International Journal of Ecology and Environmental Sciences 49: 353-361, 2023
ISSN: 2320-5199 (Online); https://doi.org/10.55863/ijees.2023.2519

Studies on the Toxic Effects of Synthetic Pyrethroid Insecticide, Cyphenothrin on Protein Metabolic Profiles of Indian Major Carp, *Cirrihinus mrigala*

SAPNA S. ANIGOL, SRINIVAS B. NEGLUR AND MUNISWAMY DAVID*
Department of P.G.Studies in Zoology, Karnatak University Dharwad, 580003, Karnataka, India
E-mail: sapnaanigol11@gmail.com, srinivasneglur@gmail.com, davidkcd@gmail.com
***Corresponding author**

ABSTRACT
Pyrethroids are used in almost all agricultural crops, nurseries, various urban structural and landscaping sites, construction sites (pre-construction termiticides), the pyrethroids is their efficacy against a broad range of insects, pests and mites, low mammalian and avian toxicity, low potential to contaminate ground water, and relatively low application rates like several desirable characters to contribute. Cyphenothrin is the synthetic pyrethroid insecticide widely used throughout the world to control domestic pests, public health and industrial location. Cyphenothrin is more toxic to insects and mammals and last longer in the environment. Freshwater fish, *Cirrihinus mrigala* were exposed to sublethal concentration one fifth of LC_{50} 30 μg/L (6 μg/L) of cyphenothrin for 10, 20, 30 and 40 days to analyze various parameters of protein metabolism in functionally different tissues. Total, structural and soluble proteins showed decrease; whereas free amino acids and the activities of protease, aspartate aminotransferase and alanine aminotransferase significantly increased in cyphenothrin exposed fish. Interestingly, ammonia content decreased but urea and glutamine increased at all periods of exposure. It was also observed that alterations steadily increased with the period of exposure and exhibited tissue specificity. Thus variation in the protein metabolism of the fish exposed to cyphenothrin indicates its toxic effect on the cellular metabolism thereby leading to impaired protein synthetic machinery.

Key words: Cyphenothrin toxicity; Protein metabolism; *Cirrihinus mrigala*

INTRODUCTION

Water contamination is normally brought about by different human sources, regularly modern offices and agrochemicals particularly in aquatic biological system, has turned into a genuine natural issue now a days. These agrochemicals and industrial wastes releases may diverted successfully by rains, winds, streams and floods into the huge water bodies and change their physico-chemical properties with high harmfulness. The water pollution make harms aquatic life particularly to fishes which are extremely sensitive to wide scope of toxins in the water (Herger et al. 1995).

Various types of fish show dynamic take-up and aggregation of numerous toxic chemicals like herbicides, pesticides, heavy metals and polychlorinated biphenyls from water bodies. Among every one of these agrochemicals, pesticides viewed as very poisonous to species and furthermore to the established pecking order of aquatic environments. The amassing of pesticides delivers some physiological, biochemical and as well as morphological reactions in the freshwater fauna by impacting a few activities of metabolites and compounds shown by Ramamurthy et al. (1987).

The presence of xenobiotic, synthetic compounds and their subsidiaries in the aquatic water bodies and biological system due to their agrarian and modern applications are well documented. Most of these chemical compounds in the type of toxins and their subsidiaries are not degradable and have the chance of endogenous restricting particles as they enter the natural water bodies may prompt ominous harmful impacts (Amdur et al. 1991). The presence of these chemicals in a natural system can achieve bothersome changes in a cell which might be flowed to the tissue or the organ (Bernet et al. 1991). The possibility of these metals to amass in the aquatic biological system, especially fish, adversely affects the order of things. Human populaces who consume fish as an essential wellspring of food are additionally important for the food chain, consequently producing a reason for worry in broad daylight wellbeing (Di Giulio and Hinton 2008). The effect and bioaccumulation of toxins and their subordinates in the organs of aquatic species like fish rely upon an assortment of variables like closeness of the species

from the toxins, metabolic action and membrane transport potential, and climatic conditions (Abhijith et al. 2016).

