined that in the absence of interactions with other pollutants, all 22 locations used in the model would experience an increase in total growth of sugar beets. Seasonal average increases in H_2S concentrations over background levels ranged from 0.1 ppb to 250 ppb. Enhanced levels of CO_2 generally enhance photosynthetic rates of plants, and increase growth rates and dry weight accumulation. Kercher (1982) determined that the combined exposure with CO_2 would provide the plants with protection against the potential impacts from H_2S alone.

Khalil *et al*. (1996) investigated the effects of a H$_2$S and sulphur dioxide (SO$_2$) mixture on alkaline and acid phosphatases in radish and tomato plants (Table 18). Results showed that 5,000 ppb of each gas increased the activity of glutamate-oxalate (GOT) and glutamate-pyruvate transaminase (GPT) in tomato plants during the first part of the experiment, while 25,000 ppb of each gas slowed GOT activity and enhanced GPT activity in tomatoes. In radish, little effect was noted except for exposure to 15,000 ppb of each gas, which increased GPT activity at 17, and 47 days of age.

6.3 Effects Levels for RSC Effects on Vegetation

The phytotoxicity of RSC compounds is dependent upon the compound, its concentration and the duration of exposure. The degree of plant response is dependent upon species, cultivar and genotype as well as on a variety of environmental factors such as light, temperature, humidity, water availability, CO$_2$ concentration and nutrient availability. The review of the literature revealed a general lack of dose-response models for RSC compounds for a variety of species under a variety of environmental conditions. There was a limited amount of information available for RSC compounds other than H$_2$S.

6.3.1 Hydrogen Sulphide

The National Research Council of Canada (NRCC, 1981) completed a review of the scientific literature and concluded that vegetation was relatively insensitive to short-term exposure to high concentrations of H$_2$S. Studies published since 1981 have not examined the effect of acute exposures of H$_2$S on vegetation. NRCC (1981) suggested that long-term exposures of concentrations less than 280 ppb (392 μg/m^3) generally stimulated plant growth whereas long-term exposures of concentrations greater than 280 ppb (392 μg/m^3) H$_2$S were more likely to inhibit growth and cause visible injury.

This review of the literature suggests that impairment to plant growth and physiological processes generally begins at 250 ppb (350 μg/m^3) but at higher concentrations of H$_2$S for several plant species as indicated. Long-term exposures to concentrations up to 100 ppb (140 μg/m^3) did not result in detectable impacts for a variety of plant species. One exception was the reduced dry weight accumulation noted in grapes exposed to 100 ppb (140 μg/m^3) for 145 days by Thompson and Kats (1978). However, the limited sample size (5 plants) exposed to this concentration raises concerns about the validity of these data.

The lowest observable effect concentration (LOEC) for H$_2$S is 250 ppb (350 μg/m^3) for 3 days. The no observable effect concentration (NOEC) is 100 ppb (140 μg/m^3) for a variety of long-term exposure periods. From this review, the highest level without demonstrated or observed effects for H$_2$S is 100 ppb (140 μg/m^3).

6.3.2 Carbonyl Sulphide

Banana, avocado and mango exposed to carbonyl sulphide concentrations ranging from 10,000 to 60,000 ppm for 24 hours resulted in an increase in flesh softening and some skin injury while

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fumigation of papaya showed skin injury and flesh softening. Red ginger was observed to be the least tolerant of the species studied, withstanding 20,000 ppm carbonyl sulphide for only 0.75 hours. Lemons fumigated with 70 ppm carbonyl sulphide for 20 hours showed a slight amount of peel injury after 12 hours, but increased in offensive juice odours and rind injury with increasing exposure duration. Wheat exposed to 24, 50, 100, 250, and 500 ppm carbonyl sulphide for 24 to 96 hours had reduced germination rates at longer exposures and lower moisture conditions, but was not affected in plumule length (Ren *et al.*, 1996). A lack of further dose-response information prevents the identification of a vegetation effects level for carbonyl sulphide.

6.3.3 Methyl Mercaptan

Methyl mercaptan was the only mercaptan or thiol compound for which there was information on the response of plants (Table 23; Taylor and Selvidge, 1984). Bush beans were exposed for six hours to methyl mercaptan concentrations ranging from 6,100 ppb (8,540 μg/m^3) to 81,800 ppb (11,340 μg/m^3) and no impairment to the rate of photosynthesis was detected at any of the concentrations. The lack of further dose-response information prevents the identification of an effect level for methyl mercaptan.

6.3.4 Interactions Between Reduced Sulphur Compounds and Other Substances

There is evidence that co-exposure of plants to RSC and other pollutants may enhance the response to RSC exposure. However, there is insufficient evidence available with which to support the identification of an effect level for any RSC in the presence of one or more other air pollutants.

Table 22: Effect of CH$_3$SH on the Growth, Yield and Physiology of Plant Species

Reference	Horticultural Species Examined	CH$_3$SH (ppb)	Exposure Duration	Measured Response	Comments
Taylor and Selvidge (1984)	Bush bean (*Phaseolus vulgaris* cv. Blue Lake 274)	6100 12300 20500 32700 40900 81800	6 hours (Teflon film chambers)	• No effect on rate of photosynthesis at any of the concentrations tested • No effect on transpiration rate at any of the concentrations tested	

7.0   MONITORING OF REDUCED SULPHUR COMPOUNDS

Monitoring methods are generally described in terms of the monitoring of an individual RSC, or in terms of monitoring for the total reduced sulphur (TRS) concentration in air. Often, the individual components of the analysis are not stated, making it difficult or impossible to separate out and discuss the concentrations of individual RSC substances in the complex family of measured total RSC concentrations.

