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RESEARCH ARTICLE

CARBOHYDRATE METABOLIC PATHWAY ENZYMES RECOVERY FROM HEAVY METAL DOSE FRESHWATER FISH, *ANABAS TESTUDINEUS* (BLOCH, 1792)

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Abstract

Glycogen phosphorylase a & ab is important amongst the several molecules available in the cells and carbohydrates plays 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.

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 (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, leads and sinkers used in sport fishing (De Francisco et al., 2003) .

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 weight 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 of glycogen phosphorylase. The values of different parameters were expressed as mean with standard error. Significance of the values obtained was tested using student ‘t’ test . Glycogen phosphorylase activity was assayed by the method of Cori et al., (1955).

RESULTS AND DISCUSSION

1) Glycogen Phosphorylase 'a':

The activity of glycogen phosphorylase 'a' was found elevated throughout the exposure period and maximum increase in activity was observed on 15th day of exposure. Liver and muscle exhibited maximum elevation, in comparison to other tissues. The activity patterns of glycogen phosphorylase 'a' are clearly reflected in the levels of glycogen. The activity of this enzyme was tissue-specific and time-dependent.

On 1st day of exposure maximum activity was recorded in kidney (+7.97% for lead nitrate, +7.25% for lead acetate, $P < 0.05$), followed by muscle (+3.92% for lead nitrate and +6.45% for lead acetate, $P < 0.01$ and $P < 0.001$ respectively and liver (+3.35% for lead nitrate $P < 0.05$ and +7.48% for lead acetate $P < 0.001$). Gill and brain exhibited insignificant rise in phosphorylase 'a' activity.

On 4th day of exposure maximum activity was recorded in gill (+13.64% for lead nitrate, +16.23% for lead acetate; $P < 0.05$) followed by liver (+11.90% for lead nitrate, +14.59 for lead acetate $P < 0.001$), kidney (+8.23% for lead nitrate, $P < 0.05$ and +17.35% for lead acetate $P < 0.001$), brain (+11.59% for lead nitrate, $P < 0.01$; +16.91% for lead acetate $P < 0.001$), and muscle (+8.07% for lead nitrate +11.04% for lead acetate, $P < 0.01$).

On 8th day of exposure maximum elevation was found in liver (+25.53% for lead nitrate, +24.38% for lead acetate $P < 0.001$) followed by kidney (+22.53% for lead nitrate, +29.94% for lead acetate, $P < 0.001$), muscle (+21.75% for lead nitrate, +23.58% for lead acetate; $P < 0.001$); gill (24.14% for lead nitrate, +22.41% lead acetate $P < 0.05$) and brain (+14.91% for lead nitrate $P < 0.01$; +27.63% acetate $P < 0.001$).

On 12th day of exposure kidney exhibited more enhancement in the activity of phosphorylase 'a' (+35.02% for lead nitrate, +43.43% for lead acetate $P < 0.001$) and brain exhibited minimum enhancement (+18.67% for lead nitrate, +33.61% for lead acetate, $P < 0.001$). The percent enhancement ranged between + 18.67% to +39.53% for lead nitrate and +30.23% to +43.43% for lead acetate. In the remaining tissues also the percentage variation was significant at $P < 0.001$.

On 15th day of exposure maximum enhancement was observed in all the tissues and percent enhancement over control was statistically significant at $P < 0.001$ in all the tissues. Liver and muscle exhibited maximum enhancement, followed by Kidney, Gill and Brain. The present enhancement ranged from +31.16% to 42.78% for lead acetate.

On transferring fish to toxicant free water the present enhancement in glycogen phosphorylase 'a' was gradually reduced in all the tissues. The values between experimental and control exhibited statistically insignificant variation indicating the maximum recoveries in the tissues. The recoveries were found more in the lead nitrate intoxicated fish in comparison to lead acetate intoxicated fish. tissues. Early recovery was witnessed in the brain on 8th day of recovery period in the lead nitrate intoxicated fishes, and on 12th day in lead acetate intoxicated fishes (Fig.1).

2) Glycogen Phosporylase "ab" :

The total glycogen phosphorylase activity was found enhanced throughout the exposure period in all the tissues. The enhancement was tissue-specific and time-dependent. However maximum activity was noticed on 15th day of exposure period.

