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IMPACT OF PESTICIDE MONOCROTOPHOS ON SELECTED BIOCHEMICAL PARAMETERS AND HISTOLOGY OF LIVER OF *MYSTUS GULIO* (HAMILTON)

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ABSTRACT: The biochemical composition and liver histology of *Mystus gulio* were investigated after exposing the fish to the sublethal concentrations of $(1/10, 1/30 \text{ of } 96\text{h LC}_{50})$ monocrotophos. The 96h LC₅₀ value of the monocrotophos during acute exposure was found to be 0.40 ppm. Exposure of the fish to monocrotophos showed a significant decrease in carbohydrate, protein and lipid content at the end of 10, 20 and 30 days as compared to control. In the fish exposed to the monocrotophos, the most common histological lesions were dilation of blood sinusoids, vacuolization, hypertrophy and disintegration of cell boundaries in the liver. The lesions were comparatively most severe in 30 days exposed fish.

KEYWORDS: Mystus gulio, monocrotophos, biochemical parameters, histology of liver.

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1.INTRODUCTION

Pesticides may be categorized as insecticides, herbicides, defolients, fungicides and rodenticides [7]. Monocrotophos, an organophosphate pesticide, was observed to be less injurious than thiodon. The presence of pesticides in the aquatic system can obviously lead to multi- fold interaction with other forms of pollution. In India, scores of studies have been undertaken to estimate the acute toxicity level of various pesticides on aquatic fauna [4, 6, 36, 46]. Though the acute toxicity is an important parameter for toxicity tests, many workers emphasise more no sublethal toxicity than on acute toxicity [28]. The carbohydrate metabolism is disturbed when the animals are subjected to toxic tree

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications [9, 26, 27, 41]. Stressful situations in fish elicit neuroendocrine responses which in turn induce disturbances in carbohydrate metabolism [25]. Studied the impact of Malathion on protein content in the fish *Clarias batrachus* [5]. Studies were done on the toxic and sublethal effects of endosulfan on Barbar stigma [24]. Freshwater fish Channa punctatus was exposed to subacute concentrations of synthetic pyrethroid insecticides (cypermethrin and lambda-cyhalothrin) for 96 h to evaluate their impact on the levels of nucleic acids and protein in its different organs [19]. The results clearly indicated that both of these pyrethroids exerted their effects in a similar manner in fish liver but differed in other tissues. Considerable decrease in total lipid in the tissues of Tilapia mossambica exposed to methyl parathion was reported [38]. The decrease in liver lipid content of Barbus chonchonius exposed to Aldiocarb for 15 and 30 days were reported [30]. Decrease in lipid content of muscle and liver of Mystus vittatus exposed to parrysulfan and sicocil was reported [39]. Decreasing total lipid under the stress of pesticide reflects on their immediate utilization to meet the energy demands [14]. Due to the decrease in total lipid content under the stress of methyl parathion, the free fatty acids showed an increase in the tissues of fishes [20]. Fluctuation in lipid content between muscle and liver is apparent in Barbus chonchonius exposed to Aldiocarb for 15 and 30 days [30]. Reports were submitted on the changes in lipid and cholesterol in the fish C. punctatus under endosulfan exposure [29]. The inhibition of these enzymes was, however, far greater when the fish was exposed to methyl parathion. Increased activity of alkaline and acid phosphatases, nonspecific esterases and lipase in the cells of the gills, liver, kidney and intestine of C. faciata and Notopterus notopterus were recorded [11]. The liver of H. fossilis exposed to a sublethal concentration (0.03 ppm) of methyl parathion for 15 days showed an inhibition of the activities of both acid phosphatase and glucose 6-phosphatase [35]. Histopathological studies conducted with animals exposed to acute levels of pesticides revealed that, while DDVP degenerated gill epithelium, damaged intestinal villi, induced swelling, vacuolation and degeneration of parenchymal cells of liver and many renal tubules of kidney in L.rohita, it induced severe effects in H.fossilis causing eruption of gill epithelium from secondary gill filaments, rupture of the mucous membranes of stomach and intestine and degeneration of epithelial and subepithelial connective tissues and swelling and vacuolation of the parenchymal cells of the liver [18]. With this in view an attempt has been made in the present study to investigate the toxicity impact of pesticide monocrotophos on Mystus gulio since very little information is available in this important edible marine fish.

