

**Recommendation from the Scientific Committee on
Occupational Exposure Limits
for Hydrogen Sulphide**

8 hour TWA:	5 ppm (7 mg/m ³)
STEL (15 mins):	10 ppm (14 mg/m ³)
Further notation:	none

Substance identity and properties

IUPAC name:	Hydrogen sulphide
Common name:	Hydrogen sulphide
CAS number:	7783-06-4
Synonyms:	Dihydrogen monosulphide ; dihydrogen sulphide, hydrogen sulphuric acid ; sewer gas ; stink damp ; sulphuretted hydrogen ; sulphur hydrogen
EINECS	231-977-3
EC number	016-001-00-4
Molecular formula :	H ₂ S
Molecular weight:	34.09
Freezing point at 101.3 kPa:	-85.5 °C
Boiling point at 101.3 kPa:	-60.7 °C
Vapour density (air=1):	1.19
Vapour pressure at 25.5 C:	2026 kPa
Explosive limits in air (vol/vol):	Lower limit: 4.3% Upper limit: 45.5%
Solubility (w/w) at 20 C:	0.4% in water 2.1% in ether
Odour threshold:	0.13 ppm
Conversion factors at 25 C:	1 ppm = 1.394 mg/m ³ 1 mg/m ³ = 0.717 ppm
EU classification:	F+; R12 Extremely flammable T+; R26 Very toxic by inhalation. N; R50 Very toxic to aquatic organisms

Hydrogen sulphide (H₂S) is a colourless gas with a strong odour of 'rotten eggs' (odour threshold 0.18 mg/m³, 0.13 ppm). The substance is flammable and explosive in air and may even be ignited by static discharge.

Occurrence/use

Hydrogen sulphide is one of the principal compounds involved in the natural cycle of sulphur in the environment. The substance is often present in volcanic gases. It is also produced by bacterial processes during the decay of both plants and animal protein or through the direct reduction of sulphate.

Occupational exposure to hydrogen sulphide is primary 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 H₂S are used in the production of deuterated water. Examples of industries where H₂S can be generated include petroleum refineries, natural gas plants, petrochemical plants, coke over plants, kraft paper mills, viscose rayon manufacture, sulphur production, iron smelters, food processing plants and tanneries.

Health significance

Toxicokinetics (Beauchamp 1984)

Hydrogen sulphide is primarily absorbed via the respiratory tract. It enters the circulation and partly dissociates to HS⁻. It is distributed to the brain, liver, kidney, pancreas and small intestine. The net uptake of sulphide is the greatest in the brainstem. The primary metabolic fate of hydrogen sulphide is oxidation to (conjugated) sulphate and excretion via urine. Methylation is another detoxification pathway.

The main detoxification pathway of H₂S is oxidation to thiosulphate. Caecal and proximal colonic mucosa metabolized H₂S to thiosulphate (and sulphate) about 10 times more rapidly than the upper part of the gastro intestinal tract mucosa and 5 times more rapid than liver tissue (Furne, 2001).

Mechanism of toxicity (Beaucamp 1984)

Toxicity of H₂S is most likely related to inhibition of metal-containing enzymes. Important target enzymes are cytochrome oxidase, the final enzyme of the mitochondrial respiratory chain, and carbonic anhydrase. By this mechanism H₂S affects cellular energy production and respiration. Most susceptible tissues are mucous membranes and tissues with a high oxygen demand, like nervous and cardiac tissues. In addition, sulphide also seems to act on the respiratory drive through other mechanisms. Among these are inhibition of monoamine oxidase, suppression of synaptic activity, a direct action on respiratory centres in the brain and stimulation of the glutamate receptors in the brain.

Acute or short term toxicity

Human data

In humans the targets of acute toxicity of hydrogen sulphide are the nervous system and the lung (ACGIH 1991, Beauchamp 1984, Arnold 1985, Guidotti 1996, Hessel 1997, Mehlman 1994). (Temporary) unconsciousness and (severe) effects on the respiratory system (with or without neurological changes) are the main symptoms (Aalst, 2000). Pulmonary oedema is also relatively often seen in patients after H₂S

exposure (Schneider 1998, Tvedt 1991, Vuorela 1987, Wasch 1989). At low exposure concentrations, the characteristic odour of “rotten eggs” can be an early warning for exposure (odour threshold 0.13 ppm, 0.18 mg/m³) (Beliles 1993, Deng 1992). At concentrations above 100 ppm (140 mg/m³) humans are not able to smell H₂S most probably due to olfactory fatigue (Glass 1990, Reiffenstein 1992). Several case reports are available that describe persistent neurological and neuropsychological abnormalities following acute hydrogen sulphide exposure (exposure levels not known) (Wasch 1989, Callender 1993, Snyder 1995, Schneider 1998, Kilburn 1993, Vuorela 1987, Tvedt 1991, Chaturvedi 2001, Kage 2002, Nelson 2002). However, complete recovery of victims of H₂S poisoning is also possible (Deng 1992, Glass 1990, Guidotti 1994))

