

ACEPHATE

TECHNICAL FACT SHEET

NPIC Technical Fact Sheets provide information that is complex and intended for individuals with a scientific background and/or familiarity with toxicology and risk assessment. This document is intended to promote informed decision-making. Please refer to the General Fact Sheet for less technical information.

Chemical Class and Type:

- Acephate is an organophosphate insecticide.¹ The International Union of Pure and Applied Chemistry (IUPAC) chemical name for acephate is *O,S*-Dimethyl acetylphosphoramidothioate², and the Chemical Abstracts Service (CAS) registry number is 30560-19-1.¹
- Acephate was first registered for use by the United States Environmental Protection Agency (U.S. EPA) in 1973. Reregistration for acephate was granted in July 2006.¹ See the text box on **Laboratory Testing**.
- In soil, plants, and insects, acephate is converted to methamidophos. Methamidophos is another organophosphate insecticide that is registered by the U.S. EPA.¹

Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term (acute) and long-term (chronic) health effects. Laboratory animals are purposely given high enough doses to cause toxic effects. These tests help scientists judge how these chemicals might affect humans, domestic animals, and wildlife in cases of overexposure.

Physical / Chemical Properties:

- Technical grade acephate is a white or transparent solid.¹ Acephate has a strong odor similar to mercaptan, which smells like sulfur.³
- Vapor pressure¹: 1.7×10^{-6} mmHg at 24 °C
- Octanol-Water Partition Coefficient (K_{ow})⁴: 0.13 at 25 °C
- Henry's constant^{5,6}: 3.1×10^{-7} atm·m³/mol; 5.1×10^{-13} atm mole/m³
- Molecular weight¹: 183.16 g/mol
- Solubility (water)^{1,6}: 79 - 83.5 g/100 mL
- Soil Sorption Coefficient (K_{oc})^{2,6}: 2; 2.7

Uses:

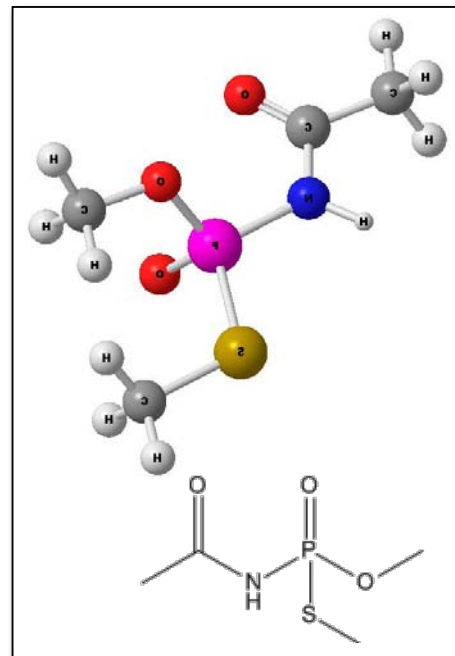
- Acephate is a general-use insecticide registered for use on food crops, agricultural seed and non-bearing plants, institutions and commercial buildings including public health facilities, sod, golf course turf, ant mounds, and horticultural nursery plants.¹
- Signal words for products containing acephate may range from Caution to Danger. The signal word reflects the combined toxicity of the active ingredient and other ingredients in the product. See the pesticide label on the product and refer to the NPIC fact sheets on [Signal Words](#) and [Inert or "Other" Ingredients](#).
- To find a list of products containing acephate which are registered in your state, visit the website http://npic.orst.edu/reg/state_agencies.html and search by "active ingredient."

Mode of Action:

Target Organisms

- Acephate is a systemic insecticide used to control sucking and biting insects by direct contact or ingestion.^{7,8}

Molecular Structure - Acephate



- Organophosphates such as acephate bind to and inhibit the enzyme acetylcholinesterase (AChE) in nervous system tissues. As a result, the neurotransmitter acetylcholine accumulates and repeatedly activates cholinergic receptors.^{9,10} Acephate itself is a weak acetylcholinesterase inhibitor.¹¹ Methamidophos is a more potent organophosphate than acephate.¹⁰ Insects metabolize acephate into methamidophos by hydrolysis, whereas mammals metabolize acephate more readily into des-O-methylacephate, accounting for acephate's relatively high selectivity against insects.^{2,12,13}
- Methamidophos inhibits acetylcholinesterase through phosphorylation.¹² Acetylcholine is the prominent insect stimulatory neurotransmitter for motor, sensory, and intermediate neurons,¹⁴ and is broken down by acetylcholinesterase.¹⁵ Organophosphates cause acetylcholine levels to increase and over-excite target nerves, muscles or tissues.¹⁰

