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REVIEW

An overview of pyrethroid insecticides

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BACKGROUND: Pesticides are used to control various pests of agricultural crops worldwide. Despite their agricultural benefits, pesticides are often considered a serious threat to the environment because of their persistence. Pyrethroids are synthetic derivates of pyrethrins, which are natural organic insecticides procured from the flowers of *Chrysanthemum cinerariaefolium* and *C. coccineum*. Pyrethroids are classified into two groups—class I and class II—based on their toxicity and physical properties. These pyrethroids are now used in many synthetic insecticides and are highly specific against insects; they are generally used against mosquitoes. The prominent site of insecticidal action of pyrethroids is the voltage-sensitive sodium channels.

METHODS and RESULTS: Pyrethroids are found to be stable, and they persist in the environment for a long period. This article provides an overview of the different classes, structure, and insecticidal properties of pyrethroid. Furthermore, the toxicity of pyrethroids is also discussed with emphasis on bioremediation to alleviate pollution.

CONCLUSIONS: The article focuses on various microorganisms used in the degradation of pyrethroids, the molecular basis of degradation, and the role of carboxylesterase enzymes and genes in the detoxification of pyrethroid.

Keywords pyrethrin, carboxylesterase enzyme, mineralization, microbial degradation, toxicity

Introduction

During the last four decades, pyrethroids and pyrethrins have become a predominant class of insecticides used against pests in agricultural lands, gardens, and houses, accounting for 25% of the insecticide market, worldwide. Pyrethroids have been used as a substitute for highly toxic and recalcitrant organochlorine and organophosphorus pesticides (Katsuda, 1999). Presently, in the United States, 16 pyrethroids are registered for use in a variety of agricultural products (Bryant and Bite, 2003). Some pyrethroid insecticides are considered potential carcinogens to humans by the US Environmental Protection Agency (EPA) (Shukla et al., 2002; Tallur et al., 2008; Zhang et al., 2010). The present article focuses on the structure and chemical properties of pyrethroids, role of carboxylesterase enzymes in the degradation of pyrethroids, mechanism of insecticidal action, and molecular changes in the organisms during the process of pyrethroid degradation.

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Classes of pyrethroids and their uses

Natural pyrethrins, organic compounds with insecticidal activity, are extracted from *Chrysanthemums cinerariaefolium* (Casida and Quistad, 1998). Pyrethroids are synthetic analogs of naturally occurring pyrethrins, which were developed to enhance the insecticidal activity of pyrethrins by increasing their stability in the presence of light and resident time in the environment (Gosselin, 1984). Pyrethroids are derivatives of pyrethrin, which lack a cyano group. They are classified into two groups namely class I and class II based on their toxicity and physical properties. Class I pyrethroids have a basic structure of cyclopropane carboxylic ester. These include allethrin, bifenthrin, permethrin, phenothrin, resmethrin, tefluthrin, and tetramethrin. Class II pyrethroids have a cyano group and cause choreoathetosis and salivation. These include cyfluthrin, cyhalothrin, cypermethrin, deltamethrin, fenvalerate, fenpropathrin, flucythrinate, flumethrin, fluvalinate, and tralomethrin.

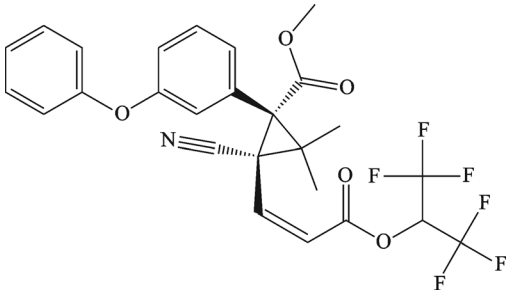
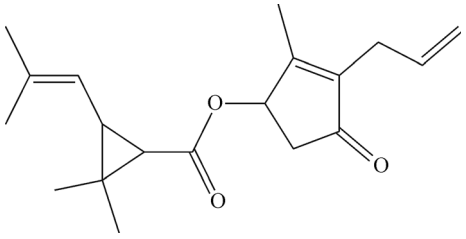
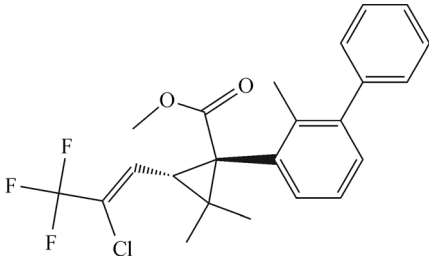
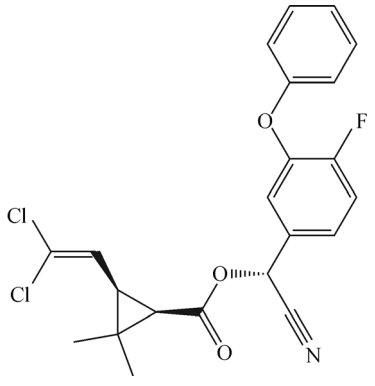
Pyrethroids are efficient against a wide range of insect pests belonging to the order Coleoptera, Hemiptera (Homoptera and Heteroptera), Diptera, Hymenoptera, Lepidoptera, Orthoptera, and Thysanoptera. They are also used as a household insecticide, mostly as a grain protectant. Pyre-