Different sampling techniques are available for measuring H_2S and other compounds containing sulphur in its lowest oxidation state (reduced sulphur compounds), including methyl mercaptan, dimethyl sulphide and dimethyl disulphide. Two general measuring classes have been developed. One is used for measuring industrial stationary source emissions (stack sampling) and the other for ambient air quality monitoring.

Continuous monitoring of stack emissions is usually carried out using automatic instrumented methods, where the combination of a gas delivery and conditioning line and an analyzer constitutes a continuous emissions monitoring system (CEMS). The measurements may be made *in situ* or be extractive. Continuous monitoring is necessary when pollutant levels vary so frequently and with such significance that an accurate emissions profile cannot be gained from intermittent sampling. When emissions are on a relatively constant level, manual analysis methods can be used with monthly, quarterly or annual frequencies of measurements. Another technique of monitoring is by grab sampling involving collection of stack exhaust into a proper container (e.g., Tedlar bags) for subsequent analysis.

A preferred technique for ambient air analysis is continuous ambient monitoring survey with direct reading analyzers. This is because of its ability to resolve short-term, infrequent episodes of high pollution that are often a primary cause of concern. As with stack monitoring, it is also possible to sample continuously with a non-direct-reading method (e.g., multiple impingers), but the quantification of short, peak episodes will not be possible. Periodic sampling and grab sampling are also used for ambient air monitoring.

The analytical principles behind the techniques used to analyze the collected samples are the same whether stack gas or ambient air has been sampled. Usually, only the changes in the details of the implementation of the principle (e.g., changes in range, sample conditioning) are required.

7.1   RSC Measurement Methods

There are currently no published international standards for the measurement of RSC in point-source ducts and stacks. The ISO 6326 series deals with the measurement of reduced sulphur species in natural gas but not in ambient air.

National standard methods exist in some countries. The U.S. EPA has published two gas chromatography-flame photometric detector (GC-FPD) methods (one semi-continuous and the other non-continuous) and a manual extractive impinger titration method. The German Verein Deutscher Ingenieure (VDI) has published five methods: an automatic paper-tape photometric method, and four manual extractive methods using adsorption or impingers followed by titration

or photometric analysis. Summary of these methods are given in Environment Agency of the National Compliance Assessment Service (2001). In Alberta stack sampling for TRS is performed according to Alberta Stack Sampling Code of 1985.

7.2   RSC (TRS) Analyzers

One class of analyzers is based on fluorescence technique. Initially, all SO_2 in the air sample is removed; H_2S in the sample is then converted to SO_2. The sample is then drawn through a sample chamber where it is irradiated with pulses of ultra-violet light. This exposure causes any SO_2 in the sample to release a characteristic wavelength of light or fluorescence. The amount of fluorescence measured is proportional to the SO_2 (converted from H_2S) concentration. The measurement process in the sample chamber is described by the following equation:

$$SO_2 + hu_1 = SO_2^*$$
$$SO_2^* = SO_2 + hu_2$$
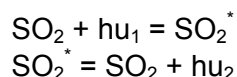
where hu_1 is the incident UV energy at 214 nm and hu_2 is the resultant fluorescence, which is directly proportional to the SO_2 concentration in the sample chamber.

Total reduced sulphur (TRS) analyzer measures total reduced sulphur concentrations continuously by pulsed fluorescence concentration of all the sulphur compounds except SO_2. Initially, all SO_2 in the air sample is removed. The remaining sulphur compounds in the sample are then converted to SO_2 in a high temperature converter. Next, the sample is irradiated with pulses of ultra-violet light in a sample chamber. As in an H_2S analyzer, the amount of fluorescence measured is proportional to the concentration of total reduced sulphur in the sample, which is reported as concentration equivalent to amount of sulphur in H_2S. In terms of sensitivity, the practical detection limit for total RSC analyzers is 1 ppb.

Another class of analyzers employs gas chromatography (GC) technique. It is especially useful in the presence of other numerous compounds in the atmosphere and to eliminate the interference among substances, separation of RSC compounds before quantitation is essential. An example of application of gas chromatography for RSC analysis is State of California *Method 44 Determination of Reduced Sulphur Gases and Sulphur Dioxide in Effluent Samples by Gas Chromatographic Method*. Effluent from source samples containing sulphur dioxide collected in Tedlar bag, Teflon bag or "SilcoCan" canisters can be analyzed using a gas chromatograph fitted with a flame photometric detector (FPD). The RSC in effluent samples may include numerous RSC, but will primarily be a mixture of H_2S, methyl mercaptan, ethyl mercaptan, dimethyl sulphide, carbon disulphide and carbonyl sulphide. The detection limit of this method is 0.5 ppm (500 ppb) for all gases. The interference within this method is SO_2 which has the same retention time as COS, high concentration hydrocarbons which can have a quenching effect on the detector, and high concentrations (>10%) of carbon monoxide and carbon dioxide which have desensitizing effect on the FPD. The operating parameters of a GC-FPD system (fitted with a, a sampling valve with a Teflon sample loop system) are 40ºC oven temperature, 10 min. holding time, 200ºC detector temperature and N_2 carrier gas. Any analytical column that can separate reduced sulphur gases and SO_2 should be used.