Short-term exposure of humans (workers, duration not reported) led to lung function impairment (Richardson, 1995) and changes in neurobehavioral functions at unknown exposure levels (De Fruyt 1998, Hessel 1997). At 1.4-16 mg/m³ (exposure during 30 minutes), no significant changes in respiratory function and bronchial responsiveness were found in healthy paper mill workers (pre-exposed daily to 14 mg/m³ H₂S). However, asthmatic subjects showed a (non- significant) increased airway resistance after exposure at 2.8 mg/m³ for 30 minutes (Jäppinen 1990).

16 healthy male volunteers were randomly exposed to 0, 0.5, 2.0, 5.0 ppm (> 16 min) on four separate occasions. The results of this study indicated that healthy young males were able to safely exercise at their maximum metabolic rates while orally breathing 5.0 ppm (7 mg/cm³) H₂S. (Bhambhani 1991). The primary physiological response under these conditions was an increased lactate accumulation during submaximal and maximal exercise. This increase in blood lactate concentration, however, did not significantly reduce the maximum physiological work capacity of the volunteers during short-term incremental exercise. Exposure to 10 ppm (14 mg/m³) for 15 minutes during submaximal exercise revealed no significant changes in routine pulmonary function parameters (Bhambhani et al, 1996). However, a significant decrease in oxygen uptake, with a concomitant increase in blood lactate was observed in healthy men and women exposed to 10 ppm H₂S for 30 minutes during submaximal exercise at 50% of VO₂max. No significant changes were observed in arterial blood parameters and the cardiovascular responses under these conditions. Muscle lactate, as well as the activities of lactate dehydrogenase, citrate synthetase, and cytochrome oxidase, were not significantly altered by H₂S exposure. (Bhambhani et al, 1997). However, a major limitation of these studies was that the volunteers inhaled the gas through the mouth from a bag (mouth only exposure). Therefore, the possible effects on eyes found in other studies, could not be detected.

Table 1. Dose-effect relationships in man after short term exposure

Effect level mg/m ³ (ppm)	NOEL mg/m ³ (ppm)	Effects	Ref.
0.028 (0.02)		Minimum perception threshold	Beliles 1993
0.07 – 7.3 (0.05-5.2)		Changes in haem synthesis in pulp production workers	Tenhunen 1983
0.18 (0.13)		Generally accepted smell threshold	Deng 1992
2.8 (2)		Non significant effects in asthmatic subjects (exposure for 30 min)	Jäppinen 1990a
4.2-7 (3-5)		Offensive smell	Beliles 1993
7 (5)	2.8 (2)	Increased muscle lactate levels during exercise (exposure > 16 min) and increased oxygen uptake	Bhambhani 1991
14 (10)		Exposure for 15 minutes did not alter the pulmonary function significantly.	Bhambhani 1996
14 (10)		Reduced oxygen uptake during exercise (exposure two times 30 minutes)	Bhambhani 1997
> 140 (>100)		No smell due to olfactory fatigue	Glass 1990, OSHA 2000
700-1400 (500-1000)		Stimulation of carotid bodies	ACGIH 1991
1400-2800 (1000-2000)		Paralysis of respiratory center and breathing stops	ACGIH 1991

Animal data

Inhalation exposure of rats during 4 hours to hydrogen sulphide gave a LC₅₀ of 444-501 ppm (622-701 mg/m³). Acute effects included oedema in the lungs (Prior, 1988). Sublethal concentrations produced cytotoxic lesions in the lungs with depression of the activity of cytochrome oxidase (Warenycia 1989). Amino neurotransmitter levels in the respiratory centers in the brainstem were increased (Kombian 1988).

In several studies, rodents were exposed to H₂S at 25-100 ppm (35-140 mg/m³). The observed effects include inhibition of cerebral cytochrome oxidase activity (Savolainen 1980,1982), increased L-glutamate levels in the hippocampus and concomitant changes in the EEG (Nicholson 1998, Skrajny 1992), various cardiac arrhythmias (Kosmider 1967) and effects on blood parameters (increased number of reticulocytes) (Savolainen 1982).