throids are employed in animal houses, fields, and green houses. They are also extensively used in veterinary medicine (Agency for Toxic Substances and Disease Registry, 2003) (Table 1). The efficacy of the pyrethroids and their selectivity for insect species depends on factors, such as shape, key structural features, including specific chirality and cis or trans stereochemistry across the cyclopropane ring, ester or nonester, and other physical properties. For instance, volatile compounds are effective against flying insect pests. Furthermore, chemical properties also affect the efficacy and selectivity of pyrethroids. For instance, polar compounds are efficient for knockdown of insects and high lipophilicity for reduced fish toxicity (Khambay, 2002).

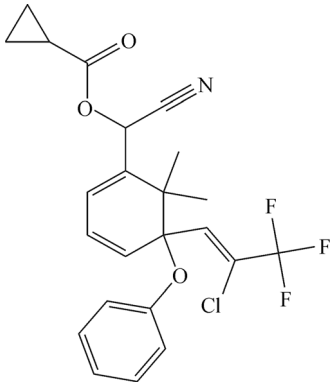
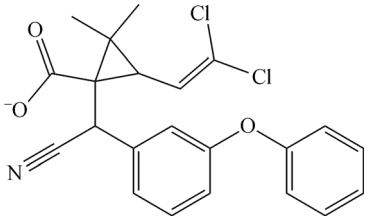
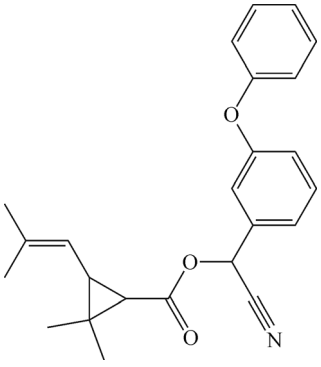
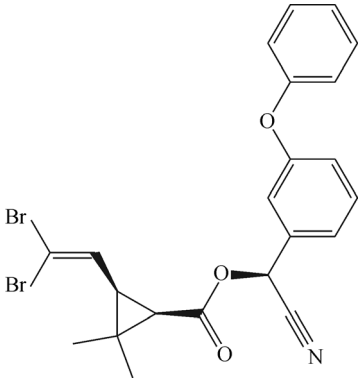
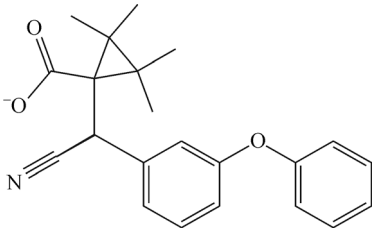
Chemical structure of pyrethroids

Pyrethroids consist of one to three asymmetric carbon atoms (chiral centers); therefore, they have high chirality. The chirality of pyrethroids might arise due to the acid moiety, alcohol moiety, or both (Kurihara and Mayamoto, 1998; Ali, 2004). All pyrethroids have at least four stereoisomers, each with different biological activities. Each pyrethroid has 2 or 4 diastereoisomer or enantiomer pairs. In general, isomer selectivity has been extensively observed in the insecticidal activity of a pyrethroid compound. Studies claim that the biodegradation of pyrethroids also exhibits significant isomer selectivity (Stok et al., 2004; Liu et al., 2005). Pyrethroids are

