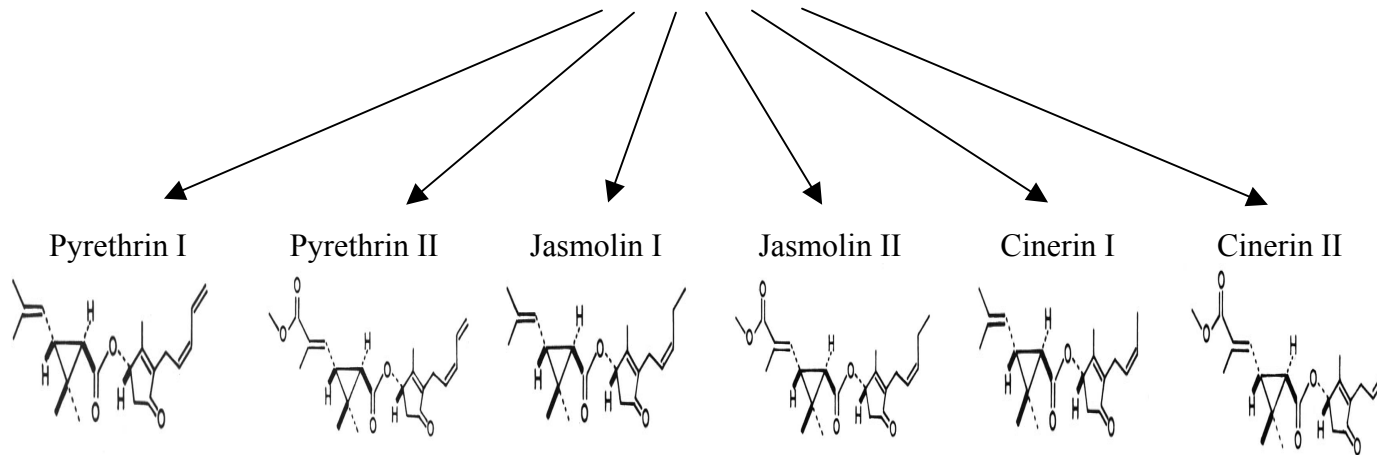# Environmental Fate of Pyrethrins

Amrith S. Gunasekara
Environmental Monitoring Branch
Department of Pesticide Regulation
1001 I Street
Sacramento, CA 95812

November 2004
(Revised 2005)

PYRETHRINS

| Pyrethrin I | Pyrethrin II | Jasmolin I | Jasmolin II | Cinerin I | Cinerin II |

Table of Contents

Introduction

Pyrethrins are a combination of six natural chemicals extracted from the commercial flowers *Chrysanthemum cinerariaefolium* and *Chrysanthemum cineum* (McLaughlin, 1973; Todd et al., 2003). Crushed and powdered *Chrysanthemum* plants were used as an insecticide by the Chinese as early as 1000 BC. Pyrethrins from the *Chrysanthemum* flower were first identified as having insecticidal properties around 1800 in Asia. Since their discovery, pyrethrins have been sold under several trade names, including Buhach®, Ofirmotox®, Dalmation Insect Flowers®, Firmotox®, Parexan® and NA 9184®. In the United States, McLaughlin Gormley King in Minnesota and SureCo Incorporated in Georgia are the two main producers of pyrethrins (Todd et al., 2003). The popularity of the compounds stems from their effective paralysis action against a wide range of insect pests (Gnadinger, 1936). These pests include mosquitoes, sawfly larvae, caterpillars, leafhoppers, aphids, and beetles. Pyrethrins are often used as an agricultural pre-harvest treatment on fruits, forage crops, vegetables, and ornamental plants and applied as a dust, spray or emulsified substance. Pyrethrins are often applied with synergists such as piperonyl butoxide and piperonyl sulfoxide to increase the effectiveness of the insecticide (Todd et al., 2003).

Pyrethrins are naturally released from *Chrysanthemum* flowers but the quantities released are very small compared to the amount in commercial products (Todd et al., 2003). They are extracted as an oil or dry powder shortly after the flower blooms. The flowers contain about 1-2% pyrethrins, relative to its dry weight, but approximately 94% of the total yield is concentrated in the seeds of the flower (Casida and Quistad, 1995). The flowers have historically been grown in commercial quantities in Kenya, Tanzania, Rwanda and Papua New Guinea. However, over the past decade, Tasmania and Uganda began harvesting *Chrysanthemum* flowers. Kenya and Tasmania are two of the largest pyrethrins producing nations (Jones, 1973). About 200,000 kg of pyrethrins are used as an insecticide each year (Crosby, 1995). This high demand led Barthomeuf et al. (1996) to study the potential of using cultivated plant cells (calli) as an alternative to obtaining pyrethrins from *Chrysanthemum cinerariaefolium* flowers. They found that the callus of

C. cinerariaefolium are able to synthesize pyrethrins at high concentrations, more than 30 mg/100 g dry biomass.

Pyrethrins were declared an effective mosquito control agent for reducing malaria in 1942. However, there was a reduction in pyrethrins use for such purposes after advances in synthetically manufactured insecticides led to the discovery of dichlorodiphenyltrichloroethane (DDT) in 1945 and later to the synthetic version of pyrethrins, pyrethroids.
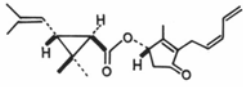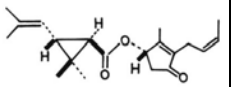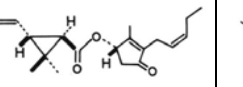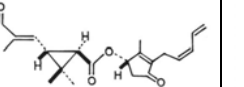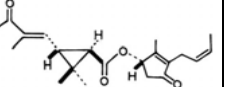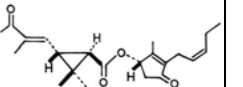
Chemical Properties