The increase of biochemical substance in various tissues of fish because of harmful effect of various agrochemicals and insecticides has been accounted for by number of scientists (Nagrathnamma and Ramamurthy 1982, Desai et al. 2002, Remia et al. 2008, Hadi et al. 2009, Ganeshwade 2011). Organophosphates are most favoured bug sprays in horticulture because of their viability, less diligent life and simple detoxification in creature tissues which straightforwardly hinder AchE (acetylechelenesterase) action saw by (Rao et al. 2005) in fish and other amphibian organic entities.

To reduce pest resistance and undesired toxicity to non-target organisms, newer generations of insecticides, such as pyrethroids, have been developed to replace older classes of pesticides including organochlorine, organophosphate and carbamate insecticides (Denholm et al. 2002). A new synthetic pyrethroid Cyphenothrin (cyano-(3-phenoxyphenyl) methyl 2,2-dimethyl-3-(2-methylprop-1-enyl)cyclopropane1carboxylate), Cyphenothrin has been classified as moderately toxic by International Programme on Chemical Safety (IPCS). Trials carried out elsewhere showed promising results against mosquito (*Culex pipiens*), housefly (*Musca domestica*), and German cockroach (*Blatella germanica*).

Huge quantities of these poisonous chemical compounds launched from untreated effluents from factories and from runoffs of pesticide-treated agricultural fields enter our bodies of water the place they are dangerous to individuals of the ecosystem, predominantly fishes. In last few years, synthetic pyrethroid pesticides have been developed for use in agriculture and public health. Current investigation on merchandise like pesticide developed from natural Pyrethrins possessing excessive insecticidal potency, low mammalian toxicity, and very quick persistence. Nevertheless, this compound is highly poisonous to fish and to positive aquatic invertebrates.

In animals, physiological and biochemical changes can lead to physiological stress and can be correlated to structural and functional changes in cell proteins. Protein metabolism is one of many compensatory mechanisms in phrases of metabolic homeostasis in any stress condition. Although lots statistics is on hand on the biotransformation of pesticides in distinct animals, facts are inadequate about synthetic-pyrethroid-induced adjustments in cellular metabolism with reference to proteins. Hence, right here we tried to study the impact of Cyphenothrin on protein metabolism in tissues of the freshwater fish *Cirrhinus mrigala*.

MATERIALS AND METHODS

Experimental animals

Healthy freshwater fish, *Cirrhinus mrigal* weighing 9±2 g were obtained from State Fisheries Department, Saundatti Belgaum District. India and were maintained in the laboratory tanks with continuous aeration. Fish were maintained in large cement tanks (6×3 feet) which were duly aerated. Water in the tanks was treated with 1% KCl solution prior to the introduction of the animals into the tank. Fish were fed with balanced nutritious food pellets (Nova, Aquatic P. Feed) and allowed to acclimatize for a period of 14 days at 24 ºC temperature and 12-14 hours of photoperiod. Water in the tanks was renewed daily and the Physico-chemical parameters of water were examined according to the guidelines of APHA.

Experimental pesticide

Cyphenothrin (Type II pyrethroid) (Sumitomo Chemical India Pvt. Ltd., Gujarat) was procured from the local agricultural market of Dharwad, Karnataka, India. The expiry date of the test substance was confirmed prior to the initiation of the exposure. Stock solution was prepared by mixing the calculated volume of the commercial solution with distilled water. Test concentrations for acute toxicity test (i.e. 30 mg/l) and sub-lethal biochemical toxicity test (6 mg/l) were prepared by serial dilution of the stock solution using variable micropipette (Neglur et al. 2020).

Bioassay of cyphenothrin toxicity

Toxicity evaluation was conducted by static bioassay method (Finney 1971) and the LC_{50} for 96h value of cyphenothrin to *Cirrhinus mrigal* was found to be 30µg/L. One fifth of LC_{50} was selected as nominal sublethal concentration (6µg/L) and used in the

present investigation to analyze the sublethal effects at exposure periods (EPs) of 10, 20, 30 and 40 days.

***In vivo* exposure**

Fish in batches of six each were exposed to sublethal concentration of cyphenothrin (6µg/L) in glass aquaria with each fish having one litre of water. The aquaria containing fish were aerated to prevent hypoxic or anoxic conditions. Fish in tanks without the addition of cyphenothrin served as control group. After the stipulated time of 10, 20, 30 and 40 days, fish were killed with a blow on the head and the tissues (gill, kidney and liver) were isolated and transferred to cold fish Ringer solution and used for the estimation of different parameters.