An impinger technique for the determination of TRS emissions from Kraft pulp mills and other sources is employed at *Method 16A Determination of Total Reduced Sulfur Emissions from Stationary Sources*, State of California, Air Resources Board (1987, amended 1999). The method is equivalent to Environment Canada Reference Method EPS 1/RM/6 for TRS. The TRS compounds include hydrogen sulfide, methyl mercaptan, dimethyl sulfide, and dimethyl disulfide. The flue gas must contain at least 1% oxygen for complete oxidation of TRS to SO_2. In this method a gas sample is extracted from the stack. The SO_2 is removed selectively from the sample using a citrate buffer solution. The TRS compounds are then thermally oxidized to SO_2 and analyzed as SO_2 by gas chromatography (GC) with an FPD. Utilizing a 1 ml sample size, the maximum SO_2 limit of the GC-FPD system is approximately 10 ppm. This limit is expanded by dilution of the sample gas before analysis or by reducing the sample size. For sources with emission levels between 10 and 100 ppm, reducing the sample size can best extend the measuring range. Carbonyl sulfide, which is partially oxidized to SO_2 and may be present in a lime kiln exit stack, would be a positive interferent. The schematic diagram of Method 16A is shown in Figure 2.
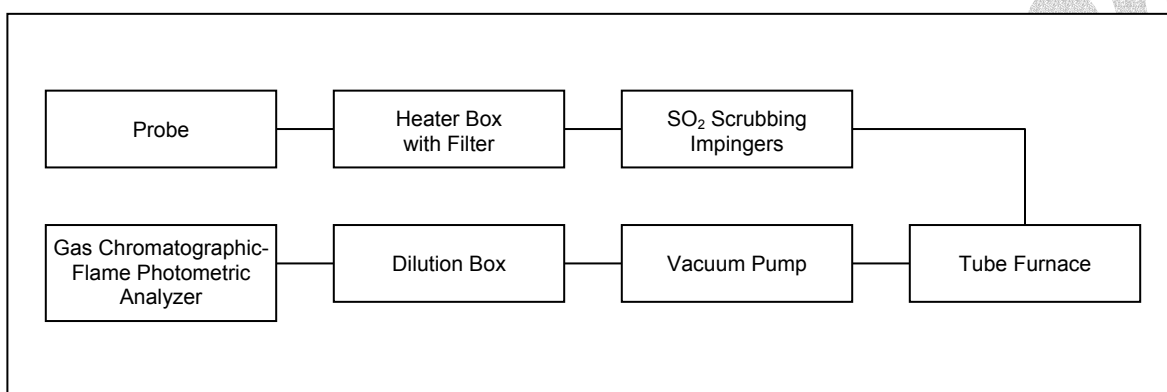
Figure 2: Schematic Diagram of Method 16A

The specificity of the RSC monitors depends on the thermal oxidizer temperature. Low temperature oxidation (300 to 400ºC) is relatively efficient at converting H_2S to SO_2 but is much less effective in oxidizing other RSC. The conversion efficiency of low temperature oxidation for methyl mercaptan and dimethyl disulphide is on the order of 45% and 15%, respectively. Analyzers with low thermal oxidizer temperature are considered to be more H_2S specific. High temperature (900ºC) thermal oxidation analyzers, which are used at RSC monitoring stations, oxidize all RSC to SO_2 with an efficiency of 90-100%. It represents a disadvantage at pulp and paper operations, but efficient at sour gas plants because it oxidizes sulphur containing compounds such as carbon disulphide and carbonyl sulphide to SO_2. Therefore the presence of these compounds will result in an overestimation of the amount of RSC at pulp and paper operations where the gases of interest are H_2S, methyl mercaptan, dimethyl sulphide and dimethyl disulphide, but efficient at sour gas plants where the gases of interest are H_2S, carbon disulphide and carbonyl sulphide.

In addition to gas chromatography with a flame photometric detector (FPD), flame-ionization detector (FID), or photoionization detector (PID), the following instrumental measurement techniques for RSC are used:

- GC-thermal conductivity detector;
- GC-chemiluminescence;
- non-dispersive infrared (NDIR) absorption;
- non-dispersive ultraviolet (NDUV) fluorescence;
- electrochemical cells;
- titrimetric analysis (manual monitoring); and
- uv-visible spectrometry (manual monitoring).

Detailed description of the above techniques is provided by Environment Agency of the National Compliance Assessment Service (2001).

Examples of currently available H_2S and TRS analyzers are provided in Table 24.

Table 23: Characteristics of Selected Analyzers for
H_2S and TRS Commercially Available on the Market

Manufacturer/Model	Monitored Parameters	Principle of Operation	Mode of Operation	Concentration Ranges	Detection Limit
Teledyne Analytical Instruments/6200 A	Total sulphides	UV fluorescence	Continuous – ambient air	0-50 ppb to 0-20,000 ppb full-scale, user selectable	0.4 ppb
Thermo Environmental Systems/AR100	TRS	UV fluorescence	Continuous – stacks	0-12.5 ppm or 0-30 ppm as SO_2	0.1 ppm
GASTEC/Tubes	H_2S	Adsorption/ colour reaction	Manual – periodic	0.25 ppm to 20% depending on tube selected	0.25 ppm
Analytical Systems International/HS	H_2S	Blackening tape	Continuous – ambient	0.1-100 ppm or 100-10,000 ppm	0.1 ppm
ENMET Corporation/Spectrum 3E	H_2S	UV fluorescence	Continuous – stacks/ ambient	0-200 ppm	0.1 ppm
API Advanced Instrumentation/102 A	TRS	UV fluorescence	Continuous – ambient	0-50 ppb or 0-20 ppm	0.4 ppb

8.0   REDUCED SULPHUR COMPOUNDS MANAGEMENT

8.1   Options and Approaches for Air Quality Management of RSC

There are a number approaches available as the basis of an ambient air quality guideline for RSC. In general, ambient air quality guidelines are based upon the scientific investigation of the responses to measured concentrations of individual substances, given under controlled conditions. Use of the data obtained from controlled experiments may be applied in a number of different ways. These include:

- guidelines based upon the concentration of a single RSC;

- guidelines based upon the aggregate total concentrations of RSC;

- guidelines based upon the concentration of a single RSC in air, which is meant to represent more than the single RSC (surrogate approach); and

- guidelines based upon a calculated concentration of RSC, expressed as equivalents to a single RSC (index approach).

