
Chemical Terrorism Fact Sheet

Pulmonary Irritants - Phosphine

Protective Equipment/Detection

Positive-pressure, self-contained breathing apparatus (SCBA) is advised in situations that involve exposure to potentially unsafe levels of phosphine. Chemical-protective clothing is not generally required because the gas is not absorbed through the skin, and skin irritation is unlikely. Use rubber gloves and aprons with victims exposed to phosphides.

Decontamination

Remove exposed individuals from the contaminated area as soon as possible. For eye exposure, flush the eyes with cool water for at least 15 minutes. Remove and double-bag contaminated clothing and personal belongings. For skin exposure, especially to the metallic phosphides, brush all visible particles from the skin and hair, flush with water for 5-10 minutes, and then wash well with soap and water, followed by thorough rinsing. Use caution to avoid hypothermia in children and the elderly.

Persons exposed only to phosphine gas do not pose substantial risks of secondary contamination. Metallic phosphides on clothes, skin, or hair can off-gas phosphine after contact with water or moisture, so a risk of secondary contamination may be present. Vomitus containing phosphides can also off-gas phosphine.

Signs and Symptoms

Phosphine is a respiratory tract irritant that attacks primarily the cardiovascular and respiratory systems causing peripheral vascular collapse, cardiac arrest and failure, and pulmonary edema. Children are at higher risk because of their greater lung surface area to body weight ratio, increased minute volume to weight ratio, and shorter stature.

Phosphine interferes with enzymes and protein synthesis, primarily in the mitochondria of heart and lung cells. In the heart, metabolic changes cause cation disturbances that alter transmembrane potentials. Clinical manifestations may include hypotension, reduced cardiac output, tachycardia, and irregular heart beat. Phosphine also affects the small peripheral vessels, causing a profound decrease in systemic vascular resistance that can lead to severe hypotension that does not respond well to pressor agents. Ultimately, cardiac arrest and peripheral vascular collapse can occur. Laboratory tests may reveal abnormal myocardial enzymes.

Respiratory toxicity is characterized by chest tightness, cough (with fluorescent green sputum), and shortness of breath. Severe exposure can cause pulmonary edema, which may have a delayed onset of up to 72 hours after exposure. Pulmonary edema and pneumonitis are believed to result from direct cytotoxicity to the pulmonary cells.

Most deaths occur within 12 to 24 hours after exposure and are cardiovascular in nature. If the patient survives the initial 24 hours, the ECG typically returns to normal, indicating that heart damage is reversible. Liver failure is the prime cause of death after 24 hours.

Phosphine also produces serious central nervous system (CNS), gastrointestinal (GI), and renal effects. Phosphine is a CNS depressant, producing headache, restlessness, dizziness, loss of feeling, impaired gait, trembling of the extremities during movement, and double vision upon initial exposure. Severe exposure can cause seizures and coma. GI symptoms are usually the first to occur after exposure and may include nausea, vomiting, abdominal pain, and diarrhea. Liver damage can also be prominent, although liver injury does not usually become evident until 48 to 72 hours after exposure. Jaundice, hepatomegaly, elevated serum transaminases, and increased serum bilirubin may be

Chemical Overview

Phosphine – PH_3 – is a colorless gas that is heavier than air and may cause asphyxiation in enclosed, poorly ventilated, or low-lying areas. It is extremely flammable and explosive and may ignite spontaneously on contact with air. Upon burning, it produces a dense white cloud of phosphorus pentoxide, also a severe pulmonary irritant due to the rapid formation of orthophosphoric acid on contact with the moist respiratory tract. Pure phosphine is nearly odorless, but commercially available phosphine has an odor of garlic or decaying fish. However, the odor threshold of 0.15 ppm may not provide adequate warning of hazardous concentrations.

Phosphine may be released during acetylene production by the action of water on calcium carbide which is commonly contaminated with calcium phosphide. Phosphine is used in the semiconductor industry, as a fumigant, and as a polymerization initiator. Its metallic salts, aluminum phosphide and zinc phosphide, are solids used as grain fumigants and rodenticides.

Most phosphine exposures occur by inhalation of the gas or ingestion of metallic phosphides, but dermal exposure to phosphides can also cause systemic effects.

Phosphine gas produces no adverse effects on the skin or eyes, and contact does not result in systemic toxicity. Frostbite may occur from contact with liquefied or compressed gas. The ingestion of phosphine is unlikely because it is a gas at room temperature, but the ingestion of metallic phosphides can produce phosphine toxicity, with its systemic symptoms, when the solid phosphide contacts gastric acid.

