d-PHENOTHRIN
TECHNICAL FACT SHEET

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1.800.858.7378

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:

- d-Phenothrin, also referred to as phenothrin or sumithrin, is a type I pyrethroid insecticide.[1] Pyrethroids are synthetic chemicals modeled after the pyrethrin components of pyrethrum, which is derived from plants.[2] The International Union of Pure and Applied Chemistry (IUPAC) name for d-phenothrin is 3-phenoxybenzyl (1*RS*,3*RS*;1*RS*,3*SR*)-2,2-dimethyl-3-(2-methylprop-1-enyl)cyclopropanecarboxylate, and the Chemical Abstracts Service (CAS) registry number is 26002-80-2.[3]

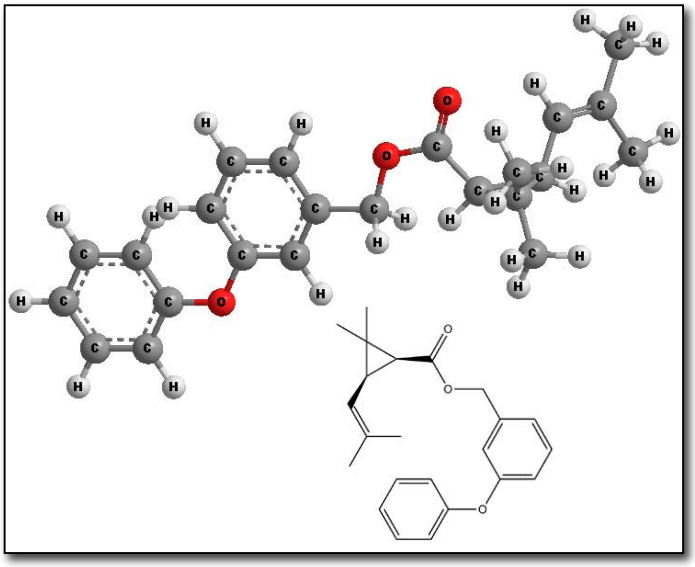
Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term (acute) and long-term (chronic) health effects. Laboratory animals are purposely given high enough doses to cause toxic effects. These tests help scientists judge how these chemicals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

- Phenothrin was first synthesized in 1969.[4] d-Phenothrin was first registered with the United States Environmental Protection Agency (U.S. EPA) in 1976.[1] See the text box on **Laboratory Testing**.

Physical / Chemical Properties:

- d-Phenothrin and phenothrin are similar in chemical structure but differ by isomeric configuration. Phenothrin is made up of equal parts of four isomers (1R-cis, 1R-trans, 1S-cis, 1S-trans), while d-phenothrin contains ≥ 95% 1R isomers, and is a 1:4 mixture of the 1R-cis and 1R-trans isomers. The 1R-trans isomer is more toxic than the 1R-cis isomer.[3,4]
- d-Phenothrin is a colorless, or pale yellow to yellow-brown liquid with a faint characteristic odor.[1,3]
- Vapor pressure[1]: 1.43 x 10^{-7} mmHg at 21 °C
- Octanol-Water Partition Coefficient (log K_{ow})[1,3]: 6.01
- Henry's constant[1]: 6.80 x 10^{-6} atm·m^3/mol
- Molecular weight[1]: 350.46 g/mol
- Solubility (water)[1,3]: <9.7 µg/L at 25 °C
- Soil Sorption Coefficient (K_{oc})[5,6]: 1.25 x 10^5 to 1.41 x 10^5

Molecular Structure - d-Phenothrin



Uses:

- d-Phenothrin is registered for use in commercial and industrial settings, medical institutions, and other settings including aircraft, railroad cars, ships, trailers, homes, animal kennels, gardens, greenhouses, and pet products.[1,7] Uses for individual products containing d-phenothrin vary widely. Always read and follow the label when applying pesticide products.
- d-Phenothrin is also registered for application by aircraft or truck-mounted sprayers as an ultra-low volume (ULV) application for mosquito control in vector abatement programs.[1,7]
- Signal words for products containing d-phenothrin 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 d-phenothrin 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

- d-Phenothrin kills insects by direct contact and ingestion.[1,3] d-Phenothrin is a Type I pyrethroid that affects the insect's central and peripheral nervous system.[1] Following exposure to d-phenothrin, voltage-gated sodium channels are kept open for prolonged periods of time, causing repetitive nerve discharge and increased excitation.[8,9]

Non-target Organisms

- d-Phenothrin and other pyrethroid insecticides are generally lower in toxicity to mammals than insects because they interfere with sodium channels more strongly at lower temperatures. The body temperature of insects and other invertebrates is approximately 10 degrees below that of mammals. Higher mammalian body temperatures also contribute to the increased metabolic degradation of pyrethroids.[8]
- Cats may have an increased sensitivity to the effects of pyrethroid insecticides, including d-phenothrin.[1,10]

Acute Toxicity:

Oral

- d-Phenothrin is very low in toxicity when ingested by rats, with an acute oral LD_{50} >5,000 mg/kg.[1] See the text boxes on **Toxicity Classification** and **LD_{50}/LC_{50}**.

