Original Research Article

Lipid peroxidation and antioxidant effects of Lead acetate: An experiment in various organs of *Oreochromis mossambicus* (Peters 1852)

ABSTRACT

The effect of heavy metal Lead acetate exposure of fingerlings of *Oreochromis mossambicus*. The fish were exposed to sub lethal concentration LC_{50} of Lead acetate for 96 hrs. for 21 and 28 days and the various organs viz. Liver, Gill, Kidney and Muscle tissues were studied for lipid peroxidation (LPO), Reduced glutathione (GSH), glutathione peroxidase (GPx), Catalase (CAT), and superoxide dismutase (SOD). These observed mean data were subjected to student 'T' test. The Lead acetate exposed to *O. mossambicus* fingerlings which tested on various organs of Liver, Gill, Kidney and Muscle for assessing the effect of LPO, GSH, GPx, CAT and SOD on control and treated fish. The predominant effects were observed at 28 days of exposer period, the Liver, Gill, Kidney and Muscle has drastically suffered by exposer of Lead acetate. Liver: 2.89, 2.40, 1.21, 1.33 and 0.42μmole/mg of protein/hr, Gill: 0.948, 1.18, 0.326, 0.62 and 0.24μmole/mg of protein/hr, Kidney: 0.968, 1.56, 1.006, 0.46 and 0.26 μmole/mg of protein/hr and Muscle: 0.988, 1.12, 0.226, 0.62 and0.12 μmole/mg of protein/hr were recorded on tissue of treated fish. Moreover, the respective concentration of Lead acetate exposed against fingerlings which compared with control and experimental group, the percentage of concentration on LPO, GSH, GPx, CAT and SOD of various organs of treated fish were increased as well as decreased in the experimental group then the control group. The huge exposure of Lead acetate on *O.*

mossambicus fingerlings which showed unpredictable negative health defects on various system. Beyond the threshold level of $Pb(C_2H_3O_2)_2$ it may interfere the metabolic activity of organism. Hence, the present experiment which gives the great knowledge about effects of Lead acetate on aquatic organism.

Keywords:

$Pb(C_2H_3O_2)_2$, *Oreochromis mossambicus*, Various organs, Metal toxicant, lipid peroxidation, Antioxidant effects

1. INTRODUCTION

The industrial effluents are having rich amounts of heavy metals as the results the metal contamination may occurred in different states of biosphere [1]. The rapid settlement of various pollutant which promoting countless of disorder to both faunal and floral communities [2]. Among the various eco-system, particularly the aquatic ecosystem predominantly receives a wide range of pollutants. The pollutant in aquatic environments which promote unavoidable hazards, disturbs the survival and growth rate, reproduction, biochemical, physiological effects in the various aquatic organisms [3]. Lead acetate ($Pb(C_2H_3O_2)_2$) is a heavy metal which causing many different health defects: skin allergy and irritation, organ and system failure, acquit and chronic toxicity as well as countless of reports have investigated towards hazardous capabilities of $Pb(C_2H_3O_2)_2$ in many experimental faunas [4]. Lead acetate is a prime toxic metal which has been used in different industrial properties and it has hugely abundance in dyes, batteries, insecticides, fuels, and other industrial properties [5]. It extensively exposed by polluted nutrients and aquatic mediums [6]. They mainly give toxic effect in different internal organs including liver, kidney, nerves system, reproductive and other side effects also [7]. The enzymes

are very importance for regular metabolic activities including many organs and it any changes occurred that could be led into many different side effects [8]. The degenerative changes due to the combined metal toxicity exhibited in the liver, gill, kidney and muscle alter level of a number of its enzymes [9]. The enzymes are biomarkers of acute hepatic damages and its bioassay can serve as a diagnostic tool for assessing the functions of the liver, gill, kidney and muscle [10]. Very few works only have reported on aquatic organisms to Lead acetate exposure. Henceforth, the present study focuses on, heavy metal Lead acetate on the lipid peroxidation and antioxidant level in the liver, gill, kidney and muscle tissue of *O. mossambicus*.

2. MATERIALS AND METHODS

2.1 Experimental organism

The *Oreochromis mossambicus* were collected from the fish farm located at Poompuhar Village, Mayiladuthurai District, Tamil Nadu, India. The fish were brought to the laboratory and transferred to the rectangular cement tanks (100X100X100cm) of 1000 liters capacity containing chlorine free aerated well water and acclimatized to the food and laboratory conditions with 12 hrs. dark and 12 hrs. light cycles, pH range of 6.90 to 7.10 and temperature ranging from 18 to 23°C for 15 days.