Non-target Organisms

- Mammals are less sensitive to acephate than insects.^{2,11} The selectivity or potency of acephate is tied to mammalian metabolism.¹³ Mammalian metabolism of acephate into the cholinesterase inhibitor methamidophos is minor, compared to the main pathway of its conversion into des-O-methylacephate, also known as S-methylacetyl phosphoramidothiolate (SMPT), which is not a cholinesterase inhibitor.^{2,11}
- Organophosphates such as acephate and methamidophos inhibit cholinesterase in mammals.¹⁰ However, acephate is a weak cholinesterase inhibitor.^{10,11} Methamidophos is more toxic to invertebrates and vertebrates alike, compared to acephate.⁶
- Other studies and authors have suggested that acephate blocks methamidophos from binding to acetylcholine directly or by an allosteric site in mammals.¹⁶ See the section on Metabolism.
- In rat studies *in vitro*, methamidophos inhibited erythrocyte and brain acetylcholinesterase a thousand times more effectively than acephate.¹⁷ Another study indicated methamidophos is ten times stronger at inhibiting cholinesterase than acephate in mice.¹¹
- One study on white throated sparrow indicated brain cholinesterase inhibition after ingestion of acephate.¹⁸ A study in which hens were given acephate by gavage showed inhibition of acetylcholinesterase and, to a much lesser extent, neuropathy target esterase.¹⁹
- Acephate is generally considered non-phytotoxic, but leaf burn on Red Delicious apples after acephate application has been observed.⁷

Acute Toxicity:

- Acephate breaks down to methamidophos in the environment by microbes in the presence of oxygen.⁴ Methamidophos is more toxic to birds and mammals than its parent compound, acephate.¹

Oral

- The oral LD₅₀ for acephate is 1.4 g/kg in male rats, and 1.0 g/kg in female rats.¹ Acephate is low in toxicity when eaten. See the text boxes on **Toxicity Classification** and **LD₅₀/LC₅₀**.
- The lowest lethal dose observed in a study of Beagle dogs was 680 mg/kg.²⁰
- Human volunteers ingested a single dose of acephate. No physiological changes were observed at the highest doses tested, 1.2 mg/kg in men and 1.0 mg/kg in women.²⁰ See the text box on **NOAEL, NOEL, LOAEL, and LOEL**.

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.

ACEPHATE

TECHNICAL FACT SHEET

TOXICITY CLASSIFICATION - ACEPHATE

	High Toxicity	Moderate Toxicity	Low Toxicity	Very Low Toxicity
Acute Oral LD ₅₀	Up to and including 50 mg/kg (≤ 50 mg/kg)	Greater than 50 through 500 mg/kg (> 50 – 500 mg/kg)	Greater than 500 through 5000 mg/kg (> 500 – 5000 mg/kg)	Greater than 5000 mg/kg (> 5000 mg/kg)
Inhalation LC ₅₀	Up to and including 0.05 mg/L (≤ 0.05 mg/L) (aerosol)	Greater than 0.05 through 0.5 mg/L (>0.05 – 0.5 mg/L)	Greater than 0.5 through 2.0 mg/L (> 0.05 – 2.0 mg/L)	Greater than 2.0 mg/L (> 2.0 mg/L) (dust)
Dermal LD ₅₀	Up to and including 200 mg/kg (≤ 200 mg/kg)	Greater than 200 through 2000 mg/kg (> 200 - 2000 mg/kg)	Greater than 2000 through 5000 mg/kg (>2000 – 5000 mg/kg)	Greater than 5000 mg/kg (> 5000 mg/kg)
Primary Eye Irritation	Corrosive (irreversible destruction of ocular tissue) or corneal involvement or irritation persisting for more than 21 days	Corneal involvement or other eye irritation clearing in 8 – 21 days	Corneal involvement or other eye irritation clearing in 7 days or less	Minimal effects clearing in less than 24 hours
Primary Skin Irritation	Corrosive (tissue destruction into the dermis and/or scarring)	Severe irritation at 72 hours (severe erythema or edema)	Moderate irritation at 72 hours (moderate erythema)	Mild or slight irritation at 72 hours (no irritation or erythema)

The highlighted boxes reflect the values in the “Acute Toxicity” section of this fact sheet. Modeled after the U.S. Environmental Protection Agency, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling. <http://www.epa.gov/oppfead1/labeling/lrm/chap-07.pdf>

Dermal

- The dermal LD₅₀ was greater than 10,000 mg/kg in male rabbits.¹
- Acephate is not a skin sensitizer in guinea pigs.^{1,17,21}
- Acephate is not considered an eye irritant by the EPA, based on a study in rabbits.¹
- In another study, acephate caused inflammation of the eye membrane and iris, and minor corneal opacity in rabbits. Symptoms were reversed within two weeks.^{17,22}
- After administering up to 0.1 ml of acephate, slight eye redness and fluid discharge were observed in rabbits. When eyes were rinsed a minute after treatment, researchers noticed swelling. In both cases, rabbits’ eyes recovered within 24 hours.^{17,23}

NOAEL: No Observable Adverse Effect Level
NOEL: No Observed Effect Level
LOAEL: Lowest Observable Adverse Effect Level
LOEL: Lowest Observed Effect Level

