

MALATHION

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:

- Malathion is an organophosphate insecticide. The Chemical Abstracts Service (CAS) registry number is 121-75-5 and the International Union of Pure and Applied Chemistry (IUPAC) name for malathion is O,O-dimethyl dithiophosphate of diethyl mercaptosuccinate.¹
- Malathion was first registered for use in the United States in 1956 by the United States Department of Agriculture (USDA), and it is now regulated by the United States Environmental Protection Agency (U.S. EPA).¹ See the text box on **Laboratory Testing**.

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.

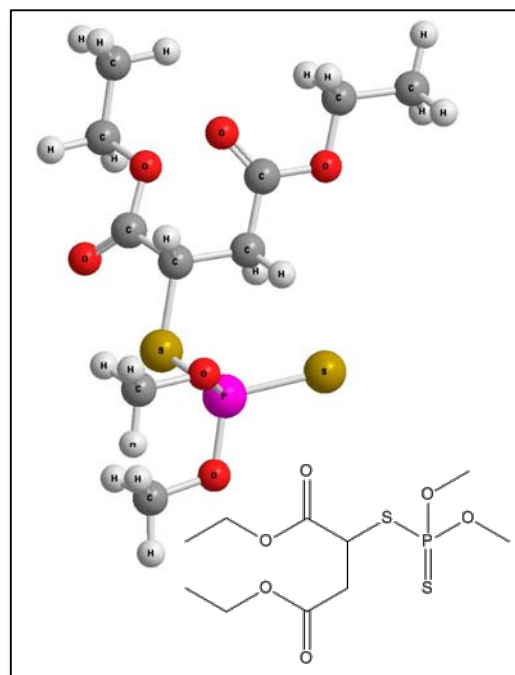
Physical / Chemical Properties:

- Malathion is a colorless to amber liquid with a skunk- or garlic-like odor.²
- Vapor pressure^{2,3,4}: 1.78×10^{-4} mmHg at 25 °C or 5.3 mPa at 30 °C; also 1.2×10^{-4} to 8×10^{-6} mmHg at 20 °C
- Octanol-Water Partition Coefficient ($\log K_{ow}$)^{5,6}: 2.75, 2.36-2.89
- Henry's constant may be estimated or derived experimentally. An experimental value of $2.0 (\pm 1.2) \times 10^{-7}$ (n = 6 experimental values, dimensionless units) is reported based on a wetted-wall column, concentration/concentration method.⁷ This value has been cited elsewhere as 4.9×10^{-9} atm·m³/mol.² Additional estimated values range from 2.4×10^{-7} to 1.0×10^{-6} at varying temperatures.⁷ An additional value of 5.68×10^{-8} mmHg has been reported.⁸
- Molecular weight³: 330.4 g/mol
- Solubility (water)³: 145 mg/L
- Soil Sorption Coefficient (K_{oc})^{2,4}: 30, 93-1800 depending on soil type and environmental conditions.

Uses:

- Malathion is a broad-spectrum insecticide used to control a variety of outdoor insects in both agricultural and residential settings. Malathion is registered for use on food, feed, and ornamental crops and in mosquito, boll weevil and fruit fly eradication programs.¹ Uses for individual malathion products vary widely. Always read and follow the label when applying pesticide products.
- Malathion is also an ingredient in shampoos regulated by the United States Food and Drug Administration (FDA) to control head lice.¹
- Signal words for products containing malathion 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 malathion which are registered in your state, visit the website http://npic.orst.edu/reg/state_agencies.html and search by "active ingredient."

Molecular Structure - Malathion



Mode of Action:

Target Organisms

- Malathion is toxic via skin contact, ingestion, and inhalation exposure.³
- Malathion and other organophosphate insecticides bind to the enzyme acetylcholinesterase (AChE) at nerve endings throughout the bodies of insects and other organisms.⁹ Under normal circumstances, AChE binds to the neurotransmitter acetylcholine (ACh) at the nerve junction, effectively ending the stimulation of the next neuron. When AChE is bound by malathion's metabolite malaoxon, ACh accumulates at the nerve junction and results in overstimulation of the nervous system.⁹
- Bioactivation of malathion is necessary for it to exert its toxic effect. Bioactivation is primarily mediated by cytochrome P450 enzymes in the liver, which create the active metabolite malaoxon through oxidative sulfuration.^{5,10}
- Malaoxon is considered to be 22 times more toxic than the parent malathion from acute dietary exposure and 33 times more toxic by all routes of exposure from short-term and medium-term exposures.¹¹
- The organophosphate pesticides, including malathion, share a common mode of action. Exposure to multiple organophosphates can lead to additive toxicity. However, the different organophosphates vary widely in their potency and how well they are absorbed by the body depending on the route of exposure.⁹
- Storage of malathion products for a long period of time may allow the accumulation of degradation products that inhibit the liver enzymes responsible for malathion detoxification.⁹ Heating malathion may also lead to the formation of isomalathion, which is a potent AChE inhibitor.⁵ See the section on Metabolism for more information.

