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Blood Glucose and Glycogen Levels as Indicators of Stress in the Freshwater Fish, *Cirrihinus mrigal* under Cyphenothrin Intoxication

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Abstract

Pesticides are non-biodegradable substances used to control bugs, weeds, snails, and plant diseases. They are widely utilised in forestry, farming, and veterinary practices and are of incredible general well-being significance. Pesticides can be ordered by their utilisation into three significant sorts (in particular insect sprays, herbicides, and fungicides). Water defilement by these pesticides is known to prompt harmful effects on the creation, proliferation, and survivability of aquatic life forms, like green growth, amphibian plants, and fish (shellfish and finfish species). The information presented in this study is used to assess the negative effects of exposing fish species to pesticide concentrations. Pyrethroids are regularly utilised around the home and in agricultural production to control bugs. Human contact with at least one pyrethroid bug spray is reasonable. These synthetic substances cause serious harm to aquatic biological systems, particularly fish. The principle points and objective of the present investigation are to assess the effect of cyphenothrin on carbohydrate metabolism in the fish. *Cirrihinus mrigal* is a monetarily significant Indian major carp. A freshwater fish, *Cirrihinus mrigal,* was exposed to bug spray, i.e., cyphenothrin a manufactured pyrethroid. The LC50 for cyphenothrin at 96 hrs was 6 µg/L by probit analysis. One-fifth of LC50 (120 µg/L), was chosen as the sublethal focus. The fish were subjected to sub-lethal concentrations for 10, 20, 30 and 40 days, and the changes in starch digestion, for example, absolute glycogen, as well as the activities of catalysts Lactate Dehydrogenase (LDH) and Succinate Dehydrogenase (SDH) in critical organs, such as the gill, kidney, and liver, were studied.

Keywords: Carbohydrate Metabolism, Cyphenothrin, LDH, SDH

1. Introduction

The contemporary era of the 'green transition', which is characterised by a rapid rise in the human population worldwide, illustrates how dependent people are on readily available everyday resources. Innovative developments have been motivated by the current circumstances to adjust to the requirements of social orders. Thus, this is ensured by the environment's gradually expanding breakdown of various synthetic substances that have been introduced there. This causes contamination, especially in aquatic bodies used as disposal sites in many parts of the world[1-3]. The primary general hazardous component, contamination, is caused by human development and rapid industrialization[4]. Water

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pollution is a risky problem in general, and seepage of agricultural, industrial, and synthetic substances into the waterways has had some unfavourable effects on aquatic life[5]. Additionally, these poisons may simply accumulate in fish tissue and contaminate the food chain, affecting human intake[6].

Pyrethroids are subsidiaries of regular pyrethrins delivered from the flowers of the pyrethrum plant (*Chrysanthemum cinerariaefolium* and *C. coccineum*)[1]. They include esters of chrysanthemum acidic (ethyl2,2-dimethyl-3-(1-isobutenyl) cyclopropane-1-carboxylate) and halogenated subordinates of their acids and alcohols[7,8]. Despite the fact that pyrethroids and pyrethrins are synthetically and toxicologically comparable, the last option goes through quick decay when exposed to heat, light, and dampness[9]. Consequently, pyrethrins were supplanted with manufactured subsidiaries (pyrethroids), initially thought to be good for people[10]. These pesticides were acquainted due to their lesser level of perseverance when contrasted with organochlorine, organophosphate and carbamate pesticides which are related to long-term biological dangers. Pyrethroids typically crumble within the sight of daylight and the surrounding environment in a couple of days. Likewise, they don't fundamentally influence ground-water quality making them favoured substitutes to conventional more constant pesticides[11].

Pyrethroids' component of activity depends on communication with sodium channels and the enlistment of delayed depolarization in neurons in the sensory system[12]. WHO World Health Organization 1999 believed that pyrethroids are neuro-harms following up on the axons in the fringe and focal sensory system by associating with sodium directs in warm-blooded animals and additionally insects. When compared to warm-blooded animals, they have an extremely high specific poisonousness for insects and have been shown to be multiple times more poisonous to insects than to higher species[13]. This is because insects have more delicate sodium channels, a more modest construction, and lower internal heat levels, as well as lower skin retention and more proficient hepatic digestion in vertebrates[14]. Be that as it may, they have been considered poisonous to aquatic species, e.g., shellfish[15], and finfish[16,17] influencing ions in both neuronal and mitochondrial layers. They have additionally been accounted for as being poisonous to higher vertebrates[18,19].

