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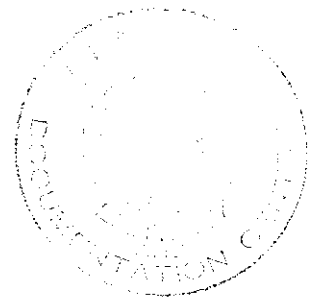
Environmental Health Criteria 19

HYDROGEN SULFIDE

Published under the joint sponsorship of
the United Nations Environment Programme,
the International Labour Organisation,
and the World Health Organization



World Health Organization
Geneva, 1981

ISBN 92 4 154079 6

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PRINTED IN FINLAND

81/4931 — VAMMALAN KIRJAPAINO OY, VAMMALA — 6500

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NOTE TO READERS OF THE CRITERIA DOCUMENTS

While every effort has been made to present information in the criteria documents as accurately as possible without unduly delaying their publication, mistakes might have occurred and are likely to occur in the future. In the interest of all users of the environmental health criteria documents, readers are kindly requested to communicate any errors found to the Division of Environmental Health, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda which will appear in subsequent volumes.

In addition, experts in any particular field dealt with in the criteria documents are kindly requested to make available to the WHO Secretariat any important published information that may have inadvertently been omitted and which may change the evaluation of health risks from exposure to the environmental agent under examination, so that the information may be considered in the event of updating and re-evaluation of the conclusions contained in the criteria documents.

A WHO Task Group on Environmental Health Criteria for Hydrogen Sulfide met in Geneva from 24 to 28 March 1980. Mr G. Ozolins, Associate Manager, Environmental Health Criteria and Standards, opened the meeting on behalf of the Director-General. The Task Group reviewed and revised the second draft of the criteria document and made an evaluation of the health risks from exposure to hydrogen sulfide.

The first and second drafts were prepared jointly by Dr T. H. Milby of the Environmental Health Associates, Inc., Berkeley, CA, USA, and Dr R. C Spear of the Department of Biomedical and Environmental Health Sciences, University of California, Berkeley, CA, USA. The comments on which the second draft was based were received from the national focal points for the WHO Environmental Health Criteria Programme in Australia, Belgium, Czechoslovakia, Finland, Federal Republic of Germany, Mexico, New Zealand, Poland, USA and USSR, and from the International Labour Organisation, Geneva, the International Centre for Industry and Environment, France, and the International Petroleum Industry Environmental Conservation Association, London. Comments were also received from Professor M. Katz (Canada) and Professor R. Lilis (USA). Some comments were received after the second draft had been prepared and were reviewed by the Task Group during its meeting. These comments were from the national focal points for the WHO Environmental Health Criteria Programme in Japan and the United Kingdom and from the Commission of the European Communities, Luxembourg, and the International Union of Pure and Applied Chemistry, London.

The collaboration of these national institutions, international organizations and individual experts is gratefully acknowledged. Without their assistance this document could not have been completed.

This document is based primarily on original publications listed in the reference section. However, several recent publications broadly reviewing health aspects of hydrogen sulfide have also been used, including those of the National Research Council, USA (1979) and NIOSH (1977).

Details of the WHO Environmental Health Criteria Programme, including some of the terms frequently used in the documents, can be found in the introduction to the publication "Environmental Health Criteria 1 — Mercury", published by the World Health Organization, Geneva, 1976, now also available as a reprint.

The following conversion factors have been used in this document: hydrogen sulfide: 1 ppm = 1.5 mg/m^3, 1 mg/m^3 = 0.7 ppm.

* *
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Financial support for the publication of this criteria document was kindly provided by the Department of Health and Human Services through a contract from the National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, USA — a WHO Collaborating Centre for Environmental Health Sciences.

1. SUMMARY AND RECOMMENDATIONS FOR FURTHER STUDIES

1.1 Summary

1.1.1 Properties and analytical methods

Hydrogen sulfide is a colourless gas with a characteristic odour that is soluble in various liquids including water, alcohol, ether, and solutions of amines, alkali carbonates, and bicarbonates. It can undergo a number of oxidation reactions to yield principal products consisting of sulfur dioxide, sulfuric acid, or elemental sulfur. Reaction rates and oxidation products depend on the nature of the oxidizing agent.

The methylene blue colorimetric method has acceptable specificity, accuracy, and sensitivity for hydrogen sulfide determinations, and is generally recognized as a standard analytical procedure. It has been used successfully, in automatic continuous monitoring, but sophisticated maintenance facilities and highly trained technicians are required for this method. Gas chromatography coupled with flame photometric detection is an alternative method for hydrogen sulfide determination, either as a laboratory method or for continuous monitoring in stationary field settings.

Most of the direct-reading methods of hydrogen sulfide determination in the occupational environment are susceptible to various forms of interference. However, methods employing chemical detector tubes appear to be useful in occupational settings, where hazardous levels of hydrogen sulfide can occur. Under these conditions, reliability and accuracy compensate for a certain lack of specificity.

1.1.2 Sources of hydrogen sulfide

Hydrogen sulfide is one of the principal compounds involved in the natural cycle of sulfur in the environment. It occurs in volcanic gases and is produced by bacterial action during the decay of both plant and animal protein. It can also be produced by bacteria through the direct reduction of sulfate. Significant concentrations of hydrogen sulfide occur in some natural gas fields and in geothermally active areas.

Hydrogen sulfide can be formed whenever elemental sulfur or certain sulfur-containing compounds come into contact with organic materials at high temperatures. In industry, it is usually produced as an undesirable by-product, though it is an important reagent or intermediate in some processes. Hydrogen sulfide occurs as a by-

product in: the production of coke from sulfur-containing coal, the refining of sulfur-containing crude oils, the production of carbon disulfide, the manufacture of viscose rayon, and in the Kraft process for producing wood pulp.

1.1.3 Environmental levels and exposures

Though concentrations of hydrogen sulfide in urban areas may occasionally be as high as 0.050 mg/m³ (0.033 ppm) with averaging times of 30 min—1 h, they are generally below 0.0015 mg/m³ (0.001 ppm). Peak concentrations as high as 0.20 mg/m³ (0.13 ppm) have been reported in the neighbourhood of point sources. In a geothermal area, 1-h mean concentrations of up to 2 mg/m³ (1.4 ppm) have been observed. When hydrogen sulfide was accidentally released in an incident in Poza Rica, Mexico, in 1950, the number of deaths that followed indicated that exposure levels probably exceeded 1500—3000 mg/m³ (1000—2000 ppm).

It is believed that workers are not usually exposed to hydrogen sulfide concentrations above the occupational exposure limits of 10—15 mg/m³ (7—10 ppm) (8-h time-weighted average) adopted by many governments. There are, however, numerous reports of accidental exposures to concentrations that have ranged from 150 mg/m³ (100 ppm) to as high as 18 000 mg/m³ (12 000 ppm). Such massive exposures to hydrogen sulfide have resulted either from leaks in industrial gas streams containing high levels of hydrogen sulfide or from the slow, insidious accumulation of hydrogen sulfide in low-lying areas. The second case may arise when hydrogen sulfide of biogenic origin is generated from such sources as sewage disposal plants and cesspools.

1.1.4 Effects on experimental animals

In experimental animals, the effects of high doses of hydrogen sulfide and high doses of cyanide are very similar. Cyanide inhibits the enzyme cytochrome *c* oxidase [EC 1.9.3.1] [a], thereby interfering with tissue use of oxygen to the point where metabolic demands cannot be met. Hydrogen sulfide also exhibited an inhibitory action on a purified preparation of cytochrome *c* oxidase.

Results of studies on a number of animal species including canary, rat, guineapig, cat, dog, and goat showed that inhalation of hydrogen sulfide at a concentration of 150—225 mg/m³ (100—150 ppm)

[a] The numbers within brackets following the names of enzymes are those assigned by the Enzyme Commission of the Joint IUPAC-IUB Commission on Biochemical Nomenclature.

resulted in signs of local irritation of eyes and throat after many hours of exposure; at 300–450 mg/m³ (200–300 ppm), eye and mucous membrane irritation appeared after 1 h inhalation and slight general effects after prolonged inhalation; at 750–1050 mg/m³ (500–700 ppm), local irritation and slight systemic signs appeared within 1 h and death after several hours; at 1350 mg/m³ (900 ppm), serious systemic effects appeared in less than 30 min and death within 1 h; at 2250 mg/m³ (1500 ppm), collapse and death occurred within 15–30 min; and, at 2700 mg/m³ (1800 ppm), there was immediate collapse with respiratory paralysis, and death. There is little information on the effects on experimental animals of long-term, low-level exposure to hydrogen sulfide gas.

1.1.5 Effects on man

1.1.5.1 *General toxicological considerations*

Hydrogen sulfide is both an irritant and an asphyxiant gas. Its direct irritant action on the moist tissues of the eye produces keratoconjunctivitis, known as "gas eye". When inhaled, hydrogen sulfide exerts an irritant action throughout the entire respiratory tract, although the deeper structures suffer the greatest damage. A consequence may be pulmonary oedema. At concentrations of 1500–3000 mg/m³ (1000–2000 ppm), hydrogen sulfide gas is rapidly absorbed through the lung into the blood, which initially induces hyperpnoea (rapid breathing). This is followed by respiratory inactivity (apnoea). At higher concentrations, hydrogen sulfide exerts an immediate paralysing effect on the respiratory centres. Death due to asphyxia is the certain outcome, unless spontaneous respiration is re-established or artificial respiration is promptly provided. This sequence of events represents the most important toxic effect of hydrogen sulfide.

Acute hydrogen sulfide intoxication can be defined as the effects from a single exposure to massive concentrations of hydrogen sulfide that rapidly produce signs of respiratory distress. Concentrations exceeding about 1500 mg/m³ (1000 ppm) produce such acute effects. Subacute hydrogen sulfide intoxication is the term applied to the effects of continuous exposure for up to several hours to concentrations ranging from 150 to 1500 mg/m³ (100–1000 ppm). In this range of exposure, eye irritation is the most commonly observed effect. However, some reports have indicated that the threshold for eye irritation occurs after several hours of exposure to hydrogen sulfide at levels of 16–32 mg/m³ (10.5–21.0 ppm). Pulmonary oedema may be a more important and potentially fatal complication of subacute hydrogen sulfide intoxication. Chronic intoxication is a largely subjective state characterized by fatigue and believed by some to be a

consequence of intermittent exposure to hydrogen sulfide concentrations of 75—150 mg/m³ (50—100 ppm). Not all research workers accept the existence of such a condition.

The characteristic "rotten egg" odour of hydrogen sulfide is well known. The threshold of perception of this odour varies considerably depending on individual sensitivity, but, under laboratory conditions, it ranges from 0.0008 to 0.20 mg/m³ (0.0005—0.13 ppm). Above about 225 mg/m³ (150 ppm), the gas exerts a paralysing effect on the olfactory apparatus, thus neutralizing the value of its odour as a warning signal. At these concentrations, the odour of the gas has been reported to be sickeningly sweet.

1.1.5.2 *Occupational exposure*

Exposure to hydrogen sulfide in high concentrations occurs in numerous occupations. Workers in the oil, gas, and petrochemical industries are occasionally exposed to hydrogen sulfide in concentrations sufficient to cause acute intoxication. In one survey of the petrochemical industry, among 221 cases of hydrogen sulfide poisoning, the overall mortality was 6% and a high proportion of victims exhibited neurological signs and symptoms. Forty percent of all cases required some form of respiratory assistance; 15% developed pulmonary oedema.

Persistent sequelae following acute intoxication have been reported among workers in a number of occupations including sewer workers, chemical plant employees, farmers, shale-oil workers, and laboratory attendants. Most victims who develop sequelae experience a state of unconsciousness during the acute phase of their illness. However, sequelae following acute intoxication without unconsciousness have also been reported.

1.1.5.3 *Exposure of the general population*

Several episodes of general population exposure to hydrogen sulfide have been reported. The effects of such exposure have ranged from minor nuisance to serious illness and death. In a small community adjacent to an oilfield installation, large quantities of hydrogen sulfide were released into the air when an oilfield flare malfunctioned. Three hundred and twenty persons were hospitalized, 22 of whom died. Nine exhibited manifestations of pulmonary oedema. Four victims developed neurological sequelae. The air levels of hydrogen sulfide were not measured.

A community of 40 000 people, located in the vicinity of a large geothermal field, was exposed in some areas to hydrogen sulfide levels in air (measured on a continuous basis over a 5-month period)

exceeding 0.08 mg/m³ (0.05 ppm), for, on average, 35% of the time. Although fatal cases of hydrogen sulfide intoxication associated with improper ventilation in geothermal steam-heated dwellings in this area were occasionally reported until 1962, the major problem has been the nuisance caused by the odour of the gas. In a moderately sized community, hydrogen sulfide was released from a small industrial waste lagoon resulting in a 1-h average concentration of hydrogen sulfide in air of 0.45 mg/m³ (0.3 ppm). Complaints were mostly related to the odour of hydrogen sulfide gas. However, the severity of complaints of nausea, vomiting, headache, loss of appetite, and disturbed sleep exceeded the mere nuisance level.