Estimation of organic constituents

One percent homogenate of the tissues were prepared in 0.25 M ice cold sucrose solution using a motor-driven Yorko speed control homogenizer for the estimation of total proteins (TP), structural proteins (StP) and soluble proteins (SP) with Folin phenol reagent (Lowry et al.1951) using bovine albumen serum as standard. This homogenate was precipitated with 10% trichloro acetic acid and the protein free supernatant was processed for free amino acids (FAA) estimation by the addition of ninhydrin reagent (Moore et al. 1954). Tyrosine was used as standard.

Analysis of nitrogenous end products

Five percent homogenate of the tissues were prepared in distilled water for ammonia and glutamine and in 15% perchloric acid for urea. Levels of ammonia were known using ammonium chloride as standard, urea by diacetyl monoxime method (Natelson 1971) and glutamine by acid hydrolysis method (Colowick and Kaplon 1967).

Assay of enzymes

Five percent homogenate of the tissues were prepared in 0.25 M ice cold sucrose solution for aspartate aminotransferase (AAT) and alanine aminotransferase (ALAT); in ice cold distilled water for protease and these were centrifuged at 2500 rpm for 10 min in a refrigerated centrifuge at 4°C to remove cell debris and clear cell free extracts were used as enzyme source. Protease activity was measured (Moore et al. 1954) with the reaction mixture containing 100 lm of phosphate buffer (pH 7.0) and 12 mg of denatured protein. AAT and ALAT activities were assayed following the method of Reitman and Frankel (1957). The incubation mixture for AAT contain 100 lm of phosphate buffer (pH 7.4), 2 lm of ketoglutarate and 50 lm of L-aspartic acid (pH 7.4). For ALAT, incubation steps followed are the same as described for AAT except for the substrate used was D-alanine (50 lm). The standard graph was prepared with sodium pyruvate. All spectrophotometric measurements were determined using Baush and Lomb spectronic 20.

Statistical analysis

Average of six individual estimations were taken after pooling them and the mean values of control and experimental fishes were subjected to statistical treatment using one-way analysis of variance (ANOVA). If the difference between control and experimental the values were considered as significant at 5% level.

RESULTS AND DISCUSSION

Total, Soluble, Structural proteins

The information in Table 1 shows notable critical increment contrasted with control, in the solvent, underlying and complete proteins of three distinct utilitarian organ tissues of fish, *Cirrihinus mrigal* at all the exposed periods under sub lethal induced of cyphenothrin. Under sublethal, absolute protein level recorded critical decline in the organs of fish at day 40 (119.39) yet increased in 10 (107.42), 20 (111.33), and 30 (144.27) days in gills. In Kidney it has increased in every one of the exposed days and in liver increased level of concentration of cyphenothrin is viewed when contrasted with the control individually. In Structural protein, Gills presented to every one of the times of exposure it increased. In kidney, 40 days decreased when contrasted with different days in 10, 20 and 30 days. Liver increased at 40 days when contrasted with 10, 20 and 30 days, contrasted with control separately. In solvent proteins, Gills, kidney and liver increased in every one of the times of exposure contrasted with control individually (10≥20≥30≥40).

Table 1. Biochemical changes in different tissues of fish, *Cirrihinus mrigal* exposed to cyphenothrin.