Cyphenothrin is the ISO common name for a mixture of four diastereoisomers known as (±)--cyano-3-phenoxybenzyl (±)-cis-trans-chrysanthemate. The name d,d,trans-cyphenothrin* alludes to an enantio-advanced combination, contained basically of the single stereoisomer (S)-alpha-cyano-3-phenoxybenzyl(1R,3R)-2,2-dimethyl-3-(2-methylprop-1-enyl) cyclopropanecarboxylate, with just little extents of the other stereoisomers, as characterized by the WHO particular. Fish are bioindicator species that assume significant roles in observing water contamination since they respond with an extraordinary aversion to changes in aquatic species[20]. Pesticide exposure to fish causes pressure, which is a state of restored homeostasis and a perplexing set-up of maladaptive reactions (Salako *et al*., 2020) with the potential to cause interruptions and mortality.

In this investigation, the freshwater fish *Cirrhinus mrigala* is used for the estimation of the intense toxicity of cyphenothrin. It is a bottom-dwelling fish that is widely consumed in the Indian subcontinent. LC_{50} focus is an imperative marker of the level of obstruction of a life form and its subordinates[83]. *C. mrigal* has been widely used as a test model by numerous analysts for toxicological investigations of pesticides. Comparative investigations have been done the Pyrethroid exposure, for example, Cypermethrin[21-24]. Deltamethrin[25], Chlorpyrifos-methyl[26], Dichlorvos[27], Permethrin[28] and Lambda-cyhalothrin[29,30] shown that guppy fish is important for researching the impacts of cancer-causing as well as poisonous waterborne diseases on people. Similarly, constant cancer-causing nature of the bioassays with guppy and possibly with other little fish species is plausible and deductively legitimate.

Prolonged use of poisonous pesticides in various environments, which because of their high viability and simple use have disposed of a few natural techniques for insect control, was brought about by ecological contamination. Pesticides have been applied to protect against diseases in plants, animals and people. In any case, the acquaintance of pesticides with the regular habitat has likewise a few adverse consequences, including insects, fish, birds, warm-blooded species, and different occupants of amphibian and earthbound biocenoses. Along these lines in the current investigation, an effort has been made to investigate the impacts of cyphenothrin on the carbohydrate metabolism of the freshwater fish *Cirrihinus mrigal*, a consumable fish.

2. Materials and Methods

2.1 Experimental Animal

Healthy freshwater fish, *Cirrhinus mrigal* weighing 100-150 g

were obtained from local ponds in, Dharwad District, India and were maintained in laboratory tanks with continuous aeration. Fish were maintained in large cement tanks (6×3 feet) which were duly aerated. The water in the tanks was treated with a 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 acclimatise for a period of 14 days at 24°C and 12-14 hours of photoperiod. The water in the tanks was renewed daily and the physicochemical parameters of the water were examined according to the guidelines of APHA.

2.2 Experimental Pesticide

Cyphenothrin (Type II pyrethroid) supplied by Sumito Chemical India Pvt. Ltd., Gujarat, India, 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. The stock solution was prepared by mixing the calculated volume of the commercial solution with distilled water. Test concentrations for the acute toxicity test (i.e., 6 µg/l) and sub-lethal behavioural toxicity test (120 mg/l)[31] were prepared by serial dilution of the stock solution using a variable micropipette.+

2.3 Estimation of Carbohydrate Metabolism

The colorimetric method was used to determine glucose levels in the blood, liver, and muscles of the sample[32]. The Anthrone reagent method was used to calculate the glycogen content of fish liver and muscle[33]. Phosphorylase activity in fish muscle was measured[34]. Glucose-6-phosphatase activity in the liver of fish was estimated using the method of Yeung, *et al.* (1968). Pyruvate in the organs of fish was estimated[35,36]. Lactate in the organs was estimated using the method of Barker and Summcrson[20] as modified by Huckabee[2]. Succinate dehydrogenase activity in the organs was estimated using the colorimetric method of Nachlas, *et al.*, 1960.

