E X T O X N E T

Extension Toxicology Network

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Pesticide
Information
Profile

Dichlorvos

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TRADE OR OTHER NAMES

Apavap, Benfos, Cekusan, Cypona, Derriban, Derribante Devikol, Didivane, Duo-Kill, Duravos, Elastrel, Fly-Die, Fly-Fighter, Herkol, Marvex, No-Pest, Prentox, Vaponite, Vapona, Verdican, Verdipor, Verdisol. Trade names used outside of the U.S. include Doom, Nogos, and Nuvan (2).

REGULATORY STATUS

A Special Review of dichlorvos was initiated in February 1988 because EPA determined that the registered uses of dichlorvos may pose a risk of cancer as well as inadequate margins of safety for cholinesterase inhibition and liver effects to exposed persons (12). The Special Review was not complete as of March 1992 (10). Products containing dichlorvos must bear the signal words "Danger-Poison" (2).

INTRODUCTION

Dichlorvos is used to control household, public health, and stored product insects. It is effective against mushroom flies, aphids, spider mites, caterpillars, thrips, and white flies in greenhouse, outdoor fruit, and vegetable crops (2). Therapeutically, dichlorvos is used to treat a variety of parasitic worm infections in dogs, livestock and humans. Dichlorvos can be fed to livestock to control botfly larvae in the manure. It acts against insects as both a contact and a stomach poison (2). Dichlorvos is available in aerosol and soluble concentrate formulations (2). It is used as a fumigant (2) and has been used to make pet collars and pest strips (3).

Dichlorvos is one of a class of insecticides referred to as organophosphates. These chemicals act by interfering with the activities of cholinesterase, an enzyme that is essential for the proper working of the nervous systems of both humans and insects.

Please refer to the Toxicology Information Brief on cholinesterase-inhibition for a more detailed description of this topic.

In 1955, it was discovered that crystalline trichlorfon, another organophosphate pesticide, gave off a vapor which was capable of killing insects. That vapor was dichlorvos, which has since been developed for insect control in enclosed spaces (3).

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Dichlorvos is highly toxic by inhalation, dermal absorption and ingestion (9). Because dichlorvos is volatile, inhalation is the most common route of exposure. As with all organophosphates, dichlorvos is readily absorbed through the skin. Skin which has come in contact with this material should be washed immediately with soap and water and all contaminated clothing should be removed.

Acute illness from dichlorvos is limited to the effects of cholinesterase inhibition. Compared to poisoning by other organophosphates, dichlorvos causes a more rapid onset of symptoms, which is often followed by a similarly rapid recovery (3). This occurs because dichlorvos is rapidly metabolized and eliminated from the body. Persons with reduced pulmonary (lung) function, convulsive disorders, liver disorders, or recent exposure to cholinesterase inhibitors will be at increased risk from exposure to dichlorvos. Alcoholic beverages may enhance the toxic effects of dichlorvos. High environmental temperatures or exposure of dichlorvos to visible or UV light may enhance its toxicity (9).

Dichlorvos is mildly irritating to skin (9). Concentrates of dichlorvos may cause burning sensations, or actual burns (6). Dichlorvos can be very toxic if it is not immediately washed off, but instead left on the skin long enough for it to become absorbed through the skin and into the bloodstream. One man nearly died after spilling 4 ounces of a 3% oil solution of dichlorvos on his lap. He did not wash it off. Another man only became nauseous and dizzy after spilling a similar amount on his arm. He washed off the dichlorvos with soap and water (6). Do not use organic solvents to remove dichlorvos from the skin (DLA/DOD Hazardous Mat'ls Info. System #0014-29- 438-0000. 1982).

The organophosphate insecticides are cholinesterase inhibitors. They are highly toxic by all routes of exposure. When inhaled, the first effects are usually respiratory and may include bloody or runny nose, coughing, chest discomfort, difficult or short breath, and wheezing due to constriction or excess fluid in the bronchial tubes. Skin

contact with organophosphates may cause localized sweating and involuntary muscle contractions. Eye contact will cause pain, bleeding, tears, pupil constriction, and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours. These may include pallor, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, eye pain, blurred vision, constriction or dilation of the eye pupils, tears, salivation, sweating, and confusion. Severe poisoning will affect the central nervous system, producing incoordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids, and eventually paralysis of the body extremities and the respiratory muscles. In severe cases there may also be involuntary defecation or urination, psychosis, irregular heart beats, unconsciousness, convulsions and coma. Death may be caused by respiratory failure or cardiac arrest (9).

