Review

Zoology & Toxicology

Toxic Impacts of Cypermethrin on Humans and Animals
A Review

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Pesticides are extensively used in modern agricultural practices worldwide, albeit in varying quantities. While their application has significantly contributed to enhanced crop yields, their widespread use poses considerable environmental, health, and safety risks to both terrestrial and aquatic ecosystems including humans, animals, and plants. Numerous existing and emerging pesticides have been shown to adversely affect the stability, growth, and survival of biological systems. A substantial body of research, utilizing various animal models for risk assessment, has explored the toxicological and biosafety profiles of these chemical agents. Among the different classes of pesticides, synthetic pyrethroids have become widely popular because of their strong insecticidal efficacy and comparatively lower mammalian toxicity than traditional organophosphate and organochlorine compounds. Cypermethrin, a type II synthetic pyrethroid, is extensively utilized in agriculture, veterinary medicine, and household pest management owing to its broad-spectrum effectiveness and cost efficiency. The toxicity of cypermethrin is influenced by multiple factors, including dosage, duration of exposure, and route of entry. Its environmental persistence and bioaccumulation further amplify the risk to non-target organisms, including humans and other terrestrial and aquatic life forms. This article provides a comprehensive review of the toxicological impacts of pesticides, with a specific focus on cypermethrin. It elaborates on the mechanisms of toxicity, public health implications, and its detrimental effects on humans and animals. The primary objective of this review is to consolidate current knowledge on cypermethrin toxicity and highlight its relevance in evaluating environmental and human health risks.

Keywords: Cypermethrin; Toxicity; Human; Animals

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Introduction

PESTICIDES are extensively employed in agriculture and public health to manage insects, weeds, disease vectors, and other harmful or nuisance organisms. While they offer considerable benefits, their misuse or overuse poses significant toxicological risks to both humans and animals (Ferrer, 2003). Among them, pyrethroid pesticides a class of synthetic compounds have gained widespread application in agricultural practices and household settings. Their use has risen significantly in recent decades, partly as a result of the reduced application of organophosphate pesticides, which exhibit higher acute toxicity to birds and mammals (Shafer and Meyer, 2004). Although pyrethroids are generally considered less acutely toxic, emerging research has indicated that they may exhibit endocrine-disrupting properties, raising concerns about their long-term safety (Han et al., 2008). Of particular concern is their potential to interfere with hormonal regulation. While earlier studies primarily investigated the estrogenic activities of pyrethroids (Kojima et al., 2004), recent attention has shifted toward their anti-androgenic effects due to increasing reports of male reproductive health decline. Several studies have linked exposure to pyrethroid pesticides with adverse effects such as reduced sperm count, testicular damage, impaired sperm motility, morphological abnormalities in sperm, and genotoxicity (Song et al., 2008). These outcomes suggest that pyrethroids may exert anti-androgenic effects by disrupting androgen receptor (AR)-mediated signaling pathways. However, evidence also points to AR-independent mechanisms, including interference with testosterone synthesis, secretion, and distribution (Zhang et al., 2007). Given the pivotal role of androgens in maintaining male reproductive function, any disruption in their physiological levels or action can severely compromise fertility (Travison et al., 2008). Pyrethroids may impair androgenic activity by altering the secretion and function of endogenous hormones or by directly affecting testicular androgen biosynthesis. Therefore, greater emphasis must be placed on investigating the endocrine-disrupting potential of pyrethroids to understand and mitigate their reproductive toxicity in both humans and animals.

Pyrethroids: Overview and Toxicological Implications

Pyrethroids are a class of synthetic organic compounds that share structural similarity with naturally occurring pyrethrins, the natural insecticidal substances derived from the dried and powdered flowers of *Chrysanthemum cineraria folium*, a plant similar in appearance to daisies. Pyrethrins are the biologically active constituents responsible for the insecticidal properties of these flowers and consist primarily of three keto alcohols and two carboxylic acids. Major producers of pyrethrins include countries such as Kenya, Tanzania, Ecuador, Japan, Uganda, and Rwanda. More recently, Australia has surpassed Kenya to become the second-largest producer globally (WHO, 1989). In developing countries with agriculture-based economies, pest infestations remain a persistent challenge. As a result, the widespread and often unregulated use of pesticides has become common practice among both farmers and agricultural scientists. However, such indiscriminate application poses significant risks to human health and the environment. Pesticides encompass a variety of chemical agents, including insecticides, fungicides, herbicides (weed killers), rodenticides, and molluscicides. Among insecticides, several major chemical classes are widely used: organophosphates, carbamates, organochlorines, pyrethroids, ryanoids, and neonicotinoids.

This review focuses on pyrethroids, which are among the most commonly used insecticides across different regions of India. Common pyrethroids include allethrins, bifenthrin, cypermethrin, lambda-cyhalothrin, deltamethrin, and esfenvalerate. These compounds are extensively utilized in both agricultural and domestic settings due to their potent insecticidal action and relatively low mammalian toxicity in controlled doses. However, excessive and improper use of pyrethroids has raised concerns about their environmental persistence and potential harmful effects on human and animal health. Exposure to pyrethroids in humans and animals mainly occurs through ingestion, inhalation, and dermal contact. Once inside the body, these compounds can accumulate in various organs, leading to systemic toxicity. They have been shown to disrupt hematological profiles and alter endocrine and hormonal functions. Moreover, exposure can trigger the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), leading to oxidative stress and subsequent cellular damage. Among the organ systems affected, the endocrine and nervous systems are particularly vulnerable. For instance, deltamethrin has been found to damage DNA in thyroid cells, although natural antioxidants like lycopene may offer some protective effects. Other synthetic pyrethroids such as bifenthrin, allethrin, cyhalothrin, and cypermethrin have demonstrated detrimental impacts on neuronal and hormonal balance (WHO, 1989). In conclusion, while pyrethroids are effective an insecticidal agent, their unregulated and excessive use poses significant toxicological risks. Their accumulation through the food chain and subsequent effects on biological systems necessitate careful monitoring and stricter regulatory oversight.