2.4 Evaluation of Results

All the data presented in the study was performed using the Origin Pro 2019b statistical package. The data were subject to one-way analyses of variance (ANOVA) with Tukey's post-hoc test and the data are presented as mean ± SD values with a significance level set at $p < 0.05$.

2.5 Ethical Statement

All the experiments performed in the present study abide by the guidelines of the Institutional Animal Ethics Committee (IAEC). The experimental animals used in the study were handled with care according to the guidelines provided by the Committee for the Purpose of Control and Supervision of Experiments on Animals, Registration number 639/GO/Re/S/02/CPCSEA, New Delhi, India. Valid for five years, from August 31, 2018, to August 30, 2023.

3. Results

3.1 Blood Glucose

According to the information in Table 1, blood glucose levels increased during all exposure times at sub-lethal concentrations of cyphenothrin in comparison to controls. Day 40 shows the lowest increase above the control of 80.71, and Day 20 saw the highest increase of 88.67.

The average blood sugar level among those with sub-lethal concentrations increased on average until day 20, reaching a maximum rise of 88.67 over control. When compared to day 10, blood glucose levels decreased on days 10 and 30. On day 40 of a sub-lethal dose, the glucose levels were at their lowest. When cyphenothrin was present at sublethal concentrations, a mean value of 80.71 was recorded (Table 1).

3.2 Glycogen

Under sub-lethal concentrations of cyphenothrin at all times of exposure, the glycogen content in all tissues (muscles and liver) showed a constant decline and increase. The fish's liver tissue showed the lowest level of glycogen (19.770 mg/g wet weight) on day 20 of exposure, and the greatest level (26.327 mg/g wet weight) on day 40 (Table 1).

According to the information in Table 1, fish exposed to sub-lethal concentrations of cyphenothrin had less glycogen in their muscles. Day 30 saw a continual decline. The average maximum change in muscle tissue was found to be 4.017.

The mean value of liver glycogen in sublethal concentration decreased up to day 20, and the largest rise in comparison to control was 26.32 on day 40. When compared to day 20, hepatic glycogen increased on days 10 and 30. (Table 1). The mean value of muscle glycogen in sublethal conditions decreased up to day 40, with the largest rise of 4.01 in comparison to control occurring on day 10. When

Table 1. Levels of blood glucose (mg/100ml) and glycogen (mg/g wet wt.) in the liver and muscle of freshwater fish *Cirrihinus mrigala* on exposure to sub-lethal concentration of cyphenothrin

Estimation	Control	Exposure in Days			
		10	20	30	40
Blood Glucose	72.8417^{E}	84.8125^{B}	88.6737^{A}	82.5217^{C}	80.7137^{D}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	--------	-16.434	-21.7348	-13.2891	-10.807
Liver Glycogen	28.2606^{A}	21.7137^{D}	19.7709^{E}	24.7951^{C}	26.3276^{B}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	--------	-23.1662	-30.0408	-12.2627	-6.83991
Muscle Glycogen	4.2017^{A}	4.0179^{B}	3.9794^{C}	3.7217^{E}	3.9122^{D}
SD±	0.000374	0.000374	0.000374	0.000374	0.000374
% Change	------	-4.3744	-5.2907	-11.4239	-6.8900

All data is represented as the mean ± SD (n = 6). Values (A-E) in the row with the same superscript are not significantly different at $p < 0.05$

compared to control Table 1, day 20 showed an increase of around 3.97, day 30 showed a decline of approximately 3.72, and again increases at day 40 to approximately 3.91.

