Workplace Exposure Standard (WES) review

HYDROGEN SULPHIDE
(CAS NO: 7783-06-4)

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New Zealand Government

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1.0
Introduction

This WorkSafe New Zealand (WorkSafe) review considers whether the **WES** for hydrogen sulphide should be changed.

It considers the potential for exposures to hydrogen sulphide in New Zealand, the health effects and risks, exposure standards in other jurisdictions around the world, and the practicability of measuring hydrogen sulphide exposures.

The review includes a recommendation to change the current WorkSafe **WES**, which is currently set at a **WES-TWA** of 10 ppm (or 14 mg/m^3) and a **WES-STEL** of 15 ppm (or 21 mg/m^3), as published in the Special Guide *Workplace Exposure Standards and Biological Exposure Indices*, 9th Edition (WorkSafe New Zealand, 2017).

Terms that are **bold** (first occurrence only) are further defined in the Glossary.

Concentrations have been converted to **ppm** from **mg/m^3** (using the conversion factor in Table 1) unless the latter is specified as an occupational exposure standard.

Synonyms: Hydrogen sulphide; Hydrosulphuric acid; Stink damp; Sulphur hydride; Sulphureted hydrogen; Dihydrogen monosulphide; Dihydrogen sulphide; Sewer gas; Sulphur hydrogen; H_2S, (sulphide = sulfide).

2.0 Physical and chemical properties

Hydrogen sulphide is a colourless gas with a strong odour of 'rotten eggs'; while under pressure it is a colourless liquid.

Chemical and physical properties include:

Molecular weight	34.09
Specific gravity	1.192 (air = 1)
Formula	H_2S
Boiling point	-60.7°C at 101.3 kPa
Vapour pressure	2026 kPa at 25.5°C
Vapour density	1.19 (air = 1)
Solubility	0.4% in water; 2.1% in ether (w/w) at 20°C
Conversion factors	1 ppm = 1.39 mg/m³; 1 mg/m³ = 0.717 ppm at 25°C and 760 torr
Reactivity	Hydrogen sulphide is flammable and explosive; may be ignited by static discharge; and reacts with oxidisers. **LEL** = 4.3%, **UEL** = 45.5%; Auto-ignition temp 260°C.
HSNO classifications (EPA, 2017)	2.1.1A [Flammable Gases: High hazard]; 6.1B (inhalation) [Acutely toxic]; 6.3B [Mildly irritating to the skin]; 6.4A [Irritating to the eye]; 6.9A (inhalation) [Toxic to human target organs or systems]; 9.1A (fish, crustacean) [Very ecotoxic in the aquatic environment]

TABLE 1: Chemical and physical properties of hydrogen sulphide

It should be noted that individuals are reported to exhibit considerable variability in their odour threshold for hydrogen sulphide (0.0005 – 0.3 ppm) (**ACGIH**®, 2010) while at higher concentrations of 100 ppm or more, olfactory paralysis can occur preventing individuals from detecting the odour of hydrogen sulphide (**ATSDR**, 2016).

3.0
Exposure

Hydrogen sulphide is one of the principal compounds involved in the natural cycle of sulphur in the environment.

Hydrogen sulphide is often present in volcanic gases or in emissions from other geothermal activities. The substance is also produced by bacterial processes during the decay of both plants and animal protein or through the direct reduction of sulphate, for example in sewers, cesspools and stagnant water (**SCOEL**, 2007).

Occupational exposure to hydrogen sulphide is primarily a problem in the 'sour gas' segment of the natural gas industry, where natural gas with high concentrations of sulphur is processed. Large quantities of hydrogen sulphide are used in the production of deuterated water. Examples of industries where hydrogen sulphide can be generated include petroleum refineries, natural gas plants, petrochemical plants, coke oven plants, kraft paper mills, viscose rayon manufacture, sulphur production, iron smelters, food processing plants, tanneries, landfills, manure treatment plants and waste water treatment plants (SCOEL, 2007; ACGIH®, 2010; ATSDR, 2016).

The number of persons exposed or potentially exposed in New Zealand is expected to be relatively high, mostly as a result of natural sources of hydrogen sulphide.