Exposure of rabbits for 6 days (10 hours/day) to H₂S alone (50-100 mg/m³) did not produce corneal lesions (Masure 1950).

Male rats (CD) were exposed to target concentrations of 0, 30, 80, 200 or 400 ppm H₂S ((0, 42, 112, 280 and 560 mg/m³, 3 hours/day) for 1 or 5 consecutive days (Brenneman 2002). After a single exposure, bilaterally symmetrically mucosal necrosis in the olfactory epithelium lining the dorsal medial meatus was found in one rat at 80 ppm, in 3 rats at 200 ppm and in 4 rats at 400 ppm (Brenneman 2002). Regenerating respiratory epithelium was found in 1 rat at 80 ppm and all rats at 200 and 400 ppm. Electron microscopy revealed severe swelling of the mitochondria in both sustentacular cells and olfactory neurons. In the sustentacular cells, endoplasmatic reticulum was

extensively swollen. Dendrites and olfactory vesicles of the olfactory neurons were swollen with reduced numbers of cilia compared to controls.

Male CD rats (6/treatment) were exposed once to 0, 10, 30, 80, 200 and 400 ppm H₂S ((0, 14, 42, 112, 280 and 560 mg/m³) for 3 hours. At the end of exposure, cytochrome oxidase activity in the lung showed a dose-related decrease (significant at 30 ppm and above). In the liver, cytochrome oxidase activity was increased significantly in all dose groups without a relationship with dose (Dorman 2002).

Male CD rats were exposed to 0, 30, 80, 200 and 400 ppm H₂S for 3 hours during 1 day or 5 consecutive days. Cytochrome oxidase activity was decreased significantly at all tested concentrations in both respiratory and olfactory epithelium after a single exposure and in the olfactory epithelium after 5 exposure days (no concentration-related effects) (Dorman 2002).

An overview of the studies available can be found in table 2.

Table 2. Dose-effect and dose-response data for animals exposed (single and short-term) to hydrogen sulphide.

Effect level mg/m³ (ppm)	NOEL mg/m³ ppm)	Duration of exposure	Effects	Reference
35 (25)		Repeated, 3 h/day	Cumulative change in hippocampal type 1 EEG activity in rat	Skrajny 1992
42 (30)	14 (10)	Once for 3 hours	Cytochrome oxidase inhibition in the lung	Dorman 2002
≥ 70 (≥50)	14 (10)	4 h	Inhibition of cytochrome oxidase in rat lung cells	Khan 1990
100 (72)		1.5 h/day several days	Various cardiac arrhythmias including ventricular extrasystoles in rabbits and guinea pigs	Kosmider 1967
140 (100)		2 h, 4-day intervals, 4 times	Increasing inhibition of cerebral cytochrome oxidase activity and decreased protein synthesis in mouse brain	Savolainen 1980,1982
140 (100)		3 h/day, 5 days	Increased level of L-glutamate in hippocampus of rats	Nicholson 1998
280 (200)		4 h	Detectable histologic lesions in nasal epithelium of rats	Lopez 1988A
280 (200)		4 h	Increase in protein and lactate dehydrogenase in lavage fluids from rat lung	Green 1991
280-560 (200-400)	70 (50)	4 h	Particle-induced oxygen consumption reduced in pulmonary alveolar macrophages from rats	Khan 1991
420 (300)		4 h	Marked abnormality in surfactant activity in lavage fluids from rat lungs	Green 1991
459 (335)		6 h	LC ₅₀ and pulmonary oedema in rats	Prior 1988
560 (400)		4 h	Transient increase in protein concentration and activity of lactate dehydrogenase in nasal lavage fluids or rats	Lopez 1987
615 (439)		4 h	Transient necrosis and exfoliation of nasal respiratory and olfactory mucosal cells in rat. Reversible pulmonary oedema	Lopez 1988B
622 (444)		4 h	LC ₅₀ for rats	Tansy 1981
701 (501)		4 h	LC ₅₀ and pulmonary oedema in rats	Prior 1988
> 700 (>500)		4 h	Lethal for rats	Khan 1990
822 (587)		2 h	LC ₅₀ and pulmonary oedema in rats	Prior 1988
2317 (1655)		5 min	Pulmonary oedema and death in rats	Lopez 1989