Cypermethrin (CYP), a synthetic pyrethroid derived from natural pyrethrins found in *Chrysanthemum cinerariaefolium*, is extensively used as a broad-spectrum insecticide due to its high potency against insects approximately 2,250 times more toxic to insects than to mammals (Tsuji et al., 2012). This differential toxicity is attributed to physiological differences such as smaller body size, lower body temperature, and increased sensitivity of insect sodium channels. The primary mechanism of action of pyrethroids involves their interaction with voltage-gated sodium channels, causing prolonged neuronal depolarization that ultimately results in neurotoxic effects (Bradberry et al., 2005). However, accumulating evidence suggests that cypermethrin exhibits broader neurotoxic effects. In mammalian models, particularly in rat brain synaptosomes, cypermethrin has been shown to disrupt not only sodium channels but also calcium and chloride ion channels (Breckenridge et al., 2009). Furthermore, recent studies have reported that cypermethrin can induce mitochondrial dysfunction by collapsing mitochondrial membrane ion gradients, potentially impairing ATP synthesis and promoting oxidative stress (Paravani et al., 2019). In addition to its neurotoxicity, cypermethrin poses a risk to human health due to its various routes of entry into the body dermal absorption, inhalation, and ingestion through contaminated food

or water (Akelma et al., 2019). Its lipophilic nature facilitates bioaccumulation in several tissues, including adipose tissue, skin, liver, kidneys, adrenal glands, and ovaries. Prolonged exposure has been associated with disruptions in immune responses, cardiovascular function, hepatic metabolic pathways, and enzymatic activities in mammals (Ji et al., 2019). As a consequence of widespread pesticide use, the risk of food contamination with harmful chemicals has increased, posing potential threats to both domestic animals and humans. Although insecticides like pyrethroids are generally considered to have low mammalian toxicity, evidence suggests that they can still cause significant harm (Usmani and Knowles, 2001). In rats, acute exposure to type-II pyrethroids via oral or intravenous routes has been shown to induce symptoms such as excessive salivation, jaw clenching, a rolling gait due to increased hindlimb extensor tone, tremors, impaired coordination, followed by tonic seizures and eventual death. Additionally, memory impairments have been reported in rats exposed to type-II pyrethroids (Nieradko-Iwanicka and Borzęcki, 2008).

Human exposure, particularly among vulnerable groups such as children, newborns, and pregnant women, has also been documented (Berkowitz et al., 2003). Pyrethroid exposure during pregnancy is associated with increased risks of embryonic resorption and fetal toxicity (Joya and Sangha, 2016). Maternal exposure to cypermethrin has been shown to negatively affect the body and organ weights of offspring (Huang and Li, 2014), while reproductive toxicity studies in rats have revealed increased pre- and post-implantation losses, reduced corpus luteum counts, fewer implantation sites, and higher early mortality rates (Ahmad, 2010). Epidemiological data indicate that women engaged in agricultural work before or during the first trimester are more likely to give birth to infants with congenital anomalies such as oral clefts, neural defects, and other developmental malformations (Ramon-Yusuf et al., 2017). Cypermethrin, a type-II synthetic pyrethroid, can cross the placental barrier, potentially disrupting fetal weight regulation and neurological development (Madu, 2015; Dewailly et al., 2014). The primary toxicological mechanism of cypermethrin involves its neurotoxic action through the prolonged opening of sodium channels, leading to sustained membrane depolarization and excessive neurotransmitter release. This causes hyper excitation of the central nervous system and affects other voltage-gated channels, including those for calcium, potassium, and chloride, resulting in oxidative stress and DNA damage (Kumar Singh et al., 2012). Due to its rapid degradation rate, cypermethrin is widely utilized in agriculture, veterinary practice, and household pest control. It is commonly found in insect repellents (e.g., Lal Hit, Baygon) and is extensively used against ectoparasites such as termites, fleas, cockroaches, and moths affecting cotton, fruits, and vegetables. Furthermore, cypermethrin-treated mosquito nets are employed in malaria prevention programs across West Africa (Lim et al., 2011; Guessan et al., 2014). The alpha-cyano group characteristic of type-II pyrethroids contributes to the prolonged depolarization of nerve membranes, exacerbating their neurotoxicity (Soderlund et al., 2002).

Toxicological Effects of Cypermethrin on Reproductive and Neurological Systems

Cypermethrin, a widely used synthetic pyrethroid, exerts a range of deleterious effects on embryonic development, reproductive function, and neural systems in mammals and aquatic species. These effects are often mediated by oxidative stress, endocrine disruption, and interference with neurotransmitter systems.

Embryotoxic and Teratogenic Effects

Cypermethrin exposure disrupts normal embryonic development, significantly increasing the incidence of embryo toxicity. Sharma et al. (2018) reported a 27% rise in embryo mortality and a 68% increase in developmental abnormalities following CYP exposure. These alterations may result from direct damage to embryonic tissues or interference with maternal endocrine signaling pathways.

Male Reproductive Toxicity

In male rats, cypermethrin disrupts testicular architecture and decreases serum concentrations of essential reproductive hormones such as testosterone, follicle-stimulating hormone (FSH), and luteinizing hormone (LH). This is accompanied by reduced daily sperm production, indicating compromised spermatogenesis (Elbetieha et al., 2001).

Female Reproductive Toxicity

Cypermethrin exposure in female rodents leads to:

- Increased atresia of ovarian follicles and reduction in healthy follicles and corpora lutea.
- Significant declines in serum estradiol levels and disrupted estrous cycles (Hamdani & Yajurvedi, 2017).
- Altered ovarian and uterine morphology, including decreased ovarian weight, increased uterine weight, and thickened myometrium (Sangha et al., 2013).
- Degenerative ovarian changes like follicular atresia, reduced follicular diameter, and vascular regression (Molavi et al., 2014).

Furthermore, CYP impairs ovulation rates, causes ovarian atypia (Khatab et al., 2016), and lowers implantation success and fetal viability when exposed males impregnate females (Hallenbeck & Cunningham-Burns, 1985). Histopathological findings reveal yolk vesicle disruption, germinal cell vacuolation, and connective tissue necrosis in ovaries (Mukadam & Kulkarni, 2014). Cypermethrin also causes hormonal imbalances such as increased serum estradiol (E2) and decreased progesterone (P4) levels, further contributing to impaired fertility (Zhou et al., 2018).

Neurotoxic Effects

Cypermethrin's primary mechanism of neurotoxicity involves alteration of voltage-gated sodium channels, leading to prolonged neuronal depolarization and hyper excitation. At the synaptic level, this results in:

- Elevated release and eventual depletion of neurotransmitters such as dopamine and GABA.
- Inhibition of monoamine oxidase, increasing neurotransmitter accumulation (Miyamoto et al., 1995).
- Blockade of chloride channels and disturbance in calcium and potassium ion homeostasis.

These neurophysiological alterations manifest as motor dysfunction, behavioral abnormalities, and neurodegeneration, as observed in multiple animal models (Singh et al., 2012).