On 1st day of exposure maximum enhancement was witnessed in liver (+6.06% for lead nitrate, + 6.53% for lead acetate $P < 0.001$) followed by muscle (+4.27% for lead nitrate., +6.28% for lead acetate; $P < 0.01$ and $P <$

0.001 respectively) and kidney (+4.27% for lead nitrate, +5.19% for lead acetate, $P < 0.01$) gill and brain exhibited an insignificant elevation over controls.

On 4th day of exposure significant elevation was noticed in all the tissues ($P < 0.001$). The percent enhancement ranged from +9.09 to +13.45% for lead nitrate and +11.78 to +17.39% for lead acetate. Maximum activity was witnessed in muscle (+13.45% for lead nitrate, +17.39% for lead acetate) follwed by liver (+13.21% for lead nitrate, +14.18% for lead acetate), kidney (+10.80% for lead nitrate, +11.78% for lead acetate), brain (+10.25%) for lead nitrate and + 12.53% for lead acetate) and gill (+9.09% for lead nitrate, +13.90% for lead acetate).

On 8th day of exposure the elevation in activity was significant at $P < 0.001$ in all the tissues. Maximum activity was observed in liver (+28.75% for lead nitrate, +29.98% for lead acetate). Minimum enhancement was noticed in gill (+14.48% lead nitrate, +17.27% lead acetate). The percent enhancement ranged between +14.48% to +28.75% for lead nitrate and +17.27% to +29.98% for lead acetate.

On the 12th day of exposure all the tissues recorded significant enhancement in total phosphorylase activity at $P < 0.001$. The magnitude of response was more in lead acetate in comparison to lead nitrate. The highest amount of activity was recorded in the liver (+34.75% for lead nitrate +40.98% for lead acetate) followed by kidney (+30.18% for lead nitrate, +28.25% for lead acetate), brain (+23.05% for lead nitrate, +24.28% lead acetate) and gill (+18.59% for lead nitrate, +23.37% for lead acetate).

On the 15th day of exposure maximum enhancement was observed over all exposure periods. Liver witnessed high enhancement (+40.34% for lead nitrate, +42.74% for lead acetate), followed by kidney (+37.35% lead nitrate, +41.83% lead acetate) , muscle (+33.84% lead nitrate +39.50% lead acetate) brain (+35.20% lead nitrate, +37.36% lead acetate) and gill (+35.20% for lead nitrate and +37.36% lead acetate). The present enhancement was significant at $P < 0.001$ in al the tissues.

During recovery period all the tissues recovered progressively. Brain and Gill recovered rapidly than muscle, kidney and liver. At the end of 15th day the difference between control and experimental values were statistically insignificant indicating recovery of this enzyme in all the tissues. However the brain and gill exhibited an early recovery in comparison to other tissue (Fig.1 & 2).

The activity patterns of glycogen phosphorylase a & ab in all the tissues are clearly reflected in the glycogen levels throughout the exposure. The maximum enhancement in the activity of glycogen phosphorylase with the consequential depletion in the glycogen levels in the liver and kidney, indicate the hepatotoxic and nephrotoxic nature of lead ions.

The differences between active and total phosphorylases could be attributed to the variation in the a/ab ratios during toxic manifestations. Enhancement in the glycogen pohosphorylase activity in the tissues of the present model was in agreement with the observations recorded in prawn (Sujay Kumar et.al, 2001) in fishes, *Anabas Scandens* (Sadath 1990) . The activity patterns of glycogen phosphorylase 'a' and 'ab' in all the tissues are clearly reflected in the glycogen levels throughout the exposure period .

(Mali et.al.,2010) Studied cadmium induced alterations in the glycolytic potential of freshwater female crab, *Barytelphusa guerini.*

The maximum enhancement in the activity of phosphorylase with consequential depletion in the glycogen levels in the liver and kidney indicate the hepatotoxic and nephrotoxic nature of lead. Another possible reason for glycogen depletion may be due to impairment of aerobic respiration .Under such conditions energy is derived from anaerobic respiration resulting in rapid glycogen utilization leading to its depletion. Arrival of anoxic or hypoxic conditions in the fish during lead toxicity may also be one of the factors responsible for the observed glycogen depletion. Impairment of hormonal release and accumulation of biogenic amines may also cause the glycogen depletion in the tissues.