Workers may be occupationally exposed to hydrogen sulphide via inhalation during its manufacture or use, or at work sites where hydrogen sulphide is released by chemical reaction or from natural sources. Exposure may also occur in the vicinity of facilities that manufacture or use this compound.

Statistics New Zealand data for 2016 indicate that 38,610 New Zealand workers were working in the industries identified as sites where hydrogen sulphide exposure could occur during manufacture or use (Table 2) (Statistics New Zealand, 2017). Other more specialised work sites with fewer staff may also exist. However, it is unlikely that all of these individuals are exposed, or potentially exposed, to hydrogen sulphide.

ANZSIC06	BUSINESS	EMPLOYEE COUNT
A016000	Dairy Cattle Farming	26,500
A019200	Pig Farming	400
B070000	Oil and Gas Extraction	820
C132000	Leather Tanning, Fur Dressing and Leather Product Manufacturing	1,400
C151000	Pulp, Paper and Paperboard Manufacturing	1,500
C170100	Petroleum Refining and Petroleum Fuel Manufacturing	730
C211000	Iron Smelting and Steel Manufacturing	1,250
D281200	Sewerage and Drainage Services	610
D29	Waste Collection, Treatment and Disposal Services	5,400
Total		**38,610**

(Statistics New Zealand, 2017)

TABLE 2:
Statistics New Zealand Business Demography Statistics 2016

4.0 Health effects of hydrogen sulphide

IN THIS SECTION:

The health effects of hydrogen sulphide have been investigated.

4.1 Non-cancer

Humans

ATSDR report that:

> "The human data suggest that the respiratory tract and nervous system are the most sensitive targets of hydrogen sulfide toxicity. The most commonly reported nonlethal effect found in individuals acutely exposed to high concentrations of hydrogen sulfide is unconsciousness followed by apparent recovery, colloquially referred to as "knockdown". In most cases, actual exposure concentrations and durations are not known; estimates suggest that the concentrations exceed 500 ppm and the durations are short, typically <1 hour. Although there is an apparent recovery, many individuals report permanent or persistent neurological effects including headaches, poor concentration ability and attention span, impaired short-term memory, and impaired motor function. Respiratory distress or arrest and pulmonary edema are also associated with exposure to very high concentrations of hydrogen sulfide; it is believed that these respiratory effects are secondary to central nervous system depression or due to tissue hypoxia. Cardiovascular effects (eg cardiac arrhythmia and tachycardia) have also been observed following an acute exposure to high concentrations of hydrogen sulfide.
>
> Exposure to lower concentrations of hydrogen sulfide can result in less severe neurological and respiratory effects. Reported neurological effects include incoordination, poor memory, hallucinations, personality changes, and anosmia (loss of sense of smell); the respiratory effects include nasal symptoms, sore throat, cough, and dyspnea. Impaired lung function has also been observed in asthmatics acutely exposed to 2 ppm hydrogen sulfide; no alterations in lung function were observed in studies of non-asthmatic workers." (ATSDR, 2016).

> "There are limited human data suggesting that maternal or paternal exposure to hydrogen sulfide can increase the risk of spontaneous abortion among rayon textile, paper products, or petrochemical workers (or their spouses). However, the subjects (or their spouses) were exposed to a number of other hazardous chemicals that may have contributed to the increased risk." (ATSDR, 2016).

A series of studies in healthy men and women exercising on a cycle ergometer for 15 – 30 minutes at different work rates while inhaling 0.5, 2.0, 5.0 or 10 ppm hydrogen sulphide have identified **NOAEL/LOAEL**s (Bhambhani and Singh, (1991); Bhambhani *et al*., (1994, 1996, 1997) cited in ACGIH® (2010) and ATSDR (2016)). No significant alterations in lung function were observed in individuals exposed to 10 ppm for 15 minutes (Bhambhani *et al*., (1996) cited in ACGIH® (2010) and ATSDR (2016)), but increases in respiratory exchange ratio (**RER**) and blood lactate levels were observed in subjects exposed to 5 or 10 ppm (Bhambhani and Singh (1991); Bhambhani *et al*., (1997) cited in ACGIH® (2010) and ATSDR (2016)). No significant effects of hydrogen sulphide exposure were reported in men at 0.5 or 2 ppm, though there were trends in data suggesting the beginning of a dose-response relationship. At 2 ppm, $\mathbf{VO_2}$ and blood lactate increased, while $\mathbf{VCO_2}$ decreased (Bhambhani and Singh (1991) cited in ACGIH® (2010) and ATSDR (2016)). The study authors noted that the increase in lactate levels suggested an increased dependence on anaerobic metabolism, which may have resulted from reduced oxygen availability due to detoxification of hydrogen sulphide by oxyhaemoglobin or inhibition of cytochrome oxidase in exercising tissue (Bhambhani, (1999) cited in ATSDR (2016)). Collectively the data from Bhambhani and associates indicates an apparent NOAEL and LOAEL of, respectively, 2.0 and 5.0 ppm (ACGIH®, 2010).

