



DELTAMETHRIN



A sneak peek into the toxicological aspects, data, reviews, reports and facts about one of the most commonly used insecticides in today's world!!!

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INTRODUCTION

Deltamethrin is a pyrethroid insecticide that kills insects on contact and through digestion. It is used to control apple and pear suckers, plum fruit moth, caterpillars on brassicas, pea moth, aphids (apples, plums, hops), winter moth (apples and plums), codling and tortrix moths (apples), in the control of aphids, mealy bugs, scale insects, and whitefly on glasshouse cucumbers, tomatoes, peppers, potted plants, and ornamentals. It also controls numerous insect pests of field crops. Formulations include emulsifiable concentrates, wettable powders, ULV and flowable formulations and granules.

Deltamethrin is a synthetic insecticide based structurally on natural pyrethrins, which rapidly paralyze the insect nervous system giving a quick knockdown effect. Deltamethrin has a rapidly disabling effect on feeding insects and for this reason there is hope that it may be useful to control the vectors of "non-persistent" viruses (viruses that can be passed on by the vector

within a few minutes of starting to feed on the plant). Deltamethrin's mode of action is thought to be mainly central in action, or at least originates in higher nerve centers of the brain. Death of insects seems to be due to irreversible damage to the nervous system occurring when poisoning lasts more than a few hours. Deltamethrin poisoning occurs through cuticular penetration or oral uptake. The susceptibility of insects is dependent on a variety of factors and can vary, as with many insecticides, according to the environmental conditions. Flies are most susceptible to pyrethroid poisoning shortly before dawn. The LD50 drops by the factor of 2 as compared to full daylight activity. Many pyrethroids are not very active against cattle ticks, but some alpha cyano compounds (of which deltamethrin is one) have higher activity than organophosphates or amidines, the former standard compounds for this purpose. Deltamethrin has very good residual activity for outdoor uses (field crops, cattle dip, tsetse) and for indoor uses (mosquitoes, stable flies, horseflies, fleas, cockroaches, stored product insects). Deltamethrin has very broad spectrum control. It is considered the most powerful of the synthetic pyrethroids. It is up to three orders more active than some pyrethroids.

Mode of Action:

Target Organisms

- Deltamethrin is effective against insects via ingestion and direct contact.
- Pyrethroids, in general, interfere with normal production and conduction of nerve signals in the nervous system. Pyrethroids act on nerve membranes by delaying the closing of the activation gate for the sodium ion channel.
- Researchers distinguish between two classes of pyrethroids based on electrophysiological studies with nerves and symptoms of toxicity.

Type II pyrethroids, including deltamethrin, have an α -cyano group that induces “long-lasting” inhibition of the sodium channel activation gate. This results in prolonged permeability of the nerve to sodium and produces a series of repetitive nerve signals in sensory organs, sensory nerves, and muscles.

- Researchers observed that deltamethrin and other Type II pyrethroids may also affect ion channels in the nervous system other than sodium channels, possibly due to their phosphorylation state.

Non-target Organisms

- The mechanism of action of pyrethroids, including deltamethrin, is the same for target and non-target organisms.

Usage

Deltamethrin products are among some of the most popular and widely used insecticides in the world[citation needed] and have become very popular with pest control operators and individuals in the United States in the past five years. This material is a member of one of the safest classes of pesticides: synthetic pyrethroids. While mammalian exposure to deltamethrin is

classified as safe, this pesticide is highly toxic to aquatic life, particularly fish, and therefore must be used with extreme caution around water.

There are many uses for deltamethrin, ranging from agricultural uses to home pest control. Deltamethrin has been instrumental in preventing the spread of diseases carried by tick-infested prairie dogs, rodents and other burrowing animals. It is helpful in eliminating and preventing a wide variety of household pests, especially spiders, fleas, ticks, carpenter ants, carpenter bees, cockroaches and bedbugs.

Deltamethrin plays a key role in controlling malaria vectors, and is used in the manufacture of long-lasting insecticidal mosquito nets. It is used as one of a battery of pyrethroid insecticides in control of malarial vectors, particularly *Anopheles gambiae*, and whilst being the most employed

pyrethroid insecticide, can be used in conjunction with, or as an alternative to, permethrin, cypermethrin and other organophosphate-based insecticides, such as DDT, malathion and fenthion. Resistance to deltamethrin (and its counterparts) is now extremely widespread and threatens the success of worldwide vector control programmes.

Recently, in South Africa, residues of deltamethrin were found in breast milk, together with DDT, in an area that used DDT treatment for malaria control, as well as pyrethroids in small-scale agriculture.

Resistance to deltamethrin

Resistance has been characterized in several important vectors of malaria, including *Anopheles gambiae*. Methods of resistance include thickening of the cuticle of the vector to facilitate less permeation of the insecticide, metabolic resistance via over expression of metabolizing P450 mono-

oxygenases and glutathione-S-transferases, and the kdr sodium channel mutations which render the action of insecticides ineffectual, even when co-administered with piperonyl butoxide. Characterization of the different forms of resistance has become a top priority in groups studying tropical medicine due to the high mortality of those who reside in endemic areas (Muller, Pie, et al. (2008).

