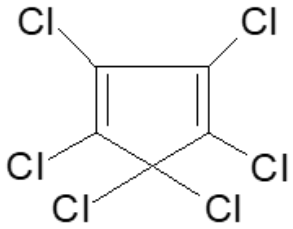
HEXACHLOROCYLCOPENTADIENE- TOXGUIDE™

CHEMICAL AND PHYSICAL INFORMATION



Hexachlorocyclopentadiene (HCCPD; CASRN 77-44-4) is a yellow or yellow-green liquid with a pungent, unpleasant odor. It is a highly volatile, reactive compound.

HCCPD is used during the production of certain flame-retardant chemicals and, to a lesser extent, nonflammable resins, polyester resins, pharmaceuticals, unbreakable plastics, acids, esters, ketones, fluorocarbons, and dyes . HCCPD was previously used in the production of chlorinated pesticides that are now banned in the United States and most other countries.

HCCPD is an anthropogenic chemical that has entered the environment primarily because of its past use in the production of chlorinated pesticides and flame-retardant chemicals.

ENVIRONMENTAL FATE AND DETECTED LEVELS

Air: HCCPD was not detected in air monitoring stations throughout the United States between 2017 and 2024.

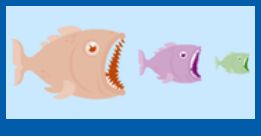
HCCPD is expected to react rapidly with hydroxyl radicals, nitrate radicals, and ozone, and to be degraded rapidly by direct photolysis. Since it exists in the vapor phase, atmospheric transport may occur, but transport distance will be limited by high reactivity of the chemical in the atmosphere. There is little potential for HCCPD washout in preciptiation due to relatively low water solubility.

Water: HCCPD was not detected in groundwater or surface water samples collected throughout the United States between 2019 and 2024.

HCCPD in water may volatilize to air or adsorb onto soil and sediment particles. HCCPD release to waterways is also subject to degradation. In shallow or flowing waters, photolysis is the predominant fate process; in deeper waters, hydrolysis and biodegradation may be more important environmental fate processes.

Sediment and Soil: HCCPD was not detected in sediment samples collected across the United States between 2019 and 2024 or in soil samples collected across the United States in 2023.

HCCPD released to soil and sediments is readily adsorbed and is unlikely to migrate to groundwater. It may volatilize to air from surface soils or sediments. Degradation of HCCPD in soils and sediments may also occur via both abiotic and biotic degradation processes. Potential exists for significant losses via photolysis of HCCPD on soil surfaces, while hydrolysis may also occur in moist soils.



Bioconcentration: Bioconcentration of HCCPD in biota is not expected due to its rapid degradation.

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GENERAL POPULATION EXPOSURE

General population exposure to HCCPD is expected to be extremely low.

Most likely routes of potential exposure: Inhalation and Oral

- Inhalation of contaminated air; however, since HCCPD is highly reactive, it is unlikely to remain in air (as HCCPD) for extended periods.
- Ingestion of contaminated water; however, since HCCPD is highly volatile, it is unlikely to remain in water for extended periods.

Possible route of potential exposure: Dermal

- Direct contact with compound or vapor in contaminated air.

POPULATIONS WITH POTENTIALLY HIGH EXPOSURE

Individuals employed in industries that make or use HCCPD may be exposed via:

- Inhalation of contaminated workplace air (predominant route of exposure).
- Direct skin contact with HCCPD or HCCPD vapors in contaminated workplace air.

Compared to the general population, the following groups may also have higher exposure to HCCPD:

- Populations living near industrial releases (via ambient air).

BIOMARKERS

Primary: HCCPD in blood, urine, or feces.

While several metabolites are excreted, the metabolites of HCCPD have not been fully identified; therefore, they cannot be used as biomarkers.

BIOMONITORING LEVELS

There are no data regarding levels of HCCPD in the general population.

TOXICOKINETICS

Absorption: HCCPD is readily absorbed via the inhalation route (80.7–86% of administered dose) but is absorbed to a lesser extent following oral exposure (25–40% of administered dose). No estimate of dermal absorption is available.

Distribution: HCCPD is rapidly and widely distributed following inhalation exposure. Following oral exposure, the highest levels have been detected in the kidneys in rats and livers in mice.

