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Impact of Acetamiprid Toxicity on Lactate Dehydrogenase in Some Tissues of the Fish *Oreochromis mossambicus*

S. Justin Raj and Baby Joseph
Interdisciplinary Research Centre, Department of Biotechnology, Malankara Catholic College, Mariagiri, Kaliakkavilai, Tamil Nadu, India

Corresponding Author: S. Justin Raj, Interdisciplinary Research Centre, Department of Biotechnology, Malankara Catholic College, Mariagiri, Kaliakkavilai, Tamil Nadu, India Tel: 09751358502

ABSTRACT

Experiment was conducted on fish, *Oreochromis mossambicus* to study the effect of acetamiprid on tissue biomarker lactate dehydrogenase. Acetamiprid is a neonicotinoid pesticide and it is used by the farmers to protect their crops. This pesticide residue reaches to the aquatic ecosystem by many ways and affects the aquatic fauna. Lethal Concentration (LC_{50}) of acetamiprid for *O. mossambicus* has been calculated by probit analysis and recorded as 5.99 ppm at 96 h. Chronic exposure shows increased activity of LDH in liver, brain and gill tissues during all the exposure periods when compared with the control. This significant increase in enzyme activity was observed due to toxic effect of acetamiprid. Long term exposure of organisms to pesticides means a continuous health hazard for the fish population and it is on high risk by consuming these toxicated fishes.

Key words: Biomarker, neonicotinoid, acetamiprid, lactate dehydrogenase, sub-lethal toxicity, osmoregulation

INTRODUCTION

Pesticides become part of the water column and fish ingest the pesticides, usually through their gills, although sometimes through their scales. The pesticides diffuse into their organs and fat tissues and sequestered there causes severe alterations in the tissue biochemistry and histology of fish (John, 2007; Velisek *et al*., 2006; Banaee *et al*., 2011; Muthukumaravel *et al*., 2013). It is true that every animal has its own detoxification mechanism to get rid of foreign substances entering their body. The pesticides induce its effects first at cellular or even at molecular level, but ultimately it tends to create biochemical disorder that may even cause death. Enzyme analysis is widely used for rapid detection to predict early warning of pesticide toxicity (Dutta and Areids, 2003; Gabriel *et al*., 2012). Fish is considered as the most important and vital link in the food chain of ecosystem and the inland fisheries are important sources of protein in a nation's diet, a thorough understanding of pesticide effects on fish would be really vital for fish conservation and fisheries development. Many pesticides have been reported to produce a number of biochemical changes in fish both at lethal and more often, at sublethal level changes in ion concentrations, organic constituents, enzyme activity, endocrinal activity and osmoregulation (Banaee *et al*., 2008). Insecticides can cause serious impairment to physiological and health status of fish. Therefore, biochemical tests are routine laboratory tests useful in recognizing acute or chronic toxicity of insecticides (Al-Kahtani, 2011) and can be a practical tool to diagnose toxicity effects in target

organs and to determine the physiological status in fish. Biochemical changes induced by pesticidal stress lead to disturb the metabolism, inhibition of important enzymes, retardation of growth and reduction of fecundity and longevity of organisms (Murthy, 1986).

Lactate Dehydrogenase (LDH) is an enzyme found in almost all body tissues, such as heart, kidneys, liver, skeletal muscle, brain, erythrocyte and gills (Ahmad and Absar-ul Hasnain, 2005). LDH measurement is used to detect tissue disorders and as an aid in the diagnosis of tissue damage (Ahmad and Absar-ul Hasnain, 2005; Rao, 2006) . A significant decrease in LDH activity under the sub-lethal toxicity of quinolphos in different tissues of freshwater fish, was reported in *Channa punctatus*. Moreover, changes in LDH activity is a maker for tissue damage in fish and as a good diagnostic tool in toxicology studies (Min and Kang, 2008; Gabriel *et al*., 2012). The impact of these pesticides on aquatic organisms is due to the movement of pesticides from various diffuse or point sources. These pesticides are posing a great threat to aquatic fauna especially to fishes, which constitute one of the major sources of protein rich food for mankind (Sharma and Singh, 2007). In this study, the fish, *O. mossambicus* was investigated to evaluate the impact of acetamiprid on LDH activity in various tissues.