TOXICOLOGICAL ASPECTS

Toxic to humans:

Pesticide products containing synthetic pyrethroids are often described by pest control operators and community mosquito management bureaus as “safe as chrysanthemum flowers.” While pyrethroids are a synthetic version of an extract from the chrysanthemum plant, they were chemically engineered to be more toxic with longer breakdown times, and are often formulated with synergists, increasing potency and compromising the human body’s ability to detoxify the pesticide.

Deltamethrin belongs to a class of chemicals called pyrethroids, described by the Agency for Toxic Substances and Disease Registry (U.S. Department of Health and Human Services) as “manufactured chemicals that are very similar in structure to the [naturally occurring] pyrethrins, but are often more toxic to insects, as well as to mammals, and last longer in the environment” (ATSDR Public Health Statement, pg. 1). Deltamethrin is not a natural

product. It is one of many synthetic pyrethroids developed for use as an insecticide based on the chemistry of the pyrethrum flowers.

The Tests:

Acute toxicity:

Deltamethrin produces typical type II motor symptoms in mammals. Type II symptoms include a writhing syndrome in rodents, as well as copious salivation. The acute oral LD₅₀ in male rats ranged from 128 mg/kg to greater than 5,000 mg/kg depending on the carrier and conditions of the study (2, 10); the LD₅₀ for female rats was 52 mg/kg and other published values range from 31 to 139 mg/kg. Values ranging from 21 to 34 mg/kg were obtained for mice; while dogs had a reported LD₅₀ of 300 mg/kg. The intravenous LD₅₀ in rats and dogs was 2 to 2.6 mg/kg, and the dermal LD₅₀ was greater than 2,940 mg/kg. The acute percutaneous LD₅₀ for rats was reported to be greater than 2,000 mg/kg; greater than 10,000 mg/kg for quail; and greater than 4,640 mg/kg for ducks. The acute dermal LD₅₀ for rabbits was greater than 2,000

mg/kg. No skin irritation and slight eye irritation were reported. Another study indicated skin irritation in rats and guinea pigs.

The signs of poisoning produced in rats by deltamethrin are not the same as those produced by other pyrethroids. Especially characteristic are rolling convulsions. The site of action is considered to be central with little or none of the peripheral component demonstrated for other pyrethroids. The sequence of signs is clearly defined, progressing from chewing, salivation, and pawing to rolling convulsions, tonic seizures, and death. Blood pressure begins to drop promptly, but slowly; it tends to normalize about the time choreoathetosis (abnormal movements of the body of a combined choreic and athetoid pattern) begins but falls precipitously prior to death. The early signs, including choreoathetosis, are reversible, but rats that exhibit a tonic seizure and shock almost always die promptly.

Acute exposure effects in humans include the following: ataxia, convulsions leading to muscle fibrillation and paralysis, dermatitis, edema, diarrhea, dyspnea, headache, hepatic microsomal enzyme induction,

irritability, peripheral vascular collapse, rhinorrhea, serum alkaline phosphatase elevation, tinnitus, tremors, vomiting and death due to respiratory failure. Allergic reactions have included the following effects: anaphylaxis, bronchospasm, eosinophilia, fever, hypersensitivity pneumonia, pallor, pollinosis, sweating, sudden swelling of the face, eyelids, lips and mucous membranes, and tachycardia.

Studies have shown many cases of dermal deltamethrin poisoning after agricultural use with inadequate handling precautions, and many cases of accidental or suicidal poisoning by the oral route at doses estimated to be 2-250 mg/kg. Oral ingestion caused epigastric pain, nausea, vomiting and coarse muscular fasciculations. With doses of 100-250 mg/kg, coma was caused within 15-20 minutes.

Chronic toxicity:

Suspected chronic exposure effects in humans include the following: choreoathetosis, hypotension, prenatal damage and shock. Workers exposed to deltamethrin during its manufacture over 7-8 years experienced transient cutaneous and mucous membrane irritation.

A DPR review of the toxicology database on the effects of deltamethrin has identified potential adverse responses. This compound has been associated with clinical signs characteristic of autonomic nervous system dysfunction in humans and in laboratory animals. The human data were from reports from accidental poisonings and attempted suicides. The laboratory animal data were primarily from studies submitted in support of product registration under Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) guidelines.

Acute toxicity: Laboratory animal studies have demonstrated toxicity in response to acute exposure to technical grade deltamethrin, as well as, to acute exposure to product formulations containing deltamethrin as the active ingredient. The primary toxic signs were characteristic of agents that disrupt the autonomic nervous system and many of these signs were consistent with the neurotoxicity associated with pyrethroids. These signs included excessive salivation, decreased activity, labored breathing, gasping, impaired limb function, ataxia, loss of righting reflex, tremors, convulsions, and lethality. Furthermore, signs of autonomic nervous system dysfunction (e.g., liquid feces, vomiting, and tremors) have been reported in studies designed to examine the effects of multiple exposures to deltamethrin. No evidence of delayed neurotoxicity was reported in domestic hens, however, the study was

rejected by DPR as a FIFRA guideline study because no repeat dosing was conducted.