The ACGIH® review of hydrogen sulphide in 2010 noted that animal and human data showed similar qualitative and quantitative responses following single and repeated exposures to hydrogen sulphide. The ACGIH® also noted a growing consensus that hydrogen sulphide functions as an endogenously produced substance with important physiological functions (Kimura et al., 2005 cited by (ACGIH®, 2010).

However they state that:

> "Concentrations of 1000 to 2000 ppm produced loss of consciousness and possible lethality in both animals and humans (US EPA, 2003; ATSDR, 1999). Concentrations of 100 to 1000 ppm were generally involved in serious effects in the respiratory, central nervous, and cardio-vascular systems (Beauchamp et al., 1984; Milby, 1962; Milby and Baselt, 1999). Olfactory fatigue occurred in humans at concentrations of 150 to 200 ppm (Nordic Council of Ministers, 2001; Fuller and Suruda, 2000; van Aalst et al., 2000). Moderate irritation of the eyes occurred between 5 and 30 ppm in both animals and humans (Grant, 1986; Nesswetha, 1969; Elkins, 1950, Masure, 1950; Barthemy, 1939). Metabolic changes were seen in exercising individuals exposed to 5 or 10 ppm hydrogen sulphide during short-term exposures (15-30 minutes) (Bhambhani and Singh, 1991; Bhambhani et al., 1994, 1996a, b, 1997). The workplace does include exercising individuals and metabolic changes, though relatively minor, were not detected in exercising individuals who were not also exposed to hydrogen sulphide. In these exercising individuals, changes were not considered clinically significant but were seen at exposures less than 5 ppm. A study of 74 healthy individuals found an increase in symptoms related to anxiety following a single exposure to 5 ppm, but not to 0.05 or 0.5 ppm hydrogen sulphide (Fiedler et al., 2008). The start of the dose-response curve for short-term human exposure appears to reside around 5 ppm. Metabolic changes have also been shown in animals exposed to hydrogen sulphide at concentrations of 20 ppm and above (Blackstone et al., 2005). In rats and mice, repeated exposures at 30 ppm, but not 10 ppm, produced minimal changes in the nasal mucosae (Dorman et al., 2004; Brenneman et al., 2000)." (References cited by (ACGIH®, 2010).

> SCOEL stated: "There is limited human information concerning the health effects after prolonged exposure to H_2S as [acute exposure]. Exposure to 1 – 5.6 mg/m^3 H_2S caused eye irritation in viscose rayon workers. However, eye irritation in these industries might be a result of combined exposure to other toxic agents (CS2 or acids), which might reduce the corneal threshold for irritation. There are no data concerning the effects of H_2S alone below levels of 28 mg/m^3 [20 ppm] (**DECOS**, 2006). One epidemiological study found effects on reproduction (increased spontaneous abortion) in women exposed to petrochemicals, including H_2S. However, these (limited) data are difficult to interpret due to the simultaneous exposure to CS_2, a known teratogen." (SCOEL, 2007).

The Dutch Expert Committee on Occupational Standards (DECOS) review of hydrogen sulphide in 2006, noted that: "There is limited information concerning the effects of H_2S after acute exposure. Only a few cases have been described in which acute exposure (to concentrations exceeding 1400 mg/m^3) caused a cessation of respiration. In asthmatics, exposure to 2.8 mg/m^3 H_2S [2 ppm] for 30 minutes did not result in statistically significant respiratory effects. Mouth only exposure for 15 minutes (to 14 mg/m^3) [10 ppm] did not cause significant changes in pulmonary functions." (DECOS, 2006).

