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Total proteins effects of Decamethrin on the Liver, Muscle and Gonads of the freshwater fish *Anabas testudineus* (Bloch)

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ABSTRACT

This study includes the alterations induced by chronic (30 days) exposure of the fish *Anabas testudineus* to a sublethal concentrations (0.106 ppm) of Decamethrin on the profile of total protein in the liver, muscle and gonads.

The muscle showed maximum depletion of total protein content amounting 31% ($p < 0.001$), followed by liver 30% ($p < 0.001$), testis 19% ($p < 0.05$) and ovary 17% ($p < 0.01$). The present study therefore points towards a severe metabolitic dysfunction in response to permethrin toxicity in the fish *A. testudineus* (Bloch).

Keywords: *Anabas testudineus, Chronic, Pesticides, Gonads.*

INTRODUCTION

Recently it is quite clear that synthetic pyrethroids are being encouraged as a substitute for the organochlorine, organophosphorous and carbamate group of pesticides due to their low mammalian toxicity and high bio-efficacy and their toxicological impacts on fishes being very little explored prompted the present worker to undertake this study.

The recently introduced synthetic pyrethroids, *viz*. Deltamethrin, Cypermethrin, Fenvalerate, Permethrin *etc*. with multiple beneficiary qualities have been in much use by farmers in pest control. Agricultural runoff is a known source of pyrethroids, and they are often bound to sediments and particulate matter in runoff (Domagalski *et al.* 2010, Weston *et al*. 2009). They have gained prominence in agricultural usage because of their low mammalian toxicity and high bio-efficacy (Chatterjee *et al*. 1986). But there have been found to be highly toxic to fish (Richardson, 1988) very scanty information is available as regards the toxic effects of synthetic pyrethroid insecticides to fishes. Fenvalerate caused decrease in the haematological parameter in *Channa marulius* which suggest that the fenvalerate (synthetic pyrethroids) may weak the immune system and result in severe physiological problem ultimately to the death of fish (Patole *et al*., 2016;

Ghosh and Chatterjee, 1988; Radhiah and Rao, 1988; Reddy and Bashamoideen, 1989; Golowe and Godzi, 1994; Srivastava *et al*., 1995; Choudhary, 1996; Singh, 1999; Singh, 2002; Pandey, 2003; Mukhopadhyay, *et al*., 2005; Rita and Milton, 2006; Singh *et al.*, 2007; Muthulakshmi *et al.* 2008; Jha, 2009; Poonam *et al.*, 2010; Yadav and Jha, 2010; Kumar and Jha, 2011 and Yadav, 2013.

MATERIALS AND METHODS

The air-breathing teleost *Anabas testudineus* procured live from the local fish market were washed with 0.1% $KMnO_4$ solution to remove dermal infection if any. Healthy fish of

average length (9–12cm) and weight (21–25 g) were acclimated for 15 days to laboratory conditions. The fish were fed with chopped goat liver every day adlibtum. Running tapwater was used in all the experiments and the fish were adjusted to natural photoperiod and ambient temperature. No aeration was done.

Static acute bioassays were performed to determine LC_{50} values of fenvalerate for 24, 48, 72 and 96 hours following the methods of APHA, AWWA & WPCF (1985). The LC_{50} values for these periods were 0.0295 ppm, 0.1625 ppm, 0.0148 ppm and 0.0055 ppm respectively. The sublethal concentration was determined following the formula of Hart *et al.* (1945). Twenty acclimated fish were exposed to a sub-lethal concentration (0.165 ppm) of permethrin for 30 days. Side by side same number of fish as that of experimental one was maintained as the control group. At the end of exposure period the fish were anaesthetized with 1:4000 MS 222 (tricane, methane, sulfonate, sandoz) for two minutes. The liver, muscle, testis and ovary were quickly dissected out, weighed to nearest mg and processed for the quantitative estimation of glycogen by the methods of Varley *et al.* (1980).

RESULTS AND DISCUSSION

The glycogen profiles of liver, muscle, testis and ovary in response to decamethrin exposure showed a significant decline. The liver and muscle showed statistically more significant decline. The liver and muscle showed statistically more significant ($P < 0.001$) decline *i.e.* 31% in muscle, while 30% in liver. The testis showed significant ($P < 0.05$) while ovary showed significant at ($P < 0.01$). The testis showed decline 19% while ovary 17%. Total protein in the control liver, muscle, testis and ovary was estimated to be 169.33 ± 1.56, 145.82 ± 1.68, 74.58 ± 1.45, and 109.81 ± 1.62 respectively. As against there, the total protein profiles in the experimental lots were 118.28 ± 1.24, 100.40 ± 1.27, 60.16 ± 0.98 and 90.26 ± 0.79 respectively (Table 1).

Table 1

Profiles of total protein (mg/g wet tissue) in tissue of *Anabas testudineus* chronically exposed to decamethrin for 30 days. Values are mean ± SE 0f 5 observations.