Sub-chronic toxicity: In addition to providing information that is useful in dose selection for chronic toxicity, reproductive, and oncogenicity studies, sub-chronic toxicology studies are designed to examine the adverse effects resulting from repeated exposure of a portion of the average life span of an experimental animal (Mosberg and Hays, 1989). When extrapolated to human exposure scenarios, sub-chronic toxicity studies can aid in the assessment of potential risk to agricultural workers involved with seasonal exposure to a pesticide. Furthermore, sub-chronic studies may aid in the assessment of potential risk to humans when home use is expected to be seasonal.

Genotoxicity: Deltamethrin was tested for genotoxic potential in vitro using *Schizosaccharomyces pombe* to test for gene mutation, Chinese Hamster Ovary (CHO) cells to test for chromosome aberrations and rat primary hepatocyte to test for unscheduled DNA synthesis (UDS). In these test systems, no genotoxic potential was reported. In other recently published studies appearing in the open literature, positive genotoxicity was reported for deltamethrin technical (or formulations with deltamethrin as the active ingredient) in both in vivo (e.g., chromosome aberrations and micronucleus test) and in vitro (sister-chromatid exchange in human lymphocytes) test systems.

Developmental Toxicity: In the studies submitted in support of registration, no significant developmental toxicity was reported in rats (Schardein, 1990a) or in rabbits (Schardein, 1990b). Effects in rats have been reported, however,

in the open literature (Abd El-Khalik, et. al, 1993). These researchers reported that a 5% deltamethrin formulation results in dose-dependent early embryonic death, retardation of fetal growth, hypoplasia of the lungs, and dilation of the renal pelvis.

Reproductive Toxicity: A rat reproduction study submitted by the registrant indicated minor effects (reduced pup weight) but was considered unacceptable to DPR based on FIFRA guidelines (Wrenn, 1980). Microscopic examinations were limited to F3b weanlings and no parental microscopic data were collected. After a review of the open literature, a study was found that indicated a number of reproductive effects in rats treated with deltamethrin.

Deltamethrin significantly decreased the weight of testes, seminal vesicle, and prostate glands. Significant decreases were also noted in sperm cell concentrations, percentage of live cells and sperm motility. Furthermore,

plasma testosterone concentration was significantly decreased and the pregnancy rate was depressed (Abd El-Aziz, 1994).

Fate in Humans and Animals

Pyrethroid-poisoned mice and rats die during seizures within one or two hours after treatment. Metabolites of the cyano substituent are eliminated more slowly, and tissue levels remain relatively high, especially in the skin and stomach. Deltamethrin at an oral dosage of 50 mg/kg produces a marked increase of cGMP in the brain of rats. Metabolism of deltamethrin in rats involves rapid ester cleavage and hydroxylation. Deltamethrin has a half-life in the rat brain of 1 to 2 days, but it is more persistent in body fat, with a half-life of 5 days.

In mammals, the point of death from deltamethrin poisoning is sharply defined by respiratory or cardiac failure. Rats and dogs given oral doses of 10

mg/kg/day for 13 weeks exhibited some motor symptoms. The dogs exhibited diarrhea and vomiting. In another study, rats given 15 daily oral doses of 10 mg/kg showed severe motor symptoms.

The reports:

Pyrethrins are natural extracts derived from flowers of chrysanthemum cinerarifolium and C. coccineum. Pyrethroids are synthetic analogues of these natural abstracts. Pyrethroids are widely used as insecticides and are also used in the topical treatment of scabies and lice. A case of Deltamethrin poisoning presenting with status epilepticus is described as below.

Case Report

- A 24-year-old female was admitted to our intensive care unit (ICU) with repeated episodes of seizures following deliberate ingestion of an unknown quantity of the anti-lice medication comprising of 1.2% Deltamethrin. On

arrival in the emergency department, the patient was given a bolus dose of Lorazepam, and soon intubated, and then a loading dose of phenytoin was commenced. Gastric lavage was not performed as patient had repeated convulsions. On examination, she was deeply comatose, and was getting generalized tonic clonic seizures. Her pulse rate was 130/min regular, and her blood pressure was 136/76 mmHg. Her pupils were equal (2 mm in size) bilaterally briskly reacting to light. Other systemic examinations were unremarkable. An arterial blood gas showed mild metabolic acidosis. Hemogram, renal and liver function tests, electrocardiogram, and chest X-ray were normal. After repeat doses of Lorazepam and phenytoin, the patient was put on mechanical ventilatory support. A therapeutic coma was induced using a combination of Midazolam and Thiopentone sodium, as the patient continued to have convulsions. In the next 36 hours, Thiopentone and Midazolam were gradually tapered off after electroencephalography documentation of complete suppression of burst activity. She was gradually weaned off the ventilator after 72 hours. She was discharged from the hospital on Day 5 after psychiatry consultation.