Animals

ATSDR report that:

> "Animal studies confirm the human data suggesting that the respiratory tract and the nervous system are the most sensitive targets of hydrogen sulfide toxicity. As with humans, unconsciousness was observed in rats exposed to very high concentrations of hydrogen sulfide (800 ppm); central nervous system depression (as evidenced by lethargy) and pulmonary edema were observed in rats exposed to 400 ppm hydrogen sulfide for 4 hours. Decreased performance in neurological testing has been observed in rats exposed to 80 – 200 ppm hydrogen sulfide for 5 days to 11 weeks. Damage to the nasal olfactory epithelium is also observed in rats exposed to lower levels of hydrogen sulfide for an acute or intermediate duration; the adverse effect levels are 80 ppm (3 hours/day for 5 days) and 30 ppm (6 hours/day, 7 days/week for 10 weeks) following acute- or intermediate-duration exposure, respectively." (ATSDR, 2016).

A key intermediate-duration study identifying the NOAEL/LOAEL in rats was reported by Brenneman et al. (2000): groups of 12 male Sprague-Dawley rats were exposed to 0, 10, 30, or 80 ppm hydrogen sulfide for 6 hours/day, 7 days/week for 10 weeks. Parameters used to assess toxicity were limited to extensive histopathological examination of the nasal cavity. Nasal lesions occurred only in the olfactory mucosa in rats exposed to 30 or 80 ppm and consisted of multifocal, bilaterally symmetrical olfactory neuron loss and basal cell hyperplasia affecting the lining of the dorsal medial meatus and the dorsal and medial regions of the ethmoid recess. No olfactory lesions were observed in the controls or rats exposed to 10 ppm (Brenneman et al., (2000) cited in ATSDR (2016); ACGIH® (2010); SCOEL (2007); DECOS (2006)).

ATSDR report that:

> "No significant alterations in reproductive performance were observed in rats exposed to 10 – 80 ppm hydrogen sulfide for an intermediate duration. The available animal data suggest that hydrogen sulfide is not a developmental toxicant at concentrations of 80 ppm and lower. No structural anomalies, developmental delays, performance in developmental neurobehavioral tests, or alterations in brain histology were observed in a well-conducted rat study." (ATSDR, 2016).

The SCOEL review of hydrogen sulphide in 2007, in relation to studies in rats, noted that:

> "There is limited information concerning the effects of hydrogen sulphide (H_2S) after acute exposure. Only a few cases have been described in which acute exposure (to concentrations higher than 1,400 mg/m^3) caused breathing stops. Mouth only exposure for 15 minutes (to 14 mg/m^3) did not cause significant changes in pulmonary functions. In experimental animals, acute or short-term exposure to H_2S, resulted in inhibition of cytochrome oxidase in the lung cells, and local irritation of eyes and throat. Since the limited data available suggest that the dermal route is of minor importance, a skin notation is not needed." (SCOEL, 2007).
>
> "In rats, subchronic exposure to hydrogen sulphide (6 h/day, 7 days/week for 10 weeks) causes nasal lesions (olfactory neuron loss and basal cell hyperplasia) (Brenneman 2000; Moulin 2002, Dorman 2004). The NOAEL for this effect was 14 mg/m^3. Inhibition of cytochrome oxidase has been observed in rat lung cells after short exposure (3-4 hours for 1 to 4 days) to levels of H_2S of 42 mg/m^3 and higher, with a NOAEL of 14 mg/m^3 as well". References cited in SCOEL (2007).
>
> "No effects on reproduction and development were reported in rats exposed to H_2S (14, 42 and 112 mg/m^3) during mating, gestation and lactation (Dorman et al, 2000). In the same study no effects on growth, development and behaviour of the pups were found. No gross or microscopic abnormalities were observed in the central nervous system of the offspring. In the studies from Hannah et al (1989 and 1991) and Skrajny et al (1992), slight neurological effects on offspring were found at levels of 20 ppm (7 h/day during pregnancy until 21 days postnatal) and higher" (References cited in SCOEL (2007).
>
> "Acute or short-term exposure of experimental animals to hydrogen sulphide, resulted in inhibition of cytochrome oxidase in the lung cells, and local irritation of eyes and throat.