Oxidative Stress and Endocrine Disruption

Cypermethrin exposure induces excessive generation of reactive oxygen species (ROS), resulting in:

Increased levels of malondialdehyde (MDA), a marker of lipid peroxidation. Decreased activities of key antioxidant enzymes, including superoxide dismutase (SOD), catalase, and glutathione (GSH) (Kaushik et al., 2018; Sharma et al., 2018). In ovarian tissue, this oxidative damage results in lowered lactate dehydrogenase and 3β-hydroxysteroid dehydrogenase levels, impairing steroidogenesis. Increased acid and alkaline phosphatase activity further reflects tissue degeneration (Sangha et al., 2013). At the molecular level, CYP induces expression of apoptotic markers such as Apaf1 and lowers the Bcl2/Bax ratio in liver tissue of zebrafish (Jin et al., 2011), indicating pro-apoptotic activity. Additionally, CYP increases p53 mRNA expression, leading to follicle cell cycle arrest and reduced follicular growth.

Implications in Humans

Human populations, particularly agricultural and industrial workers, are at risk of chronic CYP exposure through dermal contact, inhalation, and ingestion. Documented health outcomes include:

- Reproductive organ damage (infertility, sterility, miscarriages).
- Blood disorders, neurotoxicity, and hepatic fibrosis.
- Genotoxic effects, respiratory illness, and hormonal disruption.

Cypermethrin's lipophilic nature facilitates its accumulation in fatty tissues of organs like the brain, liver, kidneys, adrenal glands, and ovaries (Tao et al., 2008). Notably, its transplacental transfer affects fetal neurological and physiological development (Dewailly et al., 2014), raising concerns over prenatal exposure.

The widespread application of cypermethrin, a synthetic pyrethroid, has been associated with adverse effects on non-target species, despite its classification by the U.S. EPA (2006) as neither a developmental nor reproductive toxicant. However, experimental studies have demonstrated mild to moderate toxic symptoms in both male and female rats following oral administration of cypermethrin at doses of 5 and 20 mg/kg/day over a 30-day period. Notably, female rats exhibited reproductive toxicity, including damage to the uterus and ovaries at repeated doses. At 20 mg/kg/day, loss of follicular cells and oocytes in the ovaries was observed (Grewal et al., 2010).

Further investigations by Sangha et al., (2013) reported a decrease in ovarian weight and a simultaneous increase in uterine weight, length, and myometrial thickness after cypermethrin administration (50 mg/kg body weight) over two and four weeks. These effects were accompanied by enhanced follicular atresia and significant reductions in ovarian protein (38%), lipid (20%), phospholipid (18%), and cholesterol (37%) content. Enzymatic assays showed reduced activities of lactate dehydrogenase and 3β-HSDH, with a concurrent increase in acid and alkaline phosphatase activity, indicative of ovarian degeneration.

In vitro studies conducted by Gill et al., (2011) revealed that cypermethrin exposure led to pronounced luteal cell degeneration, including vacuolation, necrosis, and reduced cell viability, ultimately resulting in decreased progesterone production in bovine corpus luteum cells.

Histopathological evaluations by Garoussi et al., (2010) of infertile Holstein dairy cows demonstrated endometrial glandular atrophy following pyrethroid exposure. Similarly, Ullah et al., (2006) documented microscopic changes in the ovaries and uteri of rabbits given intraperitoneal doses of 25, 50, and 75 mg/kg body weight, noting connective tissue proliferation in the ovarian cortex at higher doses. These uterine alterations, including glandular atrophy and congestion, were likely due to cypermethrin-induced disruption of the ATP-dependent energy pathway.

Cypermethrin is extensively used in veterinary medicine for the topical treatment of ectoparasitic infestations, including lice, ticks, and blowflies, in animals such as cattle, sheep, rabbits, dogs, and poultry (Roberts, 1987; EMEA, 2004). It is commercially available in multiple formulations liquid, semi-liquid, and powder (WHO, 1989; Sudakin, 2006; USEPA, 2010).

Due to its lipophilic nature, cypermethrin is capable of crossing the blood-brain barrier, causing neurotoxicity and motor dysfunction (Singh et al., 2012). Its primary toxicological mechanism involves modification of voltage-gated sodium channels, leading to prolonged neuronal depolarization. Additionally, cypermethrin affects other ion channels (chloride, calcium, potassium) and disrupts neurotransmitter levels, particularly dopamine and γ-aminobutyric acid (GABA).

The objective of this review is to synthesize and evaluate existing literature concerning the toxicological impacts of cypermethrin on human and animal health.

Methodology

The purpose of this study was to systematically compile and review the current scientific literature regarding the toxicological impacts of cypermethrin on humans and animals. A comprehensive literature search was conducted using reputable and authenticated academic databases, including PubMed, ScienceDirect, Elsevier, and other peer-reviewed sources were searched for relevant articles published between 2000-2024. Relevant research articles, review papers, and toxicological reports were identified using specific keywords such as "cypermethrin toxicity," "pyrethroid effects," "human exposure," "animal models," and "environmental health impacts." Studies were selected based on their relevance, recency, and the quality of experimental evidence they presented. Both in vivo/in vitro studies involving mammalian and non-mammalian species were considered. Articles that discussed biochemical, neurological, reproductive, and systemic effects of cypermethrin were prioritized. The gathered data were critically analyzed, summarized, and interpreted to provide an overview of the known mechanisms, observed toxic effects, and the extent of cypermethrin's impact on biological systems. A total number