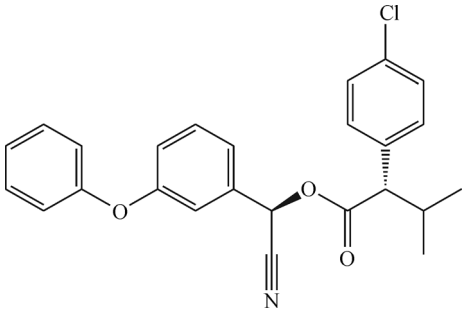
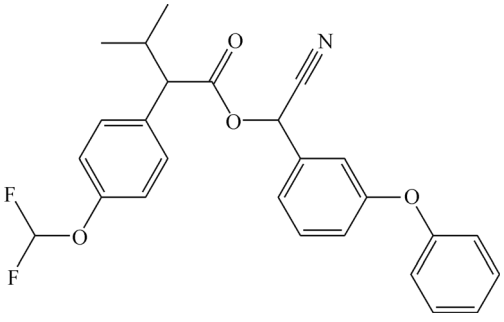
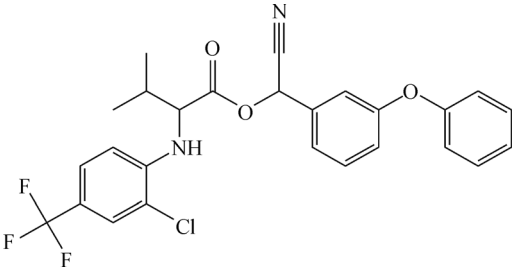
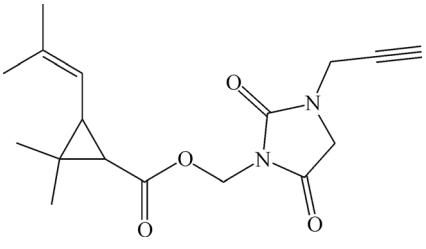
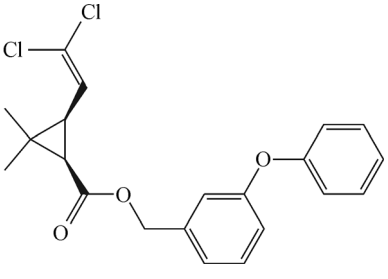
Table 1 List of pyrethroids usually detected in environmental samples

Pyrethroid (acronym)	Molecular structure	Insects
Acrinathrin (ester)		Codling moth, oriental fruit moth, leafhoppers, Red Spider Mite, two-spotted mite, and African red mite
Allethrin		Flies, mosquitoes, and ants
Bifenthrin		Beetles, weevil, houseflies, mosquitoes, lice, bedbugs, aphids, moths, cockroaches, and locust
Cyfluthrin		Aphids, cabbage stem flea beetle, houseflies, cockroaches, mosquitoes, and rape winter stem weevil

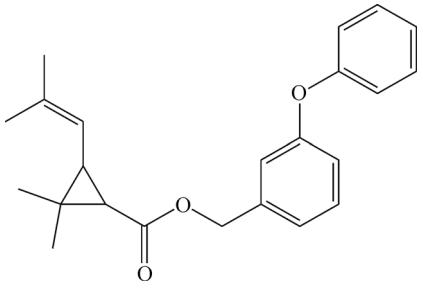
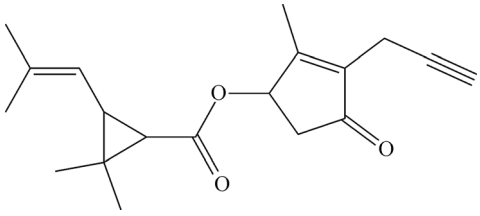
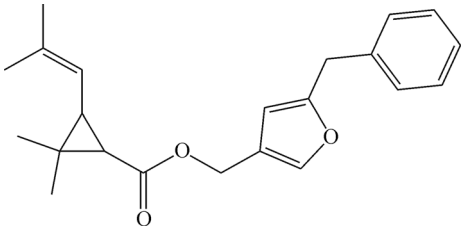
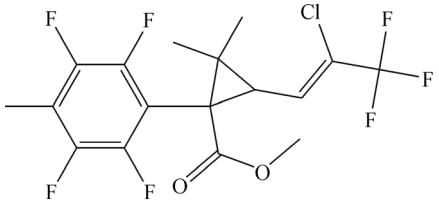
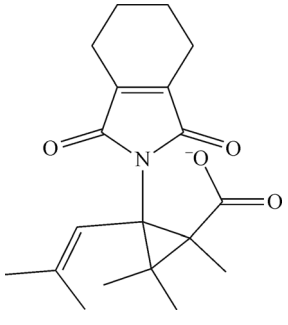
(*Continued*)

