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Pyrethroid Insecticides: Use, Environmental Fate, and Ecotoxicology

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1. Introduction

There was little public concern over potential nontarget effects of pesticides until the 1962 publication of Rachel Carson's *Silent Spring* (Delaplane, 2000; Moore et al., 2009). Organochlorine insecticides (such as DDT) had been used intensively and were credited for many of the problems highlighted by Carson. Because of their accumulation in the environment and reproductive toxicity in people, their use was eventually phased out in most industrialized countries in the late 1970s and early 1980s (Moore et al., 2009). Organophosphate (OPs) and carbamate pesticides (based on the same chemistry and mode of action as nerve gas used by the military) took the place of the organochlorine products and have been widely used since the late 1970s.

For the past three decades, OP pesticides have been the insecticides most commonly used by professional pest-control bodies and homeowners (Feo et al., 2010). However, in the late 1980s, OPs in California came under scrutiny when they began to show up in groundwater samples. In addition, because of their potentially toxic effects on people, The U.S. Environmental Protection Agency (EPA) considered a ban on both OP and carbamate insecticides as part of the Food Quality Protection Act (FQPA) of 1996. The FQPA mandated that the EPA reassess all pesticide tolerance levels considering aggregate exposures, cumulative effects from pesticides sharing common mechanisms of toxicity, and special protection for infants and children (Metcalfe et al. 2002). During the early 2000s, the EPA began phasing out residential uses of the two primary OPs, diazinon, and chlorpyrifos (CDPR 2008). EPA's decision to eliminate certain uses of the OP insecticides because of their potential for causing toxicity in people, especially children, has led to their gradual replacement with another class of insecticides, the pyrethroids (Oros & Werner, 2005).

2. Use, chemistry and mode-of action of pyrethroids

2.1 Brief history of pyrethroids

Pyrethroids are synthetic analogues of pyrethrins, insecticidal substances obtained from the flowers of a species of chrysanthemum (*Chrysanthemum cinerariaefolium*). The valuable insecticidal properties of pyrethrum (a mixture of pyrethrins, cinerins, and jasmolins) were recognized in the 19th century, and their properties stimulated detailed examination of the chemical constitution of the active esters in the first quarter of the 20th century (Elliot, 1977; Ecobichon, 1996). Chemical research was conducted initially by British, Japanese and French

scientists to find more stable and effective compounds with a mammalian toxicity as low as that of the model Pyrethrin I (Elliott et al. 1979, Pap et al. 1996). Although the acid moieties of the esters were correctly identified early on, only in 1947 were the structures of the alcohols settled. The first synthetic pyrethroid, allethrin, was synthesized in 1949, and marketed in 1952 for the control of household insects (Elliot, 1977; 1980a)

The first set of synthetic compounds containing chrysanthemic acid showed only moderately higher insecticidal potency than that of Pyrethrin I, but they proved to be potent knockdown agents. Pyrethroids and pyrethrins affect nerve impulse transmission in insects mainly through their action on voltage-sensitive sodium channels (Soderlund & Bloomquist, 1989). Efficacy was increased by changing the alcoholic component to the 5-benzy-3-furylmethyl moiety, resulting in tetramethrin, which was first synthesized in 1964 and marketed in 1965 for indoor pest control; and resmethrin, first produced in 1967 and marketed in 1969 for control of household and public health insects (WHO, 1989a). These were the first synthetic compounds with good knockdown activity, greater insecticidal activity and lower mammalian toxicity than the natural esters (Elliot, 1977). Esterifying (1RS, *cis,trans*)-2,2-dimethyl-3-(2,2-dimethylvinyl)cyclopropanecarboxylic acid (chrysanthemic acid) with 3-phenoxybenzyl alcohol resulted in a relatively cheap molecule and further improved insecticide activity, leading to phenothrin in 1969, which has been in commercial use since 1977 to control insects in the home and for public health, and to protect stored grain (Pap et al., 1996; WHO, 1990a). Halogenation of the vinyl group on the cyclopropane ring led to permethrin, which was synthesized in 1973 and produced the photostability required for field use. Permethrin was marketed beginning in 1977 mostly for agricultural purposes, but also for body lice, mosquito nets and other household uses (Pap et al., 1996; WHO, 1990a). Fenvalerate and deltamethrin came on the market in 1976 and 1977, respectively, for use on cotton and other crops, and also for control of cattle insects. In 1974, the introduction of the alpha-cyano group resulted in cypermethrin, which was initially marketed in 1977 as having exceptionally high efficacy against pests in the household, in agriculture, public health, and animal husbandry (WHO, 1989b; Pap et al, 1996). Many second generation pyrethroids (e.g., bifenthrin, cyfluthrin, lambda cyhalothrin) became available in the 1980s (Weston et al., 2004). Original patents expired in the early 1990s, allowing many other manufacturers to offer new active ingredients by the mid-1990s (Pap et al., 1996).

2.2 Pyrethroid mode of action and chemical properties

Unlike the OP insecticides that act on the central nervous system of exposed animals, pyrethroid insecticides disrupt the normal function of the peripheral nervous system. Pyrethroids react with voltage-gated sodium channels on nerves, prolonging the time during which the channels are open. This results in altered nerve function, which manifests either as a series of short bursts or a prolonged burst, and is caused by repetitive discharge of nerve signals or stimulus-dependent nerve depolarization. In general, exposure to toxic doses of these compounds causes incoordination, convulsions, and paralysis (Soderlund & Bloomquist, 1989). Based on observed symptoms and mode-of-action on sodium channels, pyrethroid insecticides can be further categorized into separate classes, type I and type II. Type I pyrethroids, which include allethrin, bifenthrin, d-phenothrin, permethrin, resmethrin and tetramethrin, cause restlessness, hyperexcitation, prostration, and body tremors; Type I pyrethroids produce repetitive nerve discharges. Conversely, exposure to Type II pyrethroids, such as cyhalothrin, cypermethrin, cyfluthrin, deltamethrin,

esfenvalerate, fenvalerate, and fluvalinate, and lambda-cyhalothrin, results in hyperactivity, incoordination, convulsions and writhing; Type II pyrethroids produce stimulus-dependent nerve depolarization and blockage (Ecobichon, 1996: Soderlund & Bloomquist, 1989). This distinction was primarily derived using mammalian studies, and although exposed insects also exhibit different symptoms between the two types, these are not so distinct (Stenerson, 2004).

Also in contrast to OP insecticides, pyrethroids exhibit low toxicity to mammals and birds, while also demonstrating strong selectivity for insects and invertebrates (Ecobichon, 1996; Fishel, 2005). Chronic animal feeding studies have produced high no-effect levels, indicating both a low potential to bioaccumulate and proficient detoxicification in mammalian receptors. In addition, although these compounds are categorized as highly toxic to very highly toxic to nontarget fish and invertebrate species, they are reportedly practically non-toxic to birds (Fishel, 2005). In addition to low avian and mammalian toxicity, physical properties of pyrethroids also are an improvement over their predecessors. The low vapor pressures and high octanol-water coefficients of pyrethroids indicate a low propensity to volatilize and a high affinity for organic matter, soils and clay (Oros & Werner, 2005). Sorption to particulates reduces bioavailability to nontarget organisms, and also makes it more likely that these insecticides will be retained at the application site.

2.3 Current uses of pyrethroids

Synthetic pyrethroid insecticides have been used for more than 20 years to control insect pests in a variety of crops (Maund et al., 2001), but they have become increasingly popular following outright bans or limitations on the use of cholinesterase-inhibiting insecticides (Luo & Zhang, 2011; Feo et al., 2010). In 2000-2001, EPA withdrew the residential registrations for two commonly applied OP pesticides, chlorpyrifos and diazinon (EPA 2000; 2001), resulting in a significant increase in the market penetration of the pyrethroid products. The pyrethroids have a high share of the insecticide market because their activity profile indicates high efficiency, wide spectrum, low mammalian and avian toxicity and biodegradability (Pap et al., 1996). Today, pyrethroids are used in agriculture, forestry, horticulture, public health (i.e., hospitals) and are active ingredients of many insect-control products intended for indoor home use (Feo et al., 2010).

Pyrethrins and synthetic pyrethroids are also considered safe by the World Health Organization (WHO) for use in aircraft for vector control (Rayman, 2006). WHO also recommends certain pyrethroids (bifenthrin, cyfluthrin, deltamethrin, lambda-cyhalothrin) for indoor residual spraying against malaria vectors (Walker, 2000; Raghavendra et al., 2011). In addition, WHO recommends treating bed nets with pyrethroids, as a crucial role in vector-based malaria intervention strategies (Raghavendra et al., 2011; WHOPES, 2005). Pyrethroids are also commonly used in developing countries, such as China, for pest control and disease vector eradication (Chen et al., 2011). Because pyrethroids are considered safer alternatives to OP pesticides, they assumed many roles formerly held by OPs, particularly for pest control in urban environments (Amweg et al., 2006). Pyrethroids are now widely used by professional pest control applicators as termiticides, for landscape application, or as perimeter treatments to keep pests out of structures. In addition, they are the dominant insecticides among retail sales to consumers (Weston et al., 2009). According to EPA usage data, approximately 1 million kg of permethrin are applied annually to agricultural, residential, structural and public health sites (Feo et al., 2010). Of this, more than 630,000 kg

are annually applied in nonagricultural settings, with 41% of applications made by homeowners on residential areas (EPA, 2006; Moore et al., 2009).

2.4 Pyrethroid use patterns

Databases that provide information on use patterns of pyrethroids are not readily available to the public in the United States. One exception is the California Department of Pesticide Regulation's (CDPR) Pesticide Use Reporting (PUR) database. CDPR began full use reporting of all agricultural pesticide applications in 1990 (CDPR, 2000). The program requires monthly reporting of any agricultural pesticide use to the county agricultural commissioners, who transfer the information to PUR. Data obtained from the PUR database suggest that in 2008 (the most recent year for which all data are available), the amount of pyrethroids professionally applied in non-agricultural situations in California was greater than for agricultural areas. A few pyrethroids, such as bifenthrin, fenpropathrin, and lambda-cyhalothrin, are used more frequently in agricultural settings. Overall, however, larger amounts of pyrethroids are used in non-agricultural settings than agricultural settings.

Using the same data from the PUR database reveals that landscape maintenance, public health pest control, and structural pest control are the three applications where pyrethroids were used most often in 2008. Data describing the non-agricultural uses of pyrethroids can be further classified into either "indoor," "outdoor," or "structural" use categories. The term "indoor uses" relates to disease control for humans and pets in buildings and homes. Pest control related to landscaping needs is covered by "outdoor uses". The "structural uses" category refers to pyrethroids being used to prevent structural damage to structures such as housing or commercial development from pests such as carpenter ants and termites. The amount of pyrethroids reported in the PUR database for each of these three use categories was summed, and the results suggested that "structural" use was greater than "outdoor" use, and both structural and outdoor uses of pyrethroids were two orders of magnitude greater than "indoor" uses of pyrethroids.

2.5 Emerging concerns

Pesticide resistance is the adaptation of pest populations targeted by a pesticide resulting in decreased susceptibility to that chemical. Over the past decade, a resurgence of bed bugs (*Cimex lectularius*) has occurred in North America, Europe, and Australia, with infestations now being common occurrences (Romero et al., 2007). Romero et al. (2007) suggested that the difficulties associated with controlling bed bug infestations, and the survival of field-collected bed bugs after direct spray applications with label-rate formulated pyrethroids including deltamethrin and lambda-cyhalothrin, indicates that bed bug resistance to pyrethroid insecticides may be widespread. Malaria vector control is dependent on pyrethroid insecticides, as this is the only class of chemicals approved for use on insecticide-treated nettings. However, there has been a dramatic increase in reports of pyrethroid resistance in malaria vectors over the past decade; the resistance alleles are spreading at an exceptionally rapid rate throughout Africa (Ranson et al., 2011).

