Toxicology of Hydrogen Sulfide

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Introduction: Hydrogen sulfide (H_2S) is a colorless gas. At low concentrations it has an obnoxious odor similar to rotten eggs. It is soluble in water. It is produced in nature primarily through the decomposition of organic matter by bacteria. It is a constituent of natural gas, petroleum, sulfur deposits, volcanic gases and sulfur springs. Hydrogen sulfide is oxidized by photo chemically-generated free radicals, especially hydroxyl radicals. It has a half-life in air ranging from 12 to 37 hours, but varies depending upon photoactive pollutants and temperature. The half-life in air during very cold and dry winter conditions can exceed 37 hours. In the United States about 125,000 employees in 73 industries are potentially exposed to Hydrogen sulfide. However, domestic exposure can occur from various sources as follows: Ambient air near petroleum refineries, and sewage treatment plants; sewers (sewer gas); hot water tanks; and septic tanks.

Sewer gas refers to the odor associated with sewers, waste treatment plants and septic tanks. Sewer gas contains hydrogen sulfide and reduced sulfur compounds, such as methyl and dimethyl sulfide, ethyl and diethyl sulfide. These organo-sulfur compounds add to the toxicity of the hydrogen sulfide in the sewer gas.

The most dangerous aspect of hydrogen sulfide results from olfactory accommodation and/or olfactory paralysis. This means that the individual can accommodate to the odor and is not able to detect the presence of the chemical after a short period of time. Olfactory paralysis occurs in workers who are exposed to 150 ppm or greater. This occurs rapidly, leaving the worker defenseless. Unconsciousness and death have been recorded following prolonged exposure at 50 ppm.

Hydrogen sulfide is a mitochondrial poison. Its action on mitochondria is similar to that of cyanide through inhibition of cytochrome oxidase (iron containing protein). This prevents the utilization of oxygen with an uncoupling of oxidative phosphorylation. In addition, hydrogen sulfide binds to hemoglobin in red blood cells interfering with oxygen transport.

Exposure to hydrogen sulfide occurs primarily by inhalation but can also occur by ingestion (contaminated food) and skin (water and air). Once taken into the body, it is rapidly distributed to various organs, including the central nervous system, lungs, liver, muscle, etc.

Odor and Concentrations: Subjective olfactory responses to various concentrations of hydrogen sulfide are summarized as follows:

0.02 ppm	No odor
0.13 ppm	Minimal perceptible odor
0.77 ppm	Faint, but readily detectable odor
4.6 ppm	Easily detectable odor, moderate odor
27.0 ppm	Strong, unpleasant odor, but not intolerable.

Physiological Responses to Acute Exposures: Physiological responses to acute exposure to hydrogen sulfide have been reported as follows:

10 ppm Beginning of Eye Irritation

50-100 ppm Slight conjunctivitis and respiratory tract irritation after one hour

100 ppm Coughing, eye irritation, loss of sense of smell after 2-15 minutes. Altered respiration, pain the eyes, and drowsiness after 15-30 minutes followed by throat irritation after one hour. Several hours exposure results in gradual increase in severity of symptoms and death may occur within the next 48 hours.

200-300 ppm Marked conjunctivitis and respiratory tract irritation after one hour exposure.

500-700 ppm Loss of consciousness and possibly death in 30 minutes to one hour of exposure.

700-1000 ppm Rapid unconsciousness, cessation of respiration, and death.

1000-2000 ppm Unconsciousness at once, with early cessation of respiration and death in a few minutes. Death may occur if individual is removed to fresh air at once.

OSHA and Other Agencies: Remember that OSHA permissible concentrations do not pertain to
Domestic Exposure situations. OSHA and other agency's regulations only pertain to the workplace for healthy adult males. Therefore, these regulations do not cover the more sensitive population, which includes the elderly, the very young and those with pre-existing illness. In addition, domestic exposure (in the home situation) is considered different than the workplace, because humans spend about 80 % of their time at home. Thus, the domestic exposure is many hours longer (168 hr x 0.8 = 134 hrs per week) versus occupational exposure (40 hours per week). It is common to divide the OSHA PEL by the ratio of number of hours per week 168 hrs) to the number of work hours per week (40), which comes to 4.2. The product of this division is considered to equate in home exposure to work exposure regulatory concentrations.

OSHA 10 ppm divided by 4.2 = 2.38 ppm. Additional practice is to divide this new figure by a Factor of 10 for precautionary reasons. Thus, in home exposures equivalent to OSHA 40 hour work week would then be 2.38 divide by 10 = 0.238 ppm.

OSHA General Industry PEL (permissible exposure level). 20 ppm ceiling for 10 minutes once, only if no other measurable exposure occurs; 50 ppm peak.

