Short Communication(NS-3)

SHORT TERM TOXIC EFFECT OF LEAD NITRATE, LEAD ACETATE AND MERCURIC ACETATE ON TADPOLE OF *Rana tigrina*

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Received December 10, 2011 *Accepted March 2, 2012*

ABSTRACT

Present paper describe the effect of lead nitrate, lead acetate and mercuric acetate on tadpole of fresh water Rana tigrina at the five different concentrations (50 µg/L, 100 µg/L, 200 µg/L, 400 µg/L and 800 µg/L) for 120 hours exposure. Mercuric acetate caused 100% mortality within six hour in the three different concentration (200 µg/L, 400 µg/L and 800 µg/L) used while in 50 µg/L concentration 100% tadpole were survived up to 24 hours. However, gradual decrease in survival percentage was noted in further duration of exposure i.e. 80% survived up to 48 hours, 60% survived up to 72 hours and 20% survived up to 96 hours. In 120 hours exposure of the same concentration all the tadpole were observed died. In both, lead nitrate and lead acetate, in 50 µg/L concentration all the tadpole survived up to 72 hours. However, in 96 hours and 120 hours exposure of the lead nitrate and lead acetate 80% and 60% tadpoles were survived respectively.

Key Words : Frog tadpole, Mercuric acetate, Lead nitrate, Lead acetate, *Rana tigrina*.

INTRODUCTION

Amphibians are ideal for genotoxicity monitoring of aquatic ecosystem due to their high sensitivity of toxic substances and widely used as bioindicator to detect the presence of toxic substances in aquatic system[1-5]. The study of toxic effect of metal will help to understand the percentage of species survival and the cause of decline in amphibian population in aquatic ecosystem. Since Amphibians are regarded as key components of many ecosystems, there disappearance may create a complexity to sustain an ecosystem[6]. Mercury is a mutagen and carcinogen, their toxicity depends upon the form of mercury, dose route of ingestion and with the exposed organism's species, sex, age, and general condition[7-8]. Mercury has a high potential for bioaccumulation and biomagnification, their biomagnified concentration reported in fish up to 1, 00000 times the ambient water concentration[9-10].

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The toxicity of mercury to amphibian's tadpole is similar to those of the fish. Mercury (II) or mercuric salts are much more common in the environment as compared to mercury (I) or mercurous salts. If these salts are soluble in water, considered toxic. Organomercury compounds, such as methyl or butyl mercury chloride are more toxic to aquatic plants than inorganic forms[11].

Direct exposure of lead to the algae, benthic invertebrates embryos, fingerlings of freshwater fishes and amphibians creates main potential ecological impact that is it can be bioconcentrated and tends to decrease with increasing trophic levels in freshwater habitats but does not bioaccumulate[12]. Loss of sodium, reduced learning capability, and developmental problems arise in amphibians is due to a limited adverse effects of Lead[13]. Muscular and neurological degeneration and destruction, growth inhibition, mortality, reproductive problems and paralysis in fish is caused by high level of lead exposure[14]. Anthropogenic practices are the primary cause of lead poising in fish in contaminated water[15].

AIMS AND OBJECTIVES

The aim of this study was to detect the short term toxic effect of lead nitrate, lead acetate and mercuric acetate on the survival of tadpole of fresh water *Rana tigrina* at different concentration and to see that in lead acetate and lead nitrate which one is more toxic.

MATERIAL AND METHODS

Tadpoles of *Rana tigrina* were collected from Holkar science college (Indore), during rainy season with the help of fish net and bucket. Then they were acclimatized to the lab condition (for five days) and grouped into control and experimental. The experimental tadpoles (five in water) were placed in separate Petri dishes containing 50 µg/L, 100 µg/L, 200 µg/L, 400 µg/L and 800 µg/L concentrated tests chemical. Experimental and control petri dishes were covered with small piece of mosquito net so that tadpoles were not come out from the dish.

