

Recovery of Alanine aminotransferase from Lead exposed freshwater fish, *Anabas testudineus* (Bloch, 1792)

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Abstract: Alanine aminotransferase is important amongst the several molecules available in the cells and proteins play an important role in the cellular process. In the present investigation, fish, *Anabas testudineus* treated with an equitoxic dose of 11 ppm of lead nitrate and lead acetate were scarified on 1, 4, 8, 12 and 15 days for recovery patterns in liver, muscle, kidney, gill and brain. Lead toxicated fishes recovered after 15 days which depends on the natural physical condition of the fish.

Keywords: *A.testudineus*, lead poisoning, Alanine aminotransferase, Recovery, proteins

1. Introduction

Proteins are important organic substances required by an organism in tissues building and repair. They are the most abundant chemical compounds of the organisms. They are versatile, complex and fragile macromolecules with high molecular weights. The modern industries are making use of various heavy metals such as iron, copper, nickel, platinum and lead. Chemical pollution threatens the living systems and aquatic environment. Some of these metals are biologically essential, but others like cadmium, lead and mercury are highly hazardous to aquatic biota and normally occur in low concentrations. It is known that common forms of lead poisoning results from mining, processing and commercial dissemination of lead (Hammond, 1969). The primary source of lead exposure to animals is contaminated soils, that remains on older structures, water from plumbing systems that contain lead, and lead based products, especially batteries and linoleum (Waldner et.al., 2002). A major source of lead to waterfowl and other wildlife is spent lead shot, bullets, cartridge, lead and sinkers used in sport fishing (De Francisco et al., 2003).

2. Material and Methods

Anabas testudineus selected as test species is a representative of Anabantoid fishes in South India. They are well known for their air breathing ability, and can survive out of water in moist air for six days. It is selected as the test animal because of its euryhaline and eurythermal nature, and unique position in food chain. They are quite sturdy and ideally suited for experimentation in laboratory for longer periods.

Biochemical assays were done in different tissues from both experimental and control fishes. Fish, approximately of same size and weights were grouped into 6 batches. 2 batch of fish served as controls, 2 exposed to lead nitrate and the remaining two exposed to lead acetate for a period of 15 days. After a period of 15 days of exposure, a fish from each batch were transferred to lead-free water and

scarified at the same intervals to observe the recovery. The values of different parameters were expressed as mean with standard error. Significance of the values obtained was tested using student 't' test. Alanine aminotransferase was estimated by Reitman and Frankel (1957) as described by Bergmeyer (1965).

3. Results and Discussion

The AAT activity was also found enhanced progressively throughout the exposure period in all the tissues exposed to lead nitrate and lead acetate. The percent enhancement in the activity was found to be tissue – specific and time – dependent. On the 1st day of exposure the highest enhancement in activity was found in the liver exposed to lead nitrate and in kidney exposed to lead acetate. The percent enhancement in liver was +4.70% for lead nitrate and +5.59% for lead acetate, $P < 0.001$, which is followed by kidney (+4.00% for lead nitrate $P < 0.05$ and +6.55% for lead acetate $P < 0.01$), gill (+3.61% for lead nitrate $P < 0.01$; +4.74% for lead acetate $P < 0.05$) and brain (+1.61%) for lead nitrate with insufficient variation over control, and +3.02% for lead acetate $P < 0.01$).

On the 4th day of exposure similar elevatory response was observed, with more percent variation in the tissues. Liver witnessed maximum enhancement (+12.5% for lead nitrate, +18.10% for lead acetate, $P < 0.001$) followed by kidney (+10.34% lead nitrate, +15.51% for lead acetate; $P < 0.001$), muscle (+10.47% for lead nitrate; +15.30% for lead acetate, $P < 0.001$), gill (+6.25% for lead nitrate; $P < 0.01$; +15.30% lead acetate $P < 0.001$) and brain (+6.60% for lead nitrate $P < 0.05$; +0.05; +9.48% for lead acetate $P < 0.01$).

On 8th day of exposure elevation ALAT activity was observed in all the tissues. The percent enhancement was significant at $P < 0.001$ level for all the tissues. The maximum elevation in activity was recorded in liver (+23.39% for lead nitrate, +27.27% for lead acetate $P < 0.001$) followed by gill (+21.81% for lead nitrate, +26.35% for lead acetate), kidney (+18.60% for

lead nitrate, +27.19% for lead acetate), muscle (17.52% for lead nitrate, +20.42% for lead acetate) and brain (+9.20% for lead nitrate, +13.29% for lead acetate).

