Environment Conservation Journal 2 (2&3) 63-66, 2001
(ISSN 0972 - 3099)

Fenvalerate: A Synthetic Pyrethroid Toxicity to Fresh Water Fishes

K. S. Tilak and K. Yacobu

Department of Zoology, Nagarjuna University, Nagarjuna Nagar-522 510 A. P., India

Abstract

Synthetic Pyrethroids are the new major class of broad spectrum organic insecticides used in Agriculture. Among the Pyrethroids, Fenvalerate (RS a cyano 3 Phenoxy Benzyl RS 2-[4 Chlorophenyl] 3-Metyl Butyrate) is used extensively and is most effective Pyrethroid. The technical isomer is a mixture of four optical isomers 2 S, a S, 2R and a S. The 2S isomers are more toxic than 2R. They are toxic to aquatic organisms. When they are being transported into aquatic environment. The toxic action of the toxicant will depend on metabolism and mode of action in aquatic organism. Hence to study it static and continuous flow through tests were conducted to determine LC_{50} values to the freshwater fish. The possible mechanism of toxic action will be presented.

Key words: ***Fenvalerate -Toxicity - Freshwater Fishes - Toxicological Mechanism.***

Introduction

The pyrethroids are the esters of cyclopropane carboxylic acids with alkenyl methyl cyclopentenolene alcohols. The activity depends on the ester, and is influenced by the absolute configuration of the asymmetrical carbon atom at C1 of the cyclopropane ring and at C4 of the cyclopentenolene ring. All very active pyrethroids have R configuration at C1 and 'S' configuration at C4. The presence of gemdimethyl group at C2 of the cyclopropane ring is essential for a high insecticidal activity. The substituents at C3 of the cyclopropane ring either as C1s or trans position are not an absolute requirement because even withoutsubstituents the insecticidal action is present but only saturated side chains give high activity. The replacement of the isobutenyl group by an isobutyl decreases activity whereas a butadienyl group may enhance potency (Wouters and Berchen 1978). The important pyrethroids are Allethrin, Bioresmethrin, Permethrin, decamethrin, cypermethrin and fenvalerate. All of them are highly active and stable insecticidal pyrethroids, hence being used extensively in agriculture, to control the insect pests, to control the vectors of endemic diseases, to protect the seeds during storage and to fight household insects.

Due to this widespread usage, they will be transported into aquatic environment, which is the ultimate sink for all pesticides, and aquatic toxicity has become an integral part of the process of environmental hazard evaluation of toxic chemicals. Generally, the potential impact of pollutants is more on the aquatic organisms, than terrestrial environment (Murty 1986).

Among the Pyrethroids, the neurotoxic organic insecticide fenvalerate (alfa cyano-3-phenoxy-phenyl) methyl-4-chloro-(1methyethyl Benzene acetate) is used to control boll warms on cotton, tobacco plants and vegetables. It is being extensively used in the local areas and keeping the view of the transportation, accumilation, concentration, the toxic impact of the toxicant to the fresh water fish is attempted. The portenous, palatable precious table item can be linked to the human food chain and the possible deleterious effects can be given a due consideration.

Materials and Methods

The freshwater fishes (sizes of the fish are given in Table- 1) were brought from local fish farms and by fisherman. The fishes were acclimatized to the laboratory conditions in well-aerated non-chlorinated tap water at test medium conditions. During the period of acclimatization and experimentation the fish were not fed. If the number of deaths exceeded 5% in any batch of fish during acclimatization, the concerned batch was discarded. The studies on toxicity were conducted for technical as well as 20 Emuslifiable Concentrate (EC) employing static and continuous flowthrough systems as recommended in the report of the committee on methods of toxicity tests with aquatic organisms (Amon 1975).

The solutions of desired concentrations were prepared in acetone to yield a concentration of 100mg/mL. The control fish received an equal quantity of acetone to that of the highest concentration of the toxicant tested.

For flowthrough system, test solutions of desired concentrations were prepared once in five hours in glass reservoirs and were let into the test containers through thin-walled polythene tubes. The flow rate was adjusted with regulators such that four litres of water passed through containers in one hour. The conditions of the test medium were temperature 28 ± 2 °C, oxygen 6-8 ppm, Hardness 80 mg/L, Alkalinity - 425 mg/L and pH 8.3. All the precautions laid down in the report of committee on toxicity tests were followed (Amon 1975).