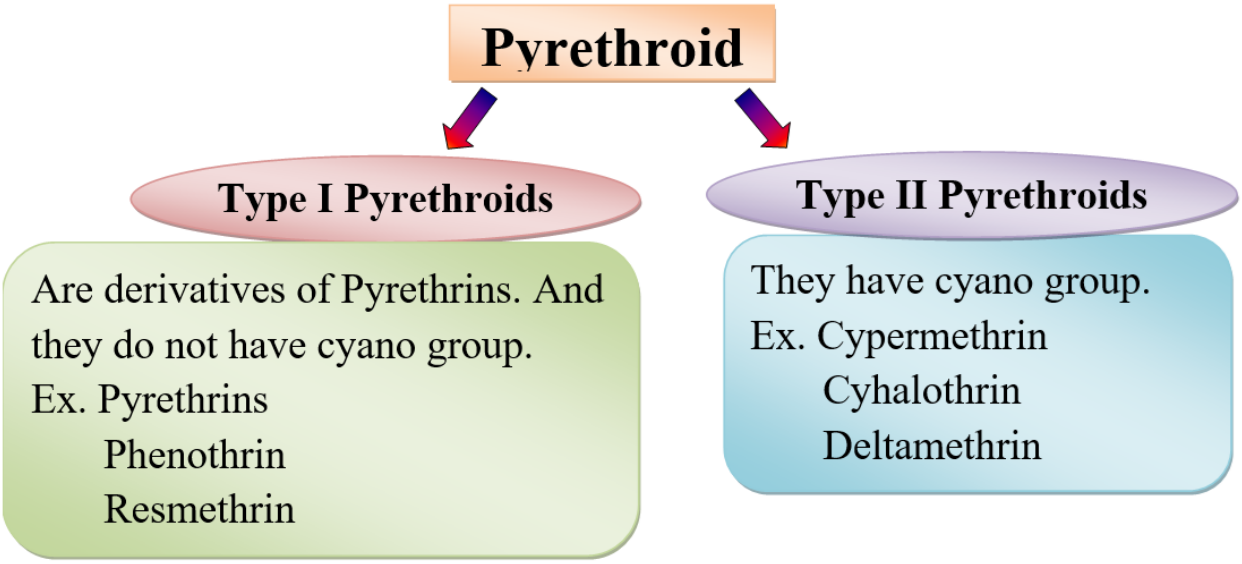

Figure1. Types of Pyrethroids.

of 236 records were obtained when combinations of all the keywords were made, andafter excluding the duplicates, the remaining articles were classified according the main focus of each article. About literature reviews, a total number of 348 focused on cypermethrin and toxicity were found. The abstracts of all articles were carefully studied, and the articles reporting the mode of action and/or toxic effects (animals and humans) of the cypermethrin were included. A final number of 49 studies were found relevant that constituted the main structure of the present review article.

Cypermethrin: A Synthetic Pyrethroid Insecticide

Cypermethrin is a synthetic pyrethroid insecticide widely employed in agriculture, veterinary medicine, and public health sectors. Notably neurotoxic, it is primarily used to protect cotton crops globally and is one of the most prevalent pyrethroids used for residential, commercial, and agricultural pest management. It is also utilized in veterinary medicine to treat ectoparasitic infestations such as lice, ticks, fleas, and blowflies in cattle, poultry, sheep, dogs, and other domestic animals (EMEA, 2001; Robin et al., 2019). In public health applications, cypermethrin is effective in controlling common pests like mosquitoes, houseflies, and cockroaches (Neskovic et al., 2013).

History and Uses

Pyrethrins, the natural compounds from which pyrethroids are derived, constitute approximately 25% of the active insecticidal content in dried Chrysanthemum flowers (Harlod et al., 2003). Cypermethrin, synthesized in 1974, has been extensively used since the late 1970s (Yousef et al., 1998; C.A.C, 2003). Its global usage is evident from data such as Iraq's importation of pesticides, where insecticides including cypermethrin constitute 80% of total imports (Heamza, 2009). In the USA, over a billion pounds of pesticides are used annually, while Egypt imports more than 30,000 metric tons each year (Yousef et al., 1999). Globally, cypermethrin accounts for roughly 25% of the pesticide market (Khan et al., 2009). Cypermethrin is predominantly applied through spraying or dipping to eliminate pests like ticks and lice. It is also used in household applications, persisting on furniture and walls for up to three months (Harlod et al., 2003). In California, it ranks as the fourth leading cause of pesticide-related illnesses among structural pest control workers (Kegley et al., 2016).

Physicochemical Properties

Cypermethrin is known for several favorable insecticidal properties:

- Rapid Neurotoxicity: It acts instantly on insect nervous systems.
- Low Persistence: Decomposes rapidly in sunlight and air, reducing environmental risks.
- Repellency and Knockdown Effect: Efficient at repelling and immobilizing insects.
- Low Mammalian Toxicity: Considered among the least toxic to domestic animals.

Cypermethrin typically appears as a pale yellow, viscous oil or dust, insoluble in water but soluble in organic solvents. Upon exposure to air and light, it oxidizes and loses its insecticidal potential. It is highly lipophilic, resists being washed away by rain, and hydrolyzes in alkaline environments (Tomlin, 1994) (**Figure 1**).

What is Cypermethrin?

Cypermethrin is a synthetic analog of natural pyrethrins extracted from Chrysanthemum flowers. Upon contact with insect exoskeletons, it acts as a fast-acting neurotoxin (WHO, 1989). Although it persists for weeks on indoor inert surfaces, it rapidly degrades when exposed to soil, sunlight, and water. The compound is extremely toxic to aquatic organisms, bees, and

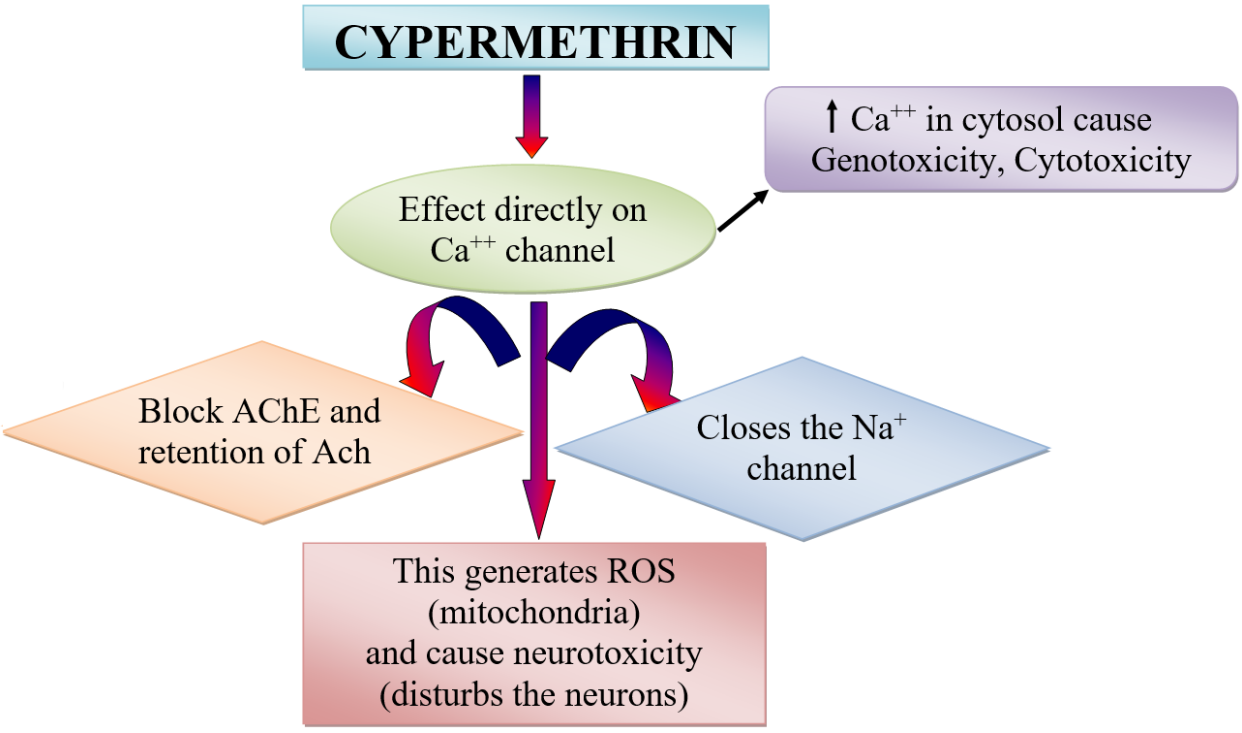

Figure 2. Mechanism of Action of Cypermethrin.

fish (NPTN). It is commonly found in commercial insecticidal products such as Raid, Ortho, Combat, and ant chalk (Gammon et al., 1981).

