



e-ISSN: 2319-8753 | p-ISSN: 2320-6710

# IJRSET

International Journal of Innovative Research in  
**SCIENCE | ENGINEERING | TECHNOLOGY**

# INTERNATIONAL JOURNAL OF INNOVATIVE RESEARCH

IN SCIENCE | ENGINEERING | TECHNOLOGY

Volume 10, Issue 3, March 2021

**ISSN** INTERNATIONAL  
STANDARD  
SERIAL  
NUMBER  
INDIA

**Impact Factor: 7.512**

9940 572 462

6381 907 438

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# Fenvalerate: Properties, Mode of Action, and Safety Issues

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**ABSTRACT:** Synthetic pyrethroids are a group of insecticides. Two different types of synthetic pyrethroids have been recognized, based on different behavioural neuropathological, chemical, and biochemical profiles. The pyrethroid insecticides are derived from a naturally occurring compound; the pyrethrin. Present paper deals with the types, metabolism, and excretion, environmental fate of synthetic pyrethroid.

**KEYWORDS:** Pyrethroids, Synthetic Pyrethroid, Types, Metabolism, Environmental fate.

## I. INTRODUCTION

Man has polluted the environment in his quest for modernization and industrial growth. As the world's population grows, so does the demand for food and fibre. To meet this demand, our agricultural system has gone entirely chemicalized. High yielding crop varieties, which yield best under the influence of chemicals and pesticides, are influencing modern farming.

Pesticides are man-made synthetic chemicals that are used in large quantities to produce low-cost food. In India, 90000 MT of technique-grade high-level pesticides are used annually to protect plants from pest attacks and diseases. Insecticides, Fungicides, Weedicides, Herbicides, Nematocides, and Rodenticides are some of the pesticides used worldwide; insecticides account for 77 percent of the total pesticides used in various agricultural and animal husbandry practices. It's also used in almost every aspect of public health. Pesticides are commonly used to improve the quality and quantity of agricultural commodities. The widespread and extensive use of synthetic chemical pesticides has negative consequences, including odor, flavor, and lethal effects on a variety of non-target organisms in the aquatic environment, as well as direct and indirect effects on users.<sup>1,2</sup>

Synthetic pyrethroids are a group of insecticides, manufactured in 1970. At that time organochlorine insecticides like DDT exposure considered harmful. Now, this pyrethroid replaces the DDT as pyrethroid has insecticidal properties like their natural counterpart, pyrethrin, originate from chrysanthemum, but also enhanced in their environmental stability. A pyrethroid is extensively used in agriculture, industries, and public health sectors worldwide.<sup>3</sup>

A synthetic pyrethroid is a group of insecticides that owe their significance to the outstanding rapid knockdown action on insects and low mammalian toxicity due to their rapid metabolic conversion to non-basic produced<sup>4,5</sup>. They are potent neurotoxicants. A synthetic pyrethroid is esters of chrysanthemic acid halo substituted chrysanthemic acid 2-(4-chloophenyl)-3-methyl butyric acid and alcohol (eg. Allethrolene, 3-phenoxy benzyl alcohol).

## II. TYPES OF PYRETHROID

Two different types of synthetic pyrethroids have been recognized, based on different behavioral neuropathological, chemical, and biochemical profiles.

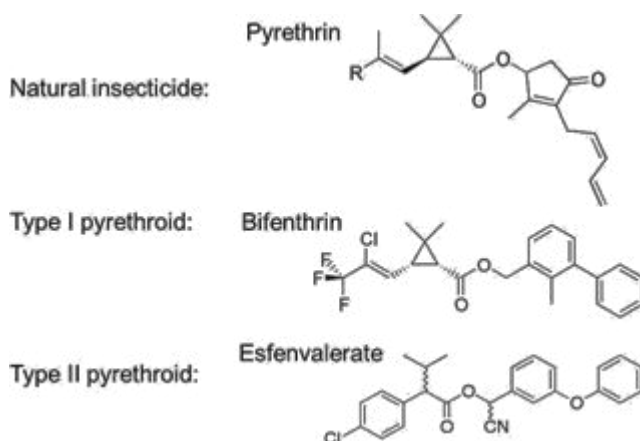
Type I have also known as class 1 or T for tremors and type II is also known as class 2 or cs for choreoathetosis-salivation.<sup>6,7</sup> In general these authorities agree that pyrethroid containing both a halogenated acid esterified with the -cyano-3-phenoxybenzyl alcohol such as fenvalerate, deltamethrin, and cypermethrin produce the type II poisoning syndrome and that pyrethroids lacking either or both of these moieties i.e. permethrin, resmethrin, cismethrin, allethrin, tetramethrin tends to produce the type 1 syndrome. Type 1 is characterized by sparring aggressive behavior, rapid onset of tremor in the extremities increased body temperatures, and whole-body tremors the type I syndrome is very similar to that produce by P, P- DDT.