OSHA's permissible exposure limit, averaged over an 8-hour work shift, is 0.3 ppm. The NIOSH IDLH

Signs and Symptoms (Continued)

evident; and centrilobular necrosis has been reported. Phosphine can produce hematuria and proteinuria, as well as acute kidney failure.

Laboratory: Blood gas analysis may reveal a combined respiratory and metabolic acidosis. In addition to abnormal cardiac isoenzymes, liver enzymes, and urine findings, there have been reports of significant hypomagnesemia and hypermagnesemia associated with massive focal myocardial damage.

Chronic exposure to very low concentrations may result in anemia, bronchitis, nausea and other gastrointestinal disturbances, inflammation of the nasal cavity and throat, weakness, dizziness, jaundice and other liver effects, increased bone density; and visual, speech, and motor disturbances. Chronic exposure may be more serious for children because of their potential longer latency period.

Treatment

There is no antidote for phosphine poisoning. Treatment consists of support of respiratory and cardiovascular functions. Steroids have no proven efficacy. Hemodialysis is recommended only if renal failure develops, and the effectiveness of exchange transfusions is questionable.

Follow the ABCs of evaluating and supporting the airway, breathing, and circulation. Patients who are comatose, hypotensive, or seizing should be provided supportive care with intravenous fluids, pressor agents, sodium bicarbonate, or anticonvulsants as indicated. For adult victims in shock or with severe hypotension (systolic pressure under 80 mmHg), give a 1,000 mL bolus of intravenous saline or lactated Ringer's solution over one hour, followed by an infusion at 150 to 200 mL/hour. If the systolic pressure is over 90 mmHg, begin fluid infusion without the initial bolus. For children, administer a 20 mL/kg bolus of normal saline over 10 to 20 minutes, followed by an infusion at 2 to 3 mL/kg/hour. For significant acidosis, administer sodium bicarbonate intravenously (adult dose = 1 ampule; pediatric dose = 1 mEq/kg), with additional therapy guided by arterial blood gas measurements.

Symptomatic patients should receive supplemental oxygen for dyspnea and should be observed for at least 72 hours with repeated chest examinations and other appropriate studies. Hypoxia may be controlled by O₂ supplementation, and the early use of positive airway pressure intermittent positive pressure breathing (IPPB), a positive end-expiratory pressure (PEEP) mask or, if necessary, intubation (with or without a ventilator) may delay and/or minimize the pulmonary edema and reduce the degree of hypoxia.

Aerosolized bronchodilators should be administered for acute bronchospasm, with consideration of the health of the myocardium in choosing which type of bronchodilator should be used.

Consider racemic epinephrine aerosol for children who develop stridor. A dose of 0.25–0.75 mL of 2.25% racemic epinephrine solution in water, repeated every 20 minutes as needed, should be used.

If phosphides have been ingested, it is important to remove them as quickly as possible from the GI tract, but **do not induce emesis**. Gastric lavage with a potassium permanganate solution (1:10,000) is recommended. Permanganate oxidizes phosphine in the stomach to form phosphate, thus reducing the available phosphine. Follow lavage with a slurry of activated charcoal at 1 gm/kg (usual adult dose 60–90 gm, child dose 25–50 gm). A mineral oil cathartic (100 mL) is recommended rather than a saline cathartic.

Routine laboratory studies for all victims include CBC, basic metabolic panel, renal function tests, liver-function tests and serial cardiac isoenzymes. Diagnostic studies include ECG, chest radiography, pulse oximetry, blood gases, and PFI3.

Additional information and references available at <http://www.bioterrorism.slu.edu>

Chemical Overview (continued)

(immediately dangerous to life or health) is 50 ppm. A level of 0.5 ppm has been set as the maximum airborne concentration below which one can be exposed for up to 1 hour without experiencing or developing serious adverse health effects that could impair the individual's ability to take protective action.

Long-term Medical Sequelae

Most survivors of acute phosphine exposure show no permanent disabilities. Secondary sequelae, such as myocardial infarct or stroke, are possible, however, when there has been insufficient blood supply to the heart and brain. Subacute poisoning, resulting from exposure over a few days, may cause reactive airways dysfunction syndrome months later. The EPA has determined that phosphine is not classifiable (Group D) as to its carcinogenicity and no teratogenic effects are known.

Environmental Sequelae

Phosphine in air is changed to less harmful chemicals in less than a day. Phosphine is also removed from the air by contact with moist soil which promotes oxidation to orthophosphate. Zinc phosphide disappears from soils of 50% or more water content in less than 5 weeks, and the majority is recoverable as orthophosphate.

Disclaimer

Information contained in this fact sheet was current as of October 2002, and was designed for educational purposes only. Medication information should always be researched and verified before initiation of patient treatment.