3.3 Glycogen Phosphorylase and Glucose-6-Phosphatase

The fish *Cirrihinus mrigala's* liver and muscle glucose-6-

Table 2. Activities of glycogen phosphorous and glucose-6-phosphate (µM Pi/mg protein/h) of freshwater fish *Cirrihinus mrigala* exposed to sub-lethal concentration of cyphenohrin

Estimation	Control	Exposure in Days			
		10	20	30	40
Liver glycogen Phosphosrylase	6.4665^{E}	6.9022^{D}	7.3119^{A}	7.1114^{B}	6.9922^{C}
SD±	0.000374	0.000374	0.000374	0.000374	0.00041
% Change	-------	-6.7378	-13.0735	-9.9729	-8.1306
Muscle Glycogen phosphorylase	4.9877^{E}	5.6325^{B}	5.7723^{A}	5.3092^{D}	5.1132^{C}
SD±	0.00028	0.00033	0.000374	0.000432	0.000374
% Change	---------	-12.9285	-15.7307	-6.4465	-2.5202
Liver Glucose-6-Phosphotase	4.1717^{E}	4.9136^{D}	5.8776^{A}	5.4607^{B}	4.9915^{C}
SD±	0.000374	0.000374	0.000374	0.000374	0.00033
% Change	---------	-17.7841	-40.8922	-30.8987	-19.6523

All data is represented as the mean ± SD (n = 6). Values(A-E) in the row with the same superscript are not significantly different at $p < 0.05$

phosphatase activities in response to sublethal cyphenothrin concentrations are shown in Table 2. Increased activity of fish liver and muscle glucose-6-phosphatase and glycogen phosphorylase was seen during some exposure times.

Up until day 40, a rising trend in the activity of both glucose-6-phosphatase and glycogen phosphorylase was seen at sublethal concentrations. Day 10 saw a regression, however. The liver's Glycogen phosphorylase activity peaked on day 20 (7.311), reaching its lowest level on day 40 (6.902). Day 40 shows a regression in muscle glycogen phosphorylase. On day 20, activity was at its peak (5.772), and on day 40, it was at its lowest (5.113). Glucose-6-Phosphatase activity in the liver marked a mean value increase of 5.877 with a maximum noticed on day 40 and a minimum increase (4.913) observed on day l0.

3.4 GSuccinate Dehydrogenase (SDH) Activity

The fish's gill, kidney, and liver SDH activity was considerably increased at all exposure levels investigated in the sub-lethal concentration of cyphenothrin, according to the data shown in Table 3. Additionally, a considerable increase in SDH activity was seen at sub-lethal concentrations (Table 3).

It was less at day 10 (gill -0.207, kidney 1.161, and liver 0.2019), at day 20, (gill 0.226, kidney 0.193, and liver 0.211), at day 30, (gills 0.247, kidney 0.2019, and liver 0.2447), and at day 40, (gills 0.263, kidney 0.223, and liver 0.291), with a significant difference between the four exposure periods of the sub-lethal concentration. Fish organs showing a significant change (P<0.05) between days 10 and 40 showed a drop in sub-lethal concentration that was less pronounced at day 10 (gill 0.207, kidney 0.161, and liver 0.201), and more pronounced at day 40 (gill 0.263, kidney 0.223, and liver 0.291). The variation in the organs between 10 and 40 days, showed significant variation (P < 0.05) in the fish organs.

3.5 Lactate Dehydrogenase (LDH) Activity

According to the information in Table 4, the fish's gill, kidney, and liver activity considerably increased at 10, 20, 30, and 40 days after exposure to a sublethal dosage of cyphenothrin. LDH activity increased in the fish's organs at sub-lethal concentrations across all exposure times (Table 4). The order of 10 < 20 < 30 < 40 days was among the exposure times for the fish organs to the sublethal dose. The increased LDH activity at 10 days increased further at 20 and 30 days in the sub-lethal levels. However, it regressed at 40 days, which caused the enzyme activity to decline at 20 days. Consequently, the rise was in the 10 > 20 > 30 > 40 day period. The rise in LDH activity varied in intensity among

Table 3. SDH activity (μ M formozan/mg protein/h) of freshwater fish *Cirrihinus mrigala* exposed to sub-lethal concentration of cyphenothrin

Estimation	Control	Exposure in Days			
		10	20	30	40
Gills	0.1702^{E}	0.2077^{D}	0.2267^{C}	0.2478^{B}	0.2637^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.00041
% Change	-------	-6.7378	-13.0735	-9.9729	-8.1306
Kidney	0.1472^{E}	0.1612^{D}	0.1935^{C}	0.2019^{B}	0.2235^{A}
SD±	0.00028	0.00033	0.000374	0.000432	0.000374
% Change	---------	-12.9285	-15.7307	-6.4465	-2.5202
Liver	0.1955^{E}	0.2019^{D}	0.2117^{C}	0.2447^{B}	0.2916^{A}
SD±	0.000374	0.000374	0.000374	0.000374	0.00033
% Change	---------	-17.7841	-40.8922	-30.8987	-19.6523

All data is represented as the mean ± SD (n = 6). Values(A-E) in the row with the same superscript are not significantly different at p < 0.05.