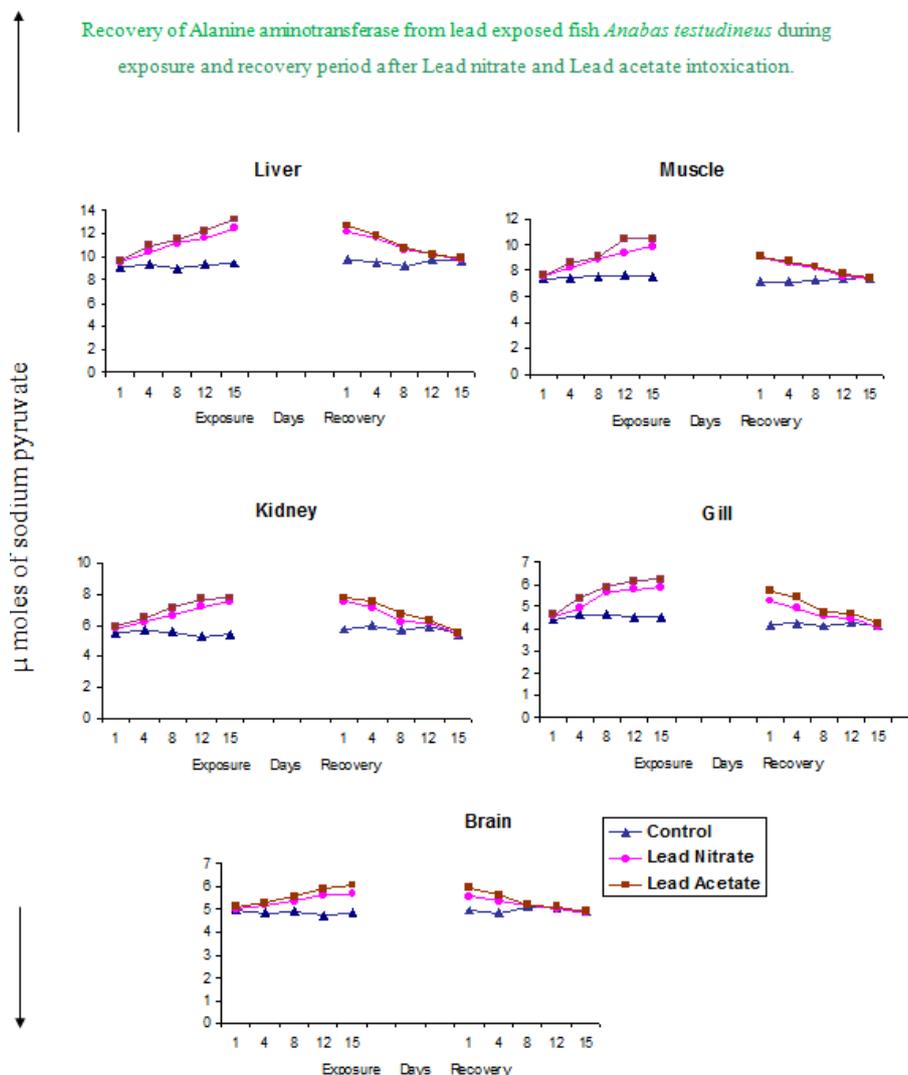
On 12th day of exposure elevation in ALAT activity was more when compared to preceding exposure periods. The maximum elevation was observed in kidney (+35.98 % for lead nitrate and +44.57 % for lead acetate) which is followed by gill (+28.06 % for lead nitrate, +36.53% for lead acetate), muscle (+22.83 % for lead nitrate, +34.38 % for lead acetate), liver (+24.15 % for lead nitrate, +30.88% for lead acetate) and brain (+18.53% for lead nitrate, +24.42% for lead acetate). Values of all the tissue were found significant at P < 0.001 and brain at P < 0.01.

On 15th day of exposure maximum elevation in activity was recorded in all the tissues when compared to preceding exposure periods. All the tissues exhibited the percent enhancement which was significant at P < 0.001. Maximum enhancement in activity was noticed in

kidney (+39.03% for lead nitrate, +41.97% for lead acetate) followed by liver (+31.22% for lead nitrate +39.87% for lead acetate), muscle (+31.69 for lead nitrate, 39.01% for lead acetate), gill (+30.31% for lead nitrate, +37.61% for lead acetate) and brain (+17.60 for lead nitrate, +25.67% for lead acetate).

During recovery period all the tissues witnessed a progressive drop in the enhanced ALAT activity. The recovery responses were found fast in the lead nitrate treated fish in comparison to lead acetate treated. The difference between the control and experimental values was gradually reduced during the recovery period in all the tissues and the differences appeared insignificant from 8th day onwards in brain, and rest of the tissues from 15th day onwards, however the differences appeared insignificant from 12th day onwards in muscle, kidney and gill exposed to lead nitrate (Fig.1).

Figure - 1



Alanine aminotransferase in the present study were found progressively elevated in all the tissues throughout the exposure period. The percent enhancement was more in the liver and kidney indicating the remarkable similarity of these enzymes with other parameters and this may be due

to more accumulation of lead in these tissues. Elevations in the activity of these enzymes indicate the utilization of amino acids. The similar observations were recorded in several animal species under heavy metal intoxication in Crabs (Reddy et al., 1982) in fish *Oreochromis niloticus*

(Almeida et.al.,2001);*Sparus aurata* (Vaglio and Landriscina, 1999). Similar observations were also recorded in mammalian models(Chougule Priti et. al.,2005;Kavitha,2010)and in some selected molluscs(David et.al.,2003; Satyaparameshwar et.al.,2006).The enhanced ALAT activities provides the oxaloacetic acid and pyruvate required for the gluconeogenic pathway to meet the additional supply of glucose for the production of energy under reduced factor of oxidative metabolism. In evidence to this the Krebs cycle enzymes in the present study were found inhibited. Heavy metal induced hypoxic condition in *Barytelphusa guerini* (Mali, 2009) offers an evidence to the compensatory role of ALAT under lead induced toxic manifestations. The activity of alanine amino transferases (ALAT), which serve as strategic links between protein and carbohydrate metabolisms, which is known to alter under several physiological and pathological conditions (Shivakumar,2005).

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