Chemical Terrorism Fact Sheet

Pulmonary Irritants - Sulfur Dioxide

Protective Equipment/Detection

Positive-pressure, self-contained breathing apparatus (SCBA) is advised in situations that involve exposure to potentially unsafe levels of sulfur dioxide. Chemical-protective clothing is indicated when there is potential exposure to the liquefied gas, or in situations where high concentrations of the gas may combine with water or sweat on the skin. Otherwise, safety glasses or face shield, and rubber gloves should provide adequate eye and skin protection.

Decontamination

Remove exposed individuals from the contaminated area as soon as possible. For eye exposure, flush the eyes immediately with water for at least 15 minutes, then irrigate each eye continuously with normal saline during transport. Use proparacaine hydrochloride to assist eye irrigation if necessary. Remove and double-bag contaminated clothing and personal belongings. For skin exposure, 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. Cover skin burns with sterile dressings after decontamination.

Signs and Symptoms

Workplace exposure to SO_2 can cause both acute and chronic effects, and exposure to very high levels can be life threatening. With acute exposure to 5 ppm, the victim will notice only dryness of the nose and throat, but a measurable increase in airway restriction is already developing. At levels of 6-8 ppm, there is a measurable decrease in tidal volume. Sneezing, cough, expectoration, and eye symptoms develop at levels of 10 ppm, while exposure to 20 ppm causes severe bronchospasm. At 50 ppm, the victim will experience extreme upper respiratory symptoms, but no significant injury will occur with exposures limited to less than 30 minutes. Exposures at 100 ppm can cause an immediate health risk and at 1000 ppm death can occur within 10 minutes from severe respiratory depression.

Since sulfur dioxide is so readily dissolved in water, it is intensely irritating to the eyes and respiratory tract, with its predominant effects being seen in the upper respiratory tract. Sulfur dioxide's adverse effects are believed to be increased by the formation of sulfates or higher sulfur oxides from interactions between SO_2 and water or SO_2 and particulate matter. The acute effects include upper respiratory tract irritation, rhinorrhea, choking, expectoration, nosebleeds, difficulty in swallowing, oropharyngeal erythema, and coughing. Within 5 to 15 minutes from the onset of exposure, workers develop temporary reflex bronchoconstriction and increased airway resistance. Continued exposure can result in high pitched rales, thoracic pain, nasopharyngitis, tracheitis, laryngeal edema, chemical bronchopneumonia, pulmonary edema, cyanosis, asphyxia and death. Destruction of the ciliated epithelium also leads to increased risks of pulmonary infection. The most common form of death is asphyxiation from severe glottal and bronchiolar constriction, so it is imperative that the airway be secured immediately in any victim displaying pulmonary symptoms.

The eyes will develop lacrimation, conjunctival injection, and blepharitis initially.

Chemical Overview

Sulfur dioxide – SO_2 – is a colorless, non-flammable gas with a strong, suffocating odor. It is a liquid when under pressure, and it dissolves in water very easily.

Sulfur dioxide in the air comes mainly from activities such as the burning of coal and oil at power plants or from copper smelting. Over 65% of SO_2 released to the air, more than 13 million tons per year, comes from electric utilities, especially those that burn coal. On a global basis, fossil fuel combustion accounts for 75 to 85% of man-made **sulfur dioxide** emissions, and industrial processes such as refining and smelting account for the remainder. As a result, SO_2 is a major component of acid rain. Exposure can also occur in the manufacturing of sulfuric acid, paper, food preservatives, and fertilizers, as well as from smoke inhalation and industrial and vehicular air pollution. In nature, sulfur dioxide can be released to the air from volcanic eruptions. In addition, hydrogen sulfide, from the natural decay of vegetation on land, marsh lands and in the oceans, is probably oxidized to **sulfur dioxide** within hours.