Non-target Organisms

- Acetylcholinesterase is found in mammals, amphibians, fish, reptiles, and birds.¹² In these organisms, the binding of AChE with malathion allows the accumulation of ACh at the nerve junction. This accumulation of ACh leads to overstimulation of glandular cells, autonomic ganglia, the central nervous system, and both smooth and skeletal muscles.⁹
- Uptake and metabolism of organophosphates such as malathion are similar in insects and mammals.¹³
- Mammals and birds have greater carboxylesterase activity relative to levels in insects. This enables birds and mammals to degrade malathion more quickly than it is oxidized to the malaoxon form. Higher vertebrates therefore detoxify and excrete malathion more readily than do insects. This accounts for the relatively low toxicity of malathion to mammals and birds.^{10,14,15}
- Greater carboxylesterase production with consequent increased detoxification of malathion appears to be the underlying mechanism in resistant insect pests.¹⁵
- Microorganisms such as bacteria may use malathion as a source of carbon and phosphorus.¹⁵
- Plants metabolize malathion to malaoxon although this appears to be a minor pathway, and malaoxon is rapidly eliminated.^{5,15} Malathion is not expected to be toxic to plants or aquatic algae because its mode of action targets nervous systems.¹

Acute Toxicity:

Oral

- Malathion is very low in toxicity when ingested. The acute rat LD₅₀ is 5400 mg/kg in males and 5700 mg/kg in females.¹ The low toxicity is due to rapid carboxylesterase enzyme metabolism of malathion.¹⁰ See the text boxes on **Toxicity Classification** and **LD₅₀/LC₅₀**.
- The acute LD₅₀ in mice ranges from 400-4000 mg/kg.^{16,17}

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- The acute LD₅₀ in rats ranged from 1000 to 12,500 mg/kg.^{16,17}

Dermal

- Malathion is low in toxicity when applied to the skin. The acute dermal LD₅₀ in rats is >2000 mg/kg. Based on this value, the U.S. EPA considers malathion to be low in toxicity.¹
- Additional dermal LD₅₀ values were greater than 4000 mg/kg in rats and 4100-8800 mg/kg in rabbits.^{3,16}
- In a skin-irritation study, malathion caused slight skin irritation in rabbits. The U.S. EPA considered malation to be very low in toxicity based on these results. Malathion is not considered a skin sensitizer based on studies with guinea pigs.¹
- In an eye-irritation study with rabbits, malathion caused slight eye irritation that cleared within 7 days. The U.S. EPA considered malathion to be low in toxicity regarding dermal eye irritation.¹

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.

Inhalation

- Malathion is very low in toxicity when inhaled, with an acute LC₅₀ in rats of >5.2 mg/L.¹
- Researchers exposed mice to aerosolized technical grade malathion and determined that the LD₅₀ for the rats and mice combined was 6.9 mg/L for inhalation exposure.¹⁸
- In another study, rabbits and quail were exposed to aerosols of technical grade malathion at concentrations of 6, 34, 65, and 123 mg/m³ via an ultra low volume spraying apparatus.¹⁹ Quail showed reduced plasma CHe activity after exposure to concentrations of malathion of 34 mg/m³ or greater, although no quail died and none showed outward signs of toxicity.¹⁹

Signs of Toxicity - Animals

- Malathion disrupts the cholinergic system, and basic clinical signs will be similar in humans and other mammals.²⁰ Cholinergic receptors including the muscarinic, nicotinic, and central nervous system receptors are all affected by exposure to malathion.⁹

TOXICITY CLASSIFICATION - MALATHION

	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 (> 0.5 – 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>

- Muscarinic effects resulting from overstimulation of the nervous system, specifically the postganglionic parasympathetic receptors, include salivation, lacrimation (production of tears), urination and defecation (the SLUD syndrome), vomiting, dyspnea (shortness of breath), bradycardia (reduced heart rate), abdominal pain, miosis (constriction of the pupils), and anorexia.²¹
- Overstimulation of the nicotinic acetylcholine receptors in the nervous system results in muscle tremors and rigidity, weakness and loss of limb mobility, and paralysis.²¹
- Central nervous system overstimulation may lead to depression, anxiety, hyperactivity or restlessness, reduced respiration, seizures, and coma.²¹
- Both organophosphate-induced delayed neuropathy (OPIDN) and Intermediate Syndrome have been reported in animals²¹ as well as people following very high exposure to some organophosphates (see below). However, no specific information was found linking malathion exposure in animals or people with either syndrome.