Pyrethroid (acronym)	Molecular structure	Insects
Cyhalothrin		Bedbugs, beetles, houseflies, ked, lice, mosquitoes, moths, and weevils
Cypermethrin		Cockroaches, mosquitoes, moths, and flies
Cyphenothrin		Flies, mosquitoes, and cockroaches. It is also used to control insects that attack wood and fabrics
Deltamethrin		Aphids, beetles, bollworm, budworm, caterpillars, cicadas, coding moths, totrix moths, weevils, whitefly, and winter moths
Fenpropathrin		Mites, aphids, beet armyworm, mealybug, potato leafhopper, moths, leafrollers, and lacebugs.

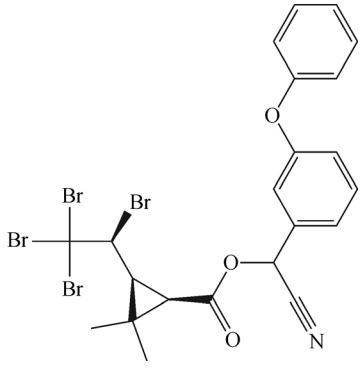
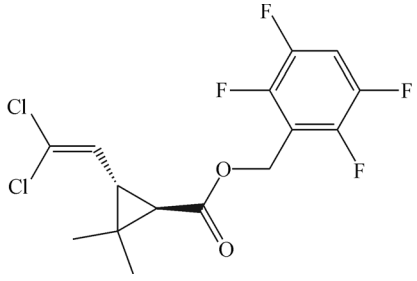
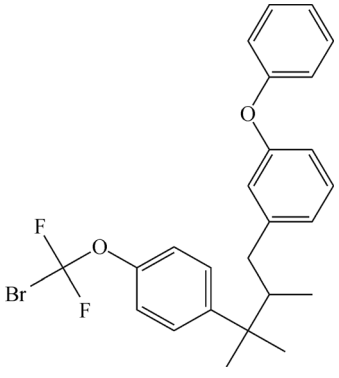
(*Continued*)

Pyrethroid (acronym)	Molecular structure	Insects
Fenvalerate		Beetles, cockroaches, flies, locusts, Mosquitoes, and moths
Flucythrinate		Bollworms, leafworms, sucking insects, whiteflies, and beetles
Fluvalinate		Aphids, leafhoppers, moths, spider mites, thrips, and white-flies
Imiprothrin		Roaches, waterbugs, ants, silverfish, crickets, and spiders
Permethrin		Ants, beetle, bollworm, bud-worm, fleas, flies, lice, moths, mosquitoes, termites, and weevils

(*Continued*)

Pyrethroid (acronym)	Molecular structure	Insects
Phenothrin		Flies, gnats, mosquitoes, cockroaches, and lice
Prallethrin		Ants, bees, carpet beetle, clover mite, and cockroaches
Resmethrin		Flies, mosquitoes, gnats, fleas, ticks, and black flies
Tefluthrin		White grub, southern corn leaf beetle, flea beetle, and chinch bug
Tetramethrin		Wasps, hornets, roaches, ants, fleas, and mosquitoes.

(*Continued*)

Pyrethroid (acronym)	Molecular structure	Insects
Tralomethrin		Ants and cockroaches
Transfluthrin		Mosquitoes and flies
Halfenprox (ether)		Mites

persistent compounds with high hydrophobicity (logK_{ow} 5.7–7.6) and low water solubility.

Most pyrethroids consist of cyclopropane carboxylic acid moieties (or an equivalent group) linked to aromatic alcohols through a central ester (or ether) bond. Alterations in this basic pyrethroid structure might increase the insecticidal potency or photostability; however, these changes might also result in insecticidal activity against non-target species (Yang et al., 1987; Valentine, 1990; Vijverberg and van den Bercken, 1990; Naumann, 1998; Soderlund et al., 2002). One such alteration is the addition of α-cyano group to the alcohol moiety, which significantly increases the insecticidal activity (Zerba, 1999). In addition to this, other alterations in the fundamental acid and alcohol moieties of pyrethroids have been introduced, including variations in the identity and position of halogenated and hydrophobic chemical groups, and variations in the stereochemical arrangement of these groups. In general, there are about 1000 different structures. Some of them are very different from the original structure of pyrethrins, including structures that lack dimethylcyclopropane ring.