Dermal

- d-Phenothrin is low in toxicity to rats when applied to the skin, with an acute dermal LD_{50} >2,000 mg/kg.[1]
- d-Phenothrin is not a skin irritant or skin sensitizer in rabbits and guinea pigs.[1]
- d-Phenothrin is a mild eye irritant in rabbits.[1]

LD_{50}/LC_{50}: A common measure of acute toxicity is the lethal dose (LD_{50}) or lethal concentration (LC_{50}) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD_{50} is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC_{50} 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 LD_{50}/LC_{50} is small and practically non-toxic when the value is large. However, the LD_{50}/LC_{50} 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

- d-Phenothrin was very low in toxicity when inhaled by rats, with a 4-hour inhalation LC_{50} >2.1 mg/L.[1]

Signs of Toxicity - Animals

- Type I pyrethroids including d-phenothrin have been associated with acute signs of neurotoxicity when administered to rats via oral or intravenous routes of exposure. Signs of acute toxicity in animals may include aggression, increased stimulus response, convulsive twitching, tremors, coma, and death.[11]
- Other signs of toxicity in animals include excessive salivation, ear twitching, and paw flicking from oral and dermal sensory nerve stimulation. Animals may also experience vomiting, diarrhea, and topical allergic reactions.[12]
- Cats may be particularly sensitive to d-phenothrin. Neurotoxic symptoms including tremors, excess salivation and seizures have been reported following treatment of cats with spot-on flea and tick products containing d-phenothrin. As a result, flea and tick spot-on products containing d-phenothrin were canceled for use on cats and kittens in 2005.[1]

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TOXICITY CLASSIFICATION - d-PHENOTHRIN

	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. EPA, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling. http://www.epa.gov/oppfead1/labeling/lrm/hap-07.pdf

Signs of Toxicity - Humans

- Poison Control Center (PCC) data collected from 1993-2005 showed that on average, 180 exposures to d-phenothrin were reported each year. About one quarter of those cases involve reported symptoms. These included nausea, vomiting, throat irritation, headache, dizziness, and skin and eye irritation.[13]
- The National Institute for Occupational Safety and Health (NIOSH) Sentinel Event Notification System for Occupational Risks (SENSOR) reported four cases of occupational exposure to phenothrin between 1998 and 2003. The reports involved mild symptoms, including exacerbation of nasal allergies, itching, and asthmatic responses.[13]
- Paresthesia is associated with exposure to pyrethroids, though more commonly with type II pyrethroids containing a cyano-group.[14] Paresthesia is characterized by sensations of itching, stinging, burning, and tingling of the skin, which may progress to numbness. Symptoms usually occur within 1-2 hours of exposure, and do not typically last more than 24-48 hours.[14,15]
- In an analysis of worker exposure data, the most common symptom reported for individuals working with pyrethroids was paresthesia following dermal contact. Paresthesia was generally limited to the direct point of contact with the skin, and occurred without signs of skin irritation. Paresthesia effects generally resolved within 48 hours of exposure, and no clinical signs of systemic toxicity were observed.[15]
- Systemic toxicity through skin contact and inhalation of pyrethroids is low. Effects to the central nervous system (e.g. seizures) are rare, but have been reported in cases of severe exposure. Seizures are more common with exposure to type II pyrethroids.[14]
- 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 discuss an incident with the National Pesticide Information Center, please call 1-800-858-7378.

Chronic Toxicity:

Animals

- Researchers fed phenothrin to dogs at doses of 0, 100, 300, 1000, or 3000 ppm for 52 weeks. At the highest dose researchers observed anemia, decreased serum proteins, and increased liver weights in both sexes. The LOAEL was 1000 ppm (26.8 mg/kg/day) based on enlargement of liver cells and focal degeneration in the adrenal cortex in male and female dogs. The systemic NOAEL was 300 ppm (7.1 mg/kg/day).[7,16] See the text box on **NOAEL, NOEL, LOAEL**, and **LOEL**.