Synthetic pyrethroid insecticides have generated public concerns due to their increasing use and potential effects on aquatic ecosystems (Luo & Zhang, 2011). In recent studies, residues of pyrethroid insecticides have been detected in the sediment from a number of urban streams in California at levels toxic to *Hyalella azteca* (Weston et al., 2005; Amweg et al., 2006;

Oki et al., 2007; Budd et al., 2007; Weston & Lydy, 2010). Although pyrethroids offer lower toxicity to human applicators and nontarget mammals and birds than OPs, they are highly toxic to invertebrates and fish (Palmquist et al. 2010). This lead the CDPR, which regulates pesticide sales and use in California, to initiate a re-evaluation of more than 600 products containing pyrethroid pesticides in 2006 (CDPR, 2007).

The increasing and widening use of pyrethroid insecticides necessitates a thorough understanding of the environmental fate and ecological effects of their use. In many ways, these insecticides represent a significant improvement over other insecticide classes, exhibiting lower mammalian and avian toxicity and better selectivity to target species than the OPs and less persistence than the organochlorine insecticides. Additionally, the unique mode-of-action and chemical behavior of pyrethroids suggests that ecotoxicological effects and fate in the terrestrial and aquatic environment are likely to be significantly different from that of their OP precursors. Subsequently, a review of the current literature regarding environmental fate and bioavailability of the pyrethroid insecticides and their ecotoxicity, with special reference to sublethal and ecological effects is presented.

3. Environmental fate in the terrestrial environment

Pyrethroid releases into the terrestrial environment occur largely via spray drift from both agricultural and non-agricultural applications, although accidental spills and direct application to soil surfaces can also be considered sources of release. However, because of their strong tendency to adsorb to soils and organic matter, these compounds are unlikely to undergo significant migration from areas of direct application, except on particulates that are carried by wind or water.

Pyrethroid persistence under aerobic soil conditions (e.g., expected field conditions) is highly variable, with half lives ranging from 11.5 days for cyfluthrin to 96.3 days for bifenthrin (Oros & Werner, 2005). Photolysis is likely a significant degradation pathway for pyrethroids in the soil, and is influenced by soil characteristics. The half life of esfenvalerate in different soil systems was significantly increased under dark conditions, with half-lives of 7.8 to 100.0 days under continuous irradiation versus 150.0 to 553.4 days in the dark (Katagi, 1991). Analysis of soils indicated that esfenvalerate was largely present in complexes with humic acid. Further, the fraction of fenvalerate bound to soil particles was greater in silty clay loam soil as compared to sandy loam soil (Lee, 1985). Consequently, it is probable that photolytic degradation of pyrethroids could proceed more slowly in highly organic soils.

Microbial degradation also appears to be a significant breakdown route. Degradation of pyrethroid insecticides was observed to be more rapid in natural versus sterilized soils, indicating that biological processes do contribute to breakdown in soil. Eight weeks after treatment, fenpropanate, cypermethrin, and permethrin concentrations were reduced to 20% of the original amount in natural mineral and organic soils, while sterilized systems retained more than 80% of initial concentrations (Chapman et al., 1981). Authors concluded that soil microorganisms were largely responsible for the more rapid rate of degradation in natural soils. There is some evidence that the byproducts of pyrethroid degradation are more mobile in soils than are the parent compounds (Kaufman 1981, Lee 1985). However, these compounds are likely to be significantly less toxic than the parent insecticide.

Soil concentrations of pyrethroids may be reduced as a result of interception by the plant canopy. For instance, the residues of cypermethrin in agricultural field soils under crop cover were determined to be approximately one-tenth of those in bare soil following a spray

event (Wiles & Frampton, 1996). Subsequently, a large fraction of spray drift may become associated with plant surfaces and undergo different degradation processes. The observed half life of cypermethrin on elm bark was approximately 50 days, and resulted in improved efficacy against bark beetles, reducing the need for additional applications. Wiles & Jepson (1994) also determined that pyrethroid residues are more persistent on leaves, and that plant-dwelling insects may therefore be exposed to pyrethroid residues longer than soil-dwelling invertebrates.

4. Effects on terrestrial invertebrates

Potential terrestrial invertebrate pyrethroid receptors can be grouped in two categories—pest invertebrates and nontarget beneficial species that include pollinators and predaceous insects. Applications to pest invertebrates are intentional and designed to result in maximum mortality of target organisms; exposures to nontarget organisms, however, are incidental and largely unintentional. In both cases, this review will focus on the sublethal effects observed following pyrethroid exposure, as sublethal exposures are much more common under environmentally-relevant exposure scenarios than are lethal concentrations.

4.1 Pest invertebrates

Pyrethroids provide excellent control of pest invertebrates as a result of residual activity, strong repellency, and high toxicity (Hammond, 1996). For example, when compared with OP insecticides, pyrethroids provide more effective control of the apple pest, the tufted bud moth (*Platynota idaeusalis)* (Hull et al., 1985). Hall (1979) reported that pyrethroids provided equivalent or superior control of plum curculio (*Conotrachelus nenuphar*), codling moth (*Cydia pomonella*) and redbanded leafroller (*Argyrotaenia velutinana*) when compared with OPs.

Sublethal pyrethroid exposure has been shown to interfere with both physiological growth processes and the ability to feed in insect pests. Sublethal doses of d-phenothrin (5 ng/day) were linked to significant reductions in lipid synthesis in the assassin bug (*Triatoma infestans*) (Juarez, 1995), indicating that processes critical to insect growth were disrupted by pyrethroid exposure. Similarly, exposure to sublethal doses of deltamethrin altered glucose metabolism in the migratory locust, with significant increases in the conversion of glucose to carbon dioxide, potentially as a result of metabolic hormone release following stimulation of neurohemal organs by pyrethroid exposure (Moreau et al., 1987). Food acquisition also is affected by consumption of pyrethroid residues. Dietary exposure to synthetic pyrethroids including cypermethrin, deltamethrin, fenvalerate and permethrin decreased feeding rates in the red flour beetle (*Tribolium castaneum*), a pest of stored grain, which resulted in reduced larval weight, delayed pupation and delayed adult emergence (Ishaaya et al., 1983). Together, these results suggest that non-lethal pyrethroid exposures can significantly alter normal invertebrate growth processes.

In addition to interfering with growth processes, pyrethroid exposure has also been linked to disruption of invertebrate chemical communication. Adult tobacco hornworm (*Manduca sexta*) females exposed to permethrin were less likely to attract males than were non-exposed females (Haynes, 1988). Permethrin also reduced male oriental fruit moth (*Grapholita molesta*) response to pheromones in a wind tunnel (Haynes, 1988). Similarly, permethrin was found to interfere with the chemical communication of male and female

pink bollworm moth (*Pectinophora gossypiella*) (Haynes and Baker, 1985). In both males and females, disruption of sex pheromone communication was a more sensitive endpoint than disruption of flight abilities.

There is also evidence that pyrethroid exposure can reduce the number and viability of eggs produced by pest invertebrates. Egg development of engorged female African ticks (*Amblyomma hebraeum*) was significantly altered by cypermethrin-exposure. Doses as low as 0.05 µg cypermethrin per tick reduced average ovary weight, average oocyte length, and ovary vitellogenin (Friesen and Kaufman, 2003). Likewise, deltamethrin also reduced the production of both ootheca and viable offspring by German cockroaches (*Blattella germanica*) (Lee et al., 1998). Longevity of exposed male and female roaches was significantly reduced by deltamethrin exposure, an effect that is likely to further reduce fecundity.

4.2 Beneficial invertebrate species

Application of pyrethroids to agricultural fields can result in the unintentional exposure of beneficial invertebrate species, such as pollinators, parasitoid wasps, generalist predators, and soil decomposers, as a result of their proximity to sprayed areas. Although such species may be exposed to acutely lethal concentrations of insecticides, it is far more probable that the majority of nontarget invertebrates will encounter sublethal concentrations. These levels of exposure can impact foraging and prey-location behaviors, development, growth, and reproduction.

4.2.1 Honeybees

As domesticated pollinators, honeybees (*Apis mellifera*) are common residents of agricultural areas. Despite measures to prevent their direct exposure to pesticides (e.g., forgoing pesticide application during peak blooming and bee foraging times), this proximity to sprayed areas increases the probability of both cuticular and dietary exposure of honeybees to insecticides, including pyrethroids. Pyrethroid insecticides, in particular, have been demonstrated to interfere with important honeybee behaviors and with reproductive output.

Low exposures of permethrin alter honeybee behavior associated with maintenance, feeding, and communication. Bees receiving topical applications of 0.001 µg permethrin were more likely than non-exposed bees to engage in self-cleaning, leg-rubbing, trembling dances, abdomen tucking and rotation (Cox & Wilson, 1984). Permethrin-intoxicated bees also spent less time walking, giving food, and antennae touching. Similarly, forager honeybees exposed to 2.5 ng deltamethrin/ bee (27 times less than the LC_{50}) exhibited altered flight patterns and homing abilities as compared to non-exposed bees (Vandame et al., 1995). While approximately 90% of control bees returned to the hive within 30 seconds of flight, only 9.0% of treated bees were able to return within this time period. Authors also noted that this delay was most likely related to bees' inability to correctly orient themselves using the sun after being exposed to deltamethrin.

Dietary exposures to sublethal pyrethroid concentrations (i.e., as in nectar or syrup) also cause behavioral aberrations, and decrease fecundity. Feeding on syrup containing 940 µg/L deltamethrin reduced the proportion of bees exhibiting learned orientation towards an odor stimulus by approximately 11% to 24% (Decourtye et al., 2005). Consumption of diet containing bifenthrin or deltamethrin at concentrations of 4.0, 7.9, 15.5, 30.6, and 60.2 mg/L or 20.0, 36.0, 64.8, 116.6, and 210.0 mg/L, respectively, also caused sublethal effects in

honeybees. Daily egg production was reduced by consumption of both bifenthrin and deltamethrin, and time in the egg stage was extended. Deltamethrin exposure also reduced capping rate and extended the duration of the immature stage (Dai et al., 2010). Other pyrethroid effects occurred sporadically, as effects on rate of development, number of feeding larvae (unsealed brood stage), hatch rate, and number of post-feeding larvae and pupae (sealed brood stage), were not significant in every year tested.

4.2.2 Parasitoids

Like honeybees, parasitoid wasps are beneficial hymenopterans that frequent agricultural fields or other commonly sprayed areas. These insects use many common agricultural pests as "nests" for their developing offspring, laying their eggs inside aphids, larval moths, and beetles. After hatching, the parasitoid larvae consume the host from the inside, eventually causing death. The species-specific nature of the parasitoid-host relationship increases their economic benefit: whereas generalist predators may consume non-pest as well as pest prey items, parasitoids are host-specific, thereby providing better control of targeted pest species. However, exposure to sublethal concentrations of pyrethroids has been observed to interfere with parasitoid host-finding behaviors, reaction to mating pheromones, and reproduction.

Olfactory orientation toward prey populations was reduced in aphid parasitoids following exposure to sublethal concentrations of lambda-cyhalothrin (Desneux et al., 2007). Similarly, the addition of deltamethrin to honeydew residues also caused significant parasitoid repellency (Longley & Jepson, 1996). In addition to altering the ability of parasitoids to locate their hosts, deltamethrin exposure at 10% of the LD_{50} also altered sex pheromone production by the female moth egg parasitoid *Trichogramma brassicae*; males localized less precisely to pheromone extracts from exposed females than from unexposed females (Delpuech et al., 1999).