Some organophosphates may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure which may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness and cramping may appear in the lower limbs and progress to incoordination and paralysis. Improvement may occur over months or years, but some residual impairment will remain (9).

The administration of slow-release formulations of dichlorvos to domestic animals to treat for internal parasites has caused some inhibition of cholinesterase and mild symptoms such as nausea or diarrhea, but no serious signs of illness. Repeated, small doses generally have no effect on treated animals. Doses of up to 4 mg/kg of a slow release formulation, given to cows to reduce flies in their feces, had no visibly adverse effects on the cows. Blood tests of these cows indicated cholinesterase inhibition (3).

Dichlorvos is very volatile, meaning that it readily forms vapors which may be inhaled. Inhalation is the most common way to be exposed to dichlorvos. Low, repeated doses may be non-toxic. High doses of dichlorvos may be very toxic, especially if inhalation exposure is continuous (6). Dichlorvos produces irritating gases, such as phosphorous and chlorine oxides, when heated (NIH/EPA 1984).

Eye protection should be worn when handling dichlorvos. Application of 1.67 mg/kg in rabbits' eyes produced mild redness and swelling, but no injury to the cornea (9). Dichlorvos may cause eye burns. Organophosphates cause the pupils to constrict (pin point pupils).

The amount of a chemical 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 oral LD50 for dichlorvos in mice is 61 to 175 mg/kg, 100 to 1090 mg/kg in dogs, 15 mg/kg in chickens, 25 to 80 mg/kg in rats, 157 mg/kg in pigs, and 11 to 12.5 mg/kg in rabbits

(2, 6, 9). The dermal LD50 for dichlorvos in rats is 70.4 to 250 mg/kg, 206 mg/kg in mice, and 107 mg/kg in rabbits (2, 3, 6, 9).

The lethal concentration fifty, or LC50, is that concentration of a chemical in air or water that kills half of the experimental animals exposed to it for a set time period. The 4-hour LC50 for dichlorvos in rats is 15 mg/m3, and 13 mg/m3 in mice (9).

CHRONIC TOXICITY

Feeding studies indicate that a dosage of dichlorvos very much larger than doses which inhibit cholinesterase are needed to produce illness. Rats tolerated dietary doses as high as 62.5 mg/kg/day for 90 days with no visible signs of illness, while a dietary level of 0.25 mg/kg/day for only 4 days produced a reduction in cholinesterase levels (3).

Rats were exposed to air concentrations of 0, 0.05, 0.5 and 5 mg/m3 of dichlorvos over a 5 week period. Rats in the 0.5 and 5 mg/kg groups exhibited significantly decreased cholinesterase activity in the plasma, red blood cells, and brain. The NOEL for this study was 0.05 mg/m3. In dogs fed dietary doses of 0.0095, 0.016, 0.16, 1.6 or 12.5 mg/kg/day for 2 years, decreased red blood cell cholinesterase activity, increased liver weights and increased liver cell size occurred in the two highest doses tested. The NOEL was 0.08 mg/kg/day (12). Chronic exposure to dichlorvos will cause fluid to build up in the lungs (pulmonary edema) (NIH/EPA; OHM/TADS 1984).

Repeated or prolonged exposure to organophosphates may result in the same effects as acute exposure including the delayed symptoms. Other effects reported in workers repeatedly exposed include impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise has also been reported (9).

Reproductive Effects

When male and female rats were given a diet containing 100 ppm (5 mg/kg/day) dichlorvos just before mating, and with this dosage continued through pregnancy and lactation for females, there were no effects on reproduction or on the survival or growth of the offspring, even though severe cholinesterase inhibition occurred in the mothers and significant inhibition occurred in the offspring. The same results were observed in a 3-generation study with rats fed dietary levels up to (25 mg/kg/day) (3). Once in the bloodstream, dichlorvos may cross the placenta (9).

Teratogenic Effects

A dose of 12 mg/kg was not teratogenic in rabbits and did not interfere with reproduction in any way. There was no evidence of teratogenicity when rats and rabbits were exposed to air concentrations of up to 6.25 mg/m3 throughout pregnancy. Dichlorvos was not teratogenic when given orally to rats (3).

Mutagenic Effects

Dichlorvos can bind to molecules such as DNA. For this reason, there has been extensive testing of dichlorvos for mutagenicity. Several studies reviewed by EPA have shown dichlorvos to be a mutagen (12). Dichlorvos is reported positive in the Ames mutagenicity assay (Mut. Res. 87:211 (1981); 76:169 (1980); 40 (1):19 (1976) and in other tests involving bacterial or animal cell cultures. However no evidence of mutagenicity has been found in tests performed on live animals. Its lack of mutagenicity in live animals may be due to rapid metabolism and excretion of dichlorvos (3).