- The Anti-poison Centre in Marseille received 230 000 telephone calls concerning intoxications during 1973–86, of which 6700 involved agricultural products and 89 (0.04%) referred to deltamethrin (Jouglaud & Boulet, 1987).

Eighty-four cases of poisoning with deltamethrin-based products were recorded in the 5 years 1988–92 at the Anti-poison Centre of Paris. In 1992, the proportion of accidents due to deltamethrin accounted for 0.02% of all poisoning cases recorded at this Centre. Sixty-three of the cases involved deltamethrin in petroleum solvents, and eight involved deltamethrin and an organophosphorous compound in petroleum solvents (Chataigner & Garnier, 1995).

- A review of 573 cases of acute pyrethroid poisoning reported in the Chinese medical literature during 1983–88 showed that 325 cases were due to a 2.5% deltamethrin emulsifiable concentrate. Of these, 153 were due to occupational exposure and 167 were accidental. Two patients died after convulsions; all the others recovered, after symptomatic and supportive treatment, within 1–6 days (He et al., 1989).

The symptoms vary according to the circumstances and route of exposure. Ingestion is the most frequent route in cases of accidental intoxication and suicide. Adverse effects occur mainly after cutaneous and respiratory exposure. After dermal exposure, symptoms such as tingling or itching of the face develop, similar in nature and duration to the paraesthesia described in plant personnel. They are always transient and are usually not associated with objective cutaneous signs. Chataigner & Garnier (1995) observed no systemic signs after cutaneous exposure. Splashing of a formulation containing deltamethrin and petroleum distillates into the eye induced pain and conjunctival hyperemia. Recovery was complete within 4 days. Inhalation of deltamethrin-based formulations was followed by nausea, vomiting, headache, and irritation of the upper respiratory tract resulting in rhinorrhea, cough, and dyspnea. These signs of irritation were more intense with aerosols than with vapours. Only the paraesthesia could clearly be attributed to exposure to deltamethrin, whereas nausea, headache, and dizziness are known to be induced by organic solvents. The signs reported by He et al. (1988, 1989) were therefore not typical of deltamethrin poisoning but may have been due to the emulsifiable concentrate as a whole.

To summarize:

On the basis of their chemical structure, pyrethroids are divided into two groups: Type I pyrethroids are devoid of a cyano moiety at the alpha-position of the basic cyclopropane carboxylic ester structure (e.g., Allethrin) while Type II pyrethroids have an alpha-cyano moiety (i.e., Fenvalerate and Deltamethrin). Pyrethroids produce prolonged opening of membrane sodium channels resulting in membrane depolarization, repetitive discharges, and synaptic disturbances leading to hyperexcitatory symptoms of poisoning. Only low pyrethroid concentrations are necessary to modify sensory neurone function. Type II pyrethroids also decrease chloride currents through voltage-dependent chloride channels and this action probably contributes the most to the features of poisoning with Type II pyrethroids. At relatively high concentrations, pyrethroids can also act on gamma-aminobutyric acid-gated chloride channels, which may be responsible for the seizures seen with severe Type II poisoning. There are suggestions that voltage-sensitive calcium (Ca^{2+}) channels (VSCC) may also be important targets of pyrethroid action. However, currently the data available neither

supports nor refutes conclusively the hypothesis that effects on VSCC are important to the acute neurotoxicity of pyrethroids. Type I pyrethroids cause a Type I poisoning syndrome characterized by reflex hyperexcitability and fine tremor, whereas Type II pyrethroids produce salivation, hyperexcitability, choreoathetosis, and seizures. Both produce potent sympathetic activation. Local contamination of the skin produces paresthesias - the face being most commonly affected. The paresthesiae are exacerbated by sensory stimulation such as heat, sunlight, scratching, sweating, or the application of water. Ingestion produces gastric irritation. The possibility that they also induce hypersensitivity reaction, which may be fatal when the respiratory tract is involved, has been debated for many years.

Because there is no antidote for pyrethrin and pyrethroid poisoning, treatment is symptomatic and supportive. Pyrethroid paresthesias are treated by decontamination of the skin. Seizures due to systemic poisoning are sometimes difficult to control with anticonvulsants. Seizures are a known manifestation of pyrethroid poisoning.

OCCUPATIONAL HAZARDS

Synthetic analogs of the pyrethrins, extracts from the ornamental *Chrysanthemum cinerariaefolium*, have been developed to circumvent the rapid photodegradation problem encountered with the insecticidal natural pyrethrins. The pyrethroids are widely used in field pest control and household use and as veterinary and human pediculicides and are among the most potent insecticides known (Smith and Stratton, 1986). The widespread use of these pesticides consequently leads to the exposure of manufacturing workers, field applicators, the ecosystem, and finally the public to the possible toxic effects of these pesticides.

