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Research Article

Mercury toxicity on lipid peroxidation in gill and hepatopancreas of freshwater bivalve *Lamellidens corrianus*

Mestry Urmila D and Bhosale Tanaji S.

Department of Zoology,
Yashwantrao Chavan Institude of Science, Satara (Maharashtra) India
Pinka84@rediffmail.com

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Abstract

The present investigation was aimed at the assessment of mercury chloride burden on lipid peroxidation in gills and hepatopancreas of freashwater bivalve *Lamellidens corrianus*. The lipid peroxidation measured in term of malondialdehyde (MDA) was increased in all groups like Control, Lc0,Lc50,1/10th and 1/20th Lc50. Increased level of MDA denote oxidative stress which damage cell. Maximum level was found in group treated with Lc50 Hg (2.97ppm) concentration. The gill tissue was more susceptible for mercury toxicity which showed maximum MDA level in chronic exposure to 1/20th Lc50 group as compared to hepatopancreas.

INTRODUCTION

The aquatic life is always under stress due to anthropogenic activity such as industrial production, agricultural as well as domestic waste and heavy metals (Kaplan *et al.*, 2011). Plenty of study reveals that those hazardous chemicals are harmful to aquaculture (Turja *et al.*, 2013). They directly enter food chain through aquatic animals. They are also a intermediatory host of many diseases. Oysters, mussels and fishes are consumed in either raw or cooked condition, had reported as a main sources of food poisoning (Gourmelon *et al.*, 2010). Suspension feeding aquatic animals like bivalve, widely used as an indicator of pollution due to their capacity to bioaccumulation and concentrate organic and metabolic pollutant (Amagliani *et al.*, 2012; Fleming *et al.*, 2006).

It is well known that mercury is one of most dangerous non essential heavy metal. It exists in environment in varieties of forms with different toxicity (Clarkson and Magos, 2006). Mercury also used for making different pesticides. Over use of these pesticides drain off into water systems and accumulates into animals. In animals it generates oxidative stress through production of reactive oxygen species (ROS), as superoxide anion radical (O_2^-), hydrogen peroxide (H_2O_2) and hydroxyl radicals (OH) (Ahmad *et al.*, 2011; Nicholson, 2003). They also do decrease the antioxidant system that modifies ROS to less reactive intermediates (Thirumavalavan, 2010).

The membrane lipids then steal by ROS formed oxidative degradation, lipid peroxidation. The malondialdehyde (MDA) is reactive aldehyde considered as byproduct of lipid peroxidation (Wu *et al.*, 2011; Richardson *et al.*, 2008; Company *et al.*, 2008). So the paucity of literature on assessments of MDA level in specific tissues like gill and hepatopancreas of mercury toxicodynamics in bivalve the present study was aimed.

MATERIALS AND METHODS

The freshwater bivalve *Lamellidens corrianus* was procured from the Madkhol, local water body of Sawantwadi (Maharashtra). The bivalves were acclimatized to laboratory condition in glass aquaria for 10 days. The acclimatized bivalves were divided into group of 10 each as under.

Gruop I:- control with normal water.

Group II:- Lc0 concentration after 96hr.(0.20ppm) treated $Hgcl_2$ water.

Group III:- Lc50 (2.97ppm) treated HgCl2 water.

Group IV:- LC50/10^{th} (0.29ppm) treated HgCl2 water for 30 days.

Group V:- LC50/20^{th} (0.14ppm) treated water for 30 days.

The lipid peroxidation was estimated in homogenates of hepatopancreas and gill. The sample was prepared by homogenizing the tissues in 0.8% NaCl solution. The homogenate prepared was centrifuged for 10 min. at 1000 x g. The supernatant was used as sample. To 1.0 ml of the tissue sample 2.0 ml of a TCA-TBA-HCl (in the ratio 15% w/v Tricarboxylic acid: 0.375% W/V Thiobarbituric acid: and 0.25 N HC1) was added. The mixture was heated in boiling water bath for 10 min., cooled to room temperature, and the precipitate was removed by centrifugation at 1,000g for 10 min. The absorbance of TBA reactive substances (TBARS) was measured at 535 nm against a blank. The TBARS were quantified using an extinction coefficient of 1.56 x 10^5 $M^{-1}cm^{-1}$. Results are expressed as nmol MDA/mg protein. Protein in the samples was estimated by Lowry method (1951).