Mechanism of Action

Cypermethrin acts primarily by altering nerve signal transmission. It binds to voltage-gated sodium channels on neuronal membranes, preventing them from closing during repolarization. This results in continuous nerve stimulation, repetitive discharges, and eventual paralysis (Lawrence et al., 1982). At low doses, insects show hyperactivity, while higher concentrations lead to paralysis and death. The compound is metabolized into cyanohydrins, which decompose into cyanide and aldehydes. This metabolic breakdown generates reactive oxygen species (ROS), leading to oxidative stress, lipid peroxidation, and increased intracellular calcium levels factors contributing to cytotoxicity and genotoxicity (Wielgomas & Krechniak, 2007; Ullah, 2018). Additionally, cypermethrin inhibits acetylcholinesterase (AChE), resulting in acetylcholine accumulation at synapses and disrupting normal nerve signal termination (Dermot et al., 1998) (**Figure 2**).

Toxic Symptoms of Cypermethrin Poisoning

Cypermethrin poisoning primarily impacts the nervous and muscular systems. Common clinical symptoms include:

- Mild to Moderate Toxicity: Fatigue, drooling, vomiting, diarrhea, urinary incontinence, hyperreactivity, tremors, ataxia, and paresthesia.
- Severe Toxicity: Disorientation, muscle spasms, dyspnea, fever or hypothermia, and respiratory complications.

Symptoms typically develop a few hours after exposure, with severity depending on formulation, dosage, and route (ingestion, inhalation, dermal contact). Prolonged skin exposure may result in erythema, pruritus, or dermatitis. Mucous membranes especially eyes, respiratory tract, and genitalia are vulnerable to irritation and inflammation. Repeated inhalation may cause allergic sensitization and asthmatic symptoms. In extreme cases, especially with prolonged or high-dose inhalation, respiratory paralysis and death may occur (Lawrence et al., 1982; Klassen et al., 1996) (**Figure 3**). Young animals and pets are particularly sensitive to cypermethrin. Misuse of agricultural or vector control formulations on pets, especially cats and dogs, can result in accidental overdose due to the absence of appropriate dosing instructions and presence of potentially harmful inert ingredients.

Products Used Which Contain Cypermethrin

Termiticides, Household insecticides, Outdoor insecticides, Ammo TM, Cybush R, Cynoff TM, Cyper kill, Demon R (United States Environmental Protection Agency, 1989).

Environmental Toxicity of Cypermethrin

Cypermethrin, a synthetic pyrethroid insecticide, poses significant environmental risks, particularly to aquatic ecosystems. Like other synthetic pyrethroids, it is highly toxic to fish and aquatic invertebrates. Consequently, the disposal of cypermethrin residues or dip wash into watercourses is strictly prohibited due to its potentially devastating impact on aquatic

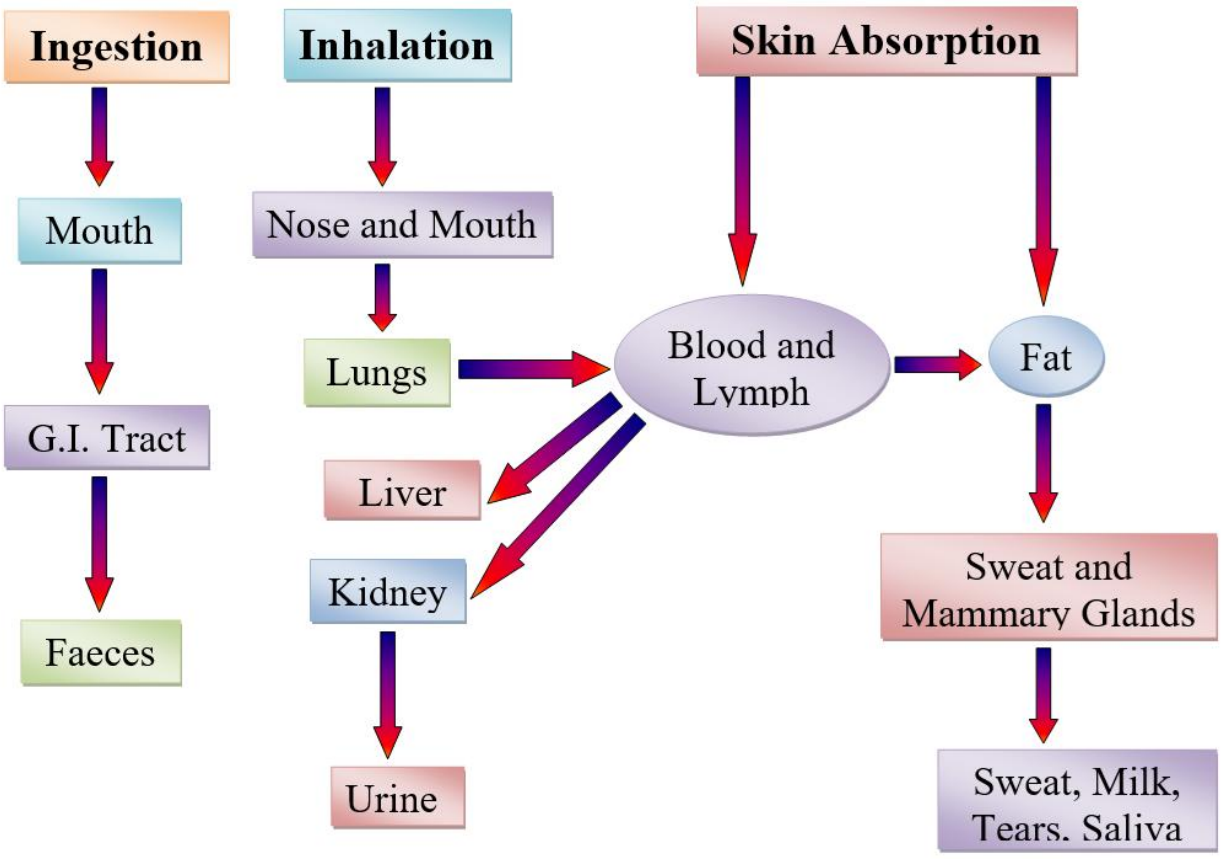

Figure 3. Animal Exposure to Cypermethrin.

life (United States Environmental Protection Agency, 1997).