Inhalation

- Acephate is very low in toxicity when inhaled, with an LC₅₀ of greater than 61.7 mg/L.¹
- The LC₅₀ value for inhalation of aerosol containing acephate dissolved in distilled water is greater than 15 mg/L air over 4 hours in rats.^{17,24}
- Rats were exposed exclusively to air passed over acephate crystals for four hours. No fatalities or changes in cholinesterase levels were observed.²⁵
- Rats were exposed to aerosol droplets containing from 0-4% acephate for an hour every day, five days a week for two weeks. No effects were observed at 1% acephate. Increases in uric acid were observed in females after exposure to 4% acephate. No other clinical changes were observed.²⁵

Signs of Toxicity - Animals

- Acephate ingestion in mice caused muscular weakness, tremors, and reduced activity. Signs resolved within nine days in survivors.²⁰

ACEPHATE

TECHNICAL FACT SHEET

- Acephate ingestion in rats caused depression, decreased food consumption, lethargy, diarrhea, excess salivation, urinary incontinence, tremors, ataxia, and collapse. Signs in survivors resolved within eight days of administration.²⁰
- Acephate ingestion in beagle dogs resulted in muscular tremors, diarrhea, emesis, dyspnea, ataxia, clonic convulsions and bloody diarrhea until six hours after acephate administration.²⁰
- Dermal exposure to acephate on rabbits caused tremors, diarrhea and dermal redness and inflammation of the treated skin.²⁰
- Acephate inhalation in rats caused excess salivation, tremors, ataxia and lethargy. Signs resolved within four days of administration.²⁰
- Signs of toxicity noted in birds were incoordination, depression, shortness of breath, feather puffing, dropped wings, falling rigidly with outspread wings, lying down, tremors, and convulsions. Signs and death have been observed as soon as 30 minutes and five hours, respectively, after acute ingestion.²⁶

Signs of Toxicity - Humans

- Symptoms appear rapidly if the organophosphates are inhaled, somewhat slower if ingested, and more delayed following dermal exposure. Symptoms from organophosphates can become apparent within minutes to hours after exposure, depending on the exposure route.¹⁰
- Symptoms include headache,¹⁰ nausea, dizziness and confusion.¹ Incident reports to the EPA about exposure to acephate-containing pesticides were most frequently associated with gastrointestinal, neurological, respiratory, or dermal symptoms such as nausea, diarrhea, abdominal cramps, tremors, tachycardia, sweating, disorientation, coughing, wheezing, congestion, and pneumonia. Skin irritation and reactions such as swelling, hives, redness, and rashes have also been reported.²⁷
- Overexcitation of central nervous system from organophosphates may result in moodiness or agitation, confusion, lethargy, weakness, convulsions, incoordination, memory loss, cyanosis or coma.⁹ When the muscarinic receptors are over-excited in the parasympathetic nervous system, cholinergic signs and symptoms including abdominal cramps and diarrhea, hypersecretion, urination, tightening of the bronchi, decreased or increased heart rate, and miosis may occur.⁹ Severe exposures to acephate can cause respiratory paralysis and death.¹
- Children can experience different symptoms than adults if exposed to organophosphates, including seizures, lethargy, and coma, flaccid muscle weakness, miosis and excessive salivation.¹⁰
- Always follow label instructions and take steps to avoid exposure. If any exposures occur, be sure to follow the First Aid instructions on the product label carefully. For medical emergencies or diagnosis and treatment advice, contact the Poison Control Center at 1-800-222-1222. If you wish to report an incident, please call the National Pesticide Information Center at 1-800-858-7378.

Chronic Toxicity:

Animals

- Acephate was given to rats in their diets at concentrations of 0, 2, 5, 10, and 150 ppm for 13 weeks. After 13 weeks, cholinesterase activity in the brain and red blood cells was inhibited at all doses. The red blood cell and plasma cholinesterase inhibition NOEL was 0.58 mg/kg/day and 0.76 mg/kg/day for male and female rats, respectively. Brain cholinesterase inhibition was detected in all treated rats. No inhibition of plasma or red blood cell cholinesterase was observed at 10 ppm doses.²⁸
- Rats were given 1 or 10 mg/kg acephate by gavage daily for 15 weeks.²⁹ Brain acetylcholinesterase activity changes were not observed in either group.²⁹ Decreases in plasma and red blood cell cholinesterase activities, and increased catecholamine were observed in the 10 mg/kg treatment group.²⁹ Brains from acephate-treated rats showed increased epinephrine, nor-

epinephrine, glutamine and asparagine and decreased GABA, dopamine, glutamic acid decarboxylase and tyrosine compared to controls.²⁹ Rats in the higher treatment group had increased urination, fasciculation, and hyperactivity in the last few weeks of treatment.²⁹

