E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

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Aluminum Phosphide

TRADE OR OTHER NAMES: Current trade or other names include Fastphos, Fumitoxin, Gastoxin, Max-Kill, Phosfume, Phostoxin and Weevilcide (1, 213). Al-phos, Celphide, Celphine, Celphos, Detia-Gas-Ex, and Quick Tox may have been used in previous formulations (1, 212).

REGULATORY STATUS: Aluminum Phosphide is a Restricted Use Pesticide so may be purchased and used only by certified applicators (211). It is in EPA Toxicity Class I and products containing it must bear the signal word DANGER (1, 211). Aluminum Phosphide was first registered for use in the United States in the late 1950s (213).

INTRODUCTION: Aluminum phosphide is an inorganic phosphide used to control insects and rodents in a variety of settings. It is mainly used as an indoor fumigant at crop transport, storage or processing facilities (or in shipholds, railcars, etc.) for both food and non-food crops (1, 212). It may also be used as an utdoor fumigant for burrowing rodent and mole control, or in baits for rodent control in crops (212). Aluminum Phosphide is available in pellet and tablet form, and is also available in porous blister packs, sachets or as dusts (212, 213). As in the case of Phostoxin, it may be formulated as 55% active ingredient along with ammonium carbamate and inert ingredients (212, 214).

TOXICOLOGICAL EFFECTS:

- **Acute Toxicity**: Phostoxin and aluminum phosphide are not absorbed dermally; main routes of exposure are through ingestion and inhalation (214). They are highly toxic via both these routes. The reported rodent oral LD50 is 11.5 mg/kg for Phostoxin, with that for the technical compound presumably lower (213, 214). Aluminum phosphide ingested orally reacts with water and stomach acids to produce phosphine gas, which may account in a large part for observed toxicity (188, 215). Phosphine generated in the gastrointestinal tract is readily absorbed in to the bloodstream, and it is readily absorbed through the lung epithelium (188). Phosphine may cause denaturing of oxyhemeglobin (the carrier for systemic distribution of oxygen) as well as enzymes important for respiration and

metabolism, and may also have effects on cellular membranes (217). Inhaled aluminum phosphide dust undergoes the same reaction in the moist air sacs of the lung, although at a lower rate, resulting in similar local and systemic effects (188, 215). The rodent 4-hour inhalation LC50 for phosphine gas (the product of phosphide reaction with water) is widely reported as 15 mg/meters cubed (15 ug/L, or approximately 10.7 ppm) (188,215). Recent study indicates that the rodent 4-hour inhalation LC50 may exceed 15 mg/meters cubed (216). In this study, male and female rats experienced no mortality at one-time 6-hour exposure levels of 15 mg/meters cubed (216). Red mucous discharge from the nostrils ceased during a 14-day recovery period; postmortem examination revealed no gross or microscopic treatment-related effects (216). Symptoms of mild to moderate acute aluminum phosphide toxicity include nausea, abdominal pain, tightness in chest, excitement, restlessness, agitation and chills (214, 188). Symptoms of more severe toxicity include, diarrhea, cyanosis, difficulty breathing, pulmonary edema, respiratory failure, tachycardia (rapid pulse) and hypotension (low blood pressure), dizziness and/or death (188, 215). Convulsions have been reported in lab animals exposed to high concentrations of phosphine (188). Severe exposure may also result in proteinuria or glucosuria (low molecular weight proteins or glucose in the urine) indicating kidney damage (215). Pathological examination of exposed laboratory animal tissue and results of post-mortem examinations of phosphine poisoning victims generally indicate hypoxia, with evidence of local trauma in the gastrointestinal tract or lungs, liver, kidneys and central nervous system (188). Data from a cohort of occupationally-exposed Indian agricultural fumigation workers undergoing single exposures of approximately 1-3 mg/meters cubed (0.71 - 2.22 ppm) revealed reversible (within 2 weeks) symptoms of mild acute exposure (of the types noted above) (215).

- **Chronic Toxicity**: Rats fed aluminum phosphide-fumigated chow averaging 0.51 ppm phosphine residues (approximately 0.43 mg/kg/day) showed no differences from the control animals with respect to blood or urine chemistry and no observable differences in tissue structure (215). It was reported that workers had probably encountered similar exposures on an intermittent basis (in some cases over as long as a 20 year period) and had yet to show signs of toxicity (215), which suggests that chronic effects may be minor or have a very long latency period. Inhalation studies were conducted on the effects of phosphine gas on male and female rats exposed at levels of 0.5, 1.5, and 4.5 mg/meters cubed for six hours per day over a 13 week period (216). Higher exposure groups (7.5 and 15 mg/meters cubed) were added following preliminary acute test results (216). Results indicated that 15 mg/meters cubed was lethal to 4 out of ten female rats following three days of exposure (216). Significant treatment-related effects on body weight and decreased food consumption were seen across all treatment groups and sexes, but were reversible (216). Decreases in red-blood cell counts, hemoglobin, hematocrit and increased platelet counts were seen in male rats of the 4.5 mg/meters cubed group (216). Dose-related changes in blood urea nitrogen and other clinical parameters were also seen across exposure groups (216). Post-mortem examination of test animals revealed microscopic lesions in the outer cortex of the kidneys of rats exposed to 15 mg/meters cubed, but not at lower exposure levels (216). All of these effects were apparently reversible following a four-week recovery period (216).
- **Reproductive Effects:** Post-mortem examination of test animals revealed apparently reversible damage to seminal vesicles in male rats exposed to 1.5 mg/meters cubed phosphine (216). Pregnancy rates for female rats exposed to 4.5 mg/meters cubed on days 6-10 of gestation were comparable to those in the unexposed group (216). No adverse effects on uterine implantation were seen in the 0.3, 3 and 4.5 mg/meters cubed exposure groups, although a statistically significant elevation in resorptions was seen in the 0.015 mg/meters cubed exposure group (216). Thus, this effect may not be dose-related as it there was not increased effect with increased dose. The available evidence for reproductive effects in animals suggest that reproductive effects are not likely in humans under normal conditions.
- **Teratogenic Effects:** No effects on fetal birthweights or sex ratios were seen in offspring of rats exposed to up to 4.5 mg/meters cubed for six hours a day on days 6-10 of gestation (216). No statistically significant differences in development or morphology were seen in the offspring of rats in the

exposed groups versus unexposed groups upon external, visceral or skeletal evaluation (216). The available evidence for teratogenic effects in animals suggests that such effects are not likely in humans under normal conditions.

- **Mutagenic Effects:** No evidence was available regarding the ability of aluminum phosphide or phosphine to cause mutations or increase the mutation rate. Studies of human lymphocyte cultures exposed under laboratory conditions showed significant increases in phosphine-induced total chromosomal aberrations (e.g. gaps, deletions, breaks or exchanges) with increasing phosphine concentrations (217). In the same study, analysis of lymphocyte cultures drawn from fumigators (using phosphine exclusively) exposed to phosphine showed significant increases in the same types of chromosomal aberrations.
- **Carcinogenic Effects:** No data are currently available; it is possible that some testing on the oncogenicity may be initiated in the near future (213).
- **Organ Toxicity**: Acute toxicity resulting from aluminum phosphide exposure is apparent most immediately in the heart and lungs; it may also affect the central nervous system, liver and kidneys (188).
- **Fate in Humans & Animals:** As stated above, aluminum phosphide rapidly reacts with water to form highly toxic phosphine gas. It has been reported that aluminum phosphide may be absorbed directly into the bloodstream, although this is probably a very minor route of entry (218). That phosphine which is not expired through the lungs may be metabolized to phosphates, hypophosphite and phosphite (1, 218).

ECOLOGICAL EFFECTS:

- **Effects on Birds:** The precise oral or inhalation median lethal doses for aluminum phosphide or phosphine in birds are not known. It is reported that exposure of turkeys and hens to 211 and 224 mg/meters cubed for 74 and 59 minutes respectively resulted in labored breathing, swelling of organs, tonic-clonic convulsions and death (219). Due to the mechanism of action it is likely that it could similarly affect other bird species at similar levels of exposure. Fortunately, such exposure is not very likely, as phosphine is rapidly dissipated in open air.
- **Effects on Aquatic Species**: The reported acute LC50 is 4.1 ug/L in rainbow trout, indicating very high toxicity (220). No data were available regarding the specific toxicity of aluminum phosphide or of phosphine to other fish or aquatic species (e.g. LC50 or EC50 values), but due to the mechanism of action it is likely that it will be very highly toxic to them as well. Such exposure is unlikely; aluminum phosphide will rapidly react to form phosphine gas, which is somewhat soluble in water, but will mainly bubble up into the air (211).
- **Effects on Other Animals (Non target species)**: No data were available.

ENVIRONMENTAL FATE:

- **Breakdown of Chemical in Soil and Groundwater:** Aluminum phosphide will breakdown spontaneously in the presence of water to form a gaseous product, and so it is non-persistent and non-mobile in the soil environment, and poses no risk to groundwater.
- **Breakdown of Chemical in Surface Water:** It is highly unlikely that aluminum phosphide or phosphine will be found in surface waters.
- **Breakdown of Chemical in Vegetation:** No data were available.

PHYSICAL PROPERTIES AND GUIDELINES:

Physical Properties:

- **Appearance:** Dark grey or yellowish crystals, has an odor similar to garlic or decaying fish (211, 214).
- **Stability:** Hydrogen phosphide (phosphine) gas, produced by reaction with aluminum phosphide in contact with water (even at ambient humidity), reacts with moist air, vilolently with acids, producing phosphine (211,214).
- **CAS Number:** 20859-73-8
- **Molecular Weight:** 57.95 (212)
- **Water solubility**: Insoluble; reactive with water to form hydrogen phosphide (phosphine), H3P (212)
- **Solubility in other solvents:** Not Available
- **Melting Point:** 1,000 degrees C (214)
- **Vapor Pressure:** negligible @ 25 degrees C (214)
- **Partition Coefficient (octanol/water):** Not Available
- **Adsorption Coefficient**: Not Available

Exposure Guidelines:

- **ADI:** NA
- **HA:** NA
- **RfD:** 0.004 mg/kg/day (221)
- **PEL/TLV:** For phosphine 0.42 mg/meters cubed (0.3 ppm) (222)

BASIC MANUFACTURER

Degesch America, Inc.
275 Triangle Dr.
P. O. Box 116
Weyers Cave, VA 24486 USA

- **Telephone**: 740-234-9281

REFERENCES

References for the information in this PIP can be found in Reference List Number 10

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Bacillus thuringiensis

Trade and Other Names: Trade names include Acrobe, Bactospeine, Berliner (variety kurstaki), Certan (variety aizawai), Dipel, Javelin, Leptox, Novabac, Teknar (variety israelensis), Thuricide, and Victory. Bacillus thuringiensis is also known at B.t.

Regulatory Status: This microbial insecticide was originally registered in 1961 as a General Use Pesticide (GUP). It is classified as toxicity class III - slightly toxic. Products containing B.t. bear the Signal Word CAUTION because of its potential to irritate eyes and skin.

Chemical Class: bacterium

Introduction: Bacillus thuringiensis (B.t.) is a naturally-occurring soil bacterium that produces poisons which cause disease in insects. B.t. is considered ideal for pest management because of its specificity to pests and because of its lack of toxicity to humans or the natural enemies of many crop pests. There are different strains of B.t., each with specific toxicity to particular types of insects: B.t. aizawai (B.t.a.) is used against wax moth larvae in honeycombs; B.t. israelensis (B.t.i.) is effective against mosquitoes, blackflies and some midges; B.t. kurstaki (B.t.k.) controls various types of lepidopterous insects, including the gypsy moth and cabbage looper. A newer strain, B.t. san diego, is effective against certain beetle species and the boll weevil. To be effective, B.t. must be eaten by insects during their feeding stage of development, when they are larvae. B.t. is ineffective against adult insects. More than 150 insects, mostly lepidopterous larvae, are known to be susceptible in some way to B.t.

B.t. forms asexual reproductive cells, called spores, which enable it to survive in adverse conditions. During the process of spore formation, B.t. also produces unique crystalline bodies. When eaten, the spores and crystals of

B.t. act as poisons in the target insects. B.t. is therefore referred to as a stomach poison. B.t. crystals dissolve in the intestine of susceptible insect larvae. They paralyze the cells in the gut, interfering with normal digestion and triggering the insect to stop feeding on host plants. B.t. spores can then invade other insect tissue, multiplying in the insect's blood, until the insect dies. Death can occur within a few hours to a few weeks of B.t. application, depending on the insect species and the amount of B.t. ingested. Typical agricultural formulations include wettable powders, spray concentrates, liquid concentrates, dusts, baits, and time release rings.

Formulation: Typical agricultural formulations include wettable powders, spray concentrates, liquid concentrates, dusts, baits, and time release rings.

