Arsine

June 2020

CAS No. 7784-42-1

Arsine (AsH_3) is a colorless, and extremely flammable gas with a garlic-like odor. The arsine gas is 2.5-times denser than air.

Arsine is a highly toxic gas that is generated upon exposure of arsenic-containing ores to acids, and it is a by-product of refining of non-ferrous metals.

Arsine is water soluble. It is generally shipped in cylinders as a liquefied compressed gas [ATSDR].

Arsine and arsine gas/air mixtures are flammable and explosive and may be ignited by heat, sparks, or flames. The gas may travel along the ground and migrate to distant ignition sources and flash back. Arsine is stable at room temperature and begins to decompose to arsenic and hydrogen at about 230 °C with complete decomposition at about 300 °C.

Arsine gives off arsenic oxides (posing a risk to health) in a fire that can accumulate or move with the wind in the form of dust.

Usage and exposure

Arsine is used most commonly in the semiconductor and metals refining industries. Most reports of exposure to arsine have occurred after unintentional formation of arsine in the workplace [CDC].

Exposure frequently occurs when arsine gas is generated while metals or crude ores containing arsenic impurities are treated with acid and this is a common source of exposure [ATSDR].

Routs of exposure

Breathing in the gas (inhalation) is the most likely route of exposure after arsine is released into the air [CDC].

The substance can be absorbed into the body by inhalation**.** A harmful concentration of this gas in the air will be reached very quickly on loss of containment [WHO].

Arsine has **not** been known to be absorbed into the body through the eyes and the skin [CDC].

In humans and animals, arsine is metabolized to trivalent arsenic (As(III)) as well as pentavalent arsenic (As(V)). As(III) is methylated to monomethylarsonate and dimethylarsinate. Arsine metabolites are mainly excreted via urine [WHO].

Target organs

The target organ of arsine poisoning is the haematopoietic system, in particular the erythrocytes. Arsine induces haemolysis, causing haemoglobinuria and subsequent renal damage; a large number of fatal intoxications have been described, and they continue to occur [WHO].

Health hazards

Arsine is a strong reducing agent and reacts vigorously with oxidizers such as potassium permanganate, sodium hypochlorite, oxygen, ozone, chlorine, fluorine, and nitric oxide.

Effects of short-term exposure:

Rapid evaporation of the liquid may cause frostbite. The substance may cause abdominal pain, confusion, dizziness, headache, nausea, shortness of breath, vomiting, weakness. Arsine may effects on the blood, resulting in destruction of blood cells and kidney failure. The effects may be delayed.

Rapidly progressing intravascular haemolysis within a few hours is characteristic of arsine poisoning.

Toxic pulmonary edema or acute circulatory failure has been reported as the cause of death in arsine poisoning. Severe liver damage has occasionally been found as a consequence of arsine poisoning. Symptoms of effects on the central and peripheral nervous systems include disorientation, chills, convulsion, and paraesthesia, which may appear shortly after exposure at high concentrations [WHO].

Effects of long-term or repeated exposure:

Late consequences of acute arsine poisoning include chronic renal damage, haematological changes, polyneuritis, and neuropsychological symptoms (e.g., irritation, confusion, memory losses, agitation, and disorientation [WHO].

There is no reported evidence for the carcinogenic effect of arsine per se in humans [WHO].

References:

- ATSDR. Agency for Toxic Substances Disease Registry. Toxic Substances Portal. Arsine. https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=278
- CDC. Center for Disease Control and Prevention. Emergency Preparedness and Response. Facts about arsine. https://emergency.cdc.gov/agent/arsine/facts.asp
- WHO. International Programme on Chemical Safety (IPCS). Concise International Chemical Assessment Document 47. Arsine: Human Health Aspects. World Health Organization Geneva, 2002. https://www.who.int/ipcs/publications/cicad/en/cicad47.pdf?ua=1