Chapter 6.6 Hydrogen sulfide

General Description

Sources

Hydrogen sulfide (H_2S) is a colourless gas, soluble in various liquids including water and alcohol. It can be formed under conditions of deficient oxygen, in the presence of organic material and sulfate. Most of the atmospheric hydrogen sulfide has natural origins. Hydrogen sulfide occurs around sulfur springs and lakes, and is an air contaminant in geothermally active areas. Saline marshes can also produce sulfide *(1).* The estimated global release of hydrogen sulfide from saline marshes into the atmosphere is 8.3×10^5 tonnes per year.

Human activities can release naturally occurring hydrogen sulfide into ambient air. For instance, some natural gas deposits contain up to 42% hydrogen sulfide *(2).* In industry, hydrogen sulfide can be formed whenever elemental sulfur or sulfur-containing compounds come into contact with organic materials at high temperatures. Hydrogen sulfide is formed, for instance, during coke production, in viscose rayon production, in waste-water treatment plants, in wood pulp production using the sulfate method, in sulfur extraction processes, in oil refining and in the tanning industry. In Canada, in 1978, the kraft pulping industry was estimated to be responsible for 97% of the country's total anthropogenic hydrogen sulfide emissions *(3).* However, only 10% of the total global emissions of this compound are of anthropogenic origin.

Occurrence in air

In one report *(2),* the average ambient air hydrogen sulfide level was estimated to be 0.3 $\mu g/m^3$ (0.0002 ppm). In north-west London, over a period of 2.5 years, air levels of hydrogen sulfide were generally below 0.15 $\mu g/m^3$ (0.0001 ppm) under clear conditions *(2).* In and around the city of Rotorua, New Zealand, where there is geothermal activity, there is usually a sufficient hydrogen sulfide concentration to cause odours. During continuous monitoring in Rotorua a hydrogen sulfide concentration of 0.08 mg/m^3 (0.05 ppm) was exceeded more than 55% of the time in the mid-winter months *(2).* Rather high concentrations of hydrogen sulfide have been measured near point sources. Near a pulp and paper-mill in California, peak concentrations of up to 0.20 mg/m^3 (0.13 ppm) were measured *(2).* In a Finnish town with two sulfate pulp mills (annual emissions of 1993 tonnes and 794 tonnes of hydrogen sulfide, respectively) concentrations of hydrogen sulfide near the mills were estimated, using the emission data and a dispersion model for the spread of gaseous sulfur compounds *(4).* The average annual concentrations were calculated to be up to 55 $\mu g/m^3$, monthly average concentrations up to 100 $\mu g/m^3$, 24-hour concentrations up to 540 $\mu g/m^3$, and 1-hour concentrations up to 1600 $\mu g/m^3$.

In another Finnish town, hydrogen sulfide concentrations near a viscose rayon mill were partly measured and partly estimated by using a dispersion model *(5).* When the smokestack of the mill was only 55 m high, the average annual concentrations exceeded 10 $\mu g/m^3$, 24-hour concentrations were approximately 200 $\mu g/m^3$ and short-term concentrations were up to 450 $\mu g/m^3$. When a higher smokestack was installed the annual concentrations were reduced to 4 $\mu g/m^3$, 24-hour concentrations to 35 $\mu g/m^3$ and 1-hour concentrations to a maximum of 80 $\mu g/m^3$.

During accidental exposures, concentrations from 150 mg/m^3 (100 ppm) to 18 000 mg/m^3 (12 000 ppm) have been reported *(2).* In a Finnish study *(6),* a health survey for hydrogen

sulfide and other sulfides was carried out at six kraft mills. The hydrogen sulfide concentrations varied from less than 0.075 mg/m^3 (0.05 ppm) to 30 mg/m^3 (20 ppm), the highest concentrations being found near vacuum pumps. A Japanese study in 18 viscose rayon plants showed occupational exposure levels of hydrogen sulfide ranging from 0.45 to 11.7 mg/m^3 (0.3-7.8 ppm), with a mean of 4.5 mg/m^3 (3 ppm) *(7).*

Hydrogen sulfide is the main toxic substance involved in livestock rearing systems with liquid manure storage *(8).* It is also a hazard at waste treatment facilities.