Tissue	Control	Sub lethal Exposure Periods in Days			
		10	20	30	40
Total Proteins					
Gills	103.2513^{E}	107.4217^{D}	111.3302^{C}	144.2784^{A}	119.3933^{B}
SD±	0.003327	0.000374	0.000374	0.000374	0.000374
% Change	————	-4.0390	-7.8245	-39.7352	-15.6337
Kidney	101.7712^{D}	96.6428^{E}	105.3467^{C}	106.9707^{B}	109.2717^{A}
SD±	0.000374	0.000374	0.000374	0.000402	0.000374
% Change	————	-5.0391	-3.5132	-5.1090	-7.3699
Liver	144.4634^{E}	147.6248^{D}	148.6117^{C}	152.4232^{B}	157.38^{A}
SD±	0.5167	0.000374	0.000374	0.000374	5.16088
% Change	————-	-2.1883	-2.8715	-5.5099	-8.9410
Structural Proteins					
Gills	36.6239^{E}	38.3467^{D}	40.2351^{C}	41.9319^{B}	43.7937^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————	-4.7040	-9.8602	-14.4933	-19.5768
Kidney	50.7447^{E}	51.4746^{D}	56.6417^{B}	59.9837^{A}	52.7650^{C}
SD±	0.000374	0.000349	0.000374	0.000374	22.4534
% Change	————	-1.4385	-11.6209	-18.2068	-3.9813
Liver	70.9707^{E}	72.7102^{D}	74.1247^{C}	74.9767^{B}	77.4237^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————	-2.4510	-4.4440	-5.6445	-9.0924
Soluble Proteins					
Gills	67.9375^{E}	69.7093^{D}	71.6978^{C}	73.3987^{B}	76.8937^{A}
SD±	0.00037	0.000392	0.000374	0.000418	0.000374
% Change	————-	-2.6081	-5.5349	-8.0386	-13.1829
Kidney	55.7393^{E}	58.9312^{D}	60.9646^{C}	62.8293^{B}	64.9772^{A}
SD±	0.000349	0.000374	0.000374	0.000463	0.000418
% Change	————-	-5.7264	-9.3745	-12.7199	-16.5735
Liver	74.2475^{E}	76.3092^{D}	78.1474^{C}	79.2834^{B}	82.4275^{A}
SD±	0.000374	0.000463	0.000374	0.000374	0.000374
% Change	————	-2.7768	-5.2525	-6.7825	-11.0172
Free amino acids					
Gills	12.1132^{E}	15.2832^{D}	17.1162^{C}	19.7612^{B}	21.5732^{A}
SD±	0.000498	0.000681	0.000787	0.000693	0.000675
% Change	————	-26.1698	-41.3021	-63.1377	-78.0966
Kidney	16.5722^{E}	17.2726^{D}	19.2621^{C}	21.3216^{B}	23.7662^{A}
SD±	0.000498	0.000681	0.000787	0.000693	0.000675
% Change	————-	-4.2263	-16.2314	-28.6588	-43.41
Liver	14.8372^{E}	18.2783^{D}	23.1672^{C}	28.1173^{B}	31.1074^{A}
SD±	0.001045	0.001151	0.001075	0.001158	0.000829
% Change	————-	-23.1924	-16.2314	-89.5054	-109.658

Means for a parameter in a row followed by the same letter are not significantly different ($P \leq 0.05$) from each other according to one-way analysis of variance (ANOVA) test

Table 2. Enzymological changes in different tissues of fish, *Cirrihinus mrigal* exposed to cyphenothrin.

Tissue	Control	Sub lethal Exposure Periods in Days			
		10	20	30	40
Protease Activity					
Gills	0.3562^{E}	0.3775^{D}	0.3913^{C}	0.4317^{B}	0.4414^{A}
SD±	0.000374	0.000374	0.000349	0.000374	0.000374
% Change	————-	-5.9797	-9.8587	-21.196	-23.9191
Kidney	0.3967^{E}	0.4024^{D}	0.4182^{C}	0.4507^{B}	0.4724^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————	-1.4368	-5.4197	-13.6123	-19.0824
Liver	0.3074^{E}	0.3267^{D}	0.3994^{C}	0.4024^{B}	0.4222^{A}
SD±	0.004343	0.000374	0.000374	0.000374	0.000374
% Change	—————	-6.2784	-29.9284	-30.9044	-37.3455
AAT					
Gills	0.2674^{E}	0.2791^{D}	0.2891^{C}	0.3011^{B}	0.3174^{A}
SD±	0.000942	0.000738	0.002745	0.00109	0.001642
% Change	————-	-4.3754	-8.1151	-12.6028	-18.6986
Kidney	0.2550^{E}	0.2714^{D}	0.2941^{C}	0.3119^{B}	0.3441^{A}
SD±	0.000942	0.000912	0.000982	0.000167	0.000721
% Change	——-	-6.4314	-15.3333	-22.3137	-34.9412
Liver	0.2410^{E}	0.02497^{D}	0.2549^{C}	0.2917^{B}	0.3017^{A}
SD±	0.001444	0.000829	0.000525	0.000518	0.00074
% Change	————-	-3.6099	-5.7676	-21.0373	-25.1867
ALAT					
Gills	0.08185^{C}	0.09077^{B}	0.09918^{A}	0.1017^{E}	0.1178^{D}
SD±	0.000515	0.000374	0.000374	0.000374	0.000374
% Change	—————	-10.898	-21.1729	-24.2517	-43.9218
Kidney	0.1017^{E}	0.1022^{D}	0.1139^{C}	0.1222^{B}	0.1392^{A}
SD±	0.00037	0.00037	0.000374	0.000374	0.000418
% Change	—————	-0.4916	-11.9961	-20.1573	-36.939
Liver	0.06917^{D}	0.07967^{C}	0.09819^{B}	0.115167^{A}	0.1541^{E}
SD±	0.000374	0.000374	0.000374	0.000333	0.000374
% Change	———	-15.18	-41.9546	-66.4985	-122.784