There are six biologically active chemicals in pyrethrins that are responsible for the knockdown properties of the insecticide (Moorman and Nguyen, 1997). They are divided into two groups: pyrethrins I and pyrethrins II. Table 1 shows the distribution of the active chemicals found in pyrethrins. Pyrethrin I, cinerin I, and jasmolin I are esters of chrysanthemic acid whereas pyrethrin II, cinerin II, and jasmolin II are esters of pyrethric acid. Chrysanthemic acid and pyrethric acid combine with one of three alcohols (pyrethrolone, cinerolone, and jasmololone) to form the respective six active ingredients (Head, 1973). The chemicals are clear, viscous, high boiling gums in pure form (Head, 1973). Group I pyrethrins are insoluble in water (Table 1) but soluble in hydrocarbons and organic solvents such as alcohol (WHO, 1975; Todd et al., 2003). According to Todd et al. (2003), pyrethrins are non-volatile at ambient temperatures (WHO, 1975), with low vapor pressures and low Henry's constants, and large octanol/water coefficients (Table 1). Synthesis of pyrethrin I, jasmolin I, and cinerin I from (*S*)-Allethrolone has been reported by Ando and Casida (1983).

Toxicity

Pyrethrins affect the nervous system of insects, causing paralysis and a "knockdown" effect. They bind to sodium channels of nerve cells, prolonging their opening, and thereby causing possible death (Tomlin, 2000). The effect of pyrethrins on the production of ATP was studied by Kakko et al. (2000), who found that pyrethrins decreased the activity of total ATPase in cerebral synaptosomes of rat brain by 15% and 40% at 0.1-10 µM and 100-1000 µM concentrations, respectively. Also, at pyrethrins

concentration of 0.1-100 μM, no effect on the activity of Mg^{2+}-ATPase was observed in rat brain synaptosomal membranes but the activity increased 100% when the

Table 1. The distribution of the active ingredients of pyrethrins and some of their important physical-chemical properties.

Source	*Chrysanthemum* flower extract					
Group	Pyrethrins I			Pyrethrins II		
% composition[1]	14.8			15.2		
Chemical compound	pyrethrin I	cinerin I	jasmolin I	pyrethrin II	cinerin II	jasmolin II
% composition[1]	11.4	2.2	1.2	10.5	3.5	1.2
Chemical structure						
Chemical formula[1]	$C_{21}H_{28}O_3$	$C_{20}H_{28}O_3$	$C_{21}H_{30}O_3$	$C_{22}H_{28}O_5$	$C_{21}H_{28}O_5$	$C_{22}H_{30}O_5$
Molecular weight[1]	328.4	316.4	330.4	372.4	360.4	374.4
CAS number	121-21-1	25402-06-6	4466-14-2	121-29-9	121-20-0	1172-63-0
Boiling point in °C[2] (mm Hg)	170 (0.1)	136-138 (0.008)	-	200 (0.1)	182-184 (0.001)	-
Vapor pressure (mm Hg) [2,3]	2.02×10^{-5}	1.1×10^{-6}	4.8×10^{-7}	3.9×10^{-7}	4.6×10^{-7}	1.9×10^{-7}
Water solubility[4] (mg/L)	0.35	3.62	0.60	125.6	1038	214.8
K_{ow} (Log)[4]	5.62	4.77	5.43	3.56	2.71	3.37
Henry's Law(atm-m³/mol)	7.7×10^{-7}	9.6×10^{-7}	1.3×10^{-6}	7.4×10^{-10}	9.2×10^{-10}	1.2×10^{-9}
BCF[4]	11,000	2500	4700	300	70	210
Volatilization[4] (µg/cm³/h)	0.89	1.98	1.18	0.65	1.38	1.80
Henry's Lawconstant[4]	4.3×10^{-3}	9.4×10^{-4}	3.3×10^{-3}	8.9×10^{-6}	2.2×10^{-6}	1.4×10^{-5}
Log K_{oc}[4]	4.43	3.97	4.33	3.31	2.85	3.21
Volatilization t½ soil[4] (days)	1.8	2.7	1.9	73.2	97.0	36.8

[1] Head, 1973.
[2] Tomlin, 2000
[3] Todd et al., 2003
[4] Estimated. Crosby, 1995

concentration of pyrethrins was increased to 1000 μM. Thus, the study showed that the activity of total ATPase may be a target for the neurotoxic action of pyrethrins. The effects of pyrethrins on insects, mammals, and aquatic organisms have been studied in some detail and are summarized below.

Insects

Pyrethrins are highly toxic to insects because they are contact poisons that rapidly penetrate into the nervous system. Numerous studies on the effectiveness of pyrethrins against insects have been conducted and they have proved to be a highly effective "knockdown" agent for a wide range of insect species. Insects, however, can become resistant to the compound via the production of enzymatic detoxifiers as found in a study by Farnham (1971). Therefore, it is not considered a satisfactory insecticide against some agricultural pests (Atkinson et al., 2004) but the activity of pyrethrins can be enhanced by additive compounds that suppress detoxification within the insect (Elliot and Janes, 1973). For instance, the use of pyrethrins (5%) with poperonyl butoxide (15%) at ultralow volume (ULV) spray concentrations on caged mosquitoes placed in and near houses in the malarious coastal regions of El Salvador resulted in high *Anopheles albimanus* mortalities; *A. albimanus* is the vector of malaria that is highly resistant to DDT, dieldrin, and malathion (Hobbs, 1976). Results after 1 hour following ULV pyrethrins spraying showed that *A. albimanus* females were knocked-down in percentages that ranged from 81.5 to 100 during eight weekly trials; thus, the vector population in the test village was markedly reduced. Subsequently, the number of malaria cases in the village decreased by 42% over one year while in the control village (no spraying), the number of malaria cased increased by 75% (Hobbs, 1976). A similar study, using 0.6% pyrethrins and 1.4 % piperonyl butoxide as an aerosol at a rate of 9.4 and 7.6 g/100 m^3 by Sullivan et al. (1976) against *A. albimanus* in Panama showed 100% effectiveness within 24 hours. Darwazeh and Mulla (1981) observed good control (96%) of *Culex tarsalis* mosquito larvae in experiment ponds by using pyrethrins in toss-it form (briquettes) at a rate of 44 g a.i. (active ingredient)/ha. However, they found at the applied rate, non-target organisms were also affected. Following five successive weekly treatments of pyrethrins and 42 days after the first treatment (44 g/ha), a 37% reduction of *Gambusia affinis*, the mosquito fish, was observed. At the rate of 88 g/ha, a 67%