Although an inability to locate hosts or mates can cause a reduction in parasitoid fecundity, there is evidence that pyrethroid exposure can also alter other aspects of parasitoid reproduction. When exposed to lambda-cyhalothrin prior to oogenesis, the Trichogramma wasp (*T. pretiosum*) experienced a reduction in fecundity (Desneux et al., 2007). Interestingly, sublethal deltamethrin and lambda-cyhalothrin exposures resulted in decreased number of female offspring and altered male:female ratios in exposed wheat aphid parasitoids (*Aphidius uzbekistanicus*). Authors noted that in this hymenopteran species, females are produced by fertilized eggs, while unfertilized eggs produce males, and that the behaviors associated with egg fertilization may have been impacted by the insecticide exposure (Desneux et al., 2007). However, data from field studies indicate that sublethal effects of pyrethroid exposure may not have large-scale impacts on overall parasitoid population viability (Li et al., 1992, Longley, 1999).

4.2.3 Other beneficial invertebrate species

Similar effects on insect behavior and feeding have been demonstrated in generalist predator species. Increased grooming has been observed in ladybird beetles (*Coccinella septempunctata*) following sublethal pyrethroid exposure. When released in areas previously sprayed with deltamethrin, these insects were observed to groom and walk more frequently (Desneux et al., 2007). Further, the prey attack rate of the 'sit-and-wait' assassin bug (*Acanthaspis pedestris*) was reduced by a factor of 2.4 to 6.4 following exposure to cypermethrin at suggested field application rates (Desneux et al., 2007). Pyrethroid exposure of prey items can also cause anti-feeding responses in invertebrate predators. Predatory

carabid beetles (*Nebria brevicollis*) were more likely to regurgitate deltamethrin-exposed aphids as compared to untreated prey. Exposure to sublethal concentrations of permethrin decreased the developmental time of female stink bugs (*Supputius cincticeps*), but extended that of the males (Desneux et al., 2007).

However, field cypermethrin soil residues were found to cause less than 10% mortality to Collembola in laboratory studies, and this insecticide was less toxic than chlorpyrifos, an OP insecticide, to Collembola (Wiles & Frampton, 1996). Toxicities of leaf and soil deltamethrin residues to beneficial insect species, including predatory beetles and parasitoids, were determined to be different, with leaf residues exhibiting higher toxicity. The LD_{50} values for ground beetles (*Demetrias atricapillus)*, rove beetles (*Tachyporus hypnorum)*, ladybird beetles (larvae and adults), hoverflies (*Episyrphus balteatus)*, and brachonid wasps (*Aphidius rhopalosiphi)* exposed to deltamethrin residues on leaf surfaces were >50, 1.2, 0.4, 2.0, 4.8 and 7.1 g a.i./ha, respectively, (Wiles & Jepson, 1994). Tested species appeared less susceptible to soil residues. These results indicate that plant-active predators are likely to be more sensitive to pyrethroid applications than are soil-dwelling predatory insects.

5. Effects on terrestrial vertebrates

One of the primary benefits of the use of pyrethroids is the compounds' low inherent toxicity to a number of nontarget terrestrial vertebrates, including mammals and birds. Given this, a discussion of avian and mammalian pyrethroid toxicity is unnecessary in the context of environmental exposures, as use rates are low enough to preclude any effects on these receptors. However, less is known regarding the toxicity of pyrethroids to terrestrial reptilian species.

Brown tree snakes (*Boiga irregularis*) were sensitive to the effects of commercially available pyrethroid insecticide formulations applied dermally as a 2-second spray. Within one hour of exposure, intoxicated snakes exhibited muscle tremors, disorientation, and eventual paralysis, followed by death (Brooks et al., 1998). Unsurprisingly, the pyrethroid synergist piperonyl butoxide was observed to enhance the toxicity of pyrethroid formulation to snakes, and a 2-second spray application delivered a high amount of active ingredient (102 to 320 mg/kg bw). Sensitivity to pyrethins was determined to be temperature-dependent in green anole lizards (*Anolis carolinensis*), a phenomenon also observed in exposed invertebrates. Lizard mortality following a 2-second bath in 300 mg /L pyrethrin solution was 30% at 38°, but increased to 100% at temperatures below 20°C (Talent, 2005). These results may indicate an increased reptilian susceptibility of pyrethroids in cooler temperate climates.

6. Effects on terrestrial communities

Little research has been conducted concerning the effects of pyrethroids on nontarget terrestrial communities. Because bird and mammals species are largely unaffected by pyrethroids, it may be that the impacts upon terrestrial ecosystems are limited to invertebrate communities. In fact, Ali et al. (2011) report that adverse effects of pyrethroid use on bird populations almost always occurs through a destruction of the food supply of insectivorous birds. Hence, effects in the vertebrate community will most likely only be observed following extremely heavy and widespread uses of these compounds that would result in the near-obliteration of resident invertebrates. Further, while the effects of

pyrethroids on nontarget invertebrate fitness have been extensively investigated, most studies focus on the effects to specific beneficial species; fewer studies have been conducted concerning the effects upon field invertebrate communities.

Although effects on nontarget soil arthropod communities are likely to be minimal and transient, pyrethroid impacts on plant-dwelling insect communities are more significant. Cypermethrin applied at levels sufficient to control aphids (25 g a.i./ha) also suppressed numbers of predaceous carabid beetle populations for 6 days post-spray. However, the pyrethroid had no effects on staphliniid beetles and only minor effects on predatory flies and parasitic wasps (Inglesfield, 1989). Applications of deltamethrin at 6.23 g a.i./ha similarly reduced ladybird beetle populations in wheat crops, but had no impact on fly or parasitic wasp species. Communities in areas adjacent to sprayed fields may also come into contact with pyrethroids in the form of spray drift. The survival of the aphid parasitoid *Aphidius colemani* in edge-of-field areas was impacted by spray drift from two applications of the lamba-cyhalothrin formulation Trafo® at the recommended rate of 10 g a.i./ha (Langhof et al., 2003). Proportions of hoverfly (Syrphidae) eggs to aphids and percentages of aphid mummies increased with distance from edge-of-field, although these trends were not significant. Authors concluded this was likely a result of the non-homogenous distribution of lamba-cyhalothrin spray drift residues (Langhof et al., 2003). Subsequently, effects observed in edge-of-field areas probably will not be consistent between different fields, as the variables affecting the magnitude and distance of insecticide drift are highly site-specific.

7. Environmental fate and bioavailability in aquatic systems

Pyrethroids are most commonly introduced into aquatic systems via runoff from sprayed fields, lawns, parking lots, etc., during rainstorm events, and, to a lesser extent though spray drift. Regression modeling of pesticide loading in the Sacramento River indicated that the frequency and magnitude of pyrethroid use as well as precipitation patterns are critical factors that govern pyrethroid transport to surface waters (Oros & Werner, 2005). However, field-specific variables determining breakdown rates (e.g. canopy cover, temperature, persistence on soil surface) in relation to precipitation events also play a role in determining pyrethroid concentration in runoff. It has also been proposed that transportation through concrete drainage systems that are present in suburban and urban areas, may result in the transport of greater concentrations of aqueous-phase pyrethroids, as compared with particulate-rich agricultural runoff channeled through earthen ditches (Weston & Lydy, 2010).

7.1 Fate of pyrethroids in aquatic systems

Pyrethroid insecticides are strongly hydrophobic; the log K_{ow} values for 11 different pyrethroid compounds ranged from 4 for esfenvalerate to 7.6 for tralomethrin (Oros & Werner, 2005). As such, the water-soluble fraction of pyrethroids introduced into an aquatic system will be short-lived and quickly reduced. A mesocosm study of the effects of lambda-cyhalothrin spray drift indicated that the pyrethroid insecticide rapidly dissipated from the water column (half life of approximately one day), leading authors to theorize that aqueous pyrethroid exposure would cause only short-term community stress (Arts et al., 2006). Subsequently, much of the fate and transport of pyrethroids in aquatic systems is governed by particulate adsorption.

Pyrethroid transport within aquatic systems occurs through movement of pyrethroid-absorbed fine particulates (Gan et al., 2005). Although the half-lives of most pyrethroid insecticides are in the order of days to weeks in the water column, pyrethroids adsorbed to particulates are considerably more persistent, with reported half-lives on sediments of 150 to 200 days (Amweg et al., 2005). Pyrethroids in stream water were most frequently associated with suspended solids and particulates, with only 0.4% to 1.0% of added pyrethroids present in the freely dissolved phrase. In runoff, the freely dissolved phase accounted for 10% to 27% of the total pyrethroid mass (Liu et al., 2004). Following pyrethroid addition to stream water, greater than 97% of the total mass added was sorbed to suspended solids and particulates.

Ditches receiving agricultural runoff mitigated bifenthrin and lambda-cyhalothrin contamination by promoting their retention and adsorption. Although runoff initially contained 666 µg/L bifenthrin and 374 µg/L lambda-cyhalothrin, this was reduced to 7.24 and 5.23 µg/L, respectively, 200 m downstream. At 400 m, no pyrethroid residues were detected (Bennett et al., 2005). In general, pyrethroid concentrations in sediment increased with distance along a drainage channel downstream from a sedimentation pond. While bifenthrin sediment concentrations in the pond averaged 0.33 mg/kg, these increased to 2.27 mg/kg 104 m downstream and 10.64 mg/kg at 145 m downstream. A similar pattern was observed for the distribution of *cis*-permethrin, with sedimentation pond concentrations of 0.77 mg/kg increasing to 1.1 and 4.45 mg/kg at 104 and 145 m downstream, respectively (Gan et al., 2005). Enrichment of pyrethroid concentrations downstream from a sedimentation pond correlated to increasing organic carbon and clay content of the sediment. As pyrethroids tightly adhere to fine organic particulates, it is probable that pyrethroid-bound fines were more likely to be transported by downstream flow, resulting in downstream enrichment.

Concentrations of cypermethrin, deltamethrin, fenvalerate, and permethrin in the interstitial (pore) water of sandy sediment were five to six times as high as concentrations measured in the overlying water. However, the differences between pyrethroid concentrations in overlying and pore water in silt or clay sediments were considerably lower, a 1.3 to 1.5-fold difference (Muir et al., 1985). Consequently, benthic and epibenthic invertebrates in systems with clay or silt sediments are likely to experience similar pyrethroid exposures. However, exposure of benthic organisms in sandy substrates may not be greater despite the higher total concentration of pyrethroids a result of chemical adsorption to the larger particles; the mass adhered to sand is likely not as bioavailable to sediment-dwelling invertebrates (You et al., 2008).

7.2 Bioavailability of pyrethroids in aquatic systems

Although pyrethroids exhibit strong hydrophobicity and therefore would be expected to bioaccumulate in aquatic organisms (and even to biomagnify in the food chain), they are rapidly depurated, and consequently do not bioaccumulate (Oros & Werner, 2005). Moreover, the hydrophobic nature of these chemicals results in binding to dissolved organic carbon (DOC), which removes them from the aqueous phase of the water column and causes them to bind to many sediments, thus reducing their bioavailability in aquatic systems.

Addition of DOC to aqueous laboratory systems reduced the bioaccumulation and toxicity of deltamethrin, fenvalerate, and cyhalothrin in the cladoceran *Daphnia magna*. Low DOC

concentrations (1.76 mg/L) sequestered up to 76.4% of added fenvalerate and 80.8% of added deltamethrin after 24 hours (Day 1991). Similarly, exposure to lambda-cyhalothrin in a water-sediment system reduced toxicity to *Daphnia magna* and carp (*Cyprinus carpio*) by factors of 175 and 74, respectively, over that observed in water-only exposure scenarios (Maund et al., 1998). This indicates that laboratory studies are likely to overestimate the toxicity of pyrethroids to field populations. Following introduction into a water-sediment system, nearly 99% of cypermethrin was adsorbed to sediment within the first 24 hours, and binding to sediments reduced the bioavailability of the insecticide to *Daphnia magna* and *Chironomus tentans* (Muir et al., 1985).

