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# Impact of Imidacloprid 17.8% SI on Biochemical Parameters of the Fresh Water Fish *Catla Catla*

**Sumaiya Ismayil, Aneykutty Joseph**

Department of Marine Biology, Microbiology and Biochemistry School of Marine Sciences Cochin University of Science and Technology, Cochin, Kerala, India

**ABSTRACT**

Use of pesticide in integrated farming in paddy field is recognized as a potential threat in aquatic organisms. The effect of sublethal toxicity of imidacloprid 17.8% SL pesticide on some biochemical parameters of a freshwater fish *Catla catla* were studied after 24 hr, 48 hr, 72 hr and 96 hr of exposure. The lethal concentration ($LC_{50}$) of imidacloprid 17.8% SL for 96 hr was 4.11 ppm. The study suggests that exposure to imidacloprid 17.8% SL at low concentration results in significant biochemical alterations. The biochemical response of *C. catla* to the exposure to sub lethal concentrations of imidacloprid 17.8% SL showed a significant decrease ($p<0.001$) of proteins and glycogen in muscle and liver tissues compared to the control group. The observations from the present study showed that, imidacloprid 17.8% SL altered the biochemical composition of the various organs of test fish, due to utilization of biochemical energy to counteract the toxic stress.

***KEYWORDS***: *IMIDACLOPRID, BIOCHEMICAL, CATLA CATLA*

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**INTRODUCTION**:

Even though effects of environmental pollutants on mortality of aquatic animals have been studied by many workers, very little is known about the disturbed physiological and biochemical processes within the organism following exposure to environmental pollutants. The respiratory system of fish seems to be the prime target of many pollutants. When tissues of the animal do not receive sufficient oxygen they must either reduce the overall energy demand or respire anaerobically. Since glycogen is the ready source of energy even in aerobic condition, the depletion of glycogen from the tissue is exposed to be an immediate manifestation of hypoxia. During severe hypoxia, flounder reduces its oxygen consumption and partially compensates by increasing anaerobic energy metabolism based on fermentation of glycogen and glucose with lactic acid as the major anaerobic end product (Jorgensen and Mustafa, 1980). This strategy is also employed by other fishes such as *Cyprinus carpio* (Johnston, 1975), *Carassius auratus* (Thillary, 1982) and *Oncorhynchus mykiss* (Burston and Spehar, 1971). A decrease in the glycogen content with the prevalence of hypoxic condition at the tissue level since anoxia or hypoxia increases carbohydrate consumption thereby creating a sort of stress in the fish even at sub lethal level, result in extra expenditure of energy.

The biochemical and physiological adaptation made by fish in response to changes in environmental oxygen levels can be correlated to the ecology of the species. Species such as *Cyprinus carpio*, which have been shown to live for several months in water of very low oxygen content (Blaska, 1958) are able to survive by reducing their oxygen uptake and changing to anaerobic metabolism (Johnston, 1975) other species such as salmonoids adapted to environments of high oxygen tension are less able to survive hypoxia (Itazawa, 1971). Since different fishes react differently to hypoxic situation, the procedure of using oxygen consumption as a yard-stick to measure the metabolic activities of the body may not produce satisfactory results. Although a number of methods are now available to measure the sub lethal effects of pollutants (Sprague, 1971) most of them are long term and not suitable for routine analysis.

Extensive investigations have revealed that different tissue of fish can sustain varying levels of anaerobic metabolism. In most teleost fermentation of glucose to lactose provided the main source of energy under hypoxic condition (Heath and Pritchard, 1965). Black *et al*. (1961) found that the endurance of fast swimming fish is limited by the anaerobic energy released when stored glycogen is transformed by Embden-Meyerhoff cycle to form lactate within muscle cells. Johnston (1975) postulated that while skeletal muscle of fish, in common with most vertebrate tissues responds to periods of anoxia by an increase in anaerobic glycogenolysis.