Carcinogenic Effects

Dichlorvos has been classified as a possible human carcinogen by EPA because of the results of tests on rats and mice (11). When dichlorvos was administered by gavage to mice for 5 days per week for 103 weeks at doses of 10 or 20 mg/kg to males and 20 or 40 mg/kg to females, there was an increased incidence of benign tumors in the lining of the stomach at the high dose for both sexes. When rats given daily doses of 0, 4 or 8 mg/kg for five days per week for 103 weeks, there was an increased incidence of benign tumors of the pancreas and of leukemia in male rats at both doses. At the highest dose, there was also an increased incidence of benign lung tumors in males. In female rats, there was an increase in the incidence of benign tumors of the mammary gland (12). No tumors caused by dichlorvos were found in rats fed up to 25 mg/kg/day for 2 years or in dogs fed up to 11 mg/kg/day for 2 years. No evidence of carcinogenicity was found when rats were exposed to air containing up to 5 mg/m3 for 23 hours/day for 2 years (3). A few tumors were found in the esophagus of mice given dichlorvos orally, even though tumors of this kind are normally rare (9).

Organ Toxicity

Dichlorvos primarily affects the nervous system through cholinesterase inhibition, by which there is a deactivation of cholinesterase, an enzyme required for proper nerve functioning.

Dichlorvos causes fluid to accumulate in the lungs (6). Liver enlargement has occurred in pigs maintained for long periods of time on high doses (500 ppm) (3, 6). Dichlorvos caused adverse liver effects in dogs (12). Lung hemorrhages may occur (14). Cholinesterase inhibition may affect the nervous system. In mice, a single oral dose of 40 micrograms (ug)/kg caused changes in the testes. In male rats, repeated doses caused abnormalities in the tissues of the lungs, heart, thyroid, liver and kidneys (9).

Fate in Humans and Animals

Amongst the organophosphates, dichlorvos is remarkable for its rapid metabolism and excretion by mammals. Dichlorvos was not detected in the blood of rats, mice or people after exposure to atmospheric concentrations of up to 17 times that normally reached for insect control in homes. Exposure of rats to 11 mg/m3 (250 times the normal exposure) for 4 hours was required before dichlorvos was detectable in the rats. Even then, it was detected only in the kidneys. At 90 mg/m3 (2000 times normal exposure), dichlorvos was detected in most tissues of the rat. Following exposure to 50 mg/m3, the half-life for dichlorvos in the rat kidney was 13.5 minutes. The reason for this rapid disappearance of dichlorvos is the presence of degrading enzymes in both tissues and blood plasma. From the gastrointestinal tract, dichlorvos is absorbed into the portal blood, rather than into the general bloodstream. From the portal blood, it is moved to the liver where it is rapidly detoxified. Thus poisoning by nonlethal doses of dichlorvos is usually followed by rapid detoxification in the liver and recovery. Rats given oral or dermal doses at the LD50 level either died within one hour of dosing or recovered completely (3, 6).

Dichlorvos does not accumulate in body tissues and has not been detected in the milk of cows or rats, even when the animals were given doses high enough to produce symptoms of severe poisoning (3).

ECOLOGICAL EFFECTS

Effects on Birds

Dichlorvos is highly toxic to birds including ducks and pheasants (4, 8). The LD50 for wild birds fed dichlorvos is 12 mg/kg (NIOSH RTECS Online File #82/8110).

Effects on Aquatic Organisms

UV light makes dichlorvos more toxic to aquatic life by 5-150 times (15). NIH/EPA found the grass shrimp to be more sensitive to dichlorvos than the sand shrimp, hermit crab and mummichog (in that order) (1984). For ocean-dwelling species they found:

scud > Atlantic silverside > striped killfish > striped mullet > bluehead > American eel > northern puffer; where ">" indicates a greater sensitivity to dichlorvos. The 96-hour LC50 for dichlorvos in fathead minnow is 11.6 mg/l, 0.9 mg/l in bluegill, 5.3 mg/l in mosquito fish, 0.004 ppm in sand shrimp, 3.7 ppm in mummichogs, and 1.8 ppm/96 hours in American eels (NIH/EPA 1984). The 24-hour LC50 for dichlorvos in bluegill sunfish is 1.0 mg/l (2).

Dichlorvos does not significantly bioaccumulate in fish (4).

Effects on Other Animals (Nontarget species)

Dichlorvos is toxic to bees (2).