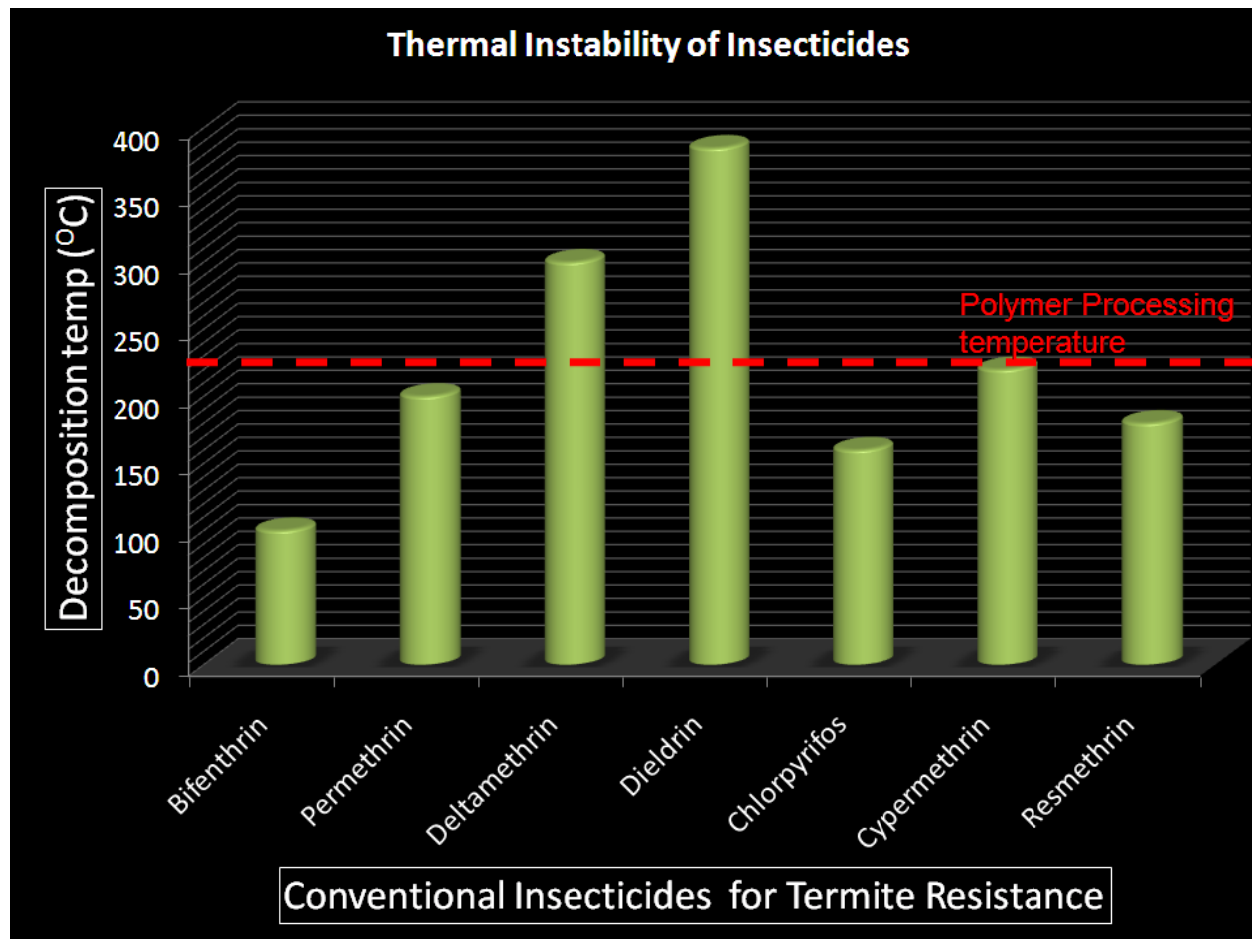
Excessive exposure to deltamethrin can cause nausea, headache, muscle weakness, excessive salivation, shortness of breath, and seizures. Worker exposure to the chemical can be monitored by measurement of the urinary metabolites, while severe overdosage may be confirmed

by quantitation of permethrin in serum or blood plasma. These harmful insecticide additives volatilize at polymer processing temperatures and release extremely toxic fumes. These toxic fumes are many times more lethal than the original. This poses fatal hazards to workers handling such products at the shop floor. As (air) temperature increases, vapour hazards will increase. The vapours from many pesticides increase three to four times for each 10° C increase in temperature. This is one reason why pesticide should be stayed away from sunlight and why it is typically recommended that pesticides not be applied when air temperatures are above 30° C. Extrusion temperatures are as high as 100-300° C.

Toxic Vapours!!!

Deltamethrin has a low thermal decomposition temperature of 320°C. When such chemicals are used in extrusion for manufacturing of wire and cables, pipelines etc, they decompose during the process only. The decomposition leads to the formation of toxic fumes. These fumes are highly toxic especially

for the personnel working on the shop floor. The graph below shows the thermal instability of various pesticides currently used worldwide.