Type II is characterized by pawing and burrowing behavior profuse salivation a decreased body temperature (due partially to evaporation of saliva) tremors progressing to choreoathetosis (i.e. a sinuous, writhing movement), muscular contractions and seizures, and death. Type-I pyrethroid does not contain  $\alpha$ -cyano group in their molecules. Type-II pyrethroid contains  $\alpha$ -cyano group at the carbon portion.

The pyrethroid insecticides are derived from a naturally occurring compound; the pyrethrin which is extracted from

the chrysanthemum genus.<sup>8</sup> Even though natural pyrethrins inherit insecticidal activity, they are also characteristically unstable to light. Hence, the structure of pyrethrin was altered to get the more stable product with required toxicological as well as insecticidal properties<sup>9</sup>.

The insecticidal nature of pyrethrins is resulting from keto alcoholic esters of chrysanthemic and pyrethroic acid. Pyrethrins' insecticidal properties are derived from chrysanthemic and pyrethroic acid keto alcoholic esters. These acids quickly penetrate many insects and pests and paralyze their nervous systems due to their strong lipophilic existence.<sup>10</sup> Generally, all pyrethroids have numerous related features like an acid and alcohol moiety, a central ester bond. Pyrethroid shows typically as stereoisomeric compound as they have acid moiety containing two chiral carbons<sup>11</sup>.



### III. METABOLISM AND EXCRETION

The most important metabolism degradation pathways for synthetic pyrethroid are oxidation on the phenoxy ring. All metabolic degradation products of synthetic pyrethroids pharmacologically inactive or inferior to the parent compound, implying that metabolic modifications lead to detoxication<sup>12</sup>.

Synthetic pyrethroid passes through the liver before they enter the systemic circulation and exert the inherent actions such as choline esterase inhibition many pesticides except their toxic action primarily by interference with mitochondria energy production<sup>13</sup>. Pyrethroid has an action at or near the sodium channel in the nerve resulting in greatly altered ionic currents and disrupted nerve function through membrane depolarization based on studies with insects, molluscan, amphibians, and small mammals there is a general agreement that the sodium channel in the nerve membrane is the major target site for all synthetic pyrethroid insecticides; that synthetic pyrethroid extends the temporary increase in sodium permeability of the nerve membrane during excitation resulting in spontaneous depolarization and repetitive release that persistent lead to muscular fasciculation acetylcholine depletion, and muscular weakness, that effects are enhanced at low temperature and that  $\alpha$ - cyano (Type II) pyrethroid are more potent neurotoxicants than non (Type I) pyrethroid.

The liver is the principal site of pyrethroid such as fenvalerate metabolism through hydrolysis by one or more hepatic microsomal esterase's inhibition of these enzymes results in enhanced toxicity<sup>14</sup>. Hydrolysis has also been demonstrated in plasma, kidney, stomach, and brain tissues except for the brain these tissues were relatively unimportant in the detoxification process<sup>12</sup>. Degenerative changes in liver cells cause increased permeability of cell membrane and lead to the release of transaminases in the circulation<sup>15,16</sup>.

**ENVIRONMENTAL FATE:** Pyrethroids can be degraded chemically, mainly by hydrolysis and photolysis, as well as by biodegradation. Hydrolysis rates rise sharply with increasing pH, as they do for many esters, with half-lives on the order of days to weeks at pH 9, weeks to months at pH 7, and reduced reactivity for most compounds at pH 5<sup>17</sup>.

The ester bond is hydrolyzed to create an aldehyde (e.g., 3-phenoxy benzaldehyde) and a carboxylic acid with a structure that depends entirely on the pyrethroid. A significant distinction between naturally occurring pyrethrins and synthetic pyrethroids is that the synthetic analogs are much more resistant to photolysis<sup>18</sup>.

The majority of the compounds have aqueous photolytic half-lives of weeks to months, with a few having half-lives of over a year. (e.g., 408 d for bifenthrin and 603 d for fenprothrin)<sup>19</sup>. In soils and sediments, all pyrethroids degrade, but the rates vary considerably depending on the pyrethroid structure.

## IV. EFFECT OF TOXICANT ON THE ENVIRONMENT

In water: Synthetic pyrethroids are characterized by low solubility and high adsorption coefficient. Upon entering the aquatic environment it quickly sorbs onto sediments, where it is degraded ultimately to carbon dioxide, as already described. They may pose a toxic risk because of spray drift, but with proper application, this can be minimized. Entering water via drift would also be quickly absorbed to sediments and other dissolved particulate matter in the water column. In one study when synthetic pyrethroid was directly applied to a water surface, 74% - 80% was found sorbed to sediments, where it remained for over 30 days<sup>20</sup>.