2.2 Experimental design

Fish were selected for the experiment from the stock irrespective of the sex. The size selected for the experiments were 80-100mm length and 5-10g of weight fish were divided into two equal groups each comprising of 20 fishes. Each group was kept in separate plastic trough. The first groups were kept as control and were maintained in normal water without any treatment. The

second group was exposed to a sub-lethal concentration of 96 hrs. LC_{50} of Lead acetate for 21 and 28 days. Solution was renewed once in 24hrs. exposure period. The fish from the respective experimental as well as control groups were sacrificed and liver, gill, muscle and kidney tissue were isolated from the fish and used for the estimation lipid peroxidation and antioxidant parameters.

2.3 Estimation of lipid peroxidation and antioxidants

The isolated liver, gill, kidney and muscle tissue of the control and experimental fish was used for the level of lipid peroxidation in liver tissue by the method [11], reduced glutathione was determined by the method [12], glutathione peroxidase activity was determined by the method [13], Catalase was determined by the method [14] and Superoxide dismutase activity was assayed by the method [15]. The Statistical significance of control and experimental means were analyzed by student 'T' test.

3. RESULTS AND DISCUSSION

The selected concentration of heavy metal Lead acetate exposed to fingerlings of *Oreochromis mossambicus* which tested on various organs such as Liver, Gill, Kidney and Muscle in various interval periods (21 and 28 days) for assessing treatment and control against lipid peroxidation and antioxidants level. The LPO, GSH, GPx, CAT and SOD of various organs of control fish: Liver: 1.23, 6.78, 1.98, 3.5 and 1.56 µmole/mg of protein/hr; Gill: 0.234, 1.98, 0.988, 1.82, and 0.95 µmole/mg of protein/hr; Kidney: 0.289, 2.86, 1.856, 1.92 and 0.88 µmole/mg of protein/hr and Muscle: 0.244, 1.68, 0.86, 1.46 and 0.64 µmole/mg of protein/hr were recorded on tissue of control fish. Furthermore, Lead acetate exposed to fingerlings which tested on various organs at

21 hrs. interval periods for assessing of LPO, GSH, GPx, CAT and SOD of various organs of treated fish: Liver: 2.74, 3.29, 1.35, 1.64 and 0.6 µmole/mg of protein/hr; Gill: 0.856, 1.42, 0.422, 0.82 and 0.42 µmole/mg of protein/hr; Kidney: 0.856, 2.08, 1.084, 1.32 and 0.51 µmole/mg of protein/hr and Muscle: 0.824, 1.21, 0.322, 0.72 and 0.20 µmole/mg of protein/hr were recorded on tissue of treated fish. Furthermore, Lead acetate exposed to fingerlings which tested on various organs at 28 hrs. interval periods for assessing of LPO, GSH, GPx, CAT and SOD of various organs of treated fish: Liver: 2.89, 2.40, 1.21, 1.33 and 0.42µmole/mg of protein/hr; Gill: 0.948, 1.18, 0.326, 0.62 and 0.24 µmole/mg of protein/hr; Kidney0.968, 1.56, 1.006, 0.46 and 0.26 µmole/mg of protein/hr and Muscle: 0.988, 1.12, 0.226, 0.62 and 0.12 µmole/mg of protein/hr were recorded on tissue of treated fish. Moreover, the respective concentration of Lead acetate exposed against fingerlings which compared with control and experimental group, the percentage of concentration on LPO, GSH, GPx, CAT and SOD of various organs of treated fish were increased as well as decreased in the experimental group then the control group. At 21 and 28 days exposer period the percentage of LPO, GSH, GPx, CAT and SOD were considerably increased and decreased levels: Liver: 43.61, -33.60, -16.30, -30.24, -33.29 and 82.29, -30.22, -22.38, -41.80 and -45.28; Gill: 38.49, -12.39, -18.50, -38.22, -21.08 and 53.64, -21.78, -28.63, -42.28 and -36.90; Kidney: 24.62, -22.39, -16.81, -42.60, -31.06 and 53.28, -38.74, -24.40, -48.32 and -42.06 and Muscle: 38.44, -18.60, -12.48, -25.44, -32.28 and45.28, -22.46, -17.12, -37.66 and -39.62 were recorded on tissue of treated fish. The mean values of LPO, GSH, GPx, CAT and SOD values of control and Lead acetate treated group was compared for their statistical significance at $P<0.05$. Previously, the similar kind of study reported, the SiO_2 nanoparticles at 5 mg/L for 96 hrs. showed the various biological changes in the liver tissues, by the exposure they were changed the disorganized hepatic parenchyma,