No community studies of the long-term, low-level effects of hydrogen sulfide exposure have been reported.

1.1.6 Evaluation of health risks

Hydrogen sulfide in ambient air in concentrations of the order of the odour threshold has not been shown to have any significant biological activity in man or animals. In controlled laboratory studies, the odour threshold for hydrogen sulfide has been reported to range from 0.0008 to 0.20 mg/m³ (0.0005—0.13 ppm). Little information is available on the odour detection limits for hydrogen sulfide either under experimental field conditions or in the ambient air. However, the Task Group considered that a level of 0.008 mg/m³ (0.005 ppm) averaged over 30 min should not produce odour nuisance in most situations. In the occupational setting, the earliest toxic response appears to be eye irritation, which has been reported to occur at 16—32 mg/m³ (10.5—21.0 ppm) after several hours' exposure. The occupational exposure guidelines for hydrogen sulfide recommended by the Task Group included the adoption of a level of 10 mg/m³ (7 ppm) as a workshift time-weighted average value together with a short-term exposure limit of 15 mg/m³ (10 ppm), the latter to be determined as a 10-min or less, average value.

1.2 Recommendations for Further Studies

Measurements of hydrogen sulfide concentrations in the ambient air should be included in studies of the levels in air of other common gaseous contaminants, such as the oxides of sulfur and nitrogen. Studies in areas remote from man-made emission sources would provide background data for the development of models for long-distance transport and diffusion, for the evaluation of biological decay processes from natural sources, and for developing a clearer

understanding of global sulfur cycles. More studies are required to elucidate processes involving chemical and photochemical oxidation reactions of hydrogen sulfide. Studies are also necessary to develop methods for the personal dosimetry measurement of hydrogen sulfide that do not require wet chemical techniques.

Studies should be conducted in experimental animals on the cumulative neural effects of repeated and/or continuous long-term hydrogen sulfide exposure at concentrations that induce subacute or chronic intoxication. The cardiac sequelae after acute intoxication should be investigated in intact animals and in those with pre-induced cardiac damage. Studies of the toxicokinetics of absorbed hydrogen sulfide are needed and other studies should be initiated to test for the metabolic generation of hydrogen sulfide from sulfur-containing organic compounds.

Case studies should be made of patients who have suffered acute hydrogen sulfide intoxication to examine the long-term effects on the myocardium. Efforts should be made to estimate the dose of hydrogen sulfide associated with acute poisoning. Prospective studies of new workers to investigate the effects of long-term exposure to concentrations of hydrogen sulfide likely to be encountered in the work place would be valuable. These studies should include considerations of morbidity and mortality, the incidence of cancer and teratogenic effects, and studies of changes in pulmonary function with time. Continuing environmental studies should play a major part in these prospective studies, in order to provide dose-response data, where possible. Similar studies should be initiated among the general population in a geothermal area, taking advantage of the natural conditions provided, for example, by the situation in Rotorua, New Zealand.

2. PROPERTIES AND ANALYTICAL METHODS

2.1 Chemical and Physical Properties

Hydrogen sulfide is a flammable colourless gas with the characteristic odour of rotten eggs. It burns in air with a pale blue flame and, when mixed with air, its explosive limits are 4.3% to 46% by volume. Its autoignition temperature is 260°C. The relative molecular mass of hydrogen sulfide is 34.08. Its density is 1.5392 g/litre at 0°C and 760 mm. The ratio density of hydrogen sulfide compared with air is 1.19. One gram of hydrogen sulfide dissolves in 187 ml of water at 10°C, in 242 ml of water at 20°C, in 314 ml of water at 30°C, and in 405 ml of water at 40°C (calculated from Weast, 1977–78). It is also soluble in alcohol, ether, glycerol, and in solutions of amines, alkali carbonates, bicarbonates and hydrosulfides. The vapour pressure of hydrogen sulfide is 18.75×10^5 Pa at 20°C and 23.9×10^5 Pa at 30°C. Its melting point is −85.5°C and its boiling point is −60.3°C (Macaluso, 1969; Windholz, 1976).

Hydrogen sulfide can undergo a large number of oxidation reactions, the type and rate of the reaction and the oxidation products depending on the nature and concentration of the oxidizing agent. The principal products of such reactions are sulfur dioxide, sulfuric acid, or elemental sulfur. Aqueous solutions of chlorine, bromine, and iodine may react with hydrogen sulfide to form elemental sulfur. In the presence of oxides of nitrogen, the oxidation of hydrogen sulfide in the gas phase may result in the formation of sulfur dioxide or sulfuric acid but, in aqueous solution (pH 5–9), the primary product is elemental sulfur (Macaluso, 1969).

Hydrogen sulfide dissociates in aqueous solution to form 2 dissociation states involving the hydrosulfide anion (HS^-) and the sulfide anion ($S^=$). The pKa in 0.01–0.1 mol/litre solutions at 18°C is 7.04 for HS^- and 11.96 for $S^=$. At the physiological pH of 7.4, about one-third of the total sulfide remains as the undissociated acid and about two-thirds as the HS^- ion. The undissociated hydrogen sulfide in solution is in dynamic equilibrium at the air-water interface with gaseous hydrogen sulfide (National Research Council, USA, 1977).

2.2 Atmospheric Chemistry

The atmospheric chemistry of hydrogen sulfide and other sulfur compounds involves chemical and photochemical oxidation reactions of emissions from both natural and man-made sources. The eventual oxidation products are sulfuric acid (H_2SO_4) and/or sulfate ion ($SO_4^=$).

There have been relatively few studies of the persistence and conversion of hydrogen sulfide under atmospheric conditions. Krasovitskaja and her co-workers (Krasovitskaja et al., 1965) studied the relationship between concentrations of hydrogen sulfide, sulfur dioxide, carbon monoxide, and hydrocarbons, and the distance from their industrial sources. Hydrogen sulfide concentrations dropped by a factor of 2 between the immediate neighbourhood of the source and a 2.5 km radius. A further decrease in concentration ranging from 30% up to a factor of 8 occurred between the 2.5 km and 20 km radii. These decreases were, in general, greater than those observed for any of the other pollutants measured. Andersson et al. (1974) reported studies concerning the photolysis of hydrogen sulfide and its reaction with sulfur dioxide, as well as its reactions with atomic and molecular oxygen and with ozone.

Junge (1963) calculated that the residence time of hydrogen sulfide was approximately 1.7 days in the presence of an ozone level of 0.05 mg/m³. A similar residence time was estimated by Katz (1977) using data from the global budget of the sulfur cycle presented by Kellogg et al. (1972). Robinson & Robbins (1970) found a residence time in relatively clean air of about 2 days, compared with only about 2 h in a polluted urban atmosphere.

Considerably lower values than those of the previously mentioned investigators, based on the global sulfur budget, have been presented by Granat et al. (1976), on the basis of a very much lower release of sulfur compounds from the biological dacay of organic matter from land and sea. Clearly, this represents a subject that requires further studies involving the measurement of atmospheric concentrations in relatively clean areas on land and elsewhere.

2.3 Sampling and Analytical Methods

Because levels of hydrogen sulfide in air, which are of interest as far as human health in concerned, range from highly concentrated industrial gas streams to ambient air pollution levels, numerous analytical methods have been applied. A recent review of current methods can be found in Becker (1979). A further complication in determining air levels of hydrogen sulfide is that according to the air monitoring application, sampling may be on either an intermittent or a continuous basis. Intermittent samples have been taken in plastic bags, evacuated bottles, Tutweiler burets (Shaw, 1940), and detector tubes (West, 1970). Continuous samples have been taken by exposing chemically treated paper tapes (Sanderson et al., 1966; Peregud et al., 1971) or ceramic tiles (Gilardi & Manganelli, 1963) to air, by pumping air through a lead acetate solution, by bubbling air through impingers containing absorbing or colou-

metric solutions (Goldman et al., 1940), and by using long-duration detector tubes or electronic detectors (West, 1970; ACGIH, 1972; Thompkins & Becker, 1976). Qualitative hydrogen sulfide detection has been based on the blackening of coins, keys, lead-based paint, and lead-acetate treated papers. More recently, direct reading instruments have been developed that make real-time monitoring possible. In some of these instruments, a 2-step absorption-reaction procedure is involved whereas in others, the gas reacts directly with, for example, metal-oxide-coated chips, the electrical properties of which change in response to various gas concentrations. Gas chromatographic methods of analysis have been developed and are particularly used by oil and gas production companies (Stevens et al., 1971). A recent report of the US National Institute for Occupational Safety and Health (NIOSH, 1977) summarizes the current situation with special reference to automatic and/or portable samplers. The report concludes that wet chemical methods are attractive because of their specificity and precision, but that they are less desirable on the basis of the portability and maintenance characteristics of the equipment. Direct reading solid state devices, on the other hand, are portable and relatively rugged but are often nonspecific and susceptible to cross-sensitivities.

However, the practical importance of such cross-sensitivities depends a great deal on the type of study. In ambient air pollution studies in which hydrogen sulfide can be excepted to be in the 0.0015—0.075 mg/m³ (0.001—0.050 ppm) concentration range, interference may be of much greater practical concern than in industrial settings in which concentrations may reach from 30 to 75 mg/m³ (20 to 50 ppm) or more, on occasion, and in which the presence and identity of interfering compounds are often known.

Because of the diversity of circumstances under which hydrogen sulfide has to be determined, only the two principal methods of analysis for hydrogen sulfide are described in detail in the following section. Questions of sampling and analysis suitable for several specific practical applications are also discussed.

2.3.1 The methylene blue method

The methylene blue colorimetric method has been evaluated and recommended by various research workers (Jacobs, 1965; Bamesberger & Adams, 1969) and by some institutions such as the US National Institute for Occupational Safety and Health (NIOSH, 1977). This method has also been proposed by the International Organization for Standardization (ISO, 1978). The Intersociety Committee of the American Public Health Association has published detailed procedures of this method for assessing hydrogen sulfide both in the ambient air and in workplace air (Intersociety Committee, 1977a).

Although light, mercaptans, sulfides, nitrogen dioxide, and sulfur dioxide can cause interference, and instruments incorporating both the absorption and reaction functions are not portable, the methylene blue method appears to combine adequate specificity with good accuracy and precision and extreme sensitivity. It can be used with either manual or automatic sample collectors and, in the latter case, with continuous sampling, levels of hydrogen sulfide as low as 0.003 g/m^3 (0.002 ppm) can be detected (Levaggi et al., 1972).

In the Intersociety Committee method, hydrogen sulfide is collected by aspirating a measured volume of air through an alkaline suspension of cadmium hydroxide. The sulfide is precipitated as cadmium sulfide to prevent air oxidation of the sulfide, which occurs rapidly in an aqueous alkaline solution. Arabinogalactan is added to the cadmium hydroxide slurry to minimize the photodecomposition of the precipitated cadmium sulfide. The collected sulfide is subsequently determined by spectrophotometric measurement of the methylene blue produced by the reaction of the sulfide with a strongly acid solution of *N*, *N*-dimethyl-p-phenylenediamine and ferric chloride. The analysis should be completed within 24–26 h of collection of the sample.

This method is intended for the determination of hydrogen sulfide concentrations in the range of 0.0012–0.1 mg/m^3 (0.0008–0.07 ppm). For concentrations above 0.08 mg/m^3 (0.05 ppm), the sampling period can be reduced or the volume of liquid increased either before or after aspirating. Excellent results have been obtained using this method for air samples containing hydrogen sulfide concentrations in the range of 7.5–75 mg/m^3 (5–50 ppm). This method is also useful for the measurement of source emissions. For example, 100 ml cadmium sulfide-arabinogalactan medium in Greenberg-Smith impingers and 5-min sampling periods have been used successfully.

The methylene blue reaction is highly specific for sulfide at the low concentrations usually encountered in ambient air. Strong reducing agents (e.g., sulfur dioxide) inhibit colour development. Even solutions containing several micrograms of sulfide per millilitre show this effect and must be diluted to eliminate colour inhibition. If sulfur dioxide is absorbed to give a sulfite concentration in excess of 10 μg/ml, colour formation is retarded. Up to 40 μg/ml of this interference, however, can be overcome by adding 2–6 drops (0.5 ml/drop) of ferric chloride instead of a single drop for colour development, and extending the reaction time to 50 min. On the other hand, nitrogen dioxide gives a pale yellow colour with the sulfide reagents at concentrations of 0.5 μg/ml or more. No interference is encountered when 0.57 mg/m^3 (0.3 ppm) of nitrogen dioxide is aspirated through a midget impinger containing a slurry of cadmium hydroxide-cadmium sulfide-arabinogalactan. If hydrogen sulfide and nitrogen dioxide are simultaneously aspirated through cadmium hydroxide-arabinogalactan, slurry, lower results

are obtained, probably because of the gas-phase oxidation of the hydrogen sulfide prior to precipitation as cadmium sulfide.