The thermal degradation temperature of Deltamethrin is 320°C which is in the same range as the process of extrusion. Elevated as well as localized temperatures in an extruder can go as high as 400°C and even beyond. This can result in serious problems for workers which will be handling the product. Moreover the temperatures are quite high near the tube exits of the

extruder which could be a major source of toxic fumes. In case of insufficient ventilation which is normally the case in case of compact arrangements near the exits, these fumes can accumulate thus increasing the toxicity in general 30° C. Extrusion temperatures are as high as 100-400° C.

Symptoms, reports and data:

Physical signs of deltamethrin poisoning can include dermatitis after skin contact; exposure to sunlight can make it worse. Severe swelling of the face including lips and eyelids can occur. Symptoms and consequences of poisoning include: **sweating, fever, anxiety and rapid heartbeat.** If **swallowed, symptoms are likely to include feeling sick, vomiting, diarrhea, twitching of arms and legs, and convulsions if poisoning is severe.**

- A health survey of 199 workers who repacked pyrethroid insecticides into boxes by hand indicated that about two-thirds of the workers had a burning sensation and tightness and numbness on the face, while one-third had sniffs

and sneezes. Abnormal sensations in the face, dizziness, tiredness and red rashes on the skin were more common in summer than in winter.

- Cold burning and numbness of the skin occurred to two-thirds of humans in a Chinese factory exposed to about 5-12 mg deltamethrin per cubic meter of air. The other third suffered from sneezing and eye-watering. In addition, headache, heartburn and skin spots were reported, and these symptoms were dependent on the time of the year.

- A trial lasting about five weeks involved three baggers, two mixers, two helpers, and nine spraymen. Six of the nine spraymen, all baggers, and one of two mixers complained of "heat around the eyes," "heat in the face," or "heat in the face and upper shoulders" plus "burning of the eyes" and tiredness. Complaints lasted until evening of each work day. There were no positive clinical signs of exposure to deltamethrin by any of the workers.

- In a plant at Romainville, France, where deltamethrin and many other pyrethroids were produced, 185 consultations for subjective cutaneous sensations were recorded between 1982 and 1990. Some persons were reported to have presented with this symptom at least 10 times after their first exposure. Up to 90% of the workers in the workshops presented once with acute subjective cutaneous sensations. The onset appeared to be more frequent in summer. The clinical description at this plant was similar to that at the previous one. Thus, the predominant sites were the jaws and periorbital area. In one man, paraesthesia occurred on the external genital organs. No tingling or burning sensations on the hands or forearms were reported. In most cases, non-concomitant cutaneous lesions were reported. Some objective signs were sometimes observed at the same time as the subjective sensations, either as slight erythema followed by discrete desquamation or as slight, transient dermal oedema. It is not clear that these effects were due to deltamethrin, as the organic solvent in which it was dissolved may have been responsible for some local symptoms. Transient tingling of the mouth and some gustative effects were also reported (Boggio, 1994).

A delay generally occurred before the onset of symptoms, which varied from 30 min after the beginning of exposure to 3–4 h after cessation. The sensations were usually increased by sensory stimulation (heat, sun, or water).

Acute toxicity values!!!

The average lethal dose (LD₅₀ in mice) of deltamethrin is 85 mg/kg.

According to **WHO Recommended Classification of Pesticides by Hazard**, this compound falls in **Class 2** meaning a “**hazardous substance**”.

R- and S-Phrases:

R-phrases (short for Risk Phrases) are defined in Annex III of European Union Directive 67/548/EEC: Nature of special risks attributed to dangerous substances and preparations. The list was consolidated and republished in Directive 2001/59/EC, where translations into other EU languages may be found. R-phrases for Deltamethrin are:

R25 Toxic if swallowed;

R36 Irritating to eyes;

R37 Irrigating to respiratory system;

R38 Irritating to skin;

R41 Risk of serious damage to eyes;

S-phrases are defined in Annex IV of European Union Directive 67/548/EEC: Safety advice concerning dangerous substances and preparations. The list was consolidated and republished in Directive 2001/59/EC, where translations into other EU languages may be found. S-phrases for Deltamethrin are:

S22 Do not breathe dust;

S26 In case of contact with eyes, rinse immediately with plenty of water and seek medical advice.

S28- After contact with skin, wash immediately with plenty of water;

S36/37/39 Wear suitable protective clothing, gloves and eye/face protection.

S45 In case of accident or if you feel unwell; seek medical advice immediately (show the label where possible).

TOXICITY IN ANIMALS

Deltamethrin is effective against insects via ingestion and direct contact. It interferes with the normal production and conduction of nerve signals in the nervous system by acting on nerve membranes by delaying the closing of the activation gate for the sodium ion channel. Deltamethrin has a α -cyano group that induces "long-lasting" inhibition of the sodium channel activation gate. This results in prolonged permeability of the nerve to sodium and produces a series of repetitive nerve signals in sensory organs, sensory nerves, and muscles.

