Hydrogen Sulfide

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- Name: Hydrogen sulfide
- Chemical Abstracts Service Registry Number: 7783-06-4, Other Registry Number: 11144-15-3, Related Registry Number: 13465-07-1 (H_2S_2)
- Synonyms: Acide sulfhydrique, Acide sulphhydrique, Dihydrogen monosulfide, Dihydrogen sulfide, EINECS 231-977-3, FEMA No. 3779, HSDB 576, Hydrogen sulfure, Hydrogen sulfide (H_2S), Hydrogen sulfuric acid, Hydrogen sulphide, Hydrogene sulfure, Hydrogene sulphure, Hydrosulfuric acid, Idrogeno solforato, RCRA waste number U135, Schwefelwasserstoff, Sewer gas, Siarkowodor, Stink DAMP, Sulfur hydride, Sulfureted hydrogen, UN 1053, UNII-YY9FVM7NSN, Zwavelwaterstof.
- Molecular Formula: H_2S
- Chemical Structure:

Background (Significance/History)

Hydrogen sulfide has been known since early times and its chemistry has been studied since the 1600s. In the nineteenth century, Petrus Johannes Kipp invented a convenient device for the generation of a variety of gases in which a liquid and solid were the reagents. This generator was especially useful for the generation of hydrogen sulfide and hydrogen.

Uses

Hydrogen sulfide is used as a reagent and an intermediate in the preparation of other reduced sulfur compounds. It is used in the production of thioorganic compounds including methanethiol, ethanethiol, and thioglycolic acid. Hydrogen sulfide is used in the manufacturing of alkali metal sulfides such as sodium hydrosulfide and sodium sulfide, which are used in the degradation of biopolymers. It is important in analytical chemistry, in the qualitative inorganic analysis of metal ions. Hydrogen sulfide is a precursor to metal sulfides and this is widely exploited. For example, waters or gases contaminated by hydrogen sulfide can be cleaned with metal sulfides. In addition, endogenous hydrogen sulfide has therapeutic applications. Many new technologies have been developed to detect endogenous hydrogen sulfide production, and novel hydrogen sulfide-delivery compounds have been invented to aid therapeutic intervention of diseases related to abnormal hydrogen sulfide metabolism. It should be noted that this therapeutic approach is just now being investigated.

Environmental Fate and Behavior

Routes and Pathways, and Relevant Physicochemical Properties (e.g., Solubility, Pow, Henry Constant)

Hydrogen sulfide is a colorless, flammable compressed liquid gas with a characteristic odor of rotten eggs. The solubility of hydrogen sulfide in water is 3980 mg l^{-1} at 20 °C and it is soluble in certain polar organic solvents, notably methanol, acetone, propylene carbonate, sulfolane, tributyl phosphate, various glycols and glycol ethers, gasoline, kerosene, crude oil, and carbon disulfide. The calculated vapor pressure at 21.9 °C is 1929 Pa. Boiling point and melting point of the substance are −60.33 °C and −85.49 °C, respectively. Based on the estimated Henry's law constant of 468 atm mol^{-1} for hydrogen sulfide, volatilization from water and soil is high.

Partition Behavior in Water, Sediment, and Soil

Considering the boiling point of hydrogen sulfide, when it is spilled onto soil much will evaporate. Since hydrogen sulfide is very soluble in water, the presence of water in soil, or falling as precipitation at the time of the spill, may contribute to movement in the soil. If the soil surface is saturated with moisture at the time of the spill, for example after a rainfall, the spilled chemical will run off and/or evaporate away.

Environmental Persistency (Degradation/Speciation)

The lifetime of hydrogen sulfide is affected by ambient temperature and other atmospheric variables including sunshine, humidity, and the presence of other pollutants. As an example, the decreased levels of hydroxide and decreased temperatures in northern regions in winter increase the residence time of hydrogen sulfide in air. Hydrogen sulfide after released into the atmosphere will behave like many other gaseous pollutants and be dispersed and eventually removed. Residence times in the atmosphere are about 1 day to more than 40 days, depending upon latitude, season, and atmospheric conditions. Oxidation–reduction reactions that oxidize hydrogen sulfide to elemental sulfur were performed in the presence of microorganisms in soil and water. Hydrogen sulfide does not absorb solar radiation reaching the troposphere, therefore it does not undergo photolysis or react photochemically with oxygen. The primary chemical transformation of hydrogen sulfide in the atmosphere is oxidation to sulfur dioxide and sulfates by oxygen-containing radicals.

Long Range Transport

The boiling point of −60.33 °C suggests that when hydrogen sulfide is spilled onto soil, much will evaporate.

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Bioaccumulation and Biomagnifications

Hydrogen sulfide does not have bioaccumulation or food chain contamination potential.