The EPA has established an air quality standard of 0.03 ppm for long-term, 1-year average concentrations of sulfur dioxide and short-term, 24-hour air concentrations should not exceed 0.14 ppm more than once a year. OSHA has set a limit of 2 ppm over an 8-hour workday, 40-hour workweek, and 5 ppm as the short-term (15 minute) exposure limit. The NIOSH IDHL (immediately dangerous to life and health) is 100 ppm, while victims with chronic pulmonary disease, particularly asthma, have been shown to be sensitive to much lower concentrations. Children may be at higher risk because of their greater lung surface area to body weight ratio, increased minute volume to weight ratio, and shorter stature.

Signs and Symptoms (Continued)

Iritis may develop with higher exposures. Fortunately, severe eye injury is only seen with contact to the liquid form of SO_2. After being sprayed, the corneal epithelium turns gray and irregular, but it remains attached to the stroma. After several hours, the lids become edematous and the conjunctiva appears white and opaque due to thrombosed blood vessels. This corneal damage can result in blindness.

Dermal exposure can result in irritation, urticarea, and burns. The major risks to the skin are from exposure to liquid sulfur dioxide.

Due to the high solubility of SO_2, it is rapidly distributed throughout the body and produces a metabolic acidosis. This results in a reduction of blood alkali reserves and a compensatory elimination of ammonia in the urine. The generalized toxic effect is one of protein and carbohydrate metabolism disorders, along with possible effects on the hemopoietic system and the production of methemoglobin. Vomiting, diarrhea, abdominal pain, fever, headache, vertigo, agitation, tremor, convulsions, and peripheral neuritis also have been noted.

The chronic effects of exposure include permanent pulmonary impairment, as a result of repeated episodes of bronchoconstriction. A worker's exposure to high concentrations of SO_2 (80 to 100 ppm) may cause an increased incidence of nasopharyngitis, shortness of breath on exertion (dyspnea), and chronic fatigue. Concentrations of SO_2 from 2 to 36 ppm produced a significantly higher frequency of respiratory disease symptoms, including chronic coughing, expectoration, and dyspnea.

Treatment

There is no antidote for sulfur dioxide exposure and care is supportive. Monitor the cardiac rhythm and treat arrhythmias as appropriate. A precautionary intravenous line should be started in all patients, using D_5W at a "to keep open" rate. If signs of hypovolemia or shock are present, use normal saline or lactated Ringer's at 150-250 mL/hr and consider vasopressors, remaining mindful of the potential for pulmonary edema and the need to avoid fluid overload. In the acidotic patient, anticipate seizures and treat with diazepam if necessary. Skin irritation and burns should be treated topically.

It is critical that a patent airway be established, and intubation at the first sign of upper airway obstruction may be necessary. An open airway must be maintained with suctioning as necessary. Watch for signs of respiratory insufficiency and consider assisted ventilation if indicated. Hypoxia is another concern so administer oxygen by a non-rebreather mask at 10 to 15 L/min and monitor the patient's oxygenation status with arterial blood gases or pulse oximetry. Also watch closely for signs of pulmonary edema, which may be delayed, and treat accordingly. The early use of positive airway pressure intermittent positive pressure breathing (IPPB), a positive end-expiratory pressure (PEEP) mask or, intubation (with or without a ventilator) may delay and/or minimize pulmonary edema and reduce the degree of hypoxia.

For ingestion, rinse the mouth and administer 5 mL/kg, up to 200 mL, of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. **Do not use emetics.** Administer activated charcoal as a slurry at 1 gm/kg (usual adult dose 60–90 gm, child dose 25–50 gm) and consider a cathartic.

Additional information and references available at http.www.bioterrorism.slu.edu

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Long-term Medical Sequelae

Acute high-dose **sulfur dioxide** exposures have resulted in severe obstructive and restrictive defects 3 months post-exposure, which failed to respond to bronchodilators. There are no studies that definitively link sulfur dioxide to cancer and the EPA has classified it in Group D, not classifiable as to human carcinogenicity. Its reproductive and teratogenetic effects are not known.

Environmental Sequelae

When released into the air, sulfur dioxide can be converted to sulfuric acid, sulfur trioxide, and sulfates. Easily dissolved in water, it can form sulfurous acid. Sulfur dioxide can also be absorbed into the soil, but its changes there are not precisely known.

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.

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