

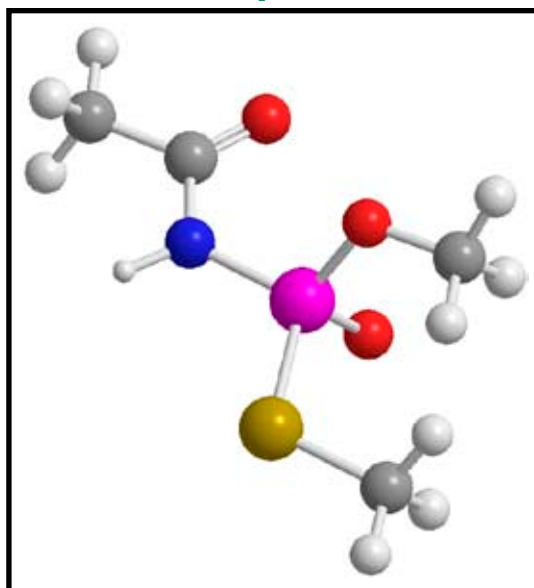
Thank you for visiting the National Pesticide Information Center's fact sheets.

Some of the information in the following fact sheet (scroll down) is out-of-date. NPIC has started a *NEW* set of fact sheets, and acephate is high on our list of priorities. If you would like to be notified when NPIC releases new publications, send an email to npicupdates@ace.orst.edu with "subscribe" in the subject line.

Check out our new technical fact sheet on [resmethrin!](#)

Please call NPIC with any questions you have about pesticides at **1-800-858-PEST (7378)**.

Molecular Structure - Acephate



NPIC Technical Fact Sheets are designed to provide information that is technical in nature for individuals with a scientific background or familiarity with the regulation of pesticides by the U.S. Environmental Protection Agency (U.S. EPA). This document is intended to be helpful to professionals and to the general public for making decisions about pesticide use.

National Pesticide Information Center

Acephate

(Technical Fact Sheet)

For less technical information please refer to the **General Fact Sheet**.

The Pesticide Label: Labels provide directions for the proper use of a pesticide product. *Be sure to read the entire label before using any product.* A signal word, on each product label, indicates the product's potential hazard.

CAUTION- low toxicity

WARNING- moderate toxicity

DANGER- high toxicity

What is acephate?

- Acephate is an insecticide that was registered in the United States Environmental Protection Agency (EPA) in 1974 (1).
- Acephate belongs to a class of insecticides known as organophosphates.

How does acephate work?

- Acephate can kill insects by direct contact or ingestion (2). Acephate kills insects by disrupting their normal nervous system functions.
- Acephate disrupts the nervous system by phosphorylating the active site of the acetylcholinesterase enzyme, rendering it inactive (3, 4).
- The acetylcholinesterase enzyme is necessary for stopping the transmission of the chemical neurotransmitter acetylcholine across the nerve synapse. When the enzyme is inhibited, a surplus of acetylcholine builds up, overstimulating the nervous system (3, 4).

What types of products contain acephate?

- Lawn and garden granules, dusts, sprays, pellets, and wettable powders
- Household sprays
- Agricultural sprays, pellets, and granules

What are some products that contain acephate ?

- Orthene™
- Lancer™
- Pinpoint™
- Payload™

How toxic is acephate?

Animals

- Acephate is low in toxicity to both male and female rats. The oral LD50 in male and female rats is 945 mg/kg and 866 mg/kg, respectively (5). See boxes on **Laboratory Testing, LD/LC50, and Toxicity Category**.
- Inhaled acephate has very low toxicity to rats. The acute LC50 in rats is >61 mg/L (5).
- A female chicken dosed with 785 mg/kg of acephate was negative for acute delayed neurotoxicity (5). A single dose high enough to cause delayed neurotoxicity would probably kill the test animal, even with atropine protection (6).
- Skin-applied acephate has very low toxicity to rabbits. The dermal LD50 in rabbits is >10,000 mg/kg. Acephate did not cause dermal sensitization in guinea pigs (5).
- Acephate caused mild eye irritation in rabbits (5).
- The signs of acephate poisoning include behavioral changes, muscle tremors, twitching, diarrhea, salivation, breathing difficulties, and in more severe cases, paralysis and death (7).