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

- Researchers exposed male and female rats to d-phenothrin via inhalation at concentrations of 0, 0.030, 0.104, 0.291, or 1.066 mg/L, 6 hours/day, 5 days/week, for 13 weeks and observed no adverse effects at 0.30 or 0.104 mg/L. The LOAEL was 0.291 mg/L based on changes in nasal tissue in male and female rats. Changes were also observed in the thyroid, liver, and adrenals at 1.066 mg/L.[17]

- Scientists applied phenothrin to the skin of rats at doses of 0, 100, 300, or 1000 mg/kg/day for 21 days. No systemic effects were observed.[18]

Humans

- No human data were found on chronic effects of d-phenothrin.

Endocrine Disruption:

- Endocrine-related effects in dogs that were fed 1000 ppm d-phenothrin included liver cell enlargement after 6 months and vacuole formation in the adrenal cortex after 1 year.[7,16]

- Researchers performed a 90-day inhalation study in rats.[7]g Endocrine-related effects included minor vacuole formation in the adrenal cortex and follicular thyroid cell enlargement in males at 0.291 and 1.066 mg/L, and liver cell enlargement at 1.066 mg/L.[7,17]

- Researchers conducted separate studies on female and male rats to determine if d-phenothrin exhibits adverse hormone activity. In the first study, immature female rats were fed d-phenothrin for 3 days at doses of 0, 100, 300, or 1000 mg/kg/day. Researchers found no estrogenic effects at any dose. In the second study, scientists fed d-phenothrin to castrated peripubertal male rats for 10 days at the same doses and found no androgenic/antiandrogenic effects.[19]

Carcinogenicity:

Animals

- d-Phenothrin is classified by the U.S. EPA as "not likely to be carcinogenic to humans" based on liver tumors and growths being observed only at excessively toxic doses (20,000 ppm) in rats, and the lack of statistical significance of these findings.[7] See the text box on Cancer.

- Researchers fed d-phenothrin to mice at doses of 0, 300, 1000, or 3000 ppm for 2 years. They observed no evidence of carcinogenicity.[20]

- Researchers often use studies designed to test for mutagenicity to screen chemicals for carcinogenicity. The U.S. EPA concluded that d-phenothrin "does not pose a mutagenic concern" based on negative findings in bacterial and mammalian gene mutation studies, and a chromosomal aberration study using mammalian cells.[7] 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.

Humans
- No human data were found on carcinogenic effects of d-phenothrin.

Reproductive or Teratogenic Effects:

Animals
- In a two-generation reproductive study, researchers fed phenothrin to rats (initial and first generation offspring following weaning) in the diet at doses of 0, 300, 1000, or 3000 ppm for 13 weeks. The first generation offspring mated to produce a second generation. At doses below 3000 ppm, no systemic toxicity was observed in the parental generation. At 3000 ppm (177.2 mg/kg/day), researchers observed decreased food consumption, decreased body weight, increased liver weights and cellular changes in the liver. At the same dose, the offspring had lower body weights during lactation. The reproductive/offspring LOAEL is 3000 ppm (177.2 mg/kg/day) based on decreased body weight of offspring during lactation.[21]
- Researchers gave phenothrin to pregnant rabbits by gavage during days 7-19 of gestation at doses of 0, 30, 100, 300, or 500 mg/kg/day. They observed signs of maternal toxicity, including decreased food consumption and weight gain at 300 and 500 mg/kg/day, and decreased elimination and increased abortions at 500 mg/kg/day.[22] The developmental NOAEL is 30 mg/kg/day based on spina bifida in one fetus at 100 mg/kg/day.[23]
- Scientists fed phenothrin to pregnant rabbits at doses of 0, 3, 10, or 30 mg/kg/day during days 6-18 of gestation and observed no birth defects in the offspring.[24]

Humans
- No human data were found on the teratogenic or reproductive effects of d-phenothrin.

Fate in the Body:

Absorption
- Pyrethroids are rapidly absorbed by the gastrointestinal tract following ingestion.[15]
- Pyrethroids are likely to be efficiently absorbed from the respiratory tract following inhalation.[15]
- Pyrethroids are poorly absorbed through the skin following dermal exposure. Researchers applied phenothrin isomers to the skin of rats as a dust or as an emulsifiable concentrate (EC) at 0.2 mg/rat and 2 mg/rat for a duration of 24 hours. After 6 days, 78-85% of the EC and 86-94% of the dust was recovered from the skin, unabsorbed.[25]
- Phenothrin isomers absorbed more quickly across the skin when applied as an emulsifiable concentrate compared to a dust formulation.[25]