Using permeation tubes as a source of hydrogen sulfide, a relative standard deviation of 3.5% and a recovery of 80% have been established. The overall sampling and analytical precision is 12.1% relative standard deviation.

Hydrogen sulfide is readily volatilized from an aqueous solution, when the pH is below 7.0. Alkaline aqueous sulfide solutions are very unstable because the sulfide ion is rapidly oxidized by exposure to the air.

Cadmium sulfide is not appreciably oxidized, even when aspirated with pure oxygen in the dark. However, exposure of an impinger containing cadmium sulfide to laboratory or more intense light sources produces immediate and variable photodecomposition. Losses of 50%–90% of sulfide have been routinely reported by a number of laboratories. Even though the addition of arabinogalactan to the absorbing solution controls the photodecomposition, it is necessary to protect the impinger from light at all times. This is achieved by the use of low actinic glass impingers, paint on the exterior of the impingers, or aluminium foil wrapping.

The Intersociety Committee (1977a) has described the apparatus, reagents, and calibration methods suitable for use with midget impinger samplers and appropriate for determining air concentrations of 0.0012–0.1 mg/m^3 (0.0008–0.07 ppm). In this range, calibration is recommended using PTFE permeation tubes. In the higher concentration range relevant to workroom air, the National Institute for Occupational Safety and Health recommends that calibration be carried out using commercially available cylinders of hydrogen sulfide in dry nitrogen (NIOSH, 1977).

2.3.2 Gas chromatography with flame photometric detection

An Intersociety Committee method also exists for the determination of hydrogen sulfide using gas chromatography (Intersociety Committee, 1977b). This method requires the use of a gas chromatograph equipped with a flame photometric detector. A narrow-band optical filter selects the 394 ± 5 mm sulfur line. Gas chromatography separates sulfur compounds of low relative molecular mass before detection, and thereby allows individual quantitative measurement of sulfur-containing gases such as hydrogen sulfide, sulfur dioxide, methyl mercaptan, and dimethyl sulfide.

In situations where minimal quantities of reduced sulfur compounds other than hydrogen sulfide are present, flame photometry can be used directly, in which case the hydrogen sulfide concentration is approximately the same as the total sulfur compounds measured. An absorbant is usually required to selectively remove

sulfur dioxide, when flame photometry is used without separation of individual compounds by gas chromatography before detection.

The limits of detection of this method, at twice the noise level for hydrogen sulfide, sulfur dioxide, methyl mercaptan, and dimethyl sulfide, range from 0.005 to 0.013 mg/m³ (0.0035–0.009 ppm). Sensitivity can be increased by the use of sample concentration techniques such as a freeze-out loop in the gas chromatograph sampling line. The upper limit of detection is 0.5 mg/m³ (0.35 ppm) but it can be extended by sample dilution to higher ranges, if necessary. Although no data are available on the precision and accuracy of the method for atmospheric samples, repetitive sampling of standard reference gases containing a hydrogen sulfide concentration of 0.08 mg/m³ (0.055 ppm) and a sulfur dioxide concentration of 0.104 mg/m³ (0.036 ppm) gave a relative standard deviation of less than 3% of the amount present (Intersociety Committee, 1977b).

An advantage of flame photometric detection is that chemical solutions are not necessary, and the only required reagent is hydrogen for the flame. However, the need for a compressed hydrogen supply may be a disadvantage in certain situations. The analyser is calibrated using hydrogen sulfide, sulfur dioxide, methyl mercaptan, and dimethyl sulfide permeation tubes, and a dual-flow gas dilution device capable of producing reference standard atmospheres as low as the limits of detection of the method. Because the photomultiplier tube output is logarithmically proportional to the sulfur concentration, conversion can be done by either plotting the response against concentration on a logarithmic scale or by using a logarithmic-linear amplifier. Using either of these techniques, the range has been established at approximately 0.13–0.5 mg/m³ (0.09–0.35 ppm) with a 1% noise level (Intersociety Committee, 1977b).

Several commercial flame photometric detection analysers are now available (with and without separation of the sulfur compounds by gas chromatography before detection). This method of analysis for hydrogen sulfide is suitable for use as a laboratory method for calibration purposes or for continuous monitoring in stationary field settings.

2.3.3 Automatic monitors in stationary field settings

Paper tapes impregnated with lead acetate have been widely used for making measurements in the field (Denmead, 1962; Thom & Douglas, 1976; Institute of Hygiene and Epidemiology, 1978). A measured volume of air is filtered through the tape and the optical density of the discoloured area is compared with an unexposed area of the same tape. Numerous criticisms of these procedures have been reported and it is clear that the presence of any substance

capable of oxidizing the lead sulfide can lead to substantial errors (Sanderson et al., 1966). Various modifications of the basic method have been suggested to minimize errors and increase sensitivity by Siu et al. (1971), including the substitution of mercuric chloride for lead acetate as proposed by Paré (1966). Natusch et al. (1974) evaluated various paper tape methods and concluded that tapes impregnated with silver nitrate are highly suitable for the determination of hydrogen sulfide concentrations in the range of 0.0015—75 mg/m³ (0.001—50 ppm). Moreover, they state that monitors using silver nitrate tape are simple, specific, portable, capable of unattended operation and inexpensive. However, silver nitrate tape systems remain subject to photodecomposition, an important deficiency for many field uses.

Continuous monitors based on various wet chemical procedures have been developed. For example, some sulfur dioxide monitors based on amperometric principles can be used to monitor hydrogen sulfide by replacing a silver screen (which normally filters out H_2S) with a barium acetate scrubber that removes any sulfur dioxide in the influent airstream. These devices are reported to have good maintenance characteristics and are suitable for use in remote areas. However, like most continuous instruments based on wet chemistry, they are relatively expensive (Lawrence Berkeley Laboratory, 1976).

Continuous monitors using the methylene blue method have also been developed (Levaggi et al., 1972). These units are attractive in that they possess the inherent sensitivity of the methylene blue method, but they require sophisticated support facilities and highly trained personnel for reliable operation. In this respect, the newer metal oxide-coated-chip semiconductor devices appear promising for field use. However, there are few published reports of field experience to date (Thompkins & Becker, 1976).

2.3.4 Direct-reading portable detection systems

Semiquantitative methods of hydrogen sulfide detection based on lead acetate-treated papers on tiles have been reported and are said to be sensitive to levels of about 1 mg/m³ (0.7 ppm) (Gilardi & Manganelli, 1963). However, long-duration detector tubes for hydrogen sulfide, suitable for use in the occupational environment, are now available, which are inexpensive and responsive over a wide range of concentrations (1—84 mg/m³; 0.7—56 ppm) and overcome some of the deficiencies of the lead acetate detectors (Leichnitz, 1977). The US National Institute for Occupational Safety and Health has investigated the quality and reproducibility of detector tubes available from various US manufacturers and reports that it is possible to obtain tubes that meet the quality specifications of the Institute (Johnson, 1972). Detector tubes for hydrogen sulfide are susceptible to interference from other sulfides, sulfur dioxide, and

nitrogen dioxide, but, generally such interference would result in false positive readings.

Various portable direct-reading hydrogen sulfide meters available on the US market have been evaluated by the National Institute for Occupational Safety and Health (Thompkins & Becker, 1976). These instruments are intended principally for industrial hygiene surveys and, in particular, for ascertaining the degree of general compliance with occupational health standards for hydrogen sulfide. The instruments surveyed operated on solid-state electrochemical principles, wet electrochemical principles, and in one case, on a photoionization principle. In terms of response time, calibration stability, and reliability, the photoionization instrument was regarded as superior, but it was the least specific of the instruments evaluated. The solid-state instruments tended to have slow response times and accuracy deficiencies but were very reliable and rugged. The wet electrochemical meters ranked highly in terms of accuracy, response time, and calibration stability but were somewhat less reliable.

2.3.5 Manual collection and analysis of air samples in occupational settings

In 1943, the American Public Health Association Sub-Committee on Chemical Methods in Air Analysis recommended collecting hydrogen sulfide with cadmium chloride in 2 simple petticoat bubblers in series followed by titration with iodine, using starch as an indicator or using an excess of iodine and back-titrating with sodium thiosulfate solution (Goldman et al., 1943). In 1965, the AIHA Analytical Guide (AIHA, 1965) listed 3 methods for determining hydrogen sulfide in air: *(a)* iodine oxidation using a Tutweiler buret; *(b)* cadmium sulfate and iodine in a midget impinger; and *(c)* formation of cadmium sulfide colloid using 2 midget impingers in series followed by conversion to methylene blue. The iodine methods are susceptible to interference at hydrogen sulfide levels expected in occupational settings. More recently, to ascertain employee exposure to hydrogen sulfide, the National Institute for Occupational Safety and Health recommended the collection of breathing-zone samples with a midget impinger and analysis by the methylene blue method (NIOSH, 1977).

In recent years, there have been developments in the use of solid adsorbents for the collection of sulfur gases. This technique for sample collection could be used in association with the gas chromatography using flame photometric detection for the measurement of hydrogen sulfide and other low relative molecular mass or self-containing gases (Black et al., 1978). This could ultimately lead to the development of solid-state personal dosimeters for hydrogen sulfide as an alternative to those that involve wet chemistry.

3. SOURCES OF HYDROGEN SULFIDE

3.1 Natural Sources

Hydrogen sulfide is one of the principal compounds involved in the natural sulfur cycle in the environment (National Research Council, USA, 1979). As indicated in Fig. 1, it occurs in volcanic gases

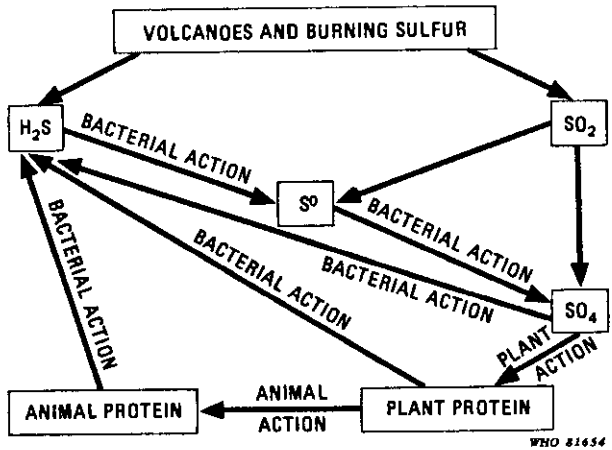

Fig. 1. The sulfur cycle (From: Cooper et al., 1976).

and is produced by bacterial action during the decay of both plant and animal protein (Cooper et al., 1976). Many bacteria, fungi, and actinomycetes release hydrogen sulfide into the environment during the decay of compounds containing sulfur-bearing amino acids and in the direct reduction of sulfate. The heterotroph *Proteus vulgaris* is an example of a common bacterium that produces hydrogen sulfide, when grown in the presence of protein (National Research Council, USA, 1979).

The reduction of sulfate to hydrogen sulfide can be accomplished by members of 2 genera of anaerobic bacteria, *Desulfovibrio* and *Desulfotomaculum*. The organic substrates for these organisms are usually short chain organic acids that are provided by the fermentative activities of other anaerobic bacteria or more complex organic material. Hence, hydrogen sulfide production can be expected in conditions where oxygen is depleted, organic material is present, and sulfate is available (National Research Council, USA, 1979).

From a microbiological point of view, the production of hydrogen sulfide is balanced by processes involving a variety of bacteria, found in soil and water, that can oxidize hydrogen sulfide to elemental

sulfur. Among these are the filamentous bacteria, *Beggiatoa* and *Thiothrix*. Photosynthetic bacteria belonging to the families Chromatiaceae and Chlorobiaceae oxidize hydrogen sulfide to elemental sulfur and sulfate in the presence of light and the absence of oxygen. Reduced sulfur compounds are also oxidized in nature by members of the genus *Thiobacillus*. The end result of this oxidative activity is the production of sulfate which, once formed, is extremely stable to further chemical activity in nature (National Research Council, USA, 1979).

As a result of these various biogeochemical processes, hydrogen sulfide occurs in and around sulfur springs and lakes and is almost continuously present as an air contaminant in some geothermally active areas.

