



HYDROGEN SULFIDE

Identity

Name (parent)	Hydrogen sulfide
UN number	1053
CAS number	7783-06-4
Intervention value (AGW in mg/m ³)	50
Structure	H ₂ S

Occurrence

Chemical state (at 20°C)	Gas
Physical appearances	Under pressure (at 20°C >18.1 bar) colorless fluid
Industrial products	Sewers (product of anaerobic bacteria), crude oil, coal pits, chemical synthesis and metallurgy

Physicochemical properties

Molecular weight	34.1
Vapor pressure (mbar at 20°C)	18 800
Octanol/water partition coefficient (log Po/w)	0.5
Water solubility (in mg/L at 25 °C)	3000

Toxicokinetics (parent)

Uptake by inhalation	Hydrogen sulfide is readily taken up by inhalation and gas exposure can result in intoxication within minutes.
Uptake by skin absorption	Negligible
Uptake via gastrointestinal tract	Not relevant
Distribution	Quickly distributed throughout the body.
Metabolism	Oxidation by cytochrome oxidases; a major part of inhaled hydrogen sulfide was metabolized within 15 h [1]
Excretion via lungs	Low concentrations are eliminated in healthy subjects [4]. This substance is found in exhaled breath of patients with cystic fibrosis [5] and chronic pancreatitis [6].
Excretion via urine	Approx. 33 % is excreted within 15 h after exposure as calculated from [1]
Excretion via feces	Negligible
Elimination kinetics	Sulfides are in part used for protein synthesis but a surplus is readily eliminated as thiosulphate [4]. No specific kinetic patterns are reported.

Toxicodynamics

Mechanisms of toxicity	Irritating to eyes and conjunctiva. At higher exposures also irritating to mucous membranes, upper and lower airway, and also alveoli but only at high exposure. Asphyxiant due to interruption of the electron transport chain by inactivation of cytochrome oxidase aa3 caused by binding to ferric ion (Fe ³⁺) and thus inhibiting aerobic metabolism and decreasing production of ATP by oxidative phosphorylation. This causes an almost immediate loss of consciousness at high concentrations. Mild symptoms of intoxication include headache, muscle cramp, lung edema.
Classifications for carcinogenicity	Not classified
Classifications for reprotoxicity	Occupational exposure caused reproduction toxic effects (classification A: unconfirmed human reproductive hazard) [7]; classified in The Netherlands for effects on fertility (cat 3: possible hazard for reduced fertility) and for developmental effects (cat 3: possible hazard for adverse effects on the unborn child) [8]
Classifications for sensitizing properties	Not classified

**HYDROGEN SULFIDE****Biological monitoring**

Biomarkers	Thiosulphate in urine (detected as bis(pentafluorobenzyl) disulfide or as bromobimane complex)
Molecular weight	158.11
Involved enzymatic metabolism	cytochrome oxidase aa3
Biological material	urine
Type of sample	spot sample
Sampling strategy	Collection of samples within 15 hours after exposure [1]
Excretion pattern	Urinary excretion follows zero kinetics. After 17 hours, the urinary concentration is equal to those in controls [1].
Materials	polypropylene bottles with screw cap
Transportation	at ambient temperature if within 3 days [2]
Storage	-20°C [2]
Stability	3 days at room temperature and up to 18 month at -20°C [2]
Pretreatment	An aliquot of 0.2 mL of the sample was vortexed with a reagent consisting of 0.5 mL of 20 mM solution of pentafluorobenzyl bromide in acetone, 0.05 mL of 200 mM ascorbic acid solution and 0.05 mL of 5% sodium chloride. Then, 2 mL of 25 mM iodine solution in ethyl acetate, 0.5 mL of internal standard solution (40 mM 1,3,5-tribromobenzene in ethyl acetate) were added. The preparation was again vortexed for 30 s and the mixture was centrifuged at 2500 rpm for 15 min and left to stand for 1 h [2].
Measurement principle	GC-ECD [2] GC-MS
Aliquot for 1 analysis	1 mL
Limit of quantification	LOD: 336 µg / L (GC-ECD) [2]
Recommended adjustments	creatinine correction to adjust for urine density
Preferred units for expression of results	µmol / mol creatinine
Conversion factor	1 nmol/L = 8.92 x µg/L; µmol/mol creatinine = 1.00 x µg/g creatinine
Biological exposure value	n/a
Background level	30 µmol/L [2]; 2.9 ± 2.5 mmol/mol Creatinine [3]
Possible confounders	Hydrogen sulfide is formed endogenously. Possible enhanced endogenous production in cystic fibrosis [5] and chronic pancreatitis [6] and higher production in persons with Down syndrome.
Remarks	Thiosulphate was detected in the urine of a non-fatal victim of poisoning (and blood thiosulphate and sulfide were not detected) Determination of thiosulphate in urine appeared more useful than the determination of sulfide [3].



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References

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2. Kage S, Takekawa K, Kurosaki K, Imamura T, Kudo K. The usefulness of thiosulphate as an indicator of hydrogen sulfide poisoning: three cases. *Int J Legal Med*. 1997;110(4):220-2.
3. Kage S, Kashimura S, Ikeda H, Kudo K, Ikeda N Fatal and nonfatal poisoning by hydrogen sulfide at an industrial waste site. *J. Forensic Sci* 47:652-655.
4. Olson KR (2009) Is hydrogen sulfide a circulating "Gasotransmitter" in vertebrate blood? *Biochim Biophys Acta* [published ahead of print]
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6. Morselli-Labate AM, Fantini L, Pezzilli R (2007) Hydrogen sulfide, nitric oxide and a molecular mass 66 u substance in the exhaled air of chronic pancreatitis patients.
7. Reptext database by Micromedia. <http://csi.micromedex.com/X/Rera.htm>
8. SZW list of reprotoxic substances (Staatscourant, January 15, 2009)