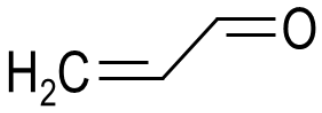
ACROLEIN - TOXGUIDE™

CHEMICAL AND PHYSICAL INFORMATION



Acrolein (CASRN 107-02-8) is a colorless or yellowish liquid with a disagreeable odor.

It is a reactive aldehyde primarily used as an intermediate in the chemical manufacturing of acrylic acid to make synthetic glycerol, acrolein polymers, polyurethane, and polyester resins. Acrolein is also used as an herbicide (trade name Magnacide H) to control algae and aquatic weeds, and as a biocide (trade name Magnacide B) to control mollusks in recirculating process water systems. Acrolein is used as an intermediate in the production of methionine, which is a protein supplement used in animal feed. Due to its foul odor, acrolein has also been used in military poison gas mixtures.

ENVIRONMENTAL FATE AND DETECTED LEVELS

Air: In 2024, the average concentrations of acrolein from 30 monitoring stations across the United States ranged from 0.030 to 0.30 ppbv (0.069–0.69 µg/m^3), with maximum values of 1.19 ppbv (2.73 µg/m^3). The respective ranges for 2022 and 2023 were 0.062 to 0.591 ppbv (0.14–1.36 µg/m^3) and 0.001 to 1.41 ppbv (0.002–3.23 µg/m^3).

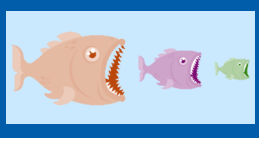
Acrolein is released to the environment in emissions from manufacturing and use facilities, combustion processes (including automobile emissions and smoke from any type of fire), degradation of other pollutants, and direct release. It is a reactive compound and is unstable in the environment. Acrolein is not persistent in the atmosphere and reacts with hydroxyl radicals, with a half-life of 15–20 hours.

Water: Acrolein was not detected above analytical method quantification limits in 72 surface water samples across the United States from 2016 to 2023; from 2005 to 2015, acrolein was detected in 20% of 69 surface water samples at average concentrations of 1.16–4.44 µg/L.

Acrolein is expected to volatilize rapidly from surface water. It can be removed from water by volatilization, abiotic processes, and biodegradation processes. When applied to surface water as an herbicide, the half-life was reported to be <1–3 days.

Sediment and Soil: In sediment samples, acrolein was found at a maximum of 1.9 µg/kg in 8 of 105 sediment samples reported for 2005–2014. Acrolein was not detected in five soil samples reported in the Water Quality Portal database from 2005 to 2009.

Acrolein is expected to volatilize rapidly from soil. It can be removed from soil by volatilization, abiotic processes, and biodegradation processes.



Bioconcentration: Acrolein is not expected to bioaccumulate.

GENERAL POPULATION EXPOSURE

Primary route of potential exposure: Inhalation

- The main source of acrolein exposure for the general population stems from indoor air; smoking (cigarettes, marijuana, e-cigarettes, vaping), secondhand smoke, cooking with oils and fats, and building materials all contribute to acrolein levels in the air.
- Environmental tobacco smoke, including primary, secondhand, and thirdhand smoke, is a major source of exposure for many people in the general population.

Possible route of potential exposure: Oral

- Ingestion of some foods and beverages and consumption of contaminated drinking water can be routes of exposure.

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POPULATIONS WITH POTENTIALLY HIGH EXPOSURE

Occupational exposures: People who work in heavy traffic or parking garages may be exposed to acrolein by breathing vehicle exhaust. Firefighters are at high risk of exposure to acrolein when battling house fires, wildfires, and industrial fires. Workers involved the production of acrylates, methionine, perfumes, plastics, refrigerants, rubber, or textile resins may potentially be exposed. Individuals working in a restaurant kitchen or a bar/tavern that allows indoor smoking may have higher exposure levels to acrolein. Occupational exposure may occur via:

- Inhalation of contaminated air

The following groups may also have higher exposure to acrolein, compared to the general population:

- People living or working near dense traffic areas, due to acrolein in the exhaust from gasoline or diesel vehicles.
- People living near a landfill or water source being treated with acrolein to eliminate unwanted plants.

BIOMARKERS

The primary urinary metabolites of acrolein, carboxyethyl mercapturic acid (CEMA) and 3-hydroxypropylmercapturic acid (3-HPMA), are used as biomarkers of exposure. However, neither metabolite is specific for acrolein exposure.

BIOMONITORING LEVELS

There are no data regarding levels of acrolein in the general population; however, higher urinary levels of 3-HPMA and CEMA were seen in tobacco smokers compared to non-tobacco users (NHANES 2005–2006).

TOXICOKINETICS

Absorption: Studies in animals indicate that acrolein is absorbed in the respiratory tract, primarily the upper respiratory tract, following inhalation exposure. Human and animal studies demonstrate that acrolein is absorbed from the gastrointestinal tract following oral exposure.

Distribution: Animal studies indicate that distribution of acrolein after inhalation and oral exposure is limited due to the strong reactivity of acrolein with tissues at the exposure site.

Metabolism: The main metabolic pathway is through acrolein conjugation with reduced glutathione (GSH) followed by enzyme-catalyzed conversion to mercapturic acid products for urinary excretion. The major urinary products of this pathway are 3-HPMA and CEMA. Minor metabolic pathways are postulated to yield glyceraldehyde and malonic acid.