An exception to this generalization is the use of some chemical or physical characteristic of the substance(s) as a surrogate for concentration measurements. As some RSC are generally odourous, the perception of odour at low chemical levels has been used to establish limits or guidelines on some RSC concentrations in air.

8.1.1   Guidelines Based Upon the Concentration of a Single RSC

Most ambient air quality guidelines are based upon the concentration of a single substance in air, expressed as a concentration averaged over a stated duration. Alberta's current H_2S guideline is in this form: 14 $\mu g\ m^{-3}$ for a 1-hour period. This form of a guideline is most straightforward to apply in air quality management, since it is relatively easy to use as a modeling benchmark, to reference in approvals, and to calculate on the basis of monitoring data.

Sufficient cause and effect data must be available to support the development of this style of guideline. Should more than one averaging period (e.g., hourly, daily, annual) be desired, there must be sufficient scientific literature to support each of the desired averaging periods. Alternatively, in the presence of information describing the responses of a receptor to the substance over a range of exposure durations it may be apparent that two or more averaging periods are needed within a guideline for a single substance, or RSC.

The advantages of a guideline based upon the concentrations of a single RSC are (i) the ease of application in air quality management and monitoring programs, and (ii) the generally larger numbers of scientific and related publications that are available to support the development of such a guideline. The disadvantages are usually limited information with which to use in determining the appropriate averaging period for the guideline, and a difficulty in handling intermittent exposures in deriving a guideline, or in evaluating potential impacts due to intermittent exposures. Nevertheless, this is the simplest form of the guideline, and should be the first form investigated for any substance, including RSC.

8.1.2   Guidelines Based Upon the Aggregate Total Concentrations of RSC

For families of substances, such as RSC, it may be possible to develop a guideline based upon the sum of the concentrations of all or some members of the chemical family.

While the total concentration of substances in the chemical family may be the same from one place or time to another, differences in the relative concentrations among family members may result in different effects or impacts over time and space. This may result in a low level of confidence in the guideline. The range of potential impacts at a single total RSC concentration, but at different relative concentrations of substances within the composite RSC exposure, may make it impossible to derive a defensible guideline.

An advantage to this form of guideline is that monitoring of ambient levels may be easier, and it is possible to measure the concentration of the entire family at one time. Measuring total reduced sulphur in air, expressed as a concentration of RSC, yields a single monitoring value. If it can be shown that the environmental and health impacts due to exposure to each of the individual substances are approximately equivalent, a guideline based upon the total concentration of the family of substances may be justified.

The review of RSC management approaches in various jurisdictions (discussed below) indicates that this is the most common approach to RSC management. Many jurisdictions manage RSC on the basis of total RSC in air, generally presumed to be the sum of concentrations of four individual RSC substances: H_2S, dimethyl disulphide, dimethyl sulphide, and methyl mercaptan. In many cases, it is presumed that the primary substance in RSC measurements is H_2S (Nebraska, British Columbia and Ontario, pers. comm.). This strategy balances the need to measure and manage the total RSC in air, while recognizing that a subset of species in the RSC family are the primary emissions that are generally responsible for the majority of impacts. The primary deficiency is that the actual substances within the sample are unknown, and therefore, potential impacts due to exposure to air containing these substances at various concentrations cannot be predicted.

8.1.3   Guidelines Based Upon the Concentration of a Single RSC Which is Meant to Represent More than the Single RSC (Surrogate Approach)

This is an approach similar in concept to the use of total RSC concentrations as the basis of a guideline. If emissions and/or atmospheric chemical reactions can be demonstrated to result in a consistent relationship in concentrations of two or more RSC in air, the selection of the most easily measured of these RSC as the basis of a guideline may be possible. The selected RSC would then act as a surrogate for either another RSC of interest or for the family as a whole.

The advantages of this method are similar to the derivation of a guideline for total RSC. The disadvantages are also similar: generally insufficient data upon which to select and properly derive a guideline using a surrogate approach, and uncertainty regarding the protective nature of a guideline meant to prevent impacts from a range of substances that is based upon a single substance.

8.1.4 Guidelines Based Upon a Calculated Value that is Based Upon Concentrations of RSC and Known Effects of Various RSC (Index Approach)

Integration of the concentrations at which different RSC cause different impacts into a single value may provide an opportunity to address some of the disadvantages associated with the approaches discussed above. It may be possible to derive a mathematical expression within which different weights are given to different RSC on the basis of their relative toxicity. In the situation where sufficient data are available upon which to develop such an equation, or index, a guideline that would be relatively protective and defensible could be developed. A simple illustration, using three substances, shows the attraction of an index.

In the situation where RSC_1 is twice as toxic (or damaging) as RSC_2, and five times more toxic than RSC_3:

$$RSC_1 = 2RSC_2, \text{ and}$$
$$RSC_1 = 5RSC_3$$

and it can be shown that:

$$2RSC_2 = 5RSC_3,$$

an index in the form of

$$RSC_1 + 2RSC_2 + 5RSC_3 < RSC_t$$

where RSC_t is "total RSC concentration" can be derived as the basis of a guideline (for a defined time period, e.g., 1 hour, 1 day).

This approach allows for integration of relative toxicity and ambient concentration into a single value (RSC_t), something that can be used relatively easily as a reference point in monitoring programs, or referenced in approvals or other regulatory documents.

The disadvantages of this approach are also similar to those discussed above for other approaches. In addition, there must not only be sufficient cause and effect data available for each individual substance in the index, but also sufficient data to support the assignment of relative weightings to the various substances in the index. With sufficient data it may be possible to define the relationships among RSC_1, RSC_2, and RSC_3 under one set of conditions and/or for one receptor at a particular growth stage or physiological condition. However, one or more of these relationships may not hold for a different receptor, or for the same receptor at a different growth stage or physiological condition. In this case the relationships may be more complex that can be determined from limited scientific effects data. Unless there is a very large amount of scientific information available to support the development of such relationships among RSC, it is suggested that an attempt to develop a RSC guideline based upon an index would be a futile effort.