Irritation and sensitisation

Human data

In viscose rayon workers, eye irritation ('spinners eye') has been reported to occur after 6-7 hours of exposure to 10 ppm (14 mg/m³) H₂S (Nesswetha 1969). Prolonged exposure led to irritation and keratoconjunctivitis in workers in 'sour gas' plants (ACGIH 1991, Beauchamp 1984, Deng 1992, Reiffenstein 1992). In another study concerning workers in the rayon viscose industry, increased prevalence of eye irritation was seen after prolonged exposure to 0.7-4 ppm (1-5 mg/m³) (Vanhoorne 1995). However, all these H₂S -exposed workers were co-exposed to CS₂ (concentration at least 26 mg/m³) and a combined effect cannot be excluded. Irritant effects of H₂S as a single agent at exposure levels below 20 ppm (28 mg/m³) are not well documented.

Olfactory fatigue is reported at high concentrations of H₂S (>140 mg/m³) and/or after prolonged exposure (Glass 1990, Reiffenstein 1992). One person has been described who lost his smell for 3 years after exposure to a high (not specified) concentration of H₂S (Tvedt 1991).

No information on skin irritation and sensitisation is available.

Animal data

Hydrogen sulphide leads to irritation of the eyes in laboratory animals (few hours exposure to 100-300 ppm, 139-417 mg/m³). Moreover, effects on the mucous membranes of the throat and nasal cavity are reported in laboratory animals (IPCS 1981, Lopez 1988A).

No information on skin irritation and sensitisation is available.

Repeated dose toxicity

Human data

Epidemiological studies of workers who have been exposed to H₂S are difficult to interpret because of the combined exposure to other toxic agents. After prolonged exposure, eye-irritation, hazy sight and photophobia (at concentrations from 1-5 mg/m³) (Vanhoorne 1995, Masure 1950, Legator 2001), lung function impairment (no concentrations indicated) (Richardson 1995, Melbostad 1994, Buick 2000), effects on enzyme levels in reticulocytes and erythrocyte protoporphyrin concentration (at concentrations between 0.07 and 7.2 mg/m³ as 8-hour TWA) (Tenhunen 1983). These effects were seen in single studies and were confirmed in other studies as the main effects related to H₂S exposure. Furthermore, an excess mortality from cardiovascular

^a CS₂ causes adverse effects in the eye (retinal microaneurysms and haemorrhages) in US-workers exposed to 3-48 mg/m³ CS₂. In Chinese workers no such effects were observed. In addition, Japanese workers exposed to CS₂ (60-95 mg/m³) showed an increased incidence of retinopathy, but these effects were not observed in Finnish workers exposed to CS₂ under similar occupational conditions (Health Council of the Netherlands, 1994).

disease and coronary heart disease was reported (Jäppinen 1990b) in a Finnish pulp mill (no exposure data available). Exposure measurements of H₂S (and mercaptans and sulfur dioxide) in this pulp mill were performed 20-40 years later by Kangas et al (1984) and showed a level of 0-28 mg/m³ H₂S.

An overview of the dose-effect relationship of hydrogen sulphide after prolonged exposure can be found in table 3.

Table 3. Dose-effect relationships in man after prolonged exposure.

Effect level mg/m³ (ppm)	NOEL mg/m³ (ppm)	Effects	Ref.
1-8.9 (0.7-6.4)		Increased prevalence of eye irritation symptoms in viscose rayon workers (co-exposure) to CS ₂ (4-112 mg/m ³)	Vanhoorne 1995
28 (20)		Effects on the cornea and conjunctiva	Masure 1950
>70 (>50)		Effects on the epithelia of the conjunctiva and the cornea of the eye	Ammann 1986
350-740 (250-600)		Pulmonary oedema after prolonged exposure	ACGIH 1991

Animal data

Brenneman et al (2000) exposed rats to H₂S, 6 hours/day, 5 days/week for 10 weeks. Dose related lesions of the olfactory mucosa were found after exposure to 30 and 80 ppm. No effects were observed after exposure to 10 ppm H₂S.

Repeated exposure resulted in 100% incidence of olfactory lesions (located at the dorsal meatus and the ethmoid recess) at 80 ppm and above (Brenneman 2002). No lesions of the respiratory epithelium were observed. After 2 weeks the olfactory epithelium was partly regenerated and after 6 weeks complete recovery was observed.

