
Information and recommendations for doctors at hospitals/emergency departments

- Patients whose clothing or skin is contaminated with liquid acetic acid can cause secondary contamination of rescue and medical personnel by direct contact or through evaporation of acetic acid. Patients exposed only to acetic acid vapor do not pose a significant risk of secondary contamination.
 - Acetic acid is rapidly corrosive to all tissues. Eye contact may cause severe burns and loss of vision. Contact with the skin may cause severe burns which may be delayed in onset. Acetic acid vapor is irritating to the skin, eyes, nose, throat and respiratory tract, causing irritation, coughing, chest pain and dyspnea. Laryngospasm and pulmonary edema (shortness of breath, cyanosis, expectoration, cough) may occur.
 - There is no antidote to be administered to counteract the effects of acetic acid. Treatment consists of supportive measures.
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1. Substance information

Acetic acid (CH₃-COOH), CAS 64-19-7

Synonyms: ethylic acid, ethanoic acid

At room temperature, acetic acid is a clear, colorless liquid with a sour, vinegar-like odor. Acetic acid is used in the manufacture of acetic anhydride, cellulose acetate, acetylsalicylic acid, and chloroacetic acid. It is also used in the production of plastics, pharmaceuticals, dyes, insecticides, and photographic chemicals; as a food additive, as a natural latex coagulant; and in textile printing. Solutions of lower concentrations may be used in disinfection applications.

2. Routes of exposure

Inhalation

Exposures may occur by inhalation. Acetic acid's odor and upper respiratory tract irritant properties generally provide adequate warning of hazardous concentrations.

Skin/eye contact

Most exposures occur by direct contact of the skin and the eyes with liquid acetic acid. Contact with the skin and the eyes causes severe burns which may be delayed in onset.

Ingestion

Ingestion is rare in occupational settings.

3. Acute health effects

Respiratory

Acetic acid exposure usually causes mucous membrane irritation, sore throat, and coughing. Rapid development of respiratory distress with chest pain, dyspnea, laryngospasm and pulmonary edema (shortness of breath, cyanosis, expectoration, cough) may occur. Lung injury may progress over several hours. Acetic acid poisoning may cause respiratory failure. Systemic absorption in humans is rare since both the liquid and vapor are irritating or corrosive.

Dermal

Deep burns of the skin and mucous membranes may be caused by contact with acetic acid; disfiguring scars may result. Contact with less concentrated acetic acid vapor or mist can cause burning pain, redness, inflammation, and blisters.

Ocular

Eye contact causes severe burns and loss of vision. Contact with less concentrated acetic acid vapor or mist cause burning discomfort, spasmodic blinking or involuntary closing of the eyelids, redness, and tearing.

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Ingestion Ingestion causes severe corrosive injury of the mucous membranes of the throat and esophagus.

Dose-effect relationships Dose-effect relationships are as follows:

<u>Acetic acid concentration</u>	<u>Effect</u>
0.48 - 1 ppm	- Odor detection
10 ppm	- TLV-TWA (USA, NIOSH)
20 ppm	- TLV-STEL (USA; NIOSH)
50 ppm	- IDLH (USA, NIOSH)
60-70 ml	- calculated lethal dose for concentrated acetic acid by ingestion

Potential sequelae If the patient survives the initial 48 hours after exposure, recovery is likely. After acute exposure, pulmonary function usually returns to normal in 7 to 14 days. Complete recovery is usual; however, symptoms and pulmonary deficits may persist. Airways hyperreactivity to non-specific irritants may persist, resulting in bronchospasm and chronic inflammation of the bronchi. Pulmonary tissue destruction and scarring may result in chronic dilation of the bronchi and increased susceptibility to infection. Chronic or prolonged exposure to acetic acid gas or mist has been associated with abnormal pulmonary function and chronic bronchial inflammation.

4. Actions

Self-protection

Patients exposed only to acetic acid vapor do not pose a significant risk of secondary contamination. Patients whose clothing or skin is contaminated with liquid acetic acid or acetic acid mist can secondarily contaminate other people by direct contact or through evaporation of acetic acid.

Decontamination

Patients exposed only to acetic acid vapor or mist who have no evidence of skin or eye irritation do not need decontamination. All others require decontamination.

Patients who are able and cooperative may assist with their own decontamination. If the exposure involved liquid acetic acid and if clothing is contaminated, **remove immediately** and double-bag the clothing.

Assure that exposed or irritated eyes have been irrigated with plain water or saline for at least 20 minutes, and that the pH of the conjunctival fluid has returned to normal (7.0). If not, continue eye irrigation during other basic care and transport. If eye irrigation is impaired by blepharospasm, one to two drops of oxybuprocaine 0.4% may be instilled into affected eyes to allow adequate irrigation.

Remove contact lenses if present and easily removable without additional trauma to the eye.

Assure that exposed skin and hair have been flushed with plain water for at least 15 minutes. If not, continue flushing during other basic care and transport. Protect eyes during flushing of skin and hair.

After ingestion do not induce emesis. If the patient's consciousness is impaired or if a large dose has been ingested less than 30 minutes before evaluation of the patient's condition, careful gastric lavage with a small-bore tube may be considered.

Initial treatment

Therapy will be empiric; there is no antidote to be administered to counteract the effects of acetic acid.

The following measures are recommended if the airborne exposure dose is 15 ppm or greater (depending on time exposed), if symptoms, e. g. eye irritation or pulmonary symptoms have developed, or if no exposure dose can be estimated but exposure has possibly occurred:

- Administration of oxygen
- Administration of 8 puffs of beclomethasone (800 µg beclomethasone dipropionate) from a metered dose inhaler.