In most all these circumstances the major share of energy comes from the carbohydrate or glycogen reserves. Thus carbohydrate forms the central point in energy production because of great mobility in the living systems, together with its capacity to get compartmentalized within cell and tissues. The mobility is provided by glucose and compartmentalization by glycogen and glucose-6-phosphate.

Effects of environmental stress due to chemical pollution on tissue glycogen levels of fish have also been reported by Leay and Brown (1975). These studies involving exposure of fishes to different pollutants have indicated that the pollution stress stimulates glycogenolysis in fish tissues. From a biochemical point of view, life is uniquely characterized by its association with proteins. Tissue protein as energy source for fishes during thermal stress, spawning and muscular exercise have been demonstrated by several investigators. Though considerable information is available dealing with the determination of acute toxic levels of several pollutants and their influence on oxidative metabolism studies on the tissue energy sources are relatively few.

Adrenocortical hormones are known to influence mammalian intermediary metabolism by stimulating protein metabolism (Freeman and Idler, 1973). It now known that these hormones are produced during stress. Hence stress created by the exposure of toxic substances interferes with the intermediary metabolism and affect the protein content of the body. Glycogen and protein present in liver and muscles provide energy to the body. Animal under stress deplete the energy sources at different rates. A study was designed to observe the influence of imidacloprid 17.8% SL on tissue level glycogen and protein in *C. catla*.

## MATERIALS AND METHODS

The test organisms are collected and acclimatized in the laboratory for 15 days. The temperature in the tank during the experiment was maintained at 26-27$^0$c, pH at 7-7.5, Dissolved oxygen 6-6.8 mg/L and salinity at 0 ppt. The saturation of oxygen was maintained by giving aeration in the tank. During acclimatization period the fishes were fed with commercial fish feed.

The fishes of size range 5-6 cm in length were selected for the experiment irrespective of the sex. Feeding of fishes was done one hour before changing the water. Fishes are transferred into each experimental tank. Each tank contained 20 L of water. Based on the $LC_{50}$ values, three sub lethal concentrations of imidacloprid 17.8% SL were selected and added to each experimental tank. One tank kept as control. Replicates were run for each concentration. The medium was renewed every 24 hours. On 10$^{th}$, 20$^{th}$ and 30$^{th}$ days of experiment the fishes caught from the tank and liver and muscle tissues were collected from the dissected fishes. The tissue are weighed and then homogenized with 5% TCA. The homogenate was made into a known volume with 5% TCA and centrifuged at 3000 rpm for 15 minutes. The residue was taken for the estimation of protein and the supernatant was use for the estimation of glycogen. Glycogen in the liver and muscle tissueswas estimated using the method of Montgomery (1957) and protein concentration was determined using the method of Lowery et al. (1951).

## RESULT

The results of the experiment showed significant reduction in the level of glycogen and protein in liver and muscle of imidacloprid 17.8% SL treated fish (Table 1.). During the exposure the changes reported in control were insignificant but in case of fishes treated with sub lethal concentrations of imidacloprid 17.8% SL the observed changes were significant.

After 10 days of exposure to sublethal concentrations of imidacloprid 17.8% SL the changes noted in the amount of muscle protein (Fig 1.) and liver protein (Fig 2.) showed a significant variation from the control fishes. The liver muscle glycogen (Fig 3.) and liver glycogen content (Fig 4.) showed similar results. The decrease in muscular protein and liver protein content of imidacloprid 17.8% SL exposed fish after 20 days was accompanied by a depletion of musculsr glycogen and hepatic glycogen content. The decrease in protein was more for muscle than for liver where as the depletion of glycogen was almost equal in both the tissues.

The ANOVA of the protein concentration in the treated and control fishes, after 30 days of exposure showed significant reduction in all the sub lethal concentrations. As with protein content, the amount of glycogen in muscle and liver tissues of fishes of which were survived in 0.27 pp. 0.41 ppm and 0.82 ppm concentrations imidacloprid 17.8% SL best explained a significant reduction in ANOVA analysis.