6

reduction of *G. affinis* was observed by Darwazeh and Mulla (1981). Dragonfly naiads were found to withstand an application rate of 66 g/ha. The authors recommended not exceeding an application rate of more than 44 g/ha in order to avoid toxicity to surface-frequenting fish.

In addition to effectively controlling mosquitoes, pyrethrins have been found in several studies to be effective against numerous other insects. For instance, the chemical is effective at controlling *Dermanyssus gallinae*, the poultry red mite, in birds (budgerigars) according to a study by McGarry and Trees (1991); an 87 and 70% reduction of *D. gallinae* was observed for pyrethrins treated budgerigars and pigeons, respectively, after within 48 hours. The mite has been found to occasionally bite humans and is a common ectoparasite in aviaries and pigeon lofts where it can breed rapidly. Another pest known to bite humans and animals is *Culicoides variipennis* (midget fly). The fly has been found to be the vector for bluetongue viral disease in sheep and cattle (Luedke et al., 1967). Woodward et al. (1985) determined that the pyrethrins LC_{90} (added as pyrenone) for midget fly larvae in an aqueous medium was 16.8 μg/L at 23°C. Field pond tests at a concentration of 131 part per billion (ppb) resulted in a 98% reduction of the larvae and successful suppression of adult emergence. Due to the potential of rapid photolysis in the daytime, the authors suggested a nighttime application of pyrethrins.

Pyrethrins are also effective against common houseflies. Sheppard and Swedlund (1999) determined the LD_{50}'s of each of the six chemicals in pyrethrins (Table 2). The table clearly shows that the individual chemicals are less effective against the common housefly than in their mixed state as pyrethrins (Sheppard and Swedlund, 1999).

Table 2. Average LD_{50}'s of the individual compounds in pyrethrins for *Musca domestica L.* (housefly).

Chemical compound	Average LD_{50} (μg/fly)
Pyrethrin I	0.20
Pyrethrin II	0.49
Cinerin I	1.77
Cinerin II	0.43
Jasmolin I	1.28
Jasmolin II	0.46
25% pyrethrins	0.11

Table 3 shows that bees are highly sensitive to pyrethrins (57.8% a.i.) administered orally and through contact. Given that bees play a vital role in the pollination of agricultural crops as well as fulfill important niches in natural ecosystems, pyrethrins use near bee populations should be carefully evaluated.

Table 3. Pyrethrins toxicity to bees.

Insect	Test	Concentration (µg/bee)	Reference and Notes
Honey bees	Oral LD$_{50}$	22	USEPA PED[*]
Bees	Contact LD$_{50}$	130-290	Tomlin, 2000

[*] U.S. EPA PED = U.S. EPA Pesticide Ecotoxicity Database

Birds and Mammals

Pyrethrins display relatively low acute mammalian and avian toxicity (Table 4 and 5). Animal studies associate mortality with only relatively high oral exposures of pyrethrins (Todd et al., 2003).

Table 4. Pyrethrins toxicity to birds at 20% a.i. unless stated (U.S. EPA PED[*]).

Bird	Test (a.i.)	Concentration (mg/kg)
Bobwhite Quail	Dietary LC$_{50}$ (57.8%)	5620
Japanese Quail	Dietary LC$_{50}$	5000
Mallard Duck	Dietary LC$_{50}$	5000
Mallard Duck	Dietary LC$_{50}$ (57.8%)	5620
Pheasant	Dietary LC$_{50}$	5000
Mallard Duck	NOEL[a] (57.8%)	1780
Bobwhite Quail	NOEL[a]	3160

[a] NOEL = no observable effect level
[*] U.S. EPA PED = U.S. EPA Pesticide Ecotoxicity Database

According to a WHO report (1975), the skin system and respiratory tract of mammals do not absorb large amounts of pyrethrins. However, it is poorly absorbed through the gastrointestinal tract (WHO, 1975). A study by Wester et al. (1994) showed that pyrethrins and piperonyl butoxide (used as a synergist) applied to the ventral forearm of six human volunteers at a concentration of 0.3% and 3%, respectively, resulted in 7 day urinary accumulations of 1.9% pyrethrins and 2.1% piperonyl butoxide of the applied doses. The main mode of absorption was via percutaneous absorption. Percent absorption for pyrethrins varied among six human subjects between the forearm (0.6 to 4.1%

sorption) and the scalp (2.4 to 16.4% sorption). In Rhesus monkeys, however, administration of pyrethrins into the body (100% bioavailability) by avoiding the digestive canal (parenteral studies) showed that 22.5% of the chemical was excreted in the urine seven days after administration (Wester et al., 1992). The compound has been found to rapidly degrade in the stomach by hydrolysis of the ester bond to harmless metabolites (Class et al., 1990).