The bioavailability of pyrethroids to sediment-dwelling species is commonly understood to be largely influenced by the sediment organic carbon (OC) content. Consequently, sediment pyrethroid concentrations are frequently OC-normalized. However, bioavailability of pyrethroids to benthic invertebrates also is influenced by the presence of leaf and plant material. Toxicity of bifenthrin to *Hyalella azteca* was reduced by the addition of leaf material to sediments, even when normalized to OC content. Authors noted that this amphipod preferentially inhabits leaf material, and that this association may reduce exposure to the pyrethroid (Maul et al., 2008b).

In addition to the quantity of OC in sediments, the type of the OC (e.g., in terms of the number of binding sites and general affinity for pyrethroids) is an important determinant of bioavailability. Subsequently, differently sourced sediments can exhibit different adsorptive capacities for pyrethroids despite having the same OC content. Amweg et al. (2005) collected sediments from several different field sites in the Central Valley of California to determine the pyrethroid-mediated toxicity to *Hyalella azteca* in a series of laboratory bioassays. Despite normalizing the pyrethroid LOAEC and LC_{50} values to organic carbon content, the toxicity threshold was different for different sources (Amweg et al., 2005). Similarly, the toxicity of permethrin to chironomid larvae in artificial sediments was greater than in naturally-derived sediments. In natural systems containing 200 ng/g permethrin, chironomid emergence was reduced to approximately 80% of controls, while in artificial peat and α-cellulose sediment, midge emergence at the same pyrethroid concentration was approximately 62% and 8%, respectively (Fleming et al., 1998). In addition, toxicity of deltamethrin to *Chironomus riparius* larvae in spiked sediment systems was reduced by use of natural sediments versus artificial sediment. This difference was attributed to the higher organic content in the natural sediment (12.5%, as opposed to 4.8% in artificial sediment). Additionally, during the 10-day bioassay, 50% of the added deltamethrin was degraded in the natural sediment system, while no degradation was observed in the artificial sediment. However, analysis of sediment pyrethroid concentrations indicated that lower toxicity in natural sediments did not result from rapid degradation, but from reduced bioavailability (Åkerblom et al., 2008). Survival of *Chironomus riparius* larvae in permethrin-spiked sediments was greater in peat sediments than in α-cellulose sediments, likely due to different binding capacities and affinities for the pyrethroid between the peat and α-cellulose (Fleming et al., 1998). Subsequently, pyrethroid bioavailability may be highly site-specific and dependant on OC source as well as content.

8. Aquatic ecotoxicity of pyrethroids

Although reasonably non-toxic to avian and mammalian receptors, pyrethroids exhibit very high toxicity to nontarget invertebrates and fish, making them vulnerable to low

concentrations of pyrethroids entering surface waters via runoff or spray drift. In addition, aquatic species may be chronically exposed to sediment-associated pyrethroid insecticides, as sediments function as a sink for these compounds, although this depends upon sediment type and organic matter, as discussed above. Consequently, the aquatic toxicity of this class of insecticides is of significant concern.

8.1 Effects of pyrethroids on nontarget aquatic invertebrates

8.1.1 Impacts on aquatic invertebrate behavior

Given that the pyrethroid mode of action is on the peripheral nervous system, it is not surprising that behavioral effects in exposed invertebrates are numerous and distinctive, often occurring within minutes of exposure. This disruption of behavioral homeostasis can alter feeding rates, cause loss of coordination and paralysis, or stimulate hyperactivity that may increase exposure to predators, both of which can have ecologically-relevant consequences.

Surface insects were observed to be susceptible to pyrethroid over-spray, exhibiting a rapid loss of coordination and paralysis in response to esfenvalerate only a few hours after application (Samsøe-Petersen et al., 2001). Following sublethal aquatic exposures to cypermethrin, cyfluthrin, and deltamethrin, red swamp crayfish exhibited similar responses (Morolli et al., 2006). Likewise, sublethal permethrin exposure (0.03 to 0.05 µg/L) altered behavior in exposed caddisflies (*Brachycentrus americanus*) and stoneflies (*Pteronarcys dorsata*). Caddisflies exhibited increased "pawing" motions followed by a cessation of feeding behaviors, while intoxicated stoneflies lost equilibrium and became immobilized (Anderson, 1982). In both cases, mortality occurred several days after exposure, and the author theorized that disrupted feeding and starvation may have caused mortality.

Pyrethroid exposure has also been demonstrated to interfere with the complex building behaviors exhibited by case-building caddisflies (*Brachycentrus americanus*); these cases provide protection from predation, improve respiration and act as a refuge during pupation. Following 48-hour esfenvalerate exposures of 0.05 µg/L and greater, caddisfly larvae were observed to exit their cases, an abnormal behavior not observed in non-exposed larvae (Johnson et al., 2008). More significantly, case-rebuilding behaviors were also negatively affected by esfenvalerate exposure.

8.1.2 Impacts on aquatic invertebrate growth

Sublethal exposures to toxicants can lead to a reduction in growth. Impaired organisms may experience a reduction in feeding, either through chemical deterrence or via disrupted feeding behaviors. Conversely, exposure to toxicants may require the expenditure of additional energy for detoxification, metabolism, and restoration of behavioral and physiological homeostasis. Alteration of organisms' energy budgets following pyrethroid exposure can be observed as changes in growth and dry mass.

Short-term (24-hour) fenvalerate exposures as low as 0.3 µg/L reduced *Daphnia magna* food filtering rates (Reynaldi et al., 2006). Although individuals were eventually able to recover from the effects of the pulsed pyrethroid exposure and resume normal feeding behavior, exposed individuals were smaller and required more time to mature than unexposed organisms. Sediment-bound pyrethroids also adversely affected the growth of *Hyalella azteca* amphipods in laboratory experiments. Amphipod growth LOAECs ranged from 0.08 to 0.21 µg/g OC for bifenthrin, 0.46 to 0.77 µg/g OC for cyfluthrin, 0.20 to greater than 1.57 µg/g

OC for deltamethrin, 0.29 to 0.49 µg/g OC for esfenvalerate, 0.14 to 0.23 µg/g OC for lambda-cyhalothrin, and 0.68 to 5.3 for permethrin (Amweg et al., 2005). Although not tested, these reductions in growth are likely to affect later size and fecundity. Maul et al. (2008a) noted that pyrethroid-mediated reductions in growth had later effects on Chironomid fitness. Both larval ash free dry mass (AFDM) and instantaneous growth rate (IGR) were negatively impacted by exposure to sediment contaminated with bifenthrin, lambda-cyhalothrin, or permethrin, with respective 10-day LOAECs of 2.2, 2.0 and 74.2 µg/g OC. Reduced growth at these exposures was linked to reduced emergence, adult size, and fecundity (Maul et al., 2008a).

Exposure to pyrethroids has been demonstrated to impact specific invertebrate growth processes. The timing of mysid and pink shrimp mortality during acute toxicity tests with cypermethrin, permethrin, and fenvalerate appeared to be linked to molting, suggesting an increase in sensitivity to pyrethroid during this growth process (Cripe, 1994). Likewise, molting failures were noted in *Daphnia magna* exposed to cypermethrin, and this appeared to contribute to the mortality rates associated with the insecticide exposure (Kim et al., 2008). It is probable that young or rapidly growing arthropods may be more susceptible to the toxic effects of these insecticides, because they are likely to undergo more frequent molts than slow-growing and larger invertebrates. Similarly, those invertebrates that undergo specialized growth and re-generation may also be especially susceptible to the effects of sublethal pyrethroid exposure. For instance, chronic exposures to permethrin (<2 µg/L) disrupted male fiddler crab (*Uca pugnax*) limb re-generation, although female limb development was unaffected (Stueckle et al., 2009).

8.1.3 Effects on aquatic insect emergence

While delayed toxicity is rarely measured during the course of standard acute aquatic toxicity testing, this endpoint is critical in understanding the total effect of toxicants on aquatic organisms. For semi-aquatic insects, delayed or suppressed emergence has been observed following sublethal exposures to pyrethroids. This effect can alter normal male:female ratios and reduce reproductive output, resulting in delayed consequences of exposure.

Timing of caddisfly (*Limnephilus lunatus*) emergence was altered following larval exposures to 0.001 µg/L fenvalerate for one hour, although a reduction in emergence rate was only observed after exposure to a one-hour pulse of 0.1 µg /L fenvalerate (Schulz & Liess, 2000). Adult dry weight was reduced by larval exposure to 0.01 µg /L fenvalerate for one hour. Although all of these delayed effects have the potential to alter reproductive output and population dynamics, these data suggest that emergence timing is the most sensitive endpoint. Likewise, pulsed esfenvalerate exposure also had significant long-term impacts on the mayfly *Cloeon dipterum*. Reduced adult emergence of mayflies was observed 29 days after one-hour exposures to 0.01 µg/L esfenvalerate, a concentration three orders of magnitude lower than concentrations determined to result in acute mortality in this species (Beketov & Liess, 2005). Interestingly, the emergence of *Chironomus riparius* larvae from artificial ponds was stimulated at low esfenvalerate concentrations (0.1 and 0.2 µg/L), but was delayed by concentrations of 0.8 µg/L and greater (Samsøe-Petersen et al., 2001). However, given that the life cycles of semi-aquatic insects are finely attuned to seasonal and environmental cues, improperly timed emergence can result in new adults emerging into inhospitable climatic conditions.

There is some evidence that a reduced emergence rate following pyrethroid exposure may be related to physical or physiological difficulties in exoskeleton shedding. Reductions in mayfly (*Cinygmula reticulata*) emergence following 48-hour esfenvalerate exposures of 0.005 to 0.015 µg/L were a result of increases in unsuccessful molting (Palmquist et al., 2008b). Affected insects appeared unable to shed nymphal exoskeletons, which led to death; a similar response was observed for *B. americanus* caddisfly emergence after 48-hour pupal exposures of 0.1 µg/L esfenvalerate and greater (Palmquist et al., 2008b).

8.1.4 Effects on aquatic invertebrate reproductive success and output

Exposure to sublethal concentrations of pyrethroids affects the reproductive output of a number of aquatic invertebrate species, through simple reductions in the number of eggs produced, disruption of complex mating behaviors, or alteration of male to female ratios.

Short-term esfenvalerate exposures reduced the fecundity of both mayfly and caddisfly species. One-hour exposures to esfenvalerate (0.01 to 0.1 µg/L) followed by low food availability reduced egg production in female *Cloeon dipterum* mayflies (Beketov & Liess, 2005). Similarly, 48-hour pupal exposures to 0.05 µg/L esfenvalerate and greater reduced the ratio of egg weight to total female body weight in *B. americanus* caddisflies (Palmquist et al., 2008b). Chronic exposure to fenvalerate concentrations of 0.005 µg/L increased *Daphnia galeata mendotae* longevity by an average of 14 days, but reduced brood size by approximately 5 individuals. Exposures of 0.01 µg /L fenvalerate and greater over the daphnid life cycle reduced longevity, total reproductive output, brood size, and brood number (Day, 1989). Continuous (21-day) exposure to 0.3 µg/L fenvalerate was also demonstrated to have a greater adverse impact on *D. magna* reproductive rate than pulsed exposures to higher concentrations, and prevented the recovery of the exposed population (Reynaldi & Liess, 2005).

Sublethal pulsed exposure to esfenvalerate negatively affected both the fecundity and mating behavior of *Gammarus pulex* amphipods. Although 100% of test organisms survived one-hour pulses of up to 2 µg/L esfenvalerate, pulsed concentrations as low as 0.01 µg/L reduced the number of intact mating pairs (Cold & Forbes, 2004). Exposure to short-term pulses of 0.05 µg /L esfenvalerate and greater also increased mating pair reformation time by a factor of six. Delayed mating pairs also produced fewer offspring than non-intoxicated amphipods (Cold & Forbes, 2004).