In the olfactory epithelium of control rats only a limited number of cells responded to cytochrome oxidase immunostaining. According to the author, a low level of cytochrome oxidase may explain the lack of reserve against cytochrome oxidase toxicity due to H₂S in the olfactory epithelium in contrast to the respiratory epithelium (Brenneman 2002).

Adult male CD rats were exposed to H₂S at 0, 10, 30 and 80 ppm (0, 14, 42 and 112 mg/m³, 6 hours/day) for 70 consecutive days. Bilaterally, symmetrical olfactory neuronal loss and basal cell hyperplasia were observed in the mucosa lining the dorsal medial meatus, the nasal septum, dorsal wall of the nasal cavity and margins of the ethmoturbinates. These findings increased with concentration (50% effect at 30 ppm and 70% effect at 80 ppm). No effects were found after exposure to 10 ppm. Comparison with modelled H₂S fluxes showed a correlation between flux and lesion incidence (Moulin 2002).

In male CD rats used in a reproduction and developmental study (70 days exposure to 0, 10, 30, and 80 ppm H₂S; 0, 14, 42 and 112 mg/m³ during 6 hours/day) cytochrome

oxidase activity was significantly decreased in lungs of animals treated at 80 ppm, but not at the lower concentrations tested (Dorman 2002). Reduction of cytochrome oxidase activity is a very sensitive biomarker for H₂S exposure. An effect is seen in the lung and nose after exposure to 30 ppm.

In 2004, Dorman et al described the results of a re-assessment of the nasal and lung histologic specimens obtained from a subchronic CIIT inhalation study (Morgan et al 1983). Rats (Fischer-344 and Sprague Dawley) and mice (B6C3F1) were exposed to 0, 10, 30 or 80 ppm H₂S (whole body) for 6 hours/day, for at least 90 days. Exposure to 80 ppm was associated with reduced feed consumption during the first exposure week (rats) or throughout the 90 day exposure (mice). Rats (male Fischer and female Sprague Dawley) and female B6C3F1 mice exposed to 80 ppm had depressed terminal body weights when compared to controls. Inhalatory exposure did not result in toxicological relevant alterations in haematological indices, serum chemistries or gross pathology. Histological evaluation of the nose showed an exposure related increased incidence of olfactory neuronal loss after exposure to 30 or 80 ppm (except for male Sprague Dawley rats which showed effects after exposure to 80 ppm). In addition, rhinitis was observed in all mice exposed to 80 ppm. Finally, exposure to 30 ppm H₂S and higher was associated with bronchiolar epithelial hypertrophy and hyperplasia in male and female Sprague Dawley rats. Comparable effects were observed in male Fischer-344 rats exposed to 80 ppm.

An overview of the studies available can be found in table 4.

Table 4. Dose-effect and dose-response data for animals repeatedly exposed to hydrogen sulphide.

Effect level mg/m³ (ppm)	NOEL mg/m³ ppm)	Duration of exposure	Effects	Reference
1.4 (1)		8 h/day, 5 weeks	Some rats with hyperreactive response in the airways	Reiffenstein 1992
112 (80)	30 (42)	6 h/day for 70 days	Decreased cytochrome oxidase activity in the lung of CD rats	Dorman 2002
42 (30)	14 (10)	6 h/d for 90 days	Olfactory neuronal loss, bronchiolar epithelial hypertrophy and hyperplasia	Dorman 2004
42 (30)	14 (10)	6 h/d, 70 days	Olfactory neuronal loss and basal cell hyperplasia	Moulin 2002
42 and 112 (30 and 80)	14 (10)	6 h/day, 7 days/week 10 weeks	Dose related olfactory neuron loss and basal cell hyperplasia in rats	Brenneman 2000, 2002

Genotoxicity

No data are available

Carcinogenicity

There are no studies on the carcinogenic effect of H₂S alone. Concerning combined exposure in pulp and paper as well as in viscose rayon manufacture, there is no support for a carcinogenic effect of H₂S (IARC 1987, MacMahon 1988, Swaen 1994, Peplonska 1996, Zambon 1994).

Reproductive toxicity

Human data

In a retrospective epidemiological study in 106 non-smoking pregnant women working in a Chinese petrochemical company, an increased risk of spontaneous abortion was found (Odds Ratio 2.5, (95% 1.7-3.7) for exposure to unknown level of H₂S. Corrections were included for exposure to benzene, gasoline, MN and NH₃. In addition the influence of age, educational level, shift work, noise level, hours with standing and kneeling, hours at work, passive smoking and diet was included in the evaluation (Xu 1998). Other reports on the fertility and developmental effects are difficult to interpret, because of the co-exposure to CS₂, a known teratogen (Reiffenstein, 1992).