Table 5. Pyrethrins toxicity to mammals

Mammal	Test	Concentration (mg/kg)	Reference
Rat	Oral LD_{50}	500-1000	WHO, 1975
Rat	Dermal LD_{50}	>1800	WHO, 1975
Rat (male)	Oral LD_{50}	2370	Schoenig, 1995
Rat (female)	Oral LD_{50}	1030	Schoenig, 1995
Rat (male)	NOAEL[b]	710	Schoenig, 1995
Rat (female)	NOAEL[b]	320	Schoenig, 1995
Rat	NOAEL[b]	150	USEPA, 1988
Rat (male)	2 yr. NOAEL[b]	130	USEPA, 1994
Rat (female)	2 yr. NOAEL[b]	173	USEPA, 1994
Rabbit	NOAEL[b]	150	USEPA, 1988
Rabbit	11% weight loss	300	USEPA, 1988
Rat	4 hour Inhalation LC_{50}	3.4 mg/m^3	Schoenig, 1995
Mouse (male)	18 month NOAEL[b]	686	USEPA, 1994
Mouse (female)	18 month NOAEL[b]	834	USEPA, 1994

[b] NOAEL = no observable adverse effect level

The metabolic fate of pyrethrins in rats has been partially defined in microsomal oxidase system (Casida et al., 1971; Elliott et al., 1972). The sites of metabolic attack on the pyrethrins and the metabolites (Figure 1 and 2) were determined by Class et al. (1990). They found that for cinerin I, jasmolin I and pyrethrin I, the major sites of oxidation are the methyl groups at 10, 10' and 11', methylene groups at 7' and 10', and double bonds at 7, 8, 8', 9', 10', and 11' (Figure 1). The cinerin II, jasmolin II and pyrethrin II compounds are oxidized in mouse liver via the methyl groups at 5 and 6, and double bond at 8' and 9' (Figure 2). The oxidation and hydrolysis products are excreted from the body in urine but non-degraded parent compounds can be excreted via the small intestinal tract. Pyrethrins are not known to accumulate in mammalian tissues; male rats receiving 3 mg/kg orally demonstrated almost complete metabolism within 100 hours (WHO, 1975). Urine analysis showed no presence of pyrethrins but metabolites were present.
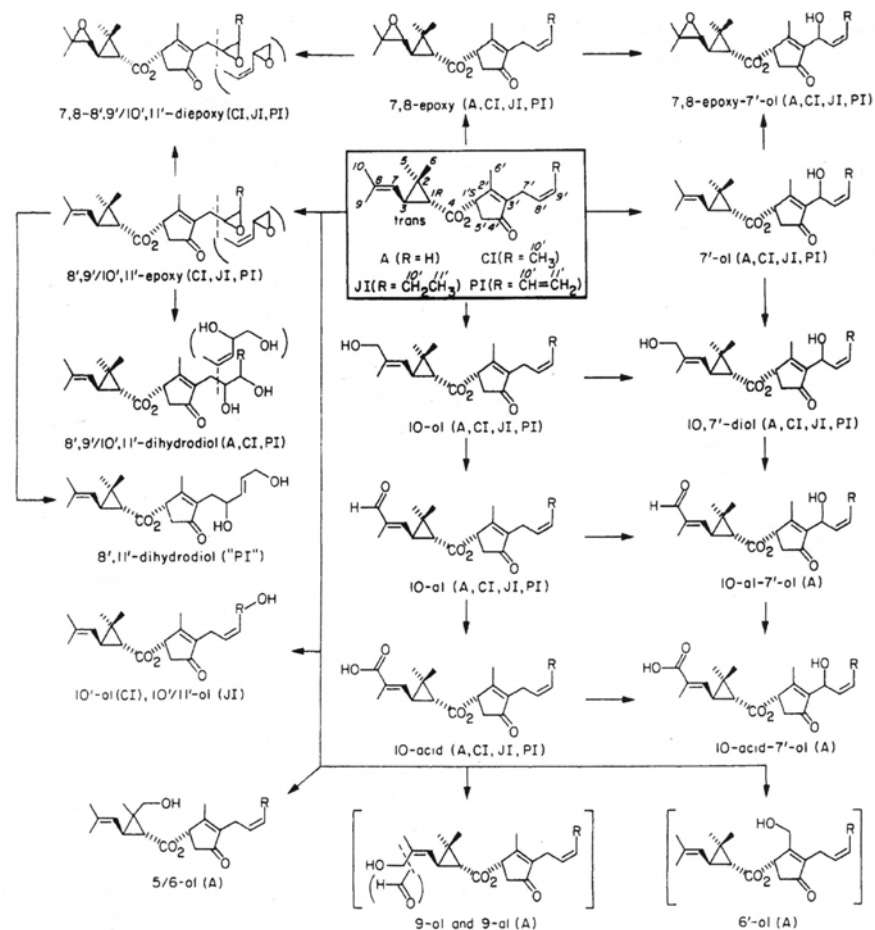
Figure 1. Partial metabolic pathways for cinerin I (A), jasmolin I (CI), and pyrethrin I (PI) in mouse and rate live microsomal oxidase systems (Class et al., 1990).
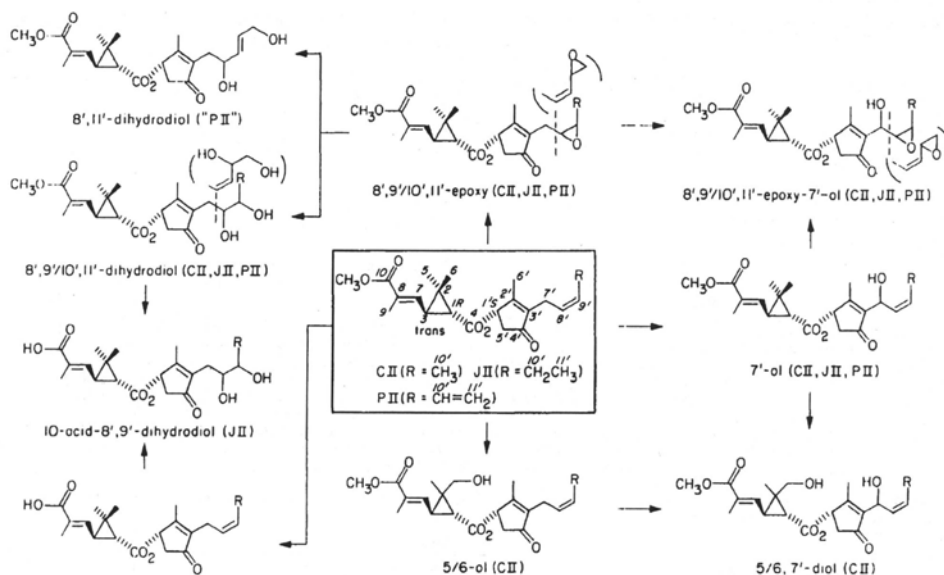