8.1.5 Effects of dietary exposure to pyrethroids

Given the hydrophobicity of pyrethroid insecticides, the primary route of field exposure for water column invertebrates may be consumption of insecticide-contaminated diet. Despite this, few studies have been conducted on the dietary toxicity of this class of compounds. Undoubtedly, some fraction of toxicity observed in invertebrate sediment exposure studies does result from consumption of pyrethroid-contaminated material, but separation of dietary uptake from cuticular uptake under these scenarios is difficult.

Palmquist et al. (2008a) examined the impact of consumption of esfenvalerate-exposed dietary items on 3 species of aquatic insects. No evidence of feeding deterrence was observed, as none of the species tested rejected the esfenvalerate contaminated food. After three weeks, mayfly nymphs (*Cinygmula reticulata*) fed on algae pre-exposed for 24-hours to 0.5 to 1.0 µg/L esfenvalerate were smaller than control organisms. Further, consumption of diet exposed to 1.0 µg/L esfenvalerate for 10 days by final instar *C. reticulata* nymphs reduced the number and length of eggs in emerged females (Palmquist et al., 2008a).

Caddisfly (*B. americanus*) larvae feeding on dead esfenvalerate-contaminated *C. tentans* larvae exhibited characteristics of pyrethroid toxicity, including case abandonment. Pre-exposure of dietary items to 0.75 and 1.0 µg/L esfenvalerate for 24 hours resulted in increased rates of case-abandonment and mortality following consumption (Palmquist et al., 2008a). Although the dietary pyrethroids concentrations necessary to affect aquatic invertebrates appear to be significantly higher than aqueous concentrations, these results suggest that pyrethroids may still be toxicologically active when adsorbed to organic material.

8.2 Effects on aquatic vertebrates

Although designed to control pest invertebrate populations, pyrethroid insecticides are also toxic to nontarget aquatic vertebrates such as fish and amphibians (Ali 2011, Fishel 2005). Fish are considered especially sensitive to pyrethroid insecticides; similar behavioral and developmental effects have been observed in amphibians.

8.2.1 Amphibians

Documented symptoms in amphibians exposed to sublethal concentrations of pyrethroids include incoordination, lack of limb control, hyperactivity, tremors, and writhing. Leopard frog tadpoles exhibited convulsions and twitching following sublethal (1.3 µg/L) esfenvalerate exposure, with behavioral homeostasis re-established only one week after removal to clean systems (Materna et al., 1995). These behavioral aberrations, though not lethal alone, may increase predation of affected amphibians and fish.

Pyrethroid exposure also affects early amphibian development. Hatching success of moor frog (*Rana arvalis*) eggs was reduced following exposure to concentrations of α-cypermethrin as low as 1.0 µg/L, and a number of the exposed embryos exhibited convulsions and tail kinking following hatch. Cypermethrin concentrations as low as 0.1 µg/L during the egg stage also reduced length at metamorphosis (Greulich & Pflugmacher, 2003). Most notably, the jelly mass surrounding the amphibian eggs did not prevent embryos from absorbing cypermethrin from the surrounding aqueous medium. For instance, although exposure to either permethrin or fenvalerate at 0.01 to 2 ppm for 22 to 96 hours caused no increase in mortality, green frog (*Rana clamitans*) embryonic development was stunted at pyrethroid concentrations greater than 1 ppm, and operculum growth was also adversely affected (Berrill et al., 1993). Exposure to pyrethroids also causes reductions in tadpole biomass production 24 days following esfenvalerate exposures of 3.6 to 10 µg/L (Materna et al., 1995).

When exposed in a mesocosm study, amphibian fitness may actually improve as a result of decreased competition from invertebrate species. Green frog larval development was altered by a single pulsed exposure to 9 µg/L permethrin, and the tadpole Gosner developmental stage was increased, as was body mass (Boone, 2008). These responses indicated an increase in population fitness, and larger size and enhanced development would lead to earlier metamorphosis and larger metamorphic size. The author surmised that permethrin-mediated reductions in invertebrate populations led to increased algal food resources for developing amphibians (Boone, 2008). However, increases in amphibian biomass were also observed during single-species exposures. Permethrin concentrations of 1 µg/L increased the weight and size at metamorphosis of the common frog (*Rana temporaria*) tadpoles,

although exposure to esfenvalerate had no similar positive impacts on frog growth (Johansson et al., 2006).

8.2.2 Effects on fish

Impacts on normal behavior. As with aquatic invertebrates and amphibians, a number of significant behavioral effects have been observed in fish exposed to pyrethroids.

Low levels of pyrethroids caused neurobehavioral effects in young zebrafish (*Danio rerio*). Body spasms and uncontrolled swimming were observed following 6 day post fertilization exposures to either 1 µg/L deltamethrin, 1 µg/L cypermethrin, 10 µg/L cyhalothrin, 50 µg/L permethrin, or 100 µg/L bifenthrin (DeMicco et al., 2010) and Japanese medaka exhibited spinal curvatures and abnormal body shapes when exposed to cypermethrin concentrations as low as 100 µg/L (Kim et al., 2008). Although 4-hour esfenvalerate exposures as high as 20 µg/L caused no immediate mortality, abnormal behavior was observed in larval fathead minnows (*Pimephales promelas*) exposed to 0.455 and 1.142 µg/L esfenvalerate, including impaired swimming and feeding behaviors even after removal of exposed fish to clean systems (Floyd et al., 2008). Similarly, permethrin exposure was determined to induce a distinct set of behavioral effects in Japanese medaka (*Oryzias latipes*) juveniles, such as hyperactive swimming and midbody bends, followed by hypoactivity, reduced reaction to stimuli, and eventually death (Rice et al., 1997).

Chronic esfenvalerate exposure (0.01 to 0.05 µg/L) reduced the number of aggressive interactions between bluegill (*Lepomis macrochirus*) after 30 and 90 days of exposure; reductions persisted for at least 21 days following exposure (Little et al., 1993). Authors surmised that an increase in tremors interfered with normal inter-species interactions. Pulsed exposure to esfenvalerate also resulted in reduced aggression with increasing number of pulses (Little et al., 1993). However, bluegill sunfish inhabiting mesocosm systems dosed with a maximum 1.07 µg/L cyfluthrin (as the commercial product Baythroid®) exhibited no indications of behavioral sublethal responses to the exposure, as exposed fish were observed to swim, feed, and flee predation similarly to control fish (Morris et al. 1994).

Effects on fish reproduction and development. Sublethal pyrethroid exposures altered a number of reproductive and early developmental processes in fish. Low-level cypermethrin concentrations interfered with olfactory cues critical to Atlantic salmon reproduction. Five-day exposures of less than 0.004 µg/L inhibited the response of male fish to prostaglandin-type pheromones produced in the urine of reproductively-receptive female fish (Moore & Waring, 2001). In addition, when salmon milt and eggs were exposed to cypermethrin concentrations as low as 0.1 µg/L, the fertilization rate was reduced. Similarly, two pulsed exposures of 1 µg/L esfenvalerate delayed bluegill sunfish spawning (Little et al., 1993).

Gonadal development and structure in the freshwater snakehead fish, *Channa punctatus*, was altered by exposure to Devicyprin®, a commercial insecticide formulation containing 25% cypermethrin. Exposure equivalent to 1/10 of the LC_{50} for 10 days caused inflammation and intertubular vacuolization of testis, while necrosis of testis tissues was evident after 30 days of exposure (Srivastava et al., 2008). Ovary tissues exhibited vacuolization, stromal hemorrhage, and inflammation after 30 days exposure to 1/10 of the LC_{50}. In addition to effects on reproductive viability, pyrethroid insecticides also have been shown to alter embryonic and early life stage development in fish. Sublethal bifenthrin exposure caused increased spontaneous movement of zebrafish embryos that was linked to accelerated

hatching (Jin et al., 2009). In addition, 96-hour exposure to 109 µg/L or 256 µg/L bifenthrin increased incidences of curved body axis and pericardial edema. Later stages of Japanese medaka embryos were more sensitive to cypermethrin exposure, as most of the effects of the insecticide were observed at hatching (González-Doncel et al., 2003).

8.3 Community and ecosystem-level effects of aquatic pyrethroid exposures

Data garnered from higher-tier field and mesocosm studies provide an assessment of the true impacts of pyrethroid exposure in aquatic systems. Such experiments incorporate realistic measures of toxicant sequestration and degradation and also encompass interspecific competition and density-dependent compensatory (or depensatory) responses that can alter both toxic effects and long-term population dynamics.

8.3.1 Species sensitivity distributions

The risk of a specific toxicant to an aquatic community can be estimated through the construction of a species-sensitivity distribution (SSD) (Posthuma et al., 2002). SSDs combine toxicity values for a number of different species into a cumulative probability distribution that can be used to derive a community hazard level. These values ("hazard concentrations") are used to estimate the environmental concentrations that would be protective of 90 to 95% of the resident species (e.g., HC_5 or HC_{10} values).

Maund et al. (1998) compared the acute toxicities of lambda-cyhalothrin to a range of fish and arthropod species, including standard laboratory test species. For the fish species tested, the resulting HC_{10} was determined to be 0.087 µg/L, and the most sensitive species tested was the golden orfe (*Leuciscus idus*, 96-hour LC_{50} of 0.078 µg/L). This was in accordance with the lambda-cyhalothin HC_5 value reported in Maltby et al. (2005) for both fish and arthropod species, 0.003 µg/L; the maximum HC_5 value for pyrethroids was reported as 0.21 µg/L for permethrin. Interestingly, the range of fish sensitivities to lambda-cyhalothrin was much narrower than that for arthropods, as amphipods and isopods were roughly 100 times as susceptible to the pyrethroid insecticide as *D. magna* (Maund et al., 1998). However, the HC_{10} for the tested arthropod community was determined to be much lower than that for fish, 0.0017 µg/L. Mesocosm results presented by Maund et al. (1998) supported these findings, with the reported NOAECs for invertebrate communities and bluegill sunfish of 0.017 and 0.17 µg /L lambda-cyhalothrin, respectively. Authors concluded that adsorption of lambda-cyhalothrin to sediments reduced bioavailability in natural systems and decreased the risk to aquatic communities.

SSDs constructed from acute toxicity values for bifenthrin and permethrin gave approximate HC_{10} values for aquatic communities of 0.02 µg/L and 0.1 µg/L, respectively (Palmquist et al., 2011). The most sensitive species in both cases were *H. azteca* and *Ceriodaphnia dubia*. Dwyer et al. (2005) determined the toxicity of permethrin to 18 threatened and endangered aquatic species. Atlantic sturgeon, shortnose sturgeon, Lahontan cutthroat trout, spotfin chub, and Apache trout were determined to be the five taxa most susceptible to permethrin, with 96-hour LC_{50} values of less than 1.2 to 1.71 µg/L (Dwyer et al., 2005). By way of comparison, the LC_{50} values for the standard laboratory test species rainbow trout (*Oncorhynchus mykiss*), fathead minnow, and sheepshead minnow (*Cyprinodon variegates*) were 3.31, 9.38, and 17.0 µg/L permethrin, respectively. This suggests that some non-standard fish species may be more sensitive to pyrethroid exposure than standard laboratory species. Similarly, species sensitivity distributions also indicated that

saltwater species are significantly more sensitive to the pyrethroid insecticide permethrin than are freshwater species (Maltby et al., 2005).

8.3.2 Community effects in high-tier mesocosm studies

While SSDs can give an estimation of expected effect concentrations, mesocosm studies provide a more realistic assessment of the community-level toxicity of pyrethroid insecticides. Mesocosm experiments not only incorporate realistic exposure scenarios (as opposed to the simplified laboratory exposure systems used to derive toxicity values for SSDs), but are also able to examine the interspecific interactions that can either mediate or exacerbate the effects of pyrethroid exposure (e.g., with predation pressures, interspecific competition for resources, etc.).