All values are express as mean ± SD. The statistical analysis was performed using one way analysis of variance (ANOVA) followed by Tukey's Post Hoc test. A value of $p<0.01$ was considered statistically significant.

RESULTS AND DISCUSSION

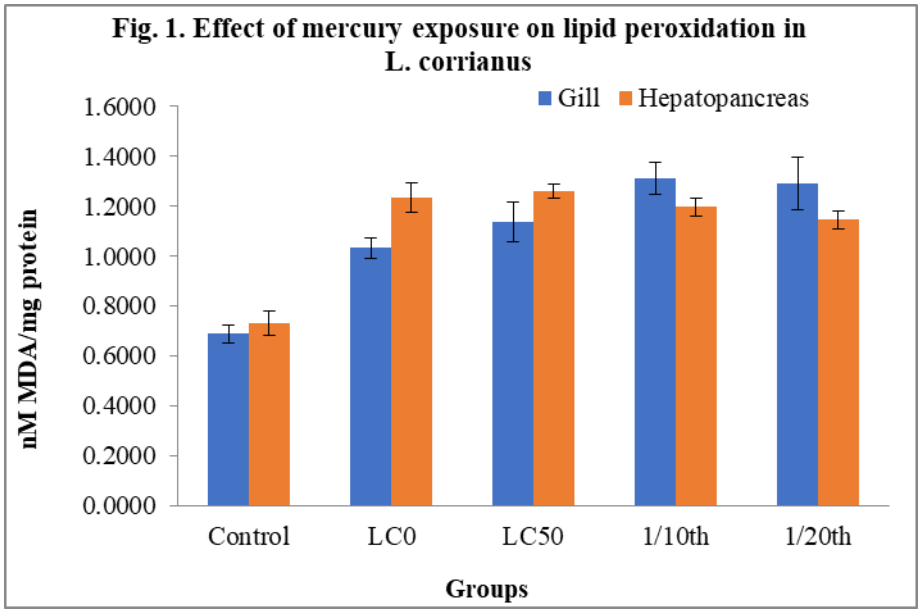
Lipid peroxidation was considered as major indicator of oxidative stress, and MDA was to be ideal biomarker for this process. Bivalves showed increased lipid peroxidation in all groups exposed to mercury chloride. In gill tissues of Group II exhibited increase in MDA content by 50% whereas in group III it was 65% as compared with control group. The maximum significant increase in MDA content was observed in chronic group IV by 90% and in chronic group V showed increased by87% as compared with control. The similar results were observed in a recent past (Xiaoyu *et al.*, 2011; Ahmad *et al.*, 2011). In fact, highest level of MDA enhanced antioxidant defense system which not protects gill tissues from ROS mediated damage.

Table1. Effect of mercury exposure on lipid peroxidation in *L. corrianus*

Group	Gill	Hepatopancreas
Control	0.6885 ±0.0356	0.7315 ± 0.0500
LC0	1.0336 ±0.0412 ***	1.2355 ±0.0585 ***
LC50	1.1360 ±0.0800 ***	1.2603 ±0.0286 ***
1/10th	1.3113 ±0.0655. ***	1.1964 ±0.0338 ***
1/20th	1.2914 ±0.1059 ***	1.1457 ±0.0351 ***

Data are mean ± S.D. mM MDA/mg protein. *** indicates $p<0.001$.

In case of hepatopancreas MDA content was found 68% in group II and 72% in group III of acute exposure. Whereas in Group IV showed 63% MDA and Group V it was 56% MDA recorded which is significantly low to gill. The hepatopancreas of bivalve is a key organ of metabolism and it is main site of digestive enzymes, detoxication and excretion. Due to detoxication of mercury in same tissue result showed low contents of MDA. Similar finding also do support to above results. (Rashmi *et al.*,2005).



The finding of this investigation revealed that the intoxication of Hg caused increased MDA level significantly this may due to induced formation of reactive oxygen species. It can be concluded that Hg is nonessential heavy metal caused damage to cells.

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