Table 4. LDH activity (μ M formozan/mg protein/h) of freshwater fish *Cirrihinus mrigala* exposed to sub-lethal concentration of cyphenothrin

Estimation	Control	Exposure in Days			
		10	20	30	40
Gills	0.78067^{A}	0.082^{E}	0.0981^{D}	0.010716^{C}	0.11121^{B}
SD±	0.002833	0.000374	0.000374	0.004021	0.00074
% Change	--------	-5.03798	-25.6613	-37.2757	-42.4631
Kidney	0.054^{E}	0.0598^{D}	0.0692^{C}	0.0818^{B}	0.09766^{A}
SD±	0.003742	0.000374	0.000374	0.000374	0.000374
% Change	-------	-10.7407	-28.1481	-51.4815	-80.8519
Liver	0.0315^{E}	0.03848^{B}	0.03856^{A}	0.0476^{D}	0.0502^{C}
SD±	0.000374	0.000349	0.00071	0.000374	0.000374
% Change	------	-22.1683	-22.4349	51.1111	-5.3651

All data is represented as the mean ± SD (n = 6). Values(A-E) in the row with the same superscript are not significantly different at $p < 0.05$.

the investigated organs, as seen from the mean values. As a result, the order of fish organs at sublethal concentration was liver, kidney, then gill. These variations weren't substantial, and they weren't consistent across the exposure times.

3.6 Lactate

From the information in Table 6, it is clear that all exposure times studied at the sub-lethal concentration of cyphenothrin resulted in a significant ($P < 0.05$) increase in the lactate level in the gill, kidney, and liver. However, the level of increase was lower than the increase in the sub-lethal concentration (Table 6).

The increase in lactate level in the fish organs exposed to the sub-lethal dose rose over the time of exposure in the order of 10 < 20 < 30 days, during the exposure periods. The increase in lactate level at day 10 in sub-lethal concentration progressed up to 40 days in the order of 10 < 20 < 30 days. At 40 days, there was a decline in the kidney and liver.

3.7 Pyruvate

From the information in Table 5, it is clear that during all exposure periods at sub-lethal concentrations, the pyruvate level in the fish's gill, kidney, and liver increased significantly. Among the exposure times, the drop in pyruvate level rose over the time of exposure at 10, 20, 30, and 40 days, whereas the pyruvate level increased in the organs of the fish exposed to the sub-lethal dosage.

4. Discussion

Of all the energy reserves, carbohydrates are typically used quickly and are therefore consumed quickly. Glucose is the main product of carbohydrate absorption, and research has revealed that it plays a role in a fish's transitional digestion[37]. Changes in carbohydrate digestion are made to meet the fluctuating energy requirements that can be expected in species under stress. The results of the current concentrate unmistakably show that exposure to cyphenothrin disturbs the digestion of carbohydrates in fish, *Cirrhinus mrigala*.

With one animal variety followed by the next, fishes' blood glucose levels change in accordance with their levels of activity and, consequently, metabolism[38]. Blood glucose levels are low in passive fish like *Laphius discatorlus* with slow digestion rates, while blood glucose levels are raised in more active fish with rapid digestive rates[39]. This has led people to accept that the blood glucose level reflects how well a fish digest carbohydrates. The increase in blood glucose levels in *Cirrhinus mrigala* fish subjected to sublethal levels of cyphenothrin showed that carbohydrate digestion advanced under adverse conditions. This hyperglycemic syndrome may cause glycogenolysis, glyconeogenesis, or both to progress. The forward motion in the way that the increased blood glucose is accompanied by a decrease in the glycogen level in *Cirrhinus mrigala* is indicative of glycogenolysis.