Toxicological Effects:

- **Acute toxicity:** B.t. is practically non-toxic to humans and animals. Humans exposed orally to 1000 mg/day of B.t. showed no effects [146]. A wide range of studies have been conducted on test animals, using several routes of exposure. The highest dose tested was 6.7 x 10^11 spores per animal. The results of these tests suggest that the use of B.t. products causes few, if any, negative effects. B.t. was not acutely toxic in tests conducted on birds, dogs, guinea pigs, mice, rats, and humans. No oral toxicity was found in rats, or mice fed protein crystals from B.t. var. israelensis [147]. The LD50 is greater than 5000 mg/kg for the B.t. product Javelin in rats and greater than 13,000 mg/kg in rats exposed to the product Thuricide [147,148]. Single oral dosages of up to 10,000 mg/kg did not produce toxicity in mice, rats, or dogs [148]. The dermal LD50 for a formulated B.t. product in rabbits is 6280 mg/kg. A single dermal application of 7200 mg/kg of B.t. was not toxic to rabbits [148]. B.t. is an eye irritant; 100 grams of formulated product applied in each eye of test rabbits caused continuous congestion of the iris as well as redness and swelling [149]. Very slight irritation from inhalation was observed in test animals. This may have been caused by the physical rather than the biological properties of the B.t. formulation tested [8]. Mice survived 1 or more 1-hour periods of breathing mist that contained as many as 6.0 x 10^10 spores B.t. per liter [143].
- **Chronic toxicity:** No complaints were made by 8 men after they were exposed for 7 months to fermentation broth, moist bacterial cakes, waste materials, and final powder created during the commercial production of B.t. [143]. Dietary administration of B.t. for 13 weeks to rats at dosages of 8400 mg/kg/day did not produce toxic effects [143]. Some reversible abnormal redness of the skin was observed when 1 mg/kg/day of formulated B.t. product was put on scratched skin for 21 days. No general, systemic poisoning was observed [8].
- **Reproductive effects:** There is no indication that B.t. causes reproductive effects [143].
- **Teratogenic effects:** There is no evidence indicating that formulated B.t. can cause birth defects in mammals [143,148].
- **Mutagenic effects:** B. thuringiensis appears to have mutagenic potential in plant tissue. Thus, extensive use of B.t. on food plants might be hazardous to these crops [143]. There is no evidence of mutagenicity in mammalian species.
- **Carcinogenic effects:** Tumor-producing effects were not seen in 2-year chronic studies during which rats were given dietary doses of 8400 mg/kg/day of B.t. formulation [148]. It is unlikely that B.t. is carcinogenic.
- **Organ toxicity:** There is no evidence of chronic B.t. toxicity in dogs, guinea pigs, rats, humans, or other test animals.

- **Fate in humans and animals:** B.t. does not persist in the digestive systems of mammals that ingest it [149].

Ecological Effects:

- **Effects on birds:** B.t. is not toxic to birds [8,150]. The LD50 in bobwhite quail is greater than 10,000 mg/kg. When autopsies were performed on these birds, no pathology was attributed to B.t. Field observations of 74 bird species did not reveal any population changes after aerial spraying of B.t. formulation [148].
- **Effects on aquatic organisms:** B.t. is practically nontoxic to fish [150]. Rainbow trout and bluegills exposed for 96 hours to B.t. at concentrations of 560 and 1000 mg/L did not show adverse effects. A small marine fish (Anguilla anguilla) was not negatively affected by exposure to 1000 to 2000 times the level of B.t. expected during spray programs. Field observations of populations of brook trout, common white suckers, and smallmouth bass did not reveal adverse effects 1 month after aerial application of B.t. formulation [148]. However, shrimp and mussels may be affected adversely [8].
- **Effects on other organisms:** Applications of formulated B.t. are not toxic to most beneficial or predator insects [148]. Treatment of honeycombs with B.t. var. aizawai does not have a detrimental effect upon bees, nor on the honey produced [151]. Very high concentrations (108 spores/ ml sucrose syrup) of B.t. var. tenebrionis, which is used against beetles such as the Colorado potato beetle, reduced longevity of honey bee adults but did not cause disease [151]. B.t. applied at rates used for mosquito control may cause the death of some non-target species [8]. Users of B.t. are encouraged to consult local officials or the nearest EPA regional office responsible for protecting endangered species before using B.t. products in counties where susceptible endangered species of Lepidoptera are known to be present [146]. It did not have negative effects on frogs and salamanders [150].

Environmental Fate:

- **Breakdown in soil and groundwater:** B.t. is a naturally-occurring pathogen that readily breaks down in the environment. Due to its short biological half-life and its specificity, B.t. is less likely than chemical pesticides to cause field resistance in target insects. B.t. is moderately persistent in soil. Its half-life in suitable conditions is about 4 months [152]. B.t. spores are released into the soil from decomposing dead insects after they have been killed by it. B.t. is rapidly inactivated in soils that have a pH below 5.1 [148]. Microbial pesticides such as B.t. are classified as immobile because they do not move, or leach, with groundwater. Because of their rapid biological breakdown and low toxicity, they pose no threat to groundwater.
- **Breakdown in water:** The EPA has not issued restrictions for the use of B.t. around bodies of water. It can be effective for up to 48 hours in water. Afterwards, it gradually settles out or adheres to suspended organic matter [150].
- **Breakdown in vegetation:** B.t. is relatively short-lived on foliage because the ultraviolet (UV) light of the sun destroys it very rapidly. Its half-life under normal sunlight conditions is 3.8 hours [153]. It is not poisonous to plants and has not shown any adverse effect upon seed generation or plant vigor [150].

Physical Properties:

- **Appearance:** The insecticidal action of B.t. is attributed to protein crystals produced by the bacterium. The vegetative cells of B.t. are approximately 1 micron wide and 5 microns long, and are motile [146]. The commercial product contains about 2.5 x 10^11 viable spores per gram. B.t. products lose some of their effectiveness when stored for more than 6 months [8]. B.t. is incompatible with alkaline materials. Formulated products are not compatible with captafol, dinocap, or, under some conditions, leaf (or foliar) nutrients [8].
- **Chemical Name:** Bacillus thuringiensis [1]
- **CAS Number:** (B.t. variety kurstaki) 68038-71-1

- **Molecular Weight:**
- **Water Solubility:** Not Applicable
- **Solubility in Other Solvents:** Not Applicable
- **Melting Point:** Not Applicable
- **Vapor Pressure:** Not Applicable
- **Partition Coefficient:** Not Applicable
- **Adsorption Coefficient:** Not Applicable

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** Not Available
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Sandoz Agro, Inc., and Abbott Laboratories
1300 E. Touhy Ave.
Des Plaines IL 60018

and

Chem. and Agric. Prod. Div.
1401 Sheridan Rd.
North Chicago, IL 60064

- **Phone:** 708-699-1616 ; 708-937-2739
- **Emergency:** 708-699-1616; Not Available

References:

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Diphacinone

Trade and Other Names: Common names include diphacin (Italy and Turkey), ratindan (in the former U.S.S.R.), dipazin, diphenadione, and diphenacin. Trade names include Diphacine, Ditrac, Gold Crest, Kill-Ko, P.C.Q., Promar, Ramik, Rat Killer, Rodent Cake, and Tomcat.

Regulatory Status: Diphacinone is an highly toxic compound in EPA Toxicity Class I. All formulations of diphacinone are Restricted Use Pesticides (RUPs). RUPs may be purchased and used only by certified applicators. The Signal Word required on products containing diphacinone varies, depending on the type of formulation: DANGER applies to the technical material, WARNING to concentrate formulations, and CAUTION to bait formulations.

Chemical Class: Not Available

Introduction: Diphacinone is a rodenticide bait used for control of rats, mice, voles, and other rodents. It is available in meal, pellet, wax block, and liquid bait formulations, as well as in tracking powder and concentrate formulations. It may also be used as a anticonvulsant drug under the name of diphenadione. It has also been used successfully in controlling vampire bats, a vector for rabies.

Formulation: It is available in meal, pellet, wax block, and liquid bait formulations, as well as in tracking powder and concentrate formulations.

Toxicological Effects:

- **Acute toxicity:** Diphacinone is highly toxic by ingestion, with oral LD50 values of 0.3 to 7 mg/kg in rats,

3.0 to 7.5 mg/kg in dogs, 14.7 mg/kg in cats, 150 mg/kg in pigs, 50 to 300 mg/kg in mice, and 35 mg/kg in rabbits [1,172]. It is highly toxic by skin exposure, with reported dermal LD50 values of less than 200 mg/kg in rats, 340 mg/kg in mice, and greater than 3.6 mg/kg in rabbits [1,172]. It is not reported to be a skin or eye irritant in rabbits, nor a skin sensitizer in guinea pigs [1,172]. The 4-hour inhalation LC50 of diphacinone in rats of less than 2 mg/L also indicates high toxicity [1,173]. Diphacinone works by inhibition of liver-synthesized coagulation proteins, leading to internal hemorrhaging [172]. If the dose is sufficient, this may result in death [172]. Effects exhibited in test animals during acute LD50 testing included labored breathing, muscular weakness, excitability, congested blood flow to the lungs, and irregular heartbeats. Other signs of poisoning include spitting of blood, bloody urine or stools, and widespread bruising or bleeding into the joints [172]. Use in humans as an anticoagulant drug has been apparently discontinued, possibly due to its structural similarity to another compound (phenindone). Phenindone was noted to pose risks of hepatitis with jaundice, damage to kidneys, severe skin irritation, and and massive tissue swelling [172]. The use history of diphacinone indicates that it produced no adverse health effects except occasional nausea and some hemorrhagic effects that persisted for 6 to 10 days [172]. In other case reports, the anticoagulant effects are reported to persist for several weeks to months [8].

- **Chronic toxicity:** No permanent or life-threatening effects occurred in humans on recommended dose regimes of an initial 20 mg dose (ca. 0.29 mg/kg in a 70 kg human), followed by successive 2 to 4 mg daily doses (ca. 0.03 to 0.06 mg/kg/day in a 70 kg person) for several days to weeks [172]. All test animals exposed at dietary levels of 0.1 and 0.2 mg/kg/day in a 21-day study showed fatal massive internal hemorrhaging, although at doses of 0.05 mg/kg/day, they were unaffected [172]. In a 90-day study in which rats were given dietary doses of 0.002 to 0.025 mg/kg/day, single rats in each of the 0.003 and 0.013 mg/kg/day dose groups died from internal hemorrhage, but the others remained unaffected by treatment [172]. The effects due to chronic exposure are similar to those which may be expected from acute exposure, but animal studies and human use experience suggest that there is a level of chronic exposure at which no adverse health effects may occur. The available studies also suggest that some individuals in any population may be much more succeptible to effects than others. These individuals may include those with poor nutritional status and/or Vitamin K deficiency, liver or kidney disorders, or infectious diseases [8].
- **Reproductive effects:** No data are currently available [8].
- **Teratogenic effects:** No data are currently available [8].
- **Mutagenic effects:** Diphacinone was not mutagenic in the Ames test [1,172]. No other data regarding mutagenic effects are currently available [1,174].
- **Carcinogenic effects:** No data are currently available
- **Organ toxicity:** The principal target of diphacinone is the blood (specifically the clotting factors), but effects on the liver, kidneys, heart, and musculature have been seen, probably as secondary effects.

- **Fate in humans and animals:** Rats eliminated 70% of the administered oral dose via the feces and 10% in the urine within 8 days [172]. A similar pattern of elimination occurred in mice [172]. Animal studies indicate that little metabolism takes place, and that diphacinone which is not eliminated may concentrate to varying degrees in the liver, kidneys, and lungs [8,172]. The half-life of diphacinone in humans is 15 to 20 days [8]. It was determined that cattle dosed with the compound as an anti-bat measure were safe to use for dairy and/or meat production [8].

Ecological Effects:

- **Effects on birds:** Diphacinone is slightly toxic to birds. The oral LD50 for diphacinone in mallard ducks is 3158 mg/kg [1,8], and in bobwhite quail is 1630 mg/kg [8].

- **Effects on aquatic organisms:** Diphacinone is moderately toxic to fish species. The 96-hour LC50 for technical diphacinone in channel catfish is 2.1 mg/L, in bluegills is 7.6 mg/L, and in rainbow trout is 2.8 mg/L [1,174]. The 48-hour LC50 in Daphnia, a small freshwater crustacean, is 1.8 mg/L [174].
- **Effects on other organisms:** Studies with cattle indicate a high degree of tolerance for the compound, hence its use against vampire bats preying on cattle in Latin America [8].

Environmental Fate:

- **Breakdown in soil and groundwater:** Diphacinone has a low potential to leach in soil [173].
- **Breakdown in water:** Diphacinone is rapidly decomposed in water by sunlight [172].
- **Breakdown in vegetation:** No data are currently available.