Means for a parameter in a row followed by the same letter are not significantly different ($P \leq 0.05$) from each other according to one-way analysis of variance (ANOVA) test

Effect on FAA and protease activity

The expansion in FAA was seen in every one of the tissues of cyphenothrin-induced fish (Table 1). Liver displayed displayed by gill. Among the group liver showed most noteworthy protein movement. This augmentation was reliable with expansion in exposure periods. Critical modifications were seen at all periods besides in the liver tissue of fish exposed for 10 days. More rise of FAA levels when contrasted with different tissues at every one of the exposures. Protease movement was additionally raised in every one of the tissues and at all times of exposure (Table 2). Most extreme increase was

Effect on AAT and ALAT activities

Fish displayed altogether higher AAT and ALAT

movements in every one of the tissues during cyphenothrin toxicity (Table 2). The augmentation of ALAT in muscle at tenth day exposure period was unimportant. Movement of ALAT and its addition was more when contrasted with AAT in every one of the tissues.

Effect on ammonia, urea and glutamine levels

Ammonia level was decreased in the tissues of *C. mrigal* exposed to cyphenothrin (Table 3). Decrease of ammonia was critical in every one of the tissues and at the entire exposed test tried; a special case was in liver at all exposure period. Difference to this, levels of urea and glutamine showed an increasing pattern (Table 3). Addition of glutamine was more when contrasted with urea. Changes in both these nitrogenous excretory activities were huge in all the exposure period besides at day 10 exposure. During this time urea and glutamine levels of tissues and urea level of gill was inconsequential.

Proteins being associated with the structure and physiology of the cell, they appear to involve a vital activity in cell digestion. Catabolism of proteins and amino acids make a significant commitment to the all-out energy creation in fishes. The exhaustion of all the protein parts seen in this investigation (Table 1) can be corresponded to this reality. These outcomes are in concurrence with the previous report of Ravider et al. (1988) who showed what was going on in *Clarius batrachus* exposed to decis. Bradbury et al. (1987) brought up that the decreased protein content could likewise be ascribed to the obliteration or rot of cell digestion and ensuing hindrance in protein manufactured. Protein exhaustion in tissues might comprise a physiological component and may assume a part of compensatory system under cyphenothrin stress, to give intermediates to the Kreb's cycle. It has likewise been accounted for that this pattern of proteins was to upgrade osmolality to remunerate osmoregulatory issues experienced because of the spillage of ions and other required molecules during pyrethroid effects (Rafat Yasmees 1986).

Tissue explicit increase in protease action (Table 2) seen at all exposure periods was obviously reflected in the breakdown of proteins. Under proteolysis, improved breakdown rules over combination. While on account of anabolic cycle, increased union overwhelms the protein breakdown (Harper, 1979). In addition, histopathological harm and hydromineral unevenness during pesticide stress has been accounted for to represent the raised protease action (Moorthy et al. 1984). Upgraded protease action and decreased protein level has brought about a noticeable height of FAA content in every one of the tissues and at unsurpassed spans (Table 1).

The recognition of transaminases like aspartate transaminase (AAT) and alanine transaminase (ALAT) in blood have been clinically utilized to relate to any tissue harm. Presence of these biomarkers can likewise be coupled to the catalyst restraint in metabolic endless supply of pressure. There was a striking expansion in both AAT/ALAT fixations in treated fishes. These can be credited to the tissue and organ harm brought about by exposure to cyphenothrin (Abhijith et al. 2016). In like manner, showing the expected activity of transaminases as major biomarkers during exposure to toxins and following in metabolic pressure. AST and ALT are markers in liver capacity tests, as AST is blended by liver hepatocyte and commonly seen as in liver and heart, while ALT fundamentally is available in liver and kidney. In this way, higher movement of these compounds has been accounted for in fish presented to pesticides (Rao 2006).