Tissue	Control	Decamethrin
Liver	169.33±1.56	*** 118.28±1.24 (– 30.14)
Muscle	145.82±1.68	*** 100.40±1.23 (– 31.14)
Testis	74.58±1.45	* 60.16±0.98 (– 19.33)
Ovary	109.81±1.62	** 90.26±0.79 (– 17.80)

Decrease (–) over control values significant at

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

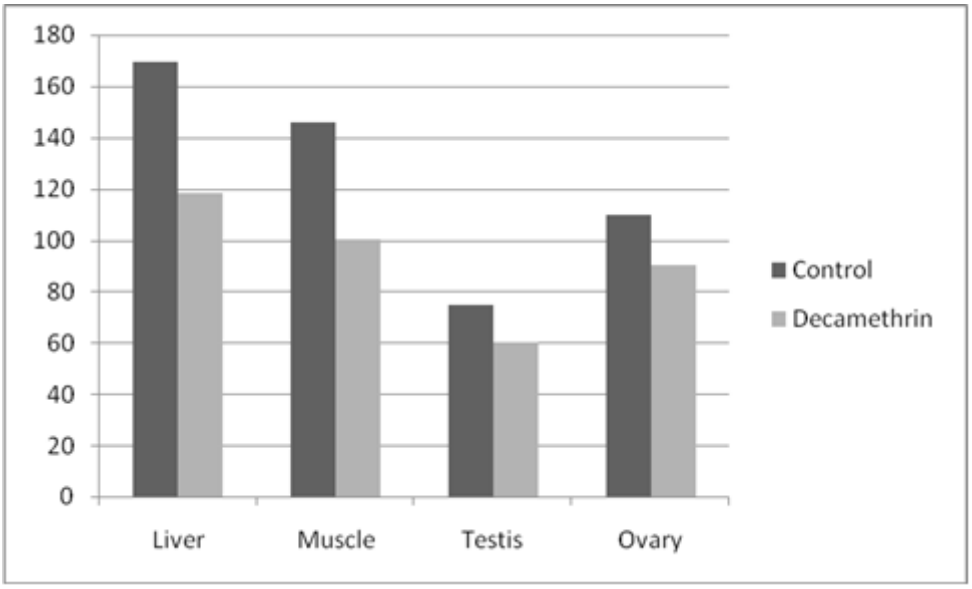

Fig. 1: Profiles of total protein (mg/g wet tissue) in tissue of liver, muscle, testis and oary of *Anabas testudineus* chronically exposed to decamethrin for 30 days.

The level of tissue protein in control fish recorded in the present study indicates that proteins are the largest contributors to the wet weight of the tissues after water. Previous workers have also reported decline in tissue protein profiles in a number of fish species exposed to various pesticides and pyrethroids. Murty and Devi (1982) found reduced protein content in liver of *Channa punctatus* exposed to endosulfan and associated the same with degradation as also the detoxification of endosulfan. Ramalingam and Ramalingam (1982) noted a steady decline in the total protein of liver and muscle after 7 and 15 days exposure of the fish, *Sarotherodon mossambicus* to malathion and mercury and correlated it with an intensive proteolysis. Similarly, a significant decrease in the protein content was recorded by Kumar and Ansari (1984) in the Zebra fish, *Brachydanio rerio,* exposed to malathion and suggested inhibition of protein synthesis by the toxicant. The liver of *Clarias gariepinus* exposed to the cypermethrin showed hyperplastic hepatic and necrosis of hepatic cells (Andem A. B. *et al.* 2016).

The present findings on tissue proteins are in conformity with those of Ravinder *et al.* (1988); Reddy and Bhagyalakshmi (1994) and Jabde *et al.* (1995). A steep decline in protein content was noted by Ravinder *et al.* (1988) in brain, liver, kidney, gill and muscle of the decis (decamethrin) exposed *Clarias batrachus* and suggested increased proteolysis and possible utilization of the products of their degradation for metabolic purposes. Similar decline in protein content of hepatopancreas and muscle tissue of fresh water crab *O. senex* exposed to sublethal concentration of fenvalerate was reported by Reddy and Bhagyalakshmi (1994) who have also pointed out an acceleration of protein catabolism during fenvalerate intoxication. Similarly, in an earlier study, Jeba Kumar *et al.* (1990) observed that when *L. thermalis* was exposed to sublethal concentration of cypermethrin, a decreased protein level was evident from first day onwards.

Proteins being involved in the architecture and physiology of the cell seem to occupy a key role in the cell metabolism. The observed significant depletion of tissue protein in the present case denotes high catabolic potency of those organs and may be attributed to the intensive proteolysis and utilization of their degradation products for metabolism under the toxic influence of Herbiclon. They might have been fed into TCA cycle through aminotransferase system to cope with the excess demand of energy during stressful situations

as suggested by Jha (1991). The observations of Saleem and Shakoori (1985) on the loss of tissue proteins during permethrin intoxication offer support for the observed decrease of protein level in tissues. Moreover, the decreased protein contents might also be attributed to the tissue destruction, necrosis, or disturbance of cellular function and consequent impairment in protein synthetic machinery (Bradbury *et al.,* 1987). Further, the loss of gonadal proteins may also be associated to the direct action of pyrethroids leading to arrest of vitellogenesis in ovary and loss of germ cells in testis (Jha and Jha, 1995 b), Choudhary (1996), Singh (1999), Singh (2002), Rita and Milton (2006), Singh *et al* (2007), Muthulakshmi *et al*. (2008), Jha (2009), Poonam *et al.* (2010), Yadav and Jha (2010), Kumar and Jha (2011), Yadav (2013). Thus we see that protein metabolism is also impaired under the sublethal chronic stress of pyrethroids which induced compensatory mechanism in the tissues resulting in proteolysis.

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