In recognition of these environmental hazards, regulatory authorities in several countries have banned the use of synthetic pyrethroid-containing livestock dips. However, cypermethrin exhibits low toxicity to birds, making it relatively safer than organophosphates in avian species. When used according to guidelines in dogs and livestock, cypermethrin poses minimal risk of environmental contamination. Nevertheless, environmental pollution through runoff following pour-on treatments in large cattle herds remains a concern, although it is significantly less hazardous than its use in crop spraying or plunge dipping practices (U.S. EPA, 1997). Cypermethrin is highly resistant to photo degradation, with a half-life of 8 to 16 days under sunlight. It has a strong affinity for soil particles and is nearly insoluble in water, thus minimizing the risk of groundwater contamination. Its persistence in water is influenced by temperature and pH, with a half-life exceeding 50 days under typical conditions.

In soils, the persistence of cypermethrin is moderate, highly dependent on soil structure and organic content. It degrades more rapidly in sandy soils with low organic matter than in clayey or organic-rich soils. Under aerobic conditions, the half-life ranges from 4 days to 8 weeks, whereas under anaerobic conditions, degradation is considerably slower. Soil microorganisms play a crucial role in the biodegradation of cypermethrin. Importantly, cypermethrin does not bioaccumulate, reducing long-term ecological concerns in terrestrial food webs (Knisel et al., 1993).

Pharmacokinetics of Cypermethrin

Absorption

Cypermethrin is rapidly absorbed through the gastrointestinal tract (GIT) following oral administration (Adriana, 2004). Absorption also occurs via the pulmonary and gut membranes (Kegley et al., 2016). When orally administered in a 50:50 cis:trans isomer ratio at 3.4 mg in soy oil, the absorption rate ranges from 26% to 56% of the administered dose (Smith et al., 1996). Dermal absorption, however, is relatively slow due to the skin's barrier properties (WHO, 1996; Adriana, 2004).

Distribution

Studies in mammals indicate that cypermethrin distributes rapidly and extensively to multiple tissues, particularly lipid-rich areas and the peripheral and central nervous systems (Khanna, 2002). It also accumulates in organs such as the ovaries and adrenal glands (Smith et al., 1996; Temple & Smith, 1996). In rats, peak concentrations in the nervous system occur within five minutes of intravenous administration (Iwanika et al., 2008).

Table1. Results of Studies of Acute Toxicity with Cypermethrin.[a]

Species	Sex	Strain	Route	Vehicle/comments	LD50 (mg/kg bw)/ LC50 (mg/m3)	Reference
Mouse	M, F	NS	Oral	5% in cornoil	82	Coombs et al.,1976
Mouse	M, F	CD	Oral	5% in cornoil[e]	88	Rose, 1982
Mouse	M, F	NS	Oral	DMSO	138	Coombs et al.,1976
Mouse	M, F	CD	Oral	40% in DMSO[e]	1,126	Rose, 1982
Mouse	M, F	NS	Oral	Aqueous suspension	779[b]	Jaggers, 1979
Mouse	M, F	CD	Oral	50% aqueous suspension[e]	657	Rose, 1982
Mouse	M, F	CD	Dermal	5% in cornoil[e]	>100	Rose, 1982
Mouse	M, F	NS	Intraperitoneal	Corn oil	485	Coombs et al.,1976
Rat	M, F	NS	Oral	Corn oil	251–992	Coombs et al.,1976
Rat	MF	Alderley Park	Oral	Corn oil[f]	247 309	Henderson & Parkinson, 1980
Rat	M, F	Tac:N (SD)fBR	Oral	5% in cornoil[g]	334	Freeman, 1987
Rat	M	NS	Oral	Glycerol formal	200–400	Coombs et al.,1976
Rat	F	NS	Oral	Glycerol formal	Approximately 200	Coombs et al.,1976
Rat	M	NS	Oral	Aqueous suspension	400–800	Coombs et al.,1976
Rat	F	NS	Oral	Aqueous suspension	Approximately 400	Coombs et al.,1976
Rat	F	NS	Oral	Aqueous suspension	4,123[b]	Jaggers, 1979
Rat	M	NS	Oral	Aqueous suspension	3,000[c]	Jaggers,1979
Rat	M, F	Wistar	Oral	50%aqueous suspension[e]	4,000	Rose, 1982
Rat	M, F	NS	Oral	DMSO	303	Coombs et al., 1976
Rat	M, F	Wistar	Oral	40%inDMSOe	4,000	Rose, 1982
Rat (3 wks) d	M, F	NS	Oral	DMSO	163	Rose & Dewar, 1978
Rat (6 wks) d	M, F	NS	Oral	DMSO	322	Rose & Dewar, 1978
Rat (12 wks) d	M, F	NS	Oral	DMSO	526	Rose & Dewar, 1978
Rat	F	NS	I.p.	Aqueous suspension	>500[b]	Jaggers, 1979
Rat	M, F	NS	I.p.	Propylene glycol	1,000-2,000[b]	Jaggers, 1979
Rat	F	NS	Dermal	Undiluted	>4,800[b]	Jaggers, 1979
Rat	M, F	Alderley Park	Dermal	Undiluted	>4,920[j]	Henderson & Parkinson, 1980
Rat	M, F	NS	Dermal	40%inxylene	>1,600	Coombs et al., 1976
Rat	M, F	Alpk: APfSD	Inhalation (4hexp)[h]	MMAD 3.95–5.20µm; 16.9–28.8%, respirable	1,260[i]	Brammer, 1989
Rat	M, F	NS	Inhalation (4hexp)[i]	MMAD2.3–2.5µm;	>1,320-2,500	Mount, 1992
Syrian hamster	M, F	NS	Oral	Corn oil	>400	Combs et al., 1976
Chinese hamster	M, F	NS	Oral	5%incornoil	203	Coombs et al., 1976
Guinea-pig	M	NS	Oral	20%incornoil	Approximately 500	Coombs et al., 1976
Guinea-pig	F	NS	Oral	Corn oil	>1,000	Coombs et al., 1976
Guinea-pig	M	NS	Oral	Aqueous suspension	>4,000[b]	Jaggers, 1979
Rabbit	F	NS	Oral	Undiluted	>2,400[b]	Jaggers, 1979
Rabbit	F	NS	Dermal	Undiluted	>2,400[b]	Jaggers, 1979
Rabbit	M, F	NZW	Dermal	Undiluted	>2,460[j]	Henderson & Parkinson, 1980
Domestic fowl	M, F	NS	Oral	DMSO	>2,000	Coombs et al., 1976
Partridge	M, F	NS	Oral	DMSO	>3,000	Coombs et al., 1976