The rise of AAT and ALAT activities seen in this investigation (Table 2) offers a great help to the above pattern. This is an obvious sign of shunting of amino acids into TCA cycle through oxidative deamination and dynamic transamination. Such a peculiarity was important to adapt up to the energy emergency during pyrethroid stress. It has additionally been recommended that pressure conditions overall instigate rise in the transamination pathway (Awasthi et al. 1984). Contribution of substitute pathways like aminotransferase responses are likewise conceivable because of restraint of oxidative chemicals like succinate dehydrogenase, malate dehydrogenase, isocitrate dehydrogenase and cytochrome-c-oxidase, as also reported by Ghosh (1989) in *Labeo rohita* under cyphenothrin toxicity.

The most conspicuous nitrogenous excretory units in teleostean fishes are ammonium salts and urea with ammonia in mass, as seen in this investigation. Ammonia, a harmful nitrogenous product, is

Table 3. Biochemical changes in different tissues of fish, *Cirrihinus mrigal* exposed to cyphenothrin

Tissue	Control	Sub lethal Exposure Periods in Days			
		10	20	30	40
Ammonia					
Gills	6.2806^{E}	6.8876^{B}	6.9366^{A}	6.6776^{C}	6.3677^{D}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————-	-9.6646	-10.4449	-6.3210	-1.3868
Kidney	8.1775^{E}	8.6607^{B}	8.8436^{A}	8.5517^{C}	8.3154^{D}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————-	-5.9089	-8.1455	-4.5759	-1.6863
Liver	4.1177^{E}	4.6176^{B}	4.7719^{A}	4.4892^{C}	4.2992^{D}
SD±	0.000374	0.000374	0.000374	0.000374	13.4936
% Change	——-	-12.1403	-15.8875	-9.02203	-4.40982
Urea					
Gills	0.3786^{E}	0.3993^{D}	0.4266^{C}	0.4978^{B}	0.56626^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	——	-5.46751	-12.6783	-31.4844	-49.5668
Kidney	0.5889^{E}	0.6677^{D}	0.6977^{C}	0.7078^{B}	0.7774^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	————-	-13.3809	-18.4751	-20.1902	-32.0088
Liver	0.5376^{E}	0.5993^{D}	0.6693^{C}	0.6913^{B}	0.7274^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	———	-11.4769	-24.5132	-28.59	-35.3051
Glutamine					
Gills	0.0567^{E}	0.07462^{B}	0.09432^{A}	0.1012^{D}	0.1432^{C}
SD±	0.00011	0.00014	0.00012	0.00057	0.00068
% Change	——-	-12.1605	-66.3492	-78.4832	-152.557
Kidney	0.9421^{B}	0.01092^{A}	0.1249^{E}	0.1632^{D}	0.1779^{C}
SD±	0.000074	0.00005099	0.00054	0.00059	0.00140
% Change	———	-88.4089	-32.5762	-73.23	-88.8335
Liver	0.1462^{E}	0.1732^{D}	0.1999^{C}	0.2019^{A}	0.1779^{B}
SD±	0.2762	0.00092	0.00058	0.00074	0.00077
% Change	———	-18.4679	-36.7305	-38.0985	-52.9412

Means for a parameter in a row followed by the same letter are not significantly different ($P \leq 0.05$) from each other according to one-way analysis of variance (ANOVA) test

delivered exogenously into the gastrointestinal system and endogenously into the tissues through catabolism of amino acids, pyrimidines and purines (Lowenstein and Goodman 1978). Ammonia can't be put away for longer timeframe in the body as it prompts endogenous ammonotoxicity. The decrease in ammonium salts content proposes that the ammonia could have been changed over into non-harmful mixtures, glutamine and urea, as proven in the current investigation. Srinivas Moorthy et al. (1986) recommended that the decrement of ammonia could likewise be because of decreased obsession of ammonium salts through keto acids prompting glutamate development by the activity of NADPH subordinate glutamate dehydrogenase. In this way, the arrangement of urea, glutamate and glutamine

by the particular proteins is by all accounts an elective pathway for the detoxification of ammonium salts. Further, this may likewise be credited to its fast dissemination into the surrounding medium. This can be upheld by the low measure of ammonium salts seen in gills of control fish, which fills in as the common excretory site. Prior, Cohen and Brown (1960) shown that the combination of glutamine is the significant ammonium salts detoxifying component.