8.1.5 The Use of Odour in the Development of an RSC Guideline

Some members of the RSC family of substances are odourous at low concentrations. This property differentiates them from many other air pollutants that cannot be detected by smell at levels that may cause harm. This opens the possibility for the development of a guideline based upon odour, rather than on demonstrated cause and effect relationships. Panels of individuals, either randomly selected or selected for an enhanced ability to smell the substance of interest, are generally used to establish odour thresholds and limits.

In this way, odour becomes a surrogate for instrumentation to estimate concentrations of RSC in air. Acceptance of odour as the basis for the establishment of an RSC guideline is predicated on the assumption that there is a relationship between the intensity of the odour, or the concentration threshold at which an odour can be detected, and defined health and/or environmental impacts. Developing and confirming such a relationship may be as difficult as conducting the experiments required to provide data in support of any form of guideline.

The use of odour as the basis of a guideline is predicated on the assumption that an ability to detect (smell) a substance is an indication of the danger present in being exposed to that substance. This assumption is essentially based upon an evolutionary argument. An ability to smell a substance at non-lethal or non-damaging levels would provide a survival advantage relative to those who are unable to detect the substance by smell. Over time, a greater percentage of the population will carry the genes that allow for detection of the substance at non-toxic levels.

This evolutionary argument can only be valid if the substance has been prevalent over a very long period of time. While H_2S and some other RSC may qualify, many substances now entering the atmosphere are of artificial, human creation. Regardless of whether the substance is relatively new, or has been a natural component of the Earth's history, any argument regarding the relationship between smell and toxicity is invalid until empirical evidence is available to establish a relationship between smell and negative effect or impact.

Exposure to the same substance for long periods, or for frequent intermittent periods of variable duration, may alter the perception of smell. In the case of RSC, and particularly H_2S, exposure is harmful to the olfactory system, and over time, the ability to smell the characteristic odour lost. When odour is used to define a threshold level for concentration, people must be selected very carefully to participate in the odour panel to avoid individuals who may have an impaired ability to smell the substance due to prior exposure to the substance, or to related substances.

While it may be stating the obvious, it is worthwhile noting that odour guidelines will only be based upon and is applicable to human health or aesthetics. It remains critical to obtain empirical cause and effect data to ensure protection of all other receptors at effects levels identified through the use of odour panels.

The remainder of this section reviews RSC management approaches used in several jurisdictions. Many jurisdictions utilize approaches based upon total RSC in air, and where possible the individual RSC species included in the total are stated. However, in many cases total RSC is presumed to be the sum of concentrations of a limited set of substances, usually

H$_2$S, dimethyl disulphide, dimethyl sulphide, and methyl mercaptan, with the primary constituent being H$_2$S.

8.2   RSC Management Approaches – Domestic

In Canada, British Columbia, Ontario and Alberta have management tools to control emissions and/or to establish ambient concentrations of reduced sulphur compounds.

8.2.1   British Columbia

In British Columbia, ambient air quality standards are used to facilitate the management of RSC (Table 25). British Columbia, the RSC definition includes H$_2$S and mercaptan compounds. A more detailed mercaptan list is not available.

Table 24: British Columbia Ambient Air Standards for H$_2$S and RSC (μg m^{-3})

RSC	Period	Maximum Desirable Level A	Maximum Acceptable Level B	Maximum Tolerable Level C
H$_2$S	1 hour	7.5 to 14	28 to 45	42 to 45
	24 hours	4	6 to 7.5	7.5 to 8
RSC	1 hour	7	28	
	24 hours	3	6	

The British Columbia ambient standards are based on prevention of human and environmental effects. There exist no province-wide ambient standards. Instead, British Columbia is divided up into management areas; each area is to comply with the ambient standards that are established for the industry that exists within that management area. The ambient air quality guidelines for petrochemical and chemical industries are provided in Table 26. Note that these guidelines are specific to the petroleum and chemical industries and fall within the concentration ranges shown in Table 25. These guidelines reflect the technological abilities of each sector to regulate emissions. The problems with this approach are that air emissions from a source could easily be dispersed into, and have impacts within other management areas, thereby potentially preventing the meeting of the management goal in the adjacent region. Also, if there exists more than one industry type within a management area, the permit manager must decide which industry standard to follow.

**Table 25: British Columbia Ambient H$_2$S Air Quality Guidelines
for the Petroleum and Chemical Industries (μg m^{-3})**

	Level A	Level B	Level C
1 hour maximum	7.5	45	45
24 hour maximum		7.5	7.5

The ambient guidelines are considered minimum desirable levels of air quality. Both H_2S and reduced sulphur compounds have desirable ambient concentration ranges within A, B and C classifications (Table 25). These ranges result from the different standards, which exists for different industries. There are no legal definitions for A, B and C classifications; they are used to assess environmental quality on a site-by-site, case-by-case basis. The three levels are described as follows:

- ***Level A (Maximum Desirable):*** Designed to provide long-term protection for all environments. This level is reasonable for polluted areas to aim for and achieve. This level represents a conservative approach of protecting the most sensitive receptor, thereby providing a large margin of safety to protect other less sensitive receptors.

- ***Level B (Maximum Acceptable):*** Intended to be the acceptable interim objective. This level provides adequate protection against adverse effects on human health, comfort, vegetation, animals, soil, water, materials, and visibility.

- ***Level C (Maximum Tolerable):*** Defines the immediate ambient objective. Due to a diminishing margin of safety, appropriate action is immediately required to protect the health of the general population when concentrations of air contaminants exceed this level.

The Regional Permit Managers are each responsible for permitting new and existing facilities and it is their responsibility to classify the regions as having to meet level A, B or C guidelines.