Conversion factors

1 ppm = 1.5 mg/m^3
1 mg/m^3 = 0.670 ppm

Routes of Exposure

The respiratory system is the main route of human exposure to hydrogen sulfide both in workplaces and in the ambient air. A recent report *(9)* criticizes the earlier belief that hydrogen sulfide can enter the body via tympanic membrane defects in workplace concentrations. There is very limited data on the sulfide levels of drinking-water *(10).*

Kinetics and Metabolism

Absorption

There are no exact data about the absorption of hydrogen sulfide through the lungs, but the absorbed proportion is probably large. Hydrogen sulfide is dissociated at physiological pH values to a hydrogen sulfide anion, in which form it is most probably absorbed *(11).*

The rate of absorption in the gastrointestinal tract is not known. However, hydrogen sulfide poisoning can be produced by infusing soluble sulfide salts in the gastrointestinal tract *(11).* Penetration through the skin is insignificant *(2).*

Distribution

Sodium sulfide, when administered parenterally, concentrates in the liver, but small amounts can also be found in the kidneys and lungs *(11).* In five lethal cases of hydrogen sulfide poisoning, sulfide concentrations in blood were between 1.7 and 3.75 mg/litre *(11);* this was due to an acute lethal hydrogen sulfide concentration of at least 750-1400 mg/m^3. Because of the metabolic process of the sulfide anion after the intoxication, it is difficult to determine the air concentration of hydrogen sulfide on the basis of blood sulfide levels. Moreover, the sulfide concentrations in other organs have not been determined.

Biotransformation

The sulfide anion is transformed by the sulfide oxidase system in the liver and kidneys mainly into thiosulfate and sulfate *(11).* In the mucosa of the gastrointestinal tract a thiol-S-methyltransferase system can also detoxify hydrogen sulfide *(11).*

Elimination

When sodium sulfide was injected intraperitoneally into mice no metabolites were found in the exhaled air *(11).*

When sodium (35)S-sulfide was administered orally to rats, 50% of the dose was found as sulfate in the urine after 24 hours *(11).* When administered parenterally, 91% of the dose was found in urine (74%) and faeces (17%).

Health Effects

Effects on experimental animals

The main toxicological effect at the cellular level in the brain is the inhibition of the enzyme cytochrome oxidase at the end of the mitochondrial respiratory chain *(11).* Because of abnormal mitochondrial function, many secondary changes appear in cells which have a great energy demand.

In acute hydrogen sulfide intoxications brain oedema, degeneration and necrosis of the cerebral cortex and the basal ganglia have been reported in rhesus apes *(11).* A rhesus monkey developed brain damage after a nonlethal hydrogen sulfide intoxication *(11).* An exposure of hydrogen sulfide at a concentration of 150 mg/m^3 for 2 hours caused inhibition of the brain protein synthesis in mice in 48 hours; this was normalized in 72 hours *(11).* It has been reported that the cerebral biochemical effects caused by repeated subclinical hydrogen sulfide intoxications are cumulative in mice *(11).*

Information on the effects of low-level concentrations of hydrogen sulfide on experimental animals is limited. The following effects have been reported on the basis of animal experiments: an increase of the reticulocytes, an increase of the blood cell volume in rats, a diminished leucocyte count, an increased lymphocyte count, and an increased thymol turbidity in rabbits *(11).* Injection of hydrogen sulfide water solution (265 mg/litre) into the ear vein of the rabbit caused prolongation of the diastole and lowered the heart frequency. There were changes in the T-wave *(11).* A decrease of the heart frequency has been reported in other mammals that were given intravenous injections of 0.5-10 mg/litre of hydrogen sulfide *(11).*

Findings in the canary, cat, dog, goat, guinea pig, rabbit and rat *(2)* are consistent as to the effects of hydrogen sulfide: at 150-225 mg/m^3 signs of local irritation of eyes and throat after many hours of exposure; at 300-400 mg/m^3 eye and mucous membrane irritation in 1 hour and slight general effects with longer exposure; at 750-1000 mg/m^3 slight systemic symptoms in less than 1 hour and possible death after several hours; at 1350 mg/m^3 grave systemic effects within 30 minutes and death in less than 1 hour; at 2250 mg/m^3 collapse and death within 15-30 minutes, and at 2700 mg/m^3 immediate collapse, respiratory paralysis and death.

Injection of sodium nitrite, inducing methaemoglobinaemia, had protective and antidotal effects against hydrogen sulfide poisoning in mice, armadillos, rabbits, and dogs *(2).* There is very limited information on chronic hydrogen sulfide intoxication in experimental animals.

No information is available on the mutagenic, carcinogenic or teratogenic effects of hydrogen sulfide in experimental animals.