Figure - 1
Activity of Glycogen Phosphorylase "a" in the tissues of *Anabas testudineus* during exposed and recovery days after Lead intoxication.

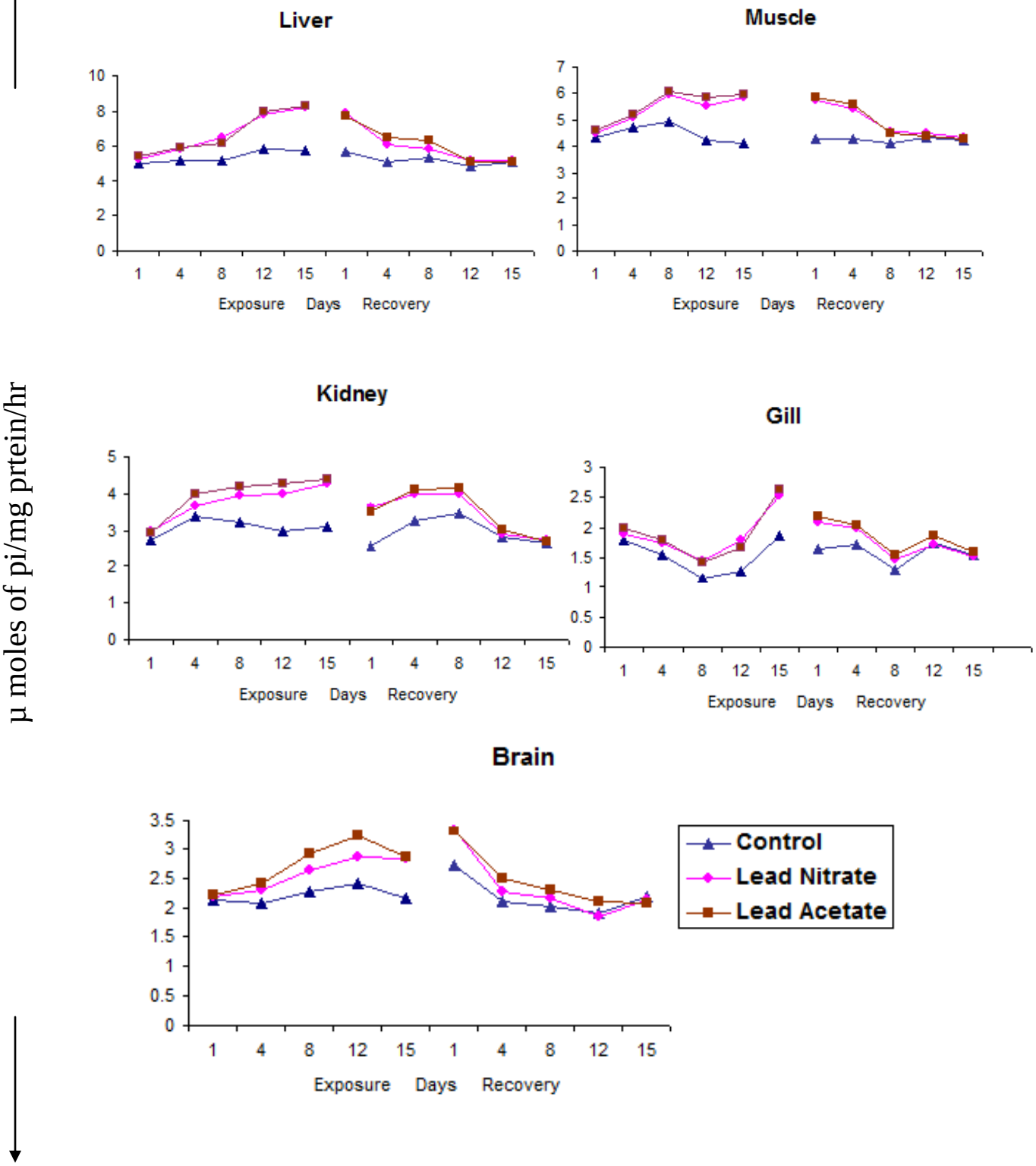
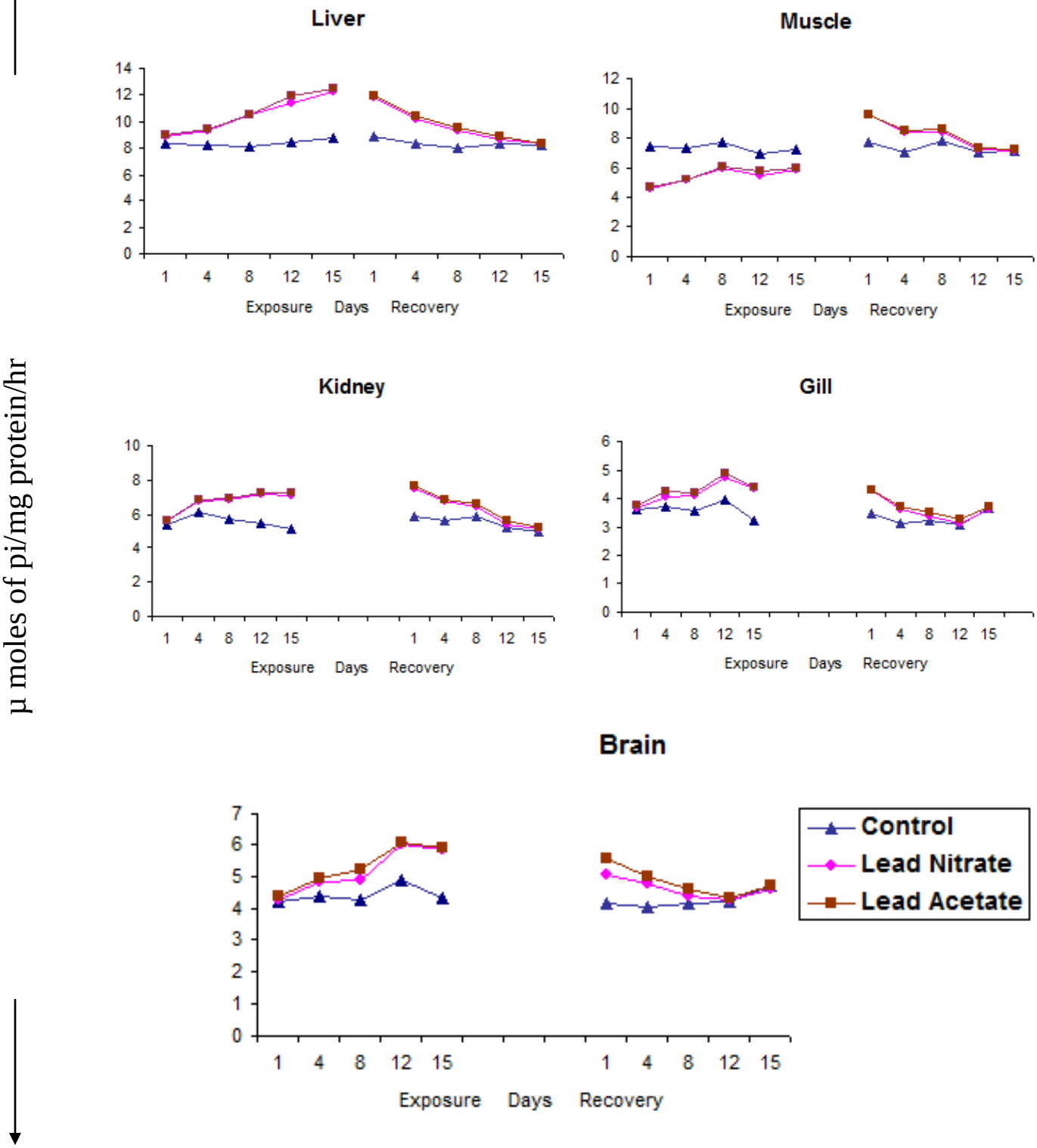


Figure - 2

Activity of Glycogen Phosphorylase "ab" in the tissues of *Anabas testudineus* during exposure and recovery period after Lead nitrate and Lead acetate intoxication.



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