Deltamethrin applications to temporary ponds located in the savannahs of West Senegal had immediate and catastrophic, but largely short-term effects, on the resident aquatic invertebrates. Significant numbers of moribund insects were evident during insecticide application, and these were largely predatory hemipterans and coleopterans (Lahr et al., 2000). Conversely, direct applications of cypermethrin at 0.7 g a.i./ha and lambda-cyhalothrin at 0.17 and 1.7 g a.i./ha produced no effect on the composition of emergent insect communities in mesocosm pond systems (Kedwards et al., 1999). The populations of benthic organisms on artificial substrates, however, were altered, and likely attributable to declines in sensitive Gammaridae and Asellidae taxa. Similarly, beta-cyfluthrin (as commercial product Baythroid) applied to artificial lentic mesocosms reduced both copepod and cladoceran populations, but did not affect community diversity indices (Heimbach et al., 1992). Subsequently, while phytoplankton were largely unaffected by the insecticide treatment, certain species exhibited increased abundance, likely as a result of reduced grazing pressures from copepods and cladocerans.

Given the notable effects on behavioral homeostasis, addition of predation pressures is likely to exacerbate the effects of sublethal pyrethroid exposure. Populations of brine shrimp (*Artemia* sp.) exposed to low levels of esfenvalerate (0.04 to 0.08 µg/L) along with simulated predatory pressure were completely eliminated from laboratory microcosms within 32 to 39 days after exposure (Beketov & Liess, 2005). Subsequently, it is possible that multiple ecological pressures (including predation and competition from less sensitive organisms) could cause greater abundance reductions and longer recovery times than observed following insecticide exposures alone. For instance, bifenthrin applications (39 to 287 ng/L) to outdoor mesocosms reduced copepod abundances, but resulted in increased rotifer and algal abundances (Hoagland et al., 1993). Rotifer abundances were also increased following simulated esfenvalerate drift and runoff events, likely as a response to decreased numbers of copepods and cladocerans from toxic pyrethroid effects and fish predation (Webber et al., 1992). Phytoplankton density also increased as a result of decreased grazing pressures from copepods and cladocerans. Smaller bluegill sunfish were present in ponds treated with high esfenvalerate applications (233 g a.i./ha) and authors theorized that this was a result of: 1) reductions in copepod and cladoceran prey and 2) benthic feeding habits of this stage of bluegill causing an increased exposure to sediment-bound esfenvalerate (Webber et al., 1992).

Esfenvalerate applications to an artificial pond system resulted in increased community chlorophyll production, likely a result of reduced grazing pressures, as cladocerans and copepods were reduced (Samsøe-Petersen et al., 2001). Bacterial and algal blooms were

observed following deltamethrin applications, resulting most likely from indirect effects of a reduction in invertebrate grazer populations (Hanson et al., 2007). Conversely, decreases in algal production have been observed following declines in top predator populations. A reduction in bluegill sunfish reproduction following exposure to pulses of either 0.67 or 1.71 µg/L esfenvalerate caused increases in the numbers of resilient zooplankton (Fairchild et al., 1992). This was observed to repress algal biomass, as invertebrate grazing pressures increased.

Although added pyrethroid mass is likely to be rapidly sequestered by organic material and sediments, short-term exposure to dissolved pyrethroids can still result in significant mesocosm effects. Despite rapid measured dissipation (water column half-life of 10.4 ± 2.0 hours), esfenvalerate negatively impacted mesocosm communities at concentrations as low as 0.25 µg/L. Six pulses of 0.25 µg/L esfenvalerate administered every 2 weeks significantly reduced the total abundance of macroinvertebrates (Fairchild et al., 1992). Authors noted that Ephemeroptera, Gastropoda and Diptera were the most sensitive invertebrate taxa. Likewise, two pulsed exposures to esfenvalerate concentrations as low as 0.08 µg/L decreased the number of multiple invertebrate taxa (including copepods, *Hyalella azteca*, and chironomids) up to 54 days after the first exposure. Conversely, numbers of cladocerans and oligochaetes increased following initial population reductions (Lozano et al., 1992). Loss of invertebrates had a positive effect on primary producers, but community metabolism was unaffected by esfenvalerate exposure.

Following pyrethroid exposure, rapid recovery has been observed, although the taxa-specific rates of recovery are variable and likely depend on life history strategies such as generation time and reproductive output. In general, recovery from pyrethroid exposure is likely to be a function of species-specific sensitivity and reproductive rates. For instance, Sherratt et al. (1999) found that recovery times following cypermethrin exposures were longest for the most sensitive taxa and for those with low reproductive rates. Populations of flying, semi-aquatic insects began to re-establish within days of deltamethrin treatment, while abundances of aquatic invertebrates, such as cladocerans and fairy shrimp, were suppressed longer, as recovery was predicated on the number of resting eggs present in the pond environment (Lahr et al., 2000). Similarly, esfenvalerate exposures had negatively impacted zooplankton communities, through reductions in sensitive cladoceran and copepod taxa, but populations recovered in less than 2 weeks following exposure (Fairchild et al., 1992); rotifers were determined to be less sensitive than other zooplankton.

Copepod and cladoceran zooplankton abundances in mesocosm systems were diminished during both simulated lambda-cyhalothrin runoff and spray drift events (maximum water concentration = 98 ng/L), but quickly rebounded following cessation of insecticide applications. Conversely, rotifers and total macroinvertebrates were increased during the dosing events (Hill et al., 1994). Simulated tralomethrin runoff (218.9 ng/L) and drift (68.5 ng/L) also did not produce any lasting effects on mesocosm ecosystems. Although densities of Caenidae mayflies and copepods were reduced, other organisms, including mollusks, fish, macrophytes, algae, and benthic invertebrates were unaffected (Mayasich et al., 1994).

Examination of the results from multiple mesocosm studies indicates that 1) a certain subset of species can be expected to be most sensitive to pyrethroid insecticides, and 2) community function is rarely, if ever, altered by pyrethroid exposure, although the community structure may be altered. For example, mesocosm community metabolism, as measured by photosynthetic and respiration rates, was unaffected by lambda-cyhalothrin treatment, despite alterations in the invertebrate community (Hill et al., 1994). Giddings et al. (2001)

analyzed the results of seven different pyrethroid mesocosm studies and determined that amphipods, copepods, cladocerans, mayflies, caddisflies and midges were consistently the most sensitive invertebrates. However, reductions in amphipod and isopods were accompanied by increases in less sensitive organisms that occupy similar ecological niches (Giddings et al., 2001). Although pyrethroids were typically associated with sediment after introduction into mesocosms, the population- and community-level impacts observed in exposed mesocosms were correlated most closely to maximum water concentrations (Giddings et al., 2001). Recovery of affected species was determined to be governed by species-specific attributes, such as generation time and reproductive rate. Zooplankton populations, for instance, recovered quickly, as they have short generation times and high reproductive output (Giddings et al., 2001). In addition to factors that influence recovery rate, other life history strategies are likely to alter the effects of pyrethroid exposure on aquatic species. Zooplankton resting egg stages located in sediments likely comprised a significant ecological reservoir, for which re-colonization following pyrethroid exposure can occur (Hanson et al. 2007). In addition, some species may inhabit microenvironments that are refugia from pyrethroid exposures, and those species with efficient or rapid immigration, are more likely to recover rapidly from pyrethroid exposures (Giddings et al., 2001).

9. Conclusions

The usage of pyrethroid insecticides increased as a response to the phasing out of other insecticide classes. In contrast to other insecticides, pyrethroids exhibit lower mammalian and avian toxicity and better selectivity to target species than OPs and less persistence than organochlorine insecticides.

Drift and runoff from sprayed outdoor areas are probably the primary routes of pyrethroid movement into nontarget aquatic and terrestrial environments. Indoor use of pyrethroids is expected to be a very small component of total applications, and therefore is unlikely to result in significant environmental concentrations. Pyrethroids do not readily volatilize and exhibit strong adsorption to organic matter, soils and clay, an aspect that largely governs their environmental fate and bioavailability.

Although pyrethroid insecticides were developed for the control of terrestrial pest insects, there is evidence that nontarget aquatic invertebrate species may be more sensitive than terrestrial invertebrates. Pyrethroids have also been demonstrated to be toxic to nontarget aquatic vertebrates, such as fish and amphibians. However, evidence from aquatic mesocosm studies suggests that adverse population- and community-level effects following pyrethroid exposure are probably short-term, and that recovery from these is rapid. In terrestrial systems, spray drift generally results in non-homogenous distribution of pyrethroids in edge-of-field and off-crop areas. Hence, impacts on nontarget invertebrates in these areas are spatially variable, and recovery can occur via immigration from non-impacted populations.

As pyrethroids provide effective control of pest insects, widespread use is expected to continue. In order to better understand the environmental impacts of this use, exposure and effects studies that incorporate environmentally relevant concentrations should continue to be conducted. In addition, research into best management practices, such as using vegetation buffers or timing applications to avoid exposure to nontarget organisms, or development of formulations that are less likely to be transported into nearly streams and

rivers, should be conducted. It is also imperative that results of that research be conveyed to the public and private sectors so that the professional pest applicators and private homeowners who use these products around their homes do so in an environmentally-friendly manner.

10. References

Åkerblom, N., Arbjörk, C., Hedlund, M. and Goedkoop, W. (2008). Deltamethrin toxicity to the midge Chironomus riparius Meigen--Effects of exposure scenario and sediment quality. *Ecotoxicology and Environmental Safety* 70, 53-60.

Ali SFM, Shieh BHH, Alehaideb Z, Khan MZ, Louie A, Fageh N, Law FCP. (2011). A review on the effects of some selected pyrethroids and related agrochemicals on aquatic vertebrate biodiversity. *Canadian Journal of Pure and Applied Science* 5(2): 1455-1464.

Amweg, E. L., Weston, D. P. and Ureda, N. M. (2005). Use and toxicity of pyrethroid pesticides in the Central Valley, California, USA. *Environmental Toxicology and Chemistry* 24, 966-972.

Anderson, R. L. (1982). Toxicity of Fenvalerate and Permethrin to Several Nontarget Aquatic Invertebrates. *Environmental Entomology* 11, 1251-1257.

Arts, G. H. P., Buijse-Bogdan L. L., Belgers J. D. M., Rhenen-Kersten C. H. , Van Wijngaarden, R. P. A. ,Van Roessink I., Maund S. J., Brink P. J. van den, Brock, T. C. M. (2006). Ecological impact in ditch mesocosms of simulated spray drift from a crop protection program for potatoes. *Integrated Environmental Assessment and Management (IEAM)* 2, 105-125.

Beketov, M. A. and Liess, M. (2005). Acute contamination with esfenvalerate and food limitation: chronic effects on the mayfly, Cloeon dipterum. *Environmental Toxicology and Chemistry* 24, 1281-1286.

Bennett, E. R., Moore, M. T., Cooper, C. M., Smith, S., Shields, F. D., Drouillard, K. G. and Schulz, R. (2005). Vegetated agricultural drainage ditches for the mitigation of pyrethroid-associated runoff. *Environmental Toxicology and Chemistry* 24, 2121-2127.

Berrill, M., Bertram, S., Wilson, A., Louis, S., Brigham, D. and Stromberg, C. (1993). Lethal and sublethal impacts of pyrethroid insecticides on amphibian embryos and tadpoles. *Environmental Toxicology and Chemistry* 12, 525-539.

Boone, M. D. (2008). Examining the single and interactive effects of three insecticides on amphibian metamorphosis. *Environmental Toxicology and Chemistry* 27, 1561-1568.

Brooks, J. E., Savarie, P. J. and Bruggers, R. L. (1998a). The toxicity of commercial insecticide aerosol formulations to brown tree snakes. *Snake* 28, 23-27.