In *Channa punctatus* under monocrotophos poisoning, hyperglycemia and a decline in the glycogen content of the

liver and muscle tissue were observed[40]. Under sublethal diazinon concentrations, similar species also showed comparative increases in blood glucose levels. It is understood that deltamethrin increases glucose fixation in bumble bee haemolymph[41]. In albino rats, it is minimum to steer the rise in blood sugar toward the termination of the exposure time mechanism with fenvalerate and decamethrin separately[42]. Reduced usage of glucose for energy-producing purposes may also cause its level in the blood to rise. This is quite clear in the fish exposed to sublethal levels of cyphenothrin in the current experiment (Table 1). Increased blood glucose levels may also be related to the transfer of glucose from tissues to the circulation. By activating the phosphorylase[43-45], various researchers using fish as models[46] for pesticide damage observed glycogenolysis and hyperglycemia. In light of this, alterations in pancreatic secretions involved in carbohydrate metabolism[47] may be a general cause of the elevation in blood glucose levels in fish exposed to cyphenothrin.

The hyperglycemic state observed in fish exposed to cyphenothrin may also be brought on by the sensation of various chemical compounds emitted from neuroendocrine physiology. Investigations into the hyperglycemic state in species that have been excitedly exposed to a variety of toxins are ongoing. The hyperglycemic substance activates phosphorylase catalysts[48,49] to accelerate glycogen decomposition. Additionally, the anaerobic weight of the fish caused by the pesticide[50] can also be attributed to hyperglycemia. In the current study, fish exposed to sublethal amounts of cyphenothrin showed decreased glycogen in a variety of organs. *Cirrhinus mrigala* was the name of the fish. A false decrease in the amount of glycogen found in fish tissues after exposure to methyl parathion in *Tilapia mossambica*[51]. *Labeo rohita*, the fish under cypermethrin stress, also showed a loss in glycogen with a corresponding rise in blood glucose levels[52]. The test fish's tissues no longer contain as much glycogen, which suggests that anaerobic glycolysis might be used to generate enough energy to justify its use. It has been discovered that "during pesticide stress[53] the combination and utilisation of glycogen are altered". The prevalence of hypoxia or anoxic conditions, which normally increase glycogen usage, may be the cause of glycogen exhaustion[54]. It was discovered that muscles exposed to methyl parathion[55] experienced anoxic conditions.

The primary cause of *Cirrhinus mrigala's* exhaustion of its carbohydrate energy reserves, when exposed to high cyphenothrin concentrations, is the rise in glucose levels and consumption of the fish's glycogen stores caused by glycogenolysis, either through hormonal irregularity or potentially through other affecting variables. A further indication of the significant metabolic stress on metabolic homeostasis is an increase in the consumption of the energy reserves provided by carbohydrates during the course of the exposure, from day 10 to day 40[56,57]. This is plausible in tissues where exposure may have severely impaired neuro-endocrine coordination centres. As a result, the liver, muscles, and fish gills would have continuously broken-down glycogen reserves due to improper activation of the phosphorylase enzyme.

The fish initially displayed significant glycogenolysis at sublethal concentrations, proving that cyphenothrin might affect energy reserves with the action of the elements even at subacute amounts. Furthermore, the fish's declining glucose level decreased glycogen content, and increased phosphorylase and glucose-6-phosphatase activities indicate that glucose will be used for energy requirements proportionate to glycogen breakdown. According to all accounts, glucose utilisation throughout this period of exposure is higher because more energy is needed to deal with the ongoing harmful pressure. The progression of obstruction in the fish under sublethal detrimental pressure is then demonstrated by the steady relapse in glucose utilisation and the breakdown of glycogen[58,59].