Figure 2. Partial metabolic pathways for cinerin II, jasmolin II, and pyrethrins II in mouse liver microsomal oxidase systems (Class et al., 1990).

Aquatic Organisms

Pyrethrins bioconcentrate in aquatic organisms and appear to be toxic to many fish (Table 6 and 7) at ppb concentrations. For example, according to the U.S. EPA, the LC_{50} for several fish (Table 6) is below 50 µg/L in static systems and even lower (less than 25 µg/L) in flow through systems (Table 7). Other aquatic organisms are sensitive to pyrethrins as well. For instance, water fleas have a 50% effect concentration of 25 µg/L. Table 7 also shows that as the percentage of active ingredient is increased (from 20 to 57.5%), the 50% lethal concentration decreases. These findings suggest that aquatic organisms are highly sensitive to pyrethrins and use of the compound near water bodies or waterways must be carefully evaluated prior to its application.

Table 6. Pyrethrins 96 hour static system aquatic toxicity at 20% a.i. (U.S. EPA PED[*])

Aquatic Organism	Test	Concentration (µg/L)
Daphnid (crustacean)	48 hour EC_{50}	42
Scud (crustacean)	LC_{50}	1.4
Water Flea	EC_{50}	25
Atlantic Salmon	LC_{50}	40
Bluegill Sunfish	LC_{50}	41
Channel catfish	LC_{50}	9.0
Chinook salmon	LC_{50}	44
Largemouth bass	LC_{50}	33

[*]U.S.EPA PED = U.S.EPA Pesticide Ecotoxicity Database

Table 7. Pyrethrins 96 hour flow through system aquatic toxicity at 20 and 57.5% a.i. (U.S. EPA PED[*])

Aquatic Organism	Test	a.i. (%)	Concentration (µg/L)
Mysid (crustacean)	LC_{50}	57.5	1.4
Water Flea	48 hour EC_{50}	57.5	11.6
Bluegill Sunfish	LC_{50}	57.5	10
Brown Trout	LC_{50}	20	19.4
Coho Salmon	LC_{50}	20	23
Lake Trout	LC_{50}	20	19.7
Rainbow Trout	LC_{50}	20	20
Rainbow Trout	LC_{50}	57.5	5.1
Sheephead Minnow	LC_{50}	57.5	16
Smallmouth Bass	LC_{50}	20	22

[*]U.S. EPA PED = U.S. EPA Pesticide Ecotoxicity Database

Environmental Degradation

Although pyrethrins are one of the oldest natural pesticides currently in use, there are limited environmental fate data available. Therefore, many of environmental fate parameters have been estimated using chemical property estimation methods as opposed to determined in laboratory or field studies (Schwarzenbach et al., 2003).

Air

One of the primary methods of applying pyrethrins is by spray application. Thus, spray drift following its application must be considered. The pyrethrins display low to moderate estimated Henry's Law constant. Consequently they have the potential to volatilize from water and moist soils. However, in natural aqueous systems the potential for volatilization would be mitigated by their sorption to soils and sediment. Their vapor pressures are also low to moderate. This, in conjunction with their moderately high estimated organic carbon normalized partition coefficient (K_{oc}) suggests they may exist in both particulate and vapor phases in the atmosphere. However, vapor phase compounds are susceptible to rapid degradation via direct photolysis and by reaction with hydroxyl radicals, ozone, and nitrate radicals (Todd et al., 2003). While particulate phase pyrethrins would be expected to degrade at a slower rate and potentially travel long distances before being removed from the air by wet (i.e., snow, rain) and/or dry (i.e., dust) deposition, there are no available data demonstrating this phenomena. Because pyrethrins are commonly used indoor, their volatilization rate from solid surfaces is important. Crosby (1995) estimated volatilization rates for pyrethrins (Table 1), concluding that group I pyrethrins are more volatile than group II pyrethrins, similar to the relative values of their estimated Henry's Law constants.

Soils and microbial interactions

Estimated pyrethrins K_{oc}'s are moderate to high (Table 1), ranging from 700 for cinerin II to 27200 for pyrethrin I (Crosby, 1995). The sorption of pyrethrins to soil increases with increasing soil organic matter. Compost amended soil, having two times greater organic matter content than native soil, was found to absorb more pyrethrins and their mobility was reduced by humic acids, a major component of organic matter (Antonious et al., 2004). It was also determined by Antonious et al. (2004) that increasing

the humic acid concentration significantly reduced the mobility of pyrethrins. Further, Antonious et al. (2001) found that compost with high organic matter content absorbed more of pyrethrin I (0.056 µg/g) than no-mulch (0.026 µg/g) and fescue strip soils (0.002 µg/g) in potato field trials. The study indicated that pyrethrin I bound strongly to soils (K_{oc} = 26915) while pyrethrin II did not (K_{oc} = 2042). These experimental data are similar to the estimates of Crosby (1995) who reported higher K_{oc}'s for the pyrethrins of group I as compared to group II.