Signs of Toxicity - Animals

Deltamethrin produces characteristic effects of choreoathetosis (sinuous writhing) and salivation, also known as CS Syndrome. In rats, this presents as pawing and burrowing behavior followed by salivation and tremors, progressing to choreoathetosis. Chronic seizures may occur in the final stage

in which case the rats exhibit motor incoordination, salivation, respiratory defects, spasms involving the limbs and tail, and chronic seizures when administered deltamethrin orally. Similarly, dogs exhibit vomiting, hyperexcitability, stiffness in the hind legs, and impaired body movement when 100 mg/kg of deltamethrin was orally administered. Guinea pigs exhibit an increase in signs of biting, scratching, and licking within 1 hour of a dermal application of deltamethrin. Symptoms from inhalation of deltamethrin in rats include grooming, hyperactivity, uncoordinated movements, and hypersensitivity to noise and touch.

Toxic to aquatic life: Deltamethrin is among the highly toxic pesticide for fish. The adverse effect depend on concentration and duration of exposure. Fish are more sensitive to deltamethrin than avian and mammalian. It is concluded that deltamethrin contamination is dangerous to the aquatic ecosystems, and this fact should be taken into consideration when this insecticide is used in agriculture or in the control of mosquito populations. Waste materials and containers should neither be disposed near water supplies, nor be emptied

into sewer drains. Biological methods could be used for controlling of mosquito and flies instead of deltamethrin in order to protect natural environment.

Bradbury and Coats (1989) also reviewed pyrethroid toxicology in mammals, birds, amphibians, and both terrestrial and aquatic invertebrates. Toxicity is highly dependent on stereochemical structure. Most products however, are mixtures of isomers. Due to their lipophilicity, pyrethroids have a high rate of gill absorption, which in turn would be a contributing factor in the sensitivity of the fish to aqueous pyrethroid exposures. Fish seem to be deficient in the enzyme system that hydrolyzes pyrethroids. The main reaction involved in the metabolism of deltamethrin, cypermethrin, or cyhalothrin in mice and rats is ester cleavage mainly due to the action of carboxyesterase. Metabolism in fish is largely oxidative (Demoute, 1989). Fish make intimate contact with the surrounding water through the gills. After short-term deltamethrin exposure, adult *Heteropneustes fossilis* (freshwater catfish) showed hypocalcemia and the researchers attribute this condition to the possible impairment of either

net electrolyte influx at the gill or renal function. Deltamethrin exposure also caused hypophosphatemia and was linked to the possible redistribution of electrolytes between intracellular or extracellular compartments and/or impairment of renal function. **Deltamethrin may disturb the calcium and phosphate homeostasis and may lead to an effect on the reproductive state of the fish (Srivastav et al., 1997).** Synthetic pyrethroids have been shown to be toxic for fish, aquatic arthropods, and honeybees in laboratory tests. Acute toxicity data for deltamethrin in fish have been summarized in a report of the World Health Organization (WHO, 1990) and classified as highly toxic to fish, being in the LC50 1.0 ppb. The potential hazard to fish is due to its heavy use in many aquatic larvicidal programs. Synergistic interactions between the active ingredient and other components of the formulation should be taken into consideration when evaluating toxicity.

Toxicity in bees:

This product is highly toxic to bees exposed to direct treatment or residues on crops or weeds. Hence it is advisable to not employ the use of this product or allow it to drift to crops or weeds on which bees are actively foraging.

Various Reports:

- Deltamethrin, a synthetic pyrethroid pesticide contaminating aquatic ecosystems as a pollutant, was investigated in the present study for toxic effects on embryos and larvae of common carp, *Cyprinus carpio* as a model. The control and five test experiments were repeated five times. The water temperature in the experimental units was kept at 24 ± 1 °C. The number of dead embryos significantly increased in response to deltamethrin concentrations 0.005, 0.05, 0.5, 5, 25, and 50 $\mu\text{g L}^{-1}$ ($p < 0.05$ for each case). Dose-response decreases in hatching success were recorded as 75.2, 64.6, 47.4,

26.0, 14.4, and 9.0%, respectively. The lowest concentration of deltamethrin ($0.005 \mu\text{g L}^{-1}$) produced a significantly decrease in number of dead larvae compared to control group ($p < 0.05$). With increasing deltamethrin concentrations, the larvae exposed duration 1–48 h significantly increased the number of dead larvae ($p < 0.05$ for each cases). The 48 h LC₅₀ values (with 95% confidence limits) of deltamethrin for common carp embryos and larvae were estimated as 0.213 (0.103–0.404) and 0.074 (0.011–0.260) $\mu\text{g L}^{-1}$, respectively. **The results provide evidence that deltamethrin pollution may have an adverse effect on the reproduction and development of carp, which should be considered when this chemical is used in agricultural areas near aquatic ecosystems.**

- Pyrethroids have been reported to be extremely toxic to fish and some beneficial aquatic arthropods, for example, lobster and shrimp (Bradbury and Coats, 1989; URL 1; Srivastav et al., 1997).