3.2 Sources Associated with Human Activity

There are various circumstances under which naturally occurring hydrogen sulfide is released by human activity. For example, hydrogen sulfide occurring in association with natural gas and/or crude oil deposits in some areas may be released during extraction and drilling operations. The sulfur content of crude oils ranges from 0 to 5% and some natural gas deposits have been reported to comprise up to 42% hydrogen sulfide (Espach, 1950). Coals can contain sulfur levels of up to 80g/kg and, occasionally, conditions arise in which hydrogen sulfide is formed within such deposits. Thus, special precautions must be taken in some mining operations as well as in the drilling and extraction of natural gas and crude oils with significant sulfur content.

Hydrogen sulfide can also be released by activities surrounding the development and use of geothermal resources. At the Cerro Prieto geothermal power generating plant in Baja California, Mexico, for example, hydrogen sulfide levels are sufficiently high to necessitate special ventilation to protect electrical systems, and alarms for the protection of personnel (Mercado, 1975).

During industrial operations, hydrogen sulfide can be formed whenever elemental sulfur or certain sulfur-containing compounds come into contact with organic materals at high temperatures. It is usually produced as an undesirable by-product, though it is also used as an important reagent or desirable intermediate compound in some industrial processes such as the manufacture of sulfides, sodium hydrosulfide, and various organic sulfur compounds. Examples of processes in which hydrogen sulfide occurs as a by-product include the production of coke from sulfur-containing coal, the production of carbon disulfide, the manufacture of viscose rayon in the Kraft process for producing wood pulp (Macaluso, 1969) and sulfur extraction by the Frasch process.

In refining sulfur-containing crude oils, about 80%—90% of the divalent sulfur compounds of hydrogen and carbon are converted to hydrogen sulfide. Both the hydrogen sulfide produced and that occurring in other industrial, geothermal, or natural gas streams can be recovered by one of a number of processes that can be classified as either absorption-desorption processes or processes involving oxidation to oxides or to elemental sulfur. The bulk of hydrogen sulfide recovered in industrial processes is used to produce elemental sulfur or sulfuric acid (Macaluso, 1969).

Large quantities of hydrogen sulfide are used in the production of heavy water, which is employed as a moderator in some nuclear power reactors. The process is based on enrichment of the deuterium content of water by hydrogen sulfide in a gas/liquid ion exchange system, followed by separation of heavy water and water by fractional distillation (McGraw-Hill Encyclopedia of Science and Technology, 1960).

In the tanning industry, hydrogen sulfide is produced in the process by which hair or wool is removed from the hides. This typically involves deliming by adding ammonium chloride or ammonium sulfate followed by pickling with sulfuric acid, and takes place in large rotating drums. The gases evolved, including hydrogen sulfide, are released from the drums on opening the hatches either to add chemicals or to unload the treated hides, and also from the waste waters (ILO, 1971).

As in the natural environment, hydrogen sulfide can be generated by bacterial action in industrial or community settings in malodorous and sometimes dangerous amounts.

In some countries, such as India and Sri Lanka, hydrogen sulfide is produced in the process by which coconut fibres are separated from the husk. This procedure involves the decomposition of the husks in shallow ponds. The hydrogen sulfide is produced as a result of microbiological decay processes.

4. ENVIRONMENTAL LEVELS AND EXPOSURE

4.1 Concentrations in Outdoor Air

There are few published data on either natural background or urban air levels of hydrogen sulfide. Robinson & Robbins (1970) estimated the average ambient air level of hydrogen sulfide to be 0.0003 mg/m^3 (0.0002 ppm). This estimate supports the data of Minster (1963) who, when sampling over a 2^1/$_2$-year period in northwest London, reported that air levels of hydrogen sulfide were generally below 0.00015 mg/m^3 (0.0001 ppm), under clear, fresh conditions. Minster (1963) also reported that average summer levels ranged from 0.00015 to 0.0007 mg/m^3 (0.0001—0.0005 ppm) and average winter

levels from 0.0007 to 0.0015 mg/m³ (0.0005—0.001 ppm). Data collected from this same station during the London fog of December 1962 indicated a hydrogen sulfide concentration of up to 0.046 mg/m³ (0.033 ppm) on 6 December during heavy fog (each sampling time was 32 min).

Measurements summarized by the US National Air Pollution Control Administration show concentrations ranging from below 0.001 mg/m³ to 0.006 mg/m³ (0.0007 to 0.0042 ppm) at various urban locations in the USA in the period 1951—64 (Miner, 1969). However, as sensitive and standardized methods of sampling and analysis for hydrogen sulfide were lacking during this period, there is some doubt about the reliability of these data. Furthermore, the averaging times for the data are not available.

Much higher concentrations of hydrogen sulfide have been measured near point sources. In California, peak concentrations as high as 0.20 mg/m³ (0.13 ppm) were measured near a pulp and papermill at the time of its commissioning (California Air Resources Board, 1970). After operating for several months, levels fell to 0.015 mg/m³ (0.010 ppm) or less. The averaging time was not reported. Near a brick works in Boom, Belgium, air levels of hydrogen sulfide were monitored over a 6-month period. During this time, the average 24-h concentration of hydrogen sulfide was 0.005 mg/m³ (0.003 ppm) with occasional daily averages in excess of 0.017 mg/m³ (0.011 ppm) (Institute of Hygiene & Epidemiology, 1978).

A major accidental release of hydrogen sulfide occurred at Poza Rica, Mexico, in 1950 (McCabe & Clayton, 1952). Although no data could be collected on environmental levels during this episode, numerous fatalities occurred indicating that exposure levels were most likely in excess of 1500—3000 mg/m³ (1000—2000 ppm). Further details of this episode are given in section 6.3.

In the geothermally active areas in and around the city of Rotorua, New Zealand, airborne concentrations of hydrogen sulfide are usually sufficient to cause noticeable odours (Thom & Douglas, 1976). At one site, for one day, a 1-h mean concentration of up to 2.0 mg/m³ (1.4 ppm) was reported (Thom & Douglas, 1976). Continuous measurements taken at another site over a period of 5 months showed that a concentration of 0.08 mg/m³ (0.05 ppm) was exceeded, on average, 35% of the time. It was also found that there were considerable seasonal variations in the hydrogen sulfide levels, reflecting the fluctuating steam-use patterns and also changes in the dispersive nature of the atmosphere. During the mid-winter months of the 1978 monitoring period, a concentration of hydrogen sulfide in air of 0.08 mg/m³ (0.05 ppm) was exceeded more than 55% of the time, whereas, during warmer months, this concentration was exceeded less than 20% of the time (Rolfe, 1980).

Also in New Zealand, the discharge of industrial and domestic liquid wastes into an inlet near Auckland created conditions in which

hydrogen sulfide levels were sufficient to cause paint blackening and complaints of offensive odours. Continuous air monitoring was conducted for 21 months. These data indicated that 40-min average hydrogen sulfide concentrations in air of from 0.8 to 1.4 mg/m³ (0.5–0.96 ppm) occurred at some time during the worst months of the year at all the sites monitored (Denmead, 1962).

4.2 Concentrations in Work Places

Under normal operating conditions, concentrations of hydrogen sulfide in the air in work places are believed to be less than 10–15 mg/m³ (7–10 ppm), the 8-h time weighted average that most national authorities have set as their occupational exposure standard (Annex).

It is well known, however, that hazardous exposures to hydrogen sulfide can occur under accidental circumstances in industries in which gas streams with a high hydrogen sulfide content exist. Furthermore, as hydrogen sulfide is slightly heavier than air, it can accumulate in toxic concentrations in low-lying areas, even when generated or leaking at very low rates. However, in such cases, the environmental levels of hydrogen sulfide have usually only been measured after the accidents in question, or have been determined by simulation or reenactment. Concentrations that have been reported range from 150 mg/m³ (100 ppm), in which a worker lost consciousness while sawing ebonite boards (Brown, 1969), to 18 000 mg/m³ (12 000 ppm) in a case in which a truck driver died while cleaning the tank of a vehicle used to transport industrial waste (Simson & Simpson, 1971). In an outdoor setting, 4 workmen lost consciousness while digging a pit in marshy land in which hydrogen sulfide concentrations in air of 442–810 mg/m³ (295–540 ppm) were measured 5 days later (Anonymous, 1952). Alexander (1974) reported hydrogen sulfide concentrations as high as 0.037 mg/litre (24.8 ppm) in a sewage stabilization pond and 10.0–13.2 mg/m³ (6.7–8.8 ppm) in the air 15 m from this pond. In a report by Ahlborg (1951) on hydrogen sulfide poisoning in the Swedish shale oil industry, concentrations of hydrogen sulfide measured at various locations in the plant ranged from 30 to 900 mg/m³ (20–600 ppm), though men seldom worked where the high levels occurred.

More recently, the US National Institute for Occupational Safety and Health reported that, in viscose rayon churn rooms, spinning tanks, and drying and storage cells, workers were mainly exposed during the working day to hydrogen sulfide concentrations of 23 mg/m³ (15 ppm) or less with occasional peaks of 150 mg/m³ (100 ppm) (NIOSH, 1977).

In the USA, it has been estimated that there are 125 000 employees potentially exposed to hydrogen sulfide (NIOSH, 1977). Table 1 is a list of occupations in which such exposure can occur

ranging according to occupation from rare exposure to low concentrations, to frequent exposure to concentrations very near those associated with adverse health effects.

Table 1. Examples of occupations with potential exposure to hydrogen sulfide[a]

Animal fat and oil processors	Lithographers
Animal manure removers	Lithopone makers
Artificial-flavour makers	Livestock farmers
Asphalt storage workers	Manhole and trench workers
Barium carbonate makers	Metallurgists
Barium salt makers	Miners
Blast furnace workers	Natural gas production and processing workers
Brewery workers	Painters using polysulfide caulking compounds
Bromide-brine workers	Papermakers
Cable splicers	Petroleum production and refinery workers
Caisson workers	Phosphate purifiers
Carbon disulfide makers	Photo-engravers
Cellophane makers	Pipeline maintenance workers
Chemical laboratory workers, teachers, students	Pyrite burners
Cistern cleaners	Rayon makers
Citrus root fumigators	Refrigerant makers
Coal gasification workers	Rubber and plastics processors
Coke oven workers	Septic tank cleaners
Copper-ore sulfidizers	Sewage treatment plant workers
Depilatory makers	Sewer workers
Dyemakers	Sheepdippers
Excavators	Silk makers
Felt makers	Slaughterhouse workers
Fermentation process workers	Smelting workers
Fertilizer makers	Soapmakers
Fishing and fish-processing workers	Sugar beet and cane processors
Fur dressers	Sulfur spa workers
Geothermal-power drilling and production workers	Sulfur products processors
Gluemakers	Synthetic-fibre makers
Gold-ore workers	Tank gaugers
Heavy-metal precipitators	Tannery workers
Heavy-water manufacturers	Textile printers
Hydrochloric acid purifiers	Thiophene makers
Hydrogen sulfide production and sales workers	Tunnel workers
Landfill workers	Well diggers and cleaners
Lead ore sulfidizers	Wool pullers
Lead removers	

[a] From: NIOSH (1977).

5. EFFECTS ON EXPERIMENTAL ANIMALS

Very little information is available on the effects of low level concentrations of hydrogen sulfide gas on experimental animals; most published data have emphasized the effects of exposure to lethal or near-lethal concentrations of the gas. According to Evans

(1967) and Smith & Gosselin (1979), the effects of high doses of hydrogen sulfide and high doses of cyanide are very similar. Both inhibit the enzyme cytochrome c oxidase [EC 1.9.3.1]. This was demonstrated in studies using purified preparations of the enzyme (Smith & Gosselin, 1979).

When sodium sulfide at 0.1, 0.25, and 0.32 mmol/kg body weight was administered intraperitoneally to mice, sulfide was not exhaled (Susman et al., 1978) suggesting that it was inactivated primarily by metabolism. In studies on the inhalation of hydrogen sulfide in rats, cats, rabbits, and dogs, the nervous centres were first excited and then paralysed; pupils first contracted and then dilated; blood pressure was first raised, then lowered; and respiration first increased and then halted (Evans, 1967; Haggard, 1925). The findings of Lehmann (1892) in the cat, dog, and rabbit, of Haggard (1925) in the dog, and of Sayers et al. (1925) in the canary, rat, guineapig, dog, and goat, are quite consistent: at 150–225 mg/m^3 (100–150 ppm), signs of local irritation of eyes and throat after many hours of exposure; at 300–450 mg/m^3 (200–300 ppm), eye and mucous membrane irritation after inhalation for 1 h and slight general effects with prolonged inhalation; at 750–1050 mg/m^3 (500–700 ppm), local irritation and slight systemic symptoms in less than 1 h and possible death after several hours' exposure; at 1350 mg/m^3 (900 ppm), grave systemic effects within 30 min and death in less than 1 h; at 2250 mg/m^3 (1500 ppm), collapse and death within 15–30 min; and at 2700 mg/m^3 (1800 ppm), immediate collapse, respiratory paralysis, and death.