The increased urea levels in liver tissue may be because of initiation of urea cycle. The presence of urea in extra hepatic tissues may be because of the vascular preparation and movement from liver. Also, decline in Na^+, K^+ and Ca^{2+} particles in the tissues of *C. mrigal* under cyphenothrin exposures could likewise represent the noticed height in urea and glutamine. It has been exhibited that both urea and glutamine repay the deficiency of osmolarity of the inward milieu under pressure condition (Sambasiva Rao 1983). Subsequently, an expansion in urea articulates the activity being played by various tissues of exposed fish in the height of ammonium toxicity other than their essential job in renewing the protein nitrogen to integrate helpful antecedents for the upkeep of homeostasis and dynamic harmony. Expanded degrees of urea and glutamine under pyrethroid stress uncover that the fish, *C. mrigal* could have adjusted to the biosynthesis of glutamine and urea as a significant pathway of detoxification of ammonia. Presumably this pathway might be useful to aquatic species particularly to fishes in detoxification and physiological remuneration or change in accordance with different exogenous and endogenous toxins.

CONCLUSIONS

Our outcomes show data about the injurious impacts of a pyrethroid insect spray, cyphenothrin on freshwater edible fish *C. mrigal*. From the outcomes, plainly the impact were portion subordinate and was not long enough for the total fix, as a few biochemical changes were as yet unique in relation to controls. This sort of data could be helpful to take preventive measure to safeguard the fishes from the dirtied regions.

ACKNOWLDGEMENTS

The Authors are thankful to PG Department of Studies in Zoology for providing animal maintenance facility.

Authors' contributions: All authors contributed equally

Conflict of interest: The authors hereby declare no conflict of interest

REFERENCES

Abhijith, B.D., Ramesh, M. and Poopal, R.K. 2016. Responses of metabolic and antioxidant enzymatic activities in gill, liver and plasma of *Catla catla* during methyl parathion exposure. The Journal of Basic and Applied Zoology, 77, 31-40.

Amdur, M.O., Dull, J. and Klaassen, C.D. 1991. Toxicology: The Basic Science of Poison. Pergamon Press.

Awasthi, M., Shaw, P., Dubale, M.S. and Gadhia, P.1984. Metabolic changes induced by organophosphates in the piscine organs. Environmental Research, 35, 320-325.

Bernet, D., Schmidt, H., Meier, W., Burkhardt-Hol, P. and Wahli T. 1991. Histopathology in fish: proposal for a protocol to assess aquatic pollution. Journal of Fish Diseases, 22, 25-34**.**

Bradbury, S.P., Mckim, J.M. and Coats, J.R. 1987. Physiological responses of rainbow trout, *Salmo gairdneri* to acute fenvalerate intoxication. Pesticide Biochemistry and Physiology, 27, 275-288.

Cohen, P.P. and Brown Jr., G.W., 1960. Ammonia metabolism and urea biosynthesis. In: Florkin, M. and Mason, H.S. (Eds.), Comparative Biochemistry. Academic Press, New York.

Colowick, S.P. and Kaplon, N.O. 1967. Methods in Enzymology.Academic Press, New York.

David, M., Mushigeri, S. B., Shivakumar, R. and Philip, G.H. 2004. Response of *Cyprinus carpio* (Linn) to sublethal concentration of cypermethrin: alterations in protein metabolic profiles. Chemosphere, 56(4), 347-352

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

Denholm, G.J. Devine, M.S. and Williamson, 2002. Insecticide resistance on the move. Science, 297, 2222-2223.

Di Giulio R.T. and Hinton D.E. 2008. The Toxicology of Fishes, CRC Press.1096 pages.

Finney, D.J. 1971. Probit Analysis, (3rd ed). Cambridge University Press, London, UK.

Ghosh, T.K. 1989. Influence of cypermethrin on the oxidative metabolism of *Labeo rohita*. Proceedings of the Indian National Science Academy. Part B Biological sciences, 55(2), 115-119.