OSHA Construction Industry PEL: 10 ppm (or 15 mg/m^3) TWA (Time Weighted Average Over 8 hours per day)

ACGIH: 10 ppm (14 mg/m^3) TWA; 15 ppm, 21 mg/m^3 STEL (Short Term Exposure Level)

NIOSH REL: 10 ppm Ceiling for 10 minutes. (Recommended Exposure Level) **Symptoms of Exposure:** Apnea; coma; convulsions; irritate eyes; conjunctivitis pain; lacrimation; photophobia; corneal vesiculation; respiratory irritation; dizziness; headaches; fatigue; insomnia; GI disturbances; irregular heartbeats, drop in blood pressure.

Health Effects: Acute systemic toxicity; CNS effects; Irritation of eyes; lung irritation.

Chronic Low Level Exposures: The effects of chronic exposure to low concentrations of Hydrogen Sulfide mixed with other organo sulfur compounds have been reported as follows:

T**he Illinois Institute For Environmental Quality reported its findings on Hydrogen Sulfide Health Effects and Recommended Air Quality Standards in 1974.** The Illinois Institute summarized the literature on human health effects and their observations on the health effects in Illinois ambient air concentrations. In general the following was reported:

Concentration of H_2S	**Symptoms**
O.12 mg/m^3 (0.08 ppm)	Increased mental depression, dizziness and blurred vision.
0.45 mg/m^3 (0.32 ppm)	Increased incidence of nausea, loss of sleep, shortness of breath, and headaches
1.0-10 mg/m^3 (0.7-6.7 ppm)	Increased incidence of decreased corneal reflex (convergence and divergence
10-70 mg/m^3 (6.7-47 ppm)	Irritation of conjunctiva, fatigue, loss of appetite, insomnia.

The Illinois Institute recommended a standard for gaseous hydrogen sulfide of 0.015 mg/m^3 (0.01 ppm) to minimize adverse health effects from chronic exposure in urban air.

Kilburn KH and Warshaw RH. Hydrogen sulfide and reduced-sulfur gases adversely affect
neurophysiological functions. Toxicology and Industrial Health, Vol 11, pp. 185-19, 1995.

Ex-workers and neighboring residents (total of 35 individuals) were compared to 33 unexposed controls. The ex-workers and residents were exposed to hydrogen sulfide and other reduced-sulfur compounds emitted from a refinery. The concentrations of hydrogen sulfide and other reduced-sulfur compounds were monitored at ground level. Depending upon the day and year hydrogen sulfide concentrations ranged from a low of 10 ppb to 8.8 ppm. Reduced sulfur compounds (dimethylsulfide, mercarptans, carbon oxide sulfide) ranged from 2 ppb to 71 ppm.

Symptoms involving the respiratory tract (chest tightness, palpitations, chest pain, dry cough, cough with blood, dryness (mouth, nose, throat,), throat irritation, eye irritation, reduced sense of smell were greater in the exposed than the controls.

Neurological symptoms were also elevated over the controls. These included: dizziness, lightheadedness, loss of balance, lack of concentration, recent and long-term memory loss, mood unstableness, irritability, exhilaration.

Sleep disturbances were also noted in the exposed, which were: cannot fall asleep, wake frequently, sleep few hours, somnolence.

Skin symptoms were itching, dryness and redness.

General Symptoms were: headache, nausea, libido decrease, excess fatigue, indigestion, loss of appetite, lack of tolerance to alcohol.

Neurophysiological deficits were found in the exposed group: simple reaction time was increased; sway speed was faster, color discrimination was reduced and psychomotor speed was time was increased.

Profile Mood States (POMS) also showed abnormalities when compared to controls. There were increased scores for anger, depression, tension, confusion, fatigue and vigor.

The automatic (subconscious) parts of the neuro-axis were impaired. Impaired performance was accompanied by reduced perceptual motor speed.

The exposure to reduced-sulfur gases, predominantly hydrogen sulfide, was considered the most plausible explanation of the neurotoxic effects in this study.

Gaitonde UB, Sellar RH and O'Hare AE. Long-term exposure to hydrogen sulphide producing sub acute encephalopathy in a child. British Medical Journal. Vol 294, pp. 614, 1989.

This is a report on a 20-month old infant exposed for a year to 0.6 ppm hydrogen sulfide downwind from a burning tip gas ignition point for a colliery. The child had subacute necrotizing encephalopathy in the basal ganglia and white matter.

Chronic Reference Dose (RfD) Based upon animal studies and the child reported by Gaitonde et al, the U.S.E.P.A. has recommended **a RfD of 0.8 micrograms per cubic meter of air** for both subchronic and chronic human inhalation exposure. The RfD is that concentration at which no adverse health effects should occur. Concentrations above the RfD may result in adverse health effects, including neurotoxicity.

Conclusion: Chronic and subchronic exposure to low concentrations of hydrogen sulfide and other organosulfur compounds (reduced sulfur compounds) do cause long-term health problems in humans. These problems appear as various symptoms of the upper and lower respiratory tract, central nervous system, skin and eyes. The central nervous system symptoms are associated with permanent neurophysiological deficits. Injury to the central nervous system includes damage to the basal ganglia and white matter