When mice were exposed repeatedly (4 times for 2 h, at 4-day intervals) to a hydrogen sulfide concentration in air of 150 mg/m^3 (100 ppm), the critical inhibition of terminal cytochrome c oxidase appeared to be cumulative. This effect was accompanied by a cumulative decrease in cerebral RNA synthesis (Savolainen et al., 1980). Experimental studies on rabbits indicated that either a single or repeated exposure for 1.5 h per day (5 consecutive days) to a hydrogen sulfide concentration in air of 105 mg/m^3 (70 ppm) caused electrocardiographic (ECG) changes (Kósmider et al., 1967).

Though some small differences in susceptibility to hydrogen sulfide gas were exhibited among the species studied by Sayers et al., (1925), canaries being the most sensitive and goats the most resistant, the interspecies differences were slight. It is agreed among investigators that the effects of hydrogen sulfide gas on the nervous system represent the most important aspect of its toxicity (Haggard, 1925; Evans, 1967). Beck et al. (1979) showed that ethanol in doses of 0.33–0.66 g/kg significantly shortened the time to loss of consciousness in rats exposed to a hydrogen sulfide concentration in air of 1200 mg/m^3 (800 ppm) for 30 min. The induction of methaemoglobinaemia by the injection of sodium nitrite had both protective and antidotal effects against hydrogen sulfide poisoning in mice, armadillos, rabbits, and dogs (Smith & Gosselin, 1979).

Water containing a hydrogen sulfide concentration as low as 0.86 mg/litre was toxic to trout after exposure for 24 h (McKee & Wolf, 1971).

6. EFFECTS ON MAN

Adequate systematic studies of the relationship between hydrogen sulfide exposure and health status in the general population have not been carried out. Controlled exposure of human subjects to concentrations of hydrogen sulfide gas exceeding about 75 mg/m³ (50 ppm) has been deemed to involve excessive risk because of the possibility of injury to the lungs (Sayers et al., 1925; National Research Council, USA, 1979). Furthermore, except for studies related to odour threshold, controlled exposures of human subjects to very low concentrations of the gas, for example, below 1.5 mg/m³ (1.0 ppm) have not been reported. Thus, the information presented in this section has mainly been derived from reports of accidental and industrial exposures to hydrogen sulfide. A general discussion of the toxicology of hydrogen sulfide has been included, because a basic understanding of the subject is necessary for a discussion of the role of the gas as an industrial and community hazard.

6.1 General Toxicological Considerations

The following observations have been derived from reports of studies involving man. However, for clarification, some studies on experimental animals have also been included. In general, both animals and man respond in a very similar fashion to toxic concentrations of hydrogen sulfide. It is both an irritant and an asphyxiant gas (Table 2) that induces local inflammation of the membranes of the human eye and respiratory tract (Yant, 1930). It has been shown that eye irritation, the most commonly reported effect of hydrogen sulfide exposure, can occur after several hours' exposure to concentrations of 16—32 mg/m³ (10.5—21.0 ppm) (Elkins, 1939; Nesswetha, 1969). However, pulmonary tract irritation is, potentially, a more serious reaction. When inhaled by dogs, hydrogen sulfide exerted an irritant action through the entire respiratory tract, though the deeper structures suffered the greatest damage

(Haggard, 1925). Inflammation of these deeper structures may result in pulmonary oedema.

Exposure to hydrogen sulfide gas did not induce important effects on the human skin nor was any appreciable absorption through intact skin observed (Yant, 1930). However, Petrun (1966) reported that when the skin of rabbits was exposed to hydrogen sulfide at concentrations of 1050 and 2100 mg/m³ (700 and 1400 ppm), trace amounts of hydrogen sulfide were found in the exhaled air of the rabbits. No quantitative information was given.

Hydrogen sulfide gas is rapidly absorbed through the lung. Like hydrogen cyanide, it is a potent inhibitor of cytochrome c oxidase that interferes with tissue use (Smith & Gosselin, 1979). As a result, the oxidative metabolism may slow to the point where tissue metabolic demands cannot be met. In the central nervous system, the result may be paralysis of the respiratory centres. Respiratory arrest and death from asphyxia would be the natural outcome.

In studies on dogs (Haggard, 1925), hydrogen sulfide at concentrations of 1500–3000 mg/m³ (1000–2000 ppm) initially stimulated excessively rapid breathing (hyperpnoea), because of a depletion in the carbon dioxide content of the blood (hypocapnia). This was followed by a period of respiratory inactivity (apnoea). Spontaneous respiration may be reestablished, if carbon dioxide depletion has not progressed beyond the point where prompt reaccumulation can act as a stimulus to the reestablishment of respiration. If spontaneous recovery does not occur and artificial respiration is not applied rapidly, death from asphyxia is the inevitable result (Haggard, 1925). At about 2250 mg/m³ (1500 ppm), the sequence of events in the dog was the same, except that the reaction was more pronounced; at 3000 mg/m³ (2000 ppm) there was respiratory paralysis after a breath or two and, in Haggard's words, "the victim falls to the ground as though struck down". When breathing ceases, generalized convulsions frequently begin. There appears to be no clear explanation of the cause of this picture of sudden collapse. According to Haggard (1925), this form of respiratory failure is not related to the carbon dioxide content of the blood but, rather, to the directly paralysing effect of hydrogen sulfide on the respiratory centre. Breathing is never reestablished spontaneously following this hydrogen sulfide-induced respiratory paralysis. Haggard noted, however, that because the heart continues to beat for several minutes after respiration has ceased, death from asphyxia can be prevented if artificial respiration is begun immediately and is continued until the hydrogen sulfide concentration in the blood decreases. This decrease is probably a consequence of metabolic processes, as shown in mice by Susman et al. (1978) rather than, as once believed, the result of the pulmonary excretion of the gas.

Smith & Gosselin (1979) have called attention to the confusion that exists in the literature with regard to the effects of hydrogen

sulfide on haemoglobin. They emphasize that many studies have proved that neither sulfhaemoglobin nor any other abnormal pigments are present in significant concentrations in the blood of animals or human subjects, fatally poisoned by hydrogen sulfide.

The characteristic "rotten egg" odour of hydrogen sulfide is an important aspect of the toxicology of the gas. The threshold of perception (odour) varies considerably depending on individual sensitivity. Several authors have reported odour detection thresholds ranging from 0.0007 mg/m³ to 0.20 mg/m³ (0.0005—0.13 ppm) (Table 2). Thus, the odour of hydrogen sulfide gas can be a very sensitive indicator of its presence in low concentrations. However, at higher concentrations (> 225 mg/m³ (150 ppm)), hydrogen sulfide exerts a paralysing effect on the olfactory apparatus (Milby, 1962), thus neutralizing the value of its odour as a warning signal. Poda (1966) reported that among 42 workers, who were rendered unconscious from overexposure to hydrogen sulfide, the majority did not smell the characteristic odour of the gas but noted a sickeningly sweet odour, very briefly, before losing consciousness.

Table 2. Effects of hydrogen sulfide exposure at various concentrations in air

Effect	Concentration		Duration of exposure	Reference
	mg/m³	ppm		
Man Approximate threshold for odour	0.0007—0.2	0.0005—0.13	A few seconds to less than 1 min	Yant (1930); Ryazanov (1962); Adams & Young (1968); Leonardos et al. (1969); Lindvall (1970); Thiele (1979); Winneke et al. (1979)
Threshold of eye irritation	16—32	10.5—21	6—7 h	Elkins (1939) Nesswetha (1969)
Acute conjuctivitis (gas eye)	75—150	50—100	> 1 h	Yant (1930)
Loss of sense of smell	225—300	150—200	2—15 min	Sayers et al. (1925)
Animals [a] Local irritation and slight systemic symptoms; possible death after several hours	750—1050	500—700	< 1 h	Haggard (1925)
Systemic symptoms; death in less than 1 h	1350	900	< 30 min	Haggard (1925)
Death	2250	1500	15—30 min	Haggard (1925)

[a] These observations were made in experimental animals. However, there are no better quantitative data available concerning man with respect to exposure to hydrogen sulfide at high concentrations.

Hydrogen sulfide intoxication in man has generally been categorized according to 3 clinical forms, acute, subacute, and chronic, depending on the nature of the predominant clinical signs and

symptoms (National Research Council, USA, 1979). The term "acute hydrogen sulfide intoxication" has been most often used to describe systemic poisoning characterized by rapid onset and predominance of signs and symptoms of nervous system involvement. The term "subacute intoxication" has been applied to episodes of poisoning in which signs and symptoms of eye and respiratory tract irritation were most prominent. "Chronic hydrogen sulfide intoxication" has been applied by some authors to describe a prolonged state of symptoms resulting from a single or repeated exposure to concentrations of hydrogen sulfide that do not produce clear-cut manifestations of either acute or subacute illness.

In a document prepared by the National Research Council of the National Academy of Sciences of the USA (National Research Council, USA, 1979), the observation was made that application of the terms "acute", "subacute" and "chronic" to hydrogen sulfide exposure was both imprecise and misleading. However, rather than abandon these frequently used terms altogether, the authors suggested a series of clarifying definitions which are quoted in the following paragraphs, and will henceforth be used in this section.

"*Acute intoxication:* Effects of a single exposure [seconds- minutes] [a] to massive concentrations of hydrogen sulfide that rapidly produce signs of respiratory distress. Concentrations approximating 1400 mg/m^3 (1000 ppm) are usually required to cause acute intoxication.

Sub-acute intoxication: Effects of continuous exposure [up to several hours] [a] to mid-level 140 to 1400 mg/m^3 (100 to 1000 ppm) concentrations of hydrogen sulfide. Eye irritation (gas eye) is the most commonly reported effect, but pulmonary edema (in the absence of acute intoxication) has also been noted.

Chronic intoxication: Effects of intermittent exposures to low to intermediate concentrations 70 to 140 mg/m^3 (50 to 100 ppm) of hydrogen sulfide, characterized by "lingering", largely subjective manifestations of illness."

It is important to note that these definitions do not include a consideration of the health consequences to man associated with prolonged low-level exposure to hydrogen sulfide gas, such as may be encountered under conditions of general urban air pollution.

A concentration of hydrogen sulfide in drinking water as low as 0.07 µg/litre (0.05 ppm) can affect its taste (Campbell et al., 1958).

6.2 Occupational Exposure

In certain occupations, workers are intermittently exposed to concentrations of hydrogen sulfide that are not only malodorous but

[a] Time factors added by WHO Task Group.

can, in some situations, cause severe adverse health effects and even death. Usually, hydrogen sulfide is encountered in the workplace as an undesirable by-product of a manufacturing process, notably petroleum refining, viscose rayon production, sugar beet processing, and tannery work (Milby, 1962; NIOSH, 1977). In other occupations, for example cesspool cleaning and work in sewers, exposure to hydrogen sulfide may occur when the gas is formed as a result of the decomposition of sulfur-containing organic matter, in the absence of complete oxidation. Deaths attributed to such exposure occurred in the sewers of Paris in the 1780s (Mitchell & Davenport, 1924) and still occur under various circumstances.

Workers in certain occupations risk exposure to naturally occurring hydrogen sulfide; geothermal energy workers and spa attendants may be included in this category.

Acute hydrogen sulfide intoxication is a dramatic, often fatal event. Three men were inadvertently enveloped in a cloud of hydrogen sulfide gas escaping from a cylinder under high pressure; all fell, as if struck down, and ceased breathing. Only as a result of prompt resuscitation by trained onlookers did the men survive, though the two most seriously affected experienced violent convulsions and did not recover consciousness for some 30 min. None of the men suffered important after-effects, and none recalled having noted the characteristic odour of hydrogen sulfide. The hydrogen sulfide concentrations to which the men were exposed were estimated to be about 2800 mg/m^3 (2000 ppm) (Milby, 1962). Twelve workmen in a plant that produced benzyl polysulfide were overcome by hydrogen sulfide gas, when a pipe used to transfer sodium sulfhydrate ruptured. The liquid sulfhydrate drained into a nearby sewer, where it reacted with acid sewage releasing hydrogen sulfide from several sewer openings in the immediate vicinity. Two of the 12 workmen died, probably as a result of respiratory arrest; 3 stopped breathing but were successfully resuscitated; 6 lost consciousness but recovered spontaneously, and 1 individual developed pulmonary oedema that responded to therapy (Kleinfeld et al., 1964).

Burnett et al. (1977) reviewed 221 cases of exposure to hydrogen sulfide associated with the oil, gas, and petrochemical industries in Canada. The overall mortality was 6%; three-quarters of all victims experienced a period of unconsciousness and 12% were comatose. A high proportion of patients had other neurological signs and symptoms, including altered behaviour patterns, confusion, vertigo, agitation, or somnolence. Respiratory tract effects were second in frequency only to neurological manifestations. Forty percent of all cases required some form of respiratory assistance and 15% of all cases developed pulmonary oedema. Less severely affected patients complained primarily of headaches, sore eyes, or gastrointestinal upsets. There were no recognizable sequelae among the survivors. Data on environmental exposure levels were not reported.