**Table1. Variations in biochemical parameters during confidor exposure in *C. catla*.**

| Duration of exposure | parameters | control | 0.27 ppm | 0.41 ppm | 0.82 ppm |
|---|---|---|---|---|---|
| 10 days | Muscle protein | 67.58±0.92 | 61.61±1.09* | 57.89±0.71* | 53.17±0.34* |
| | Liver protein | 55.43±0.71 | 52.06±0.24* | 48.15±0.60* | 43.24±0.46* |
| | Muscle glycogen | 3.72±0.27 | 3.19±0.09* | 2.91±0.03* | 2.77±0.05* |
| | Liver glycogen | 6.78±0.09 | 6.47±0.07* | 5.99±0.08* | 5.78±0.77* |
| 20 days | Muscle protein | 67.31±1.11 | 59.59±1.90* | 56.17±0.36* | 48.99±1.23* |
| | Liver protein | 55.03±1.28 | 50.13±0.41* | 42.12±1.13* | 39.18±0.33* |
| | Muscle glycogen | 3.62±0.14 | 3.02±0.01* | 2.81±0.10* | 2.47±0.07* |
| | Liver glycogen | 6.63±0.14 | 6.25±0.5* | 5.78±0.20* | 5.23±0.16* |
| 30 days | Muscle protein | 67.44±0.67 | 57.27±0.21* | 51.12±1.26* | 42.82±0.62* |
| | Liver protein | 54.89±1.34 | 45.04±0.21* | 36.95±0.94* | 32.38±0.77* |
| | Muscle glycogen | 3.52±0.12 | 2.90±0.10* | 2.18±0.06* | 1.88±0.06* |
| | Liver glycogen | 6.66±0.17 | 6.48±0.65* | 5.55±0.04* | 4.89±0.07* |

Value ± SD *Significance level at ($p<0.001$)

Dose dependent effect of imidacloprid 17.8% SL on tissue energy reserves of *C. catla* was observed after 30 days of exposure. The depletion of protein and glycogen was more for the fishes exposed to the highest concentration (0.82 ppm).