- Brain cholinesterase inhibition was observed in both sexes of beagle dogs given 120 or 800 ppm per day acephate for 52 weeks. In males, the effect was also seen at 10 ppm. No overt symptoms were observed in the dogs.²⁰
- In a study of oral acephate exposure in hens, authors concluded that organophosphate-induced delayed neuropathy (OPIDN) was implausible based on the low neuropathy target esterase inhibition at acephate levels that were life threatening or fatal to tested hens.¹⁹ In another study, hens were administered the median lethal oral dose of acephate in a delayed neurotoxicity study. No evidence of delayed neurotoxicity such as neurotoxic symptoms or neural tissue damage was observed.¹⁷

Humans

- Fourteen volunteers ingested mixtures of acephate and methamidophos. Plasma cholinesterase inhibition was observed, and the LOAEL was established for a 1:4 ratio of methamidophos:acephate at 0.2 mg/kg/day in both sexes after 16 days of administration.²⁵ No other changes were observed. Cholinesterase activities returned to normal within seven days.¹⁷ See the text box on **Exposure**.

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.

- Ten male volunteers each ingested 0.25 mg/kg/day acephate for 28 days. No acetylcholinesterase inhibition, clinical or physiological changes were observed.²⁰
- The intermediate syndrome has been attributed to methamidophos following acute, symptomatic poisoning. Health effects from intermediate syndrome may include limb, neck, and respiratory muscle weakness, or death. Intermediate syndrome onset begins a day or more after initial acute symptoms begin or resolve and may last 30 to 40 days. No cases linking these conditions to acephate were found.
- Organophosphate-induced delayed (poly)neuropathy (OPIDN) has been reported from methamidophos exposure. OPIDN onset is two or three weeks after some acute poisonings, and has only been observed in cases with acute cholinergic symptoms. OPIDN effects may include leg muscle pain, followed by numbness, weakness or paresthesia beginning in the feet and sometimes hands.³⁰

Endocrine Disruption:

- In one study, reversible hyperglycemia and adrenal cortex hyperactivity were observed two to six hours after fasted male rats were fed 140 mg/kg acephate.³¹
- In another study, rats were given 178 mg/kg acephate by gavage daily for four, fourteen, or sixty days.³² Overall, brain serotonin and metabolite levels decreased after treatment, but the pattern of change varied in different sections of the brain.³²
- Female Wistar rats were injected with 500 mg/kg acephate or 5 mg/kg methamidophos intra-peritoneally. Increases in serum corticosterone and aldosterone, and decreases in serum glutamate and histidine levels were observed after both treatments. According to the authors, the results suggest that acephate and methamidophos injection increase energy needs and gluconeogenesis in treated rats.¹⁶

- Acephate or methamidophos exposure to rat hypothalamus tissue isolated *in vitro* resulted in increased corticotropin-releasing factor (CRF) expression and release, even in the presence of a factor that normally inhibits CRF.³³
- Acephate is on U. S. EPA's June 2007 Draft List of Chemicals for Initial Tier 1 Screening list for the Endocrine Disruptor Screening Program (EDSP).³⁴ Priority for endocrine disruption testing is set for chemicals based on the likelihood of human exposure to them.³⁴

Carcinogenicity:

Animals

- Significant increases in hepatocellular carcinomas and adenomas were observed in female mice fed 1000 ppm acephate in their diet for two years. In male rats that were fed the same doses, no significant increases in tumor incidence was noticed.¹⁷
- Only male rats showed increased frequency of adrenal gland tumors at dietary doses of acephate up to 700 ppm for 28 months. The relationship between tumor occurrence and acephate dose was not linear.¹⁷
- According to one source, acephate was a weak mutagen in studies of *Salmonella*, *Escherichia coli*, and *Saccharomyces cerevisiae*.²⁸ Another source cited inconsistent genotoxicity results in acephate studies among several prokaryotic and eukaryotic cell types.³⁵
- Results from acephate genotoxicity studies using whole animals have been mixed, indicating both positive and negative results.^{17,36}
- In another study, male mice were fed acephate doses ranging from 12.25 to 392 mg/kg by intubation. DNA damage was observed in cells of all treated mice. DNA damage repair began in lower dose groups by 72 hours and was complete in all dose groups by 96 hours. The level of DNA damage correlated to the dose.³⁵

Humans

- The U.S. EPA designated acephate as Group C, possible human carcinogen based on increases in liver adenomas and carcinomas in female mice that ate acephate.²⁸ See the text box on **Cancer**.

Cancer: Government agencies in the United States and abroad have developed programs to evaluate the potential for a chemical to cause cancer. Testing guidelines and classification systems vary. To learn more about the meaning of various cancer classification descriptors listed in this fact sheet, please visit the appropriate reference, or call NPIC.