Signs of Toxicity - Humans

- Signs and symptoms of toxic exposure depend on the target enzyme and its sensitivity, the location of the affected synapse, the level of malaoxon that reaches that synapse, and the route of exposure.²⁰
- Muscarinic symptoms in humans include excessive perspiration, constriction of the pupils, lacrimation (production of tears), salivation, abdominal cramps, diarrhea, nausea, vomiting, chest tightness, and difficulty breathing.^{9,20,22}
- Nicotinic symptoms from exposure to malathion can include muscle weakness, muscle cramping or twitching, ataxia, and paralysis.^{9,20,22}
- Exposure to malathion may cause blood pressure changes with either rapid or decreased heart rate. Effects on the central nervous system's cholinergic neurons can also include headache, confusion, insomnia, decreased rate or depth of respiration, convulsions, and coma.^{9,20,22}
- Children may show somewhat different signs than adults following exposure to malathion and other organophosphate insecticides. Children are less likely to show decreased heart rate, sweating, muscle tremors, and lacrimation than adults, and more likely to show lethargy, seizures, constricted pupils, excessive salivation, muscle weakness, and coma.⁹
- Following exposure to very high doses of some organophosphates including malathion, humans may develop Intermediate Syndrome.^{23,24} The onset is typically 24-96 hours following the exposure. Signs include muscular weakness in the neck, face, and the upper arms and legs, and partial respiratory paralysis. Depressed tendon reflexes and palsies of the cranial nerves also occur frequently in this syndrome.⁹
- A few organophosphates have been linked to organophosphate-induced delayed neuropathy, or OPIDN, following very high levels of exposure. OPIDN involves weakness or paralysis of the extremities, particularly the legs.⁹ In order for OPIDN to develop, the enzyme neuropathy target esterase (NTE) must be inhibited permanently.²⁵ The chemical structure of malathion suggests that it is unlikely to bind to NTE, and malathion has not bound to NTE in animal models or experimental animal studies.²⁵
- 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 additional treatment advice, contact the Poison Control Center at 1-800-222-1222. If you wish to report an incident, please call 1-800-858-7378.

Chronic Toxicity:

Animals

- Researchers fed dogs malathion for 1 year at doses of 0, 62.5, 125.0, or 250.0 mg/kg/day. The dogs exhibited plasma and erythrocyte cholinesterase inhibition at all doses but no clinical signs of toxicity. A LOAEL was set at 62.5 mg/kg/day for plasma and erythrocyte ChE inhibition, but no overall ChE NOAEL was established.²⁶ See the text box on **NOAEL, NOEL, LOAEL, and LOEL**.
- In a 21-day dermal study, scientists exposed rabbits to doses of malathion at 0, 50, 300, or 1000 mg/kg/day for six hours/day, five days/week for three weeks. Scientists detected decreased cholinesterase activity at the two highest doses (300 and 1000 mg/kg/day) but no clinical signs of ChE inhibition. One rabbit died at the highest dose (1000 mg/kg/day). The NOAEL for cholinesterase inhibition was 50 mg/kg/day.²⁶
- In a 13-week inhalation study, investigators exposed male and female rats to malathion at concentrations of 0, 0.10, 0.45, or 2.01 mg/L for six hours/day, five days/week for 13 weeks. Investigators noted nasal and larynx lesions at the greatest dose and measured ChE inhibition at all doses. In addition, signs of cholinesterase inhibition in some animals included excess salivation, reduced grooming, and red stains around the urogenital areas. The systemic toxicity LOAEL was 0.1 mg/kg/day but the NOAEL was not established.¹⁴
- Researchers administered malathion at doses of up to 359 mg/kg/day in the diets of male rats or 415 mg/kg/day in the diets of female rats for two years. Kidney weight gain was noted. The NOAEL for male rats was 29 mg/kg/day and was 35 mg/kg/day for females.²⁷

NOAEL: No Observable Adverse Effect Level

NOEL: No Observed Effect Level

LOAEL: Lowest Observable Adverse Effect Level

LOEL: Lowest Observed Effect Level

Humans

- In an experimental study, five volunteers ingested malathion at up to 16 mg/day (0.23 mg/kg/person/day) for 47 days and displayed no significant reduction in cholinesterase activity. When consuming 24 mg/day (0.34 mg/kg/day) for 56 days, five volunteers displayed reduced cholinesterase activity two weeks after the dosing began. A maximum cholinesterase inhibition of 25% was observed three weeks after the end of the dosing period.^{20,28} See the text box on **Exposure**.