Nearly fatal cases of acute hydrogen sulfide intoxication associated with sequelae of varying severity have been reported. A 48-year-old farmer, who collapsed from hydrogen sulfide intoxication while shovelling manure, continued to have convulsive seizures after resuscitation. The ECG changes suggested myocardial infarction. The patient recovered but a slight, persistent dizziness remained (Kaipainen, 1954). A 46-year-old sewer worker, who was overcome by hydrogen sulfide in a manhole for 30 min, was cyanotic, suffering generalized spasms, and required artificial respiration. A week later, he could move and speak only with great effort; a month afterwards, he still exhibited neurological deficits. The ECG showed evidence of small anterolateral infarct and a right bundle branch block. Three months later, although ambulatory, the patient still suffered from anginal pain upon exertion (Hurwitz & Taylor, 1954). In a man who had suffered severe hydrogen sulfide intoxication with collapse and respiratory failure, ECG evidence of myocardial ischaemia was noted during the early phase of acute illness but gradually disappeared over a period of 15 days (Kemper, 1966). Each of these 3 events, in which sequelae were reported, were characterized by periods of unconsciousness during which hypoxia of vital tissues was likely to have occurred and may have been the basis for the observed prolonged effects. Many other instances of sequelae following acute hydrogen sulfide intoxication have been reported. Most resemble the cases just mentioned, in which serious poisoning with unconsciousness preceded the appearance of sequelae.

Ahlborg (1951) described 58 cases of acute hydrogen sulfide intoxication in Sweden's shale oil industry. There were no fatalities. Symptoms were generally uniform: sudden fatigue, dizziness, and intense anxiety followed by unconsciousness with or without respiratory failure. Several cases of acute poisoning were not associated with unconsciousness, otherwise symptoms were similar to those of the other cases. The diagnosis of "sequelae after acute hydrogen sulfide poisoning" was made in 15 cases. The majority of these had a history of repeated acute intoxications followed in each case by neurasthenic problems (fatigue, somnolence, headache, lack of initiative, irritability, anxiety and poor memory, and decreased libido), though some developed sequelae following acute intoxication without an intercurrent epidsode of unconsciousness. Many stricken workers developed an increase in sensitivity (aversion) to the odour of gas of any type, even pure gasoline vapour (Ahlborg, 1951).

Numerous case histories of fatal hydrogen sulfide intoxication have been reported (Larson et al., 1964; Adelson & Sunshine, 1966; Simson & Simpson, 1971). Oedema of the lungs and brain are common post-mortem findings. The presence of detectable concentrations of hydrogen sulfide in the blood has been reported on several occasions (Larson et al., 1964; Adelson & Sunshine, 1966) and con-

centrations in the blood of fatally poisoned victims have ranged from 1.70 mg to 3.75 mg/litre (McAnalley et al., 1979).

In acute hydrogen sulfide intoxication, cessation of respiration is an immediate threat to life. Accordingly, the provision of artificially assisted respiration on an emergency basis is absolutely critical. There is some question as to whether mouth-to-mouth resuscitation may create a potential health hazard to the rescuer, because of the presence of hydrogen sulfide in the expired air or on the clothing of the victim. Thus, methods of artificial respiration requiring less direct contact (for example, back-pressure-arm lift) may be prudent.

6.3 General Population Exposure

There are several reports of episodes of general population response to air contamination by hydrogen sulfide. Information derived from these events is consistent with observations reported among workers occupationally exposed to hydrogen sulfide. Table 2 demonstrates the wide range of odour perception thresholds for hydrogen sulfide reported by various investigators. In view of the magnitude of these differences, it is not possible to state with certainty the concentration at which odour-related complaints can be expected.

A catastrophic exposure episode involving the release of large quantities of hydrogen sulfide into a small community was reported by McCabe & Clayton (1952). This occurred in 1950 in Poza Rica, Mexico, a city of 22 000 people located about 210 km northeast of Mexico City. Poza Rica was then the centre of Mexico's leading oil-producing district and the site of several oil field installations, including a sulfur-recovery plant. An early morning malfunction of the waste gas flare resulted in the release of large quantities of unburned hydrogen sulfide into the atmosphere. The unburned gas, aided by a low-level temperature inversion and light early morning breezes, was carried to a residential area adjacent to the plant area. Residents of the area were overcome while attempting to leave the area and while assisting stricken neighbours. Within 3 h, 320 persons were hospitalized and 22 died. The most frequent symptom was loss of the sense of smell. More than half of the patients lost consciousness, many suffered signs and symptoms of respiratory tract and eye irritation and 9 developed pulmonary oedema. Four of the 320 victims exhibited neurological sequelae; 2 experienced neuritis of the acoustic nerve; 1 developed dysarthria; the fourth patient suffered aggravation of pre-existing epilepsy. The duration of these neurological sequelae was not reported.

There have been reports of other episodes of general atmospheric pollution by hydrogen sulfide evolved from both natural and

industrial sources, but none has been as severe as the Poza Rica incident.

In the geothermal areas of Rotorua, New Zealand, a city of 40 000 people, steam and hot water from approximately 500 active bores are used to heat houses, buildings, and swimming pools, and to provide domestic hot water supplies and steam for cooking boxes.

In these areas, accidental fatal cases of acute hydrogen sulfide intoxication associated with improper ventilation of geothermal steam-heated dwellings have occasionally been reported. For example, at least 3 deaths from acute hydrogen sulfide poisoning occurred in 1962. However, with the introduction of legal requirements regarding the safe domestic use of the geothermal resources, and possibly also as a result of increased public awareness of the dangers involved, no fatalities attributable to hydrogen sulfide have occurred in Rotorua since 1962 (Thom & Douglas, 1976).

Air pollution monitoring for hydrogen sulfide has been conducted, during several periods over the past 15 years, at various sites in Rotorua. However, there has been considerable variation in the results obtained between the various sites, and furthermore, the concentrations measured showed considerable seasonal variations. Some of the results of these measurements are mentioned in section 4.1.

In 1964, the Division of Air Pollution, US Public Health Service, reported that in the city of Terre Haute, Indiana, biodegradation of industrial wastes in a 14.5-ha lagoon caused the atmospheric concentration of hydrogen sulfide to reach a 1-h mean concentration of 0.45 mg/m^3 (0.3 ppm). As a result, 81 complaints were registered by the public, 41 of which were health-related. The most common complaints were concerned with the perception of a foul odour. The most common effects were nausea, interruption of sleep, burning of the eyes, and shortness of breath. Less common manifestations were cough, headache, and anorexia. Several acute asthma attacks were reported, but the association of these attacks with the hydrogen sulfide incident was not clearly established. Though no grave physical illnesses could be directly related to the air pollution incident, the investigators stressed that the odours emanating from the lagoon caused more than a mere nuisance (United States Public Health Service, 1964).

The Poza Rica tragedy provides ample evidence that the accidental release of hydrogen sulfide into a community can be expected to cause systemic intoxication of varying severity. The Rotorua experience is notable in that it emphasizes the fact that the potential for serious, even fatal, hydrogen sulfide intoxication is present in active geothermal areas. The more common picture of general population exposure is exemplified by the Terre Haute incident where an industry-related source of low-level concentrations of hydrogen

sulfide gas created a health problem which, although not grave, exceeded the level of mere nuisance.

The potential of long-term, low-level exposure to hydrogen sulfide to cause pulmonary changes of the type known to be associated with other irritant gases such as oxides of nitrogen and sulfur has scarcely been studied. No epidemiological data are available, at present, upon which to base any sound conclusions.

7. EVALUATION OF HEALTH RISKS TO MAN FROM EXPOSURE TO HYDROGEN SULFIDE

By far the most important recognized toxic effect of hydrogen sulfide is its ability to induce acute intoxication, characterized by immediate collapse, frequently accompanied by respiratory arrest and, without treatment, death. The scientific literature abounds with cases of this type, most often associated with industrial overexposure. However, a few cases of acute hydrogen sulfide exposure have been recorded in the general population as a result of the release of hydrogen sulfide either from an industrial process or from natural sources. A second form of injury associated with exposure to hydrogen sulfide is caused by the irritative action of the gas on the mucous membranes of the eyes and respiratory tract. Keratoconjunctivitis (gas eye) and pulmonary oedema are two most serious manifestations of this local irritative effect. The malodorous property of hydrogen sulfide gas is well recognized and this characteristic alone is believed by many to be capable of producing impairment of human health and well-being.

Most of the information available on human health effects associated with exposure to various concentrations of hydrogen sulfide gas has come from observations on accidental and industrial exposures. With the exception of the Poza Rica catastrophe, information on general population exposures and associated health effects is sketchy at best. There is also little information on controlled human exposure to hydrogen sulfide gas, and, except for data from odour threshold studies, the information that is available is more than 50 years old. Although a small amount of information is available on the effects on experimental animals of high concentrations of hydrogen sulfide gas, there is virtually no information on the long-term, low-level effects of the gas on experimental animals.

Furthermore, epidemiological data are lacking concerning the health consequences of long-term, low-level exposure to hydrogen sulfide, in both the general and industrial populations.

7.1 Exposure Levels

General population air pollution problems associated with hydrogen sulfide arise mainly in connexion with malodorous conditions, traceable to point sources. Such sources can be industrial or, in some cases, polluted bodies of water. Peak levels as high as 0.20 mg/m^3 (0.13 ppm) have been reported in the air in the neighbourhood of industrial sources. Hydrogen sulfide is also a common pollutant in geothermally active areas. At one site in a geothermal area in New Zealand, where continuous measurements were carried out over a 5-month period, a level of 0.08 mg/m^3 (0.05 ppm) was exceeded, on average, for 35% of the time.

Concentrations of hydrogen sulfide in the workplace also vary widely. In the shale oil industry and in viscose rayon production, for example, maximum levels of exposure during the work day have been reported to range from 23 to 30 mg/m^3 (15–20 ppm). In general, however, massive accidental exposure to hydrogen sulfide has constituted the principal hazard of this gas in industrial settings. In many cases, such exposure has occurred because of equipment breakage or malfunction. However, because hydrogen sulfide is heavier than air, it can accumulate in lethal concentrations in low-lying or enclosed areas. Numerous fatalities have occurred from the slow, insidious accumulation of the gas in the air in both ambient and industrial environments.

7.2 Experimental Animal Studies

The toxic effects of hydrogen sulfide gas have not been studied extensively in experimental animals. However, in studies on a number of animal species including the mouse, rat, cat, dog, and goat, it has been shown that the primary target of hydrogen sulfide in high doses is the nervous system. Collapse, followed by respiratory arrest and asphyxia resulting from the paralysing effects of high concentrations of hydrogen sulfide on the respiratory centres of the central nervous system, is the usual sequence of events leading to death.

Little information is available in the published literature concerning the effects in experimental animals of long-term, low-level exposure to hydrogen sulfide.

7.3 Effects of Occupational Exposure

Inadvertent and accidental exposure of human subjects to high concentrations of hydrogen sulfide has occurred among workers engaged in petroleum refining, viscose rayon production, sugar beet processing, and tannery work. Exposure levels have not been precisely documented in many of these situations. Reported effects range from the relatively less grave conditions of neurasthenic and oto-neurological symptoms and keratoconjunctivitis to the more serious effects of pulmonary oedema, respiratory failure, collapse, and even death. From the available data, it can be estimated that exposure for seconds or minutes to concentrations of approximately 1400 mg/m³ (1000 ppm) or more would cause acute intoxication, concentrations of 140–1400 mg/m³ (100–1000 ppm) with an exposure time of up to several hours would produce keratoconjunctivitis and pulmonary oedema, and intermittent exposures to concentrations of 70–140 mg/m³ (50–100 ppm) could be associated with lingering, largely subjective, manifestations believed by some to represent chronic intoxication. Various studies have associated exposure to hydrogen sulfide in concentrations as low as 16–32 mg/m³ (10.5–21.0 ppm) for several hours with eye irritation in workers.

Effects of low-level, long-term industrial exposure to hydrogen sulfide have not been systematically evaluated.

7.4. Effects of General Population Exposure

Several episodes of exposure of the general population to hydrogen sulfide emanating from a specific source have been investigated and described. For the most part, these events involved only annoyance because of the odour or, at worst, minor temporary illness such as headache, nausea, and sleeplessness. However, as described in section 6.3, on two occasions, general population exposure to hydrogen sulfide caused grave illness and even death: one at Poza Rica, Mexico, and the other in and around Rotorua, New Zealand.

Unfortunately, these incidents were not studied using epidemiological techniques, and it is not possible to establish exposure-effect relationships from the data.