Toxicity

To increase the effectiveness of insecticides, pyrethroids are formulated with some organic compounds, such as piperonyl butoxide, piperonyl sulfoxide, and sesamex, which act as synergists. These organic compounds enhance the pesticidal potency of pyrethrins and pyrethroids; however, they are not pesticides. The Agency for Toxic Substances and Disease Registry (2003) attested that the formulated commercial pyrethroids have a high proportion of highly toxic inert

ingredients. Pyrethroid insecticides are used widely for their high insecticidal activity and low level of acute toxicity in mammals, birds, and plants when compared with those of organochlorine and organophosphate insecticides. The acute effects of pyrethroids and pyrethroid formulations on non-target organisms are listed in Table 2. However, exposure to high levels of pyrethroids might affect the central nervous system, and induce reproductive toxicity due to endocrine disruption. Furthermore, they exhibit suppressive effects on the immune system affecting lymph node and spleen, and irritate the skin and eyes (Garey and Wolff, 1998; Laskowski, 2002). It has also been reported to cause acute toxicity on bees, fish, and aquatic invertebrates at a concentration of less than 1 $\mu g \cdot L^{-1}$ (Bloomquist, 1996; Gan et al., 2005; Suchismita and Anilava, 2008; Lutnicka et al., 1999 and Kumar et al., 2008).

When compared with that of other pesticides, pyrethroids are more specific toward target species. Organophosphates, organochlorines, and carbamates have a LD_{50} ratio (i.e., LD_{50} rat/LD_{50} insect) of < 100, while pyrethroids have a LD_{50} ratio of > 2000 (Katsuda, 1999). However, exposure to high levels of pyrethroids results in neurotoxicity toward non-target species, including mammals. Fishel (2005) reported that most pyrethroids are non-toxic to birds; however, they can be indirectly affected by pyrethroids through the food chain. Waterfowls, which feed mostly on aquatic invertebrates, are especially susceptible to pyrethroids. The signs of poisoning in non-target species are similar to those observed in insects (Glomot, 1982). These similarities in poisoning might be attributed to similar molecular mechanisms of toxicity across species, such as voltage sensitive sodium channels (VSSCs). The differences in pyrethroid efficiency between species have been attributed to differences in the intrinsic sensitivity of VSSCs to these compounds, body temperature, and metabolism (Ross et al., 2006). The detoxifying efficiency of pyrethroids depends on the route of administration. The metabolism and/or limited absorption play significant protective roles against pyrethroid neurotoxicity in mammals (Soderlund et al., 2002). A few studies have been carried out on pyrethroid-induced signs of poisoning in large mammalian species. The component involved in pyrethroid poisoning is similar across the mammalian species examined. Based on veterinary observations, acute, sub lethal exposure to pyrethroids induces restlessness and hyper-excitability followed by drunken gait (i.e., locomotor ataxia), mydriasis, diarrhea, and general depression. In some cases, motor incoordination, paresis, head bobbing, chewing, hypersalivation, and/or whole-body tremors (Legath et al., 1992).

Mechanism of insecticidal action

Pyrethroids can change the typical functioning of insect nerves by altering the kinetics of VSSCs, which mediate a transient increase in the permeability of sodium ions across the nerve membrane that underlies the action potential of nerves (Bloomquist, 1993a). The insecticidal activity of pyrethroids on the sodium channels of insects and other invertebrates, and the effects of interaction have been extensively investigated (Soderlund and Bloomquist, 1989;

Table 2 Acute effects of pyrethroids and pyrethroid formulations on non-target organisms Mueller-Beilsehmidt

Pyrethroid	mg pyrethroid/kg bodyweight of birds	Fish	Bees
Allethrin	2030	Toxic	-
s-Bioallethrin (Esbiol)	680	Highly toxic	-
Resmethrin	-	Toxic	Highly toxic
Bioresmethrin	-	Highly toxic	Highly toxic
Tetramethrin	> 1000	Toxic	toxic
Permethrin	> 13500	Highly toxic	Highly toxic
Fenvalerate	9932	Highly toxic	Toxic
d-Phenothrin	> 2500	Toxic	Toxic
Cypermethrin	-	Extremely toxic	Toxic
Esfenvalerate	-	Highly toxic	-
Bifenthrin	> 2150	Toxic	-
Fenpropathrin	1089	Toxic	-
Tefluthrin	4190	Highly toxic	-
Cyfluthrin	4450	Toxic	Toxic
Fluvalinate	> 5620	Toxic	Non-toxic
Tralomethrin	7716	Extremely toxic	Highly toxic
Deltamethrin	> 4640	Toxic	Highly toxic
Cyhalothrin	> 5000	Highly toxic	-
Kadethrin	-	Toxic	Toxic
Alphacypermethrin	-	Toxic	Toxic
Lambda- cyhalothrin	> 3950	Toxic	Toxic