There are a few reports that examine the effects of pyrethrins on microbial communities. One such study examined the influence of pyrethrins on soil microbial flora in relation to changes in nutrient levels. Taiwo and Oso (1997) found that pyrethrins and glucose addition increased the bacterial and actinomycetes populations up to three weeks after treatment but then the populations declined over time. The decrease suggested that some of the microbial populations that were tolerant to pyrethrins were susceptible to the degradation products of the chemical.

Crosby (1995) has predicted the microbial degradation of pyrethrins occurs via oxidative metabolism (Figure 3). These oxidative processes are expected to occur at unsaturated side-chains, reactive methylene groups, and secondary alcohol groups of pyrethrins (structures 2 and 3 in Figure 3). Degradation of the cyclic rings is expected to be slower compared to the unsaturated side chains. Figure 3 shows that the structures 2, 3, 4, 5, 6, and 7 (Figure 3), all maintain their ring structure while oxidative mechanisms affect their side-chains. Crosby (1995) predicted that mineralization of pyrethrins in normal soils and natural waters would be rapid.
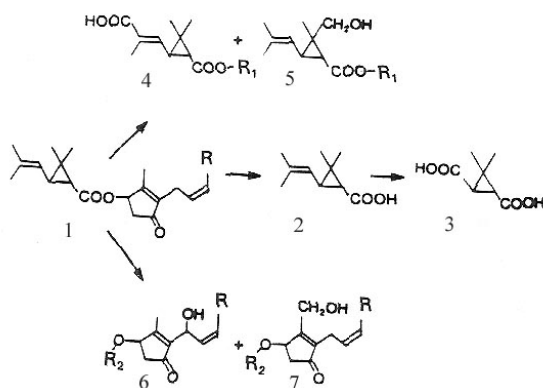

Figure 3. Predicted microbial degradation routes of pyrethrins (Crosby, 1995) where 1 is an individual compound of pyrethrins.

13

Water

The estimated octanol/water (K_{ow}) partition coefficients (Table 1) suggest that group I are more lipophilic than the group II pyrethrins that also have higher estimated water solubilities. Antonious et al. (2001) similarly found the K_{ow} for pyrethrin I (416,869) to be higher than the more soluble than pyrethrin II (3,631). The estimated bioconcentration factor (BCF) data, the ratio of the concentration of a chemical in an animal to that in the animals environment, are also consistent with the solubility and K_{ow} results, with higher BCF for the more lipophilic group I pyrethrins. These values are based on K_{ow} estimation methods using fragment constants (Crosby, 1995). However, Crosby (1995) has shown that the estimated values are very similar to the experimentally observed K_{ow} values by Briggs et al. (1983) and solubility values by Tomlin (2000) for pyrethrin I and II.

Runoff studies have not been extensive, but Antonious et al. (1997) reported the concentration of pyrethrins in runoff water averaged 36.1 ng/L after 11 days of pyrethrins application to rototilled bare soils.

Photochemistry

Pyrethrins degrade rapidly when exposed to natural sunlight and do not persist in the environment beyond a few weeks (Todd et al., 2003). Figure 4 shows the degradation of pyrethrins under light and dark environments. Clearly evident in figure 4 is that under dark conditions, there is little degradation of pyrethrins over time, however in the light there is rapid degradation from 100% to less than 1% within 5 hours. The photochemical degradation of the acid components of pyrethrins has been examined in numerous studies. Sasaki et al. (1970) and Ueda and Matsui (1971) found that the carbon 1 and 3 (Figure 2) of the main cyclopropane group gets cleaved and subsequently, according to Elliot and Janes (1973), leads to the formation of a di-radical. The compound can then undergo fragmentation to a senecioate, rearrangement to a lactone or become the parent material by the reformation of the bond between the carbon 1 and 3 (Figure 2). However, of more importance is the photochemical degradation of the esters. Photodegradation of pyrethrins is rapid in the presence of oxygen and sunlight. For instance, Chen and Casida (1969) studied the photochemical oxidation of pyrethrins and found that pyrethrin I is

14

stable for 24 hours under nitrogen and oxygen conditions in the dark but highly unstable in the presence of oxygen and light (Figure 4).
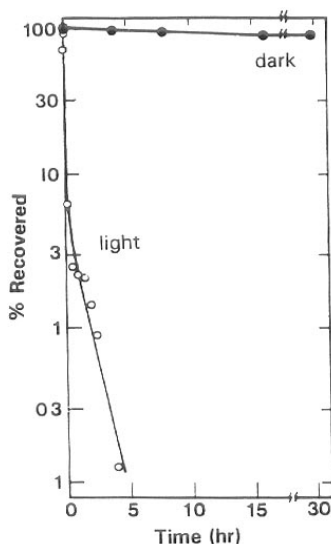

Figure 4. Photochemical reactions of pyrethrins where (●) and (○) indicates pyrethrins degradation in the dark and light, respectively (Crosby, 1995).

Other degradation variables

Temperature has been found to be a critical factor in the rate of degradation of natural pyrethrins whereas moisture, oxygen and microbial activity did not play a major role (Atkinson et al. 2004). The study was conducted because harvested Tasmanian crops of *Tanacetum cinerariaefolium* resulted in substantial loss in pyrethrins during storage. According to the study, 26, 65, and 68% of the pyrethrins were lost (before stabilization) at 20, 60, and 100°C, respectively. Since the pyrethrins did not reach zero concentration, it was suggested that plant structure might provide chemical or physical protection to the compounds.

