E X T O X N E T

Extension Toxicology Network

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

Pesticide
Information
Profile

Azinphos Methyl

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TRADE OR OTHER NAMES

Common names include azinphos methyl and metiltriazotion. Trade names include Cotnion-methyl, Gusathion, Guthion, Methyl-Guthion, Bay 17147, Carfene, Gusathion-M, Bay 9027.

REGULATORY STATUS

Azinphos methyl was initially registered as a pesticide under FIFRA in 1957 (40). All azinphos methyl liquids with a concentration greater than 13.5% are classified as Restricted Use Pesticides (RUP) by the U. S. Environmental Protection Agency (EPA), because of the human inhalation hazard and acute toxicity presented by this material, as well as its potential adverse effects to other mammalian species, birds, and aquatic organisms. RUPs may be purchased and used only by certified applicators (5, 28). A registration standard was issued for azinphos methyl in September, 1986 (27). The EPA has imposed a 24-hour reentry interval for this material. Areas that have been treated with azinphos methyl may not be reentered for at least 24 hours, unless protective clothing is worn (29, 15). Products containing azinphos methyl must bear the signal words "Danger" and "Poison" (36). Check with specific state regulations for local restrictions which may apply.

INTRODUCTION

Azinphos methyl is a highly persistent, broad spectrum insecticide. It is also an acaricide, toxic to mites and ticks, and a molluscicide, poisonous to snails and slugs. It is one of a group of organic compounds called organophosphates because of the presence of phosphorous in their molecular structures. It is a nonsystemic material, meaning that it is not transported from one plant part to another. It is used primarily as a foliar application against leaf-feeding insects. It works as both a contact insecticide and a stomach poison. Contact poisons are effective upon contact with any part of the target organism. Stomach poisons become toxic when they are eaten by an insect (15, 2, 30, 3).

Azinphos methyl is registered for use in the control of many insect pests on a wide variety of fruit, vegetable, nut, and field crops, as well as on ornamentals, tobacco, and forest and shade trees (15, 2, 30, 3). Outside of the U.S., azinphos methyl is used in lowland rice production (36). On 1986, approximately three million pounds of this active ingredient were used against more than 200 pests on about 50 different sites (29). Azinphos methyl is available in emulsifiable liquid, liquid flowable, ULV liquid, and wettable powder formulations (36).

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Azinphos methyl is one of the most toxic of the organophosphate insecticides (20). It is highly toxic by inhalation, dermal absorption, ingestion, and eye contact (35, 39). Like all organophosphate chemicals, azinphos methyl is referred to as a "cholinesterase inhibitor." It binds up, blocks, or damages, the normal functioning of cholinesterase, an enzyme which is essential to the proper working of the nervous system. Individuals with a history of reduced lung function, convulsive disorders, or recent exposure to other cholinesterase inhibitors will be at increased risk from exposure to azinphos methyl (39).

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. Azinphos methyl is easily absorbed by the skin (35). 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 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 (39).

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, and in some cases residual impairment will remain (39).

For humans, ingestion of azinphos methyl in amounts above 1.5 mg/day can cause severe poisoning with symptoms, such as dimness of vision, salivation, excessive sweating, stomach pain, vomiting, diarrhea, unconsciousness and death (20). Inhalation of the dust or aerosol preparation of azinphos methyl may cause difficulty in breathing and vision, with symptoms of wheezing, tightness in the chest, blurred vision, and tearing of the eyes. Dogs fed 300 parts per million (ppm), or about 9 mg/kg, per day showed tremors, weakness, abnormal quietness, and some weight loss (11). Complete symptomatic recovery may occur within one week after sublethal poisoning: poisoning from an exposure that is just below the amount necessary to be fatal.

Azinphos methyl has caused worker illness (15). Human exposure to azinphos methyl is most likely during the handling of its concentrated forms and with airblast applications. Five milligrams per cubic meter (mg/m3) can be immediately dangerous to life or health. Health risks from these types of exposures can be reduced by the use of a respirator and protective clothing, as well as by strictly following of the 24-hour reentry interval set for this insecticide by the EPA (30).