Narahashi, 1992; Bloomquist, 1993a; Narahashi, 1996). The prominent site of insecticidal action of pyrethroids is VSSCs (Soderlund, 1997; Soderlund and Knipple, 1999). In the housefly, the super-kdr (enhanced knockdown resistance) and kdr (knockdown resistance) attributes confer resistance to all pyrethroids by reducing the sensitivity of its nervous system. Genetic linkage analyses summarized that these attributes are close to the principal *Vssc* gene of the housefly (designated *Vssc1*). In addition, kdr-like resistance in other insect species has been mapped to sodium channel genes that are orthologous to *Vssc1*. The DNA sequence analysis of *Vssc1* coding sequences from susceptible and resistant housefly strains identified a single mutation, which is common to all resistant strains. Furthermore, a second mutation is observed only in the highly-resistant super-kdr strains.

Insertion of these mutations into a susceptible *Vssc1* cDNA, followed by functional analysis of the susceptible and point mutations in sodium channels revealed that these mutations are adequate for the resistance caused by the kdr and super-kdr attributes. Therefore, the sodium channels encoded by the *Vssc1* gene of the housefly and by orthologous genes of other insect species must be the prominent sites of pyrethroid insecticidal action. Another study demonstrated that sodium channels containing the two mutations associated with the super-kdr resistance are insensitive to pyrethroid (Lee et al., 1999; Soderlun and Lee, 2001).

Pathways of pyrethroid metabolism

The metabolic pathway of most pyrethroids in animals is illustrated by the generalized pathway shown in Fig. 1. In the 1960s, Casida identified the pathways involved in the breakdown of pyrethroid and its metabolites. The initial biotransformation of the parent compound is by the action of either esterases at the central ester bond or cytochrome P450-dependent monooxygenases at one or more sites in the acid or alcohol moieties (Casida and Quistad, 1998). Although most of the oxidative pathways yielded hydroxyester metabolites, oxidative ester cleavage might be a minor pathway for some pyrethroids. The relative importance of initial hydrolytic or oxidative attack varies with compound and isomer for each pyrethroid. Following ester cleavage, primary alcohol moieties, such as 3-phenoxybenzyl alcohol, undergo additional oxidation, via the aldehyde, to the corresponding carboxylic acids, whereas the cyano-substituted alcohols lose the cyanide non-enzymatically to form the corresponding aldehyde (Maloney et al., 1988; Maloney et al., 1993; Soderlund et al., 2002). Some of these ester cleavage products might be hydroxylated. Subsequently, some of these hydroxylated ester metabolites might be hydrolyzed, thereby, producing hydroxylated ester cleavage products. Furthermore, the metabolism of these initial products of hydrolytic or oxidative attack involves conjugation with amino acids, sugars, sugar acids, or sulfate prior to excretion (Soderlund, 1997).

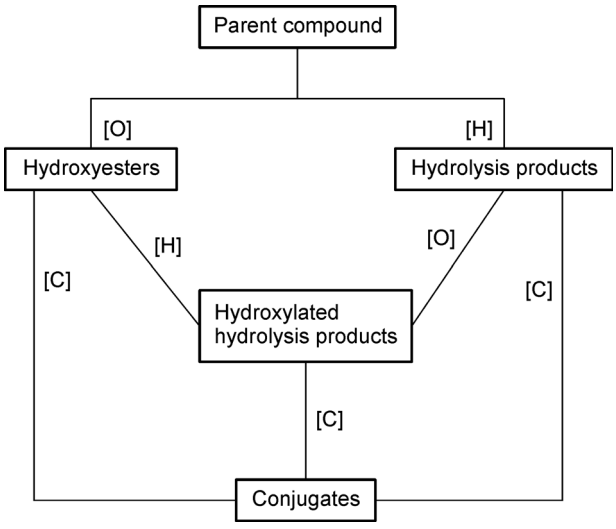


Figure 1 Generalized pathway involved in the metabolism of pyrethroids in mammals by hydrolysis [H], oxidative [O], and conjugation [C] reactions.

