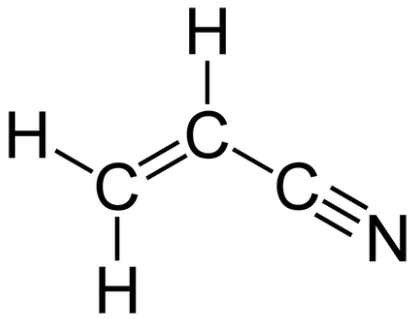
ACRYLONITRILE- TOXGUIDE™

CHEMICAL AND PHYSICAL INFORMATION

Acrylonitrile (CAS# 107-13-1) is a volatile liquid. It is used in the manufacture of acrylic fibers, plastics, and other chemicals. Significant quantities may escape into the air during the manufacture and use. In the past, it was combined with carbon tetrachloride and used as a fumigant for flour milling, bakery food processing equipment, and stored tobacco; these fumigants were voluntarily removed in the late 1970s.



ENVIRONMENTAL FATE AND DETECTED LEVELS

Air: The average ambient air level of acrylonitrile is samples taken from 63 locations in the United States in 2020–2022 was 0.1 ppbv.

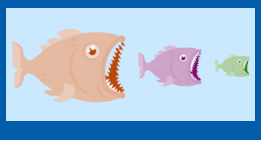
Acrylonitrile is degraded primarily by reaction with hydroxyl radicals, with an estimated half-life of 1.2–12 hours.

Water: Acrylonitrile was not detected in 87 surface water samples collected in 2020–2022. It was detected in one of the 1,121 groundwater samples collected in 2020–2022 at a concentration of 1.82 ppb.

Acrylonitrile has little tendency to adsorb to sediment but is subject to biodegradation by microorganisms.

Sediment and Soil: Acrylonitrile was not detected in 97 sediment samples collected between 2000 and 2009.

Acrylonitrile is expected to be highly mobile in soils.



Bioconcentration: An average bioconcentration factor of 30 has been estimated for edible portions of freshwater fish and marine species.

GENERAL POPULATION EXPOSURE

Likely route of potential exposure: Inhalation

- One of the primary sources of exposure for the general population is inhalation during the use of consumer products containing acrylonitrile, such as acrylic fiber clothing or carpeting and acrylonitrile-based plastics.
- The general population can also be exposed via inhalation of smoke from tobacco, marijuana, or other acrylonitrile-containing burning biomass.
- Another potential source of exposure is via ambient air.

Likely route of potential exposure: Oral

- One of the primary sources of exposure for the general population is the oral route through ingestion of acrylonitrile leached from plastic food containers.

Possible route of potential exposure: Dermal

- Dermal exposure may occur through the use of products containing acrylonitrile.

ACRYLONITRILE

POPULATIONS WITH POTENTIALLY HIGH EXPOSURE

Workers involved in the manufacture of acrylic and modacrylic fibers and the production of acrylonitrile-butadiene-styrene plastics, adipontrile, acrylamide, nitrile rubber, and carbon fiber may be exposed to higher than background levels of acrylonitrile exposed via:

- Inhalation of contaminated air
- Direct skin contact with acrylonitrile

Compared to the general population, the following groups may also have increased risk of exposure:

- Populations living near industrial releases or contaminated hazardous waste sites (via ambient air and/or groundwater contamination).

BIOMARKERS

Several biomarkers of exposure have been identified for acrylonitrile.

- Urinary levels of the metabolites, thiocyanate, 2-cyanoethylmercapturic acid (CEMA) and N-acetyl-S-s(2-cyanoethyl)-L-cysteine (2-CyEMA), can be used to evaluate acrylonitrile exposure. However, thiocyanate and CEMA are not specific to acrylonitrile.
- The hemoglobin adduct, N-(2-cyanoethyl)valine, can be measured in blood and used as a biomarker of exposure. N-(2-cyanoethyl)valine levels are high in smokers.

BIOMONITORING LEVELS

The geometric mean urinary level (creatinine adjusted) in NHANES survey years 2017–2018 of the acrylonitrile metabolite 2-CyEMA was 2.77 µg/g creatinine.

TOXICOKINETICS

Absorption: Acrylonitrile is well absorbed following inhalation and oral exposure; approximate absorption rates are 50 and 90%, respectively. Data are not available to estimate dermal absorption rates.

Distribution: Acrylonitrile is widely distributed throughout the body, with higher levels in the liver, kidneys, lungs, and stomach.

Metabolism: The primary metabolic pathway is conjugation with glutathione. It is also metabolized by the microsomal enzyme system to form 2-cyanoethylene, which is metabolized to thiocyanate or thiodiglycolic acid.