Harper, H.A. 1979. In: Harper, H.A., Rodwell, V.W. and Mayes, P.A. (Eds.), Review of Physiological Chemistry. Lange Medical Publications, California.

Herger, W., Jung S.J. and Peter H. 1995. Acute and prolonged toxicity to aquatic organisms of new and existing chemicals and pesticides. Chemosphere, 31, 2707-2726.

Jebakumar, S.R.D., Flora, S.D.J., Ganesan, R.M., Jagatheesan, G. and Jayaraman, J. 1990. Effect of short-term sublethal exposure of cypermethrin on the organic-constituents of the fresh-water fish *lepidocephalichthys-thermalis*. Journal of Environmental Biology, 11(2), 203-209.

Lowenstein, J.M. nad Goodman, M.N. 1978. The purine nucleotide cycle in skeletal muscle. Fed. Proceedings, 37, 2308-2312.

Lowry, O.H., Rosebrough, N.J., Farr, A.L. and Randall, R.J. 1951. Protein measurement with the folin-phenol reagent. Journal of Biological Chemistry, 193, 265-273.

Moore, S., Stein, W.H.J., Farr, A.L. and Randall, R.J. 1954. A modified ninhydrin reagent for the photometric determination of amino acids and related compounds. Journal of Biological Chemistry, 221, 907-913.

Moorthy, K.S., Kasi Srinivasa Moorthy, K., Kasi Reddy, B., Swami, S.K. and Chetty, C.S. 1984. Changes in respiration and ionic content in tissues of freshwater mussel exposed to methylparathion toxicity. Toxicology Letters, 21, 287-291.

Natelson, S. 1971. In: Charless, G. (Ed.) Techniques of Clinical Chemistry. Spring Field III Inois, USA.

Neglur, S.B., Sanakal, R.D. and David, M. 2020. Studies on toxicological endpoints of Fenoxaprop-P-Ethyl on behavioural changes in freshwater exotic carp *Cyprinus carpio* (Linnaeus). Journal of Advanced Scientific Research, 11.

Neglur, S.B., Sanakal, R.D. and David, M. 2022. Fenoxapro-P-Ethyl induced biochemical changes in fresh water fish *Cyprinus carpio* under sublethal exposure. East African Scholars Journal of Agricultural Life Sciences, 5(1), 1-9.

Rafat Yasmees, M. 1986. Physiological responses of freshwater fish, Anabas scandens to the toxicity of Endosulfan. Doctoral Dissertation, Osmania University, Hyderabad, India.

Ramamurthy, R., Nagaratnamma, R., Jayasundaramma, B. and Ramarao, P. 1987. Histopathological lesions in the gills of freshwater teleost, *Cyprinus carpio* induced by methyl parathion. Matsya, 12, 144-147.

Rao, V.J. 2006. Sublethal effects of an organophosphorus insecticide (RPR-II) on biochemical parameters of tilapia, *Oreochromis mossambicus*. Comparative Biochemistry and Physiology - Part C, 143 (4), 492-498.

Ravi Shankar, P., Waghray, S. and Davi, I. 1992. The effect of sublethal concentration of synthetic pyrethroid cypermethrin to the common carp *Cyprinus carpio*. Journal of Environmental Biology, 13, 89-94.

Ravider, V., Suryanarayana, N. and Narayana, N. 1988. Decis induced biochemical alteration in a freshwater cat fish, *Clarias batrachus*. Indian Journal of Comparative Animal Physiology, 6, 5-12.

Reitman, S. and Frankel, S. 1957. A colorimetric method for the determination of serum glutamic oxaloacetate and glutamic pyruvic transaminases. American Journal of Clinical Pathology, 289, 56-63.

Sambasiva Rao, K.R.S. 1983. Impact of technical and commercial grade phenthoate on some selected parameters of oxidative metabolism in the fish, *Channa punctatus*. Journal of Environmental Biology, 8, 173-177.

Srinivas Moorthy, K., Kasi Reddy, B., Swami, K.S. and Chetty, C.S. 1986. Effect of pesticide dichloruos on succinate and malate dehydrogenases activity in fish, *Sarotherodan mossambicus*. Journal of Environmental Biology, 7, 101-106.

Received:27th April 2022
Accepted:23rd November 2022