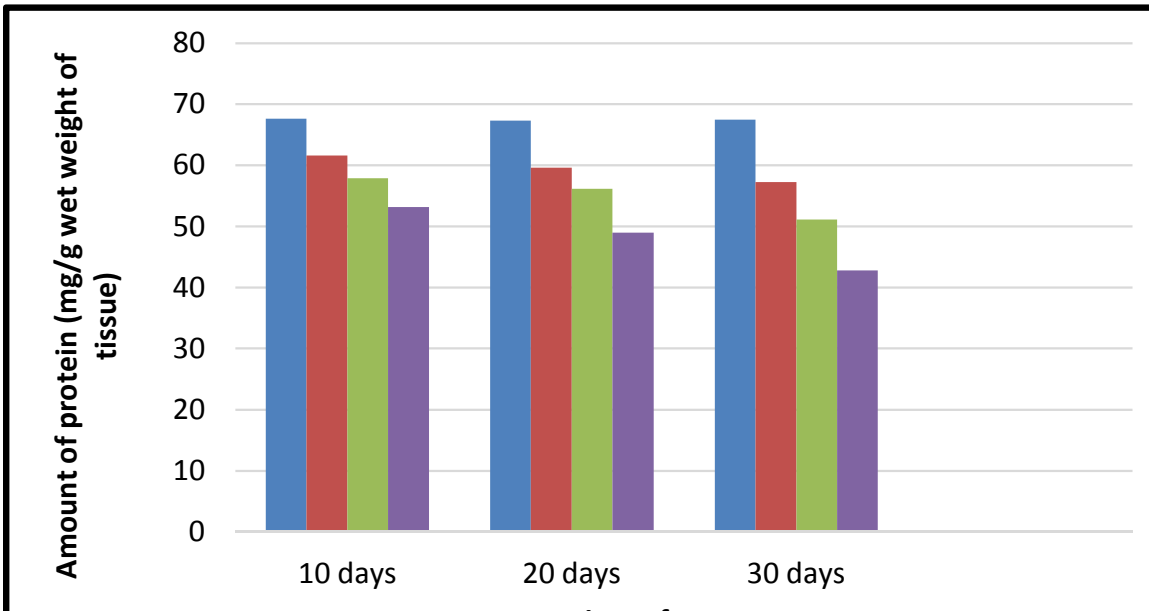


**Fig.1. Protein content of muscle in experimental fishes.**

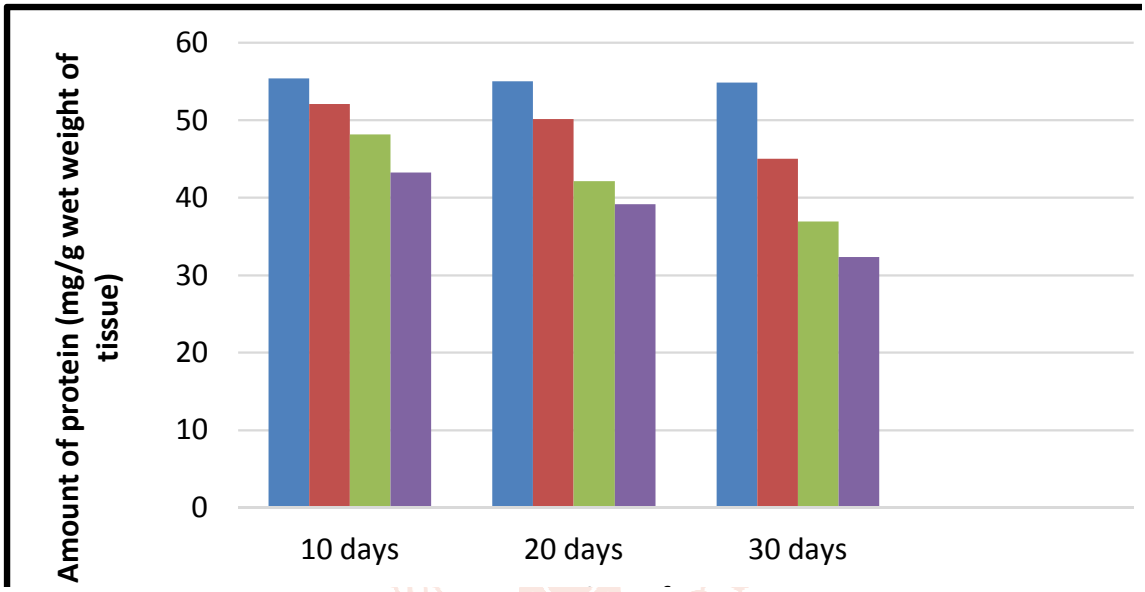


**Fig.2. Protein content of liver in experimental fishes.**

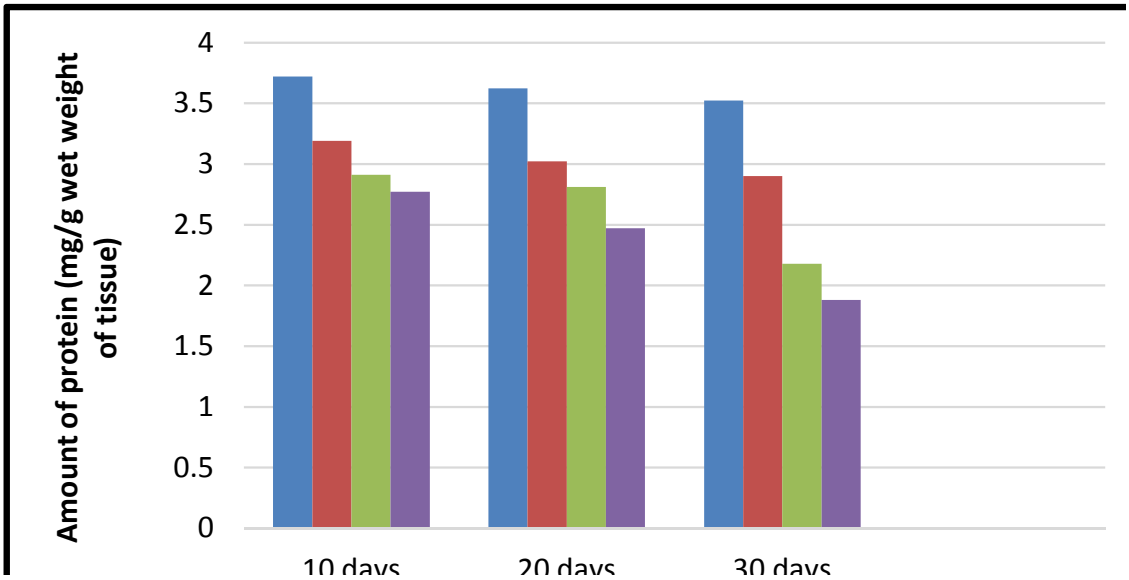


**Fig.3. Glycogen content of muscle in experimental fishes.**

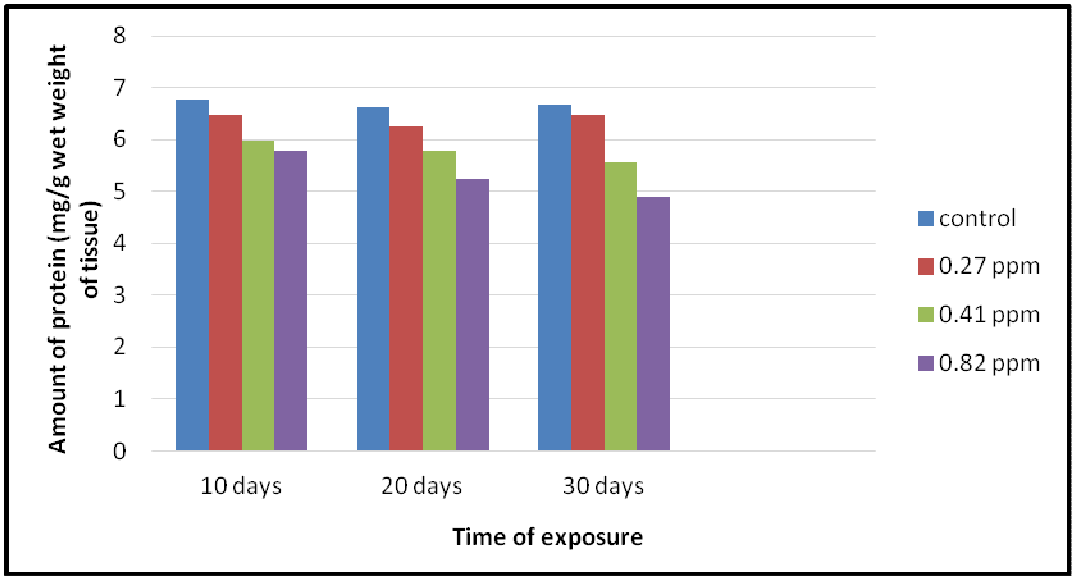


**Fig.4. Glycogen content of liver in experimental fishes.**

**DISCUSSION**

Kumar *et al*. (1994) studied on the effect of endosulfan in *Clarius batrachus* and reported that the rapid use of pesticide to control pests poses serious hazard to non target organisms including fishes in the aquatic bodies. Pesticides reaches aquatic bodies by direct application, spray from ground or aerial, atmosphere fallout, run-off from agricultural land and discharge of effluents. These pesticides persist in the environment and are found to be toxic to various aquatic animals including fishes. David *et al*. (2004) conducted a experiment on cypermethrin in *Thilapia mossambicuss* and reported that the carbohydrates are the primary and intermediary source of energy and in stress conditions carbohydrates depleted to meet energy demand. In the present study a decrease in carbohydrate energy source, glycogen in liver and muscle tissues of imidacloprid 17.8% SL treated *C. catla* as reported. The glycogen level of both the tissues showed a decreasing trend as treatment progressed. Depletion of glycogen may be due to direct utilization for energy generation, a demand caused by imidacloprid 17.8% SL induced hypoxia.