Deltamethrin is a highly toxic synthetic pyrethroid pesticide widely used in agriculture. Here special attention is drawn to its heavy use in mosquito

control programs, which necessitates in-depth subchronic and chronic toxicity tests to fish species and to non target species to be undertaken. In addition, potential risk from deltamethrin metabolites should be investigated to get a more complete picture in terms of toxicity. The low toxicity of deltamethrin to mammals may be misleading at this point in terms of ecotoxicology and lead to extrapolation problems to aquatic species. Delistraty (2000), in the study of examining relationships among physicochemical properties and acute toxicity endpoints of 231 chemicals in rats and trout, concluded that trout aquatic LC50 was predicted from rat inhalation LC50 with moderate success. Therefore, such data are useful in ecological risk assessment but there are limitations and uncertainties. Further work with toxicity testing methods directly on fish will be very useful in assessing possible ecological risk assessment of these pesticides. To overcome discrepancies and potential synergistic effects from the components of the pyrethroid formulations, toxicity tests with formulations must be included together with active ingredient tests. Using only the pyrethroid active ingredient in the tests is insufficient.

- Pest management practices may be contributing to a decline in wild bee populations in or near canola (*Brassica napus* L.) agro ecosystems. The objective of the study conducted by the Department of Environmental Biology, University of Guelph, Guelph, ON, Canada was to investigate the direct contact toxicity of five technical grade insecticides--imidacloprid, clothianidin, deltamethrin, spinosad, and novaluron--currently used, or with potential for use in canola integrated pest management on bees that may forage in canola: common eastern bumble bees [*Bombus impatiens* (Cresson); hereafter bumble bees], alfalfa leafcutting bees [*Megachile rotundata* (F.)], and *Osmia lignaria* Cresson. Deltamethrin was found to be toxic to these species. Bumble bees were generally more tolerant to the direct contact applications > *O. lignaria* > leafcutting bees. However, differences in relative toxicities between the three species were not consistent, **deltamethrin was 53 and 68x more toxic to leafcutting bees than to bumble bees and *O. lignaria*, respectively.** Laboratory assessment of direct contact toxicity, although useful, is only one measure of potential impact, and mortality under field conditions may differ greatly depending on management practices. Research conducted using only honey bees as the indicator species may not adequately

reflect the risk posed by insecticides to wild bees because of their unique biology and differential susceptibility. Research programs focused on determining non target impact on pollinators should be expanded to include not only the honey bee but also wild bee species representative of the agricultural system under investigation.

- One theory proposed to explain the global declines in amphibian populations involves contaminant-induced immune alteration and subsequent increased susceptibility to infectious disease. The goal of the study conducted by the Toxicology Centre, University of Saskatchewan, Saskatoon, Saskatchewan, Canada was twofold, to **(1) study acute oral toxicity of deltamethrin (cyclopropanecarboxylic acid, 3-(2,2-dibromoethenyl)-2,2-dimethyl cyano(3-phenoxyphenyl)methyl ester) in tiger salamanders (*Ambystoma tigrinum*), and (2) evaluate whether the insecticide deltamethrin produces immunosuppression in these animals.** In the acute toxicity study, tiger salamanders receiving single doses of deltamethrin ranging from 1 to 35 mg/kg displayed intention tremors, hypersalivation,

ataxia, choreoathetosis (writhing), severe depression (immobility with minimal response to stimuli), and death. For acute effects, based on clinical signs, the median lethal dose (LD₅₀) and lowest observed adverse effect level (LOAEL) were estimated to be 5 to 10 mg/kg and 1 mg/kg, respectively. The LOAEL in animals dosed 3 times per week for 4 wk was 400 microg/kg/d. The endpoints for the immunotoxicity study included lymphoid organ mass and histopathology, hematological variables, and functional assays of phagocytosis, oxidative burst, and lymphoblastic transformation. Tiger salamanders in 4 treatment groups (0, 4, 40, or 400 microg/kg/d) were dosed with deltamethrin via the diet 3 times per week for 4 wk. **Deltamethrin exposure resulted in increased liver mass, packed cell volume, and total plasma protein concentration, but these effects were not dose dependent.** The relative mass of kidney and spleen, plasma albumin and globulin concentrations, and circulating leukocyte numbers were not affected by deltamethrin exposure, nor were phagocytosis, oxidative burst, and lymphoblastic transformation. This study shows that at moderate levels of exposure, deltamethrin may be neurotoxic to tiger salamanders.

CONCLUSION

The above reported results show how deltamethrin has been found to be toxic and therefore extensive study of the same is the need of the hour which would then probably reveal in alarming proportions whatever has been found so far to have affected humans and animals alike! What however we can do is stop or reduce the use of such compounds as ultimately it is us the end users who suffer the consequences and not the ones who supply it or market it and develop use of better non toxic and effective alternatives.

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