DMSO: Dimethyl sulfoxide; F: Female; M: male.
NZW: New Zealand White.
a. Unless specified, all values refer to data generated using Cypermethrin (cis:trans=50:50).
b. Data generated using Cypermethrin (cis:trans=40:60).
c. Data generated using Cypermethrin (cis:trans=53:46).
d. Refers to the age of the rats.
e. Purity, 98.1%; cis:trans-isomer ratio=51:49.
f. Technical material, purity, 72.9%; cis:trans-isomer ratio not stated.
g. Purity, 93.5%; cis:trans-isomer ratio not stated.
h. Nose-only exposure.
i. Whole-body exposure; purity, 95.7%; cis:trans-isomer ratio not stated.
j. Purity, 91.5%; cis:trans-isomer ratio 53:47.

Metabolism

Cypermethrin is metabolized swiftly in mammals, primarily via ester hydrolysis, oxidation, and conjugation reactions (Atamanalp et al., 2002; Smith et al., 1996). Rapid hydrolysis of the ester bond in the digestive tract contributes to its low oral toxicity (Beasley, 1999). During biotransformation, both central and peripheral tissues are involved. The hydrolysis of ester linkages yields carboxylic acids and alcohols, which are further oxidized to phenoxybenzoic acids. These are then conjugated to glycine, sulfate, and glucuronide forms for elimination (WHO, 1996; Beasley, 1999; EMEA, 2004). However, the role of sex or age in metabolic enzyme activity remains unclear (Beyerbach, 2000).

Excretion

Cypermethrin is rapidly eliminated, primarily via the urine and feces. Following an oral dose of 3.8 mg/kg, about 61% is excreted within 50 hours 43% in urine and 22.5% in feces (Adriana et al., 2004). Approximately 65% is excreted within two days post-oral administration (Tamang et al., 1991). After topical application in sheep (2.7%), cypermethrin is cleared over six days, mostly through urine and feces. Oral administration to

Table 2. Effect of Cis:Trans Ratio on Acute Toxicity of Cypermethrin.

Species	Sex	Strain	Route	Vehicle	Cis:transratio	LD50(mg/kg bw)	Reference
Rat	M, F	?	Oral	DMSO	Cisonly	160–300	Brown, 1979a
Rat	M, F	?	Oral	DMSO	Transonly	>2,000	Brown, 1979b
Rat	F	?	Oral	Cornoil	90:10	367	Jaggers, 1979
Rat	F	?	Oral	Cornoil	40:60	891	Jaggers, 1979

DMSO: Dimethyl sulfoxide; F: Female; M: Male.

rats at 4 mg/kg in soy oil showed peak excretion between 5 and 10 hours (Temple & Smith, 1996). In dairy cows fed 0.3 mg and 12 mg/kg of cypermethrin, urinary and fecal routes were the primary excretory pathways, with less than 1% found in milk (Woolen et al., 1992). Topical administration at 22 mg/kg in sheep resulted in only 2% excretion after six days, mostly through urine, with some fecal elimination (Adriana, 2004).

Lethal Dose (LD_{50})

The LD_{50} of cypermethrin varies widely across species, influenced by the cis:trans isomer ratio and formulation vehicle used:

- Mice: Oral LD_{50} = 80-769 mg/kg b.w. (Smith et al., 1996)
- Rats:
  - Oral LD_{50} = 190-325 mg/kg (males), 160-520 mg/kg (females)
  - Dermal LD_{50} = 1,630 mg/kg
- Rabbits: Dermal LD_{50} > 2,100 mg/kg
- EPA (1998):
  - Oral LD_{50} (rats) = 260 mg/kg
  - Dermal LD_{50} = 2,400 mg/kg
  - Inhalation LD_{50} = 3 mg/L
- PMEP (1989):
  - Rats: LD_{50} = 250 (197-336) mg/kg (males), 300 (100-450) mg/kg (females)
  - Rabbits: Dermal LD_{50} > 2,500 mg/kg
- Tample & Smith (1996):
  Oral toxic dose varies with isomer type:
  - Cis isomers: 150-350 mg/kg
  - Trans isomers: > 3,000 mg/kg
- Toxicological Thresholds
  - NOEL (No Observed Effect Level) for subchronic oral toxicity in rats: 73 ppm
  - NOEL (chronic toxicity): 160 ppm
  - LOEL (Low Observed Effect Level): 510 ppm
  - Dermal irritation index in rabbits: 0.81 (mild irritant).

Toxicological Studies

Acute Toxicity

Oral, Dermal, and Inhalational Toxicity

The acute toxicity profile of cypermethrin is presented in **Tables 1** and **2**. The toxicological signs observed with cyano-containing pyrethroids, including cypermethrin, were similar and primarily neurological.

Following oral administration, symptoms appeared within a few hours and included sedation, ataxia, unsteady or splayed gait, tip-toe walking, abasia, excessive salivation, diarrhea, piloerection, tremors, and clonic convulsions. In one study, while some rats showed signs throughout the 14-day observation period, most recovered within a few days (Henderson & Parkinson, 1980; Freeman, 1987). With dermal exposure, symptoms were minimal, and acute toxicity was considered low (Jaggers, 1979). Inhalational exposure resulted in salivation, lacrimation, decreased reflexes and activity, slow deep respiration, tremors, and impaired stability, which progressed to splayed or tiptoe gait and tail erection (Brammer, 1989). In dogs, high oral doses induced tremors, vomiting, diarrhea, nervousness, anorexia, and exaggerated ataxia (Coombs et al., 1976). Notably, the cis-isomer of cypermethrin demonstrated greater acute oral toxicity in rats than the trans-isomer (see **Table 2**).

Dermal and Ocular Irritation

Cypermethrin caused moderate dermal irritation in rabbits after a single 24-hour topical application (Coombs et al., 1976). However, in rats, dermal application caused no irritation except temporary desquamation in one individual (Henderson & Parkinson, 1980). In another study, six New Zealand White rabbits exhibited mild to moderate erythema following exposure, which resolved in most cases within 72 hours. Ocular exposure to undiluted cypermethrin resulted in mild conjunctivitis, chemosis, and conjunctival redness in rabbits, all resolving within 7 days. Rinsing the eyes post-exposure expedites recovery (Henderson & Parkinson, 1980).