7.5 Guidelines for the Protection of Public Health

There are two aspects concerning the protection of public health in relation to hydrogen sulfide exposure: *(a)* the protection of the public, and occupational groups in particular, from the toxicological effects of such exposure; and *(b)* the protection of the public from

the odour nuisance that can be associated with releases of hydrogen sulfide.

The odour threshold for hydrogen sulfide has been variously reported to range from 0.0007 mg to 0.20 mg/m^3 (0.0005—0.13 ppm) (Table 2). Little information is available on the odour detection limits for ambient hydrogen sulfide either under experimental field conditions or in general population exposures. The Task Group considered that a level of 0.007 mg/m^3 (0.005 ppm) averaged over 30 min should not produce odour nuisance in most situations. Some regulatory bodies may wish to adopt longer averaging times with appropriately adjusted concentration limits.

The best estimates available suggest that eye irritation may occur in man after several hours' exposure to hydrogen sulfide concentrations of 16—32 mg/m^3 (10.5—21.0 ppm). As occupational exposure guidelines, the Task Group recommended the adoption of 10 mg/m^3 (7 ppm) as a workshift time-weighted average value together with a short-term exposure limit of 15 mg/m^3 (10 ppm). The short-term limit should be determined as a 10-min or less averaged value. These limits should prevent eye irritation in workers which represents the earliest recognized toxic response in man.

Annex tables 1 and 2 contain various national standards or recommendations for ambient air quality and occupational exposure limits for hydrogen sulfide. As can be seen, there is considerable consensus regarding occupational exposure limits and the recommendations of the Task Group are generally consistent with the national values. There is less agreement regarding ambient air quality standards, possibly because of different values placed on the nuisance value of odours.

REFERENCES

ADAMS, D. F., YOUNG, F. A., & LUHR, R. A. (1968) Evaluation of an odor perception threshold test facility. *Tappi*, **51**: 62A–67A.

ADELSON, L. & SUNSHINE, I. (1966) Fatal hydrogen sulfide intoxication. Report of three cases occurring in a sewer. *Arch. Pathol.*, **81**: 375–380.

AHLBORG, G. (1951) Hydrogen sulfide poisoning in shale oil industry. *Am. Med. Assoc. ind. Hyg. occup. Med.*, **3**: 247–266.

AIHA (1965) Hydrogen sulfide. *Anal. Abstr.*, **12**: 2207.

ALEXANDER, M. (1974) Microbial formation of environmental pollutants. *Adv. appl. Microbiol.*, **18**: 37–40.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (1972) *Air sampling instruments for evaluation of atmospheric contaminants*, 4th ed., Cincinnati., ACGIH.

ANDERSSON, G., BROSSET, C., & GRENNFELT, P. (1974) The stability of emitted odorous compounds in the atmosphere. In: Turk, A., Johnston, J. W. Jr, & Moulton, D. G., ed. *Human response to environmental odors*, New York, Academic Press.

ANONYMOUS (1952) Four workers overcome by hydrogen sulfide when digging in marshy land. *Occup. Health*, **12**: 39.

BAMESBERGER, W. L. & ADAMS, D. F. (1969) Improvements in the collection of hydrogen sulfide in cadmium hydroxide suspension. *Environ. Sci. Technol.*, **3**: 258–261.

BAMESBERGER, W. L. & ADAMS, D. F. (1969) Field comparison of the coulometric, colorimetric, and lead acetate tape analysis methods for sulfur-containing gases. *Tappi*, **52**: 1302–1306.

BEASLEY, R. W. R. (1963) The eye and hydrogen sulfide. *Br. J. ind. Med.*, **20**: 32–34.

BECK, J. F., CORMIER, F. & DONINI, J. C. (1979) The combined toxicity of ethanol and hydrogen sulfide. *Toxicol. Lett.*, **3**: 311–313.

BECKER, W. J. (1979) On H_2S emission and immission measuring techniques. *Staub-Reinhalt. Luft*, **39**: 169–174.

BERUASHVILI, S. A. (1977) Skin irritant effect of water containing hydrogen sulfide and results of population studies. In: *Annual Report of the Natadse Institute of Sanitary and Hygiene. Tbilisi*, Vol. XII, pp. 60–63.

BERUASHVILI, S. A. (1979) The long-term effects of the thermal water, used for the hot water-supply. In: *Proceedings of the 4 Conferences of the Young Scientists and Specialists on the Problem "Environmental Health", Baku*, pp. 3–5.

BLACK, M. S., HERBST, R. P., & HITCHCOCK, D. R. (1978) Solid absorbent preconcentration and gas chromatographic analysis of sulfur gases. *Anal. Chem.*, **50**: 848–851.

BROWN, K. E. (1969) Some toxicological problems in a rubber industry. *Med. J. Aust.*, **1**: 534–538.

BURNETT, W. W., KING, E. G., GRACE, M., & HALL, W. F. (1977) Hydrogen sulfide poisoning: review of 5 years' experience. *Can. Med. Assoc. J.*, **117**: 1277–1280.

CALIFORNIA AIR RESOURCES BOARD (1970) *Ambient air quality standards*, Sacramento, California, California Air Resources Board, pp. 69–75.

CAMPBELL, C. L., DAWES, R. K., DEOLALKAR, S., & MERRITT, M. C. (1958) Effect of certain chemicals in water on the flavor of brewed coffee. *Food Res.*, **23**: 575–579.

COOPER, R. C., JENKINS, D., & YOUNG, L. Y. (1976) *Aquatic microbiology laboratory manual*, Texas, University of Texas.

DENMEAD, C. F. (1962) Air pollution by hydrogen sulfide from a shallow polluted tidal inlet, Auckland, New Zealand. In: *Clean Air Conference, Sydney, Australia.*

ELKINS, H. B. (1939) Toxic fumes. *Ind. Med.*, **8:** 426–432.

ESPACH, R. H. (1950) Sources of hydrogen sulfide in Wyoming. *Ind. Eng. Chem.*, **42:** 2235–2237.

EVANS, C. L. (1967) The toxicity of hydrogen sulfide and other sulfides. *Quart. J exp. Physiol.*, **52:** 231–248.

FEDERAL MINISTER FOR HOME AFFAIRS (1974) [*Technical instructions for prevention of air pollution,* issued 28 August 1974]. pp. 175 (in German).

FUKUI, S , NAITO, S., KANEKO, M., & KANNO, S. (1967) [Hygienic chemical studies on public nuisance by injurious gases. X. Improvement of absorption mixture and examination of the methylene blue method for determination of hydrogen sulfide.] *Hyg. Chem.*, **13:** 16–21 (in Japanese).

GILARDI, E. F. & MANGANELLI, R. M. (1963) A laboratory study of a lead acetate-tile method for the quantitative measurement of low concentrations of hydrogen sulfide. *Air Pollut. Control Assoc.*, **13:** 305–309.

GOLDMAN, F. H., COLEMAN, A. L., ELKINS, H. B., SCHRENK, H. H., & SMUCKER, C. A. (1940) II. Report of sub-committee on chemical methods in air analysis. Sampling and sampling devices. *Am. public Health Assoc. Year Book,* pp. 92–98.

GOLDMAN, F. H., COLEMAN, A. A., ELKINS, H. B., & SCHRENK, H. H. (1943) II. Report of sub-committee on chemical methods in air analysis. Cadmium and hydrogen sulfide. *Am. J. public Health,* **33:** 862–864.

GRANAT, L., RODHE, H., & HALLBERG, R. O. (1976) The global sulfur cycle. In: Svensson, B. H. & Söderlund, R., ed. *Nitrogen, phosphorus and sulfur — global cycles, SCOPE report 7. Ecol. Bull. (Stockholm),* **22:** 89–134.

HAGGARD, H. W. (1925) The toxicology of hydrogen sulfide. *J. ind. Hyg.*, **7:** 113–121.

HURWITZ, L. J. & TAYLOR, G. I. (1954) Poisoning by sewer gas with unusual sequelae. *Lancet,* **1:** 1110–1112.

ILO (1971) Tanning, leather finishing. In: *Encyclopedia of Occupational Health and Safety,* 2nd ed., Geneva, International Labour Office, pp. 1381–1382.

ILO (1971—72) Hydrogen sulfide. In: *Encyclopedia of Occupational Health and Safety,* 2nd ed., Geneva, International Labour Office, pp. 697–699.

INSTITUTE OF HYGIENE & EPIDEMIOLOGY (1978) [*Air pollution by H_2S at Boom, Belgium.*] Brussels, pp. 1–17 (L.V.M. BO.77) (in Dutch).

ISO (1978) *Proposal to the SC3 for the determination of hydrogen sulfide in air,* Geneva, International Organization for Standardization, 8 pp. (ISO/TC 146/SC3/WG3 N 2E (revised), 1978-06-14).

INTERSOCIETY COMMITTEE (1977 a) Tentative method of analysis for hydrogen sulfide content of the atmosphere. In: Katz, M., ed. *Methods of air sampling & analysis,* 2nd ed., pp. 676–681.

INTERSOCIETY COMMITTEE (1977 b) Tentative method of gas chromatographic analysis for sulfur-containing gases in the atmosphere (automatic method with flame photometer detector). In: Katz, M., ed. *Methods of air sampling & analysis,* 2nd ed., pp. 722–737.

JACOBS, M. B. (1965) Recommended standard method for continuing air monitoring for hydrogen sulfide. Ultramicrodetermination of sulfides in the air. *J. Air Pollut Control Assoc.*, **15:** 314–315.

JOHNSON, B. A. (1972) The evaluation of gas detector tube systems: hydrogen sulfide. *Am. Ind. Hyg. Assoc. J.*, **33:** 811–812.

JUNGE, C. E. (1963) *Air chemistry and radioactivity,* New York, Academic press, pp. 68–69 (International Geophysics series Vol. 4).

KAIPAINEN, W. J. (1954) Hydrogen sulfide intoxication. Rapidly transient

changes in the elcctrocardiogram suggestive of myocardial infarction. *Ann. Med. intern. Fenn.*, **43**: 97–101.
KATZ, M. (1977) *Sulfur and its inorganic derivatives in the Canadian environment*, Ottawa, Associate Committee on Scientific Criteria for Environmental Quality, National Research Council of Canada, pp. 21–68 (Publ. No. NRCC 15015).
KELLOGG, W. W., CADLE, R. D., ALLEN, E. R., LAZRUS, A. L., & MARTELL, E. A. (1972) The sulfur cycle. Man's contribution as compared to natural sources of sulfur compounds in the atmosphere and oceans. *Science*, **175**: 587–596.
KEMPER, F. D. (1966) A near-fatal case of hydrogen sulfide poisoning. *Can. Med. Assoc. J.*, **94**: 1130–1131.
KLEINFELD, M., GIEL, C., & ROSSO, A. (1964) Acute hydrogen sulfide intoxication; an unusual source of exposure. *Ind. Med. Surg.*, **33**: 656–660.
KOSMIDER, S., ROGALA, E., & PACHOLEK, A. (1967) Electrocardiographic and histochemical studies of the heart muscle in acute esperimental hydrogen sulfide poisoning. *Arch. Immunol. ther. Exp.*, **15**: 731–740.
KRASOVITSKAYA, M. L., MALJAROVA, L. K. & ZAPOROŻEC, T. S. (1965) [Contamination of the air in the area of a refinery and petrochemical plant.] *Gig. i Sanit.*, **4**: 103–105 (in Russian).
LARSON, C. P., REBERGER, C. C., & WICKS, M. J. (1964) The purple brain death. *Med. Sci. Law*, **4**: 200–202.
LAWRENCE BERKELEY LABORATORY (1976) *Instrumentation for environmental monitoring: air pollution. Ambient air monitor, Multi-component monitoring system for air pollution and SO_2 monitor*, California, University of California.
LEHMANN, K. B. (1892) [Experimental studies on the effect on the body of gases and vapours of technical and hygienic importance, Part V, hydrogen sulfide.] *Arch. Hyg.*, **14**: 135–189 (in German).
LEICHNITZ, K. (1977) Air analyses by means of long-term detector tubes. *Dräger Rev.*, **40**: 9–17.
LEONARDOS, G., KENDALL, D., & BARNARD, N. (1969) Odor threshold determination of 53 odorant chemicals. *J. Air Pollut. Control Assoc.*, **19**: 91–95.
LEVAGGI, D. A., SIU, W., & FELDSTEIN, M. (1972) Continuous determination of H_2S in the PPB range by a specific colorimetric method. In: *Proceedings of the Technicians International Congress, N.Y.*, pp. 65–67.
LINDVALL, T. (1970) On sensory evaluation of odorous air pollutant intensities. Measurements of odor intensity in the laboratory and in the field with special reference to effluents of sulfate pulp factories. *Nord. Hyg. Tidskr.*, **51** (suppl.): 36–39.
MACALUSO, P. (1969) Hydrogen sulfide. In: Mark, H. F., McKetta, J. J., & Othmer, D. F., ed. *Encyclopedia of chemical technology*, 2nd ed., N.Y., John Wiley & Sons.
McANALLEY, B. H., LOWRY, W. T., OLIVER, R. D., & GARRIOTT, J. C. (1979) Determination of inorganic sulfide and cyanide in blood using specific ion electrodes: application to the investigation of hydrogen sulfide and cyanide poisoning. *J. anal. Toxicol.*, **3**: 111–114.
McCABE, L. C. & CLAYTON, G. D. (1952) Air pollution by hydrogen sulfide in Poza Rica, Mexico. An evaluation of the incident of Nov. 24, 1950. *Am. Med. Assoc. Arch. ind. Hyg. occup. Med.*, **6**: 199–213.
McGRAW-HILL ENCYCLOPEDIA OF SCIENCE & TECHNOLOGY (1960) Heavy water, New York, McGraw-Hill Book Co. Inc., pp. 382–383.
McKEE, J. E. & WOLF, H. W. (ed.) (1971) *Water quality criteria*, 2nd ed., California, The Resources Agency of California, State Water Resources Control Board, 584 pp. (Publ. No. 3-A).
MERCADO, S. G. (1975) Geothermo-electric project of Cerro-Prieto: contamination and basic protection. In: *Proceedings of the Second UN*

Symposium on the Development and Use of Geothermal Resources, San Francisco, CA, pp. 1385–1393.