Humans

- Acephate products may have a signal word on the label of Danger, Warning, or Caution (8). The signal word is determined by the toxicity criteria in the **Toxicity Category** box (9).
- The symptoms associated with acephate poisoning include headache, nervousness, blurred vision, weakness, nausea, cramps, diarrhea, difficulty breathing, and chest pain (10).
- The signs associated with acephate poisoning include sweating, pin-point pupils, tearing, salivation, clear nasal discharge and sputum, vomiting, muscle twitching, muscle weakness, and in severe poisonings convulsions, coma, and death (10).

Is acephate metabolized and excreted by the body?

- In a rat study, acephate was rapidly and completely absorbed from the stomach and rapidly excreted in the urine. Approximately 95% of the acephate dose was excreted in the first 12 hours. The remaining acephate was found in the exhaled air (probably CO₂; 1- 4.5%), feces (1%) and tissues (0.4%). In a separate metabolism study in rats, acephate did not concentrate in blood, liver, muscle, fat, heart and brain (5).
- In another rat study, a small portion of the acephate was metabolized to the organophosphate insecticide methamidophos. The methamidophos was excreted quickly and did not accumulate in the rat (5).

Exposure: Effects of acephate on human health and the environment depend on how much acephate is present and the length and frequency of exposure. Effects also depend on the health of a person and/or certain environmental factors.

Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term and long-term health effects. In these tests, laboratory animals are purposely fed a pesticide at high doses to cause toxic effects. These tests help scientists judge how these chemicals might affect humans, domestic animals, and wildlife in cases of overexposure. When pesticide products are used according to label directions, toxic effects are not likely to occur because the amount of pesticide that people and animals may be exposed to is low compared to the doses fed to laboratory animals.

LD50/LC50: A common measure of acute toxicity is the lethal dose (LD50) or lethal concentration (LC50) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD50 is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC50 is often expressed as mg of chemical per volume (e.g., liter (L)) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD50/LC50 is small and practically non-toxic when the value is large. However, the LD50/LC50 does not reflect any effects from long-term exposure (i.e., cancer, birth defects, or reproductive toxicity) that may occur at levels below those that cause death.

Does acephate cause reproductive or teratogenic effects?

Animals

- In a three generation reproductive study, researchers fed rats acephate. Researchers observed treatment-related effects only in the high dose group, 25 mg/kg/day. Effects included decreased body weights and/or weight gains for adult and offspring rats, decreased litter sizes, and a decreased mating performance. The parental and reproductive NOAELs were 2.5 mg/kg/day (5).
- Pregnant rats fed mid to high doses (20 and 75 mg/kg/day, respectively) of acephate had pups with decreased food consumption and body weights. At the highest dose (75 mg/kg/day), scientists observed a decrease in the number of ossified vertebrae, sternal centers, metacarpals, and forelimb and hindlimb phalanges in pups. The no observable adverse effect level (NOAEL) for development effects was 75 mg/kg/day (5).

- In a rabbit study, 2 rabbits out of 16 were aborted. Researchers observed no other effects on maternal and developmental parameters. The maternal and developmental NOAELs were 3 and 10 mg/kg/day, respectively (5, 11).

Humans

- There is no evidence that human exposure to acephate interferes with pregnancy or causes birth defects (1, 5, 11).

Does acephate cause cancer?