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

- Four male volunteers per treatment inhaled malathion products at 5.3, 21.0, or 85.0 mg/m³ for one hour per exposure, two exposures per day for 42 consecutive days. The test subjects reported nasal and eye irritation at the highest dose during the first 5-10 minutes of each exposure. The authors concluded that no effects on cholinesterase activity occurred, but noted that one subject in each of the two highest dose groups exhibited reduced plasma cholinesterase activity.^{20,29}
- Researchers evaluated the health effects associated with treating areas with malathion and diazinon via ground application, followed by aerial malathion treatments for the control of the Mediterranean fruit fly (*Ceratitis capitata*). The applications occurred from April 1998 to September 1998 in an area containing approximately 132,000 people and covering 128 square miles. A total of 6285 gallons of malathion was applied during the 5 month period. There were 230 reports of pesticide-related illness, and researchers classified 34 as probable and 89 as possible cases. The most commonly reported signs and symptoms were associated with the respiratory, gastrointestinal, and neurological systems.³⁰

Endocrine Disruption:

- Researchers noted suppression of thyroid secretory function in young adult rats that were fed 0.06 mg per rat per day of malathion for 21 days. They also noted an increase in thyroid stimulating hormone (TSH), suggesting that the pituitary gland was compensating to restore normal levels of thyroid hormones.³¹

- In another study, researchers observed thyroid effects including an increased prevalence of parathyroid hyperplasia in male rats. Researchers also reported an increase in thyroid follicular cell adenomas and carcinomas and thyroid c-cell carcinomas in males only.¹⁴
- Rats that were fed high doses (200 and 400 ppm) of malathion for four weeks showed increased blood glucose levels and blood insulin concentration.³²
- Researchers fed rats 40 mg (approximately 225 mg/kg/day) of malathion for five days and noted decreased pituitary prolactin levels, increased serum prolactin levels and increased pituitary gland weight.^{20,33}
- Researchers exposed groups of catfish to 1.2 mg/L malathion for 24 to 96 hours. Investigators noted degenerative changes to ovary follicle cells, increased levels of damage and abnormalities in the oocytes and reduction in the normal level of estrogen in blood plasma.³⁴
- The effects of malathion on the binding of thyroid hormones to the protein transthyretin in blood plasma was studied in vitro using quail blood plasma. After one hour of incubation with varying concentrations of malathion, free and bound thyroid hormone levels were measured. The concentration of malathion necessary to inhibit 3,3',5'-triiodothyronine (T3) by 50% (IC₅₀) was 1400 ± 370 nM.³⁵
- Malathion is listed in the first group of substances to be tested as part of the U.S. EPA Endocrine Disruptor Screening Program (EDSP) because of the potential for people to be exposed to malathion. However, inclusion in the list does not infer that malathion is either known or suspected to be an endocrine disruptor.³⁶

Carcinogenicity:

Animals

- Evidence of the carcinogenicity of malathion in animals is mixed. Several studies have been conducted with rats and mice to determine whether malathion has the potential to cause cancer with variable results.
- In a study involving long-term dietary exposures to malathion, researchers observed an increased incidence of liver and nasal/oral tumors in rats and increased incidence of liver tumors in mice.¹⁴
- In an 80-week dietary study in rats, researchers administered malathion at doses of 0, 359, and 622 mg/kg/day. Investigators did not find statistically significant evidence of carcinogenicity.³⁷
- Researchers administered dietary doses of 0, 166, or 332 mg/kg/day to rats for 103 weeks. Investigators concluded that there was no evidence that malathion was carcinogenic to rats.³⁸
- Researchers conducted an 80-week dietary study in mice with doses of 0, 1490, or 2980 mg/kg/day of malathion. They found no evidence of an association between tumor incidence and exposure to malathion.³⁷
- In a two-year dietary study, researchers administered oral doses of 2, 359, 739, or 868 mg/kg/day to rats. They found a statistically significant increase in liver adenomas and carcinomas in females at the highest dose tested.²⁷
- In a bioassay in mice, researchers administered malathion at doses ranging from 17.4 to 3448.0 mg/kg/day. They concluded that there was evidence of carcinogenicity at doses of 1476 and 2978 mg/kg/day in males and 1707 and 3448 mg/kg/day in females based on incidences of hepatocellular adenomas and liver carcinomas.³⁹ Information on specific dose levels was not available.

Humans

- The International Agency for Research on Cancer (IARC) concluded in 1987 that the carcinogenic potential of malathion was not classifiable, and placed it in Group 3.⁴⁰

- The U.S. EPA classifies malathion as “suggestive evidence of carcinogenicity but not sufficient to assess human carcinogenic potential by all routes of exposure”. This classification was based on the occurrence of liver tumors at excessive doses in mice and female rats and the presence of rare oral and nasal tumors in rats that occurred following exposure to very large doses.¹⁴ 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.