The Dutch Expert Committee on Occupational Standards (**DECOS**) is of the opinion that the data concerning acute or short term exposure show that a short term exposure limit is not indicated. They say:

> "In rats, exposure to H_2S causes nasal lesions (olfactory neuron loss and basal cell hyperplasia) and bronchial epithelial hyperthophy (sic) and hyperplasia after exposure to H_2S for 70 – 90 days (6 hours/day, 7 days/week). The NOAEL (no observed adverse effect level) for these effects is 14 mg/m^3. The committee is of the opinion that these are the critical effect [effects] [based on studies by Brenneman (2000); Moulin (2002); Dorman (2004)]". (DECOS, 2006).

4.2 Cancer

Humans

There are limited data on the potential for hydrogen sulphide to induce cancer in humans. One study found significant increases in the risk of developing cancers of the trachea, bronchus, and lung among residents exposed to high levels of naturally occurring hydrogen sulphide. However, the elevated disease rates were consistent with exposure to high concentrations of hydrogen sulphide and mercury; the contribution of mercury to the overall respiratory tract cancer rates cannot be determined from these data. Another study did not find significant alterations in cancer incidences among residents living near natural gas refineries (ATSDR, 2016).

Animals

The carcinogenicity of hydrogen sulphide has not been assessed in animal studies (ATSDR, 2016).

SCOEL found no data concerning the carcinogenic effects of hydrogen sulphide (SCOEL, 2007).

4.3 Absorption, distribution, metabolism and excretion

There is evidence that hydrogen sulphide functions as an endogenously produced substance with important physiological functions, including as a neuromodulator and smooth muscle relaxant (Kimura et al. (2005) cited in ACGIH® (2010)). Hydrogen sulphide is primarily absorbed via the respiratory tract, with some absorption through the skin and digestive tract lining. Some of the sulphide may be trapped by natural disulphide in the blood, while some is excreted via sulphide in the faeces (ACGIH®, 2010).

Results from animal inhalation studies show that hydrogen sulphide is widely distributed in the body. Hydrogen sulphide is metabolised by oxidation, methylation, and reaction with metallo- or disulphide-containing proteins (Sullivan and Krieger, 1992 cited in ACGIH® (2010)).

Hydrogen sulphide disrupts oxidation enzyme function, significantly the cytochrome oxidase system resulting in hypoxia (Rom, 1992 cited in ACGIH® (2010)). The affinities for the binding site on cytochrome c oxidase, the last step of electron transport in mitochondria of cyanide and sulphide, were of the same order of magnitude (Wever et al., 1975 cited in ACGIH® (2010)).

In the blood, hydrogen sulphide gas is associated with alkaline sulphide and the hydrosulphide anion is excreted via the lungs and urine (Booth and McDonald, (1982) cited in ACGIH® (2010)). Hydrogen sulphide is not considered a cumulative toxin as it is rapidly oxidised to sulphate, which is readily excreted by the kidneys (US EPA (2003) cited in ACGIH® (2010)).

5.0 Exposure standards and guidance values in use around the world

IN THIS SECTION:

Table 3 below shows the hydrogen sulphide exposure standards from around the world.

The information is published by the Institute for Occupational Safety and Health of the German Social Accident Insurance (Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung, 2017). WorkSafe, ACGIH® and SCOEL exposure limits are included for reference purposes.

JURISDICTION OR ADVISORY BODY	8-HOUR LIMIT VALUE		SHORT-TERM LIMIT VALUE	
	ppm	mg/m³	ppm	mg/m³
Australia	10	14	15	21
Austria	5	7	5	7
Belgium	5	7	10	14
Canada – Ontario	10		15	
Canada – Quebec	10	14	15	21
Denmark	10	15	20	30
Finland	5	7	10	14
France[1]	5	7	10	14
Germany (AGS)	5	7.1	10	14.2
Germany (DFG)	5	7.1	10	14.2
Hungary		14		14
Ireland	5	7	10	14
New Zealand	10	14	15	21
Poland		7		14
Singapore	10	14	15	21
South Korea	10	14	15	21
Spain	1		5	
Switzerland	5	7.1	10	14.2
The Netherlands		2.3		
USA – NIOSH			10[2]	15[2]
USA – OSHA	4		20	
UK	5	7	10	14
ACGIH® (2010)	1	1.4	5	7
SCOEL (2007)	5	7	10	14

TABLE 3: Exposure standards for hydrogen sulphide from around the world

It is noted that the only organisations from whom we get information as to how and why they set occupational exposures standards are ACGIH®, SCOEL and DECOS.