Dermal Sensitization

Skin sensitization studies using the Magnusson and Kligman maximization test in guinea pigs showed weak sensitizing potential, with 2 of 20 animals exhibiting a positive response (Coombs et al., 1976). However, using the Buehler test, cypermethrin (91.5% purity, cis:trans = 53:47) was non-sensitizing in guinea pigs (Henderson & Parkinson, 1980).

Toxic Effects of Cypermethrin on Humans and Animals

Cypermethrin is a widely used synthetic pyrethroid pesticide employed in agricultural fields, households, and veterinary medicine. However, with increasing exposure, significant toxicological concerns have been raised regarding its effects on humans and animals. These include physiological disturbances, neurotoxicity, reproductive impairments, and molecular disruptions.

Effects on Humans

i. Physiological Impacts

Repeated oral administration of cypermethrin (5 and 20 mg/kg/day for 30 days) in both male and female rats resulted in a spectrum of mild to moderate toxic symptoms and behavioral changes (Grewal et al., 2010). Pathological findings include:

- Cardiac and pulmonary damage: Myocardial hemorrhages and disruption of branching structures.
- Respiratory effects: Thickening of alveolar septa.
- Reproductive organs: Degeneration of testes (loss of spermatogenic stages) and ovaries (loss of follicular cells and oocytes).

Ahmad et al., (2012) further confirmed that cypermethrin exposure in males leads to a significant decrease in testicular and epididymal sperm count. Testes showed halted spermatogenesis, connective tissue proliferation, and epididymal alterations including tailless spermatozoa and sperm-free seminal plasma.

ii. Neurological Impacts

Cypermethrin exhibits neurotoxic effects primarily by inhibiting acetylcholinesterase (AChE), disrupting antioxidant defense, and producing free radicals (Sharma et al., 2014). Key effects include:

- AChE inhibition through interaction with its anionic substrate-binding site.
- Behavioral abnormalities: Pawing, burrowing, excessive salivation, tremors, hypothermia, and reduced motor coordination.
- Neurophysiological symptoms: Neuromuscular weakness, impaired balance, lateral head movements, hypersensitivity to stimuli, and increased urination (McDaniel and Moser, 1993).

Antioxidants like resveratrol have been shown to counteract cypermethrin-induced oxidative damage and restore AChE activity.

iii. Molecular-Level Impacts

At the molecular level, cypermethrin primarily targets voltage-gated sodium channels, delaying their closure and resulting in prolonged nerve stimulation and eventual tremors or paralysis (Manna et al., 2005).

- It has minimal interaction with magnesium channels.
- Synergistic compounds like piperonylbutoxide (PBO) amplify its toxicity by inhibiting detoxifying enzymes like cytochrome P450.
- It also damages nigrostriatal brain tissue, affecting movement and cognitive functions (Kakko et al., 2003).

iv. Reproductive Toxicity

Both acute and chronic exposure to cypermethrin negatively affects the male reproductive system:

- Seminal glands exhibit increased epithelial cell height and proliferation, and inflammatory infiltration marked by mast cells (Rodriguez et al., 2009).
- Chronic toxicity leads to asymptomatic inflammation and potential infertility.
- Cypermethrin residues in food may act as endocrine disruptors, especially with prolonged exposure through agricultural produce (Mun et al., 2005).

Effects on Animals

i. Neurological and Systemic Effects

Cypermethrin targets animal nerve membranes by inhibiting closure of sodium ion channels, disrupting action potential propagation (Mun et al., 2005). Key consequences:

- Arthropods: Exhibit hyperactivity at low doses and paralysis at higher concentrations (Nasuti et al., 2007).
- Chordates: Particularly cats are highly vulnerable due to the absence of glucuronidase, which metabolizes pyrethroids.

ii. Metabolism and Sensitivity

- In most mammals, cypermethrin is metabolized rapidly by the liver into non-toxic compounds excreted via urine.
- Glucuronidase accelerates detoxification, but species lacking this enzyme (e.g., cats) are especially sensitive (Manna et al., 2005).
- Despite approval for veterinary use in dairy animals, oral ingestion of cypermethrin can still result in reproductive toxicity.

Conclusion

Pesticides continue to play a vital role in modern agriculture and public health by managing pest populations, enhancing crop productivity, and preventing vector-borne diseases. Among these, cypermethrin a type-II synthetic pyrethroid has gained widespread use due to its effectiveness and relatively low cost. It is extensively applied in agricultural fields, public health programs, households, and veterinary practices. However, the widespread and often indiscriminate use of cypermethrin raises serious concerns regarding its safety, especially with respect to human and animal health. The evidence gathered from various toxicological studies suggests that the toxic effects of cypermethrin are both dose- and time-dependent. Acute exposure may lead to neurological symptoms such as tremors, salivation, ataxia, and behavioral changes, while chronic exposure is associated with oxidative stress, immunotoxicity, reproductive dysfunctions, hepatotoxicity, and even carcinogenic risks in some cases. Cypermethrin is absorbed through oral, dermal, and inhalation routes, with the oral route being particularly significant for humans, especially those working in agriculture and pest control without adequate protective measures. Though animals may exhibit lower dermal sensitivity, systemic toxicity still occurs with prolonged exposure. The skin of certain animals may act as a partial barrier, but once absorbed, cypermethrin is capable of bioaccumulation and subsequent physiological disruption. This highlights the compound's potential to affect non-target organisms, including domestic animals and wildlife, which may be exposed through environmental contamination. Moreover, environmental persistence and bioaccumulative properties of cypermethrin contribute to its ecological impact, particularly in aquatic ecosystems where it is highly toxic to fish and invertebrates.

This underlines the need for environmentally sound disposal practices and regulatory enforcement. Given the gravity of the toxicological profile of cypermethrin, there is a pressing need to educate stakeholders particularly farmers, pest control workers, and household users about the associated risks. Implementing proper handling procedures, usage guidelines, and promoting the adoption of personal protective equipment (PPE) are critical steps in reducing exposure. Regulatory agencies should also strengthen monitoring systems and consider phasing in safer alternatives or integrated pest management (IPM) strategies where possible. In summary, while cypermethrin remains an indispensable tool in pest management, its use must be critically evaluated against its potential to harm humans, animals, and the environment. Proactive steps such as public education, policy implementation, and development of safer alternatives are necessary to mitigate its adverse effects. Continued research is essential to further elucidate its mechanisms of toxicity, establish safer exposure thresholds, and inform evidence-based regulations for sustainable and responsible pesticide use.■

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