Physical Properties:

- **Appearance:** Technical diphacinone is an odorless, pale yellow powder [1].
- **Chemical Name:** 2-(diphenylacetyl)indan-1,3-dione [1]
- **CAS Number:** 82-66-6
- **Molecular Weight:** 340.38
- **Water Solubility:** 0.3 mg/L; almost
- **Solubility in Other Solvents:** v.s. in acetone, acetic acid, toluene, xylene, and chloroform [1]
- **Melting Point:** 145-147 C [1]
- **Vapor Pressure:** 13.7 nPa @ 25 C (technical) [1]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** Not Available

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** Not Available
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Hacco, Inc.
P.O. Box 7190
537 Atlas Ave.
Madison, WI 53707

- **Phone:** 608-221-6200
- **Emergency:** 800-642-4699

References:

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Malathion

Trade and Other Names: Malathion is also known as carbophos, maldison and mercaptothion. Trade names for products containing malathion include Celthion, Cythion, Dielathion, El 4049, Emmaton, Exathios, Fyfanon and Hilthion, Karbofos and Maltox.

Regulatory Status: Malathion is a slightly toxic compound in EPA toxicity class III. Labels for products containing it must carry the Signal Word CAUTION. Malathion is a General Use Pesticide (GUP). It is available in emulsifiable concentrate, wettable powder, dustable powder, and ultra low volume liquid formulations.

Chemical Class: organophosphate

Introduction: Malathion is a nonsystemic, wide-spectrum organophosphate insecticide. It was one of the earliest organophosphate insecticides developed (introduced in 1950). Malathion is suited for the control of sucking and chewing insects on fruits and vegetables, and is also used to control mosquitoes, flies, household insects, animal parasites (ectoparasites), and head and body lice. Malathion may also be found in formulations with many other pesticides.

Formulation: It is available in emulsifiable concentrate, wettable powder, dustable powder, and ULV liquid formulations. Malathion may also be found in formulations with many other pesticides.

Toxicological Effects:

- **Acute toxicity:** Malathion is slightly toxic via the oral route, with reported oral LD50 values of 1000

mg/kg to greater than 10,000 mg/kg in the rat, and 400 mg/kg to greater than 4000 mg/kg in the mouse [2,13]. It is also slightly toxic via the dermal route, with reported dermal LD50 values of greater than 4000 mg/kg in rats [2,13]. Effects of malathion are similar to those observed with other organophosphates, except that larger doses are required to produce them [2,8]. It has been reported that single doses of malathion may affect immune system response [2]. Symptoms of acute exposure to organophosphate or cholinesterase-inhibiting compounds may include the following: numbness, tingling sensations, incoordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, and slow heartbeat. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality. The acute effects of malathion depend on product purity and the route of exposure [33]. Other factors which may influence the observed toxicity of malathion include the amount of protein in the diet and gender. As protein intake decreased, malathion was increasingly toxic to the rats [78]. Malathion has been shown to have different toxicities in male and female rats and humans due to metabolism, storage, and excretion differences between the sexes, with females being much more susceptible than males [79]. Numerous malathion poisoning incidents have occurred among pesticide workers and small children through accidental exposure. In one reported case of malathion poisoning, an infant exhibited severe signs of cholinesterase inhibition after exposure to an aerosol bomb containing 0.5% malathion [44].

- **Chronic toxicity:** Human volunteers fed very low doses of malathion for 1 1/2 months showed no significant effects on blood cholinesterase activity. Rats fed dietary doses of 5 mg/kg/day to 25 mg/kg/day over 2 years showed no symptoms apart from depressed cholinesterase activity. When small amounts of the compound were administered for 8 weeks, rats showed no adverse effects on whole-blood cholinesterase activity [2]. Weanling male rats were twice as susceptible to malathion as adults.
- **Reproductive effects:** Several studies have documented developmental and reproductive effects due to high doses of malathion in test animals [2]. Rats fed high doses of 240 mg/kg/day during pregnancy showed an increased rate of newborn mortality. However, malathion fed to rats at low dosages caused no reproductive effects [8]. It is not likely that malathion will cause reproductive effects in humans under normal circumstances.
- **Teratogenic effects:** Rats fed high doses (240 mg/kg/day) showed no teratogenic effects. Malathion and its metabolites can cross the placenta of the goat and depress cholinesterase activity of the fetus [8]. Chickens fed diets at low doses for 2 years showed no adverse effects on egg hatching [8]. Current evidence indicates that malathion is not teratogenic.
- **Mutagenic effects:** Malathion produced detectable mutations in three different types of cultured human cells, including white blood cells and lymph cells [2,8]. It is not clear what the implications of these results are for humans.
- **Carcinogenic effects:** Female rats on dietary doses of approximately 500 mg/kg/day of malathion for 2 years did not develop tumors [2]. Adrenal tumors developed in the males at low doses, but not at the high doses [80], suggesting that malathion was not the cause. Three of five studies that have investigated the carcinogenicity of malathion have found that the compound does not produce tumors in the test animals. The two other studies have been determined to be unacceptible studies and the results discounted [2,8,80]. Available evidence suggests that malathion is not carcinogenic but the data are not conclusive.
- **Organ toxicity:** The pesticide has been shown in animal testing and from use experience to affect the central nervous system, immune system, adrenal glands, liver, and blood.

- **Fate in humans and animals:** Malathion is rapidly and effectively absorbed by practically all routes including the gastrointestinal tract, skin, mucous membranes, and lungs. Malathion undergoes similar detoxification mechanisms to other organophosphates, but it can also be rendered nontoxic via another simple mechanism, splitting of either of the carboxy ester linkages. Animal studies indicate it is very rapidly eliminated though urine, feces and expired air with a reported half-life of approximately 8 hours in

rats and approximately 2 days in cows [2]. Autopsy samples from one individual who had ingested large amounts of malathion showed a substantial portion in the stomach and intestines, a small amount in fat tissue, and no detectable levels in the liver. Malathion requires conversion to malaoxon to become an active anticholinesterase agent. Most of the occupational evidence indicates a low chronic toxicity for malathion. One important exception to this was traced to impurities in the formulation of the pesticide [2].

Ecological Effects:

- **Effects on birds:** Malathion is moderately toxic to birds. The reported acute oral LD50 values are: in mallards, 1485 mg/kg; in pheasants, 167 mg/kg; in blackbirds and starlings, over 100 mg/kg; and in chickens, 525 mg/kg [2,6]. The reported 5- to 8-day dietary LC50 is over 3000 ppm in Japanese quail, mallard, and northern bobwhite, and is 2639 ppm in ring-neck pheasants [6]. Furthermore, 90% of the dose to birds was metabolized and excreted in 24 hours via urine [79].
- **Effects on aquatic organisms:** Malathion has a wide range of toxicities in fish, extending from very highly toxic in the walleye (96-hour LC50 of 0.06 mg/L) to highly toxic in brown trout (0.1 mg/L) and the cutthroat trout (0.28 mg/L), moderately toxic in fathead minnows (8.6 mg/L) and slightly toxic in goldfish (10.7 mg/L) [13,8,16]. Various aquatic invertebrates are extremely sensitive, with EC50 values from 1 ug/L to 1 mg/L [28]. Malathion is highly toxic to aquatic invertebrates and to the aquatic stages of amphibians. Because of its very short half-life, malathion is not expected to bioconcentrate in aquatic organisms. However, brown shrimp showed an average concentration of 869 and 959 times the ambient water concentration in two separate samples [12].
- **Effects on other organisms:** The compound is highly toxic to honeybees [13].

Environmental Fate:

- **Breakdown in soil and groundwater:** Malathion is of low persistence in soil with reported field half-lives of 1 to 25 days [19]. Degradation in soil is rapid and related to the degree of soil binding [12]. Breakdown occurs by a combination of biological degradation and nonbiological reaction with water [12]. If released to the atmosphere, malathion will break down rapidly in sunlight, with a reported half-life in air of about 1.5 days [12]. It is moderately bound to soils, and is soluble in water, so it may pose a risk of groundwater or surface water contamination in situations which may be less conducive to breakdown. The compound was detected in 12 of 3252 different groundwater sources in two different states, and in small concentrations in several wells in California, with a highest concentration of 6.17 ug/L [33].
- **Breakdown in water:** In raw river water, the half-life is less than 1 week, whereas malathion remained stable in distilled water for 3 weeks [12]. Applied at 1 to 6 lb/acre in log ponds for mosquito control, it was effective for 2.5 to 6 weeks [12]. In sterile seawater, the degradation increases with increased salinity. The breakdown products in water are mono- and dicarboxylic acids [12].
- **Breakdown in vegetation:** Residues were found mainly associated with areas of high lipid content in the plant. Increased moisture content increased degradation [33].

Physical Properties:

- **Appearance:** Technical malathion is a clear, amber liquid at room temperature [13].
- **Chemical Name:** diethyl (dimethoxy thiophosphorylthio) succinate [13]
- **CAS Number:** 121-75-5
- **Molecular Weight:** 330.36

- **Water Solubility:** 130 mg/L [13]
- **Solubility in Other Solvents:** v.s. in most organic solvents [13]
- **Melting Point:** 2.85 C [13]
- **Vapor Pressure:** 5.3 mPa @ 30 C [13]
- **Partition Coefficient:** 2.7482 [13]
- **Adsorption Coefficient:** 1800 [19]

Exposure Guidelines:

- **ADI:** 0.02 mg/kg/day [38]
- **MCL**: Not Available
- **RfD:** 0.02 mg/kg/day [53]
- **PEL:** 15 mg/m3 (8-hour) (dust) [39]
- **HA**: 0.2 mg/L (lifetime) [53]
- **TLV**: Not Available

Basic Manufacturer:

Drexel Chemical Company
1700 Channel Avenue
Memphis, TN 38113

- **Phone:** 901-774-4370
- **Emergency:** Not Available

References:

References for the information in this PIP can be found in Reference List Number 5

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

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Methoprene

Trade and Other Names: Trade names include Altosid, Apex, Diacan, Dianex, Kabat, Minex, Pharorid, Precor, and ZR-515.

Regulatory Status: Methoprene is a slightly to practically nontoxic compound in EPA toxicity class IV. It is a General Use Pesticide (GUP). Labels for containers of products containing methoprene must bear the Signal Word CAUTION.

Chemical Class: Not Available

Introduction: Methoprene is a compound which mimics the action of an insect growth regulation hormone. It is used as an insecticide because it interferes with the normal maturation process. In a normal life cycle, an insect goes from egg to larva, to pupa, and eventually to adult. Methoprene artifically stunts the insects' development, making it impossible for insects to mature to the adult stages, and thus preventing them from reproducing.

To be effective, it is essential that this growth inhibitor be administered at the proper stage of the target pest's life cycle. Methoprene is not toxic to the pupal or adult stages. Treated larvae will pupate but adults do not hatch from the pupal stage. Methoprene is also considered a larvicide since it is effective in controlling the larval stage of insects. Methoprene is used in the production of a number of foods including meat, milk, eggs, mushrooms, peanuts, rice, and cereals. It is also used in aquatic areas to control mosquitoes and several types of ants, flies, lice, moths, beetles, and fleas. It is available in suspension, emulsifiable and soluble concentrate formulations, as well as in briquette, aerosol, and bait form.

Formulation: It is available in suspension, emulsifiable and soluble concentrate formulations, as well as in

briquette, aerosol and bait form.

Toxicological Effects:

- **Acute toxicity:** Methoprene is practically nontoxic when ingested or inhaled and slightly toxic by dermal absorption. The oral LD50 for methoprene in rats is greater than 34,600 mg/kg, and in dogs is greater than 5000 mg/kg [1]. It is slightly toxic by skin exposure, with reported dermal LD50 values of greater than 2000 to 3000 mg/kg in rabbits [1]. Methoprene is not an eye or skin irritant, and it is not a skin sensitizer [1]. The inhalation LC50 for methoprene in rats is greater than 210 mg/L [155]. No overt signs of poisoning have been reported in incidents involving accidental human exposure to methoprene [155].
- **Chronic toxicity:** No methoprene-related effects were observed in 2-year feeding trials with rats given doses of 250 mg/kg/day, nor in mice given 30 mg/kg/day [1]. Liver changes were observed in mice fed 50 to 250 mg/kg/day of methoprene during an 18-month study [155]. Increased liver weights occurred in rats fed 250 mg/kg/day for 90 days, but not during a 24-month feeding study in which rats were fed 125 mg/kg/day [155].
- **Reproductive effects:** Experimental data indicate that no reproductive hazards are associated with methoprene [155]. No methoprene-related effects were observed in three-generation reproduction studies in rats receiving dietary doses of 125 mg/kg/day [1].
- **Teratogenic effects:** There have been no teratogenic effects in animals dosed with methoprene; teratogenic effects were not seen in rats at doses of about 25 mg/kg/day, or in rabbits at doses of about 15 mg/kg/day [156,157]. Methoprene does not appear to be teratogenic.
- **Mutagenic effects:** Methoprene does not appear to be mutagenic. No methoprene-related mutagenic effects were observed in rats following a single dose of 2000 mg/kg [158].
- **Carcinogenic effects:** No tumors were seen in an 18-month feeding study with mice, or in a 24-month oncogenicity study with rats [156]. These data suggest that methoprene is not carcinogenic.
- **Organ toxicity:** The target organ primarily affected by methoprene after long-term exposure is the liver.
- **Fate in humans and animals:** In mammals, methoprene is rapidly and completely broken down and excreted, mostly in the urine and feces [157]. Some evidence suggests that methoprene metabolites are incorporated into natural body components [155]. Methoprene is excreted unchanged in cattle feces in amounts that are sufficient to kill some larvae that breed in dung [131].