Distribution
- Pyrethroids, including d-phenothrin, are lipophilic. Following absorption from inhalation or ingestion, the chemical is distributed throughout the body primarily to fatty tissues.[15]
- Researchers administered a single oral dose of 200 mg/kg d-phenothrin to rats and it was rapidly absorbed in the gastrointestinal tract and distributed to body tissues and organs. Peak concentrations occurred at three hours after dosing, and occurred in the liver, kidneys, and blood.[26]
- Following a single oral dose of d-phenothrin in rats (10 mg/kg), researchers observed low overall residue levels of 1.0-2.5 mg/kg in tissues after seven days, with the highest concentrations in the fat.[4]

Metabolism
- In mammals, d-phenothrin is primarily degraded by hydrolysis at the ester bond, oxidation of the subsequent alcohol, followed by conjugation reactions.[4,26]

- Metabolites of d-phenothrin detected in the brain, liver, kidneys, and blood of rats following oral administration include 3-phenoxybenzyl alcohol, 3-phenoxybenzoic acid, and 3-(4'-hydroxy) phenoxybenzoic acid in descending order.[26]
- Following d-phenothrin administration in rats, researchers found the major urinary metabolites were cleaved esters, primarily 4'OH-phenoxybenzoic acid sulfate.[7]

Excretion

- Scientists administered a single oral dose of radiolabeled d-trans-phenothrin to rats at 200 mg/kg and found that >95% of the compound was metabolized and excreted within 24 hours, with the remaining amounts completely recovered within 48 hours.[26] The rats excreted approximately 57% of the administered dose in the urine, and approximately 44% in the feces.[26]
- Researchers administered a single oral dose of phenothrin to rats. Approximately 96% was recovered in the feces and urine within 6 days. Excretion of the *cis*-isomer occurred at 74% in the feces and 22% in the urine, and at 75% in the urine and 21% in the feces for the *trans*-isomer.[25]
- Excessive accumulation and persistence of pyrethroids, including d-phenothrin, in the body is not expected because they are rapidly metabolized under normal conditions.[15]

Medical Tests and Monitoring:

- Analytical methods have been developed to detect d-phenothrin metabolites in the urine using gas chromatography and mass spectrometry.[27] Detection of urinary metabolites serves as an indicator of exposure, but are insufficient to determine the level of exposure or whether an adverse effect will occur.[28]
- Pyrethroids break down rapidly in the body. Therefore, analytical methods are pertinent only when exposure has occurred within the last few days.[28] Most clinical laboratories do not offer testing services for pyrethroids due to the specialized equipment that is necessary to analyze samples.[28]

Environmental Fate:

Soil

- d-Phenothrin is degraded in the environment primarily through UV light reactions.[29]
- In upland conditions, the half-life of d-phenothrin in the soil is 1-2 days. In flood conditions, the half-life of d-phenothrin in the soil ranged from 2 weeks up to 2 months.[5,30] See the text box on **Half-life**.
- d-Phenothrin has low water solubility and binds tightly to soil. Based on these properties, d-phenothrin is relatively immobile in soil and its potential to contaminate groundwater is low.[5]

The "half-life" is the time required for half of the compound to break down in the environemnt.

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

- d-Phenothrin is not likely to contaminate groundwater due to its low water solubility and high affinity for binding to soil.[5]
- Under anaerobic conditions, d-phenothrin is stable to hydrolysis at all pH values with an anaerobic aquatic half-life of 173 days.[5,31]
- With exposure to sunlight, such as in clear shallow water, d-Phenothrin is readily degraded through aqueous photolysis with a half-life of 6.5 days.[32] However, d-phenothrin is lipophilic and has a high affinity to bind to suspended solids and bottom sentiment in water so it is only available for photolysis for a short time.[5]

- d-Phenothrin has a high potential to reach surface water through erosive runoff events due to its low water solubility and a high affinity to bind to soil.[5]

Air

- d-Phenothrin is readily degraded in the air through photolytic reactions with ozone and atmospheric hydroxyl radicals, with an estimated atmospheric half-life of 38.4 minutes and 1.2 hours, respectively.[5]

Plants

- Researchers applied phenothrin isomers to the leaves of kidney beans and rice plants grown in greenhouses, and determined a foliar half-life of less than 1 day.[30]
- Phenothrin isomers were partially taken up by the roots of bean plants grown in treated soil at concentrations of 1 ppm. Researchers observed very little translocation into untreated parts of the plants, including the shoots and edible portions (pods and seeds) 30 days after planting.[30]
- Though no phytotoxicity data were available, the U.S. EPA does not expect d-phenothrin to be toxic to plants.[5]