The pilot experiments were conducted to determine the concentrations causing mortality of test fish in the range of 10 to 90%. For each concentration, 10 fish were tested and the experiment was repeated thrice. Probit analysis (Finney 1971) as recommended by Roberts and Boyce 1972 was followed to calculate the LC_{50} values. The technical fenvalerate and 20% EC were supplied by Searle (India) Limited, Mumbai.

Results and Discussion

The earlier reports on the toxicity of fenvalerate to the fishes are: Bradbury and Coats 1982, Bradbury *et al.*, 1985, 86, 87 Coats and Jeffery 1979, Zitko et al, 1977, 79, Hya 1989 and Anitha *et al.* 1999 a & 1999 b. The LC_{50} values were given in the Table 1. The toxic action of fenvalerate is accounted in earlier reports due to its change as metabolites or due to certain biochemical changes in the tissue.

Table 1: The LC_{50} Values and their 95% confidence limits offenvalerate technical grade to freshwater fishes for 96 hours in static and continuous flow through system.

Name of the Fish and toxicant	Length of the fish	LC_{50} value for 96 hours	
		Static	Continuous flow through system mg/L
Labeo rohita			
Fenvalerate (Technical)	2-4 cm	0.012	0.0031
20% EC	2-4 cm	0.016	0.0037
Catla Catla			
Fenvalerate (Technical)	2-4 cm	0.004	0.0018
20% EC	2-4 cm	0.017	0.0006
Cirrhinus mrigala			
Fenvalerate (Technical)	2-4 cm	0.002	0.0001
20% EC	2-4 cm	0.016	0.0050
Aplochelus panchax			
Fenvalerate (Technical)	2-4 cm	0.027	0.014
20% EC	2-4 cm	0.002	0.009
Ctenopharyngodon idellus			
Fanvalerate (Technical)	3-5 cm	1.9	1.90
20% EC	3-5 cm	2.60	1.70

The values are tested for chisquare and found to be not significant at $P < 0.05$. The 95% confidential limits are also calculated.

Generally, there are three main chemical reactions involved in the degradation of a toxicant i.e. isomerization, hydrolysis and oxidation (Demoute 1989). The fenvalerate has the common degradation products in mammals, birds and Amphibians. The basic metabolic reactions are similar in all, i.e. cleavage of ester bond and oxidation of released acid and alcohol fragments. The initial transformations are followed by conjugation with sugars or amino acids which allows faster exertion.

Same type of degradation is reported in fish also, Bradbury *et al.* 1987 studied the toxicokinetics of fenvalerate in rainbow trout and reported the uptake of the gill. The qualification of the residues of the gill tissues; confirmed the isomer of venvalerate which is toxic i.e. 2s isomer the degradation product. The qualification and quantification of the residues proves that fenvalerate is highly toxic fro fish and is species specific. In vertebrates except fish pyrethroids as opposed to organochlorines, which have the same ligophilicity are readily metabolized and excreted. The half-life periods never go beyond a few days. Birds have a larger capacity to eliminate their products than mammals. The high toxicity to fish is partly explained by their poor ability to metabolize them. Takimotto *et al.* 1986 reported that the lower rates of elimination and metabolism of fenvalerate in trout is the factor responsible for higher toxicity.

Their metabolism is largely oxidative in fish. Edwards *et al.* 1987. The fenvelerate acid moiety is very different from the other groups of pesticides and the ester cleavage releases the acid fragment, which is mainly excreted or as a glucorinide conjugate. The cyanide ion is transformed into thiocyanate and also carbon dioxide. Because of this, the freshwater fish are affected due to toxic action of fenvalerate.

The toxicity varies in LC_{50} value of 96 hours in continuous flow through system as well as static. The residue analysis by TLC and GLC (Anitha Susaan *et al.* 1996 & 1999) showed the deposition in fish tissues as residues. In the Table 1, toxicity data of the experiments confirmed that the commercial formulation is more toxic than the technical compound. The metabolites formation, storage of the pesticide after manufacture and as there was no strict quality control all of them are causing synergistic effect to the fish apart from the above mentioned reasons of earlier reports. Although, the pyrethroids are safest insecticides, when administered orally. But they may become highly toxic when they reach central nervous system in sufficient concentration. The sensitivity of the fishes in the present study to technical grade fenvalerate and 20% EC formulation was in the order of

Cirrhinus mrigala > Catla Catla > Labeo rohita >
Aplocheilus panchex > Ctenopharyngodon idellus.

