

Sources of Exposure

Toxicokinetics and Normal Human Levels

Biomarkers/Environmental Levels

General Populations

- Contaminated air was a potential source of exposure to parathion among the general population living near areas where the insecticide was used; however it is no longer used in the United States.
- Contaminated food was another likely source of general population exposure to parathion; a lesser source of exposure may have been contaminated drinking water.
- However, given that EPA cancelled all registered uses in 2006, the potential for human exposure to parathion through the diet or drinking water in the United States is low.

Occupational Populations

- Contaminated air and dermal contact were sources of exposure for workers previously involved in the manufacture and/or agricultural application of the insecticide. However, given that its registered uses have been cancelled, there is no longer a potential for occupational exposure to parathion in the United States.

Toxicokinetics

- Parathion can be readily absorbed from the skin; available data indicate that parathion can be absorbed from the lungs and gastrointestinal tract as well.
- Available animal data indicate a high affinity of parathion for adipose tissue and the liver; lower levels of parathion and its active metabolite (paraoxon) were detected in muscles, lung, and brain.
- The liver is the main site for parathion metabolism; however, metabolism has been observed in kidney, lung, and brain as well. Some parathion metabolites (mainly paraoxon) are toxic.
- Most parathion is eliminated through metabolism and subsequent excretion in urine; a small proportion of metabolites may be excreted in feces.

Normal Human Levels

- Parathion is not likely to be found in blood, tissues, urine, or feces because it is no longer manufactured or used as an insecticide in the United States.

Biomarkers

- Parathion can be measured in blood or urine. Some parathion metabolites in urine may serve as biomarkers as well.

Environmental Levels

Air

- Ambient air levels of 0.017–0.089 µg/m³ were measured when parathion was used in the past in the United States.

Soil

- Parathion was not detected in most soil samples, but was sometimes detectable in dose proximity to application sites (e.g., in drainage ditches).

Water

- Parathion in surface waters generally ranged from not detectable to 160 ppt. Parathion was not detected in most drinking water samples; but was detected at 4.6 ppb in one drinking water well.

Food

- Average levels in food items between the years 1994 and 2000 ranged from not detectable to 1.6 ppm.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2017. Toxicological Profile for Parathion. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

ToxGuide™

for

Parathion

$C_{10}H_{14}NO_5PS$

CAS# 56-38-2

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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

Contact Information:
Division of Toxicology and Human Health Sciences
Environmental Toxicology Branch

1600 Clifton Road NE, F-57
Atlanta, GA 30329-4027
1-800-CDC-INFO
1-800-232-4636



Chemical and Physical Information

Routes of Exposure

Relevance to Public Health (Health Effects)

Parathion is a Liquid (pure) or Solid (formulated)

- Parathion is an organophosphorus insecticide with a garlic- or phenol-like odor that is pale yellow (pure), dark brown (technical grade liquid generally in an organic solvent), or colorless to white (formulated solid).
- Parathion has been banned for all uses in the United States.

- Inhalation and Dermal—Formerly significant routes of exposure for the general population and farm workers in areas where parathion was used, and among workers involved in production, formulation, and handling of parathion.
- Oral – Former possible route of exposure through ingestion of parathion-treated food sources; small children may have come into contact with parathion residues in soil and dust via hand-to-mouth activity.

Parathion in the Environment

- Parathion in air via agricultural spraying would be expected to exist in vapor and particulate phases; in air parathion would be expected to be degraded in the presence of sunlight and ozone.
- Parathion in surface water or soils would tend to adsorb to soils and sediments; this would limit its mobility, volatilization from water surfaces, susceptibility to photolysis, bioavailability, and biodegradation.
- Parathion in surface water may be subject to both abiotic degradation via hydrolysis and photolysis and biotic degradation by microorganisms.
- Parathion in soils and sediments can be degraded by hydrolysis, photolysis, and microorganisms.
- Parathion does not significantly bioaccumulate in aquatic organisms.

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

Minimal Risk Levels (MRLs)

Inhalation

- No acute-duration (≤ 14 days) inhalation MRL was derived for parathion.
- An intermediate-duration (15-364 days) inhalation MRL of 20 ng/m^3 was derived for parathion.
- No chronic-duration (≥ 365 days) inhalation MRL was derived for parathion.

Oral

- No acute-duration (≤ 14 days) oral MRL was derived for parathion.
- An intermediate-duration (15-364 days) oral MRL of 0.009 mg/kg/day was derived for parathion.
- No chronic-duration (≥ 365 days) oral MRL was derived for parathion.

Health Effects

- The nervous system is the main target of parathion toxicity and is typical of other organophosphates.
- Typical signs and symptoms of organophosphate intoxication include reduced plasma and red blood cell acetylcholinesterase activity, excessive bronchial secretions, respiratory distress, salivation, pinpoint pupils, bradycardia, abdominal cramps, diarrhea, tremor, fasciculations, and possibly death
- EPA concluded that parathion is a “possible human carcinogen” based on limited animal data. The International Agency for Research on Cancer (IARC) has concluded that parathion is possibly carcinogenic to humans.

Children's Health

- 1 Children exposed to parathion would be expected to experience effects characteristic of organophosphate poisoning.
- No studies were located regarding parathion in human breast milk, but there is indirect evidence for transfer of parathion via the placenta and/or breast milk in animals.
- The potential for parathion-induced developmental effects in humans has not been adequately evaluated. Oral exposure of pregnant rats and rabbits to parathion did not result in developmental effects.