8.2.2   Ontario

The Ontario Ministry of Environment (MOE) sets environmental quality standards to protect human and ecosystem health, prevent damage to the physical environment and minimize offensive odours. The MOE has both ambient air quality criteria (AAQC) and the corresponding Point Of Impingement (POI) Limits for more than 300 compounds. The AAQC are used for the assessment of general air quality and the potential for adverse effect. The POI limits are used to review emission levels and certificate of approval applications for facilities. The POI limit is defined as the maximum ground level concentration at a location in the vicinity of a facility. Air dispersion models are used to locate the appropriate place of emission measurement. Within this list of 300 compounds there are standards for the reduced sulphur compounds carbonyl disulphide, dimethyl disulphide, dimethyl sulphide, and H_2S. For Ontario, the total reduced sulphur compounds definition includes H_2S, methyl mercaptan, dimethyl sulphide, dimethyl disulphide. The POI limits and AAQC for four reduced sulphur compounds are listed in Table 27. Although, the intentions of POI limits are to protect health and the environment, they are based on odour thresholds (OME, year unknown).

**Table 26: Ontario Point of Impingement Guidelines and Air Quality Criteria
for Carbonyl Disulphide, Dimethyl Disulphide, Dimethyl Sulphide, and H$_2$S**

Contaminant Name	Point of Impingement (POI) Limit		Ambient Air Quality Criteria (AAQC)		
	Half-hour POI Limit (µg m^{-3})	POI Limiting Effect	24-hour (µg m^{-3})	1-hour (µg m^{-3})	AAQC Limiting Effect
Carbon disulphide	*330	Odour	*330		Odour
Dimethyl disulphide	40	Odour		40	Odour
Dimethyl sulphide	30	Odour		30	Odour
H$_2$S	30	Odour		30	Odour
Reduced sulphur compounds (as hydrogen sulphide)	40	Odour		40	Odour

* The higher number is due to the higher odour threshold limit for carbon disulphide.

In Ontario, POI measurements establish concentrations of harmful pollutants within source emissions (OME, 2001). However direct emission source monitoring may eliminate the cost and potential errors associated with the dispersion modeling necessary in POI measurement. The setting of air quality standards for Ontario follows a multi-step process that incorporates the key elements of priority setting, risk management, and public consultation (OME, 2001; Appendix F). The multi-step process for standard establishment includes stakeholder participation.

8.2.3 Alberta

Alberta has established ambient air quality requirements that are designed to protect human health and the environment (Alberta Environment, 1999) (Table 28).

Table 27: Alberta Ambient H$_2$S and Carbonyl Sulphide Guidelines (µg m^{-3})

Compound	Period	Guideline
H$_2$S	1 hour	14
	24 hours	4
Carbonyl disulphide	1 hour	30
	24 hours	

8.3 RSC Management Approaches – North America

In the United States, Texas, Nebraska, California, and the United States Environmental Protection Agency have management tools to control emissions and/or to establish ambient concentrations of reduced sulphur compounds.

8.3.1 Texas

Texas State regulations on H$_2$S and RSC range from limits on ambient concentration and nuisance odours to standards applied during construction and operation. The Texas Natural Resources Conservation Commission (TNRCC) through the Texas Administrative code is responsible for the establishment of allowable off-property concentrations (ambient standards)

and calculation methods. The detrimental health effects were stated as the basis for H_2S and RSC concentration and emission limits. It is also stated that odour is not used as an indicator of exposure because the sense of smell is lost upon exposure to high concentrations to some sulphur compounds. Texas Administrative Code states that, "No person shall discharge from any source whatsoever one or more air contaminants or combinations of contaminants thereof, in such concentration and of such duration as are or may tend to be injurious or to adversely affect human health or welfare, animal life, vegetation, or property, or as to interfere with the normal use and enjoyment of animal life, vegetation, or property." Table 29 provides the ambient standards for H_2S in the State of Texas.

Table 28: State of Texas Ambient Concentration Standards ($\mu g\ m^{-3}$)

	Period	Maximum Level (Residential, Business or Commercial Property)	Maximum Level (Other Properties, Not Normally Occupied by People)
H_2S	30 minutes	113	170

The state of Texas regulates emission levels based on technology-based standards. They have established specific RSC limits, as H_2S, for Kraft pulp mills. These limits are specific to mill age and design. Specifications and limits are shown in Table 30.

Table 29: State of Texas Emission Guidelines Applied to Kraft Pulp Mills ($mg\ m^{-3}$)

	Maximum Level (Residential, Business or Commercial Property)		Maximum Level (Other Properties Not Normally Occupied by People)	
	Old Designs	New Designs	Cross Recovery	Digester System
RSC	28 mg m^{-3} as H_2S	8 mg m^{-3} as H_2S	35 mg m^{-3} as H_2S	7 mg m^{-3} as H_2S

The technology-based emission standards ensure that sources implement, within feasibility reason, the most current and effective technology available in order to reduce emissions. Technology-based standards, as opposed to effects-based standards, do not require effects data (human and/or environmental). This approach may be preferred when establishing a RSC guideline, when limited effects data are available. Nevertheless, RSC effects should be studied to ensure that technology-based standard do not exceed acceptable effects levels. Monitoring sources directly (stack emissions) may be a cost effective method in regulating air quality emissions, compared to guidelines requiring air dispersion modeling and an ambient monitoring network.

8.3.2   Nebraska

Title 129, Chapter 4, 007 of the Nebraska Air Quality Regulations details the RSC ambient standards as established by the Nebraska Department of Environmental Quality. The maximum ambient RSC concentration (95% of which is presumed to be accounted for by H_2S, methyl mercaptan, dimethyl sulphide and dimethyl disulphide) is 10.0 ppm (99.2 mg m^{-3}), one-minute average concentration and 0.10 ppm (0.99 mg m^{-3}), 30-minute average (Nebraska Air Quality Division, 2000). These values were based on a literature search that focused on low-level toxic

effects, as they are concentrations that are expected in ambient air. Technical data on Nebraska's ambient guideline is available in Appendix B. Values are summarized in Table 31.