Effects on humans

In its acute form, hydrogen sulfide intoxication is mainly the result of action on the nervous system. At concentrations of 15 mg/m^3 and above, hydrogen sulfide causes conjunctival irritation, because sulfide and hydrogen sulfide anions are strong bases *(11).* Hydrogen sulfide affects the sensory nerves in the conjunctivae, so that pain is diminished rapidly and the tissue damage is greater *(11).* Serious eye damage is caused by a concentration of 70 mg/m^3. At higher concentrations (above 225 mg/m^3, or 150 ppm), hydrogen sulfide has a paralysing effect on the olfactory perception *(2),* so that the odour can no longer be recognized as a warning signal. At higher concentrations, respiratory irritation is the predominant symptom, and at a concentration of around 400 mg/m^3 there is a risk of pulmonary oedema. At even higher concentrations there is strong stimulation of the central nervous system (CNS) *(2),* with hyperpnoea leading to apnoea, convulsions, unconsciousness, and death. At concentrations of over 1400 mg/m^3 there is immediate

collapse. In fatal human intoxication cases, brain oedema, degeneration and necrosis of the cerebral cortex and the basal ganglia have been observed *(11)*.

If respiration can be maintained, the prognosis in a case of acute hydrogen sulfide intoxication, even a severe one, is fairly good. There are reports of neurasthenic symptoms after severe acute intoxication, such as amnesia, fatigue, dizziness, headache, irritability, and lack of initiative *(11)*. A decrease of delta-aminolaevulinic acid dehydrase (ALAD) synthase and haem synthase activity in reticulocytes one week after hydrogen sulfide intoxication has been reported *(12),* together with low levels of erythrocyte protoporphyrin. The ALAD and haem synthase activities returned to normal two months after the accident, erythrocyte protoporphyrin remaining low. Changes in the electrocardiogram have been reported after acute hydrogen sulfide intoxication, these changes being reversible *(11)*. No tolerance to the acute effects of hydrogen sulfide has been reported to develop *(11)*.

The mortality in acute hydrogen sulfide intoxications seems to be lower than that reported in 1977; according to a recent Canadian report it is now 2.8% *(13),* whereas formerly it was 6% *(2)*. This may be a result of improved first-aid procedures and increased awareness of the dangers of hydrogen sulfide.

Information about longer-term exposures to hydrogen sulfide is scanty. Eighty-one Finnish pulp mill workers who were exposed to hydrogen sulfide concentrations of less than 30 mg/m^3 (20 ppm) and to methyl mercaptan concentrations of less than 29.6 mg/m^3 (15 ppm), displayed loss of concentration capacity and chronic or recurrent headache more often than a nonexposed control group of 81 workers. Restlessness and lack of vigour also appeared more often, but the findings were not statistically significant. There was also a tendency towards more frequent sick leave among the exposed group *(6)*. One report cites decreased activity of haem synthesizing enzymes in reticulocytes of pulp mill workers exposed for years to organic and inorganic sulfides, with hydrogen sulfide concentrations of between 0.075 mg/m^3 and 7.8 mg/m^3 *(12)*. No information is available as to whether the observed effect was related to peak concentrations or average concentrations. It can, however, be assumed that average exposure was considerably higher than 0.075 mg/m^3 (around 1.5-3 mg/m^3). Furthermore, there is no firm proof that hydrogen sulfide was the causative agent, as there may be confounding factors (other substances).

Epidemiological data concerning longer-term exposures are limited. Seventy per cent of workers exposed to hydrogen sulfide daily, often at 30 mg/m^3 or more, complained of such symptoms as fatigue, somnolence, headache, irritability, poor memory, anxiety, dizziness, and eye irritation *(14)*. In a Finnish mortality study workers in a sulfate pulp mill showed excess mortality from cardiovascular diseases (standardized mortality rate 140), and especially from heart infarction (standardized mortality rate 142). The findings were statistically significant. In the same study population, cancer incidence was not significantly different from, that of the general Finnish population *(15)*.

Sensory effects

Hydrogen sulfide is an odorant, which in pure form has an odour detection threshold of 0.2-2.0 μg/m^3 depending on the purity *(16,17)*. Its characteristic smell of rotten eggs appears at concentrations 3-4 times higher than the odour threshold *(18)*. In practical situations, such as in the effluents of kraft pulp mills, hydrogen sulfide is accompanied by other odorous substances, such as methyl mercaptan, dimethyl disulfide and dimethyl monosulfide, and, in the case of effluents of the viscose industry, by carbon disulfide. The odour quality of these emissions changes with the specific composition of the mixtures *(19)*.