Ecological Effects:

- **Effects on birds:** Methoprene is slightly toxic to birds [1,158]. The reported 5- to 8-day LC50 values for Altosid, a methoprene formulation, are greater than 10,000 ppm in mallard ducks and bobwhite quail, and the acute oral LD50 for Altosid is greater than 4640 ppm in chickens [1,158]. In mallards an acute oral LD50 of greater than 2000 mg/kg was determined [158]. Nonlethal effects that may affect survival of the birds did appear at acute oral doses of 500 mg/kg. These effects appeared as soon as 2 hours after treatment and persisted for up to 2 days and included slowness, reluctance to move, sitting, withdrawal, and incoordination [63]. These effects may decrease bird survival by making them temporarily more susceptible to predation. No effects were observed in the reproduction of bobwhite quail and mallard ducks at 30 ppm constant feeding of Altosid [158].
- **Effects on aquatic organisms:** Methoprene is slightly to moderately toxic to fish [157]. The reported 96-hour LC50 values for the methoprene formulation Altosid were 4.6 mg/L in bluegill sunfish, 4.4 mg/L in trout, and greater than 100 mg/L in channel catfish and largemouth bass [1,8]. Methoprene residues may have a slight potential for bioconcentration in bluegill sunfish and crayfish [155]. Methoprene is very highly toxic to some species of freshwater, estuarine, and marine invertebrates, while the acute LC50

values are greater than 100 mg/L in freshwater shrimp, and it is greater than 0.1 mg/L in estuarine mud crabs [159]. Altosid had very little effect, if any, on exposed non-target aquatic organisms including waterfleas, damselflies, snails, tadpoles, and mosquito fish [159].
- **Effects on other organisms:** Tests with earthworms showed little if any toxic effects on contact [159]. It is nontoxic to bees [1].

Environmental Fate:

- **Breakdown in soil and groundwater:** Methoprene is of low persistence in the soil environment; reported field half-lives are up to 10 days [155]. In sandy loam, its half-life was calculated to be about 10 days [155]. When Altosid was applied at an extremely high application rate of 1 pound per acre, its half-life was less than 10 days [155]. In soil, microbial degradation is rapid and appears to be the major route of its disappearance from soil [155,157]. Methoprene also readily undergoes degradation by sunlight [157]. Methoprene is rapidly and tightly sorbed to most soils [155]. It is slightly soluble in water [1]. These properties, along with its low environmental persistence make it unlikely to be significantly mobile. In field leaching studies, it was observed only in the top few inches of the soil, even after repeated washings with water [155,159].
- **Breakdown in water:** Methoprene degrades rapidly in water [8]. Studies have demonstrated half-lives in pond water of about 30 and 40 hours at initial concentrations of 0.001 mg/L and 0.01 mg/L, respectively [49]. At normal temperatures and levels of sunlight, technical Altosid is rapidly degraded, mainly by aquatic microorganisms and sunlight [159,49].
- **Breakdown in vegetation:** Altosid is biodegradable and nonpersistent, even in plants treated at very high rates. It has a half-life of less than 2 days in alfalfa when applied at a rate of 1 pound per acre [159]. In rice, the half-life is less than 1 day [49]. In wheat, its half-life was estimated to be 3 to 7 weeks, depending on the level of moisture in the plant [155]. Plants grown in treated soil are not expected to contain methoprene residues.

Physical Properties:

- **Appearance:** Technical methoprene is a amber or pale yellow liquid with a faint fruity odor [1]
- **Chemical Name:** ispropyl(E,E)-(R,S)-11-methoxy-3,7,11-trimethyldodeca-2,4-dienoate [1]
- **CAS Number:** 40596-69-8
- **Molecular Weight:** 310.48
- **Water Solubility:** 1.4 mg/L @ 25 C [1]
- **Solubility in Other Solvents:** Miscible in organic solvents [1]
- **Melting Point:** Not Available
- **Vapor Pressure:** 3.15 mPa @ 25 C [1]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** Not Available

Exposure Guidelines:

- **ADI:** 0.1 mg/kg/day [12]
- **MCL**: Not Available
- **RfD:** Not Available
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Zoecon Corp.
12005 Ford Rd., Suite 800
Dallas, TX 75234

- **Phone:** Not Available
- **Emergency:** 708-699-1616

References:

References for the information in this PIP can be found in Reference List Number 10

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

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Naled

Trade and Other Names: Trade names for naled include Bromex, Dibrom, Fly Killer-D, Lucanal, and RE 4355.

Regulatory Status: Naled is a moderately toxic compound in EPA toxicity class I. Products containing naled must bear the Signal Word DANGER - POISION because it is corrosive to the eyes. Naled is a General Use Pesticide (GUP).

Chemical Class: organophosphate

Introduction: Naled is a fast acting, nonsystemic contact and stomach organophosphate insecticide used to control aphids, mites, mosquitoes, and flies on crops and in greenhouses, mushroom houses, animal and poultry houses, kennels, food processing plants, and aquaria and in outdoor mosquito control. Liquid formulations can be applied to greenhouse heating pipes to kill insects by vapor action. It has been used by veterinarians to kill parasitic worms (other than tapeworms) in dogs. Naled is available in dust, emulsion concentrate, liquid, and ULV formulations. Unless otherwise specified this profile refers to the technical product of naled.

Formulation: Naled is available in dust, emulsion concentrate, liquid, and ULV formulations. Unless otherwise specified this profile refers to the technical product of naled.

Toxicological Effects:

- **Acute toxicity:** Naled is highly to moderately toxic via the oral route, with reported oral LD50 values of 91 to 430 mg/kg in rats, and 330 to 375 mg/kg in mice [2,13]. It is moderately toxic through skin exposure; reported dermal LD50 values are 1100 mg/kg in rabbits and 800 mg/kg in rats [2,13]. Naled may cause dermatitis (skin rashes) and skin sensitization (allergies) [2,8], and may be corrosive to the skin and eyes.

Mice exposed to 1.5 mg/L in air for 6 hours showed no adverse effects [13]. Naled is used to combat parasitic infestations (such as worms) in dogs at recommended doses of 16.7 mg/kg [2]. Effects due to naled exposure will be similar to those caused by other organophosphate pesticides, including inhibition of cholinesterase and neurological and neuromuscular effects [2]. Symptoms of acute exposure to organophosphate or cholinesterase-inhibiting compounds may include the following: numbness, tingling sensations, incoordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, and slow heartbeat. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality.

- **Chronic toxicity:** Chronic exposure to organophosphates may also cause the neurological and neuromuscular effects associated with cholinesterase inhibition [2]. Rats have tolerated a dosage of 28 mg/kg/day for 9 weeks with no visible signs of poisoning and with only moderate inhibition of cholinesterase [2].
- **Reproductive effects:** No data are currently available.
- **Teratogenic effects:** No data are currently available.
- **Mutagenic effects:** Naled did not affect the ability of one bacterial species (Proteus mirabilis) to repair DNA damage, but did increase the frequency of mutations in another bacterial species (Salmonella typhimurium) [8]. These data are insufficient to determine its potential for mutagenicity.
- **Carcinogenic effects:** No data are currently available.
- **Organ toxicity:** Naled primarily affects the nervous system through cholinesterase inhibition.

- **Fate in humans and animals:** Naled is readily absorbed into the bloodstream through the skin and lung and intestinal tissue. Rat studies suggest accumulation may occur in bone [8].

Ecological Effects:

- **Effects on birds:** Naled is highly to moderately toxic to birds. The reported acute oral LD50 for naled is 52 mg/kg in mallard ducks, 65 mg/kg in sharp-tailed grouse, 36-50 mg/kg in Canadian geese, 120 mg/kg in ring-neck pheasants, and 59 mg/kg in chickens [13,6]. Reported 5- to 8-day dietary LC50 values indicate slight toxicity in species studied. These were 1328 ppm in Japanese quail, 2724 ppm in mallard duck, 2117 ppm in northern bobwhite, and 2538 ppm in ring-neck pheasant [6,13,14].
- **Effects on aquatic organisms:** Naled is highly to moderately toxic to fish [16]. Reported 96-hour LC50 values range from 0.127 mg/L in cutthroat trout, 0.195 mg/L in rainbow trout, and 0.087 mg/L in lake trout to higher values of 3.3 mg/L in fathead minnow, 2.2 mg/L in bluegill sunfish, and 1.9 mg/L in largemouth bass [16]. The reported LC50 for goldfish is 2 to 4 mg/L [13]. Naled may be very highly toxic to aquatic invertebrate species, with reported 96-hour LC50 values of 0.4 ug/L in Dapnia, 8 ug/L in stoneflies, and 18 ug/L in scuds and sideswimmers [16].
- **Effects on other organisms:** Naled is toxic to bees [13]. The reported acute oral LD50 in mule deer is 200 mg/kg [6].

Environmental Fate:

- **Breakdown in soil and groundwater:** Naled is practically nonpersistent in the environment, with reported field half-lives of less than 1 day [19]. It rapidly degrades in the presence of sunlight to dichlorvos [2,13]. For more information on the environmental fate of dichlorvos, see the pesticide profile for dichlorvos. Naled is not strongly bound to soils, but is not highly soluble in water [19]. It is rapidly broken down if wet, and it is moderately volatile [8]. Soil microorganisms break down most of the naled in the soil. It therefore should not present a hazard to groundwater.
- **Breakdown in water:** Naled is rapidly broken down in water, with a reported half-life of about 2 days [8]. Naled is moderately volatile.
- **Breakdown in vegetation:** Plants reductively eliminate bromine from naled to form dichlorvos (DDVP), which may evaporate or be further metabolized [13].

Physical Properties:

- **Appearance:** Technical naled is a colorless liquid with a slightly pungent odor [13].
- **Chemical Name:** 1,2-dibromo-2,2-dichloroethyl dimethyl phosphate [13]
- **CAS Number:** 300-76-5
- **Molecular Weight:** 380.84
- **Water Solubility:** <1 mg/L @ 20 C [13]
- **Solubility in Other Solvents:** v.s. in alcohols, aromatic solvents,; s. in aliphatic hydrocarbons, aromatic hydrocarbons, chlorinated hydrocarbons, and ketones; s.s. in mineral oils and petroleum solvents [13]
- **Melting Point:** 26-27.5 C [13]
- **Vapor Pressure:** 260 mPa @ 20 C [13]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** 180 [19]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.002 mg/kg/day [53]
- **PEL:** 3 mg/m3 (8-hour) [39]
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Amvac Chemical Corp.
4100 E. Washington Blvd.
Los Angeles, CA 90023

- **Phone:** 213-264-3910
- **Emergency:** 800-228-5635, ext. 169

References:

References for the information in this PIP can be found in Reference List Number 5

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

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Permethrin

Trade and Other Names: Trade names include Ambush, BW-21-Z, Cellutec, Dragnet, Ectiban, Eksmin, Exmin, FMC 33297, Indothrin, Kafil, Kestrel, NRDC 143, Pounce, PP 557, Pramex, Qamlin, and Torpedo.

Regulatory Status: Permethrin is a moderately to practically non-toxic pesticide in EPA toxicity class II or III, depending on the formulation. Formulations are placed in class II due to their potential to cause eye and skin irritation. Products containing permethrin must bear the Signal Word WARNING or CAUTION, depending on the toxicity of the particular formulation. All products for agricultural uses (except livestock and premises uses) are Restricted Use Pesticides (RUPs) because of their possible adverse effects on aquatic organisms. Restricted Use Pesticides may be purchased and used only by certified applicators.

Chemical Class: pyrethroid

Introduction: Permethrin is a broad spectrum synthetic pyrethroid insecticide, used against a variety of pests, on nut, fruit, vegetable, cotton, ornamental, mushroom, potato, and cereal crops. It is used in greenhouses, home gardens, and for termite control. It also controls animal ectoparasites, biting flies, and cockroaches. It may cause a mite buildup by reducing mite predator populations. Permethrin is available in dusts, emulsifiable concentrates, smokes, ULV (ultra-low volume), and wettable powder formulations.

Formulation: Permethrin is available in dusts, emulsifiable concentrates, smokes, ULV (ultra-low volume), and wettable powder formulations.