Animals

- Researchers fed rats acephate over their lifetime and noted a higher incidence of tumors in male rats. The higher incidence was dose-unrelated and within the historical control range. The U.S. EPA reevaluated the data and concluded that acephate was not carcinogenic in the study (5).
- Acephate increased the incidence of hepatocellular carcinomas and adenomas in female mice fed large amounts of acephate over their life time. The NOAELs for male and female mice were 8 and 7 mg/kg/day, respectively (5).
- Researchers often use studies designed to test for mutagenicity to screen chemicals for carcinogenicity. Acephate tested positive in mutagenicity tests using bacteria, yeast, and cultured mammalian cells. Negative mutagenicity results in whole animal studies lessens the concern for the potential mutagenic hazard of acephate (5).

Humans

- The EPA has classified acephate as a possible (group C) human carcinogen (11). This classification means that acephate has been shown to cause cancer in one strain or sex of a laboratory animal, but there is inadequate or no evidence that it may cause cancer in humans. See **Cancer** box.

Toxicity Category (<i>Signal Word</i>)				
	High Toxicity (<i>Danger</i>)	Moderate Toxicity (<i>Warning</i>)	Low Toxicity (<i>Caution</i>)	Very Low Toxicity (<i>Caution</i>)
Oral LD50	Less than 50 mg/kg	50 - 500 mg/kg	500 - 5000 mg/kg	Greater than 5000 mg/kg
Dermal LD50	Less than 200 mg/kg	200 - 2000 mg/kg	2000 - 5000 mg/kg	Greater than 5000 mg/kg
Inhalation LC50 - 4hr	Less than 0.05 mg/l	0.05 - 0.5 mg/l	0.5 - 2 mg/l	Greater than 2 mg/l
Eye Effects	Corrosive	Irritation persisting for 7 days	Irritation reversible within 7 days	Minimal effects, gone within 24 hrs
Skin Effects	Corrosive	Severe irritation at 72 hours	Moderate irritation at 72 hours	Mild or slight irritation

U.S. Environmental Protection Agency, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling
<http://www.epa.gov/oppfod01/labeling/lrm/chap-07.htm>

Cancer: The U.S. EPA has strict guidelines that require testing of pesticides for their potential to cause cancer. These studies involve feeding laboratory animals large daily doses of the pesticide over most of the lifetime of the animal. Based on these tests, and any other available information, EPA gives the pesticide a rating for its potential to cause cancer in humans. For example, if a pesticide does not cause cancer in animal tests, then the EPA considers it unlikely the pesticide will cause cancer in humans. Testing for cancer is not done on human subjects.

What happens to acephate in the environment?

- Acephate is not persistent in soil. The acephate half-life in common soils range from 0.5 days in clay soil to 4 days in loamy-sand soil (12). Acephate in highly organic-content muck soil had a 13 day half-life (12). Acephate has an average soil half-life of 3 days or less (2, 13). See **Half-life** box.
- Approximately 5-10% of applied acephate degrades to the insecticide methamidophos. Methamidophos degrades quickly with a soil half-life of 2 to 6 days (12).
- Acephate has a low potential for movement through the soil to groundwater (13).
- Acephate sprayed on forest leaves for gypsy moth control had a 2-day half-life (12). Acephate has an average foliar half-life of 2.5 days (14).

Half-life is the time required for half of the compound to degrade or be eliminated from the body.

1 half-life	=	50% degraded
2 half-lives	=	75% degraded
3 half-lives	=	88% degraded
4 half-lives	=	94% degraded
5 half-lives	=	97% degraded

Remember that the amount of chemical remaining after a half-life will always depend on the amount of the chemical originally applied.

What effect does acephate have on wildlife?

- Acephate is highly toxic to honey bees. The LD50 in bees is 1.2 µg/bee (1).
- Acephate is low to moderately toxic to birds. The oral LD50s in pheasants and mallard ducks are 140 and 350 mg/kg, respectively (2). The oral LD50 in chickens is 852 mg/kg (12).
- Acephate is practically non-toxic to freshwater fish. The LC50s in rainbow trout and bluegill sunfish are both >1000 mg/L (1, 12).

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