Hydrogen sulfide causes odour nuisance at concentrations far below those that cause health hazards. On the basis of the scientific literature, it is not possible to state a specific concentration of hydrogen sulfide at which odour nuisance starts to appear. Half-hour

average concentrations exceeding 7 μg/m^3 are likely to produce substantial complaints among persons exposed *(19,20).* A reduction in the concentration of hydrogen sulfide does not guarantee a substantial reduction of the odour nuisance, since hydrogen sulfide in many effluents provides only a small contribution to the odour strength of the total effluent *(21).* Moreover, the interaction between hydrogen sulfide and other odorous components in the effluent cannot explain the odour strength of the total effluents from pulp mills *(21).* Better short-term studies are required to elucidate the relationship between actual concentrations and reports of odour nuisance.

Evaluation of Human Health Risks

Exposure

Typical symptoms and signs of hydrogen sulfide intoxication are most often caused by relatively high concentrations in occupational exposures. There are many occupations where there is a potential risk of hydrogen sulfide intoxication and, according to the US National Institute for Occupational Safety and Health *(14),* in the United States alone approximately 125 000 employees are potentially exposed to hydrogen sulfide. Low-level concentrations can occur more or less continuously in certain industries, such as in viscose rayon and pulp production, at oil refineries and in geothermal energy installations.

In geothermal areas there is a risk of exposure to hydrogen sulfide for the general population *(2)*. The biodegradation of industrial wastes has been reported to cause ill effects in the general population *(2)*. An accidental release of hydrogen sulfide into the air surrounding industrial facilities can cause very severe effects, as at Poza Rica, Mexico, where 320 people were hospitalized and 22 died *(2)*. The occurrence of low-level concentrations of hydrogen sulfide around certain industrial installations is a well known fact (see above).

Dose-effect and dose-response relationship

The first noticeable effect of hydrogen sulfide at low concentrations is its unpleasant odour. Conjunctival irritation is the next subjective symptom and can cause so-called “gas eye” at hydrogen sulfide concentrations of 70-140 mg/m^3. Table 1 shows the established dose-effect relationships for hydrogen sulfide.

Table 1. Hydrogen sulfide: established dose-effect relationships

Hydrogen sulfide concentration		Effect	Reference
mg/m^3	ppm		
1400-2800	1000-2000	Immediate collapse with paralysis of respiration	*(2)*
750-1400	530-1000	Strong CNS stimulation, hyperpnoea followed by respiratory arrest	*(2)*
450-750	320-530	Pulmonary oedema with risk of death	*(2)*
210-350	150-250	Loss of olfactory sense	*(11)*
70-140	50-100	Serious eye damage	*(11)*
15-30	10-20	Threshold for eye irritation	*(11)*

Health risk evaluation

The hazards caused by high concentrations of hydrogen sulfide are relatively well known, but information on human exposure to very low concentrations is scanty. Workers exposed to hydrogen sulfide concentrations of less than 30 mg/m^3 are reported to have rather diffuse neurological and mental symptoms *(6)* and to show no statistically significant differences when compared with a control group. On the other hand, changes of haem synthesis have been reported at hydrogen sulfide concentrations of less than 7.8 mg/m^3 (1.5-3 mg/m^3 average) *(12)*. It is not known whether the inhibition is caused by the low concentration levels or by the cumulative effects of occasional peak concentrations. Most probably, at concentrations below 1.5 mg/m^3 (1 ppm), even in exposure for longer periods, there are very few detectable health hazards in the toxicological sense. The malodorous property of hydrogen sulfide is a source of annoyance for a large proportion of the general population at concentrations below 1.5 mg/m^3, but from the existing data it cannot be concluded whether any health effects result. The need for epidemiological studies on possible effects of long-term, low-level hydrogen sulfide exposure is obvious. A satisfactory biological exposure indicator is also needed.

Guidelines

The lowest-adverse-effect level of hydrogen sulfide is 15 mg/m^3, when eye irritation is caused. In view of the steep rise in the dose-effect curve implied by reports of serious eye damage at 70 mg/m^3, a relatively high protection (safety) factor of 100 is recommended, leading to a guideline value of 0.15 mg/m^3 with an averaging time of 24 hours. A single report of changes in haem synthesis at a hydrogen sulfide concentration of 1.5 mg/m^3 should be borne in mind.

In order to avoid substantial complaints about odour annoyance among the exposed population, hydrogen sulfide concentrations should not be allowed to exceed 7 μg/m^3, with a 30-minute averaging period.

When setting concentration limits in ambient air, it should be remembered that hydrogen sulfide is emitted from natural sources in many places.

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