Metabolism: Polar metabolites of HCCPD have been measured in the liver, kidneys, urine, and feces of rats following oral exposure, but these have not been fully characterized. Results of *in vitro* studies indicate that microbial degradation of HCCPD occurs in the digestive tract. Only a minimal amount of HCCPD is completely metabolized to carbon dioxide (CO_2).

Excretion: HCCPD and its metabolites are excreted in the urine and bile regardless of exposure route. Unabsorbed HCCPD is excreted in feces following oral exposure.

Physiologically based pharmacokinetic (PBPK) models: A two-compartment model was established to describe the fate of intravenously injected HCCPD. Existing kinetic data are insufficient for developing a model for experimental animals or humans exposed via the oral or inhalation routes.

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HEALTH EFFECTS

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

Inhalation effects:
Systematic review was conducted on sensitive effects identified following inhalation exposure in humans (respiratory) and animals (respiratory, hematological).

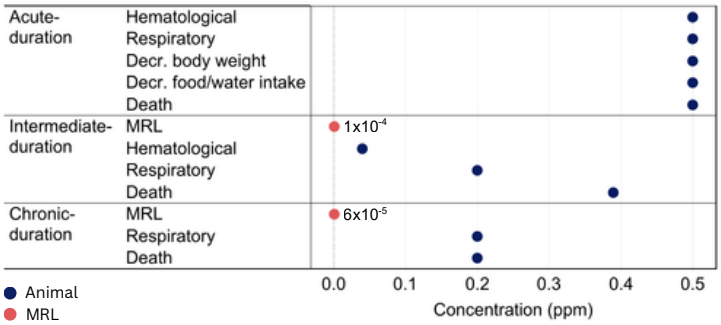
- Respiratory effects are a presumed health effect for humans following inhalation exposure. Effects are attributed to compound reactivity at portal-of-entry.
- Hematological (red blood cell) effects are suspected health effects for humans following inhalation exposure; white blood cell effects are not classifiable as to the hazard to humans.

Oral effects:
Systematic review was conducted on sensitive effects identified following oral exposure in animals (gastrointestinal, renal).

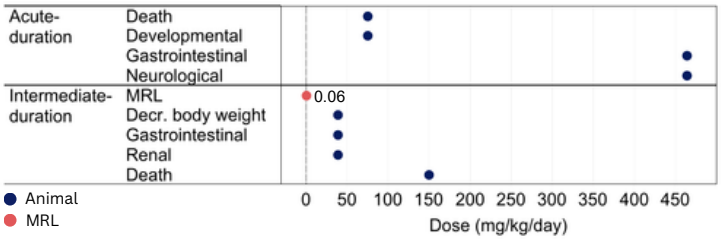
- Gastrointestinal effects are a presumed health effect for humans following oral exposure. Effects are attributed to compound reactivity at portal-of-entry.
- Renal effects are a presumed health effect for humans following oral exposure.

Dermal/Ocular effects: Results of studies in humans and animals indicate that direct contact with HCCPD liquid or vapors are irritating to the skin and eyes. Effects are attributed to compound reactivity at portal-of-entry.

Sensitive Effects of Inhalation Exposure to HCCPD



Sensitive Effects of Oral Exposure to HCCPD*



*No oral chronic data were available

MINIMAL RISK LEVELS (MRLs)

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

Inhalation:

- **Acute:** Not derived.
- **Intermediate:** A provisional intermediate-duration inhalation MRL of $1x10^{-4}$ ppm ($1x10^{-3}$ mg/m^3) was derived based on respiratory (nasal) effects in rats.
- **Chronic:** A provisional chronic-duration inhalation MRL of $6x10^{-5}$ ppm ($7x10^{-4}$ mg/m^3) was derived based on respiratory (nasal) effects in mice.

Oral:

- **Acute:** Not derived.
- **Intermediate:** A provisional intermediate-duration oral MRL of 0.06 mg/kg/day was derived based on gastrointestinal effects in rats.
- **Chronic:** Not derived.

CANCER

The U.S. Environmental Protection Agency (EPA) determined that HCCPD is not likely to be a human carcinogen by the inhalation route. The International Agency for Research on Cancer (IARC) has not evaluated the carcinogenicity of HCCPD. The U.S. Department of Health and Human Services (HHS) has not evaluated a cancer classification category for HCCPD.

REFERENCE

Agency for Toxic Substances and Disease Registry (ATSDR). 2026. Toxicological profile for Hexachlorocylcopentadiene (draft for public comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services. https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=992&tid=208.