- Researchers conducted a study involving participants from six Canadian provinces and found that exposure to organophosphates as a group and malathion alone was associated with non-Hodgkin’s lymphoma. Malathion used as a fumigant was not associated with increased cancer risk.⁴¹
- Between 1993 and 1997, as part of the Agricultural Health Study, researchers surveyed 19,717 pesticide applicators about their past pesticide exposures and health histories. No clear association between malathion exposure and cancer was reported.⁴²

Reproductive or Teratogenic Effects:

Animals

- In a developmental neurotoxicity study in rats, researchers administered malathion orally at doses of 0, 5, 50, or 150 mg/kg/day to pregnant rats from gestation day 6 to postnatal day 10. Their offspring were then administered the same doses of malathion from postnatal day 11 to postnatal day 21. Increases in startle response were noted in the offspring at all doses tested. Researchers noted abnormal gait at 50 and 150 mg/kg/day and reduced reflex response in female rats at 150 mg/kg/day.¹⁴
- Researchers conducted prenatal developmental toxicity studies with malathion in rats and did not observe any developmental toxicity at maternal doses of up to 800 mg/kg/day.¹⁴
- In a study of pregnant rabbits, researchers noted decreased maternal body weight and an increase in the incidence of fetus resorption at or above 50 mg/kg/day of malathion.¹⁴
- Malathion did not affect reproductive function in female rats in a two-generation reproduction study. However, researchers observed effects on pre-weaning pup growth at doses of 394-451 mg/kg/day. These doses were not toxic to the mothers. Cholinesterase activity was not measured.¹⁴
- Malathion and/or its metabolites were transferred across the placenta and affected the plasma cholinesterase activity of rabbit fetuses when the mothers were orally dosed with 180 mg/kg malathion for three consecutive days.⁴³
- In a postnatal study in mice, dams were given daily injections of malathion at concentrations of 20, 60, and 200 mg/kg during the entire lactation period (21 days). Brain acetylcholinesterase in the dams was significantly decreased only at the highest dose tested, but offspring showed significant reduction in brain acetylcholinesterase activity at all doses tested.⁴⁴
- Researchers exposed pig sperm to malathion for one hour at concentrations of 50, 100, and 500 µM. Sperm viability was reduced at the two highest doses, and motility was reduced following exposure to malathion at all doses.⁴⁵

Humans

- No data were found on developmental or reproductive effects of malathion in humans.

Fate in the Body:

Absorption

- After oral exposure, malathion is rapidly absorbed by the body.²⁰
- Malathion is readily absorbed through the skin, although the percent of a dose that is absorbed varies depending on the size of the dose and site of exposure.²⁰
- Researchers applied malathion to skin of male human volunteers at various points of their body, including forearm, axilla (armpit), ball of the foot, abdomen, forehead, and jaw angle. Absorption was greatest in the armpit followed by the forehead. Absorption through skin in the armpit and forehead areas was 4.2 and 3.4 times greater respectively than absorption by forearm skin.⁴⁶
- Malathion is expected to be readily absorbed when the vapor or spray mist is inhaled.²⁰

Distribution

- Researchers administered malathion orally to one group of male rats at 28 mg/kg, and dermally to another group at 41 mg/kg. In both cases, more than 90% of the dose was excreted in urine within 24 hours. The remaining malathion was found in feces, blood, intestines, liver, and kidneys.⁴⁷
- Researchers applied malathion to rats either intravenously, orally or dermally. Thirty minutes after intravenous exposure most of the malathion was in the liver, kidneys, small intestine, urinary tract and lungs. Four hours after oral administration, 75% of the malathion was still in the stomach, while 8% was in the small intestines, and 7% in the saliva. Eight hours after dermal application, 28% of the dose was still on the applied site, 29% had spread to untreated skin, and 23% had been absorbed and travelled to the small intestine and urinary bladder cavity.⁴⁸
- Based on organ weight changes during a two-week inhalation study in rats, other target organs for malathion are the liver and kidney.²⁶

Metabolism

- Researchers detected 10 metabolites in the urine and feces of rats following dosing with radiolabeled malathion. Urine contained mostly the malathion dicarboxylic acid and lesser concentrations of the - and -malathion mono acids; these three compounds comprised 80% of the radiolabel. Minor metabolites included malaoxon, desmethyl malathion, O,O-dimethyl phosphorothioate, monoethyl fumarate, O,O-dimethyl phosphorodithioate, and thiomalic acid.⁵
- Metabolites detected in humans were essentially the same as in rats, except monomethyl and dimethyl phosphate were found in humans, but thiomalic acid and monoethyl fumarate were not.⁵
- In a metabolism study conducted on rats, malathion did not bioaccumulate in any of the organs or tissues analyzed. The parent compound made up the majority of the excreted residue.¹⁴
- Certain impurities in malathion products can potentiate the toxicity of malathion by inhibiting the carboxylesterases that metabolize malathion and malaoxon in the body.¹³ These impurities may form during the manufacturing process or after long periods of storage.²⁰
- Storage of malathion at high temperatures may also lead to the formation of isomalathion, which is much more toxic than malathion itself. Isomalathion is a potent AChE inhibitor.⁵
- Both malathion and malaoxon are broken down into water-soluble compounds by carboxylesterase enzymes.¹⁴ These enzymes are found in various organs in rats, including the liver, blood serum, and kidney. In humans however, the liver contains the greatest carboxylesterase activity, but the enzyme is absent in the blood.²⁰

