Toxicol TIMES, 2026
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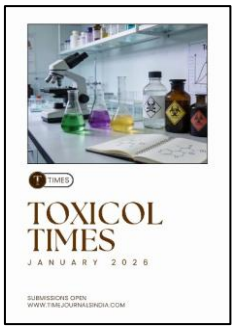
Short Communication



Use of Pyrethroid Chemicals: Toxicology and Environmental Effects

Thind PS*
Department of Toxicology, Gawalior College Rajasthan, India

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Received: March 14, 2026
Accepted: May 08, 2026
Published: May 22, 2026

***Corresponding author:**
Thind PS
E-mail:
thindpssraj@gmail.com

ABSTRACT

Pyrethroids are synthetic analogs of natural pyrethrins, widely used as insecticides due to their high efficacy and relatively low mammalian toxicity. This article reviews the toxicological profiles of pyrethroids, their mechanisms of action, human health implications, and environmental effects. Emphasis is placed on their neurotoxic effects, bioaccumulation, and impact on non-target organisms, including aquatic life and beneficial insects. Regulatory considerations and future research directions are also discussed.

KEYWORDS: Pyrethroids, Environment, Chemicals, Toxicology, Bioaccumulation

1. INTRODUCTION

Pyrethroids represent a major class of synthetic insecticides derived from pyrethrins, which are natural insecticidal compounds extracted from Chrysanthemum flowers. Since their introduction in the 1970s, pyrethroids have become widely used in agriculture, public health, and household pest control due to their potent insecticidal activity and comparatively favorable safety profile in mammals (Soderlund, 2012). However, concerns have emerged regarding their toxicological effects on humans and wildlife, as well as their environmental persistence and bioaccumulation potential (Bradberry et al., 2005).

This review aims to provide a comprehensive overview of pyrethroid toxicology and environmental effects, focusing on their mechanisms of action, exposure routes, toxicokinetics, and ecological impacts.

2. CHEMICAL STRUCTURE AND CLASSIFICATION

Pyrethroids are classified into two main types based on their chemical structure and toxicological effects:

- **Type I pyrethroids**: Lack an alpha-cyano group; examples include permethrin and allethrin. They induce the T-syndrome characterized by tremors and hyperexcitability.
- **Type II pyrethroids**: Contain an alpha-cyano group; examples include cypermethrin and deltamethrin. They cause the CS-syndrome, marked by choreoathetosis and salivation (Ray & Fry, 2006).

Both types share a common mode of action targeting voltage-gated sodium channels in nerve membranes but differ in their binding affinity and toxicological manifestations.

3. MECHANISM OF TOXICITY

Pyrethroids exert their toxic effects primarily through modulation of voltage-gated sodium channels in neuronal membranes. They prolong the opening of these channels, leading to sustained depolarization, repetitive nerve firing, and eventual neurotoxicity (Soderlund et al., 2002).

Type I pyrethroids cause repetitive firing and prolonged sodium channel opening without complete inactivation, resulting in hyperexcitation and tremors. Type II pyrethroids stabilize the sodium channel in an open state longer and may also affect other ion channels, such as calcium and chloride channels, contributing to more severe neurotoxic symptoms (Shafer et al., 2005).

4. HUMAN TOXICOLOGY

4.1 Exposure Routes

Humans are exposed to pyrethroids primarily via dermal contact, inhalation, and ingestion. Occupational exposure occurs in agricultural workers and pest control professionals, while the general population may be exposed through residues in food, water, and indoor environments (Bradberry et al., 2005).

4.2 Acute Toxicity

Pyrethroids generally exhibit low acute toxicity in mammals, with oral LD50 values typically exceeding 100 mg/kg in rats (Ray & Fry, 2006). Symptoms of acute poisoning include paresthesia, dizziness, headache, nausea, and in severe cases, seizures and respiratory distress (Shafer et al., 2005).

4.3 Chronic Toxicity and Neurotoxicity

Chronic exposure studies suggest that pyrethroids may cause neurobehavioral alterations, immunotoxicity, and endocrine disruption. Animal studies have demonstrated effects such as learning deficits, motor dysfunction, and changes in

neurotransmitter levels following prolonged exposure (Gupta, 2014). Epidemiological data on humans are limited but indicate potential associations between pyrethroid exposure and developmental neurotoxicity, particularly in children (Eskenazi et al., 2018).