Excretion: Acrylonitrile is primarily excreted in the urine as conjugates or thiocyanate. A small percentage is excreted in air as carbon dioxide.

Physiologically based pharmacokinetic (PBPK) models: PBPK models of rats and humans have been developed for predicting internal doses of acrylonitrile and cyanoethylene oxide.

HEALTH EFFECTS

Based on a systematic review, the following are presumed health effects for humans exposed to acrylonitrile:

- Respiratory effects (inhalation exposure only)
- Gastrointestinal effects (oral exposure only)
- Neurological effects
- Developmental effects

Health effects are determined by the dose (how much), the duration (how long), and the route of exposure.

The discussion of health effects is continued on Page 3.

ACRYLONITRILE

HEALTH EFFECTS (CONTINUED)

Humans exposed to high levels of acrylonitrile via inhalation exposure reported respiratory tract irritation and clinical signs similar to those associated with cyanide poisoning, including labored and irregular breathing, dizziness, cyanosis, limb weakness, and convulsions.

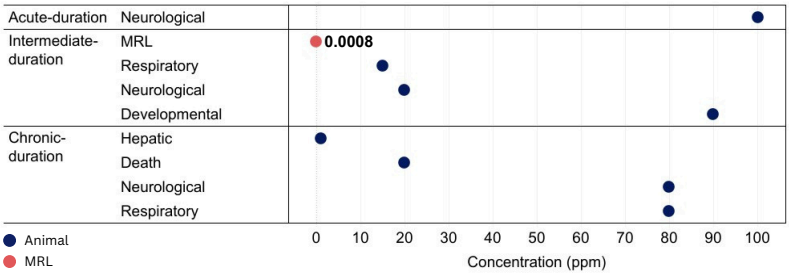
Respiratory irritation has also been observed in laboratory animals acutely exposed to high levels of acrylonitrile. Longer-term inhalation exposure has resulted in nasal cavity epithelial lesions in animals.

Lesions in the non-glandular stomach have been reported in laboratory animals following oral exposure.

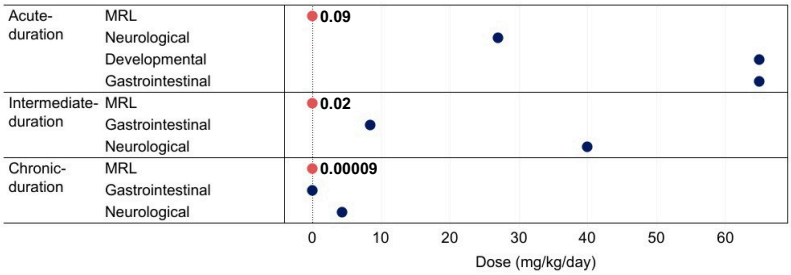
Signs of overt neurotoxicity, including hindlimb weakness, decreased activity, paralysis, and seizures, have been observed in laboratory animals. Other neurological effects included decreased nerve conduction velocity and glial cell tumors in the brain.

Decreased fetal body weights and increased malformations have been observed in animals following inhalation and oral exposure.

Sensitive Effects of Inhalation Exposure to Acrylonitrile



Sensitive Effects of Oral Exposure to Acrylonitrile



MINIMAL RISK LEVELS (MRLs)

Acute: ≤14 days; Intermediate: 15–364 days; Chronic: ≥365 days

Inhalation:

- **Acute:** Not derived.
- **Intermediate:** An intermediate-duration inhalation MRL of 0.0008 ppm was derived based on nasal effects in rats.
- **Chronic:** Not derived.

Oral:

- **Acute:** An acute-duration oral MRL of 0.09 mg/kg/day was derived based on developmental effects in rats.
- **Intermediate:** An intermediate-duration oral MRL of 0.02 mg/kg/day was derived based on forestomach effects in rats.
- **Chronic:** A chronic-duration oral MRL of 0.00009 mg/kg/day was derived based on forestomach effects in rats.

CANCER

Animal cancer studies have found increases in the incidence of several cancer types, including glial cell tumors in the brain and spinal cord of rats, Zymbal gland carcinomas in rats, and forestomach tumors in rats and mice.

The Department of Health and Human Services (HHS) has categorized acrylonitrile as reasonably anticipated to be a human carcinogen. The U.S. Environmental Protection Agency (EPA) has categorized acrylonitrile as a probable human carcinogen. The International Agency for Research on Cancer (IARC) concluded that acrylonitrile is carcinogenic to humans.

REFERENCE

Agency for Toxic Substances and Disease Registry (ATSDR). 2025. Toxicological profile for acrylonitrile. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services. https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=447&tid=78.