- Animals metabolize malathion into the α - and β -malathion monocarboxylic acids via de-esterification, and these compounds are further broken down into dicarboxylic acid.⁵ Secondary metabolic pathways include oxidative desulfuration to malaaxon, hydrolysis to phosphatases, and dealkylation to desmethylmalathion.⁵
- In insects, resistance to malathion appears to be due to the ability to induce greater levels of carboxylesterase activity. Reduced absorption and increased excretion rates may also play a role in resistance.⁵

Excretion

- In a metabolism study conducted on rats, orally administered malathion was excreted primarily in the urine (80- 90%) within the first 24 hours following exposure. Unchanged malathion was the primary residue.¹⁴
- Researchers observed ten different metabolites of malathion in the urine of rats dosed with an unspecified concentration of radio-labeled compound. A total of 85-89% of the dose was excreted in urine, whereas 4-15% of the dose was excreted in feces in the first 72 hours.⁵
- Malathion was applied to the ventral forearm skin of eight human male volunteers at 4 $\mu\text{g}/\text{cm}^2$. Excretion of the parent compound peaked 4-8 hours following the dosing, although only 8% of the applied dose was recovered in urine over the 120 hour post-application period.⁴⁹ Researchers applied the same dose of malathion to skin at various sites of the body in another study. Excretion through urine was greatest following application to skin of the axilla (armpit) and forehead, with $28.6 \pm 13.7\%$ and $23.1 \pm 9.1\%$ of the original dose recovered, respectively.⁴⁶
- Malathion has been detected in human breast milk,⁵⁰ although no studies were found that examined the relationship to exposure or if its presence could cause adverse effects in nursing infants.

Medical Tests and Monitoring:

- There are tests available to determine whether exposure to malathion has occurred. Metabolic products can be measured in urine, if the test is conducted within a few days of exposure.²⁰ However, the presence of malathion metabolites in urine does not necessarily indicate an exposure level great enough to cause adverse health effects.⁵² In addition, presence of metabolites may also result from exposure to the metabolites through diet or from the environment, not from direct exposure to the parent compound.⁵¹
- A blood test may be taken to measure cholinesterase levels in the blood relative to a person's normal level. This type of test is not specific to malathion, and can be used to determine exposure to any cholinesterase inhibitor. However, normal baseline levels of cholinesterase vary widely and can also be suppressed by other factors such as disease.⁹
- In animals, whole blood, stomach contents, hair, or vomitus may be evaluated by submitting samples for laboratory screening for AChE activity. Reduced AChE activity may indicate that exposure to malathion or other organophosphate or carbamate pesticides may have occurred.²¹

Environmental Fate:

Soil

- Reported half-lives in soil range from 1 to 17 days.^{52,53} See the text box on **Half-life** on page 10.
- The extractable residues of malathion in the soil decline rapidly due to volatilization, binding to soil, uptake by plants, and metabolism by soil microbes. Bound residues peaked at 42% of the applied dose 200 hours following application in laboratory studies.⁵³
- Although malathion may be degraded by chemical processes in soil such as chemical hydrolysis, the amount of microbial degradation is far greater than chemical degradation in natural systems.¹⁵

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- Malathion is considered to be very mobile in most soil types, including sand, loam, sandy loam, and silt loam soils.²⁰
- Malaoxon is the primary metabolite of malathion under certain abiotic environmental conditions. Malaoxon may form from environmental degradation of the parent compound, particularly if malathion is deposited on hard, dry surfaces.¹ Malaoxon formation may be greater on dry soils.⁵⁴
- Malaoxon is less stable than malathion and can be quickly degraded to non-toxic metabolites.¹⁵ Malaoxon is a minor metabolite of malathion in soil.⁵⁵