Chapman, R. A., Tu, C. M., Harris, C. R. and Cole, C. (1981). Persistence of five pyrethroid insecticides in sterile and natural, mineral and organic soil. *Bulletin of environmental contamination and toxicology* 26, 513-519.

Cold, A. and Forbes, V. E. (2004). Consequences of a short pulse of pesticide exposure for survival and reproduction of *Gammarus pulex*. *Aquatic Toxicology* 67, 287-299.

Cox, R. L. and Wilson, W. T. (1984). Effects of permethrin on the behavior of individually tagged honey bees, *Apis mellifera* L. (Hymenoptera: Apidae). *Environmental Entomology* 13, 375-378.

Cripe, G. M. (1994). Comparative acute toxicities of several pesticides and metals to *Mysidopsis bahia* and postlarval *Penaeus duorarum*. *Environmental Toxicology and Chemistry* 13, 1867-1872.

Dai, P.-L., Wang, Q., Sun, J.-H., Liu, F., Wang, X., Wu, Y.-Y. and Zhou, T. (2010). Effects of sublethal concentrations of bifenthrin and deltamethrin on fecundity, growth, and development of the honeybee *Apis mellifera ligustica*. *Environmental Toxicology and Chemistry* 29, 644-649.

Decourtye, A., Devillers, J., Genecque, E., Menach, K. L., Budzinski, H., Cluzeau, S. and Pham-Delègue, M. H. (2005). Comparative sublethal toxicity of nine pesticides on olfactory learning performances of the honeybee *Apis mellifera*. *Archives of environmental contamination and toxicology* 48, 242-250.

Delpuech, J.-M., Legallet, B., Terrier, O. and Fouillet, P. (1999). Modifications of the sex pheromonal communication of *Trichogramma brassicae* by a sublethal dose of deltamethrin. *Chemosphere* 38, 729-739.

DeMicco, A., Cooper, K. R., Richardson, J. R. and White, L. A. (2010). Developmental neurotoxicity of pyrethroid insecticides in zebrafish embryos. *Toxicological Sciences* 113, 177-186.

Desneux, N., Decourtye, A. and Delpuech, J. M. (2007). The sublethal effects of pesticides on beneficial arthropods. *Annual Review of Entomology* 52, 81-106.

Dwyer, F. J., Mayer, F. L., Sappington, L. C., Buckler, D. R., Bridges, C. M., Greer, I. E., Hardesty, D. K., Henke, C. E., Ingersoll, C. G., Kunz, J. L. et al. (2005). Assessing contaminant sensitivity of endangered and threatened aquatic species: Part I. acute toxicity of five chemicals. *Archives of environmental contamination and toxicology* 48, 143-154.

Ecobichon D.J. (1996). Toxic effects of pesticides. In: *Casarett & Doull's Toxicology: The Basic Science of Poisons*, Klaassen C.D., Amdur MO, Doull J. (Eds). McGraw-Hill, Health Professions Division. New York, NY. 1996. 643-690.

Fairchild, J. F., La Point, T. W., Zajicek, J. L., Nelson, M. K., Dwyer, F. J. and Lovely, P. A. (1992). Population-, community- and ecosystem-level responses of aquatic mesocosms to pulsed doses of a pyrethroid insecticide. Environmental Toxicology and Chemistry.11, 115-129.

Feo, M.L., Eljarrat, E., Barcelo', D. (2010). Determination of pyrethroid insecticides in environmental samples. *Trends in Analytical Chemistry* 29(7):692-705.

Fishel FM. 2005. Pesticide Toxicity Profile: Synthetic Pyrethroid Pesticides. Agronomy Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida. Report #PI-54.

Fleming, R. J., Holmes, D. and Nixon, S. J. (1998). Toxicity of permethrin to *Chironomus riparius* in artificial and natural sediments. *Environmental Toxicology and Chemistry* 17, 1332-1337.

Floyd, E. Y., Geist, J. P. and Werner, I. (2008). Acute, sublethal exposure to a pyrethroid insecticide alters behavior, growth, and predation risk in larvae of the fathead minnow (Pimephales promelas). *Environmental Toxicology and Chemistry* 27, 1780-1787.

Friesen, K. J. and Kaufman, W. R. (2003). Cypermethrin inhibits egg development in the ixodid tick, *Amblyomma hebraeum*. *Pesticide Biochemistry and Physiology* 76, 25-35.

Gan, J., Lee, S. J., Liu, W. P., Haver, D. L. and Kabashima, J. N. (2005). Distribution and persistence of pyrethroids in runoff sediments. *J. Environ. Qual.* 34, 836-841.

Giddings, J. M., Solomon, K. R. and Maund, S. J. (2001). Probabilistic risk assessment of cotton pyrethroids: II. Aquatic mesocosm and field studies. *Environmental Toxicology and Chemistry* 20, 660-668.

González-Doncel, M., de la Peña, E., Barrueco, C. and Hinton, D. E. (2003). Stage sensitivity of medaka (*Oryzias latipes*) eggs and embryos to permethrin. *Aquatic Toxicology* 62, 255-268.

Greulich, K. and Pflugmacher, S. (2003). Differences in susceptibility of various life stages of amphibians to pesticide exposure. *Aquatic Toxicology* 65, 329-336.

Hall, F. R. (1979). Effects of Synthetic Pyrethroids on Major Insect and Mite Pests of Apple. *Journal of economic entomology* 72, 441-446.

Hammond, R. B. (1996). Residual activity of lambda-cyhalothrin against bean leaf beetle (Coleoptera: Chrysomelidae) in soybeans. *Journal of Agricultural Entomology* 13, 365-373.

Hanson, M. L., Graham, D. W., Babin, E., Azam, D., Coutellec, M.-A., Knapp, C. W., Lagadic, L. and Caquet, T. (2007). Influence of isolation on the recovery of pond mesocosms from the application of an insecticide. I. Study design and planktonic community responses. *Environmental Toxicology and Chemistry* 26, 1265-1279.

Haynes, K. F. (1988). Sublethal effects of neurotoxic insecticides on insect behavior. *Annual Review of Entomology* 33, 149-168.

Haynes, K. F. and Baker, T. C. (1985). Sublethal effects of permethrin on the chemical communication system of the pink bollworm moth, *Pectinophora gossypiella*. *Archives of Insect Biochemistry and Physiology* 2, 283-293.

Heimbach, F., Pflueger, W. and Ratte, H.-T. (1992). Use of small artificial ponds for assessment of hazards to aquatic ecosystems. *Environmental Toxicology and Chemistry* 11, 27-34.

Hill, I. R., Runnalls, J. K., Kennedy, J. H. and Ekoniak, P. (1994a). Lambda-cyhalothrin: A mesocosm study of its effects on aquatic organisms. In *Aquatic Mesocosm Studies in Ecological Risk Assessment*, eds. R. L. Graney J. H. Kennedy and J. H. Rodgers Jr.). Boca Raton, Fl: Lewis Publishers.

Hoagland, K. D., Drenner, R. W., Smith, J. D. and Cross, D. R. (1993). Freshwater community responses to mixtures of agricultural pesticides: Effects of atrazine and bifenthrin. *Environmental Toxicology and Chemistry* 12, 627-637.

Hull, L. A., Beers, E. H. and Meagher, R. L. (1985). Integration of Biological and Chemical Control Tactics for Apple Pests Through Selective Timing and Choice of Synthetic Pyrethroid Insecticides. *Journal of economic entomology* 78, 714-721.

Inglesfield, C. (1989). Pyrethroids and terrestrial nontarget organisms. *Pesticide science* 27, 387-428.

Ishaaya, I., Elsner, A., Ascher, K. R. S. and Casida, J. E. (1983). Synthetic pyrethroids: Toxicity and synergism on dietary exposure of *Tribolium castaneum* (Herbst) larvae. *Pesticide science* 14, 367-372.

Jin, H. and Webster, G. R. B. (1998). Persistence, Penetration, and Surface Availability of Cypermethrin and Its Major Degradation Products in Elm Bark. *Journal of Agricultural and Food Chemistry* 46, 2851-2857.

Jin, M., Zhang, X., Wang, L., Huang, C., Zhang, Y. and Zhao, M. (2009). Developmental toxicity of bifenthrin in embryo-larval stages of zebrafish. *Aquatic Toxicology* 95, 347-354.

Johansson, M., Piha, H., Kylin, H. and Merilä, J. (2006). Toxicity of six pesticides to common frog (*Rana temporaria*) tadpoles. *Environmental Toxicology and Chemistry* 25, 3164-3170.

Johnson, K. R., Jepson, P. C. and Jenkins, J. J. (2008). Esfenvalerate-induced case-abandonment in the larvae of the caddisfly (Brachycentrus americanus). *Environmental Toxicology and Chemistry* 27, 397-403.

Juarez, M. P. (1995). The effect of sublethal doses of insecticides on *Triatoma infestans* lipid synthesis. *Pesticide Biochemistry and Physiology* 52, 81-89.

Katagi, T. (1991). Photodegradation of the pyrethroid insecticide esfenvalerate on soil, clay minerals, and humic acid surfaces. *Journal of Agricultural and Food Chemistry* 39, 1351-1356.

Kaufman, D. D., Russell, B. A., Helling, C. S. and Kayser, A. J. (1981). Movement of cypermethrin, decamethrin, permethrin, and their degradation products in soil. *Journal of Agricultural and Food Chemistry* 29, 239-245.

Kedwards, T. J., Maund, S. J. and Chapman, P. F. (1999). Community level analysis of ecotoxicological field studies: II. Replicated-design studies. *Environmental Toxicology and Chemistry* 18, 158-166.

Lahr, J., Diallo, A. O., Gadji, B., Diouf, P. S., Bedaux, J. J. M., Badji, A., Ndour, K. B., Andreasen, J. E. and van Straalen, N. M. (2000). Ecological effects of experimental insecticide applications on invertebrates in sahelian temporary ponds. *Environmental Toxicology and Chemistry* 19, 1278-1289.

Langhof, M., Gathmann, A., Poehling, H. M., Vogt, H., Heimbach, U. and Vinuela, E. (2003). Field study on the effects of insecticide drift on nontarget arthropods : residual toxicity, effect on populations and recolonisation processes. *Bulletin OILB/SROP* 26, 73-82.

Lee, C. Y., Yap, H. H. and Chong, N. L. (1998). Sublethal effects of deltamethrin and propoxur on longevity and reproduction of German cockroaches, *Blattella germanica*. *Entomologia Experimentalis et Applicata* 89, 137-145.

Lee, P. W. (1985). Fate of fenvalerate (Pydrin insecticide) in the soil environment. *Journal of Agricultural and Food Chemistry* 33, 993-998.

Little, E. E., Dwyer, F. J., Fairchild, J. F., Delonay, A. J. and Zajicejk, J. L. (1993). Survival of bluegill and their behavioral responses during continuous and pulsed exposures to esfenvalerate, a pyrethroid insecticide. *Environmental Toxicology and Chemistry* 12, 871-878.

Liu, W., Gan, J. J., Lee, S. and Kabashima, J. N. (2004). Phase distribution of synthetic pyrethroids in runoff and stream water. *Environmental Toxicology and Chemistry* 23, 7-11.

Longley, M. (1999). A review of pesticide effects upon immature aphid parasitoids within mummified hosts. *International Journal of Pest Management* 45, 139 - 145.

Longley, M. and Jepson, P. C. (1996). Effects of honeydew and insecticide residues on the distribution of foraging aphid parasitoids under glasshouse and field conditions. *Entomologia Experimentalis et Applicata* 81, 189-198.

Lozano, S. J., O'Halloran, S. L., Sargent, K. W. and Brazner, J. C. (1992). Effects of esfenvalerate on aquatic organisms in littoral enclosures. *Environmental Toxicology and Chemistry* 11, 35-47.