Toxicological Effects:

- **Acute toxicity:** Permethrin is moderately to practically non-toxic via the oral route, with a reported LD50 for technical permethrin in rats of 430 to 4000 mg/kg [12]. Via the dermal route, it is slightly toxic, with a reported dermal LD50 in rats of over 4000 mg/kg, and in rabbits of greater 2000 mg/kg [12,2]. Permethrin caused mild irritation of both the intact and abraded skin of rabbits. It also caused conjunctivitis when it was applied to the eyes [9]. The 4-hour inhalation LC50 for rats was greater than 23.5 mg/L, indicating practically no inhalation toxicity. The toxicity of permethrin is dependent on the ratio of the isomers present; the cis-isomer being more toxic [12].
- **Chronic toxicity:** No adverse effects were observed in dogs fed permethrin at doses of 5 mg/kg/day for 90 days [15]. Rats fed 150 mg/kg/day for 6 months showed a slight increase in liver weights [9]. Very low levels of permethrin in the diet of chickens (0.1 ppm for 3 to 6 weeks after hatching) have been reported to suppress immune system activity [9].
- **Reproductive effects:** The fertility of female rats was affected when they received very high oral doses of 250 mg/kg/day of permethrin during the 6th to 15th day of pregnancy [25]. It is not likely that reproductive effects will be seen in humans under normal circumstances.
- **Teratogenic effects:** Permethrin is reported to show no teratogenic activity [9].
- **Mutagenic effects:** Permethrin is reported to show no mutagenic activity [9].
- **Carcinogenic effects:** The evidence regarding the carcinogenicity of permethrin is inconclusive.
- **Organ toxicity:** Permethrin is suspected of causing liver enlargement of the liver and nerve damage [9]. Effects on the immune system have been noted in animal studies.
- **Fate in humans and animals:** Permethrin is efficiently metabolized by mammalian livers [40]. Breakdown products, or "metabolites," of permethrin are quickly excreted and do not persist significantly in body tissues [41]. When permethrin is administered orally to rats, it is rapidly metabolized and almost completely eliminated from the body in a few days. Only 3 to 6% of the original dose was excreted unchanged in the feces of experimental animals [41]. Permethrin may persist in fatty tissues, with half-lives of 4 to 5 days in brain and body fat [9]. Permethrin does not block, or inhibit, cholinesterase enzymes [40].

Ecological Effects:

- **Effects on birds:** Permethrin is practically non-toxic to birds [12]. The oral LD50 for the permethrin formulation, Pramex, is greater than 9900 mg/kg in mallard ducks, greater than 13,500 mg/kg in pheasants, and greater than 15,500 mg/kg in Japanese quail [41].
- **Effects on aquatic organisms:** Aquatic ecosystems are particularly vulnerable to the impact of permethrin. A fragile balance exists between the quality and quantity of insects and other invertebrates that serve as fish food [41]. The 48-hour LC50 for rainbow trout is 0.0125 mg/L for 24 hours, and 0.0054 mg/L for 48 hours [12]. The 48-hour LC50 in bluegill sunfish and salmon is 0.0018 mg/L [12]. As a group, synthetic pyrethroids were toxic to all estuarine species tested. They had a 96-hour LC50 of less than or equal to 0.0078 mg/L for these species [42]. The bioconcentration factor for permethrin in bluefish is 715 times the concentrations in water and is 703 in catfish. This indicates that the compound has a low to moderate potential to accumulate in these organisms.
- **Effects on other organisms:** Permethrin is extremely toxic to bees. Severe losses may be expected if bees are present at treatment time, or within a day thereafter [2,43]. Permethrin is also toxic to wildlife [9]. It should not be applied, or allowed to drift, to crops or weeds in which active foraging takes place [12].

Environmental Fate:

- **Breakdown in soil and groundwater:** Permethrin is of low to moderate persistence in the soil

environment, with reported half-lives of 30 to 38 days [12,25]. Permethrin is readily broken down, or degraded, in most soils except organic types. Soil microorganisms play a large role in the degradation of permethrin in the soil. The addition of nutrients to soil may increase the degradation of permethrin. It has been observed that the availability of sodium and phosphorous decreases when permethrin is added to the soil [44]. Permethrin is tightly bound by soils, especially by organic matter. Very little leaching of permethrin has been reported [45]. It is not very mobile in a wide range of soil types [41]. Because permethrin binds very strongly to soil particles and is nearly insoluble in water, it is not expected to leach or to contaminate groundwater.

- **Breakdown in water:** The results of one study near estuarine areas showed that permethrin had a half-life of less than 2.5 days. When exposed to sunlight, the half-life was 4.6 days [44]. Permethrin degrades rapidly in water, although it can persist in sediments [15,45]. There was a gradual loss of toxicity after permethrin aged for 48 hours in sunlight at 0.05 mg/L in water [45].
- **Breakdown in vegetation:** Permethrin is not phytotoxic, or poisonous, to most plants when it is used as directed. Some injury has occurred on certain ornamental plants. No incompatibility has been observed with permethrin on cultivated plants. Treated apples, grapes, and cereal grains contain less than one mg/kg of permethrin at harvest time [12].

Physical Properties:

- **Appearance:** Permethrin is an odorless, colorless crystalline solid or a viscous liquid that is pale brown [12].
- **Chemical Name:** 3-phenoxybenzyl(1RS)-cis,trans-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropanecarboxylate [12]
- **CAS Number:** 52645-53-1
- **Molecular Weight:** 391.30
- **Water Solubility:** ca. 0.2 mg/L @ 20 C [12], insoluble in water
- **Solubility in Other Solvents:** s. in most organic solvents except ethylene glycol [12]
- **Melting Point:** 34-35 C [12]
- **Vapor Pressure:** 0.045 mPa @ 25 C [12]
- **Partition Coefficient:** 6.1004[12]
- **Adsorption Coefficient:** 100,000 [26]

Exposure Guidelines:

- **ADI:** 0.05 mg/kg/day [29]
- **MCL**: Not Available
- **RfD:** 0.05 mg/kg/day [30]
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Zeneca Ag Products
1800 Concord Pike
Wilmington, DE 19897

- **Phone:** 800-759-4500
- **Emergency:** 800-759-2500

References:

References for the information in this PIP can be found in Reference List Number 2

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

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PYRETHRINS AND PYRETHROIDS

TRADE OR OTHER NAMES: Several trade names associated with these compounds are Buhach, Chrysanthemum Cinerariaefolium, Ofirmotox, Insect Powder, Dalmation Insect Flowers, Firmotox, Parexan and NA 9184.

INTRODUCTION: Pyrethrins are natural insecticides produced by certain species of the chrysanthemum plant. The flowers of the plant are harvested shortly after blooming and are either dried and powdered or the oils within the flowers are extracted with solvents. The resulting pyrethrin containing dusts and extracts usually have an active ingredient content of about 30%. These active insecticidal components are collectively known as pyrethrins. Two pyrethrins are most prominent, pyrethrin-I and pyrethrin-II. The pyrethrins have another four different active ingredients, Cinerin I and II and Jasmolin I and II. Pyrethrin compounds have been used primarily to control human lice, mosquitoes, cockroaches, beetles and flies. Some "pyrethrin dusts," used to control insects in horticultural crops, are only 0.3% to 0.5% pyrethrins, and are used at rates of up to 50 lb/A. Other pyrethrin compounds may be used in grain storage and in poultry pens and on dogs and cats to control lice and fleas.

The natural pyrethrins are contact poisons which quickly penetrate the nerve system of the insect. A few minutes after application, the insect cannot move or fly away. But, a "knockdown dose" does not mean a killing dose. The natural pyrethrins are swiftly detoxified by enzymes in the insect. Thus, some pests will recover. To delay the enzyme action so a lethal dose is assured, organophosphates, carbamates, or synergists may be added to the pyrethrins.

Semisynthetic derivatives of the chrysanthemumic acids have been developed as insecticides. These are called pyrethroids and tend to be more effective than natural pyrethrins while they are less toxic to mammals. One common synthetic pyrethroid is allethrin.

In this report, the term "pyrethrins" refers to the natural insecticides derived from chrysanthemum flowers; "pyrethroids" are the synthetic chemicals, and "pyrethrum" is a general name covering both compounds. The EPA classifies pyrethrin-I as a Restricted Use Pesticide (RUP). Restricted Use Pesticides may be purchased and used only by certified applicators.

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** Synthetic pyrethroid compounds vary in their toxicity as do the natural pyrethrins. Pyrethrum carries the signal word CAUTION. Inhaling high levels of pyrethrum may bring about asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, incoordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensations (102). The most severe poisonings have been reported in infants, who are not able to efficiently break down pyrethrum. The lowest lethal oral dose of pyrethrum is 750 mg/kg for children and 1,000 mg/kg for adults (102). Oral LD50 values of pyrethrins in rats range from 200 mg/kg to greater than 2,600 mg/kg (96). Some of this variability is due to the variety of constituents in the formulation. Mice have a pyrethrum oral LD50 of 370 mg/kg (102). Animals exposed to toxic amounts may experience tongue and lip numbness, nausea, and diarrhea. Symptoms may also include incoordination, tremors, convulsions, paralysis, respiratory failure, and death. Pyrethroids can cause two quite different responses at near lethal doses in rats; aggressive sparring and a sensitivity to external stimuli progressing to tremors is the one response and pawing and burrowing behavior, and salivation leading to chronic seizures is the other (105). Human response to these two different types of pyrethroids has not yet been evaluated. Recovery from serious poisoning in mammals is fairly rapid. Rats and rabbits are not affected by large dermal applications (96, 102). On broken skin, pyrethrum produces irritation and sensitization, which is further aggravated by sun exposure.
- **Chronic Toxicity:** Absorption of pyrethrum through the stomach and intestines and through the skin is slow. However, humans can absorb pyrethrum more quickly through the lungs during respiration. Response appears to depend on the pyrethrum compound used. Overall, pyrethrins and pyrethroids are of low chronic toxicity to humans and the most common problems in humans have resulted from the allergenic properties of pyrethrum (104). Patch tests for allergic reaction are an important tool in determining an individuals sensitivity to these compounds. Many of the natural and synthetic compounds can produce skin irritation, itching, pricking sensations and local burning sensations. These symptoms may last for about two days (105).
- **Reproductive Effects:** Rabbits that received pyrethrins orally at high doses during the sensitive period of pregnancy had normal litters. A group of rats fed very high levels of pyrethrins daily for three weeks before first mating had litters with weanling weights much lower than normal (96). Overall, pyrethrins appear to have low reproductive toxicity.
- **Teratogenic Effects:** The one rabbit reproduction study performed showed no effect of pyrethrins on development of the offspring (101). More information is needed.
- **Mutagenic Effects:** No information was found.
- **Carcinogenic Effects:** No carcinogenic status has been established for pyrethrins or pyrethroids.
- **Organ Toxicity:** In mammals, tissue storage has not been recorded. At high doses, pyrethrum can be damaging to the central nervous system and the immune system. When the immune system is attacked by pyrethrum, allergies can be worsened. Animals fed large doses of pyrethrins may experience liver damage. Rats fed pyrethrin at high levels for two years showed no significant effect on survival, but slight, definite damage to the livers was observed (96). Inhalation of high doses of pyrethrum for 30 minutes each day for 31 days caused slight lung irritation in rats and dogs (102).
- **Fate in Humans and Animals:** Pyrethrins, pyrethroids, and their metabolites are not known to be stored in the body nor excreted in the milk (100). The urine and feces of people given oral doses of pyrethrum contain chrysanthemumic acid and other metabolites (100, 96). These metabolites are less toxic to mammals than are the parent compounds (101). Pyrethrins I and II are excreted unchanged in

the feces (100). Other pyrethrum components undergo rapid destruction and detoxification in the liver and gastrointestinal tract (96).

ECOLOGICAL EFFECTS

Pyrethrin is extremely toxic to aquatic life, such as bluegill and lake trout while it is slightly toxic to bird species, such as mallards. Toxicity increases with higher water temperatures and acidity. Natural pyrethrins are highly fat soluble, but are easily degraded and thus do not accumulate in the body. These compounds are toxic to bees also.Because pyrethrin-I, pyrethrin-II, and allethrin have multiple sites in their structures that can be readily attacked in biological systems, it is unlikely that they will concentrate in the food chain (100).

ENVIRONMENTAL FATE

Two pyrethroid synthetic insecticides, permethrin and cypermethrin, break down in plants to produce a variety of products (103). Pyrethrins have little residual effect. In stored grain, 50% or more of the applied pyrethrins disappear during the first three or four months of storage. At least 80% of what remains is removed by handling, processing, and cooking (101). Pyrethrins alone provide limited crop protection because they are not stable. As a result, they are often combined with small amounts of antioxidants to prolong their effectiveness. Pyrethrum compounds are broken down in water to nontoxic products. Pyrethrins are inactivated and decomposed by exposure to light and air. Pyrethrins are also rapidly decomposed by mild acids and alkalis. Stored pyrethrin powders lose about 20% of their potency in one year. As the pyrethrins are purified, their stability decreases; thus, pure pyrethrin-I and pyrethrin-II are the least stable of the pyrethrins (96). Purified pyrethrins are very expensive and are only available for laboratory uses.