- No human data were found on carcinogenic effects of acephate.²⁸
- Methamidophos is not likely to be a human carcinogen according to U.S. EPA classification.³⁷

Reproductive or Teratogenic Effects:

Animals

- In multi-generational and prenatal development studies, fetuses and pups were no more sensitive to acephate than the adult rats or rabbits.¹
- Male mice were fed acephate five days a week for four weeks at doses up to 28 mg/kg/day, and then mated with untreated female mice.¹² At doses over 7 mg/kg/day, they observed decreases in mating, male fertility, sperm counts and motility, implantations and live fetuses, and increased resorptions.¹² Testes weighed less in all treated mice.¹²

- Multigenerational exposure to acephate in rats resulted in reduced body weight gain, increased food consumption, and reductions in mating and litter size.¹⁷
- No changes in nervous system development were found in multigenerational acephate exposure studies, including behavioral and cognitive effects.^{1,20} No developmental or neurotoxic effects were observed in offspring at doses up to 10 mg/kg/day.²⁰
- In considering environmental risks, the U.S. EPA concluded that chronic exposure to acephate and methamidophos may decrease offspring survival and body weight in wild mammals.¹

Humans

- No human data were found on the teratogenic or reproductive effects of acephate.

Fate in the Body:

Absorption

- When acephate is eaten, it is quickly absorbed from the gastrointestinal tract in rats and humans.^{20,38}
- Acephate is readily absorbed by the skin of rats.^{17,39,40}
- Acephate in aerosol formulations is absorbed by inhalation.¹⁷

Distribution

- In rats, dermally absorbed acephate is distributed into the blood. Blood concentrations of acephate were highest 1 to 3 hours after exposure.^{39,40}
- In rats given acephate by oral gavage, acephate evenly distributes to tissues according to their level of blood circulation. Accordingly, highest acephate concentrations were found in the skin, liver, kidney and heart, and were comparable to blood plasma acephate concentrations.²⁰
- In two human deaths involving presumed intentional ingestion, acephate was detected most in the stomach and urine, and in lesser amounts in blood. One of the subjects had ingested 100 mL or less of a product containing 17.25% acephate. The ratios of methamidophos to acephate in the stomach, urine and blood were at or below 2:100.⁴¹

Metabolism

- Acephate in mammals remains mostly unmetabolized.²⁰ Mammalian metabolism of acephate into the more potent cholinesterase inhibitor methamidophos is minor, compared to the main pathway of its conversion into the metabolite des-O-methylacephate. The des-O-methylacephate is further metabolized into molecules that are integrated into cellular biochemicals, or excreted.²
- After dermal exposure in rats, one study found 1% of administered acephate was excreted from urine as methamidophos.^{17,39}
- In one study, urinary metabolites of acephate in rats after oral administration included 73-77% acephate, 3-6% O-S-dimethylphosphorothioate, and 3-4% S-methyl-acetylphosphoramidothioate. Their assay did not indicate the presence of methamidophos.^{40,42}
- According to another study, microbes biodegraded a small percentage of the orally-given acephate to methamidophos in the rat gastrointestinal tract; 0.5-1.5% of dosed acephate was detected both in rat tissues and in rat urine as methamidophos.¹⁷

- In one study, mice fed a single 120 µg/g dose of acephate by tube were evaluated for acephate metabolites in the liver.¹¹ After thirty minutes, 19-30% acephate was detected in the liver. The major metabolite was S-methylacetyl phosphoramidothiolate, which peaked at 8-9% seven hours after feeding. After 30 minutes, 1.5% of the dose was identified as methamidophos. After 24-30 hours, only O,S dimethyl phosphorothiolate was detectable in the liver at very small levels.¹¹
- In hens given acephate by gavage, the proportion of methamidophos detected in the brain decreased as doses increased. At 25 mg/kg acephate doses, 16% methamidophos was observed; at 700 mg/kg acephate doses, methamidophos levels averaged 10%.¹⁹

Excretion

- Acephate is rapidly excreted from mammals.²⁵ After ingestion in rats, 82-95% of the acephate dose was rapidly excreted in the urine, 1-9% through exhalation as carbon dioxide, and 1-2% in feces.^{40,42} Less than 1% of orally administered acephate in rats was detected in tissues 72 hours after exposure.^{20,38}
- Acephate plasma elimination was non-linear in rats; the elimination half-life is 1.4 hours during the first two hours, and 50 hours beginning 24 hours after the exposure.²⁰
- After maternal oral exposures, rat offspring can be exposed to acephate through the placenta or milk.¹⁷
- One study measured urine levels of acephate and methamidophos in humans exposed to acephate while farming in fields. Acephate was detected in urine, but methamidophos was not. Daily maximum acephate urine concentrations ranged from 3 to 9 mg/L.⁴⁰ Urinary concentrations of acephate were undetectable in less than 48 hours after exposure ended.⁴⁰

Medical Tests and Monitoring:

