
Chemical Terrorism Fact Sheet

Lung Irritants - Phosgene

Protective Equipment/Detection

The usual techniques used to determine air concentrations include passive dosimetry, manual and automated colorimetry, infrared spectroscopy and ultraviolet spectrophotometry. Paper tape monitors capable of detecting $5 \mu\text{g}\cdot\text{m}^3$ have been described, but no automated field detectors are currently available.

A military-style respirator offers good protection.

Decontamination

The first consideration during decontamination is to remove the victim from continued exposure. Flush affected skin and eyes with running water for 15-20 minutes. If there has been contact with liquid phosgene, remove contaminated clothing and footwear; and thaw affected area with lukewarm water.

Signs and Symptoms

Phosgene's mode of action is not fully understood, and it has been suggested that it may act by inhibiting enzymes, or by producing HCl in the alveoli. The early-onset ocular, nasal, and central airway irritation from high concentration exposures is likely caused by the HCl released by the hydrolysis of **phosgene** in these moist tissues. More recently, some have suggested that, as a highly reactive molecule, it may react directly at the alveolar and capillary wall. The carbonyl group ($\text{C}=\text{O}$) participates in acylation reactions with amino ($-\text{NH}_2$), hydroxyl ($-\text{OH}$), or sulfhydryl ($-\text{SH}$) groups at the alveolar-capillary membrane and these acylations lead to leakage of fluid into the interstitium from those capillaries. As this leakage outpaces normal drainage mechanisms, edema develops.

With exposure to concentrations exceeding 3 ppm, burning and watering of the eyes, a sore or scratchy throat, dry cough, choking, nausea, headache, and chest tightness develop. Erythema of the oral and pharyngeal mucus membranes becomes evident at higher concentrations. These symptoms, however, do not accurately predict the potential for severe lung injury. Sustained exposures to just 2 ppm for roughly 80 minutes will not cause any irritation, but result in pulmonary edema 12-16 hours later. At concentrations > 200 ppm, **phosgene** passes the blood-air barrier causing hemolysis in the pulmonary capillaries, congestion by red cell fragments and stoppage of capillary circulation. Death occurs within a few minutes from acute cor pulmonale.

Moist rales in the lung fields indicate the presence of pulmonary edema. This is preceded by damage to the bronchiolar epithelium, development of patchy areas of emphysema, partial atelectasis, and edema of the perivascular connective tissue. The trachea and bronchi are usually normal in appearance. With damage to the bronchiolar epithelium, narrowing of the lumen develops, causing a lengthening of the respiratory cycle. As the edema progresses, discomfort, apprehension and dyspnea increase and frothy sputum develops. Rales and rhonchi are audible over the chest, and breath sounds are diminished. The patient may develop shock-like symptoms, with pale, clammy skin, low blood pressure and a feeble, rapid heartbeat. **Phosgene's** effects usually reach a maximum 12-24 hours after exposure. In the terminal clinical phase of lethal poisoning, extreme distress ensues with intolerable dyspnea until respiration ceases.



Phosgene smells like fresh cut grass.
Photo courtesy of the Department of Agriculture

Chemical Overview

Phosgene (CG), carbonyl chloride, is a colorless gas at ambient temperatures. It is easily liquefied under pressure, and has a boiling point 8.2°C . Its density is four times that of air, so it will settle to low-lying areas. It has been described as smelling like decaying fruit, fresh-cut grass or moldy hay, and the odor threshold is roughly 1.5 ppm (mg/m^3). Trained workers can detect it at concentrations of 0.4 ppm. However, the sense of smell is a poor guide to possible concentrations, since olfactory fatigue can develop at high concentrations and mislead the victim as to the potential danger. The LC_{50} is about $3200 \text{ mg}\cdot\text{min}/\text{m}^3$.

Inhalation is the primary route of exposure, with the lung being the main target. At high concentrations, skin and eye irritation occur. The degree of injury is the product of concentration and the duration of acute exposure (Haber's Law). Eyes, nose and throat become irritated at 3-4 ppm. Exposures damaging to the lung are $30 \text{ ppm}\cdot\text{min}$ or greater. Pulmonary edema occurs at dosages exceeding $150 \text{ ppm}\cdot\text{min}$ ($600 \text{ mg}\cdot\text{min}/\text{m}^3$). At dosages exceeding $30 \text{ ppm}\cdot\text{min}$, the initial irritation and respiratory symptoms are followed by a second (sometimes asymptomatic) phase, sometimes lasting 24-48 hours. The duration of this latent phase is inversely proportional to the inhaled dose: the larger the dose the shorter the latent period.

Signs and Symptoms (Continued)

The blood becomes viscous and coagulates easily. Methemoglobin levels increase; followed by cyanosis and reduced arterial blood pressure with tachycardia, as well as a metabolic acidosis and a compensatory hyperventilation. Arterial blood gases will reveal a significant hypoxia.

Contact with liquid **phosgene** may cause burns or frostbite.

Treatment

The treatment for exposure victims is primarily supportive care, with warmth and forced bedrest being important since activity can shorten the latent period. It is important to differentiate between early irritant symptoms and pulmonary edema evident on chest x-ray. Early edema may be detected by chest x-ray, before evident clinical signs, using 50-80 kilovolts. At 100-120 kilovolts, this may not be seen. Irritation typically precedes edema, but edema in the absence of lung irritation has been reported. Observe patients for up to 48 hours. If pulmonary edema develops, it will be apparent by this time, and its onset within 2 to 6 hours is predictive of severe injury. The early use of positive airway pressure intermittent positive pressure breathing (IPPB) or a positive end-expiratory pressure (PEEP) mask may delay and/or minimize the pulmonary edema and reduce the degree of hypoxia, but intubation is critical at the first sign of edema or pulmonary failure. Provide adequate oxygenation, and determine the appropriate mode of ventilation for each individual. Antibiotics are reserved for those with documented pulmonary infection.

An elevation of the pCO₂ greater than 45 mm Hg suggests that bronchospasm is the more likely cause of hypercarbia and bronchodilators should then be used aggressively. If the patient has a prior history of clinical bronchospasm, steroids should be added immediately.

In all others, although the effectiveness of steroids in a chemically induced pulmonary edema is not proven, they are still advised if they can be given within 15 minutes of exposure. The steroid doses used are much greater (methylprednisolone, 700-1000 mg in divided doses on day one and tapered from there) than those prescribed in asthma and should be given by inhalation or, in severe cases, intravenously.

Only cautious use of sedatives is advised and they should be withheld until adequate oxygenation is assured and facilities for possible respiratory assistance are available. Barbiturates, atropine, analeptics and antihistamines are all contraindicated.

Pulmonary function studies and a chest x-ray should be performed on each patient at a 2-3 month follow-up exam.

Long-term Medical Sequelae

Most survivors of acute exposure have a good prognosis, but shortness of breath and physical limitations may persist. Smoking worsens the chances of full recovery, as does pre-existing chronic pulmonary disease. **Phosgene** does not appear to be mutagenic, and data on carcinogenicity are insufficient for an assessment.

Environmental Sequelae

Phosgene is very persistent in the atmosphere because it does not absorb UV light and is not subject to photolysis by sunlight in the troposphere. Its half-life in the atmosphere is estimated at 113 years at sea level. The minimal water solubility and vapor pressure of **phosgene** allow it to rapidly hydrolyze in water.

Disclaimer

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

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