vacuolization and disintegrated nucleus, cytoplasmic vacuolization and leukocyte infiltration [16]. These lipid peroxides and hydroxyl radicals may cause outer skin damages and which led into destroy the entire cell membrane and its contents [17]. In the present work, the Lead acetate exposed to several hours of aquatic organism *O. mossambicus* fish which drastically reduced the free radical scavenger enzymes GPx, CAT, and SOD. The drastic destruction of GPx, CAT and SOD (free radical scavenger enzymes) by the direct influences of Lead acetate sublethal concentration on concern fish species *O. mossambicus*. The higher concentration of heavy metal directly interrelates with internal organ tissues which considerably reduced the enzyme secretion and its activities [18]. The $PbNO_3$ exposed to *O. niloticus* fingerlings with their LC_{50} values were 143.3 mg/l for *O. niloticus* as well as which drastically suppressed counts of RBCs, Hb, PCV, MCV, MCH, MCHC, AST and ALT deterioration of hepatic tissue. The $PbNO_3$ toxicity on *O. niloticus* were observed the changing of antioxidant enzymes level GPx, and CAT in hepatic tissue [19]. The industrial wastes highly suffering the aquatic organisms which constrict of various heavy metals Mn, Fe, Pb, Ni, Cr, Hg, As, Zn and Fe. They were seriously affected the bio-system of *O. niloticus* fish population, the Mn and Fe LC_{50} values were 147.36mg/L and 90.52mg/L, respectively [20]. Correspondingly, Lead (Pb^{2+}) heavy metal were very seriously suffered oxidative stress as well as which caused ROS level, lipid peroxidation, reduced immune and disease resistance, etc., [20]. Cadmium (Cd) metal rises the production of enzymatic rate in rodents and aquatic faunas [21]. The present study, resulted low production in GSH levels as well as augmented ROS and LPO levels by the exposure of Lead acetate in *Oreochromis mossambicus.* The similar observations were showed in heavy metal of Arsenic (As) exposed against catfish at lower concentration [22]. The heavy metal at low/ high level concentration which directly influenced on enzyme secretion on many of the aquatic species [23], the Arsenic

(As) heavy metal changes the secretion level of GSH, SOD and Catalase in liver tissues of tilapia fish [24]. It can be stated that the different interval exposure of Lead acetate affects the various organs of *O. mossambicus* for observing lipid peroxidation and antioxidant responses under laboratory conditions.

4. CONCLUSION

Lead acetate is a high toxic metal in many of the faunal species which are considerably making many hazards to environment as well as living species. The huge exposure of Lead acetate may cause the various health issues as well as metabolic activity also. This study may give the awareness about exposure of heavy metal in various eco-system and it could be given the great knowledge about metal contamination in aquatic fauna and various health defects on faunal communities by using of higher concentration.

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Table 1. The observation of lipid peroxidation and antioxidants level in various organs of *Oreochromis mossambicus* by the exposure of Lead acetate heavy metal.

Various organs	Parameters	Control	Exposure 21 days	% Change	Exposure 28 days	% Change
Liver	LPO	1.23±0.24	2.74±0.39	43.61	2.89±0.76	82.29
	GSH	6.78±0.22	3.29±0.27	-33.60	2.40±0.39	-30.22
	GPx	1.98±0.64	1.35±0.31	-16.30	1.21±0.62	-22.38
	CAT	3.58±0.24	1.64±0.28	-30.24	1.33±0.21	-41.80
	SOD	1.56±0.28	0.65±0.26	-33.29	0.42±0.44	-45.28
Gill	LPO	0.234±0.22	0.856±0.34	38.49	0.948±0.89	53.64
	GSH	1.98±0.46	1.42±0.86	-12.39	1.18±0.46	-21.78
	GPx	0.988±0.26	0.422±0.36	-18.50	0.326±0.22	-28.63
	CAT	1.82±0.44	0.82±0.25	-38.22	0.62±0.42	-42.28
	SOD	0.95±0.02	0.42±0.48	-21.08	0.24±0.20	-36.90
Kidney	LPO	0.289±0.25	0.856±0.28	24.62	0.968±0.22	53.28
	GSH	2.86±0.24	2.08±0.42	-22.39	1.56±0.63	-38.74
	GPx	1.856±0.21	1.084±0.12	-16.81	1.006±0.22	-24.40
	CAT	1.92±0.42	1.32±0.20	-42.60	0.46±0.28	-48.32
	SOD	0.88±0.44	0.51±0.28	-31.06	0.26±0.77	-42.06
Muscle	LPO	0.244±0.32	0.824±0.37	38.44	0.988±0.32	45.28
	GSH	1.68±0.04	1.21±0.24	-18.60	1.12±0.28	-22.46
	GPx	0.86±0.021	0.322±0.72	-12.48	0.226±0.08	-17.12
	CAT	1.46±0.22	0.72±0.02	-25.44	0.62±0.76	-37.66
	SOD	0.64±0.20	0.20±0.40	-32.28	0.12±0.44	-39.62

The data were statistically evaluated into Mean ± Standard Error
The percentage of bio-activities were calculated by replication of six times
The Statistically significance at $p<0.05$ and subjected into student 'T' test.
LPO: Lipid peroxidation (µmole/mg.of protein)
GSH: Glutathione ((µmole /mg.of protein)
GPx: Glutathione peroxidase (µmoles/mg protein)
CAT: Catalse (Unit/mg.of protein)
SOD: Superoxide dismutase (Unit/mg.of protein)