Table 30: State of Nebraska Ambient Concentration Standards

Concentration	Period	Maximum Level (ppm)	Maximum Level (mg m^{-3})
RSC*	1 minute	10.0	99.2
	30 minutes	0.10	0.99

* 95% of which is presumed to be accounted for by H$_2$S, methyl mercaptan, dimethyl sulphide and dimethyl disulphide

8.3.3   California

The State of California has not established an ambient standard for total RSC. They do however, regulate H$_2$S concentrations under the California ambient air quality standard for H$_2$S: 42 µg m^{-3}, as a 1-hour maximum (Table 32) (Collins *et al*., 2000). This standard was established in 1969 and was based on the geometric mean odour threshold measured using a panel of 16 adults. The current guideline is slated for revision.

Table 31: State of California Ambient Concentration Standards (µg m^{-3})

	Period	Maximum Level
H$_2$S	1 hour	42

Table 32: Summary of RSC Ambient Standards (µg/m^3) Used by Various Jurisdictions

Contaminant Name	Period	British Columbia			Alberta	Ontario	Texas	
		Maximum Desirable A	Maximum Acceptable B	Maximum Tolerable C	Guideline		Maximum Level Residential Commercial Or Business Property	Minimum Level Other Properties, Not Normally Occupied By People
Total RSC*	1 minute							
	1 hour	7	28			40		
	24 hours	3	6					
Carbon Disulphide	1 hour				30			
	24 hours					330		
Dimethyl Disulphide	1 hour					30		
Hydrogen Sulphide	30 minutes							
	1 hour	7.5 – 14	28 – 45	42 – 45	14	30	113	170
	24 hours	4	6 – 7.5	7.5 – 8	4			
Dimethyl Sulphide						30		

Contaminant Name	Period	US EPA	Germany	United Kingdom	World Health Organization	State of Colorado	Norway	Nebraska	California
		Regulated By Technology Bases Emission Standards, Rather Than Air Quality Standards	No Established RSC Management	No Established RSC Management	No Established RSC Management	No Established RSC Management	No Established RSC Management		
Total RSC*	1 minute							10 ppm	
	30 minutes							0.10 ppm	
	24 hours								
Carbon Disulphide	1 hour								
	24 hours								
Dimethyl Disulphide	1 hour								
Hydrogen Sulphide	30 minutes								
	1 hour								
	24 hours								42
Dimethyl Sulphide									

9.0   DISCUSSION

The Ontario Point of Impingement guideline and the Air Quality Criteria for H_2S are both 30 µg/m^3, and are both based upon detection of odour. Similarly, the California 1-hour ambient H_2S standard of 42 µg/m^3 is based upon an odour threshold. Because of the distinctive odours associated with H_2S consideration should be given to the development and application of H_2S guidelines based on odour thresholds. Other than noting that other jurisdictions have established guidelines on the basis of H_2S odour detection, discussion is necessarily limited, as a review and assessment of health effects of H_2S is being conducted independently (by Alberta Health and Wellness). Therefore, a comparison of odour thresholds to effects levels was not addressed in this report.

While a comparison between guidelines established on the basis of detection of H_2S odour and health effects is not possible, a comparison is valid between odour detection and the concentration of H_2S demonstrated to affect vegetation. The highest level without demonstrated or observed vegetation effects is 140 µg/m^3 ($\approx$100 ppb). Thus, a guideline adopted or developed on the basis of H_2S odour detection (e.g., 30 to 42 µg/m^3) would be protective of vegetation. For other RSC, establishment of guidelines on the basis of odour may be an appropriate approach. Table 34 provides a comparison of odour thresholds to effects levels, for both health and vegetation impacts.

For all RSC for which odour thresholds have been identified, the odour threshold is lower than the health and vegetation effects levels. In addition, odours are detected quickly, within seconds of exposure, while effects are not generally observable unless exposures have been for periods of minutes or hours. This difference provides an added margin of conservatism. Thus, establishment of guidelines on the basis of odours should be an acceptable approach for the protection of health and vegetation against RSC effects (for those RSC for which odour thresholds and effects levels have been identified).

It is important to reiterate that the literature regarding health effects of RSC exposure is limited, and that any guidelines based upon this literature should be supported by research to confirm that the effects levels have been appropriately identified and characterized.

The review and summary of effects literature presented in this assessment report does not support the derivation of guidelines for individual RSC. There is insufficient effects information available to support such guidelines. In the case of carbon disulphide, the database of health effects is substantial, however, there is little information regarding exposure to low concentrations; it is to low concentrations that a person would normally be exposed.

Table 33: Comparison of Odour Thresholds to Human Health and Vegetation Effects Levels for Various RSC