Synthetic pyrethroids are stable at low pH but unstable under alkaline conditions; t<sub>1/2</sub> increases with increasing application rates, and the primary degradation pathway in water (at neutral pH) is via photolysis. Photo-degradation usually occurs via ester cleavage, hydrolysis of the cyano group, or decarboxylation and usually yields 3-(4-chlorophenyl)-4-methyl-2-(3-phenoxyphenyl)-pantenenitrile (decarboxy fenvalerate) as a major product<sup>17</sup>.

In soil: pyrethroids are persistent in soil; they tightly bound to soil particles, as reflected by their high adsorption coefficient and low water solubility. It does not easily move laterally or to lower soil layers and generally remains localized within the region of application; thus risk of groundwater contamination is low<sup>19,20</sup>. Degradation rates from mineral soils are dependent on soil type, moisture, temperature, and microbial activity. Usually, the t<sub>1/2</sub> ranges between 2 and 18 day but period as long as 3 months have been observed<sup>20,21</sup>. When several types of soil were treated with fenvalerate at a rate of 1 mg/kg and stored at 25°C under aerobic conditions, the initial half-life period of fenvalerate ranged from 15 days to 3 months<sup>22</sup>.

In the air: Photo-degradation of pyrethroids is dependent on environmental temperature<sup>23</sup>. On exposure to sunlight, pyrethroids undergo rapid photolysis with approximate half-lives of 4 days and 10 days in summer and winter respectively. For E.g. Fenvalerate undergoes rapid photodegradation under the action of UV light with a half-life of 16-18 min<sup>24</sup>. After 60 minutes, the conversion was 90-95% and the major photoproduct is 2-(3-phenoxy phenyl)-3-(4-chlorophenyl)-4-methyl-pentanitrile.

**EXPOSURE LIMIT OF PESTICIDES:**

The determination of acceptable exposure limits for potentially dangerous substances is important for public health. The acceptable daily intake (ADI) has been suggested for pesticide residue in food defined as the daily exposure level of a pesticide residue which during the entire lifetime of man appears to be an appreciable risk based on all facts known at the time. The ADI is expressed in mg/kg/day the no observable effect level (NOEL) is the quantity of substance which when administered daily to a group of experimental animals demonstrate the absence of effects observed at a higher level and there is no significant difference in the test group of animals and control group of animals.

**EFFECT ON THE LIVING WORLD:** Pyrethroids are highly toxic to the nervous system. Pyrethroids are categorized into two groups based on the symptoms they cause and the presence of an alpha-cyano group in their structure. Type I pyrethroids, such as cismethrin, cause neurotoxicity primarily by interfering with sodium channel function in the nervous system, whereas type II pyrethroids, such as fenvalerate, may influence additional ion-channel targets such as chloride and calcium channels. Pyrethroids affect the release of acetylcholinesterase in the hippocampus region of the brain and can inhibit ATPases in addition to the normal mechanism of action. Furthermore, this pyrethroid can interfere with hormone functions, and its transformation products are more potent endocrine disruptors than the parent compound. Pyrethroids suppress the development of progesterone and estradiol in animals, resulting in estrogenic effects in females and anti-androgenic effects in males. The estrogen-dependent egg yolk precursor proteins vitellogenin and choriogenin and plasma estrogen have been shown to increase in fish exposed to pyrethroids<sup>25</sup>. Furthermore, pyrethroids also affect cell division by interrupt cell cycle processes, cause cell stress, and have immunosuppressive effects<sup>26</sup>.

Pyrethroids cause acute toxicity in aquatic organisms at concentrations far below 1 mg/L. Crustaceans and insects are the most vulnerable, with LC<sub>50</sub> values in the low ranges<sup>25</sup>. Significantly, Daphnia is not among the susceptible taxa, despite being the most commonly used species in aquatic toxicity research<sup>26</sup>.

Passive effects, such as food deprivation, can wipe out populations. Pyrethroids can also reduce the reproductive rate and success of aquatic organisms.

These compounds have been shown to bind to steroid receptors, with metabolites and transformation products being more effective than the parent compound. Pyrethroid disruption of the endocrine system has been shown to decrease the reproductive success of aquatic organisms.

Cypermethrin decreased fertilization success in fish<sup>27</sup> and impaired sperm motility<sup>28</sup>. In inland silverside, exposure to bifenthrin (500 ng/L) altered endocrine signaling and reduced the number of fertilized eggs per female<sup>29</sup>.

## V. CONCLUSION

Fenvalerate is an insecticide and veterinary drug that is commonly used to combat pests all over the world. The toxic effects of fenvalerate on animals and humans have received a lot of attention as its use has increased. As a result, research into the toxic effects and mechanism of fenvalerate is needed to protect non-target animals, such as humans, from damage.

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