Hence the formulation products must be viewed seriously before any pesticide is given a representation to be introduced into the environmental usage.

References

Anita Susan T., Veeeraiah K. and Tilak K. S. 1999 a. Biochemical changes in the tissues of the fish *Catla catla* exposed to fenvaleratse a synthetic pyrethroid. ***J. Ecobiology* 11(2), 109-116.**

Anita Susan T., Veeraiah K. and Tilak K. S. 1999 b. A study on the Bioaccumulation of Fenvalerate a synthetic pyrethroid in the whole body tissue of *Labeo rohita*, *Catla catla*, *Cirrhinus mrigala* by GLC. ***Enviromedia* 18(1), 57-59.**

Amon 1975. *Committee on Methods of Toxicity Tests with Fish, Macroinvertebrates and Amphibuians* EPA Oregon **61 pp.**

Bradbury S. P. and Coats J. R. 1982. Toxicity of fenvalerat4e to bobwhite quail including brain and liver residues associated with mortality. ***J. Toxicol. & Environ. Health.* 10, 307-319.**

Bradbury S. P., Coats J. R. and Mckim J. M. 1985. Differential toxicity and uptake of two fenvalerate formulation in fathead minoows (Pimephales promelas) ***Environ. Toxicol. Chem.* 4, 533-541.**

Bradbury S. P., Coats J. R., Mc Kim J. M. 1986. Toxicokinetics of fenvalerate in rainbow trout Salmo gairdneri. ***Environ. Toxicol. Chem.* 5, 567 - 576.**

Bradbury S. P., McKim J. M. and Coats J. R. 1987. Physiological response of rainbow torut to acute fenvalerate intoxificatin. Pestic Biochem. ***Physiol.*** **27, 275-288.**

Coats J. R. and O'D Jefery N. 1979. Toxicity of four synthetic pyrethroid insecticides to rainbow trout. ***Bull Environ. Contam. Toxicol.*** **23, 250 -255.**

Demoute J. P. 1989. A brief review of the environmental fate and metabolism ofpyrethroids. ***Pestic. Science.*** **27, 375-385.**

Edwards R. and Millburn P. 1985. The metaboism and toxicity of insecticides in fish. In: Hutson D. H. and Roberts T. R. (eds) *Progress in Pesticides Biochemistry and Toxicology Vol.5*. Insecticides John Wiley and Sons Ltd., New York, **pp. 249-274.**

Finney D. J. 1971. *Probit Analysis*. Cambridge University Press, **333pp.**

Haya K. 1989. Toxicity of pyrethroid insecticides to fish. ***Environ. Toxicol. Chem.***. **8, 381-391.**

Murty A. S. 1986. *Toxicity of Pesticides to Fish vol. I & II.* CRC Press, Inc.

Roberts and Boyce C. B. C. In: Norris and Ribbons, D. W. (eds.) *Methods of Microbiology vol. 7A*. Academic Press, New York, 1972.

Takimotto Y., Matsyda O. M. T. and Miyamoto M. 1986. Metabolism of several pyrethroid insecticides by carp cyprinus caarpio:Abstract The sixth international congress of pesticides chemistry August 10-15, 1986. International Union of pure and applied toxicology.

Wouters W. and Bercken J. V. D.1979. Action Pyrethroids. ***General Pharmacology***. **9, 387 398.**

Zitko V., Carson W.G. and Metcalfe C. D. 1977. Toxicity of pyrethroids to juvenile Atlantic salmon. ***Bull Environ. Contam. Toxicology***. **18, 35-41.**

Zitco V., McLeese D. W., Metcalfe C. D. and Carlson W. G. 1979. Toxicity of permethrin, decamethrin and related pyretrhroids to Salmon (*Salmo Saler*) and lobster. ***Bull. Environ. Contam. Toxicol.*** **21,338-343.**