Pure azinphos methyl is easily absorbed by the skin and lethal amounts can build up in the body after dermal exposure. Symptoms of illness caused by this type of exposure include nausea, vomiting, blurred vision and muscle cramps (11, 20).

Eye contact with concentrated solutions of azinphos methyl can pose a threat to life. Research has shown that within a few minutes of ocular exposure, this material may cause pain, blurring of distant vision, tearing and other ocular problems. Symptoms of cholinesterase inhibition may also occur, such as respiratory difficulties, gastrointestinal problems and central nervous systems disturbances (20, 39).

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. There is wide variation in the recorded LD50s for azinphos methyl depending on the route of exposure and the test animal. The oral LD50 for azinphos methyl in rats is 4.4 to 16 mg/kg (3, 11, 30, 33), in guinea pigs is 80 mg/kg (3, 10), and in mice is 8 to 20 mg/kg (11). The dermal LD50 in rats is 88 to 220 mg/kg (2, 4, 10, 30), and in mice is 65 mg/kg (39).

The lethal concentration fifty, or LC50, is that concentration of a chemical in air or water that kills half (50%) of the experimental animals given exposure to it for a set time period. The 1-hour inhalation LC50 for azinphos methyl in rats is 69 mg/m3 (39).

CHRONIC TOXICITY

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 depression, irritability, confusion, headache, speech difficulties, delayed reaction time, nightmares, sleepwalking and drowsiness or insomnia (39).

Long-term exposure to azinphos methyl, above the average 8-hour standard set by the Occupational Safety and Health Administration (OSHA), can bring about the following symptoms: impairment of concentration and memory, headache, irritability, nausea, vomiting, muscle cramps and dizziness (20).

Cholinesterase inhibition from exposure to this material may persist for two to six weeks. Repeated exposure to small amounts of azinphos methyl may result in an unexpected inhibition of cholinesterase, causing symptoms that resemble other flu-like illnesses: general discomfort, weakness, and lack of appetite. Exposure to a concentration of azinphos methyl that might not produce symptoms in a person who was not previously exposed may produce severe symptoms of cholinesterase inhibition in a previously exposed person. In a study of eight workers who were dermally exposed to concentrations of up to 9.6 mg/m3, the lowest activity of cholinesterase in the blood serum was 78% of the value before exposure, and there were no signs of illness.

Rats fed 2 or 5 mg/kg of azinphos methyl for 60 days showed no health effects, but concentrations of 10 mg/kg or greater did affect health (10). Rats tolerated this material at dietary doses of 0.25 mg/kg/day 60 days without cholinesterase inhibition. 1 mg/kg/day resulted in questionable growth effects and slight inhibition of brain and red blood cell cholinesterase. These dietary levels are approximately equal to 17 and 70 mg per human per day (1).

In chronic oral toxicity studies, rats and dogs were fed doses of 0.125, 0.25, 1, or 2.5 mg/kg/day. The 2.5 mg/kg dose was increased to 5 mg/kg after 47 weeks. The level at which cholinesterase was not affected (cholinesterase no-effect level) was found to be 0.125 mg/kg/day in both rats and dogs. At 1 mg/kg, both the plasma and red blood cell cholinesterase in the rat were initially inhibited, but both returned to normal after 65 weeks. The 5 mg/kg produced convulsions in some animals. In dogs, 0.5 mg/kg produced only a slight, irregular decrease in red blood cell cholinesterase (3, 41).

Rats fed azinphos methyl for two years at rates of 50 ppm, and later 100 ppm (about 5 to 10 mg/kg/day), ate and grew normally, and had normal kidney and red blood cell function. However, all animals had

depressed plasma, red blood cell counts, and brain cholinesterase activity. Dietary levels of about 0.5 mg/kg/day (5 ppm) or less did not have negative effects (11).