Luo, Y., and Zhang, M. (2011). Environmental modeling and exposure assessment of sediment-associated pyrethroids in an agricultural watershed. PLoS ONE 6(1):e15794. Doi:10.1371/journal.pone.0015794.

Maltby, L., Blake, N., Brock, T. C. M. and Van den Brink, P. J. (2005). Insecticide species sensitivity distributions: Importance of test species selection and relevance to aquatic ecosystems. *Environmental Toxicology and Chemistry* 24, 379-388.

Materna, E. J., Rabeni, C. F. and Lapoint, T. W. (1995). Effects of the synthetic pyrethroid insecticide, esfenvalerate, on larval leopard frogs (*Rana* spp.). *Environmental Toxicology and Chemistry* 14, 613-622.

Maul, J. D., Brennan, A. A., Harwood, A. D. and Lydy, M. J. (2008a). Effect of sediment-associated pyrethroids, fipronil, and metabolites on Chironomus tentans growth rate, body mass, condition index, immobilization, and survival. *Environmental Toxicology and Chemistry* 27, 2582-2590.

Maul, J. D., Trimble, A. J. and Lydy, M. J. (2008b). Partitioning and matrix-specific toxicity of bifenthrin among sediments and leaf-sourced organic matter. *Environmental Toxicology and Chemistry* 27, 945-952.

Maund, S. J., Hamer, M. J., Warinton, J. S. and Kedwards, T. J. (1998). Aquatic ecotoxicology of the pyrethroid insecticide lambda-cyhalothrin: considerations for higher-tier aquatic risk assessment. *Pesticide science* 54, 408-417.

Maund, S. J., Travis K Z, Hendley P, Giddings J M and Solomon K R. (2001). Probabilistic risk assessment of cotton pyrethroids: V. Combining landscape-level exposures and ecotoxicological effects data to characterize risks. *Environmental Toxicology and Chemistry* 20, 687-692.

Mayasich, J. M., Kennedy, J. H. and O'Grodnick, J. S. (1994). Evaluation of the ecological and biological effects of tralomethrin utilizing an experimental pond system. In *Aquatic Mesocosm Studies in Ecological Risk Assessment*, eds. R. L. Graney J. H. Kennedy and J. H. Rodgers Jr.). Boca Raton, FL: Lewis Publishers.

Moore, A. and Waring, C. P. (2001). The effects of a synthetic pyrethroid pesticide on some aspects of reproduction in Atlantic salmon (*Salmo salar* L.). *Aquatic Toxicology* 52, 1-12.

Moore, M.T., Cooper, C.M., Smith, S., Cullum, R.F., Knight, S.S., Locke, M.A., Bennett, E.R. 2009. Mitigation of two pyrethroid insecticides in a Mississippi Delta constructed wetland. *Environmental Pollution* 157:250-256.

Moreau, R., Carle, P. R., Gourdoux, L., Couillaud, F., Khay, A. B. and Girardie, A. (1987). Deltamethrin: In vivo effects on glucose catabolism in *Locusta migratoria* L. *Pesticide Biochemistry and Physiology* 27, 101-105.

Morolli, C., Quaglio, F., Della Rocca, G., Malvisi, J. and Di Salvo, A. (2006). Evaluation of the Toxicity of Synthetic Pyrethroids to Red Swamp Crayfish (Procambarus clarkii, Girard 1852) and Common Carp (Cyprinus carpio, L. 1758). *Bull. Fr. Pêche Piscic.* 380-381, 1381-1394.

Muir, D. C. G., Rawn, G. P., Townsend, B. E., Lockhart, W. L. and Greenhalgh, R. (1985). Bioconcentration of cypermethrin, deltamethrin, fenvalerate and permethrin by *Chironomus tentans* larvae in sediment and water. *Environmental Toxicology and Chemistry* 4, 51-61.

Oros, D. R. and Werner, I. (2005). Pyrethroid Insecticides: An Analysis of Use Patterns, Distributions, Potential Toxicity and Fate in the Sacramento-San Joaquin Delta and

Central Valley. In *White Paper for the Interagency Ecological Program*, vol. SFEI Contribution 415. Oakland, CA: San Francisco Estuary Institute.

Palmquist, K., Fairbrother, A., Salatas, J. and Guiney, P. D. (2011). Environmental fate of pyrethroids in urban and suburban stream sediments and the appropriateness of Hyalella azteca model in determining ecological risk. *Integrated Environmental Assessment and Management* 7, 325-335.

Palmquist, K. R., Jenkins, J. J. and Jepson, P. C. (2008a). Effects of dietary esfenvalerate exposures on three aquatic insect species representing different functional feeding groups. *Environmental Toxicology and Chemistry* 27, 1721-1727.

Palmquist, K. R., Jepson, P. C. and Jenkins, J. J. (2008b). Impact of aquatic insect life stage and emergence strategy on sensitivity to esfenvalerate exposure. *Environmental Toxicology and Chemistry* 27, 1728-1734.

Pap, L., Bajomi, D., and Szekely, I. (1996). The pyrethroids, an overview. *International Pest Council* 38(1):15-19.

Posthuma, L., Suter, G.W. II. and Traas, T.P. (2002). Species Sensitivity Distributions in Ecotoxicology. CRC Press. Boca Raton, FL. 616pp.,

Ranson, H., N'Guessan, R, Lines, J., Moiroux, N., Nkuni, Z., and Corbel, V. (2011). Pyrethroid resistance in African anopheline mosquitoes: what are the implications for malaria control? *Trends in Parasitology* 27(2):91-98.

Ray, D.E. (2000). Pesticide neurotoxicity in Europe: real risks and perceived risks. *NeuroToxicology* 21(1-2):219-222.

Reynaldi, S., Duquesne, S., Jung, K. and Liess, M. (2006). Linking feeding activity and maturation of Daphnia magna following short - term exposure to fenvalerate. *Environmental Toxicology and Chemistry* 25, 1826-1830.

Reynaldi, S. and Liess, M. (2005). Influence of duration of exposure to the pyrethroid fenvalerate on sublethal responses and recovery of Daphnia magna straus. *Environmental Toxicology and Chemistry* 24, 1160-1164.

Rice, P. J., Drewes, C. D., Klubertanz, T. M., Bradbury, S. P. and Coats, J. R. (1997). Acute toxicity and behavioral effects of chlorpyrifos, permethrin, phenol, strychnine, and 2,4-dinitrophenol to 30-day-old Japanese medaka (Oryzias latipes). *Environmental Toxicology and Chemistry* 16, 696-704.

Samsøe-Petersen, L., Gustavson, K., Madsen, T., Mogensen, B. B., Lassen, P., Skjernov, K., Christoffersen, K. and Jørgensen, E. (2001). Fate and effects of esfenvalerate in agricultural ponds. *Environmental Toxicology and Chemistry* 20, 1570-1578.

Schulz, R. and Liess, M. (2000). Toxicity of fenvalerate to caddisfly larvae: chronic effects of 1- vs 10-h pulse-exposure with constant doses. *Chemosphere* 41, 1511-1517.

Sherratt, T. N., Roberts, G., Williams, P., Whitfield, M., Biggs, J., Shillabeer, N. and Maund, S. J. (1999). A life-history approach to predicting the recovery of aquatic invertebrate populations after exposure to xenobiotic chemicals. *Environmental Toxicology and Chemistry* 18, 2512-2518.

Soderlund DM, Bloomquist JR.1989. Neurotoxic actions of pyrethroid insecticides. *Ann. Rev. Entomol.* 34:77-96.

Srivastava, R. K., Yadav, K. K. and Trived, S. P. (2008). Devicyprin induced gonadal impairment in a freshwater food fish, Channa punctatus (Bloch). *Journal of Environmental Biology* 29, 187-191.

Stueckle, T. A., Shock, B. and Foran, C. M. (2009). Multiple stressor effects of methoprene, permethrin, and salinity on limb regeneration and molting in the mud fiddler crab (UCA pugnax). *Environmental Toxicology and Chemistry* 28, 2348-2359.

Talent, L. G. (2005). Effect of temperature on toxicity of a natural pyrethrin pesticide to green anole lizards (Anolis carolinensis). *Environmental Toxicology and Chemistry* 24, 3113-3116.

Vandame, R., Meled, M., Colin, M.-E. and Belzunces, L. P. (1995). Alteration of the homing-flight in the honey bee *Apis mellifera* L. exposed to sublethal dose of deltamethrin. *Environmental Toxicology and Chemistry* 14, 855-860.

US EPA. (2000). Chlorpyrifos revised risk assessment and agreement with registrants. Washington, D.C.: U.S. Environmental Protection Agency.

US EPA. (2001). Diazinon revised risk assessment and agreement with registrants. Prevention, Pesticides and Toxic Substances. Washington, D.C.: U.S. Environmental Protection Agency.

Walker, K. (2000). Cost-comparison of DDT and alternative insecticides for malaria control. *Medical and Veterinary Entomology* 14:354-354.

Webber, E. C., Deutsch, W. G., Bayne, D. R. and Seesock, W. C. (1992). Ecosystem-level testing of a synthetic pyrethroid insecticide in aquatic mesocosms. *Environmental Toxicology and Chemistry* 11, 87-105.

Weston DP, Lydy MJ. 2010. Urban and Agricultural Sources of Pyrethroid Insecticides to the Sacramento-San Joaquin Delta of California. Environ. Sci. Technol. 44(5): 1833-1840.

Wiles, J. A. and Jepson, P. C. (1994). Substrate-mediated toxicity of deltamethrin residues to beneficial invertebrates: Estimation of toxicity factors to aid risk assessment. *Archives of environmental contamination and toxicology* 27, 384-391.

WHOPES. (2005). Safety of pyrethroids for public health use. WHO Pesticide Evaluation Scheme.

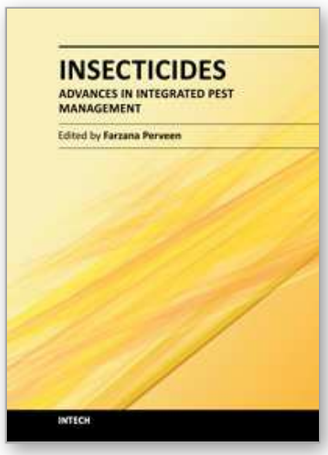
World Health Organization. (1989a). *Environmental Health Criteria 92 – Resmethrin*. International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, pp 1-79.

World Health Organization. (1989b). *Environmental Health Criteria 82 – Cypermethrin*. International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, pp 1-88.

World Health Organization. (1990a). *Environmental Health Criteria 96 – d-Phenothrin*. International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, pp 1-33.

World Health Organization. (1990b). *Environmental Health Criteria 94 - Permethrin*. International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, pp 1-58.

You, J., Pehkonen, S., Weston, D. P. and Lydy, M. J. (2008). Chemical availability and sediment toxicity of pyrethroid insecticides to Hyalella azteca: Application to field sediment with unexpectedly low toxicity. *Environmental Toxicology and Chemistry* 27, 2124-2130.



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This book contains 30 Chapters divided into 5 Sections. Section A covers integrated pest management, alternative insect control strategies, ecological impact of insecticides as well as pesticides and drugs of forensic interest. Section B is dedicated to chemical control and health risks, applications for insecticides, metabolism of pesticides by human cytochrome p450, etc. Section C provides biochemical analyses of action of chlorfluazuron, pest control effects on seed yield, chemical ecology, quality control, development of ideal insecticide, insecticide resistance, etc. Section D reviews current analytical methods, electroanalysis of insecticides, insecticide activity and secondary metabolites. Section E provides data contributing to better understanding of biological control through Bacillus sphaericus and B. thuringiensis, entomopathogenic nematodes insecticides, vector-borne disease, etc. The subject matter in this book should attract the reader's concern to support rational decisions regarding the use of pesticides.

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