- Red blood cell (RBC) acetylcholinesterase or plasma butylcholinesterase inhibition can be measured as a biomarker of effect for acute and chronic exposure to organophosphates.^{10,30,43} The level of RBC cholinesterase inhibition detected is associated with symptom severity in poisonings.³⁰
- Some agencies recommend or require periodic cholinesterase level testing for people who handle organophosphates in their work.^{43,44,45} Blood cholinesterase levels differ widely among people, and in individuals over time.^{30,43} Workers are often tested before any potential exposure to establish a baseline.^{43,44,45} Post-exposure blood cholinesterase samples can then be compared to pre-exposure samples from the same individual.⁴³
- When occupational exposures occur or a worker complains of illness, tests are optimally performed within 24 hours.⁴³ Cholinesterases are continuously resynthesized by the body.³⁰
- Other methods of testing for organophosphate exposure exist, such as detecting organophosphate metabolites in urine. However, they require advanced technology and are not usually available in clinical settings. Their clinical significance is unclear.^{30,43}

Environmental Fate:

Soil

- In soil, acephate breaks down primarily by aerobic metabolism and to a lesser degree by photolysis to methamidophos, which is another organophosphate insecticide registered by the U.S. EPA.^{1,6} Twenty three percent of applied acephate converted to methamidophos by aerobic metabolism in loam soil, and less than 10% did so in two other soil types.⁶
- By soil photolysis, 8.4% or less converted to methamidophos.⁶ Methamidophos is then mineralized to carbon dioxide and microbial biomass by soil microorganisms with a half-life of less than ten days.^{1,6} See the text box on **Half-life**.

ACEPHATE

TECHNICAL FACT SHEET

- In laboratory studies, the calculated half-life of acephate ranged from about 4.5 days in half-saturated silt loam soil to 32 days in half-saturated silt clay loam soil.⁵ The half-life of acephate is less than two days in conditions anticipated by the U.S. EPA to occur from use.¹
- In other laboratory studies, the calculated half-life of methamidophos ranged from roughly one day in saturated silt loam soil to 13 days in half-saturated silt clay loam soil.⁵ The half-life of methamidophos is expected to be less than ten days under use conditions anticipated by the U.S. EPA.¹
- Soil mobility of acephate and methamidophos is expected to be high.¹
- Field studies of acephate applied to crops found that acephate and methamidophos were undetectable at 50 cm depths in silt loam, sand, and loam soils for the duration of the study.¹ Dissipation half-lives measured in these studies were two days or less.^{1,6}
- One study conducted on soil where tomatoes were grown found 11.3% of applied radiolabeled acephate had broken down to methamidophos after one week, and it was the primary metabolite found.²

The “half-life” is the time required for half of the compound to break down in the environment.

1 half-life = 50% remaining

2 half-lives = 25% remaining

3 half-lives = 12% remaining

4 half-lives = 6% remaining

5 half-lives = 3% remaining

Half-lives can vary widely based on environmental factors. The amount of chemical remaining after a half-life will always depend on the amount of the chemical originally applied. It should be noted that some chemicals may degrade into compounds of toxicological significance.

Water

- Despite acephate's high solubility and mobility in water, acephate is considered a low risk of groundwater contamination because acephate and methamidophos do not persist in the environment.^{1,46}
- Acephate is not likely to volatilize from water because it is highly water soluble.⁴
- Acephate does not break down readily by photolysis in water. Only 1.6% broke down to methamidophos in this medium.^{4,6}
- The half-life of acephate in the anaerobic clay sediment of a creek was between six and seven days. Acephate degraded mostly to methane and carbon dioxide in this study.⁶
- Acephate hydrolysis is slow, but occurs faster at higher pHs.⁶ The half-life at pH 9 was 18 days. When mixed with distilled water of pH levels 4-6 in laboratory conditions, 82-98% acephate was recovered from water after 20 days, and 0.2% of added acephate was recovered as methamidophos.⁴
- Acephate was mixed with distilled water of pH 7.2, 8.1, and 8.6 and isolated in laboratory conditions at 37 °C for seven days. About 40-60% of acephate was hydrolyzed mainly to O, S dimethyl phosphorothiolate within the first three days. After three days decreases in acephate levels were small or unobservable.¹¹ Methamidophos and O-methylacetyl phosphoramidothiolate were minor products throughout this time course.¹¹
- A product containing acephate was applied to three sites in a stream in British Columbia, Canada for five hours at levels amounting to 1000 ppb.⁴⁷ Acephate and methamidophos levels were measured in water and in river sediment at time intervals up to 96 hours.⁴⁷ Acephate levels detected in nearby sediment were highest four or eight hours after application began, which was three or seven hours after peak concentrations of acephate were observed in the treated water.⁴⁷ Methamidophos was not detected in any of the water samples.⁴⁷

Air

- Acephate is not volatile in ambient conditions.⁴ In one study, creek and pond water with added acephate were isolated in flasks in laboratory conditions, and no acephate or methamidophos were detected from the air for up to fifty days.⁴⁸

- At air saturation in environmental temperatures, acephate would be present in the air at 0.002 ppm.⁴