Excretion: Acrolein is not excreted unchanged. Acrolein metabolites are excreted primarily in the urine and exhaled air following oral or inhalation exposure; small quantities are excreted in feces.

Physiologically based pharmacokinetic (PBPK) models: PBPK models have been developed to simulate the kinetics uptake and metabolism of acrolein in the rodent and human respiratory tract.

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

HEALTH EFFECTS

A systematic review of noncancer endpoints resulted in the following hazard identification conclusions:

- Respiratory effects are a presumed health effect for humans following inhalation of acrolein.
- Immunological effects are a suspected health effect for humans following inhalation of acrolein.
- Gastrointestinal effects are a suspected health effect for humans following ingestion of acrolein.

Other health effects were observed at similar inhalation and/or oral exposure levels (e.g., hematological, endocrine, neurological, and cardiovascular effects). However, these endpoints did not undergo systematic review due to paucity and/or inconsistency of available data. *The discussion of health effects is continued on Page 3.*

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HEALTH EFFECTS (CONTINUED)

Respiratory effects

- Rapid onset of nose and throat irritation and a reduction in breathing rate (believed to be a protective measure triggered by nose irritation) were reported by volunteers acutely exposed to low levels of acrolein.
- Epidemiology studies have reported associations between acrolein exposure and reporting of respiratory irritation symptoms, prevalence of asthma, and decrements in pulmonary function.
- In animals, nasal and pulmonary irritation, altered respiratory function, and increased lung weight were reported after inhalation exposure (all durations).

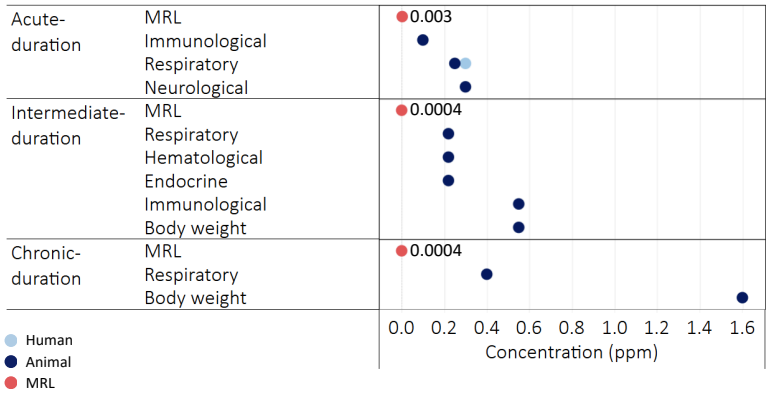
Immune effects

- Altered immune function has been seen in animals following inhalation or oral exposure.

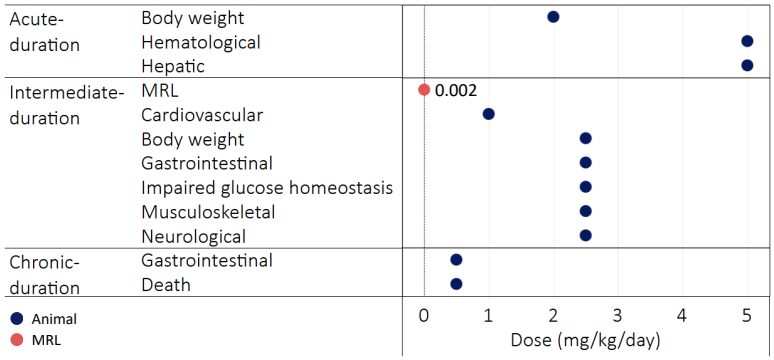
Gastrointestinal effects

- In animals, stomach lesions including ulcers, hemorrhage, hyperplasia of the forestomach, and/or erosion of the glandular mucosa were seen after intermediate-duration oral exposure.

Sensitive Effects of Inhalation Exposure to Acrolein*



Sensitive Effects of Oral Exposure to Acrolein



*Human studies in figures are limited to those with reliable exposure estimates.

MINIMAL RISK LEVELS (MRLs)

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

Inhalation:

- **Acute:** An acute-duration inhalation MRL of 0.003 ppm (0.007 mg/m^3) was derived based on nose and throat irritation and decreased respiratory rate in humans.
- **Intermediate:** The chronic-duration MRL of 0.0004 ppm (0.0009 mg/m^3) was adopted as the intermediate-duration inhalation MRL.
- **Chronic:** A chronic-duration inhalation MRL of 0.0004 ppm (0.0009 mg/m^3) was derived based on nasal respiratory gland metaplasia in rats.

Oral:

- **Acute:** Not derived.
- **Intermediate:** An intermediate-duration oral MRL of 0.002 mg/kg/day was derived based on forestomach squamous epithelial hyperplasia in male mice.
- **Chronic:** Not derived.

CANCER

Increased incidence of nasal tumors was seen in animals after chronic-duration inhalation exposure.

The Department of Health and Human Services (HHS) has not classified acrolein as to its carcinogenicity. The U.S. Environmental Protection Agency (EPA) concluded that the potential carcinogenicity of acrolein cannot be determined due to inadequate data. The International Agency for Research on Cancer (IARC) has classified acrolein as "probably carcinogenic to humans" (Group 2A) based on "sufficient" evidence of carcinogenicity in experimental animals and "strong" mechanistic evidence.

REFERENCE

Agency for Toxic Substances and Disease Registry (ATSDR). 2025. Toxicological profile for acrolein. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.
https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=557&tid=102