[1] Restrictive statutory limit values.

[2] Ceiling limit value (10 min).

5.1 New Zealand

WorkSafe's WESs values for hydrogen sulphide have been unchanged since their adoption in 1994.

The toxicological database reviewed indicates that hydrogen sulphide has a clear threshold dose/exposure level below which exposure is not expected to lead to adverse effects. The dose-response curve for short-term human exposure appears to start at around 7 mg/m^3 (5 ppm) – which may produce minor irritation and a brief change in oxygen uptake, but would not be expected to produce serious effects on the respiratory, central nervous, or cardio-vascular systems. Sub-chronic studies in rats indicated that nasal lesions (olfactory neuron loss and basal cell hyperplasia) and bronchial epithelial hypertrophy and hyperplasia were the critical endpoints, with a NOAEL of 14 mg/m^3 (10 ppm).

Based on the aforementioned documentation, WorkSafe does not consider its current WES-TWA of 10 ppm or WES-STEL of 15 ppm for hydrogen sulphide to be adequate to manage health risk from inhalation exposure.

5.2 ACGIH®

The ACGIH® **TLV-TWA** of 1 ppm and **TLV-STEL** of 5 ppm for hydrogen sulphide are based on human data that indicated the start of the dose-response curve for short-term human exposure was around 5 ppm (ACGIH®, 2010).

The ACGIH® review concluded that peak exposures of 5 ppm may produce minor irritation and a brief change in oxygen uptake, but would not be expected to produce serious effects on the respiratory, central nervous, or cardio-vascular systems. Therefore, a TLV-STEL of 5 ppm is recommended. Sufficient data were not available to recommend **Skin**, **SEN** or carcinogenicity cancer notations.

Data from Bhambhani and Singh (1991), and Bhambhani et al (1994, 1996, 1997) as referenced by ACGIH® (2010) suggested that 2 ppm and 5 ppm were apparent NOAEL and LOAELs respectively for hydrogen sulphide.

5.3 SCOEL

The SCOEL **OEL** for hydrogen sulphide is health-based, as the review of the total available scientific database leads to the conclusion that it is possible to identify a clear threshold dose/exposure level below which exposure to the substance in question is not expected to lead to adverse effects (SCOEL, 2007).

The SCOEL review (SCOEL, 2007) concluded that nasal lesions (olfactory neuron loss and basal cell hyperplasia) and bronchial epithelial hypertrophy and hyperplasia in rats after exposure to hydrogen sulphide for 70 – 90 days (6 hours/day, 7 days/week) were the critical endpoints for setting an OEL (NOAEL of 14 mg/m^3 (10 ppm) (SCOEL, 2007). The SCOEL review concluded that a compensation for differences in exposure pattern in the experimental setting (subchronic) and occupational setting (chronic), and for the limited dataset concerning the pathological effects was warranted. As such, starting from a NOAEL of 10 ppm and using an uncertainty factor of 2, SCOEL proposed an 8-h TWA of 5 ppm (SCOEL, 2007).

SCOEL has not recommended notations for sensitisation nor for the potential for absorption through the skin.

SCOEL considered an uncertainty factor to compensate for the differences between rats and humans:

> "unnecessary, as the critical effects found are local (non systemic) and rats are predominantly nose breathers which might lead to higher local (nasal) concentrations. However, a compensation for differences in exposure pattern in the experimental setting (subchronic) and occupational setting (chronic) and for the limited dataset concerning the pathological effects is warranted. For these aspects together, a factor of 2 is proposed, taking also into account that systemic effects (a significant decrease in oxygen uptake with an increase in blood lactate) have been found after short term exposure (Bhambhani et al, 1991). Considering all these aspects and the preferred value approach in setting OELs, starting from a NOAEL of 10 ppm (14 mg/m^3) and using an uncertainty factor of 2, SCOEL proposes an 8-h TWA of 5 ppm (7 mg/m^3) for H_2S" (References cited by SCOEL (2007).
>
> "Given the nature of the acute toxic effects such as eye irritation, unconsciousness and persistent neurological disorders and the fact that short-term exposures do occur in industrial settings, a STEL of 10 ppm is recommended. Moreover, it is strongly advised to avoid exposure to rapid rising high peaks." (SCOEL, 2007).