MATERIALS AND METHODS

Specimens of *O. mossambicus* were obtained from local vicinity and introduced into large glass tank (30×30×60 cm) disinfected with potassium permanganate and washed thoroughly prior to introduction of fish (to prevent fungal infection). Fishes with same weight were acclimatized for about 20 days before the commencement of the experiment. They were fed on commercial fish food which was given daily at morning hours. The LC_{50} of acetamiprid was calculated by the log-dose/Probit regression line method (Finney, 1971) and was recorded. Sublethal or safe level concentrations were derived from 96 h LC_{50} as per the procedure given by APHA (2005) to observe various responses of the test fishes on prolonged exposure to acetamiprid. In the present study 1/15 of the 96 h LC_{50} were selected as sublethal concentration and the fishes were exposed to this concentration for a period of 7, 14, 21 and 28 days. A control batch corresponding to each test group was simultaneously experimented to compare the toxicated values of LDH activity, which was estimated by the method of Kaplan and Pesce (2009). Fresh concentrations were supplied daily to maintain a constant toxic media. At the end of each exposure period, fishes were sacrificed and tissues, such as liver, brain and gill were dissected and were used for the analysis.

RESULTS AND DISCUSSION

The LDH activity in the liver, brain and gill tissues of *O. mossambicus* exposed to control and treated (acetamiprid) established an increasing trend in liver, brain and gill tissues with increasing exposure period. The LDH activity of control liver tissue was 123.6±1.15 IU L^{-1} and it increased gradually to 213±3.21 IU L^{-1} for acetamiprid treated liver tissue at 28th day and in brain and gill tissues there is a significant increase from 332±1.52-821±1.52 and 378.3±2.08-1081±1.5 IU L^{-1} for acetamiprid treated tissues at 28th day exposure (Table 1). Changes in the enzyme concentration (LDH) after exposure to acetamiprid was shown in Table 1. The increase in LDH over the control values ranged from 141-213 IU L^{-1} for acetamiprid liver, 351-821 IU L^{-1} for acetamiprid brain and 413-1081 IU L^{-1} for acetamiprid gill in 0, 7th, 14th, 21st and 28th day exposure time. Jee *et al*. (2005) found that an increase in activities of serum LDH in Korean rockfish (*Sebastes schlegeli*) exposed to cypermethrin. The results are in agreement with his findings (Jee *et al*., 2005). Exposure to cypermethrin produced a significant increase in

Table 1: Showing the LDH activity of liver, brain and gill of control and treated (acetamiprid) fish *Oreochromis mossambicus* in different exposure periods

Duration (days)	Li (C)	Li (A)	Br (C)	Br (A)	Gi (C)	Gi (A)
0	114.0±1.00	141±1.52	321.0±1.00	351±1.52	361.0±1.00	413±1.00
7	116.0±1.00	162±1.52	323.0±2.00	480±1.15	362.6±1.52	561±2.08
14	117.6±1.52	170±2.30	328.5±1.00	591±1.52	366.6±1.52	691±1.52
21	122.0±2.00	196±1.52	331.0±1.00	651±1.52	372.6±2.30	852±2.00
28	123.6±1.15	213±3.21	332.0±1.52	821±1.52	378.3±2.08	1081±1.50

Values are expressed as Means±SD for n = 3, C: Control, A: Acetamiprid treated, Li- liver, Br: Brain, Gi: Gill tissue). The results are expressed in IU L^{-1}

the activities of serum LDH in fish *Rhamdia quelen* (Borges *et al*., 2007) and *Labeo rohita* (Das and Mukherjee, 2003), respectively.

The level of tissue lactate content may also act as an indicator of anaerobic respiration as this process might enable the animal to tolerate hypoxic condition (Masopust, 2000). The enhanced LDH activity in *O. mossambicus* exposed to acetamiprid observed in this study may reflect the increased rate of conversion of lactate to pyruvate and then to glucose. This result is in agreement with those reported for other fish species exposed to metasytox, chlordane, cypermethrin and phenol (Jee *et al*., 2005; Hori *et al*., 2006). Several reports that revealed decreased LDH activity in tissues under various pesticide toxicity conditions in different fishes, such as *O. mossambicus* (Rao, 2006) and *C. punctatus* (Agrahari *et al*., 2007). Hernandez *et al*. (2006) reported some pesticides, such as organophosphates are able to cause inhibition of LDH activity. Moreover, changes in LDH activity is a maker for tissue damage in fish and as a good diagnostic tool in toxicology studies (Min and Kang, 2008).

CONCLUSION

Enzymes are easily altered by even smaller changes, either in internal or external medium. The LDH is a major oxidative enzyme in carbohydrate metabolism. It may be used for demonstrating tissue damage in fish and as a good diagnostic tool in toxicology studies. The study depicts that the pesticide, acetamiprid is highly toxic to the fish *O. mossambicus* and the stress response showed by fish are dependent on concentration and duration of exposure. The LDH is a general indicator of the existance and severity of acute or chronic tissue damage and sometime as a monitor of progressive conditions like hemolytic anemia. From the present study it may be concluded that long term exposure to acetamiprid means a continuous health hazard for the fish population. Therefore it is required to monitor the aquatic system and predict the toxic effect of pesticides on fish.

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