Tsumura-Hasegawa et al. (1992) examined the presence of pyrethrins in post harvest-treated potatoes during storage and processing into starch. A shorter half-life was observed for cinerine and jasmolin at -5°C as compared to 19.7°C. Pyrethrin I and II had two phase decay curves where the first phase displayed a shorter half-life at -5°C than at the higher temperature, while the second phase showed a longer half-life at -5°C. They also found that in unprocessed potatoes the concentration of pyrethrins ranged from 0.113 to 0.453 ppm. However, the washing, homogenization, and filtration steps during starch

15

production greatly reduced the amount of pyrethrins. Less than 1% (0.4 to 1 ppm) of the initial pyrethrins concentration was present in the dried starch. No detectable pyrethrins (pyrethrin I and II) were found at ppb levels of detection in processed apple, pear, squash, and carrot baby foods (Moore et al., 2000).

Summary

Pyrethrins are generally effective insectides that display low toxicity to mammals, breakdown quickly in the environment due to light or air interactions, and are rapidly metabolized. For instance, the photodegradation of pyrethrins in sunlight is rapid and results in the isomerization of the side-chains, photooxidation to a variety of carboxylic acids, and isomerization of the cyclopropane acids. The aqueous hydrolysis data indicate that pyrethrins are slow to degrade in water; however, in the presence of microbial communities, the degradation is expected to be faster via oxidative metabolism. The compound is toxic to some fish and aquatic invertebrates, so applications in proximity to aquatic habitats may potentially be a problem. Other parameters such as the dissipation in the soil and environment show that pyrethrins I have a higher K_{ow}, BCF, volatility, and K_{oc} and lower water solubility than pyrethrins II. This suggests that pyrethrin I, cinerin I, and jasmolin I may be more "mobile" in the environment than pyrethrin II, cinerin II, and jasmolin II.

References

Ando, T., and Casida, J. E. 1983. Conversion of (S)-Allethrolone to pyrethrin I, jasmolin I, cinerin I, and [propenyl-3-^{13}C]- and [propenyl-3-^{14}C]-(S)-bioallethrin. J. Agric. Food Chem. 31: 151-156.

Antonious, G. F., Patel, G. A., Snyder, J. C., and Coyne, M. S. 2004. Pyrethrins and piperonyl butoxide adsorption to soil organic matter. J. Environ. Sci. Health. B39: 19-32.

Antonious, G. F., Byers, M. E., and Kerst, W. C. 1997. Residue levels of pyrethrins and piperonyl butoxide in soil and runoff water. J. Environ. Sci. Health. B32: 621-644.

Antonious, G. F., Snyder, J. C., and Patel, G. A. 2001. Pyrethrins and piperonyl butoxide residues on potato leaves and in soil under field conditions. J. Environ. Sci. Health. B36: 361-271.

Atkinson, B. L., Blackman, A. J., and Faber, H. 2004. The degradation of the natural pyrethrins in crop storage. J. Agric. Food Chem. 52: 280-287.

Barthomeuf, C., Hitmi, A., Veisseire, P., and Coudret, A. 1996. Identification and assay of pyrethrins in *Chrysanthemum cinerariaefolium* calli. Biotech. Tech. 10: 639-642.

Briggs, G. G., Elliott, M., and Janes, N. F. Present status and future prospects for synthetic pyrethroids. In "Pesticide Chemistry; Human Welfare and the Environment". J. Miyamoto and P. C. Keareny (eds.). Pergamon Press. New York, NY. 1983. pp 157-164.

Casida, J. E., Quistad, G. B. Pyrethrum; a benefit to human welfare. In "Pyrethrum flowers; Production, Chemistry, Toxicology, and uses". J. E. Casida and G. B. Quistad (eds.). Oxford University Press. New York, NY. 1995. pp 345-350.

Casida, J. E., Kimmel, E. C., Elliott, M., and Janes, N. F. 1971. Oxidative metabolism of pyrethrins in mammals. Nature. 230: 326-327.

Chen, Y-L., and Casida, J. E. 1969. Photodecomposition of pyrethrin I, allethrin, phthalthrin, and dimethrin. J. Agr. Food Chem. 17: 208-215.

Class, T. J., Ando, T., and Casida, J. E. 1990. Pyrethroid metabolism: microsomal oxidase metabolites of (S)-bioallethrin and the six natural pyrethrins. J. Agric. Food Chem. 38: 529-537.

Crosby, D. G. Environmental fate of pyrethrins. In "Pyrethrum flowers; Production, Chemistry, Toxicology, and uses". J. E. Casida and G. B. Quistad (eds.). Oxford University Press. New York, NY. 1995. pp 194-213.

Darwazeh, H. A., and Mulla, M. S. 1981. Pyrethrins tossits against mosquito larvae and their effects on mosquito fish and selected nontarget organisms. Mosquito News. 41: 650-655.

Elliott, M. and Janes, N. F. Chemistry of the natural pyrethrins. In "Pyrethrum; The Natural Insecticide". J. E. Cadisa (eds.). Academic Press. New York, NY. 1973. pp 84-91.

Elliott, M., Janes, N. F., Kimmel, E. C., and Casida, J. E. 1972. Metabolic fate of pyrethrin I, pyrethrin, II, and allethrin administered orally to rats. J. Agric. Food Chem. 20: 300-313.

Farnham, A. W. 1971. Changes in cross-resistance patterns of houseflies selected with natural pyrethrins or resmethrin (5-benzyl-3-furylmethyl (±)-cis-trans-chrysanthemate). Pestic. Sci. 2: 138-143.