5.4 DECOS

The DECOS review (DECOS, 2006) also concluded that nasal lesions (olfactory neuron loss and basal cell hyperplasia) and bronchial epithelial hypertrophy and hyperplasia in rats after exposure to hydrogen sulphide for 70-90 days (6 hours/day, 7 days/week) was the critical endpoint for setting an OEL (NOAEL of 14 mg/m^3), based on the studies by Brenneman et al. (2000, 2002), Moulin et al. (2002), and Dorman et al. (2004). The DECOS review concluded that compensation for differences in exposure pattern in the experimental setting (subchronic) and occupational setting (chronic) and for the limited dataset concerning the pathological effects was warranted, a factor of 2, plus compensation for interindividual differences, a factor of 3 [6 total] (DECOS, 2006).

> "Considering all these aspects, starting from a NOAEL of 14 mg/m^3 and using an extrapolation factor of 6 the committee recommends an **HBROEL** 8 hour WES for hydrogen sulphide of 2.3 mg/m^3 (~1.7 ppm)." (DECOS, 2006)

6.0 Analytical methods for the assessment of airborne hydrogen sulphide

A common practice in New Zealand to measure hydrogen sulphide is using OSHA Method 1008 (OSHA, 2006).

Using this method, an air sample of 12 litres is collected onto a hydrogen sulphide sampler containing silver nitrate-coated silica gel using a sampling train set at a flow rate of 50 ml of air per minute. Other sampling media are also acceptable, as per manufacturers and analytical laboratory guidelines. During sampling, the hydrogen sulphide reacts to form silver sulphide. Following desorption of the sulphide using alkaline sodium cyanide solution, and subsequent oxidation with hydrogen peroxide, the resultant sulphate is determined by ion chromatography using conductivity detection. The detection limit of this method is 7.5 µg per sample for hydrogen sulphide.

This would allow a minimum concentration of 0.5 ppm of hydrogen sulphide to be quantified over a collection period of 4 hours.

Short-term exposures could also be assessed using this method by collecting 7.5 litres of air using a flow rate of 500 ml or air per minute, for 15 minutes. This would allow a minimum concentration of 0.7 ppm of hydrogen sulphide to be detected in 15 minutes.

It is noted that hydrogen sulphide can also be detected, normally at a 0.1 ppm resolution, in real-time, using electrochemical detection monitors.

7.0
Discussion and recommendation

WorkSafe does not consider that its current WES-TWA of 10 ppm and WES-STEL of 15 ppm for hydrogen sulphide are acceptable.

DECOS concluded in their 2006 review, based largely on animal data, that an 8-hour TWA HBROEL of 2.3 mg/m^3 (~1.7 ppm) was appropriate. This was based on applying an 'extrapolation factor' of 6 to an NOAEL of 14 mg/m^3 (10 ppm). DECOS did not propose a STEL value.

In 2007, SCOEL proposed an 8-hour TWA of 5 ppm, by applying an 'uncertainty' factor of 2 to a NOAEL of 10 ppm. They also proposed a STEL of 10 ppm to guard against acute toxic effects such as eye irritation, unconsciousness and persistent neurological disorders.

In their more recent 2010 review, ACGIH® recommended a TLV-TWA of 1 ppm, to protect against all the unwanted effects of hydrogen sulphide, and taking into account a NOAEL of 2 ppm and a LOAEL of 5 ppm. They also adopted a TLV-STEL of 5 ppm to protect against minor irritation and brief changes in oxygen uptake not expected to produce serious effects.

Based on the dates of publication of the reviews discussed above, and the most conservative NOAEL, WorkSafe considers the ACGIH® TLV® values to be the most appropriate for minimising the risk of adverse health effects arising from exposure to hydrogen sulphide.