PHYSICAL PROPERTIES AND GUIDELINES

Physical Properties:

- **Appearance:** The pyrethrins are viscous brown resins, liquids, or solids which inactivate readily in air.
- **Chemical Name:** n/a

- **CAS Number:** 8003347
- **Molecular Weight:** Due to differences in the types and amounts of esters in the pyrethrum mixture, its molecular weight ranges from 316 to 374.
- **Water Solubility:** considered to be insoluble in water.
- **Solubility in Other Solvents:** soluble in organic solvents like: alcohol, kerosene, nitromethane, petroleum ether, carbon tetrachloride, and ethylene dichloride.
- **Melting Point:** n/a
- **Vapor Pressure:** about 0 mm/Hg
- **Partition Coefficient:** n/a
- **Adsorption Coefficient:** n/a

Exposure Guidelines:

- **ADI:** 0.04 mg/kg body weight (humans) (101)
- **MCL**: Not Available
- **RfD:** Not Available
- **PEL:** 5 mg/m3

- **HA**: Not Available
- **TLV**: 5 mg/m3

BASIC MANUFACTURER

There are several manufacturers of products in this category.

REFERENCES

References for the information in this PIP can be found in Reference List Number 2

E X T O X N E T

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Resmethrin

Trade and Other Names: Trade manes include Chryson, Crossfire, Derringer, FMC 17370, Isathrine, NRDC 104, Pynosect, Raid Flying Insect Killer, Respond, Scourge, Sun-Bugger #4, SPB-1382, Synthrin, Syntox, Vectrin, and Whitmire PT-110.

Regulatory Status: Resmethrin is a slightly toxic to practically non-toxic compound in EPA toxicity class III. Products containing resmethrin must bear the Signal Word CAUTION on the label. All products containing resmethrin for pest control at or near aquatic sites are classified as Restricted Use Pesticides (RUP) by the EPA because of potential fish toxicity. RUPs may be purchased and used only by certified applicators.

Chemical Class: pyrethroid

Introduction: Resmethrin is a synthetic pyrethroid used for control of flying and crawling insects in homes, greenhouses, indoor landscapes, mushroom houses, industrial sites, stored product insects and for mosquito control. It is also used for fabric protection, pet sprays and shampoos, and it is applied to horses or in horse stables. Technical resmethrin is a mixture of its two main isomers (molecules with the same chemical formula but slightly different configurations); a typical blend is 20 to 30% of the (1RS)-cis-isomer and 70 to 80% of the (1RS)-trans-isomer.

Formulation: Technical resmethrin is a mixture of its two main isomers (molecules with the same chemical formula but slightly different configurations); a typical blend is 20 to 30% of the (1RS)-cis-isomer and 70 to 80% of the (1RS)-trans-isomer.

Toxicological Effects:

- **Acute toxicity:** Resmethrin is slightly to practically non-toxic by ingestion. The oral LD50 for technical resmethrin in rats is variously reported as greater than 2500 mg/kg or 1244 mg/kg [3,12]. Resmethrin is only slightly toxic through the dermal route as well. The reported dermal LD50s for technical resmethrin are: greater than 3000 mg/kg in rats, greater than 2500 mg/kg in rabbits, and greater than 5000 mg/kg in mice [3,12]. It is slightly toxic via inhalation, with a 4-hour inhalation LC50 for resmethrin of greater than 9.49 mg/L [3]. Symptoms of exposure by any route may include incoordination, twitching, loss of bladder control, and seizures [12]. Dermal exposure may lead to local numbness, itching, burning, and tingling sensations near the site of exposure. Resmethrin is reported to be nonirritating to the skin and eyes of test animals and not to cause skin sensitization in guinea pigs [3].
- **Chronic toxicity:** In a chronic feeding study with rats, 25 mg/kg/day (the lowest dose tested) caused liver enlargement. At 125 mg/kg/day, there were pathological liver changes in addition to increased liver weights. Doses of 250 mg/kg/day caused increased thyroid weight and thyroid cysts [3]. In another study over 90 days, doses of 150 mg/kg/day did not produce any adverse effects in exposed rats [12]. Increased liver weights occurred in dogs fed 30 mg/kg/day for 180 days. No effects were observed in dogs in this study at dose rates of 10 mg/kg/day [3]. In a 90-day inhalation study with rats, 0.1 mg/L, the lowest dose tested, produced behavioral changes, decreased blood glucose levels in males, and decreased body weights and increased serum urea levels in females [3]. Resmethrin was not neurotoxic to rats at doses of 62.5 mg/kg/day for 32 weeks, 250 mg/kg/day for 30 days, or 632 mg/kg/day for 7 days [4]. It is unlikely that chronic effects will be seen in humans under normal circumstances.
- **Reproductive effects:** A three-generation study with rats showed a slight increase in premature stillbirths and a decrease in pup weight at 25 mg/kg, the lowest dose tested [4]. Since these doses are much higher than expected human exposures, it is unlikely such effects will occur in humans.
- **Teratogenic effects:** No birth defects were observed in the offspring of rabbits given doses as high as 100 mg/kg/day [4]. Skeletal aberrations were seen in the offspring of rats given doses higher than 40 mg/kg/day [3]. No teratogenic effects were observed in mice at dose levels of 50 mg/kg/day over an unspecified period [12]. It is unlikely that teratogenic effects will be seen in humans under normal circumstances.
- **Mutagenic effects:** Resmethrin was not mutagenic in a test performed with the bacterium, Salmonella typhimurium [6].
- **Carcinogenic effects:** No evidence of tumor formation was observed in a 2-year rat feeding study with doses as high as 250 mg/kg/day, nor in an 85-week study with mice given doses as high as 50 mg/kg/day [3,4].
- **Organ toxicity:** Pyrethroids may cause adverse effects on the central nervous system. Long-term feeding studies have shown increased liver and kidney weights and adverse changes in liver tissues in test animals [12].

- **Fate in humans and animals:** Resmethrin is quickly eliminated by chickens. When oral doses of 10 mg/kg resmethrin were given to laying hens, 90% of the dose was eliminated in urine and feces within 24 hours [46]. In another study with hens given the same treatment, residues were low in hens sacrificed 12 hours after the treatment, with the highest levels found in the liver and kidneys. Low levels were found in the hens' eggs, with levels peaking 1 day after treatment in the whites and 4 to 5 days after treatment in the yolks [47].

Ecological Effects:

- **Effects on birds:** Resmethrin is practically nontoxic to birds. Its LD50 in California quail is greater than 2000 mg/kg [3]. In Japanese quail, the five-day dietary LC50 is greater than 5000 ppm [48].
- **Effects on aquatic organisms:** Resmethrin is very highly toxic to fish with 96-hour LC50 values

generally at or below 1 ug/L (0.001 mg/L) for most species tested. The LC50 for resmethrin in mosquito fish is 7 ug/L [49]. The LC50 for resmethrin synergized with piperonyl butoxide in red swamp crawfish, Procambarus clarkii, is 0.00082 ug/L [48]. The LC50 in bluegill sunfish is 0.75 to 2.6 ug/L, and 0.28 to 2.4 ug/L in rainbow trout [3]. Other reported 96-hour LC50s are 1.8 ug/L in coho salmon, 1.7 ug/L in lake trout, 3.0 ug/L in fathead minnow, 16.6 ug/L in channel catfish and 1.7 ug/L in bluegill sunfish [50]. Fish sensitivity to the pyrethroids may be explained by their relatively slow metabolism and elimination of these compounds. The half-lives for elimination of several pyrethroids by trout are all greater than 48 hours, while elimination half-lives for birds and mammals range from 6 to 12 hours [20].

- **Effects on other organisms:** Resmethrin is highly toxic to bees, with an LD50 of 0.063 ug per bee [3].

Environmental Fate:

- **Breakdown in soil and groundwater:** Resmethrin is of low to moderate persistence in the soil environment. Its half-life has been estimated at 30 days [51]. Observed half-lives will depend on many site-specific variables. In aerobic Kentucky loamy sand, the compund showed a half-life of nearly 200 days. Degradation end-products reported for resmethrin are chrysanthemic acid, benzaldehyde, benzyl alcohol, benzoic acid, phenylacetic acid, and various esters [52]. Resmethrin is tightly bound to soil and would not be expected to be mobile or to contaminate groundwater, especially in light of its extremely low solubility in water [51].
- **Breakdown in water:** Resmethrin may enter surface waters through particulate run-off or misapplication. In pond waters and in laboratory degradation studies, pyrethroid concentrations decrease rapidly due to sorption to sediment, suspended particles and plants. Microbial and photodegradation also occur [22]. The half-life in water is 36.5 days.
- **Breakdown in vegetation:** No information was found.

Physical Properties:

- **Appearance:** Resmethrin is a waxy, off-white to tan solid with an odor characteristic of chrysanthemums [12].
- **Chemical Name:** 5-benzyl-3-furylmethyl (1RS)-cis,trans-2,2-dimethyl-3-(2-methylprop-1-enyl)cyclopropanecarboxylate [12]
- **CAS Number:** 10453-86-8
- **Molecular Weight:** 338.45
- **Water Solubility:** <1 mg/L at 30 C [12], insoluble in water
- **Solubility in Other Solvents:** s. in hexane, kerosene, xylene, methylene chloride, isopropyl alcohol, and aromatic petroleum hydrocarbons; m.s. in methanol [12]
- **Melting Point:** 43-48 C [12]
- **Vapor Pressure:** 0.0015 mPa @ 30 C [12]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** 100,000 [51]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.03 mg/kg/day [30]
- **PEL:** Not Available

- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Roussel Uclaf Corp.
95 Chestnut Ridge Road
Montvale, NJ 07645

- **Phone:** 201-307-9700
- **Emergency:** Not Available

References:

References for the information in this PIP can be found in Reference List Number 2

E X T O X N E T

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Temephos

Trade and Other Names: Trade names for products containing the compound include Abat, Abate, Abathion, Acibate, Biothion, Bithion, Difennthos, Ecopro, Nimitox, and Swebate. The compound may also be found in mixed formulations with other insecticides including trichlorfon.

Regulatory Status: Temephos is a General Use Pesticide (GUP). Temephos containing products are slightly toxic compounds (EPA toxicity class III) that carry the Signal Word WARNING on their labels despite the relatively low toxicity of the technical compound.

Chemical Class: organophosphate

Introduction: Temephos is an nonsystemic organophosphorus insecticide used to control mosquito, midge, and black fly larvae. It is used in lakes, ponds, and wetlands. It also may be used to control fleas on dogs and cats and to control lice on humans. Temephos is available in up to 50% emulsifiable concentrates, 50% wettable powder, and up to 5% granular forms.

Formulation: Temephos is available in up to 50% emusifiable concentrates, 50% wettable powder, and up to 5% granular forms.

Toxicological Effects:

- **Acute toxicity:** Typical of other organophosphate insecticides, temephos inhibits the action of the group of enzymes called cholinesterases. These enzymes are most inportant in the nervous system, the brain, and the musculoskeletal systems in controlling nerve signal transmission. Symptoms of acute exposure are

similar to other organophosphates and may include nausea, salivation, headache, loss of muscle coordination, and difficulty breathing [8]. Temephos produces signs and symptoms typical of cholinesterase inhibition at moderate levels of exposure, but mortality does not occur unless very large doses of the compound are administered [2,8]. Reported oral LD50 values of temephos range from 1226 to 13,000 mg/kg in rats [2,13], and 460 to 4700 mg/kg in mice. The LD50 for a 2% powder formulation of temephos in dogs and cats is greater than 5000 mg/kg for both species. Temephos may potentiate (greatly increase) the observed toxicity of malathion when used in combination with it at very high doses [2].

- **Chronic toxicity:** Rats, rabbits, guinea pigs, and chickens fed temephos at doses of approximately 20 mg/kg/day for extended periods showed no clinical effects [2]. Dogs tolerated 3 to 4 mg/kg/day for an extended period although there was a slight decrease in cholinesterase activity in the blood and the brain [2]. Severe effects were seen in dogs given 14 mg/kg/day for an extended period, and 15.3 mg/kg/day produced leg weakness in chickens over a 30-day period [2]. As noted under carcinogenicity, a reduction in liver weights was noted in a study on rats fed small doses of temephos over a 2-year period. In another study of rabbits, findings of minor pathological changes in the liver at doses of 10 mg/kg/day were noted, but were not found at a dose of 1 mg/kg/day [2]. No other effects on organs have been reported. Thus, while the LD50 values for acute toxicity indicate that the compound is relatively nontoxic or only slightly toxic, the compound has the potential to cause significant toxic effects (depression of the activity of the enzyme cholinesterase in the blood and the brain) in mammals exposed over long periods of time. Temephos was used in cisterns and other potable water sources in some locations in the U.S. and in the West Indies for the control of mosquito larvae. Subsequent tests on the residents that had used the water sources showed no observable effects in the exposed individuals [2]. Humans ingested 256 mg/person/day for 5 days and 64 mg/person/day (equivalent to 0.91 mg/kg/day) for 4 weeks without any symptoms or detectable effects on blood cholinesterase activity [2].
- **Reproductive effects:** Neither of two studies of rats fed small amounts of temephos showed any reproductive difficulties in the test animals. The maximum dose (25 mg/kg/day) had no effect on the number of litters, litter size, or viability in the young, and produced no congenital defects in the offspring. The concentration of temephos in the diet of the test animals was, however, sufficient to produce cholinesterase inhibition and some toxic symptoms [2]. Low oral doses of temephos of up to 2.5 mg/kg administered in feed over 1 1/2 years caused no reproductive effects in sheep or in their offspring [2]. These data indicate that temephos does not cause reproductive toxicity.
- **Teratogenic effects:** There were no birth defects noted in the offspring of pregnant rabbits fed temephos in two separate studies utilizing different formulations of temephos, a 2% formulation and a 90% formulation. In both studies, maternal toxicity and depression of cholinesterase activity occurred during the study [97]. These data suggest that temephos poses little teratogenic risk.
- **Mutagenic effects:** The potential of a commercial product containing temephos (Abate) to cause mutations was tested on several strains of bacteria. Though the conclusion of the study was that the compound was not mutagenic, weakly mutagenic effects were noted in one of the strains. Additional tests on rabbits and on other strains of bacteria have shown the compound to be nonmutagenic [8,97].
- **Carcinogenic effects:** Only one study of the carcinogenic potential of temephos has been conducted with rats. The rats were fed doses of the compound over a 2-year interval. No tumors or cancer related changes were noted in the test animals at 15 mg/kg/day, the highest dose used [8,97]. During the study the rats experienced a reduction in liver weight at the lowest dose of 0.5 mg/kg/day [8,97]. These data suggest that temephos is not carcinogenic.
- **Organ toxicity:** Animal studies indicate that target organs include the nervous system and liver.