Patients with severe clinical respiratory symptoms (e.g. bronchospasms, stridor) should be treated as follows:

a) Nebulization of adrenaline (epinephrine): 2 mg adrenaline (2 ml) with 3 ml NaCl 0.9% and inhale through a nebulizer mask.

b) Administration of a β 2-selective adrenoceptor agonist, e.g., four strokes of terbutaline or salbutamol or fenoterol (one stroke usually contains 0.25 mg of terbutaline sulfate; or 0.1 mg of salbutamol; or 0.2 mg of fenoterol); this may be repeated once after 10 minutes. Alternatively, 2.5 mg salbutamol and 0.5 mg atrovent may be administered by nebulizer mask.

If inhalation is not possible, administration of terbutaline sulfate (0.25 mg to 0.5 mg) subcutaneously or salbutamol (0.2 mg to 0.4 mg over 15 minutes) intravenously.

c) Intravenous administration of 250 mg methylprednisolone (or equivalent steroid dose).

Patients with clinical signs of a toxic lung edema (e.g. foamy sputum, wet crackles) should be treated as follows:

- a) Start CPAP-therapy (Continuous Positive Airway Pressure Ventilation).
- b) Intravenous administration of 1000 mg methylprednisolone (or an equivalent steroid dose) is recommended.

Intubation of the trachea or an alternative airway management should be considered in cases of respiratory compromise. When the patient's condition precludes this, consider cricothyrotomy if equipped and trained to do so.

Note: Efficacy of corticosteroid administration has not yet been proven in controlled clinical studies.

If signs of hypoxemia or severe inhalation exposure are present, humidified supplemental oxygen should be administered.

Intubation of the trachea or an alternative airway management should be considered in cases of respiratory compromise. When the patient's condition precludes this, consider cricothyrotomy if equipped and trained to do so.

If acetic acid was in contact with the skin, chemical burns may result; treat as thermal burns: adequate fluid resuscitation and administration of analgesics, maintenance of the body temperature, covering of the burn with a sterile pad or clean sheet.

After eye exposure chemical burns may result; treat as thermal burns. Immediately consult an ophthalmologist.

Note: Any facial exposure to liquid acetic acid should be considered as a serious exposure.

To the standard intake history, physical examination, and vital signs add pulse oximetry monitoring and a PA chest X-ray.

Further evaluation and treatment

Spirometry should be performed. Routine laboratory studies should include a complete blood count, blood glucose and electrolyte determinations.

Evidence of pulmonary edema - hilar enlargement and ill-defined, central-patch infiltrates on chest radiography - **is a late finding that may occur 6 to 8 hours or later after exposure. The chest X-ray is typically normal on first presentation to the emergency department, even with severe exposures.**

Patients who have had significant exposure or who develop serious signs or symptoms should be observed for a minimum of 12 hours and reexamined frequently before confirming the absence of toxic effects. Delayed effects are unlikely in patients who have minor upper respiratory symptoms (mild burning or a slight cough) that resolve quickly.

If oxygen saturation is less than 90 % or if it appears to drop, immediately check arterial blood gasses and repeat the chest X-ray. If blood gasses begin to show deterioration and/or if the chest X-ray begins to show pulmonary edema start oxygen supplementation.

In case of worsening clinical signs (especially tachypnea >30/min with a simultaneous decrease of the partial pressure of carbon dioxide) CPAP-therapy (Continuous Positive Airway Pressure Ventilation) should be started within the first 24 hours after exposure.

In case of a pulmonary edema fluid intake/output and electrolytes should be monitored closely. Avoid net positive fluid balance. Central line or Swan-Ganz catheterization might be considered, to optimize fluid management.

As long as signs of pulmonary edema are present, intravenous administration of methylprednisolone (or an equivalent steroid) should be continued in intervals of 8-12 hours.

Prophylactic antibiotics are not routinely recommended but may be used based on the results of sputum cultures. Pneumonia can complicate severe pulmonary edema.

*Patient release/
follow-up instructions*

Clinically asymptomatic patients exposed to a concentration of **less than 15 ppm** (depending on the period of time exposed) **as well as patients who have a normal clinical examination and no signs or symptoms of toxicity may be discharged after an appropriate observation period in the following circumstances:**

- a) The evaluating physician is experienced in the evaluation of individuals with acetic acid or irritant gas exposure.
- b) Information and recommendations for patients with follow-up instructions are provided verbally and in writing. Patients are advised to seek medical care promptly if symptoms develop or recur.
- c) The physician is comfortable that the patient understands the health effects of acetic acid.
- d) Site medical is notified, so that the patient may be contacted at regular intervals in the 24-hour period following release from the emergency department.
- e) Heavy physical work should be precluded for up to 24 hours.
- f) Exposure to cigarette smoke should be avoided for 72 hours; the smoke may worsen the condition of the lungs.

Patients who have serious skin or eye injuries should be reexamined in 24 hours.

Post discharge spirometry should be repeated until values return to the patient's baseline values.

In this document BASF has made a diligent effort to ensure the accuracy and currency of the information presented but makes no claim that the document comprehensively addresses all possible situations related to this topic. This document is intended as an additional resource for doctors at hospitals/emergency departments in assessing the condition and managing the treatment of patients exposed to acetic acid. It is not, however, a substitute for the professional judgement of a doctor and must be interpreted in the light of specific information regarding the patient available to such a doctor and in conjunction with other sources of authority.

BASF SE
Corporate Health Management
Carl-Bosch-Straße 38
67056 Ludwigshafen
Germany

BASF Corporation
Medical Department
100 Campus Drive, M/S F 221
Florham Park, NJ 07932
USA