In observation number of tadpoles survived in

Table 1 : Mercuric acetate- survival percentage of tadpoles in different concentration and time duration

Duration of exposure	50 µg/L	100 µg/L	200 µg/L	400 µg/L	800 µg/L
6 hour	100% (5)	80% (4)	0% (0)	0% (0)	0% (0)
12 hour	100% (5)	80% (4)			
24 hour	100% (5)	0% (0)			
48 hour	80% (4)				
72 hour	60% (3)				
96 hour	20% (2)				
120 hour	0% (0)				

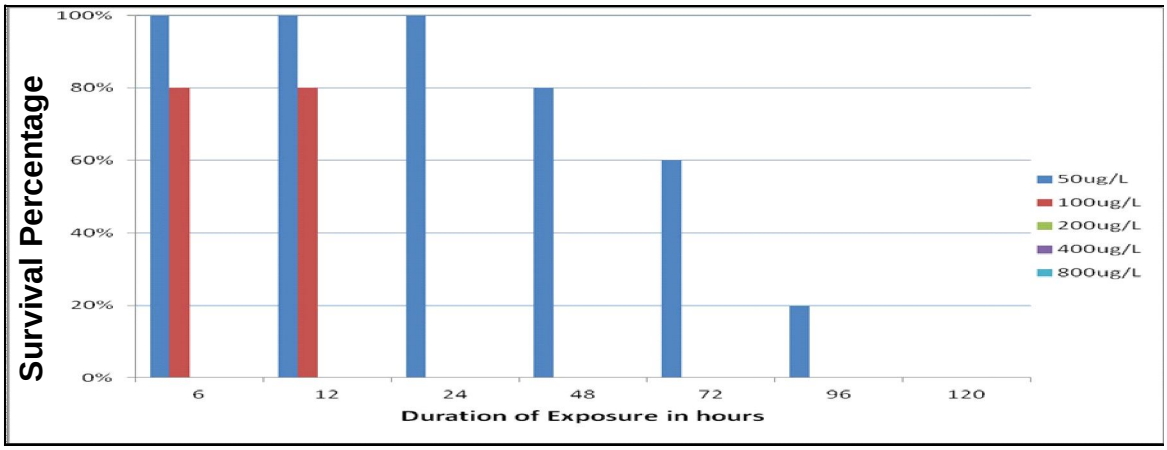

Fig. 1 : Survival percentage of tadpole on different concentration of mercuric acetate

Table 2 : Lead nitrate- Survival percentage of tadpoles in different concentration and time duration

Duration of exposure	50 µg/L	100 µg/L	200 µg/L	400 µg/L	800 µg/L
6 hour	100% (5)	100% (5)	100% (5)	80% (4)	60% (3)
12hour	100% (5)	100% (5)	100% (5)	80% (4)	60% (3)
24 hour	100% (5)	100% (5)	100% (5)	80% (4)	60% (3)
48 hour	100% (5)	100% (5)	100% (5)	60% (3)	60% (3)
72 hour	100% (5)	80% (4)	60% (3)	60% (3)	60% (3)
96 hour	80% (4)	60% (3)	40% (2)	40% (2)	20% (2)
120 hour	60% (3)	40% (2)	40% (2)	40% (2)	0% (0)

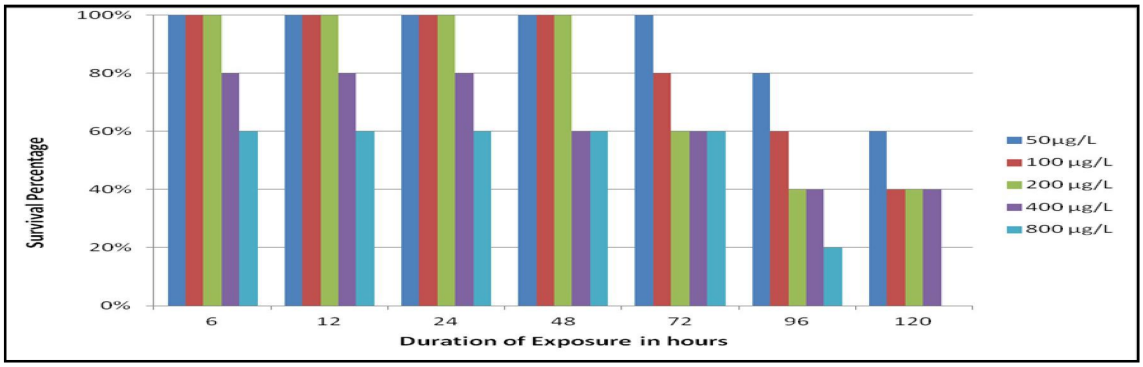

Fig. 2 : Survival percentage of tadpole on different concentration of lead nitrate

Table 3 : Lead acetate- Survival percentage of tadpoles in different concentration and time duration