According to Rand and Petrocelli (1988) layering over gill membrane and filaments in the fish alters the aerobic nature and a shift towards anaerobiosis with decrease in stored carbohydrates. Results of the biochemical studies in *C. catla* showed similar results. Decline in glycogen content of muscle and liver after toxic stress have been reported in studies with aquatic animals by Shoba *et al.* (1989) and Hori *et al*. (2006). Similar drop off in glycogen in *Clarias batrachus* was observed after fish were intoxicated with organophosphate pesticide dimethoate and roger was reported from the works of Begum and Vijayaraghavan (1999). The depletion of glycogen in the tissues is an indication of typical stress response in fish challenged with pesticides. The observed reduction in glycogen content indicates the utilization of stored glycogen to meet the highly energy requirement under pesticide stress. Significant dose dependent decrease in glycogen content of tissue was observed in all the three sublethal concentrations of imidacloprid 17.8% SL.

Shamsi and Al-Akel (1986) studied on mercuric chloride treated *Oreochromis niloticus* and reported that exposure to various toxicants considered stress full such as heavy metal. Effect of endosulfan in *Cyprinus carpio* studied by Chandransekhar and Jayabalan (1993) and results shows that, the reduction in the muscle liver glycogen of the fish. Glycolysis in the fish exposed to pollutants may be expected to meet the energy requirements of the animal for the increased level of physical activity. The decrease in tissue glycogen of the fish exposed to different concentrations of imidacloprid 17.8% SL shows maximum decrease at highest dose concentration, indicating the mobilization of energy reserve to meet energy crisis. Nakamo and Tomlinson (1967) studied on the catecholamine and carbohydrate concentration in *Salmo gairdneri* in relation to physical disturbances and reported that disturbances are related to enhance circulating levels of both catecholamines and glucocorticoids. Thus the reduction in the level of glycogen in muscle and liver exposed to imidacloprid 17.8% SL in presence study may be related to stress induced increase in circulating catecholamines and adrenocorticostreroids.

Since fish have a very little amount of carbohydrate so next alternative source of energy is protein to meet the increased energy demand (Rao, 1999). The concentration of total protein in liver and muscle of *C. catla* exposed to imidacloprid 17.8% SL were found to be lower than those in control on all sampling days. Similar findings have been noted in fresh water field crab, *Barytelphusa guerini* on exposure to endosulfan by Reddy *et al.* (1991), and in the fish *Notopterus notopterus* exposed to various forms of stress by Narasimhan and Sundararaja (1971). The feasible explanation for these observations is that proteolytic activity was induced in these organisms due to stress. The decrease in protein level in liver and muscle tissues may be due to meet the higher energy demands for metabolic purposes. Another hypothesis has been advanced to explain the reduced protein level in *B.guerini* exposed to pesticide endosulphan by Reddy *et al.* (1991) reported that the physiological compensatory mechanisms are activated to either to provide intermediate for deriving energy through Kreb's cycle or to compensate for osmoregulatory problems by increasing the free amino acid level in blood; such mechanisms would have possibly operated in the test fishes exposed to imidacloprid 17.8% SL in the present study.

## CONCLUSION

The result of the present study indicate that imidacloprid 17.8% SL exposure during sublethal treatment induces significant changes in the biochemical parameters of *C. catla.* Tissue level protein and glycogen showed a significant reduction when the fishes were treated with sub lethal concentrations of imidacloprid 17.8% SL. Reduction was more evident in muscle tissues. Under the light of this study, it could be concluded that even at sub lethal levels imidacloprid 17.8% SL can cause depletion of energy resources in fishes. These changes may be potentially disruptive on the survivability of *C. catla*. This fact should be taken into consideration when imidacloprid 17.8% SL is used for pest control applicatons.

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