Indoor

- Scientists sprayed the corners of a room with an aerosol product containing 0.9 g d-phenothrin and 1.1 g tetramethrin in 300 ml four times over an 8-week period. Air concentrations of d-phenothrin peaked immediately after spraying (752 µg/m^3) then decreased rapidly. With minimum air exchange, the half-life of d-phenothrin in the air was 20 minutes. d-Phenothrin did not accumulate in indoor air with repeat applications.[33]
- In the same study, d-phenothrin residues on the floor, ceiling, and walls gradually increased with the number of sprayings during the 8-week period. Residue levels decreased over time when spraying was discontinued. The half-life of d-phenothrin was 13-20 days on the floor and 31-41 days on the walls, and 24-75 days on the ceiling.[33]

Food Residue

- d-Phenothrin has a 0.01 ppm food tolerance for all food/feed crops following area-wide mosquito applications.[34]
- In 2009, the United States Department of Agriculture (USDA) tested almost 9,000 agricultural samples for d-phenothrin residue. d-Phenothrin was not detected in any of these samples.[35]
- The United States Food and Drug Administration (FDA) Pesticide Residue Monitoring Program conducts regulatory and incidence/level monitoring for pesticide residues in domestic and imported foods (except meat, poultry, dairy, and eggs). From 2000-2008, the FDA analyzed more than 700 domestic and 1200 imported food samples for phenothrin residue. Of the samples tested, only one sample had detectable residue at 0.022 ppm.[36]

Ecotoxicity Studies:

Birds

- d-Phenothrin is practically non-toxic to birds. The acute oral LD_{50} in bobwhite quail is >2,510 mg/kg and the dietary LC_{50} is >5,000 mg/kg.[37]
- No studies evaluating the chronic toxicity of d-phenothrin in birds were found. However, the U.S. EPA concluded that the chronic avian risk from d-phenothrin is likely to be low based on the lack of chronic risk in mammalian species and the current use pattern of d-phenothrin.[1]

Fish and Aquatic Life

- d-Phenothrin is very highly toxic to freshwater fish. The 96-hour LC_{50} is 16.7 µg/L and 15.8 µg/L in rainbow trout and bluegill sunfish, respectively.[38]

- Scientists exposed freshwater fish (rainbow trout) to d-phenothrin. Post-hatch 60-day survival in rainbow trout decreased at doses above 1.1 µg/L.[39]
- d-Phenothrin is very highly toxic to estuarine/marine fish. In separate studies, the 96-hour LC_{50} was >38.3 µg/L and 94.3 µg/L in inland silverside.[40,41]
- d-Phenothrin is very highly toxic to freshwater invertebrates. The 48-hour LC_{50} is 4.4 µg/L in *Daphnia magna*.[42]
- The chronic NOAEC for freshwater invertebrates is 0.47 µg/L based on a 21-day reproduction endpoint in *Daphnia magna*.[43]
- Researchers conducted a 96-hour static toxicity study to determine the toxicity of technical and synergized (piperonyl butoxide) formulations of d-phenothrin in crayfish. Researchers found d-phenothrin to be highly toxic to test species *Orconectes immunis* (small and large) and *Procambarus clarkii* (large) with a LOAEC of 0.08 µg/L in tests with synergized d-phenothrin in *Orconectes immunis* (small). For technical and synergized d-phenothrin formulations 96-hour lethal concentrations ranged from 0.24 µg/L in *Orconectes immunis* (small) to 2.62 µg/L in *Orconectes immunis* (large). Researchers did not find a clear pattern of the effects of synergized formulations of d-phenothrin.[44]
- d-Phenothrin is very highly toxic to estuarine/marine invertebrates. The 96-hour LC_{50} is 0.025 µg/L in mysid shrimp.[40]

Terrestrial Invertebrates

- d-Phenothrin is highly toxic to honey bees. The contact LD_{50} is 0.067 µg/bee.[5]

Regulatory Guidelines:

- The acute Reference Dose (aRfD) for 13-49 year old females is 0.03 mg/kg based on a developmental NOAEL of 30 mg/kg/day in rabbits.[7,23] See the text box on **Reference Dose**.
- The chronic RfD for d-phenothrin is 0.007 mg/kg/day based on a systemic toxicity NOAEL of 7.1 mg/kg/day in dogs.[7]

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, Technology Transfer Network, Air Toxics Health Effects Glossary, 2009. http://www.epa.gov/ttnatw01/hlthef/hapglossaryrev.html#RfD

- d-Phenothrin is classified as "not likely to be carcinogenic to humans".[7] See the text box on **Cancer** (page 4).
- The Acceptable Daily Intake (ADI) for d-phenothrin is 0.07 mg/kg.[3]

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