Reproductive Effects

In a 2-generation rat reproduction study, the NOEL for reproductive and maternal effects was 0.25 mg/kg (41).

Teratogenic Effects

In a rat teratology study the NOEL for maternal and developmental effects was greater than 2 mg/kg (41).

Mutagenic Effects

No mutagenic effects were observed in three tests submitted to the EPA. These tests were the Ames test on bacteria, an unscheduled DNA synthesis test, and a test on human cell cultures (41).

Carcinogenic Effects

A carcinogenicity study suggested that tumors of the pancreas and selected thyroid cells may have been associated with azinphos methyl (11). Upon reevaluation, however, the EPA found this study unacceptable, and it required that further tumor production, or oncogenicity, data be collected and submitted. One oncogenicity study did not show an increase in the incidence of tumors in mice from this material (30). In another study, there were no tumor-producing effects in rats fed azinphos methyl at rates of 50 ppm and 10 ppm (about 5 to 10 mg/kg/day) (11).

Organ Toxicity

Toxicity for azinphos methyl is primarily manifested in cholinesterase inhibition. Female rats given sublethal injections of this material into their abdominal cavities showed similar levels of cholinesterase inhibition in the tissue of the brain, salivary glands, and blood serum (3).

Fate in Humans and Animals

There are no valid studies on the way that azinphos methyl metabolizes, or goes through biochemical changes in structure. The EPA required that these metabolism data gaps be fulfilled (29). One study did suggest that Guthion is rapidly detoxified, or broken down into nonpoisonous forms, in the body (3). Although the submitted studies are not adequate to assess the fate of this material in animals, azinphos methyl breakdown products have been identified in chicken excreta and rat urine (30).

ECOLOGICAL EFFECTS

Effects on Birds

Azinphos methyl is moderately toxic to birds. Acute symptoms of azinphos methyl poisoning in birds include regurgitation, wing drop, wing spasms, diarrhea, lack of movement, etc. (25). When chickens were fed azinphos methyl at a dosage of 40 mg/kg, the chickens developed leg weakness. The mode of the action of this material was unknown (22).

The oral LD50 for azinphos methyl in young mallards is 136 mg/kg, 74.9 mg/kg in young pheasant, 84.2 mg/kg in young chukar partridges (30, 22), 262.0 mg/kg in chickens (3), and 32.2 mg/kg in bobwhite quail (36).

The LC50 for azinphos methyl in bobwhite quail is 488 ppm, 639 ppm in Japanese quail, 1821 ppm in ring-necked pheasant, and 1940 ppm in mallard duck (29).

Effects on Aquatic Organisms

The acute toxicity data on freshwater fish indicate that azinphos methyl is moderately to very highly toxic, depending on the species tested. Most values were in the very highly toxic range (less than 100 ug/l). The 96-hour LC50 for azinphos methyl in rainbow trout is 0.003 mg/l (36). The use of azinphos methyl in vegetable and fruit production has resulted in occasional contamination of nearby streams, leading to local fish kills. The longer the time that fish are exposed to this material, the larger the number of expected fish deaths (15).

Guthion-poisoned fish exhibit impairment of the central nervous system, a response pattern that is typical of organophosphate toxicity. Erratic swimming, accompanied by uncontrolled convulsions at varying intervals may be seen in fish that are poisoned by this material. Rapid gill movements, paralysis, and death follow in rapid succession (3).

Azinphos methyl is highly toxic to aquatic invertebrates - waterborne species that lack spinal columns. Toxicity is 0.13 to 56 ug/l, depending on the species tested (30). Studies suggest that azinphos methyl is toxic to shellfish, frogs and toads (3). The EPA required additional information on the behavior of azinphos methyl in aquatic environments, so that the extent of exposure and hazard can be fully assessed (29).