- **Fate in humans and animals:** In general, organophosphate insecticides are readily absorbed through the lungs, skin, and digestive tract [8]. A single oral dose of temephos reached peak concentration in the bloodstream of rats between 5 and 10 hours after it was administered [2], and was eliminated with a half-

life of 10 hours. Some of the compound was also found in the digestive tract and some in fat in mammals. Most of the compound is eliminated unchanged through the feces and urine, though some breakdown products have been detected [2].

Ecological Effects:

- **Effects on birds:** Tests with various wildlife species indicate that the compound is highly toxic to some bird species and moderately toxic to others. The LD50 of temephos ranges from 18.9 mg/kg in the California quail to 240 mg/kg in the chukar partridge [15]. The LD50 values in other bird species studied (Japanese quail, pheasant, and rock dove) were between 35 mg/kg and 85 mg/kg [15]. Mallards fed diets containing moderate amounts of temephos showed no changes in reproduction except in the frequency of egg-laying [98].
- **Effects on aquatic organisms:** Temephos shows a wide range of toxicity to aquatic organisms, depending on the formulation. Generally, the technical grade compound (tech) is moderately toxic and the emulsifiable concentrate (ec) and wettable powder (wp) formulations are highly to very highly toxic. The most sensitive species of fish is the rainbow trout with a temephos LD50 ranging from 0.16 mg/L (ec) to 3.49 mg/L (tech) [16]. Other 96-hour LD50 values are reported as: coho salmon 0.35 mg/L (ec), largemouth bass 1.44 mg/L (ec), channel catfish 3.23 mg/L (ec) to >10 mg/L (tech), bluegill sunfish 1.14 mg/L (ec) to 21.8 mg/L (tech), and Atlantic salmon 6.7 mg/L (ec) to 21 mg/L (tech) [6,8,13,16]. Freshwater aquatic invertebrates such as amphipods are very highly susceptible to temephos, as are some marine invertebrates such as mysids. The 96-hour LD50 of temephos in Gammarus lacustris is 0.08 mg/kg, and in stoneflies is 0.01 to 0.03 mg/kg [6,8,16]. Because the compound is an insecticide and is used effectively to control the aquatic larval stages of mosquitoes, black flies, and midges, its high toxicity to these organisms is not surprising. The product Abate 4E (46% emulsifiable concentrate) is very highly toxic to saltwater species such as the pink shrimp (LC50=0.005 mg/L) and the Eastern oyster (LC50=0.019 mg/L) [8]. The compound is nearly nontoxic to the bull frog with an LD50 of greater than 2000 mg/kg [8]. Temephos has the potential to accumulate in aquatic organisms. The bluegill sunfish accumulated 2300 times the concentration present in the water. Nearly 75% of the compound was eliminated from the fish after exposure ended [8].
- **Effects on other organisms:** The compound is highly toxic to bees, with a direct contact LC50 of 1.55 ug/bee [13].

Environmental Fate:

- **Breakdown in soil and groundwater:** There is little information available about the fate and behavior of temephos in the environment. Based on its very low solubility in water, it would probably have a high affinity for soil. Based on this, a half-life of 30 days has been estimated [19], indicating a low to moderate persistence.
- **Breakdown in water:** Weekly application of temephos at twice the normal application rates on pond water resulted in the rapid disappearance of the compound from the water and from the sediments [6]. At even higher application rates to pond water there were still only traces of the compound detected 1 week after application. Temephos will be photolyzed in water [8]. Temephos was sprayed over an intertidal mangrove community in Florida. Between 15% and 70% of the sprayed amount reaching the leaf surface entered the water below the trees. Additional amounts were washed into the water during rainfall. Pesticide residues were detected in the water 2 hours but not 4 hours after application, indicating a very short persistence in the water. However, in simulated tide pools the compound persisted for up to 4 days. It also persisted in oysters for 2 days after application [99]. Temephos has low persistence in water.
- **Breakdown in vegetation:** Breakdown in plants is very slow.

Physical Properties:

- **Appearance:** Temephos is a solid at room temperature and is composed of colorless crystals. As a liquid, it is brown and viscous [13].
- **Chemical Name:** O,O'-(thiodi-4,1-phenylene)bis(O,O-dimethyl phosphorothioate) [13]
- **CAS Number:** 3383-96-8
- **Molecular Weight:** 466.46
- **Water Solubility:** 0.001 mg/L [13]
- **Solubility in Other Solvents:** s. in common organic solvents; i.s. in hexane and methylcyclohexane [13]
- **Melting Point:** 30-30.5 C [13]
- **Vapor Pressure:** Not Available
- **Partition Coefficient:** 4.9538 [13]
- **Adsorption Coefficient:** 100,000 (estimated) [19]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.02 mg/kg/day [53]
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: 10 mg/m3 total dust; 5 mg/m3 respirable fraction (8-hour) [47]

Basic Manufacturer:

American Cyanamid
One Cyanamid Plaza
Wayne, NJ 07470-8426

- **Phone:** 210-831-2000
- **Emergency:** 210-835-3100

References:

References for the information in this PIP can be found in Reference List Number 5

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

EXTOXNET primary files maintained and archived at Oregon State University

Revised 9/95.

WARFARIN

TRADE OR OTHER NAMES: The active ingredient warfarin is found in a variety of commercial rodenticides. Some trade names for products containing warfarin include Cov-R-Tox, Co-Rax, d-Con, Dethmor, Mar-Fin, Rattunal, Rax, Rodex, Rodex Blox, Rosex, Solfarin, Tox-Hid, Warf, and Warfarat (207, 369, 375). Warfarin is called coumafene in France, zoocoumarin in the Netherlands and Russia, and coumarin in Japan (1, 207, 372).

REGULATORY STATUS: Warfarin is a general use pesticide (GUP). Check with specific state regulations for local restrictions which may apply. The Signal Word for technical and high concentrations of warfarin is "Danger". The Signal Word "Caution" is used for low concentrations and ready-to-use baits (207).

INTRODUCTION: Warfarin was the first anticoagulant rodenticide introduced and was first registered for use in the United States in 1952 (369, 377). Warfarin is used for controlling rats and house mice in and around homes, animal and agricultural premises, and commercial and industrial sites. It is odorless and tasteless and effective in very low dosages. Action is not rapid; usually about a week is required before a marked reduction in the rodent population is noticeable. Rodents do not tend to become bait-shy after once tasting warfarin; they continue to consume it until its anti-clotting properties have produced death through internal hemorrhaging. The prothrombin content of the blood is reduced and internal bleeding is induced. Repeated ingestion is needed to produce toxic symptoms. This rodenticide can be used year-after-year wherever a rodent problem exists. Mice are harder to control than rats, and complete control may take a longer period. Recently, resistant strains of rats and mice are developing (207, 369, 375, 377).Warfarin is only slightly dangerous to humans and domestic animals when used as directed, but care must be taken with young pigs, which are especially susceptible (1).

FORMULATION: Warfarin comes in water soluble, ready-to-use bait, concentrate, powder, liquid concentrate, nylon pouch, coated talc and dust formulations. The compound also comes in mixed formulations with pindone, calciferol, and sulphaquinoxaline. It is considered compatible with other rodenticides (1, 242, 207).

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** The amount of Warfarin that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD50. The acute oral toxicity for warfarin in rats is variously reported to be 3 mg/kg (207, 369, 371, 302, 375, 376); 1,600 ug/kg (370); 186 mg/kg (Hartley and Kidd, 1987) (302, 375); 58 mg/kg in female rats (373, 376). The acute oral LD50 for rats over 4-5 days is 1 mg/kg/day (1, 242). There was no development of ingestion tolerance indicated regardless of rodent sex or age (207). The acute oral LD50 for technical sodium warfarin in rats was 323 mg/kg for males and 58 mg/kg for females (376). A single, large dose of warfarin is about as toxic as a single, small dose. On a multiple-dose basis, the reported LD100 for rats is 0.2 mg/kg/day for 5 days (369, 375). The dermal LD50 for rats was 1,400 mg/kg; 420 mg/kg intraperitoneal LDlo (Lethal Dose, Low. The lowest dose which causes death in test animals.); and 320 mg/m3 inhalation LC50 (370). The same source indicated the acute oral LD50 for mice was 60 mg/kg; 800 mg/kg subcutaneous LDlo; and 165 mg/kg intravenous LD50 (370). Toxicity values for warfarin in other animals are: an oral LD50 for cats of 2.5-20 mg/kg (371, 376); an acute oral LD50 of 35 mg/kg for a single dose or 3 mg/day for 5 days (1, 242); and 12 mg/kg oral LDlo (370). The acute oral LD50 for dogs exposed to warfarin was 3 mg/kg/day for 5 days (1). Technical sodium warfarin in dogs had an LD50 of 200-300 mg/kg (376). The acute oral LD50 for warfarin in cattle was 200 mg/kg/day for 5 days (1). The LD50 for technical sodium warfarin in guinea pigs was 182 mg/kg (376). The oral LDlo for warfarin in pigs was reported to be 1,200 ug/kg (370). Death followed 5 daily doses of 1 mg/kg for pigs (242, 375). Studies done on rabbits indicated the dermal LD50 of warfarin to be greater than 8 g/kg (371, 376). Technical sodium warfarin in rabbits had an LD50 of 800 mg/kg. Rabbits exhibited mild to slight conjunctival irritation in response to technical warfarin (376). Toxicity values for humans exposed to warfarin indicated an oral-woman TDlo of 15 mg/kg/21 weeks intermittent; 10,200 ug/kg oral-man TDlo; and 6,667 mg/kg oral-human LDlo. Average or large doses of warfarin in humans may cause hemorrhage (373). Warfarin is not known to be an eye irritant. It has produced hemorrhages in the retina, however, through its systemic toxicity (375). The compound is considered highly toxic by inhalation and ingestion and moderately toxic by dermal absorption. A dose of warfarin at 200 mg/m3 is considered highly toxic and immediately dangerous to life or health (370).
- **Chronic Toxicity:** A farmer whose hands were intermittently wetted with a 0.5% solution of warfarin over a period of 24 days developed gross hematuria two days after the last contact with the solution; the following day, spontaneous hematomas appeared on the arms and legs. Within four days, there were also epistaxis, punctate hemorrhages of the palate and mouth, and bleeding from the lower lip. The bleeding time was over 30 minutes; the clotting time was 11 minutes and 30 seconds; the prothrombin index was 17; and the prothrombin percentage (thrombotest) was 5. Four days later, after treatment for two days with phytonadione, the values were in the normal range (375). Another source indicated that two human fatalities occurred after ingesting 0.25% warfarin on corn meal over 15 days (376).
- **Reproductive Effects:** No information currently available.
- **Teratogenic Effects:** Warfarin has been established as a human teratogen, because it causes birth defects in the offspring of women receiving clinical doses of the compound during any trimester of pregnancy. Therapeutic use by pregnant women has resulted in fatal hemorrhaging of the fetus and malformations and mental retardation in infants. However, the amount of warfarin contained in the rodenticide bait is very low. A single ingestion of warfarin-treated bait by an adult female would not be likely to cause teratogenic effects (370, 377, 376). Other studies also indicated fetal abnormalities in humans exposed to clinical sodium warfarin (376).
- **Mutagenic Effects**: No information currently available.
- **Carcinogenic Effects:** No information currently available.
- **Organ Toxicity:** Warfarin causes organ damage by inhibiting blood coagulation (1). Absorption by the lungs may result in hemorrhagic effects (370). Animals killed by warfarin exhibit extreme pallor of the

skin, muscles, and all the viscera. In addition, evidence of hemorrhage may be found in any part of the body but usually only in one location in a single autopsy. Such blood as remains in the heart and vessels is grossly thin and forms a poor clot or no clot (372, 374). Rats injected intraperitoneally with 14C-warfarin excreted approximately 90% of the activity in 14 days, about half in the urine and half in the feces (372). Symptoms of human exposure to warfarin include hematuria, back pain, hematoma in arms and legs, bleeding lips, mucous membrane hemorrhage, abdominal pain, vomiting, and fecal blood. One source stated that serious illness was induced by the ingestion of 1.7 mg of warfarin/kg/day for 6 consecutive days with suicidal intent. This would correspond to eating almost 1 pound of bait (0.025% warfarin) each day for 6 days. All signs and symptoms were caused by hemorrhage and, following multiple transfusions and massive doses of vitamin K, recovery was complete (374).