ENVIRONMENTAL FATE

Breakdown of Chemical in Soil and Groundwater

Dichlorvos does not adsorb to soil particles and it is likely to contaminate groundwater. When spilled on soil, dichlorvos leached into the ground with 18 to 20% penetrating to a depth of 30 cm within 5 days. In soil, dichlorvos is subject to hydrolysis and biodegradation. Volatilization from moist soils is expected to be slow. Half-lives of 7 days were measured on clay, sandy-clay, and loose sandy soil (4).

Dichlorvos is rapidly broken down in the air and in damp media such as soil. The pH of the media determines the rate of breakdown. Alkaline soils, water, etc., show rapid breakdown, whereas acidic media shows slow degradation. For instance, at a pH of 9.1 the half-life of dichlorvos is about 4.5 hours. At a pH of 1 (very acidic), the half-life is 50 hours (8). Dichlorvos is non-persistent.

Breakdown of Chemical in Water

In water dichlorvos remains in solution and does not adsorb to sediments. It degrades primarily by hydrolysis, with a half-life of approximately 4 days in lakes and rivers. This half-life will vary from 20 to 80 hours between pH 4 and pH 9. Hydrolysis is slow at pH 4 and rapid at pH 9 (4, 5). Biodegradation may occur, especially under acidic conditions which slow hydrolysis, or where populations of acclimated micro-organisms exist, as in polluted waters. Volatilization from water is expected to be slow. The volatilization half-life from river and pond waters have been estimated at 57 and over 400 days respectively (4).

Breakdown of Chemical in Vegetation

Except for cucumbers, roses, and some chrysanthemums, plants tolerate dichlorvos very well (5).

PHYSICAL PROPERTIES AND GUIDELINES

Dichlorvos is a colorless to amber liquid with a mild chemical odor. Dilute dichlorvos breaks down rapidly in the presence of moisture. Concentrated forms are readily decomposed by strong acids and bases (3). Dichlorvos is stable under normal temperatures and pressures, but it may pose a moderate fire hazard if exposed to heat or flame. It may hydrolyze on contact with moisture, and may decompose in the presence of strong acids or bases (3, 9). Thermal decomposition of dichlorvos will release toxic oxides of phosphorus and carbon, toxic and corrosive chlorides and toxic phosgene gas. Dichlorvos is corrosive to iron and steel. It may attack materials such as plastics, rubber and coatings (9). Other metals (stainless steel, aluminum, nickel) are resistant if no water is present.

Dichlorvos increases the effects of malathion (5). Alcoholic beverages promote the absorption of dichlorvos into the bloodstream (8).

Persons who work with organophosphate materials for long periods of time should have frequent blood tests of their cholinesterase levels. If the cholinesterase level falls below a critical point, no further exposure should be allowed until it returns to normal (13).

Protective clothing must be worn when handling dichlorvos. Before removing gloves, wash them with soap and water. Always wash hands, face and arms with soap and water before smoking, eating or drinking.

After work, remove all work clothes and shoes. Shower with soap and water. Wear only clean clothes when leaving the job. Wash contaminated clothing and equipment with soap and water after each use. Keep contaminated work clothes separate from regular laundry.

Exposure Guidelines:

1 mg/m3 OSHA TWA (skin) (9)
0.1 ppm (0.9 mg/m3) ACGIH TWA (skin) (9)
1 mg/m3 NIOSH Recommended TWA (skin) (9)
Air concentrations of 200 mg/m3 are immediately dangerous to life or health (9).
PADI: 8 x 10 to the minus 4 power mg/kg/day, based on a 2-year dog feeding study (12)

Physical Properties

CAS #:	62-73-7
Specific gravity:	1.44 (60 degrees /60 degrees F) (2)
Solubility in water:	1 g/100g at 25 degrees C (17)
Solubility:	Miscible in non-polar solvents such as dichloromethane, 2-propanol and toluene (2, 17). Soluble in ethanol, chloroform, acetone, and kerosene (1, 5). Miscible in alcohol and in aromatic and chlorinated hydrocarbon solvents. Solubility in kerosene and mineral oils is about 3% (3).
Boiling point:	140 degrees C at 20 mm Hg (17); 117 degrees C at 11 mm Hg (2); 35 degrees C at 0.05 mm Hg (3); 183 degrees F (84 degrees C) (9)
Flash point:	>175 degrees F (>80 degrees C) (2, 16), practically non-flammable (17).
Vapor pressure:	0.01 mm Hg at 30 degrees C (18)
Chemical class/use:	Organophosphate insecticide

BASIC MANUFACTURER

Amvac Chemical Corp.
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Review by Basic Manufacturer

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