Animal data

Effects of H₂S on amino acid neurotransmitter levels in the developing rat brain (cerebellum) were reported at 20 and 75 ppm (28 and 102 mg/m³). Affected were the levels of aspartate, glutamate and GABA (gamma aminobutyric acid) (at 75 ppm), and serotonin and noradrenaline (at 20 ppm) (Hannah 1989). Neuropathological alterations of the Purkinje cells in rat offspring were found at 20 ppm (28 mg/m³) (Skrajny 1995).

Dorman et al (2000) examined whether perinatal exposure to H₂S had an adverse effect on pregnancy outcome, offspring prenatal and postnatal development or offspring behaviour. Male and female Sprague Dawley rats (12/sex/concentration) were exposed to H₂S (0, 10, 30, 80 ppm), 6 h/day, 7 days/week. The exposure of the female rats started two weeks prior to breeding and the females were additionally exposed during the 2-week mating period, and then from gestation day 0 to 19. Exposure of the dams and their pups resumed from postnatal day 5 and 18. Adult males were exposed 70 consecutive days starting two weeks before mating. The test protocol was, to the extent possible, similar to the OECD screening test for reproductive and developmental toxicity (OECD guideline 421).

A statistically significant decrease in feed consumption was observed in F₀ male rats from the 80 ppm exposure group during the first week of exposure. There were no effects on the reproductive performance (number of females with live pups, litter size, average length of gestation, and the average number of implants per pregnant female). Exposure to H₂S did not affect pup growth, development, performance of any of the behavioural tests.

Studies are summarised in table 5.

Table 5 Summary of dose-effect data of hydrogen sulphide from reproductive and developmental studies in rats.

Exposure mg/m ³ (ppm)	Duration of exposure	Effects	Ref.
28 (20)	7 h/day during pregnancy until 21 days postnatal	Severe alterations in the architecture and growth characteristics of the purkinje cell dendritic fields of the rat offspring	Hannah 1991
28 and 98 (20 and 70)	7h/day during pregnancy until 21 days postnatal	Altered levels of serotonin (5-HT) and norepinephrine in the developing rat cerebellum and frontal cortex	Skrajny, 1992
105 (75)	7h/day during pregnancy until 21 days postnatal	Decreased level of aspartate, glutamate and GABA in the cerebrum and aspartate and GABA in cerebellum of the rat offspring.	Hannah, 1989
112 (80)	6 h/day, 7 days/week for 2 weeks prior to breeding and through the whole pregnancy	No effect on pup growth, development or performance on any of the behavioural tests on the offspring	Dorman 2000

Recommendation

There is limited information concerning the effects of hydrogen sulphide (H₂S) after acute exposure. Only a few cases have been described in which acute exposure (to concentrations higher than 1400 mg/m³) caused breathing stops. Mouth only exposure for 15 minutes (to 14 mg/m³) did not cause significant changes in pulmonary functions.

In experimental animals, acute or short-term exposure to H₂S, 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.

There is limited human information concerning the health effects after prolonged exposure to H₂S as well. Exposure to 1-5.6 mg/m³ H₂S 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 H₂S alone below levels of 28 mg/m³. One epidemiological study found effects on reproduction (increased spontaneous abortion) in women exposed to petrochemicals, including H₂S. However, these (limited) data are difficult to interpret due to the simultaneous exposure to CS₂, a known teratogen.

In rats, subchronic exposure to H₂S (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/m³. 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 H₂S of 42 mg/m³ and higher, with a NOAEL of 14 mg/m³ as well (Khan et al, 1990, Dorman 2002).

No data are available concerning the carcinogenic effects of H₂S.

No effects on reproduction and development were reported in rats exposed to H₂S (14, 42 and 112 mg/m³) 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.

The nasal lesions found in rats after exposure to H₂S are considered the critical effect. The NOAEL of 14 mg/m³ (10 ppm) found in the studies of Dorman (2004), Brenneman (2000, 2002) and Moulin (2002) is taken as a starting point for establishment of the OEL. An uncertainty factor to compensate for the differences between rats and humans is considered 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 10ppm (14 mg/m³) and using an uncertainty factor of 2, SCOEL proposes an 8-h TWA of 5ppm (7 mg/m³) for H₂S.

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.

Measurement difficulties are not foreseen at the proposed limit.

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