

## Sources of Exposure

## Toxicokinetics and Biomonitoring

## Biomarkers/Environmental Levels

### General Populations

- General population exposure to vinyl acetate is expected to be low.
- Potential sources of exposure include inhalation of contaminated ambient air and cigarette smoke, dermal contact with products containing the compound (e.g., glues and paints), ingestion of residual vinyl acetate monomers in food (that may have migrated from plastic food wraps), or food items containing the compound as a starch modifier.
- Dermal and inhalation exposure may occur during household water use (e.g., showering, bathing, washing of dishes or clothes) if the water contains vinyl acetate.
- Vinyl acetate was detected infrequently and at very low levels in the air of residence and office buildings.
- Populations living near contaminated hazardous waste sites may have increased exposure via ambient air, groundwater contamination, and/or vapor intrusion, compared to the general population.

### Occupational Populations

- Occupational exposure to vinyl acetate may occur via inhalation of contaminated workplace air.
- Workers may also be exposed by dermal contact with vinyl acetate vapor or liquids and products containing the compound.
- Workers involved in manufacturing adhesive, petrochemical, paint and coating, or plastics and resin may be exposed to vinyl acetate.

### Toxicokinetics

- Vinyl acetate is rapidly and effectively absorbed via the inhalation and oral route. However, vinyl acetate is expected to be absorbed to some degree based on lethality reported in a single rabbit study following exposure to a highly concentrated dermal dose.
- Vinyl acetate is rapidly and widely distributed in rodents, with the highest concentration in the Harderian gland, salivary glands, lacrimal glands, gastrointestinal mucosa, and respiratory tract.
- Vinyl acetate is rapidly hydrolyzed by carboxylesterases to form acetaldehyde and acetic acid.
- Vinyl acetate is eliminated rapidly from the body, primarily through expired air as carbon dioxide.
- Available *in vivo* and *in vitro* data have been utilized to develop physiologically based pharmacokinetic (PBPK) models to simulate the kinetics of vinyl acetate uptake and metabolism in the nasal cavity in rats and humans.

### NHANES Biomonitoring

- There are no data regarding levels of vinyl acetate in the general U.S. population.

### Biomarkers

- There are no specific biomarkers for vinyl acetate exposure.
- Vinyl acetate breaks down very quickly in the body to substances that are normally found in the body; thus, measurement of these break down products is not useful for determining whether exposure to vinyl acetate has occurred.

### Environmental Levels

#### *Air*

- The average vinyl acetate levels recorded from air monitoring sites across the United States were <LOD (limit of detection)–10.6 ppbv in 2020 and <LOD–7.85 ppbv in 2021.

#### *Water*

- Between 2010 and 2019, vinyl acetate was only detected in 13% of ambient surface water samples, with an average of 9.39 µg/L (range 0.5–200.00 µg/L; n=6,457).

#### *Sediment and soil*

- The most recent sediment samples were collected in 2000; vinyl acetate was detected in 11% of samples at an average level of 25 µg/kg (range 16–36 µg/kg; n=85).

### Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2023. Toxicological Profile for Vinyl Acetate (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

# ToxGuide™ for Vinyl Acetate



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U.S. Department of Health and  
Human Services  
Public Health Service  
Agency for Toxic Substances  
and Disease Registry  
[www.atsdr.cdc.gov](http://www.atsdr.cdc.gov)



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AGENCY FOR TOXIC SUBSTANCES  
AND DISEASE REGISTRY

## Chemical and Physical Information

## Routes of Exposure

## Relevance to Public Health (Health Effects)

### Vinyl Acetate

- Vinyl acetate is a colorless liquid with a sweet, fruity smell.
- It is a man-made compound that is used in the production of polymers and copolymers, including polyvinyl acetate, polyvinyl alcohol, polyvinyl acetals, ethylene-vinyl acetate copolymer, and polyvinyl chloride-acetate copolymer.
- Consumer and commercial uses include use in adhesives, paints and powder coatings, plastics and resins, rubber foam, packaging, sporting equipment (e.g., ski boots, bicycle seats), auto-related films, and intermediates in construction and building materials.
- Vinyl acetate also has a few approved uses as a food additive (masticatory substance, solvent/vehicle) and as a component in polymerized food packaging (e.g., ethylene-vinyl acetate copolymers).

- Inhalation – Primary route of exposure for the general and occupational populations.
- Oral – Possible route of exposure for the general population through ingestion of food containing vinyl acetate.
- Dermal – Primary route of exposure for occupational population; possible route of exposure for the general population.

### Vinyl Acetate in the Environment

- Vinyl acetate is a volatile compound that is released mainly to the atmosphere.
- It is highly soluble in water. Therefore, vinyl acetate released to the atmosphere is expected to redeposit to surface waters and soil through precipitation.
- Vinyl acetate is expected to be highly mobile in soils and is likely to partition to groundwater when released to subsurface soils.
- In the atmosphere, vinyl acetate is rapidly broken down by photochemical oxidation with an atmospheric lifetime on the order of hours to days.
- In soils and surface and groundwater, the compound undergoes hydrolysis and biotransformation, with half-lives on the order of hours to days.
- Vinyl acetate is unlikely to bioconcentrate/biomagnify in terrestrial or aquatic organisms/food chains.

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

### Minimal Risk Levels (MRLs)

#### *Inhalation*

- A provisional acute-duration ( $\leq 14$  days) inhalation MRL of 1 ppm ( $3.5 \text{ mg/m}^3$ ) was derived.
- A provisional intermediate-duration (15–364 days) inhalation MRL of 0.7 ppm ( $2.5 \text{ mg/m}^3$ ) was derived.
- A provisional chronic-duration ( $\geq 365$  days) inhalation MRL 0.3 ppm ( $1.1 \text{ mg/m}^3$ ) was derived.

#### *Oral*

- No acute-, intermediate-, or chronic-duration oral MRLs were derived for vinyl acetate.

### Health Effects

- Exposure to vinyl acetate via inhalation in humans resulted in irritation to the nose and throat.
- The nasal cavity appears to be the most sensitive target tissue in rats and mice following inhalation exposure.
- Degenerative, necrotic, and/or hyperplastic lesions were seen in the olfactory epithelium of the nasal cavity following inhalation exposure in rats.
- Decreased fetal growth and delayed ossification were observed in rat fetuses following maternal exposure during gestation; findings may have been secondary to decreased maternal weight.
- Chronic inhalation or oral exposure in rodents was associated with neoplastic lesions in the upper respiratory system, oral cavity, and upper gastrointestinal tract.
- The Department of Health and Human Services (HHS) (NTP) and U.S. Environmental Protection Agency (EPA) (IRIS) have not classified the potential for vinyl acetate to cause cancer in humans. The International Agency for Research on Cancer (IARC) has determined that vinyl acetate is possibly carcinogenic to humans based on inadequate evidence in humans and limited evidence in experimental animals.

### Children's Health

- It is not known if children are more sensitive to vinyl acetate exposure than adults.