The increase in the activity of the enzyme's glucose-6-phosphatase and glycogen phosphorylase in the fish *Cirrhinus mrigala* may have been caused by the pesticide's rapid influence on the pancreas, which inhibits the arrival of insulin. It was demonstrated that cypermethrin significantly increased the activity of phosphorylase action[60]. It is widely known that fish release large amounts of the stress hormones glucocorticoids[61] and catecholamines[62], which lead to rapid hyperglycemia[63-65]. By activating the phosphorylase molecule, various results in fish[66] models during pesticide toxicosis reveal glycogenolysis and hyperglycemia. As height was also observed in a species model[67], exhibited hyperglycemia coupled with a decrease in the levels of glycogen in the liver and muscle of the fish, *Sarotherodon mossambica* exposed to sumithion and postulated that haepatic glycogen is the primary source of blood glucose. Methyl parathion caused the liver and muscle glycogen to be depleted and glucose levels to rise, which stimulated the release of catecholamines in the Indian feline fish *H. fossilis*[68]. Both hormonal and neuromuscular alterations could likely demonstrate the increased glycogenolytic activity and gluconeogenesis[69].

The slowing down of SDH activity in the gills, kidneys, and liver of fish exposed to sub-lethal concentrations of cyphenothrin suggests hiding oxidative phosphorylation of

the species[70] in the current experiment linked to the slowing down of oxygen usage. Even if the carbohydrates were activated, the fish's inability to use the new energy was caused by the inhibition of oxidative metabolism. As a result, once the fish experience severe cyphenothrin stress, their glucose consumption declines. The reduced SDH mobility in rodent tissues that was caused by parathion[71] was revealed in detail. This might be attributed to decreased pyruvate formation and oxygen take-up during methyl parathion stress. Several studies linked the progressions in the respectability of mitochondria as a result of methyl parathion exposure to the lowered activities of citric acid cycle catalysts[72]. Curiously, the increase in LDH movement in the fish organs exposed to sublethal levels of cyphenothrin suggests that the fish under cyphenothrin stress may have relied less on anaerobic glycolysis to meet their energy needs. Similar perceptions under endosulfan poisoning in *Tilapia mossambica* have been demonstrated[73].

The extent of the increase in LDH activity caused by tissue damage and organ rot in carp has been demonstrated[74]. In mice treated with taurine and penicillamine and mice exposed to mercury, comparative effects have been demonstrated[75]. The mobility of LDH was also found to be elevated in professionals exposed to DDT for work purposes[76] and in rodent livers after dieldrin exposure[77]. The LDH activity was shown to be enhanced by the sub-lethal grouping of cadmium[78]. The accumulation of pyruvate and lactate in fish organs may be caused by a decrease in SDH activity and an increase in LDH activity. However, the difference between the levels of pyruvate and lactate may indicate that pyruvate has been activated. The elevated LDH activity and lactate levels suggest that some of the pyruvates that had accumulated may have been converted to lactate by anaerobic glycolysis for energy requirements. The prevalence of anaerobic glycolysis during pesticide stress is demonstrated by the lower levels of pyruvate and the increasing amounts of lactate. This suggests that Kreb's cycle is used to meet energy requirements, and it also indicates a reduction in extra pyruvate oxidation in the citric acid cycle. A comparative pattern has been demonstrated in mice that received BMC[79] treatment.

The organs of fish exposed to sub-lethal concentrations of cyphenothrin show mobility in resisting the sub-lethal toxic pressure starting at day 10, with an underlying decrease in oxygen usage and SDH activity across the entire species. It is feasible that the compound effect wouldn't be significantly curbed by the lower concentration of cyphenothrin accumulated in fish organs. As more energy is anticipated for pesticide disposal as well as for improved protein options. On delayed exposure to sublethal focus, the oxidative metabolic cycle is boosted in fish, which is further supported by increased glycogenolysis and decreased glucose levels. The purpose of anaerobic glycolysis and the underlying concealment of oxidative digestion may be to meet energy demands. However, as oxidative digestion increased, anaerobic glycolysis gradually reverted and reached the prediction by 15 days. Fish exposed to the sublethal concentration of the substance had this type of change and acclimatisation[80].

5. Conclusions

According to the findings and conclusions, the present study revealed that fish that had been exposed to cyphenothrin had changes in their carbohydrate metabolism and content in the bony fish *Cirrihinus mrigal*, which is a biochemical marker distinct from the toxic effects of the toxin. When something is harmful, it first affects cells or possibly molecules before causing biochemical changes. The changes in fish's carbohydrate composition typically have a negative impact on the nutritional value of aquatic life, diminishing the value of fish, and possibly even posing health risks to individuals who consume such fish regularly.

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