Exposure and Exposure Monitoring

Routes and Pathways (Including Environmental Release)

Hydrogen sulfide is produced naturally and also as a result of human activities. It is one of the principal compounds involved in the natural cycle of sulfur in the environment, and natural sources account for about 90% of total hydrogen sulfide in the environment. Hydrogen sulfide as a by-product of many industrial operations (e.g., coking and the hydrodesulfurization of crude oil and of coal), can result in its release to the environment.

Human Exposure

Human exposure to hydrogen sulfide may occur from exogenous sources and also its endogenous production. The general populace may be exposed to hydrogen sulfide through inhalation of ambient air in communities located near certain types of industrial sites. Also, accidental release of natural gas wells during drilling operations near residential areas can result in human exposure to hydrogen sulfide. The majority of occupational exposure to hydrogen sulfide occurs where it is produced, used, or generated, including its occurrence in petroleum, natural gas, sewer gas, soil, and as a by-product of chemical reactions.

Environmental Exposure (Monitoring Data in Air, Water, Sediment, Soil and Biota)

Hydrogen sulfide concentrations in ambient air range from 0.11–0.33 ppb and in urban areas are generally <1 ppb, although much higher concentrations (often exceeding 90 ppb) have been detected in communities located near natural sources or industries releasing hydrogen sulfide. In undisturbed anoxic sediment, hydrogen sulfide levels may be as high as 100 ppb and in disturbed sediments may range from 1–30 ppb. No data are available for hydrogen sulfide concentrations in soil. Hydrogen sulfide readily evaporates from surface water, and no data are available for hydrogen sulfide levels in drinking water.

Toxicokinetics

Hydrogen sulfide is primarily absorbed via the lungs. It can also be absorbed through the skin and gastrointestinal tract, but there are limited data for these routes. Absorbed hydrogen sulfide is rapidly distributed throughout the body and enters the circulation and partly dissociates to HS^-. It is distributed to the brain, liver, kidney, small intestine, and pancreas. Hydrogen sulfide is metabolized through three pathways: methylation, oxidation, and reactions with metalloproteins or disulfide-containing proteins, and sulfate metabolites are excreted in the urine. In the bloodstream, the gas is converted to alkali sulfide and the hydrosulfide radical is excreted by the lungs and in urine. Part of the sulfide is also oxidized to thiosulfate and sulfate.

Mechanism of Toxicity

Toxicity of hydrogen sulfide is most likely related to inhibition of metal-containing enzymes such as cytochrome oxidase, the final enzyme of the mitochondrial respiratory chain, and carbonic anhydrase. Therefore, hydrogen sulfide affects cellular energy production and respiration. Mucous membranes and tissues with a high oxygen demand, like nervous and cardiac tissues are most susceptible tissues in exposure to hydrogen sulfide. In addition, sulfide also seems to act on the respiratory drive through other mechanisms such as suppression of synaptic activity, inhibition of monoamine oxidase, a direct action on respiratory centers in the brain, and stimulation of the glutamate receptors in the brain. The hydrosulfide anion also forms a complex with methemoglobin and creates sulfmethemoglobin. On the other hand, hydrosulfide can be produced endogenously, particularly in mammalian cells, through an enzymatic pathway and in a smaller part via a nonenzymatic pathway. Among enzymes involved in hydrosulfide production, cystathionine-synthase and cystathionine-lyase have been investigated extensively; both use vitamin B_6 as a cofactor. Captopyruvate sulfurtransferase along with cysteine aminotransferase are involved in transsulfuration and reverse transsulfuration pathways in different capacities and utilize specific substrates. Of course, the regulation mechanisms for the expression and activities of these hydrosulfide-generating enzymes under physiological or pathophysiological conditions needs more research. These enzymes are differentially expressed in neuronal, immune, cardiovascular, renal, gastrointestinal, reproductive, respiratory, liver, and endocrine systems and affect the functions of these systems through production of hydrosulfide. Meanwhile, different molecular targets, such as different ion channels and signaling proteins, mediate physiological functions of hydrogen sulfide. Alternations of hydrosulfide metabolism lead to an array of pathological disturbances in the form of hypertension, diabetes, cirrhosis, atherosclerosis, heart failure, inflammation, sepsis, erectile dysfunction, asthma, and neurodegenerative disease.

Acute and Short-Term Toxicity (To Include Irritation and Corrosivity)

Animal

The inhalation LC_{50} for hydrogen sulfide in mouse is 634 ppm per hour and inhalation exposure of rats during 4 h to this compound gave an LC_{50} of 444–501 ppm. Acute effects of hydrogen sulfide in animals included cytotoxic lesions and edema in the lungs with depression of the activity of cytochrome oxidase, increase of amino neurotransmitter levels in the respiratory centers in the brainstem, and detectable histological lesions in nasal epithelium. Hydrogen sulfide leads to irritation of the eyes in laboratory animals (after a few hours exposure to 100–300 ppm), and effects on the mucous membranes of the throat and nasal cavity are also reported in laboratory animals. No data on skin irritation and sensitization are available.