Water

- Chemical degradation in water is a function of both pH and temperature. Malathion's half-life in double-deionized water was greatest at pH 4 and decreased rapidly with either increasing or decreasing pH. The researchers concluded that elimination reactions predominate at high temperatures and carboxyl ester hydrolysis reactions predominate at lower temperatures.⁵⁶
- The half-life of malathion in water was estimated as 1.65 days at pH 8.16 and 17.4 days at pH 6.0.⁵⁷
- Based on the K_{oc} values, malathion is expected to exist primarily in the water column and not bind to sediments.⁵⁸
- Malathion may dissolve in rainwater and be carried in runoff from the application site. U.S. EPA monitoring data from the Boll Weevil Eradication Program and the Mediterranean fruit fly control programs indicate that malathion concentrations in runoff water decrease as distance from the application site increases.¹
- Malathion's half-life in sediments from two creeks in Southern California were 0.8-1.4 days under aerobic conditions and 1.6-2.3 days under anaerobic conditions. The presence of oxygen affected the degradation rate in one sediment type but not the other.⁵⁸
- Of the 990 wells sampled for the U.S. EPA's groundwater database (1971-1991), 12 had positive detections of malathion.¹
- The USDA's Pesticide Data Program (PDP) found no residues of malathion or malaoxon in any of the bottled water or drinking water samples tested in 2006.⁵⁹
- Researchers tested 12 community water systems and detected malathion in 5 of 228 samples prior to standard water treatment. No malathion was detected in the finished water.⁶⁰ All malathion entering treatment facilities with surface water is expected to be converted to malaoxon by the end of the treatment process based on monitoring data.¹⁴
- Malathion was found in surface water in both urban and agricultural settings during a survey of surface and groundwater conducted by the United States Geological Survey (USGS) from 1992 to 2001. In urban streams, malathion was detected in 15% of water samples and was responsible for 30% of the incidents where pesticide concentrations were found at levels exceeding the aquatic-life benchmark dose.⁶¹ Malathion was commonly found in mixtures in urban streams. It also occurred in surface waters in agricultural areas although it was not detected in groundwater.⁶¹
- Malaoxon was found to undergo most rapid hydrolysis at pH 10.⁶²
- Microbial degradation was implicated in the degradation of malathion in seawater.⁶³
- Photodegradation of malathion in deionized water led to the formation of nine degradation products, primarily butane(a)ne diethyl esters. The half-life of malathion during this process was estimated to be 11.6 minutes.⁶⁴

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.

Air

- Malathion in the vapor phase can be degraded by hydroxyl radicals created by sunlight or by photolysis.²
- Malathion was detected in very low concentrations in air (<1 ng/m³) and surface waters between 18 and 2042 m altitude (64-83 ng/L) in the Sierra Nevada Mountains. Researchers speculated that the deposition was a result of atmospheric transport.⁶⁵ Malathion was also detected in 53% of rain and snow samples from Sequoia National Park as a result of wet deposition. Peak concentrations detected were 18-24 ng/L but did not show seasonal or altitudinal trends.⁶⁶
- Malathion was measured in fog samples collected from sites in California and urban Maryland. Concentrations ranged from 70 to 2740 ng/L. Researchers found that the Henry's Law Constant was poor at predicting the distribution of malathion and other pesticides between air and fog droplets, with fog droplets containing up to six-fold greater concentrations of malathion than expected.⁶⁷
- Malathion applied aurally or by a truck sprayer during mosquito control operations at a rate of 492 ng/cm² reached a maximum deposition rate at 36 minutes post-application, but the rate of deposition was only 20% of the applied rate. Researchers speculated that the remaining 80% either volatilized or degraded prior to settling.⁵⁷

Plants

- Half-life on foliage of various fruits, vegetables, alfalfa, and grass ranged from less than 1 to nearly 9 days.⁵²
- Hydrolysis of malathion's P-S bond is the most important degradation process in plants.²⁰
- Researchers applied radio-labeled malathion to peas in pots and found that after 6 hours, a maximum of 2.9% of the applied malathion was present in the tissues of the pea plants, and declined rapidly thereafter.⁵⁴ Residues were lost from the plants due to evapotranspiration, which eventually exceeds the rate of malathion uptake from the soil.⁵³
- Researchers noted that malathion sprayed on strawberry flowers decreased to 2.70% of the initial concentration within two days of application, 0.93% after three days, and 0.50% within seven days.⁶⁸
- Following foliar application, researchers found unmetabolized malathion residues in vegetative portions of alfalfa, lettuce and wheat, and in the seeds of cotton and wheat. The toxic metabolite malaoxon made up ≤1% of residues.¹⁴

Indoor

- No studies were found on the indoor fate of malathion.

Food Residue

- Of the 9602 food samples tested for malathion by the USDA in 2006, there were 15 detections, all below the U.S. EPA's established tolerance level. These detections occurred in kale, spinach, peaches, cranberries and eggplants. An additional 16 fruits and vegetables including applesauce, squash and peas were tested, but had no detectable residues.⁵⁹
- Malathion was found in 1 out of 739 samples of peanut butter, and at a level that was below the U.S. EPA's tolerance.⁵⁹
- Malathion was found in 433 of 687 samples of wheat tested. None of the detections exceeded the U.S. EPA's tolerance.⁵⁹
- Malathion was not found in any of 655 samples of poultry meat tested.⁵⁹

Ecotoxicity Studies:

Birds

- Malathion is slightly to moderately toxic to birds.¹
- The 5-day dietary LC₅₀ for bobwhite quail (*Colinus virginianus*) is 3500 mg/kg and 4230 mg/kg for ring-necked pheasants (*Phasianus colchicus*).³

- Studies of wild bird populations following use of malathion in grasshopper control programs concluded that there were either no effects or inconsistent effects of treatment on reproduction and survival.^{77,78} Bird densities were lower several weeks after treatment in one study, as were grasshopper densities; the researchers concluded that reduced food availability was the most plausible reason for the declines in bird densities.⁷⁸