Reduced Sulphur Compound	Odour Threshold	Health Effects Level [endpoint]	Vegetation Effects Level [endpoint]
Hydrogen Sulphide*	30 - 50 µg/m^3	30 - 50 µg/m^3	140 µg/m^3 [NOEC, for a variety of long-term exposures]
Methyl Mercaptan	31.5 µg/m^3	33,446 µg/m^3 [reduced body weight]	>11,340 µg/m^3 [NOEC, one experiment only]
n-Butyl Mercaptan	0.2 - 2 µg/m^3	369,000 µg/m^3 [maternal & embryotoxic effects]	N/A
Ethyl Mercaptan	36.9 µg/m^3	6,925,000 µg/m^3 [mortality]	N/A
t-Butyl Mercaptan	N/A	332,000 µg/m^3 [inflammatory lesions]	N/A
Propyl Mercaptan	2.3 µg/m^3	22,739,000 µg/m^3 [mortality]	N/A
Dimethyl Sulphide	2.5 - 160 µg/m^3	3,000,000 µg/m^3 [mucous membrane irritation]	N/A
Dimethyl Disulphide	N/A	30,071,000 µg/m^3 [mortality]	N/A
Carbon Disulphide	60 µg/m^3	3,400 µg/m^3 [neural conductance changes]	N/A
Methyl Disulphide	3 - 14 µg/m^3	7,500 µg/m^3 [microscopic liver damage]	N/A
Carbonyl Sulphide	N/A	123,000 µg/m^3 [histopathological effect]	172,200 µg/m^3 [lemon peel injury, limited literature]

* The average 1-hour maximum H$_2$S concentration observed in Alberta is 6.6 µg/m^3 (4.7 ppb) (from Table 9). The odour threshold stated for H$_2$S is based upon guidelines established on the basis of odour. The effects level for H$_2$S is based upon established guidelines, since a review of health effects of H$_2$S exposure was outside the scope of this report.

Additionally, in many studies, the duration exposure to carbon disulphide is undocumented, making interpretation of the information in a guideline context impossible. The absence of information also prevents the development of an RSC guideline using an index approach, with the additional constraint that the relative toxicities of the members of the RSC family under varying environmental and exposure conditions are unknown. It is necessary to understand these relative toxicities under variable conditions in order to effectively derive and apply an index approach.

For RSC guideline(s), different forms based upon relative concentrations of individual reduced sulphur species might be considered. Such guideline(s) may take the form of a "cumulative 1-hour concentration of RSC may not exceed X $\mu g/m^3$, of which no more than Y $\mu g/m^3$ may be H_2S, where Y<X".

This suggestion is based upon the availability of data and information on the effects of H_2S, and the identified need to establish guidelines, objectives or other management approaches for other RSC. The form of guideline proposed in this method is a blending of approaches to establish guidelines: one approach based upon effects as documented in the scientific literature, the second addressing the potential for management at the points of emission. The effects approach is reflected in the "Y" component, which sets a hard limit on the ambient air concentration of H_2S; a limit based upon the effects literature. The emissions management approach is reflected in the "X" component, which allows for emissions of the other RSC (and hence their presence in ambient air), while imposing limits on these emissions through the implementation of pollution control technology. The difference between "X" and "Y" can be determined on the basis of the technology to reduce RSC emissions – the more effective the technology, the closer that "X" will approach "Y".

On the basis of the available effects literature, the difference between "X" and "Y" is currently substantial. This is illustrated in a comparison of effects levels for each of the RSC discussed in this report, for both health and vegetation (Table 34). Concentrations required to induce an effect are several orders of magnitude higher for any RSC, relative to H_2S. Given the information available, an initial concentration of "Y" would likely be in the 10 to 50 $\mu g\ H_2S/m^3$ range. The concentration of "X" cannot be estimated, since in this approach it depends upon emission control technologies, which were not reviewed. If based upon the limited effects literature that is available, a concentration of 3,000 $\mu g\ RSC/m^3$ may represent a starting point, based upon the identified health effects of carbon disulphide.

The summary presented in Table 34 also illustrates that for those RSC for which sufficient information is available to compare health and vegetation effects levels, health effects generally occur at or below levels that affect vegetation. This applies to H_2S and carbonyl sulphide; a comparison for methyl mercaptan is not possible since the single paper describing results from vegetation exposure to methyl mercaptan did not use concentrations high enough to induce an impact.

It is recognized that the exposure duration is not accounted for in the information presented in Table 34. For the most part, exposures to the concentrations provided in Table 34 were short-term (24 hours to 1 week). There is insufficient information available to provide direct

comparisons for a variety of exposure periods, or to normalize the data to an expression of dose and compare normalized values.

There is insufficient health and vegetation effects literature available to derive and apply guidelines based upon a surrogate approach. No single RSC or subset of the RSC family of substances can be identified as a surrogate because of the lack of information on reduced sulphur effects.

10.0   CLOSURE

This report has been completed in fulfillment of the terms of reference for the project, and in response to requests and comments received from Alberta Environment.

Respectfully submitted,

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KF/sms

11.0   LITERATURE CITED

11.1   Published Literature

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Appendix A

Common Reduced Sulphur Compounds

Appendix A – Common Reduced Sulphur Compounds

Name	Formula
Hydrogen Sulphide	H_2S
Carbonyl Sulphide	COS
Methyl Mercaptan	CH_3SH
Ethyl Mercaptan	C_2H_5SH
Dimethyl Sulphide	$(CH_3)_2SH$
Carbon Disulphide	CS_2
i-Propyl Mercaptan	$(CH_3)_2CHSH$
n-Propyl Mercaptan	$CH_3(CH_2)_2SH$
tert-Butyl Mercaptan	$CH_3CH_2CHSHCH_3$
sec-Butyl Mercaptan	$CH_3CH_2CHSHCH_3$
i-Butyl Mercaptan	$CH_3CH_2CHSHCH_3$
n-Butyl Mercaptan	$CH_3(CH_2)_3SH$
Methyl Ethyl Sulphide	$C_2H_5SCH_3$
Diethyl Sulphide	$(C_2H_5)_2SH$
Dimethyl Disulphide	CH_3SSCH_3
Diethyl Disulphide	$C_2H_5SSC_2H_5$

Appendix C

Alberta Releases of Hydrogen Sulphide in
1999 and 2000 (NPRI Reports)

Appendix D

Alberta Releases of Carbon Disulphide in
1999 and 2000 (NPRI Reports)

Appendix E

Alberta Environment Total Reduced Sulphur Monitoring Data
1999 to 2001