2. MATERIALS AND METHODS

Mystus gulio were collected from Agniar estuary, southeast coast of Tamilnadu. The fish were exposed to different concentrations of monocrotophos and mortality was observed for 96 hr. A static renewable bioassay method was adopted for the determination of 96hr median lethal concentration [40]. Probit analysis was followed for the calculations of 96h LC₅₀ [22]. The fish were removed from each experimental group and tissues were isolated for the biochemical parameters such as total

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications carbohydrate, protein and lipids were estimated by methods of earlier researchers [8, 23, 34]. For histological studies, the liver was removed from the fish and transferred to fresh Bouin's fluid and left in it for 24 hours and processed by adopting the usual micro technique procedure [12].

3. RESULTS AND DISCUSSION

The carbohydrate in the liver of pesticide treated medium of the fish had decreased values when compared with control fish. The normal carbohydrate was 25.41-26.11 mg/g. When the fish was introduced at higher concentration of the pesticide monocrotophos the carbohydrate was in decreased order. At the sublethal concentration 10% and 30% sublethal concentration of the carbohydrate was 68.56 and 87.58% in the liver of fish exposed. The rest of treated fish carbohydrate in 10% and 30% were tabulated (Table 1). In the present study the percentage of carbohydrate content decreased in the liver tissues of fishes exposed to the sublethal concentrations of monocrotophos. The copper exposed Thalamita crenata showed a decrease in the carbohydrate content in the gill tissues [48]. Carbohydrates may be converted to glycogen or shunted in the metabolic pathway to supply the carbon chain for amino acids, or converted into fat, of these various processes formation and breakdown of glycogen appears to occupy a central position [31]. This energy yielding carbohydrate metabolism is, however, disturbed when the fish is exposed to pollutants [9, 26]. The normal total protein content was the highest in liver 26.15-26.35 mg/g. When the fish were introduced at higher concentrations of the pesticide monocrotophos the total protein content was in decreased order. At 10% sublethal concentration of the total protein content was 21.15 mg/g, 17.15 mg/g and 14.65 mg/g respectively for 10, 20 and 30 days of the exposure periods. At 30% sublethal concentration the total protein contents were 18.15 mg/g, 14.32 mg/g and 10.11 mg/g respectively for the exposure periods of 10, 20 and 30 days (Table 2). Proteins are mainly involved in the architecture of the cell. During chronic period of stress, they are also a source of energy [46]. Other workers have also reported decline in protein constituent in different fish tissues exposed to sublethal concentrations of insecticides [23, 37]. Depletion in the protein content in stomach and intestine of Clarias batrachus exposed to pesticides endosulfan, Malathion and agrofen [17]. Earlier record shows decrease in protein content of Lepidocephalichthys thermalis exposed to sublethal concentrations of fenvalerate [16]. A significant decrease has been reported in the protein content of the liver and kidney in O. mossambicus, when exposed to 20% active ingredient EC Fenvalerate [2]. The pesticides exposed Oreochromis mossambicus showed a decrease in the protein content in the muscle [10]. A similar decrease in the total and soluble protein content has been observed with fenvalerate in fish [23, 43]. The concentration of lipid in the normal fish was found to be maximum in liver (14.52-14.65 mg/g) In the fish exposed to sublethal concentrations of monocrotophos, significant decrease were observed in the content of lipid in all the tissue on pesticide monocrotophos exposure (Table. 3). In fish exposed to the highest sublethal concentration of monocrotophos (10%) for 30 days, the concentration of lipid decreased to 58.15% in the liver,

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications while the fish exposed to 30% SLC for 30 days showed decreased lipid content in the liver 71.16%. In the 10% sublethal concentration of the pesticide the decrease in lipid content was to a lesser extent (Table.3). At the sublethal concentrations the lipid levels showed decreasing trend during the period of exposure in liver. Earlier researchers also suggested that the decrease in lipid content in C. carpio may be either due to the uptake of lipid by the tissue for utilization at cellular levels or due to increased lipolysis or mitochondrial injury, which affect the fatty acid oxidation mechanism as suggested [3, 33, 49]. Earlier research reported significant decrease in lipid of L. rohita when exposed to heavy metal cadmium [13]. At the liver of the monocrotophos treated fish at the 10% sublethal concentration, the hepatic lobule displayed some loss of its hexagonal conformation and of the cells delimitation, the synusoidal spaces being not very well defined, the cells being more agglomerated. Alternately, at 30% sublethal concentration there was marked swelling of the hepatocytes in places with areas of diffuse necrosis (Figs. 1 - 7). Liver, the first organ to face any foreign molecule through portal circulation is subjected to more damage [15]. These observations are in good agreement with the earlier reports [1, 42, 44]. The same changes in liver of Catla catla, hepatic cords appeared in decreased size, nucleus became pyknotic [43]. Moderate cytoplasmic degeneration in hepatocytes, formation of vacuoles, rupture in blood vessels and appearance of blood vessels among hepatocytes, pyknotic nuclei in the liver of Mystus gulio exposed to fenvalerate [32].