Duration of exposure	50µg/L	100 µg/L	200 µg/L	400 µg/L	800 µg/L
6 hour	100% (5)	100% (5)	100% (5)	100% (5)	100% (5)
12 hour	100% (5)	100% (5)	100% (5)	100% ((5)	100% (5)
24 hour	100% (5)	100% (5)	100% (5)	100% (5)	100% (5)
48 hour	100% (5)	100% (5)	100% (5)	100% (5)	100% (5)
72 hour	100% (5)	80% (4)	80% (4)	80% (4)	80% (4)
96 hour	80% (4)	60% (3)	60% (3)	60% (3)	60% (3)
120 hour	60% (3)	40% (2)	40% (2)	40% (2)	40% (2)

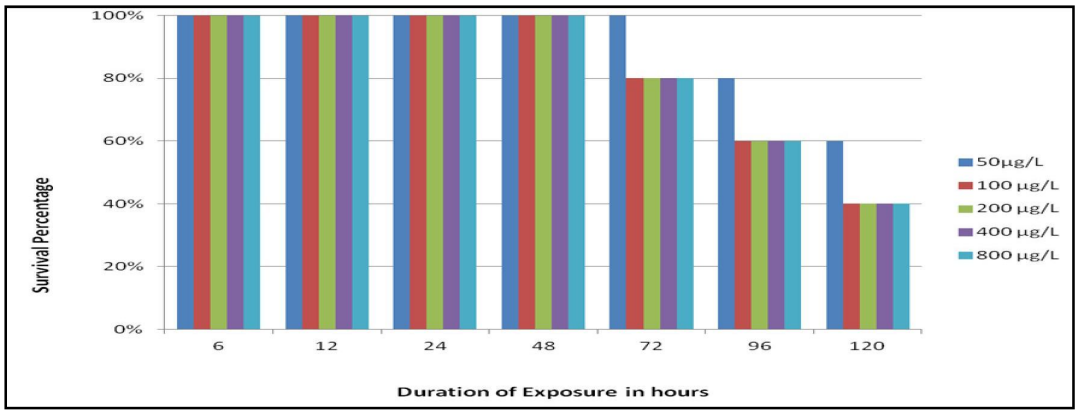

Fig. 3 : Survival percentages of tadpoles on different concentration of Lead acetate

different concentration in different time duration (6h, 12h, 24h, 48h, 72h, 96h, and 120h) are shown in (**Table 1** to **Table 3)**, then their survival percentage were calculated from these data.

RESULTS AND DISCUSSION

In the present study the exposure of five different concentrations (50 µg/L, 100 µg/L, 200 µg/L, 400 µg/L and 800 µg/L) of Lead acetate, Lead nitrate and Mercuric acetate were given to the tadpole of freshwater *Rana tigrina* to find out the percentage of survival. Data collected during different experiments conducted for the present study are summarized in **Table 1** to **Table 3** and shown **Fig. 1** to **Fig. 3**.

Comet assay after *invivo* toxicity tests (6, 24 and 96 h) shown that Pb was genotoxic for all the three tissue (blood, liver and gills cells of fishes) analysed after 96 h exposure[16].

At 50 µg/L exposure of mercuric acetate, 100% of the tadpoles were survived upto 24 h and then gradual decline in survival percentage (80% survived upto 48 h, 60% survived upto 72 h, 20% survived upto 96 h) were recorded, however all the tadpoles were died in 120 h in same concentration. In 100 µg/L concentration of mercuric acetate, 80% of tadpoles were survived upto 12 h. All the tadpoles were died within 6 h in onward concentration of mercuric acetate (i.e. for 200 µg/L, 400 µg/L and 800 µg/L). All the tadpoles of *Rana pipiens* which were raised in water containing more than 0.05 ppm of mercury, died in 48 h due to the disturbance of the osmotic regulatory system[17]. Authors also support his view.

In case of both lead acetate and lead nitrate 100% of the tadpoles were survived upto 72 h, 80% survived upto 96 h and 60% survived upto 120 h in 50 µg/L concentration. In 100 µg/L and 200 µg/L concentration of both these solution 100% percent tadpoles were survived upto 48 h and then gradual decrease in survival percentage were recorded but major difference in percentage survival were noted at 400 µg/L and 800 µg/L for both compound of Lead which are summarized in **Table 2** and **Table 3**. The toxic effect of lead on aquatic animals are associated with oxidative stress.Author agree with this statement[18], however The direct interaction between lead and DNA by covalent binding of Pb^{2+} to DNA[19]. Some recent evidence suggest the relation between lead intoxication and production of Reactive Oxygen Species (ROS) that lead to the DNA damage and depletion of cell antioxidant defense systems[20-21].

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