Degradation methods for the removal of pyrethroid residues

Epidemiological data, clinical reports, and other laboratory studies indicate that pyrethroids possess estrogenic and antiprogestagenic activities. Thus, they are classified as endocrine disruptors (Garey and Wolff, 1998). Therefore, it is important to develop rapid and efficient degradation processes to eliminate or minimize the contamination of pyrethroids in the environment. In the natural environment, the insecticide pyrethroid are degraded by biotic and abiotic pathways, including photooxidation, chemical oxidation, and biodegradation (Abraham and Silambarasan, 2014; Abraham and Silambarasan, 2016). The degradation of pyrethroids in the soil is mostly by chemicals and indigenous microbes.

The microbes in soil play the most important role in degrading pyrethroids in soils and sediments. The rate of degradation in the soil depends mainly on the type of pyrethroid, soil, and climate; the species of microorganism; and the size of their population. Many pyrethroid-degrading microbes have been isolated from soils (Liang et al., 2005; Tallur et al., 2008; Wu et al., 2006). A list of microbes with a potential to biodegrade pyrethroids has been provided in Table 3. Biodegradation is considered the most prominent and effective technique for the removal of pesticides. Microbial degradation is generally considered to be the safest, least disruptive, reliable, and cost-effective technique for pesticide degradation.

Table 3 List of microbes involved in the degradation of pyrethroid residues

Pyrethroid degrading microbe	References
Bacillus cereus SM3	Maloney et al. (1993)
Pseudomonas fluorescens	Grant et al. (2002)
Pseudomonas sp.	Halden et al. (1999)
Cladosporium sp.	Chen et al. (2011)
Rhodococcus sp. CDT3	Xu et al. (2007)
Vibrio hollisae	Lee et al. (2004)
Burkholderia pickettii	Zhai et al. (2012)
Erwinia carotovora	Liang et al. (2005)
Ochrobactrum anthropi YZ-1	Wu et al. (2006)
Aspergillus niger ZD11	Guo et al. (2009)
Klebsiella sp. ZD112	Wu et al. (2006)
Sphingobium sp. JZ-2	Maloney et al. (1988)
Achromobacter sp.	Sakata et al. (1992)
Bacillus cereus	Yu and Fan (2003)
Serratia plymuthica	Lee et al. (2004)
Pseudomonas sp. YF05	Saikia and Gopal (2004)
Stenotrophomonas acidaminiphila, *Aeromonas sobria*	Preeti et al. (2008)
Yersinia frederiksenii	Zhang et al. (2010)
Trichoderma viride	Chen et al. (2011a)
Micrococcus sp.	Chen et al. (2011b)
Serratia sp.	Chen et al. (2011c)
Streptomyces sp.	Maloney et al. (1993)
Ochrobactrum sp.	Grant et al. (2002)
Stenotrophomonas sp.	Halden et al. (1999)

Role of carboxylesterases in the detoxification of pyrethroids

The carboxylesterases (Ces) represent a group of enzymes belonging to α/β hydrolase fold family (Hosokawa 2008). They are extremely important for the hydrolysis of numerous xenobiotic and endogenous ester containing compounds. They play a crucial role in the detoxification of pyrethroids by hydrolyzing ester bonds. Carboxylesterase are highly efficient in hydrolyzing pyrethroids to their corresponding acid and alcohol, thereby significantly reducing the toxicity. The Ces are efficient in rapidly degrading class I and class II pyrethroids. Furthermore, they can be used to minimize the toxicity of ester containing pyrethroids. Pyrethroids are a large class of compounds that consist of an ester bond, which is formed by an alcohol and acid moieties. The major metabolic pathway of pyrethroid residues in resistant insects and microbes, which degrade them, involves oxidation by cytochrome P450s and hydrolysis by esterases (Kasai, 2004).

Some pyrethroid Ces, which hydrolyze permethrin into 3-phenoxybenzyl alcohol and 3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid, have been purified and characterized from pyrethroid-resistant insects (Sogorb and Vilanova, 2002). However, these enzymes often have low activity and narrow substrate specificity (Stok et al., 2004).