17

Gnadinger, C. B. Pyrethrum Flowers. 2nd Ed. McLaughlin, Gormley, King. Minneapolis, Minnisota. 1936.

Head, S. W. Composition of pyrethrum extract and analysis of pyrethrins. In "Pyrethrum; The Natural Insecticide". J. E. Cadisa (eds.). Academic Press. New York, NY. 1973. pp 25-49.

Hobbs, J. H. 1976. A trial of ultralow volume pyrethrin spraying as a malaria control measure in El Salvador. Mosquito News. 36: 132-137.

Jones, G. D. G. Pyrethrum production. In "Pyrethrum; The Natural Insecticide". J. E. Cadisa (eds.). Academic Press. New York, NY. 1973. pp 17-21.

Kakko, I., Toimela, T., and Tahti, H. 2000. Piperonyl butoxide potentiates the synaptosome ATPase inhibiting effect of pyrethrin. Chemosphere. 40: 301-305.

Luedke, A. J., Jones, R. H., and Jochim, M. M. 1967. Transmission of bluetongue between sheep and cattle by *Culicoides variipennis*. Amer. J. Vet. Res. 28: 457-460.

McGarry, J. W., and Trees, A. J. 1991. Trap perches to assess the activity of pyrethrins against the poultry red mite Dermanyssus gallinae in cage birds. Experimental Appl. Acarology. 12: 1-7.

McLaughlin, G. A. History of pyrethrum. In "Pyrethrum; The Natural Insecticide". J. E. Casida (eds.). Academic Press. New York, NY. 1973. pp 3.

Moore, V. K., Zabik, M. E., and Zabik, M. J. 2000. Evaluation of conventional and "organic" baby food brands for eight organochlorine and five botanical pesticides. Food Chem. 71: 443-447.

Moorman, R., and Nguyen, K. 1997. Identification and quantitation of the six active compounds in a pyrethrin standard. J. Assoc. Off. Anal. Chem. 65: 921-926.

Sasaki, T., Eguchi, S., and Ohno, M. 1970. Studies on chrysanthemic acid. 4. Photochemical behavior of chrysanthemic acid and its derivatives. J. Org. Chem. 35: 790-793.

Schoenig, G. P. 1995. Mammalian toxicology of pyrethrum extract. In "Pyrethrum Flowers; Production, Chemistry, Toxicology, and uses. J. E. Casida and G. B. Quistad (eds.). Oxford University Press. New Youk, NY. pp 249-257.

Schwarzenbach, R. P., Gschwend, P. M., and Imbonden, D. M. Environmental Organic Chemistry. John Wiley & Sons Inc. Hoboken, NJ. 2003.

Sheppard, D. C., and Swedlund, B. 1999. Toxicity of individual pyrethrin esters to house flies (Diptera: Muscidae). J. Entomol. Sci. 35: 279-282.

Sullivan, W. N., Gebhart, W. A., Chaniotis, B. N., and Whitlaw, J. T. 1976. The effectiveness of pyrethrin and pyrethroid aerosols against mosquitoes endemic to Panama. Mosquito News. 36: 316-320.

Taiwo, L. B., and Oso, B. A. 1997. The influence of some pesticides on soil microbial flora in relation to changes in nutrient level, rock phosphate solubalization and P release under laboratory conditions. Agric. Eco. Environ. 65: 59-68.

Todd, G. D., Wohlers, D., and Citra, M. Toxicology Profile for Pyrethrins and pyrethroids. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Atlanta, GA. 2003.

Tomlin, C, D, S. The Pesticide Manual. 12th Ed. British Crop Protection Council. Surrey, England. 2000. pp. 178-179.

Tsumura-Hasegawa, Y., Tonogai, Y., Nakamura, Y., and Ito, Y. 1992. Residue levels of dichlorvos, chlorpropham, and pyrethrins in postharvest-treated potatoes during storage or processing into starch. J. Agric. Food Chem. 40: 1240-1244.

Ueda, K., and Matsui, M. 1971. Studies on chrysanthemic acid. 11. Photochemical isomerization of chrysanthemic acid and its derivatives. Tetrahedron. 27: 2771-2774.

U.S. EPA. 1994. Memorandum. Pyrethrum extract; Review of rat chronic feeding/ carcinogenicity study (IRDC 1990) and mouse carcinogenicity study (IRDC 1990). US Environmental Protection Agency. Office of Pesticide and Toxic Substances. Tox. Review 01079.

U.S. EPA. 1988. Memorandum. Pyrethrins; Submission of rat and rabbit teratology range finding studies in response to a prior request from Toxicology Branch. US Environmental Protection Agency. Office of Pesticide and Toxic Substances. Tox. Review 006824.

U.S. EPA. Pesticide Ecotoxicity database. http://www.epa.gov/oppefed1/general/databasesdescription.htm#ecotoxicity.

Wester, R. C., Bucks, D. A. W., and Maibach, H. I. 1994. Human invivo percutaneous absorption of pyrethrin and poperonyl butoxide. Food Chem. Tox. 32: 51-53.

Wester, R. C., Maibach, H. I., Melendres, J., Sedik, L., Knaak, K., and Wang, R. 1992. In vivo and in vitro percutaneous absorption and sink evaporation of isofenphos in man. Fundamental Appl. Tox. 19: 521-526.

WHO (World Health Organization). 1975. Data Sheet on Pesticides No. 11; Pyrethrins. www.inchem.org/documents/pds/pds/pest11_e.htm.

Woodward, D. L., Colwell, A. E., and Anderson, N. L. 1985. Use of pyrethrin larvicide to control Culicoides variipennis (diptera: ceratopogonidae) in an alkaline lake. J. Am. Mosq. Control Assoc. 1: 363-368.