Plants

- Acephate was rapidly translocated and incorporated into plants when taken up from soil.² Translocation occurs in the direction of roots to foliage, not from foliage to roots.²⁵
- In plants, acephate is primarily metabolized into methamidophos, which is a more potent insecticide. Methamidophos found in leaves equaled 2-10% of the soil-applied acephate. These studies included tomatoes, beans, maize, fruit, spruce trees and cones, *Capsicum*, and cucumber. No other metabolites were identified.²
- Acephate residues on treated leafy vegetables were highest on the outer leaves.²⁵
- The half-life of acephate in tomatoes was 7-14 days.²⁵
- Surface application of acephate to the peel of citrus fruit penetrated and distributed evenly into the fruit flesh.²⁵
- Acephate applied to raspberry and cherry plants days before flowering resulted in residues of 0.15-2.84 ppm in nectar after flowering. One raspberry plant's nectar contained 14.39 ppm acephate.⁴⁹

Indoor

- No data regarding the indoor environmental fate of acephate were found.

Food Residue

- In 2008, 8686 samples of produce were tested for acephate.⁵⁰ Four hundred ninety-one samples (5.7% of tested samples) had detectable acephate residues.⁵⁰ The majority of samples with acephate were celery or green beans.⁵⁰ That year, acephate was detected infrequently in spinach, summer squash, nectarines, blueberries, collard greens, strawberries, and tomatoes.⁵¹
- In 2006, 8315 samples of produce were tested for acephate.⁵¹ Seventy-one samples (0.85% of tested samples) had detectable acephate residues. Produce with detected acephate were mostly cranberries, spinach, or watermelon, and less frequently winter squash, eggplant, and applesauce.⁵¹
- In 2008, 9694 samples of produce were tested for methamidophos. Four hundred thirty-nine samples, (4.5% of samples) showed detectable levels of methamidophos.⁵⁰ The majority of samples in which methamidophos was detected were celery, green beans, or tomatoes.⁵⁰ That year, methamidophos was detected infrequently in peaches, summer squash, asparagus, nectarines, spinach, blueberries, and potatoes.⁵¹
- In 2006, 7524 samples of produce were tested for methamidophos.⁵¹ Ninety-four samples (1.25% of tested samples) had detectable methamidophos residues. Produce with detected methamidophos most often were eggplant, watermelon, or cranberries, and less frequently peaches, winter squash, spinach, frozen potatoes, frozen sweet peas, and applesauce.⁵¹

Ecotoxicity Studies:

Birds

- Acephate is moderately toxic to birds. According to the U.S. EPA, oral LD₅₀s ranged from 51-500 mg/kg in different bird species.¹ One study found the LD₅₀ for acephate in hens was 800 mg/kg.¹⁹
- Acephate's degradation product, methamidophos, can be very highly toxic to birds on an acute basis. The LD₅₀s for methamidophos ranged from less than 10-50 mg/kg.¹
- Blackbirds (*Euphagus cyanocephalus*), robins (*Turdus migratorius*), and starlings (*Sternus vulgaris*) approached lethality two hours after oral exposure to 50-100 mg/kg acephate.^{26,52} The average LD₅₀ for acephate in dark-eyed juncos (*Junco hyemalis*)

lis) was 106 mg/kg, and for methamidophos was 8 mg/kg.²⁶ Juncos given 26.52 mg/kg acephate by gavage showed brain cholinesterase inhibition recovery between three and six days after treatment.²⁶

- In multiple studies, most birds that died after acephate exposure had about 50% or greater brain cholinesterase inhibition.^{26,53,54}
- Bobwhite quail (*Colinus virginianus*) were fed 80 ppm acephate in a chronic ingestion study. Body weight, number of eggs laid, and embryo/hatchling viability were all reduced.¹ In mallards (*Anas platyrhynchos*) chronically fed 20 ppm acephate, the number of viable embryos and living embryos after three weeks was reduced.¹ Egg thinning was observed in bobwhite quail when parents were fed 5 ppm methamidophos on a chronic basis.¹
- Field studies and incident reports indicated the potential for adverse effects to songbirds, including migratory pattern disruption or death, after acephate field applications. In the field studies, adverse effects were delayed one or two days.¹ Cholinesterase inhibition was observed in birds up to 33 days after 1.13 kg/ha treatment of a plot of land, and up to 26 days after 0.57 kg/ha treatment.^{26,53}
- In one study, white-throated sparrows (*Zonotrichia albicollis*) were fed 256 ppm acephate in their food. Adult birds selected random migratory directions compared to controls. Juvenile birds went south, whether or not they were fed acephate.⁵⁵ Authors concluded acephate affected memory.⁵⁵
- One study exposed white-throated sparrows to acephate in the diet over fourteen days. Results indicated an atypical dose response relationship.¹⁸ At doses up to 16 ppm, the birds seemed to adapt to exposure by replenishing cholinesterase activity in brain tissues. At doses between 16 and 1024 ppm, cholinesterase activity continued to decrease.¹⁸

