
Chemical Terrorism Fact Sheet

Irritants - Ammonia

Protective Equipment/Detection

Ammonia is easily detected by its distinctive odor, and a number of chemical processes are available to detect it as well. Positive-pressure, self-contained breathing apparatus (SCBA) and chemical-protective clothing are recommended in response situations with potentially unsafe levels of ammonia.

Although ammonia levels within the serum and urine can be performed, these are not of value in determining the presence or level of a toxic exposure because ammonia occurs naturally in the body.

Decontamination

Rapid skin and eye decontamination is critical. Remove contaminated clothing while flushing exposed areas. Double-bag contaminated clothing and personal belongings to avoid exposure to off-gassing vapors. Exposed skin and hair should be flushed with water for at least 5 minutes, and then washed thoroughly with soap and water. Take care to avoid hypothermia. Irrigate exposed or irritated eyes with plain water or saline for at least 15 minutes. Contact lenses should be removed, if possible without causing further injury to the eye.

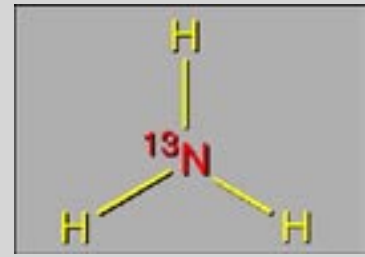
In cases of ingestion do not induce emesis, perform gastric lavage, attempt neutralization, or administer activated charcoal. Give 4 to 8 ounces of water or milk to those who are able to swallow.

Signs and Symptoms

Skin and Eyes: The extent of skin and eye injury from ammonia exposure depends on the duration of the exposure and the concentration of the liquid or gas. Frostbite injury can result from liquefied ammonia contact. Dermal contact to low concentration (5%) solutions, as typically found in the household, is irritating but rarely causes damage. Contact with concentrated ammonia solutions (25% and higher) or vapors can cause pain, inflammation, blisters, necrosis and deep penetrating burns, especially on moist skin areas, as well as permanent eye damage or blindness. The full extent of ophthalmic damage may not be evident for up to 1 week after the exposure.

Respiratory Tract: Low air concentrations (50-100 ppm) may produce rapid eye and nose irritation with lacrimation, rhinorrhea, and cough; and higher air concentrations can produce serious burns to the eyes. Inhalation exposure to high concentrations of ammonia can cause nasopharyngeal and tracheal burns, bronchiolar and alveolar edema, and airway destruction resulting in respiratory distress or failure. Bronchiolar edema can narrow the airways to the extent of causing airway obstruction. While anhydrous ammonia is lighter than air, vapors from liquefied gas are initially heavier than air and may spread along the ground causing asphyxiation in poorly ventilated or enclosed areas. 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.

Cardiovascular: Hypertension has also been reported and can reach malignant levels. As such, myocardial infarction and cardiac death have followed ammonia exposure.



Molecular Structure of Ammonia
Photo courtesy of the Department of Molecular & Medical Pharmacology at the University of California, Los Angeles

Chemical Overview

Ammonia is a colorless gas with a very distinct, sharp odor that evaporates quickly and readily dissolves in water. It is somewhat flammable at concentrations of 15 to 28% by volume in air. Prolonged exposure to concentrations greater than 300 ppm can cause permanent injury or death. Fortunately, ammonia has a low odor threshold (5 ppm) and causes eye irritation at 20 ppm, alerting most people to its presence and the need to move out of the area of contamination. However, olfactory fatigue can occur, leading to prolonged and dangerous exposure.

It occurs naturally in the soil as a product of bacterial action on decaying organic matter. As a key intermediate in the nitrogen cycle, ammonia is essential in many biological processes, such as the formation of nitrates and nitrites, as well as being a nutrient for many bacteria and plants. It is also essential for the creation of proteins and other nitrogen compounds such as the base compounds of DNA.

Ammonia is commercially manufactured for use in fertilizer, textiles, plastics, explosives, pulp and paper production, food and beverages, household cleaning products, refrigerants (as anhydrous ammonia), and other products. It is commonly available in liquid form, either pressurized in tanks or dissolved in water as ammonium hydroxide, an alkaline solution that can be highly corrosive.

Since ammonia is a naturally occurring substance whose concentrations vary based on a number of factors such as pH and temperature, it is difficult to assess uniform safe levels within a body of water. Any environmental release greater than 100 pounds of ammonia or 1,000 to 5,000 pounds of ammonium salts must be reported to the EPA. For air exposure, OSHA has set a limit of 50 ppm over an 8-hour workday, 40-hour

Signs and Symptoms (Continued)

Gastrointestinal: Although not common, the ingestion of ammonia, in the form of ammonium hydroxide, will produce nausea, vomiting, and abdominal pain and can cause serious burns of the mouth, pharynx, esophagus, and stomach at higher concentrations (10% and higher). Such ingestions do not cause system toxicity.

Treatment

General: 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, or anticonvulsants as indicated. There is no specific antidote for ammonia poisoning. Some toxicologists recommend the use of corticosteroids to limit esophageal scarring, but this treatment is unproven and may be harmful in patients who have perforation or serious infection. Hemodialysis has not been shown effective.

Cardiopulmonary: If there are signs of laryngeal edema and airway compromise, secure the airway and respiration via endotracheal intubation or cricothyroidotomy. Administer supplemental oxygen by mask to all patients who have respiratory symptoms. Observe all patients carefully for 6 to 12 hours for signs of upper-airway obstruction and pulmonary edema, while noting that pulmonary injury may continue to evolve over 18 to 24 hours. Hypertensive patients should be treated medically and closely monitored for cardiac injury.

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. Ammonia poisoning is not known to add additional risk during the use of bronchial or cardiac sensitizing agents.

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.

Skin: Ammonia-induced skin burns should be treated as thermal burns. Patients with mild to moderate skin burns should be reexamined within 24 hours.

Eyes: For eye injuries, continue irrigation for at least 15 minutes or until the pH of the conjunctival fluid has returned to normal. Test visual acuity, examine the eyes for corneal damage, and treat any injuries appropriately. An ophthalmologist should reexamine all patients with eye injuries within 24 hours, and immediate consultation should be obtained for those who have severe corneal injuries.

Chemical Overview (continued)

workweek, while the National Institute of Occupational Safety and Health (NIOSH) advises that workplace air should not exceed 25 ppm ammonia averaged over an 10-hour workday or 40-hour workweek. For short-term exposures up to 15 minutes, a maximum concentration of 35 ppm is recommended.

Long-term Medical Sequelae

Residual bronchoconstriction, bronchiectasis, small airway disease, and chronic lung disease can develop following severe inhalation injury. Inhalation victims who are initially symptomatic should be observed carefully and reexamined periodically, including annual pulmonary function testing. With ammonia-related eye injuries, ulceration and perforation of the cornea can develop weeks or even months after exposure, with the potential for permanent blindness. Cataracts and glaucoma have also been reported following acute exposures. Ingestion injuries may result in permanent damage to the mucous membranes of the alimentary canal, with bleeding, perforation, scarring, or stricture formation as potential sequelae. The reproductive, carcinogenic, and teratogenic effects of ammonia are not known.

Environmental Sequelae

Ammonia occurs naturally and readily taken up from soil and water by plants and bacteria. It is recycled naturally as part of the nitrogen cycle. However, major spills can cause environmental damage by producing overgrowth of aquatic plants and the depletion of oxygen in the water, and by the corrosive effects of ammonium hydroxide.

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>