Effects on Other Animals (Nontarget species)

Several studies have indicated that azinphos methyl causes adverse effects to wildlife. Wild mammals and aquatic organisms appear to be more vulnerable than birds to hazards created by this material (29). The EPA required extensive field monitoring data to better define the extent of exposure and hazard to wildlife. Endangered species labeling was mandated for certain azinphos methyl uses (30).

Azinphos methyl is toxic to honeybees and other beneficial insects (3, 36). Severe losses may be expected if it is used when bees are present at treatment time or within a day thereafter (18). A 90-percent mortality rate was seen in pollinating leaf-cutting bees after a nine- day exposure to greenhouse alfalfa that had been treated with this material (22).

ENVIRONMENTAL FATE

Breakdown of Chemical in Soil and Groundwater

Azinphos methyl is fairly immobile in soil because it adsorbs strongly to soil particles and is of low solubility in water. It therefore has low leaching potential and is unlikely to contaminate groundwater (29, 37, 38). It was not detected in 54 groundwater samples collected in New York state (12). Azinphos methyl is one of 118 synthetic organic chemicals that the state of Florida has designated for groundwater monitoring (19).

The disappearance of azinphos methyl from soil is more rapid in the surface layers (0-2.5 centimeters deep) than it is in the next deeper layer (2.4-7.5 cm). This disappearance of the insecticide results from many natural breakdown processes. Azinphos methyl is subject to microbial degradation, degradation by ultraviolet (UV) light from the sun, hydrolytic decomposition in the presence of water, and it volatilizes when it is exposed to air at ordinary temperatures. Biodegradation and volatilization are most likely the primary routes of disappearance for azinphos methyl applied to soil surfaces or incorporated into the upper soil layer. Chemical hydrolysis is probably not important in other than alkaline soils (38). Photodecomposition is particularly obvious with high levels of soil moisture and in the presence of UV light (33). Rapid degradation of Gusathion was observed at temperatures higher than 37 degrees C. Degradation was further enhanced when water was present (29).

Estimates vary on how quickly azinphos methyl disappears from the soil: two to four weeks (13); less than one year (33); after 30 days at 40 degrees C in sandy loam, with a half-life in sandy loam of five days. Derivatives of this material were not found in field soil after four years. Field studies on the potential of azinphos methyl to break down and dissipate in soil demonstrate that it is not persistent, and is 90% degraded within 30 days (29).

There is a lag time in both sterile and nonsterile soils, between application of azinphos methyl and its disappearance in the soil. This indicates that degradation is not strictly biological. Its half-life in nonsterile soil is reportedly 21 days in conditions with oxygen (aerobic) or 68 days under oxygen-free, 'anaerobic' conditions. In sterile soil, the half-life is reported to be 355 days (30).

The few reports on the persistence of this material in soil suggest that its metabolism can be influenced by biological or chemical processes (33). Available data on azinphos methyl indicate that there is no buildup of residue amounts in a variety of soil types. A study on the persistence of different Gusathion formulations and concentrations in sandy soil showed that undiluted insecticide can remain in soils for up to four years (14).

Breakdown of Chemical in Water

In general, organophosphates are dissipated rapidly in water. Azinphos methyl is not persistent in water (7). In pond water, azinphos methyl is subject to degradation by sunlight and microorganisms, with a half-life of up to two days (3). Biodegradation is probably the most important degradation process for azinphos methyl in natural waters. Volatilization from water is unlikely. Chemical hydrolysis is probably important only in alkaline waters (38). In one study, azinphos methyl was very stable in water below pH 10.0. Above pH 11.0, it was rapidly hydrolyzed to anthranilic acid, benzamide, and other metabolites (14). Azinphos methyl has a low to medium tendency to adsorb to sediments or suspended solids or to bioconcentrate (38).

Based on its high toxicity to fish and other aquatic organisms, extreme care should be taken not to contaminate open waters through drifting, spray application, equipment cleaning or waste disposal. Contamination of streams and local fish kills have been caused by the use of azinphos methyl in fruit and vegetable production (29).