Fish and Aquatic Life

- According to the U.S. EPA, acephate is considered practically non-toxic to slightly toxic to freshwater fish for short-term exposures. The LD₅₀ ranges from 50 to greater than 100 ppm.¹ In other studies, the 24-hour LC₅₀ for rainbow trout ranged from 900-2800 ppb.⁴⁷ Up to tenfold bioaccumulation in bluegill sunfish (*Lepomis macrochirus*) was observed after fourteen days of exposure to 0.007 and 0.7 ppm acephate.⁶
- Methamidophos is slightly toxic to freshwater fish for acute exposures with LD₅₀s ranging from 10-100 ppm.¹
- A product containing acephate was applied to a stream in British Columbia, Canada for five hours at levels amounting to 1000 ppb.⁴⁷ While no fish deaths were observed, acephate and methamidophos were detected in fish in two out of three sites up to eight hours after treatment.⁴⁷ The percentage of methamidophos compared to acephate ranged from about 4-10%, when detected.⁴⁷
- Acephate is practically non-toxic to slightly toxic in estuarine and marine fish for acute exposures; LC₅₀ ranges from 50 to greater than 100 ppm. Methamidophos is moderately toxic to estuarine or marine fish for acute exposures; LC₅₀s range from 1-10 ppm.¹
- According to U.S. EPA sources, acephate is practically non-toxic to amphibians.¹ In one study, a 24-hour LC₅₀ of 6433 ppm was reported for green frog (*Rana clamitans*) tadpoles. No behavioral changes were observed at 500 ppm acephate. In another green frog tadpole study, no toxicity or bioaccumulation was seen at 5 ppm.⁶
- A salamander (*Caudata*) study identified a 96-hour LC₅₀ of 8816 ppm acephate for salamander larvae.⁵⁶ When salamander egg masses were exposed to acephate at varying concentrations in the water, researchers observed muscle deformities and bent vertebral columns at 382 mg/L, decreased growth, feeding, and lethargy at 798 mg/L, and the most sensitive stage was the first week after hatching. Hatching time was not affected at the highest dose tested, 798 mg/L.⁵⁶

ACEPHATE

TECHNICAL FACT SHEET

- Acephate has low to moderate toxicity to aquatic invertebrates for acute exposures (LC_{50}/EC_{50} ranged from 1 to greater than 100 ppm).¹ Methamidophos is very highly toxic to aquatic invertebrates for acute exposures (EC_{50} less than 0.1 ppm).¹ See the text box on EC_{50} .
- Acephate affects daphnid reproduction, reducing the number of young. The lowest tested concentration that produced this effect was 0.375 ppm. The daphnid No Observable Adverse Effect Concentration (NOAEC) was 0.150 ppm in this study.¹

Terrestrial Invertebrates

- Acephate and methamidophos are highly toxic to bees and other beneficial insects. The LD_{50} for acephate was 1.2 μ g/honey bee. The LD_{50} of methamidophos was 1.37 μ g/honey bee.¹
- In one study, bee colonies were fed sugar syrup treated with 0.25, 0.5, or 1.0 ppm acephate for 14 days.⁴⁹ At 0.25 ppm, the number of eggs was reduced.⁴⁹ At higher doses, some queens stopped laying eggs, others were lost.⁴⁹ At 1 ppm levels, bee flight activity and foraging were slowed.⁴⁹ Over time, other effects were observed in all treatment groups, including decreases in larvae and pupae rearing, newly built comb, feeding and food storage.⁴⁹
- A product containing acephate was applied to three sites in a stream in British Columbia, Canada for five hours at levels amounting to 1000 ppb.⁴⁷ The highest level of acephate was 1189.5 ppb, detected in insects two and a half hours after the application began. About two hours after the treatment, acephate was found in insects at only trace levels and was undetectable at 24 hours.⁴⁷ Acephate levels in insects were comparable to nearby water, about ten times higher than those found in fish or sediment, and highest in stream bottom insects compared to insects in cages.⁴⁷ No insect deaths were observed in this study.⁴⁷

Reference Dose (RfD): The RfD is an estimate of the quantity of chemical that a person could be exposed to every day for the rest of their life with no appreciable risk of adverse health effects. The reference dose is typically measured in milligrams (mg) of chemical per kilogram (kg) of body weight per day.

Regulatory Guidelines:

- The aPAD/aRfD is 0.005 mg/kg/day and the cPAD/cRfD is 0.0012 mg/kg/day.¹ See the text box on **Reference Dose (RfD)**.
- The U.S. EPA has classified acephate as a Group C, possible human carcinogen. Increases in liver carcinomas and adenomas were found in acephate studies using female mice.²⁸ See the text box on **Cancer** (page 6).
- The Drinking Water Concentration of acephate that poses a risk level of one in 1,000,000 additional cancers is 4 μ g/L.²⁸
- The chronic RfD for acephate in rat studies was 4×10^{-3} mg/kg/day, based on brain cholinesterase inhibition.²⁸

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