Fish and Aquatic Life

- Malathion is highly toxic to bluegill sunfish (*Lepomis macrochirus*) and large-mouth bass with 96-hour LC₅₀ of 0.10 mg/L and 0.28 mg/L, respectively.³ It is moderately toxic to the snakehead fish (*Channa punctatus*) and mosquitofish (*Gambusia affinis*) with a 96-hour LC₅₀ of 6.60 ppm and a 48-hour LC₅₀ of 1.23 mg/L, respectively.^{69,70}
- Researchers exposed larvae of the estuarine fish red drum (*Sciaenops ocellatus*) to environmentally realistic and sublethal levels of malathion at levels of 0, 1, and 10 µg/L for up to seven days. No adverse effects were recorded.⁷¹
- The 48-hour EC₅₀ for *Daphnia* is 1.0 µg/L.³ See the text box on EC₅₀.
- Estimated LC₅₀ values for six species of North American tadpoles ranged from 1.2-5.9 mg/L for a 16-day exposure. Water was changed once every 4 days to maintain nominal concentrations.⁷²
- Bullfrog tadpoles were exposed to a constant, high level of malathion for 28 days. At concentrations of 1000 µg/L and higher, tadpole development was delayed, and at 2500 µg/L and higher, survival decreased. These levels are higher than those observed in the environment following normal use of malathion.⁷³
- Researchers applied malathion to aquatic mesocosms containing phytoplankton, zooplankton, periplankton, and tadpoles from two species of frogs. One treatment consisted of weekly applications of 10 µg/L of malathion, others of single applications of 50 or 250 µg/L. Although zooplankton recovered following single-dose exposures, time to metamorphosis in the tadpoles increased and their mass at metamorphosis declined following both the pulsed and single exposures.⁷⁴
- Other aquatic mesocosm experiments have documented altered aquatic community structure and altered predator-prey relationships following exposure to malathion at concentrations of 0.25, 0.50, or 1.00 mg/L.⁷⁵ Short-term community effects following exposure to 0.32 mg/L included a loss of 30% of the total animal species richness.⁷⁶

EC₅₀: The median effective concentration (EC₅₀) may be reported for sublethal or ambiguously lethal effects. This measure is used in tests involving species such as aquatic invertebrates where death may be difficult to determine. This term is also used if sublethal events are being monitored.

Newman, M.C.; Unger, M.A. *Fundamentals of Ecotoxicology*; CRC Press, LLC.: Boca Raton, FL, 2003; p 178.

Terrestrial Invertebrates

- Malathion is highly toxic to bees, whether from direct contact, contact with foliar residues, or contact with residues on pollen. The honey bee topical LD₅₀ is 0.71 µg/bee.³
- Malathion is toxic to other beneficial insect species, and very highly toxic to aquatic invertebrates.^{1,3}
- The LC₅₀ for worms is 613 mg/kg of soil.³

Regulatory Guidelines:

- The U.S. EPA has established an acute Reference Dose (RfD) of 0.14 mg/kg/day for the general population based on a study comparing ChE levels in rats.¹⁴ See the text box on **Reference Dose (RfD)**.
- The U.S. EPA established a benchmark dose level of 7.1 mg/kg based on studies of rats and used this to determine the chronic RfD of 0.07 mg/kg/day.¹⁴

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.

U.S. Environmental Protection Agency. Office of Water. 2002 Edition of the Drinking Water Standards and Health Advisories. EPA 822-R-02-038.
<http://www.epa.gov/ost/drinking/standards/dwstandards.pdf>

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- The U.S. EPA classifies malathion as having “suggestive evidence of carcinogenicity”.¹⁴ See the text box on **Cancer** (page 7).
- Based on a 2-year dietary study in which rats showed inhibition of ChE activity, malathion has a chronic Minimum Risk Level (MRL) of 0.02 mg/kg/day.²⁷
- The U.S. EPA has established an acute population adjusted dose (aPAD) of 0.14 mg/kg/day and a chronic population adjusted dose (cPAD) of 0.07 mg/kg/day.¹
- The workplace permissible exposure limit (PEL) for malathion established by the Occupational Safety and Health Administration (OSHA) is 15 mg/m³, for an 8-hour workday, 40 hours per week.²⁰
- The workplace recommended exposure limit (REL) established by the National Institute for Occupational Safety and Health (NIOSH) is 10 mg/m³, for a 10-hour workday, 40 hours per week.²⁰
- NIOSH has also established the level of malathion in the air that is immediately dangerous to life and health (IDLH) be set at 250 mg/m³.²⁰

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For more information contact: NPIC

Oregon State University, 310 Weniger Hall, Corvallis, OR 97331-6502

Phone: 1-800-858-7378 Fax: 1-541-737-0761

Email: npic@ace.orst.edu Web: npic.orst.edu