MILBY, T. H. (1962) Hydrogen sulfide intoxication. Review of the literature and report of unusual accident resulting in two cases of nonfatal poisoning. *J. occup. Med.*, **4**: 431–437.

MINER, S. (1969) *Preliminary air pollution survey of hydrogen sulfide. A literature review*, Raleigh, NC, National air pollution control adm. (Publ. No. APTD 69–37).

MINSTER, J. T. (1963) Meteorology. Atmospheric hydrogen sulfide concentrations during the London fog of December 1962. *Nature (Lond.)*, **199**: 474–475.

MITCHELL, C. W. & DAVENPORT, S. J. (1924) Hydrogen sulfide literature. *Public health Rep.*, **39** (1): 1–13.

NATIONAL RESEARCH COUNCIL, USA (1979) *Subcommittee on hydrogen sulfide. Hydrogen sulfide*, Baltimore, University Park Press.

NATUSCH, D. F. S., SEWELL, J. R., & TANNER, R. L. (1974) Determination of hydrogen sulfide in air — an assessment of impregnated paper tape methods. *Anal. Chem.*, **46** (3): 410–415.

NESSWETHA, W. (1969) [Eye lesions caused by sulfur compounds.] *Arbeitsmed. Sozialmed. Arbeitshyg.*, **4**: 288–290 (in German).

NEWILL, V. A. (1977) Air quality standards. In: Stern, A. D., ed. *Air pollution, Vol. V, Air quality management*, 3rd ed., New York, Academic Press.

NIOSH (1977) Occupational exposure to hydrogen sulfide, Cincinnati, OH, National Institute for Occupational Safety and Health (DHEW (NIOSH) publ. No. 77–158).

NYMAN, H. Th. (1954) Hydrogen sulfide eye inflammation — treatment with cortisone. *Ind. Med. Surg.*, **23**: 161–162.

PARÉ, J. P. (1966) A new tape reagent for the determination of hydrogen sulfide in air. *J. Air Pollut. Control Assoc.*, **16**: 325–327.

PEREGUD, Y. A., GERNET, Y. V., & BYKHOOSKAYA, M. S. (1971) *Rapid methods for the determination of noxious substances in the air*, Springfield, VA, US Dept. of Commerce, National Technical Information Service, (AD 126795).

PETRUN, N. M. (1966) [Penetration of hydrogen sulfide through skin and its influence on gaseous exchange and energy metabolism.] In: *Pharmacol. Toxicol.*, 2nd ed., Kiev, Publishing House "Health" ("Zdorovie"), pp. 247–250 (in Russian).

PODA, G. A. (1966) Hydrogen sulfide can be handled safely. *Arch. environ. Health*, **12**: 795–800.

ROBINSON, E. & ROBBINS, R. C. (1970) Gaseous sulfur pollutants from urban and natural sources. *J. Air Pollut. Control Assoc.*, **20**: 233–235.

ROLFE, K. A. (1980) The air-pollution aspects of geothermal power stations. *N.Z. energy J.*, **53**: 51–58.

RYAZANOV, V. A. (1962) Sensory physiology as basis for air quality standards. *Arch. environ. Health*, **5**: 480–494.

SANDERSON, H. P., THOMAS, R., & KATZ, M. (1966) Limitations of the lead acetate impregnated paper tape method for hydrogen sulfide. *J. Air Pollut. Control Assoc.*, **16**: 328–330.

SAVOLAINEN, H., TENHUNEN, R., ELOVAARA, R., & TOSSAVAINEN, A. (1980) Cumulative biochemical effects of repeated subclinical hydrogen sulfide intoxication in mouse brain. *Int. Arch. occup. environ. Health*, **46**: 87–92.

SAYERS, R. R., SMITH, N. A. C., FIELDNER, A. C., MITCHELL, C. W., JONES, G. W., YANT, W. P., STARK, D. D., KATZ, S. H., BLOOMFIELD, J. J., & JACOBS, W. A. (1925) *Investigation of toxic gases from Mexican and other high-sulfur petroleums and products.* Report by the Dept. of the Interior, Bureau of Mines, to the American Petroleum Institute,

Washington, DC, US Government Printing Office, pp. 59–80 (Bull. No. 231).
SHAW, J. A. (1940) Rapid determination of hydrogen sulfide and mercaptan sulfur. In gases and in aqueous solutions. *Anal. Chem.*, **12**: 668–671.
SIMSON, R. E. & SIMPSON, G. R. (1971) Fatal hydrogen sulfide poisoning associated with industrial waste exposure. *Med. J. Aust.*, **1**: 331–334.
SIU, W., LEVAGGI, D. A., POTTER, L., MARTIN, R., & FELDSTEIN, M. (1971) Modifications to an H_2S tape sampler for increasing sensitivity and accuracy in H_2S sampling. *J. Air Pollut. Control Assoc.*, **21** (10): 636–638.
SMITH, R. P. & GOSSELIN, R. E. (1979) Hydrogen sulfide poisoning. *J. occup. Med.*, **21**: 93–97.
STEVENS, R. K., MULIK, J. D., O'KEEFFE, A. E., & KROST, K. J. (1971) Gas chromatography of reactive sulfur gases in air at the parts-per-billion level. *Anal. Chem.*, **43**: 827–831.
STINE, R. J., SLOSBERG, B., & BEACHAM, B. E. (1976) Hydrogen sulfide intoxication. A case report and discussion of treatment. *Ann. intern. Med.*, **85** (6): 756–758.
SUSMAN, J. L., HORNIG, J. F., THOMAE, S. C., & SMITH, R. P. (1978) Pulmonary excretion of hydrogen sulfide, methanethiol, dimethyl sulfide and dimethyl disulfide in mice. *Drug Chem. Toxicol.*, **1**: 327–338.
THIELE, V. von (1979) [Experimental investigations to determine an odour threshold value for hydrogen sulfide.] *Staub-Reinhalt. Luft*, **39**: 159–160 (in German).
THOM, N. G. & DOUGLAS, R. T. (1976) A study of hydrogen sulfide levels in the geothermal areas of Rotorua, New Zealand. In: *Fourth International Clean Air Congress*, Tokyo, pp. 565–568.
THOMPKINS, F. C., Jr & BECKER, J. H. (1976) *An evaluation of portable, direct-reading H_2S meters, Research Report*, Cincinnati, OH, National Institute for Occupational Safety and Health (DHEW (NIOSH) Publ. No. 77–137).
UNITED STATES PUBLIC HEALTH SERVICE (1941) *Hydrogen sulfide: its toxicity and potential dangers*, Cincinnati, OH, US Dept of Health, Education, and Welfare, Public Health Service, Division of Air Pollution, pp. 684–693.
UNITED STATES PUBLIC HEALTH SERVICE (1964) *The air pollution situation in Terre Haute, Indiana with special reference to the hydrogen sulfide incident of May – June, 1964*, Cincinnati, OH, US Dept of Health, Education, and Welfare, Public Health Service Division of Air Pollution, pp. 1–28.
WEAST, R. C., ed. (1977–78) Physical constants of inorganic compounds. In: *Handbook of chemistry and physics*, 58th ed., Cleveland, OH, USA, CRC Press.
WEST, P. W. (1970) Analytical methods for the study of air pollution. *Pure appl. Chem.*, **21**: 437–447.
WINDHOLZ, M., ed. (1976) Hydrogen sulfide. In: *Merck index*, 9th ed., Rahway, N. J., Merck & Co. Inc., pp. 633–634.
WINNEKE, VON G., KOTALIK, J., KELDENICH, H.-O., & KASTKA, J. (1979) [The identification of hydrogen sulfide under laboratory and field conditions.] *Staub-Reinhalt. Luft*, **39**: 156–159 (in German).
YANT, W. P. (1930) Hydrogen sulfide in industry, occurrence, effects, and treatment. *Am. J. public Health*, **20**: 598–608.

Annex

Annex table 1. Ambient air quality standards for hydrogen sulfide from selected countries

Country	Long-term			Short-term			Reference
	mg/m³	ppm	averaging time (h)	mg/m³	ppm	averaging time (min)	
Bulgaria	0.008	0.006	24	0.008	0.006	30	Newill (1977)
China	—	—	—	0.01	0.007	20	Official Communication[d]
Czechoslovakia	0.008	0.006	24	0.008	0.006	30	Newill (1977)
German Democratic Republic	0.008	0.006	24	0.015	0.011	10—30	Newill (1977)
Germany, Federal Republic of	0.005	0.004	24	0.01	0.007	30	Federal Minister for Home Affairs (1974)
Hungary	0.008[a]	0.006	24	0.008[a]	0.006	30	Newill (1977)
Hungary	0.15	0.11	24	0.3	0.21	30	Newill (1977)
Israel	0.045	0.032	24	0.15	0.11	30	Newill (1977)
Philippines	—	—	—	0.3	0.21	30	UNEP/IRPTC[e]
Poland	0.008[b]	0.006	24	0.008[b]	0.006	30	UNEP/IRPTC[e]
Poland	0.02[c]	0.014	24	0.06[c]	0.04	20	UNEP/IRPTC[e]
Romania	0.01	0.007	24	0.03	0.02	30	Newill (1977)
Spain	0.004	0.003	24	0.01	0.007	30	Newill (1977)
USSR	0.008	0.006	24	0.008	0.006	30	Newill (1977)
Yugoslavia	0.008	0.006	24	0.008	0.006	30	Newill (1977)

[a] For highly protected and protected areas.

[b] For especially protected areas, sanatoria, health resorts, sanctuaries, and national parks.

[c] For protected areas, towns, and villages.

[d] Official communication from the Institute of Health, Chinese Academy of Medical Sciences.

[e] Private communication.

Note by the WHO Task Group

Ambient Air Quality Standards have been set according to different criteria in different countries, and these criteria may include, but are not necessarily limited to the assessment of health effects. Moreover, the limits themselves may have different meanings such as the maximum acceptable level or the permissible level over a 10 to 30-min averaging time, etc.

Annex table 2. Occupational exposure standards for hydrogen sulfide from selected countries[a]

Country	mg/m³	ppm	Standard type
Australia	15	10	8-h TWA[b]
Belgium	15	10	8-h TWA[b]
Bulgaria	10		ceiling
China	10		ceiling
Czechoslovakia — average	10		shift[c] TWA[b]
maximum	20		10-min STEL[d]
Finland	15	10	8-h TWA[b]
German Democratic Republic — average	15		8.75-h TWA [b]
short-term	15		30-min STEL[d]
Germany, Federal Republic of	15	10	8-h TWA[b]
Hungary	10		8-h TWA[b]
Italy	10		shift[c] TWA[b]
Japan	15	10	shift[c] TWA[b]
Netherlands	15	10	shift[c] TWA[b]
Poland	10		8-h TWA[b]
Romania — average	10		shift TWA[b]
maximum	15		ceiling
Sweden	15	10	shift[c] TWA[b]
Switzerland	15	10	8 to 9-h TWA[b]
USSR	10		less than 30-min STEL[d]
USA — occupational standard		20	ceiling except for one 10-min peak less than 50 ppm
ACGIH	15	10	8-h TWA[b]
	21	15	15-min STEL[d]
Yugoslavia	10	7	ceiling

[a] Abstracted from: ILO (1977).
[b] TWA = time-weighted average value.
[c] = a time-weighted value averaged over the entire shift or workday.
[d] STEL = short-term exposure limit.