Human

The targets of acute toxicity of hydrogen sulfide in humans are the nervous system and the lung. Temporary unconsciousness and severe effects on the respiratory system with or without neurological changes are the main symptoms. 'Gas eye' is a superficial inflammation of the cornea and conjunctiva from the irritant effect of hydrogen sulfide. The hydrogen sulfide is irritating to the respiratory tract, resulting in rhinitis, pharyngitis, laryngitis, bronchitis, and pneumonia. No information on skin irritation and sensitization is available for acute toxicity with hydrogen sulfide in humans. In addition, hydrogen sulfide toxicity is one of the most unusual and reliable toxidromes in medical toxicology, include 'knockdown' (acute central neurotoxicity), pulmonary edema, conjunctivitis, and odor perception followed by olfactory paralysis. These symptoms always occur singly or in combination regardless of the susceptibility or characteristics of the victim. Secondary symptoms and signs of hydrogen sulfide toxicity include headache and short-term cognitive changes (such as short-term memory loss), seizure disorders, and gastrointestinal symptoms. Of course, the prevalence and profile of these secondary effects is much weaker than that of the reported cardinal effects.

Chronic Toxicity

Animal

Inhalation exposure to hydrogen sulfide at 0, 10, 30, and 80 ppm (6 h day^{-1}) for 70 consecutive days in the rat has been reported to cause bilaterally, symmetrical olfactory neuronal loss and basal cell hyperplasia in the mucosa lining the dorsal medial meatus, the nasal septum, dorsal wall of the nasal cavity, and margins of the ethmoturbinates. No effects were found after exposure to 10 ppm but these findings increased with concentration (50% effect at 30 ppm and 70% effect at 80 ppm). Based on available animal evidence, injury to the cerebral cortex, cerebellum, and possibly the brainstem and spinal cord might occur at concentrations approaching those of humans.

Human

Epidemiological studies of humans who have been exposed to hydrogen sulfide for a long time showed eye irritation, hazy sight and photophobia (at concentrations from 1–5 mg m^{-3}), lung function impairment, effects on enzyme levels in reticulocytes, and erythrocyte protoporphyrin concentration (at concentrations between 0.07 and 7.2 mg m^{-3} as 8-h TWA). Chronic effects of higher sublethal exposure of hydrogen sulfide may be neurotoxicity and peripheral neuropathy. Short-term exposure to high concentrations of hydrogen sulfide may equate to longer term exposure to lower concentrations that is particularly misleading because toxicity for this agent is mostly driven by concentration.

Reproductive Toxicity

Based on moderate evidence, hydrogen sulfide has a low developmental and reproductive toxicity, although there are no published reports of teratogenesis attributable to hydrogen sulfide exposure.

Genotoxicity

Hydrogen sulfide exposure was associated with an increased risk of chromosome aberrations in chemical workers but other factors may be involved in these multiply exposed cohort populations. No studies were located regarding genotoxicity in humans with hydrogen sulfide and no mutagenicity was observed with this compound in Ames assays.

Carcinogenicity

The International Agency for Research on Cancer (IARC) has evaluated that there is no support for a carcinogenic effect of hydrogen sulfide.

Clinical Management

Clinical management is symptomatic and supportive. Removal from inhalational exposure is the first step. Airway management is recommended in patients with mental status depression or respiratory distress. Irrigate eyes if there is evidence of irritation and wash skin if there is evidence of irritation. Consider the administration of sodium nitrite for patients with severe poisoning who are already receiving good supportive care.

Ecotoxicology

Freshwater/Sediment Organisms Toxicity

Marine Organisms Toxicity

Hydrogen sulfide is toxic for aquatic organisms. It is very highly toxic for crustaceans, fish, and zooplankton and also slightly toxic for nematodes and flatworms.

Exposure Standards and Guidelines

Based on OSHA standards, permissible exposure limit: Table Z-2 Acceptable Ceiling Concentration: 20 ppm. Threshold Limit Values: 8-h Time Weighted Average (TWA): 10 ppm; 15 min Short-Term Exposure Limit: 15 ppm. Based on NIOSH recommendations, Recommended Exposure Limit: 10 min Ceiling Value: 10 ppm (15 mg m^{-3}); Immediately Dangerous to Life or Health: 100 ppm; Emergency Response Planning Guidelines (ERPG): ERPG(1) 0.1 ppm (no more than mild, transient effects), ERPG(2) 30 ppm (without serious, adverse effects), and ERPG(3) 100 ppm (not life threatening) for up to 1 h exposure.

Miscellaneous

Hydrogen sulfide is also used in the separation of deuterium oxide (known as heavy water) from normal water, by a process called the Girdler Sulfide process.

See also: Volatile Organic Compounds; Occupational Toxicology; Cyanide; Cyanogen Chloride; Mitochondrial Toxicity.

Further Reading

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