Hydrolysis of pyrethroids by carboxylesterases

Pyrethrins and allethrins are metabolized via oxidation of the side chain of the acid moiety, and hydrolysis of the ester bond plays an insignificant role. However, for most other pyrethroids, the hydrolysis by CbEs is more essential than oxidation for the degradation and detoxification (Lawrence and Casida, 1982). In general, the trans-isomers of pyrethroids are degraded faster by CbEs when compared with that of the cis-isomers (WHO, 1989; WHO, 1990). The slow degradation of cis-isomers might increase the toxicity in mammals, as they exhibit a high affinity for the Na^+ channel than the trans-isomers. Pyrethroids induce toxicity by changing the functions of these channels (Narahashi, 1992). It has been reported that synergist compounds, by inhibiting CbEs, significantly increase the toxicity of pyrethroids (Lawrence and Casida, 1982; Valles et al., 2000).

Aspergillus niger ZD11 has been reported to be efficient in utilizing pyrethroid pesticide as the sole carbon source (Liang et al., 2005). Chen et al. (2013) isolated a novel enzyme from *Streptomyces* sp., which could degrade β-cypermethrin. The isolate was found to utilize β-cypermethrin as the growth substrate. The enzyme was purified and identified as monooxygenase, which was found to be significantly stimulated by Fe^{2+} and strongly inhibited by Ag^+, Al^{3+} , and Cu^{2+} .

Genes encoding pyrethroid-hydrolyzing carboxylesterase

Many studies have focused on the detoxification of pyrethroid insecticides, degradation ability of microbial strains, analysis of metabolites, and purification of some related degrading enzymes. However, studies on genes responsible for pyrethroid degradation are limited. A few genes encoding pyrethroid-hydrolyzing Ces have been identified. Until now, only a few pyrethroid-degrading genes, such as *sestP*, *pytH*, *pye3*, and *pytZ*, have been reported (Wu et al., 2006; Wang et al., 2009; Li et al., 2008; Zhai et al., 2012). Pei et al. (2006) isolated pyrethroid hydrolyzing esterase (*EstP*) gene from *Klebsiella* sp. strain ZD 112, cloned it into *Escherichia coli*, and sequenced. The *EstP* gene encoded product can degrade many pyrethroid pesticides, and also malathion—an organophosphorous pesticide. It can also hydrolyze ρ-nitrophenyl esters of various fatty acids. This indicates that EstP is an esterase with a broad substrate spectrum.

Wang et al. (2009) reported a novel esterase gene *pytH* encoding a pyrethroid-hydrolyzing Ce cloned from *Sphingobium* sp. strain JZ-1. PytH can transform p-nitrophenyl esters of short-chain fatty acids and can degrade a wide range of pyrethroid pesticides. Furthermore, its hydrolysis efficiency depends on the structure of the pyrethroid molecule. Li et al. (2008) identified pyrethroid-hydrolyzing esterase gene *Pye3*,

a suitable candidate for in situ detoxification of pyrethroids, and cloned it successfully using metagenomic DNA. Fang et al. (2013) investigated on the degradation of fenvalerate using a novel strain *Sphingomonas* sp. strain F-7. The organism was found to utilize fenvalerate as the sole carbon source. It degraded fenvalerate within 72 h by hydrolyzing carboxylester linkage yielding 3-phenoxybenzoic acid (3-PBA) as the major metabolite. Furthermore, *Sphingomonas* sp. degraded permethrin, fenpropathrin, β-cypermethrin, cyhalothrin, deltamethrin, bifenthrin, and 3-phenoxybenzoic acid.

In another study, a novel pyrethroid-degrading esterase gene *pytY* was isolated from the genomic library of *Ochrobactrum anthropi* YZ-1. PytY exhibited efficient degrading ability and stability over a wide range of temperature and pH. The optimal temperature and pH were 35°C and 7.5, respectively. Cofactors were not required for the enzyme activity (Ruan et al., 2013). Until now, all pyrethroid degrading genes reported in genomic library are single genes. However, *pytY* is the second pyrethroid degrading gene isolated from the strain *O. anthropi* YZ-1, making it the first study to report two genes from the same strain, and both contributing to pyrethroid degradation.

Summary

The ever growing population and increasing demand for food have increased the dependence on pesticides and insecticides to protect the plants from pest, thus improving plant yield. However, this is at the expense of environment, which is being contaminated by pesticides. This calls for the implementation of safe methods, such as use of biopesticides. Biodegradation is a promising and reliable technique. It is also an ecofriendly method in effectively remove the contaminants from the soil. However, more attention at the molecular level and more understanding of the organism are required for effective implementation of bioremediation of pesticides.

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Conflict of interest

There is no conflict of interest to declare.

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