As such, WorkSafe proposes lowering the WES-TWA for hydrogen sulphide to 1 ppm to minimise the potential for metabolic changes and anxiety. WorkSafe further proposes lowering the WES-STEL for hydrogen sulphide to 5 ppm to protect against minor irritative effects and brief changes in oxygen uptake.

Appendices

IN THIS SECTION:

Appendix 1: Glossary

TERM	MEANING
ACGIH®	The American Conference of Governmental Industrial Hygienists (ACGIH®) is a 501(c)(3) charitable scientific organization, established in 1938, that advances occupational and environmental health. Examples of this include their annual edition of the TLVs® and BEIs® book and Guide to Occupational Exposure Values.
AGS	German Committee on Hazardous Substances (Ausschuss für Gefahrstoffe).
ATSDR	Agency for Toxic Substances and Disease Registry.
DECOS	Dutch Expert Committee on Occupational Standards.
DFG	German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area (Deutsche Forschungsgemeinschaft).
EPA	The New Zealand Environmental Protection Authority.
HBROEL	Health Based Occupational Exposure Limit (a term used in DECOS documents).
HSNO	Hazardous Substances and New Organisms Act, 1996.
LEL	Lower explosive limit.
LOAEL	Lowest Observed Adverse Effect Level.
mg/m^3	Milligrams (or thousandths of a gram) of substance per cubic metre of air.
NIOSH	The National Institute for Occupational Safety and Health (NIOSH) is the United States federal agency responsible for conducting research and making recommendations for the prevention of work-related injury and illness. NIOSH is part of the Centers for Disease Control and Prevention (CDC) within the U.S. Department of Health and Human Services.
NOAEL	No Observed Adverse Effect Level.
OEL	Occupational Exposure Limit.
OSHA	The US Occupational Safety and Health Administration.
ppm	Parts of vapour or gas per million parts of air.
RER	Respiratory Exchange Ratio: is the ratio between the amount of carbon dioxide (CO_2) produced in metabolism and oxygen (O_2) used.
SCOEL	The Scientific Committee on Occupational Exposure Limits is a committee of the European Commission, established in 1995 to advise on occupational health limits for chemicals in the workplace within the framework of Directive 98/24/EC, the chemical agents directive, and Directive 90/394/EEC, the carcinogens at work directive.
SEN	A notation indicating the subject substance is a sensitiser. DSEN and RSEN are used in place of SEN when specific evidence of sensitisation by the dermal or respiratory route, respectively, is confirmed by human or animal data. An ACGIH® term.
Skin	A notation indicating the potential for significant contribution to the overall exposure, by the cutaneous route, including mucous membranes and the eyes, by contact with vapours, liquids and solids. An ACGIH® term.
TLV®	Threshold Limit Value (see TLV-STEL and TLV-TWA below). An ACGIH® term.
TLV-STEL	TLV®-Short-Term Exposure Limit; a 15 minute TWA exposure that should not be exceeded at any time during a work day, even if the 8-hour TWA is within the TLV®-TWA. An ACGIH® term.
TLV-TWA	TLV® – Time-Weighted Average; the TWA concentration for a conventional 8-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed to, day after day, for a working lifetime without adverse effect. An ACGIH® term.
UEL	Upper explosive limit.

TERM	MEANING
VCO_2	The volume of carbon dioxide exhaled per unit of time.
VO_2	The volume of oxygen utilised per unit of time.
WES	Workplace Exposure Standard – WESs are values that refer to the airborne concentration of substances, at which it is believed that nearly all workers can be repeatedly exposed to, day after day, without coming to harm. The values are normally calculated on work schedules of five shifts of eight hours duration over a 40 hour week. See also WES-TWA and WES-STEL, below. A New Zealand term.
WES-STEL	The 15-minute time weighted average exposure standard. Applies to any 15-minute period in the working day and is designed to protect the worker against adverse effects of irritation, chronic or irreversible tissue change, or narcosis that may increase the likelihood of accidents. The WES-STEL is not an alternative to the WES-TWA; both the short-term and time-weighted average exposures apply. Exposures at concentrations between the WES-TWA and the WES-STEL should be less than 15 minutes, should occur no more than four times per day, and there should be at least 60 minutes between successive exposures in this range.
WES-TWA	The average airborne concentration of a substance calculated over an eight-hour working day. A New Zealand term.

Appendix 2: References

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