RECOVERY IN FREE AMINO ACID FROM LEAD TOXICATED FRESHWATER FISH, *ANABAS TESTUDINEUS* (BLOCH, 1792)

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Abstract

Free aminoacid 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, *A.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 physical condition of the fish.

Keywords: Free amino acid; lead; anabas

1. Introduction

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. 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. A major source of lead to waterfowl and other wildlife is spent lead shot, bullets, cartridge, lead and sinkers used in sport fishing.

2. Material and Methods

2.1 Material: *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.

2.2 Methods: 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 batches 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. Free amino acid was estimated by modified method of 7.

3. Results and Discussion

3.1 Results: Free aminoacids content was decreased progressively in all the tissues exposed to lead nitrate and lead acetate. The drop in the amino acid content was found more in the tissues of the fish intoxicated with lead acetate in comparison to lead nitrate intoxicated fishes. However, the differences in responses elicited by these two salts are not appreciable. The tissue-specific responses were observed during exposure.

On 1st day of exposure depletion in amino acid content was observed in all the tissues. Maximum amount of depletion was noticed in liver (-11.96% for lead nitrate, -17.03% for lead acetate P < 0.001) followed by kidney (-15.81% for lead nitrate; -6.52% for lead acetate P < 0.001), gill (-11.80% for lead nitrate; -15.19% for lead acetate P < 0.001), muscle (-9.45% for lead nitrate; -11.75% for lead acetate P < 0.001) and brain (-9.5% for lead nitrate; -10.86% for lead acetate). However, the values of the brains were found statistically insignificant.

On 4th day of exposure the percent depletion was more when compared to 1st day. The percent variation over control ranged between -14.54% to -22.96% for lead nitrate treated fish and -17.70% to -32.31% for lead acetate treated fish. Except for brain, all the other tissues exhibited depletory response, significant at P < 0.001.
On 8th day of exposure the percent depletion was increased than preceding exposure period. The percent depletion was significant P < 0.001 in all tissues. The maximum depletion was found in liver (-35.27% for lead nitrate; -30.15% for lead acetate) followed by gill (-25.66% for lead nitrate; -28.35% for lead acetate), brain (-22.35% for lead nitrate, -31.64% for lead acetate), muscle (-24.16% for lead nitrate, -26.98% for lead acetate) and kidney (-20.49% for lead nitrate, -23.15% lead acetate).

On 12th day of exposure tissue specific responses were observed. The percent depletion ranged from -24.58% to -40.52% for lead nitrate and -25.23% to -36.78% for lead acetate. Maximum depletion was observed in kidney (-40.52% for lead nitrate, -33.48% for lead acetate; P < 0.001). Followed by liver (-33.89% for lead nitrate; -36.78% for lead acetate; P < 0.001), brain (-36.76% for lead nitrate P < 0.001; -32.03% for lead acetate P < 0.01), muscle (-27.62% lead nitrate, -29.55% lead acetate P < 0.001) and muscle (-24.58% lead nitrate, -25.23% for lead acetate P < 0.001).

On 15th day of exposure maximum depletion was found in all the tissues when compared to preceding exposure periods. The percent depletion ranged between -29.09% to -39.29% for lead nitrate and -23.57% to -41.20% for lead acetate and the values were significant at P < 0.001 for liver, muscle, kidney and gill, and for brain at P < 0.01. The depletion in free aminoaicids were gradually reduced to normal levels during the recovery period. Recovery in free aminoacid content was found to be time dependent and tissue specific. Brain exhibited maximum recovery from the 8th day of recovery period, and gills from 12th day onwards by exhibiting statistically insignificant differences between control and experimental values. The liver, muscle and kidney witnessed the maximum recovery on 15th day of recovery period (Fig.1)

3.2 Discussion: Free aminoaicids were found depleted progressively in all the tissues throughout the exposure period indicating maximum utilization of these molecules under lead induced toxic manifestations. The drop in free aminoacid content also suggests an imbalance between proteolysis and amino acid catabolism. Drop in the aminoacid suggests their rapid utilization in the transamination and oxidative deamination, particularly to generate the amphiblastic intermediates to krebs cycle or gluconogenic pathway. As evidence to this the inhibition of Krebs cycle enzymes was observed in the tissues of the same fish after lead intoxication. The tissue-specific and time-dependent responses in free aminoacid content exhibits a striking similarity with that of proteins. The similarity in responses is due to the differential accumulation of lead salts and also on the factors responsible for accumulation in the tissues. Decrease in free aminoacid content was recorded after heavy metal intoxication in crab, Barytelphusa guerini Catla. Similar responses were also recorded in mussels after chemical stress. The loss in the aminoacids could also be attributed to its loss in Urine, (aminaciduria). Aminoaicid catabolism brought about by a series of specific amino transferases, the pyridoxal phosphate dependent enzymes. These enzymes catalyses transfer of amino group from amino acid to α-Ketoacids without the liberation of ammonia. The ketoacids formed in this reaction serve as amphibolic intermediates which may enroute into the krebs cycle for lipogenesis. The α-Ketoacids converted into amino acids in this reaction may be utilized in the protein synthesis. Therefore the reaction plays an important role in the regulation of protein and carbohydrate metabolism. Any kind of fluctuations in transamination reaction; disturb the nitrogen balance which in turn may disturb urea cycle, gluconeogenic process and fat metabolism.

Figure – 1 Recovery in free Amino acid from lead toxicated freshwater fish Anabas testudineus
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