- **Fate in Humans and Animals:** When 9 normal men and 5 normal women were given a single oral dose of 1.5 mg/kg warfarin, maximal concentration in plasma was reached in 2 to 12 hours. Maximal depression of prothrombin activity was between 36 and 72 hours. Their individual increases in prothrombin time were proportional to their half-times for disappearance of the warfarin from plasma. In other words, the pharmacological effect was greatest in those with slower excretion. The half-times for disappearance from the plasma varied from 15 to 58 hours with an average of 42 hours. Absorption of warfarin from the gastrointestinal tract was apparently complete; no warfarin was found in the stool even after massive doses, and plasma levels and prothrombin activity responses were virtually identical following oral and intravenous administration at the same rates (372). Warfarin is readily absorbed by the gastrointestinal tract; absorption in man requires about 3 hours as indicated by a comparison of the rate of action of oral and intravenous doses (374). Another study indicated that 96 hours after intraperitoneal injection of warfarin, the concentrations of activity in the kidney, liver, and pancreas were 3, 12, and 15 times, respectively, greater than that in the blood (372). Metabolites in animals include 4-, 6-, 7- and 8-hydroxycoumarin (1, 372).

ECOLOGICAL EFFECTS

- **Effects on Birds:** The acute avian toxicity of warfarin indicates that it is practically non-toxic to game birds. In subacute studies, warfarin ranged from moderately toxic to practically non-toxic to upland game birds and waterfowl (377). Another source indicated that an acute oral mallard duck study was performed with a 10% formulation of warfarin. This formulation of warfarin was considered moderately toxic to mallard ducks (LC50 greater than 120 mg/kg) when administered as a single dose. However, when exposed to 60 mg/kg for a period of 14 days, 4 out of 5 ducks died (376). Chickens are relatively resistant to warfarin (369).
- **Effects on Aquatic Organisms:** The toxicity of warfarin to aquatic organisms is felt to be of low potential due to the fact that warfarin is insoluble in water. A long field experience shows no potential hazards to aquatic organisms (377). A 96-hour rainbow trout study was performed using a 0.54% formulation of warfarin sodium salt. With a 96-hour LC50 of greater than 10,000 ppm, this formulation is considered non-toxic to rainbow trout (376).Effects on Other Animals (Nontarget species) Warfarin used as a prepared bait (0.13%) is considered non-toxic to bees when used as prescribed (1, 207). The use of warfarin as a hand-placed bait limits the potential for any secondary exposure of nontarget animals. However, because of its high degree of mammalian toxicity and its use patterns, warfarin could adversely affect endangered or threatened species (377). One study exists on a 50/50 percent formulation of warfarin-sulfaquinoxaline technical. The warfarin-sulfaquinoxaline caused secondary poisoning in mammalian carnivores such as mink and dogs when ingesting prey killed after they were provided with treated bait (carrots containing 0.025% by weight of the test material). The first death occurred after 8 days of continuous exposure to treated nutria (376). A study by Bucklew et al. investigated the short-term influence of warfarin on the growth of the gram-positive spore-forming soil microorganism, Bacillus megaterium. Impregnation of paper disks and subsequent measurement of the

zones of growth inhibition showed that spore germination for this bacterium was not affected by the presence of warfarin for 15-21 hours at 21 degrees C and at concentrations as high as 1 mg/ml (about 1,000 ppm) (376).

ENVIRONMENTAL FATE

- **Breakdown of Chemical in Soil and Groundwater:** No information currently available.
- **Breakdown of Chemical in Surface Water:** No information currently available.

PHYSICAL PROPERTIES AND GUIDELINES

Physical Properties:

- **Appearance:**
- **Chemical Name:** 4-hydroxy-3-(3-oxo-1-phenylbutyl)coumarin; 4-hydroxy-3-(3-oxo-1-phenylbutyl)-2H-1-benzopyran-2-one (1, 242)
- **CAS Number:** 81-81-2
- **Molecular Weight:** 308.3
- **Water Solubility:** Practically insoluble (1.7 mg/100 ml at 20 degrees C) (1, 370, 373)
- **Solubility in Other Solvents:** Soluble to very slightly soluble in acetone, benzene, ethanol, ether, toluene, xylene, methyl ethyl ketone and cyclohexane. Moderately soluble in methanol, ethanol, and isopropanol. In acetone 6.5, chloroform 5.6, dioxane 10.0 (all in g/100 ml at 20 degrees C). Dissolves in aqueous alkalis with the formation of water-soluble salts (1, 242, 302, 372, 373)
- **Melting Point:** 161-162 degrees C (1, 302); 159-165 degrees C (207); 318-322 degrees F (370)
- **Vapor Pressure:** 9 x 10 to the minus 2 mbar at 21.5 degrees C
- **Partition Coefficient:** 3.20 (calculated) (302)
- **Adsorption Coefficient:** 2.96 (calculated) (302)

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** Not Available
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: 0.1 mg/m3 (OSHA) (207, 302, 375)

BASIC MANUFACTURER:

SHACCO, Inc.
P. O. Box 7190
537 Atlas Avenue (53714)
Madison, WI 53707

- **Phone:** 608-221-6200
- **Fax**: 608-221-6208

Prentiss, Inc.

CB 2000
Floral Park, NY 11001

- **Phone:** 516-326-1919
- **Fax:** 516-326-2312

REFERENCES

References for the information in this PIP can be found in Reference List Number 10

E X T O X N E T

Extension Toxicology Network

Pesticide Information Profiles

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EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Zinc phosphide

Trade and Other Names: Trade names for commercial products containing zinc phosphide include Arrex, Commando, Denkarin Grains, Gopha-Rid, Phosvin, Pollux, Ridall, Ratol, Rodenticide AG, Zinc-Tox and ZP.

Regulatory Status: Zinc phosphide is a Restricted Use Pesticide (RUP) because of its hazard to non-target organisms and its acute oral toxicity. RUPs may be purchased and used only by certified applicators. Some formulations of this rodenticide are classified as highly toxic and require the Signal Word DANGER - POISON on the label. Others are either moderately toxic or only slightly toxic, and thus require the Signal Words WARNING or CAUTION, respectively.

Chemical Class: inorganic compound

Introduction: Zinc phosphide is an inorganic chemical that is used to control rats, mice, voles, ground squirrels, prairie dogs, nutria, muskrats, feral rabbits, and gophers. It is also used as a tracking powder for the control of house mice. It is applied to crop areas and non-crop areas including lawns, golf courses, highway medians, and areas adjacent to wetlands. It may be formulated as a grain based bait, as scrap bait, or as a paste. Rodenticide baits usually contain 0.5 to 2.07% zinc phosphide, pastes approximately 5 to 10%.

Formulation: It may be formulated as a grain based bait, as scrap bait, or as a paste. Rodenticide baits usually contain 0.5 to 2.07% zinc phosphide, pastes approximately 5 to 10%.

Toxicological Effects:

- **Acute toxicity:** Zinc phosphide ingested orally reacts with water and acid in the stomach and produces phosphine gas, which may account in a large part for observed toxicity [160]. Symptoms of acute zinc phosphide poisoning by ingestion include nausea, abdominal pain, tightness in chest, excitement, agitation, and chills [160,8]. Other symptoms include vomiting, diarrhea, cyanosis, rales, restlessness, and fever [8,160]. The inhalation of zinc phosphide or its breadkown product phosphine gas may result in vomiting, diarrhea, cyanosis, rapid pulse, fever, and shock [160]. There are documented cases of adults dying from massive oral doses of 4000 to 5000 mg (approximately 55 to 70 mg/kg), although others have survived acute exposure of as high as 25,000 to 100,000 mg (approximately 350 to 1400 mg/kg) if vomiting occurred early and exposure to phosphine was limited [160]. In rats, the LD50 for the technical product (80 to 90% pure) is 40 mg/kg, while the LD50 values for lower concentration formulations are slightly higher, indicating lower acute toxicity [160]. In sheep the LD50 ranges from 60 to 70 mg/kg [160]. The compound is nonirritating to the skin and eyes [160].
- **Chronic toxicity:** Rats fed zinc phosphide over a wide range of doses experienced toxic effects. Increased liver, brain, and kidney weights, and lesions on these organs, were noted in rats exposed to around 14 mg/kg/day. Body hair loss, reduction in body weight, and reduction of food intake were all noted at 3.5 mg/kg/day. The study was conducted over 13 weeks [8]. There have been no observed symptoms of chronic poisoning due to zinc phosphide exposure in humans [1]. However, it has been suggested that chronic exposure to sublethal concentrations for extended periods of time may produce toxic symptoms [8].
- **Reproductive effects:** No data are currently available.
- **Teratogenic effects:** No data are currently available.
- **Mutagenic effects:** No data are currently available regarding the mutagenicity of zinc phosphide. However, its metabolite, phosphine, has shown a concentration-dependent increase in chromosomal aberrations in studies using human lymphocyte cultures [8]. Thus, its mutagenicity is unclear.
- **Carcinogenic effects:** No data are currently available.
- **Organ toxicity:** Damage to the kidneys, the liver, and the stomach have been noted in humans, but only at high acute doses of the rodenticide. Zinc phosphide reacts with water and stomach juices to release phosphine gas, which can enter the blood stream and adversely affect the lungs, liver, kidneys, heart, and central nervous system [8].

- **Fate in humans and animals:** Small amounts of the rodenticide fed to experimental animals may have produced an 80% absorption of zinc as well. Zinc in sufficient concentrations may have an emetic effect [8]. Hypophosphite may be excreted in the urine as a metabolite of zinc phosphide [160]. There is little tendency for the compound to concentrate in living tissue, as it is readily converted to phosphine.

Ecological Effects:

- **Effects on birds:** Zinc phosphide is highly toxic to wild birds The most sensitive birds are geese (LD50 of 7.5 mg/kg for the white-fronted goose). pheasants, mourning doves, quail, mallard ducks, and the horned lark are also very susceptible to this compound. Blackbirds are less sensitive [8].
- **Effects on aquatic organisms:** Zinc phosphide is highly toxic to freshwater fish. The fish species which have been evaluated include bluegill sunfish (LC50 of 0.8 mg/L) and rainbow trout (LC50 of 0.5 mg/L) [1]. Carp were also found to be susceptible to zinc phosphide, especially in weakly acidic water.
- **Effects on other organisms:** Zinc phosphide is also toxic to non-target mammals when ingested directly [8]. Nearly 60 studies have been conducted on the toxicity of this rodenticide to wild animals. Secondary toxicity to mammalian predators (animals eating other animals that had been exposed to the compound) from zinc phosphide is rather low, primarily because the compound does not significantly accumulate in the muscles of target species [8]. Some of the toxic effects to predators have been due to

the ingestion of zinc phosphide that was in the digestive tract of the target organism. Studies on secondary organisms have focused on coyotes, fox, mink, weasels, and birds of prey. Under field conditions, most of the toxic effects to non-target wildlife are due to direct exposures resulting from misuse or misapplication of this rodenticide [8].

Environmental Fate:

- **Breakdown in soil and groundwater:** Zinc phosphide may be applied as an active ingredient in either bait or a dust. Under average conditions, toxic activitity persists for approximately 2 weeks [8]. Soil acidity and moisture tend to accelerate the breakdown of the compound [8]. Phosphine gas may be liberated as a result of this process.
- **Breakdown in water:** No data are currently available.
- **Breakdown in vegetation:** No data are currently available.

Physical Properties:

- **Appearance:** Zinc phosphide is an amorphous black-grey powder with a garlic-like odor [1]. It is stable when dry and decomposes in moist air [1].
- **Chemical Name:** trizinc diphosphide [8]
- **CAS Number:** 1314-84-7
- **Molecular Weight:** 258.09
- **Water Solubility:** Practically insoluble in water (decomposes slowly) [8]
- **Solubility in Other Solvents:** Practically i.s. in alcohol; s.s. in benzene and carbon disulfide [8]
- **Melting Point:** >420 C [8]
- **Vapor Pressure:** Negligible in the dry state (as solid) [8]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** Not Available

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.0003 mg/kg/day [13]
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Hacco, Inc.
P.O. Box 7190
537 Atlas Ave.
Madison, WI 53707

- **Phone:** 608-221-6200
- **Emergency:** 800-642-4699

References:

References for the information in this PIP can be found in Reference List Number 10