5. METABOLISM AND TOXICOKINETICS

Pyrethroids are rapidly metabolized in mammals primarily via hydrolysis by carboxylesterases and oxidation by cytochrome P450 enzymes, leading to less toxic metabolites excreted mainly in urine (Ray & Fry, 2006). However, variability in metabolic capacity may influence individual susceptibility.

6. ENVIRONMENTAL FATE AND EFFECTS

6.1 Persistence and Bioaccumulation

Pyrethroids are generally lipophilic and tend to adsorb strongly to soil and sediment particles, which limits their mobility but increases persistence in these compartments (Li et al., 2017). Their half-lives in soil range from days to weeks depending on environmental conditions.

Bioaccumulation in aquatic organisms has been documented, raising concerns about trophic transfer and ecosystem impacts (Minguez et al., 2018).

6.2 Effects on Non-target Organisms

- **Aquatic toxicity**: Pyrethroids are highly toxic to fish and aquatic invertebrates due to their neurotoxic mode of action. Sublethal effects include behavioral changes, impaired reproduction, and developmental abnormalities (Beketov & Liess, 2008).
- **Pollinators**: Exposure to pyrethroids adversely affects bees and other pollinators, causing mortality, impaired foraging behavior, and colony decline (Sánchez-Bayo & Goka, 2014).
- **Beneficial insects**: Natural predators and parasitoids of pest species may also be harmed, potentially disrupting integrated pest management strategies (Desneux et al., 2007).

7. ENVIRONMENTAL MONITORING AND RISK ASSESSMENT

Due to their widespread use, pyrethroids are frequently detected in surface waters and sediments. Risk assessments emphasize the need to consider chronic and sublethal effects on aquatic life and non-target insects (Li et al., 2017).

8. REGULATORY STATUS AND SAFETY MEASURES

Regulatory agencies such as the US Environmental Protection Agency (EPA) and the European Food Safety Authority (EFSA) have established maximum residue limits and guidelines for pyrethroid use to mitigate risks to human health and the environment (EPA, 2020). Personal protective equipment, proper application techniques, and adherence to recommended dosages are critical to reducing exposure risks.

9. FUTURE DIRECTIONS AND RESEARCH NEEDS

Key areas for future research include:

- Long-term epidemiological studies to clarify human health risks.
- Development of pyrethroids with reduced environmental persistence and non-target toxicity.
- Improved understanding of mechanisms underlying developmental neurotoxicity.
- Enhanced environmental monitoring using advanced analytical techniques.

10. CONCLUSION

Pyrethroids remain important tools in pest control owing to their high efficacy and relatively favourable safety profile compared with older insecticides. Their rapid action, high potency at low application rates, and lower acute toxicity to mammals have made them widely used in agriculture, public health, and household settings. However, their neurotoxic effects—mediated through disruption of voltage-gated sodium channels—raise concerns for both occupational exposures and vulnerable populations such as children. In addition, many pyrethroids are lipophilic and can persist in soils and sediments, leading to bioaccumulation and chronic exposure risks for non-target organisms, including aquatic invertebrates and beneficial insects such as pollinators and natural enemies of pests. Evidence also indicates that some pyrethroids can impair aquatic ecosystems and contribute to biodiversity loss when runoff carries residues into streams and wetlands. Balancing effective pest management with the protection of human health, non-target species, and environmental quality therefore remains a critical challenge, underscoring the need for integrated pest management, judicious use, and ongoing research on safer alternatives and mitigation strategies.

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Author Contributions
Author conceived the concept, wrote and approved the manuscript.

Acknowledgements
Not applicable.

Funding
Not applicable.

Availability of data and materials
Not applicable.

Competing interest
The author declares no competing interests.

Ethics approval
Not applicable.

Citation: Thind PS (2025) Use of Pyrethroid Chemicals: Toxicology and Environmental Effects. Toxicol TIMES 1(1): 38-40.