 Table 1: Total carbohydrate in Liver of Mystus gulio exposed to sublethal concentrations of Monochrotophos (mg/g)

Exposure Period	10 Days	20 Days	30 Days
Control	25.41±0.34	26.11±0.35	25.92±0.36
10% SLC	17.25±0.28	11.62±0.31	8.15±0.28
%Variation	-32.11	-55.50	-68.56
30% SLC	10.65±0.31	6.14±0.28	3.22±0.34
% Variation	-58.09	-76.48	-87.58

Values are mean \pm SD of six observations. – or + indicate percent decrease or increase over control

Table 2: Total protein in Liv	er of Mystus gulid	exposed to sublethal
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concentrations of Monoemotophos (mg/g)					
Exposure Period	10 Days	20 Days	30 Days		
Control	26.35±0.41	26.15±0.28	26.21±0.37		
10% SLC	21.15±0.39	17.15±0.36	14.65±0.31		
%Variation	-19.73	-34.42	-44.11		
30% SLC	18.15±0.35	14.32±0.33	10.11±0.29		
% Variation	-31.12	-45.24	-61.43		

concentrations of Monochrotophos (mg/g)

Values are mean \pm SD of six observations. – or + indicate percent decrease or increase over control

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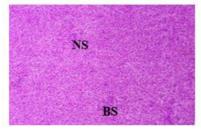
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Exposure Period	10 Days	20 Days	30 Days	
Control	14.52±0.34	14.65±0.23	14.60±0.22	
10% SLC	11.28±0.26	7.68±0.26	6.11±0.27	
%Variation	-22.31	-47.58	-58.15	
30% SLC	8.16±0.24	6.19±0.25	4.21±0.22	
% Variation	-43.80	-57.25	-71.16	

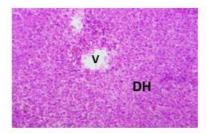
concentrations of Monochrotophos (mg/g)

Values are mean \pm SD of six observations. – or + indicate percent decrease or increase over control

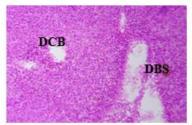
Histopathological lesions in the liver of *M. gulio* exposed to 10% sublethal concentration of monochrotophos at different durations













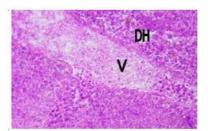
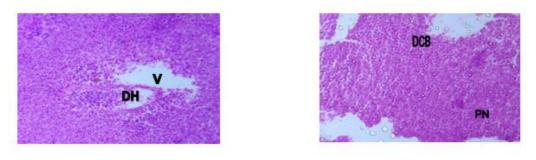


Fig 4.

Sathick et alRJLBPCS 2019www.rjlbpcs.comLife Science Informatics PublicationsHistopathological lesions in the liver of M. gulioexposed to 30% sublethal concentration of
monochrotophos at different durations







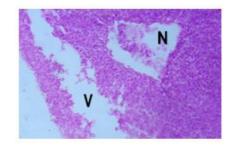




Fig 1. Control: NH - Normal hepatocytes; BS - Blood sinus

Fig 2. 10 days treated: DCB-Disintegration of cell boundaries; DBS-Dilation of blood sinusoids

Fig 3. 20 days treated: V-Vacuolization; DH-Degeneration of hepatocytes

Fig 4. 30 days treated: V-Vacuolization; DH-Degeneraiton of hepatocytes

Fig 5. 10 days treated: V-Vacuolization; DH-Degeneration of hepatocytes

Fig 6. 20 days treated: DCB-Disinterpretation of cell boundaries; PN-Pyknotic nuclei

Fig 7. 30 days treated: N-Necrois; V-Vacuolization

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CONFLICT OF INTEREST

Authors have no conflict of interest.

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