Breakdown of Chemical in Vegetation

Residue levels of azinphos methyl in crops are dependent on a number of factors, including rate and frequency of application, nature of the plant surface and weather conditions such as rainfall, temperature, sunlight, humidity and wind (3). The approximate residual period of this material on plants is 1 to 3 weeks. It gives effective protection for two or more weeks (9, 15). Field tests with azinphos

methyl indicated that, on treated apple trees, the half-life of this pesticide was about 2.6 to 6.3 days (17). Hawthorn and American Linden trees have been injured by this material. It has also caused russeting on certain varieties of fruit (24). The half-life for Guthion on vegetable and forage crops ranges from three to five days under field conditions (3).

PHYSICAL PROPERTIES AND GUIDELINES

Pure azinphos methyl is a white-crystalline solid. Technical azinphos methyl is a brown waxy solid (30, 35). Azinphos methyl is chemically stable under normal temperatures and pressures, and can be stored under ordinary conditions for an unlimited period of time. However, high temperatures may cause gas evolution and the development of pressure in enclosed containers. Azinphos methyl is unstable above 200 degrees C. It poses a negligible fire hazard when exposed to heat or flame, but poses a fire and explosion hazard in the presence of strong oxidizers. It may hydrolyze in the presence of acids or alkalis. Thermal decomposition of azinphos methyl may release toxic oxides of nitrogen, phosphorus, sulfur and carbon (16, 39).

Azinphos methyl can be applied together with most other insecticides and fungicides, but it is incompatible with alkaline preparations and formulating materials (10). A recent study indicates that the addition of calcium to azinphos methyl slows its chemical breakdown enough for it to provide more effective insect control. Without the added calcium, azinphos methyl often breaks down before it comes into contact with vulnerable stages of the target pest's life cycle. For example, the coddling moth has a life cycle of 21-days (26).

Exposure Guidelines:

0.2 mg/m3 OSHA TWA (skin) (39)

0.2 mg/m3 ACGIH TWA (skin) (39)

0.2 mg/m3 NIOSH Recommended TWA (skin) (39)

Concentrations of 5 mg/m3 or more in air are immediately dangerous to life and/or health (39)

TLV: 0.2 mg/m3 (1, 11), indicating that azinphos methyl is considered safe for occupational intake at a rate of about 0.03 mg/kg/day (11)

ADI: 0.0025 mg/kg (6)

PADI: 0.0013 mg/kg/day, based on a NOEL of 0.125 mg/kg/day in a 2-year feeding study with dogs and a 100-fold safety factor (41)

STEL: 0.6 mg/m3 (1)

Physical Properties:

CAS #:	86-50-0
Specific gravity:	1.44 at 20 degrees C (35)
H2O solubility:	30 mg/l at 25 degrees C (10)
Solubility in other solvents:	Soluble in dichloromethane, ethanol, methanol, n-hexane, 2-propanol, propylene glycol, toluene, xylene and other organic solvents except aliphatics (4, 36).

Can recrystallize from methanol and isopropanol (10)

Melting point:	(pure) 73 - 74 degrees C (1, 11, 10); (technical) 65 - 68 degrees C (11)
Flashpoint:	150 degrees F (36)

Decomposition temperature:	> 200 degrees (11). Decomposes at elevated temperatures with gas evolution (36).
Vapor pressure:	less than 3.8 x 10 to the minus 4 power mm Hg at 20 degrees C (11); less than 10 to the minus 5 power mbar at 20 degrees C (10); 2.2 x 10 to the minus 7 power mm of Hg at 20 degrees C (16)
Chemical Class/Use:	Organophosphate insecticide, acaricide, molluscicide
Koc:	1,000 g/ml (37)

BASIC MANUFACTURER

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Review by Basic Manufacturer:

Comments solicited: October, 1992
Comments received:

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Questions regarding the development of this web site should be directed to the PMEP Webmaster