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RESEARCH ARTICLE

Pyruvic acid content recovery from metal dose freshwater fish, *Anabas testudineus* (Bloch, 1792)

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Abstract

Pyruvic acid 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, *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.

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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, lead 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. The values of different parameters were expressed as mean with standard error. Significance of the values obtained was tested using student 't' test . Pyruvate was estimated by the method of Friedman and Hangen (1942).

RESULTS AND DISCUSSION

Pyruvic acid levels were found decreased in the initial exposure periods, later on there was an enhancement in the pyruvate content. Almost all tissues exhibited similar response to lead toxicity.

On 1st day of exposure the pyruvate content was found significantly decreased in all the tissue at $P < 0.01$ and < 0.05. Maximum depletion was found in kidney (-17.44% for lead nitrate, -18.60% for lead acetate) followed by brain (-15.09% for lead nitrate, -16.98% for lead acetate), liver (-15.49% for lead nitrate, -14.79% for lead acetate), muscle (-12.5% for

lead nitrate, -16.67% for lead acetate), and gill (-10.42% for lead nitrate, -12.5% for lead acetate).

On the 4th day of exposure a further depletion in pyruvate content was recorded in all the tissues. The values were found statistically significant at ($P < 0.001$, $P < 0.01$ and $P < 0.05$). The maximum depletion was recorded in kidney followed by brain, liver, gill and muscle. The depletion ranges from -14.67 to -20.73% for lead nitrate and -17.33% to -23.17% for lead acetate.

On the 8th day of exposure an exactly opposite response was witnessed in comparision to its early exposure periods. Accumulatory values were found significant at $P < 0.001$; $P < 0.01$; $P < 0.05$. The accumulation was found to be tissue-specific and in general the accumulation was more in the tissues of fish intoxicated with lead acetate. The order of accumulation for lead nitrate : Brain < gill < muscle < kidney < liver and for lead acetate brain < liver < muscle < gill < kidney.

On 12th day of exposure maximum enhancement in pyruvate content was noticed in kidney (+23.33% for lead nitrate, +26.67% for lead acetate $P < 0.001$) followed by liver (+22.29% for lead nitrate, +24.00% for lead acetate $P < 0.001$); muscle (+22.06% for lead nitrate $P < 0.001$; +23.53% for lead acetate $P < 0.01$); brain (+20.83% for lead nitrate $P < 0.05$; +22.92% lead acetate $P < 0.01$); and gill (+18.00% for lead nitrate, +22.00% for lead acetate $P < 0.05$).

On 15th day of exposure enhancement in the pyruvate was found increased in all the tissues. Kidney accumulated more pyruvate (+31.96% lead nitrate +34.02% lead acetate $P < 0.001$) followed by liver (+30.41% lead nitrate, +33.78% lead acetate, $P < 0.001$). Muscle (+29.38% lead nitrate, +32.31% lead acetate, $P < 0.01$) Gill (+26.09% lead nitrate, +28.26% lead acetate, $P ,< 0.05$) and Brain (+26.92% lead nitrate $P < 0.05$, +30.77% lead acetate $P < 0.01$).

During the recovery period, all the tissues recovered, and the recovery was gradual and progressive. Brain pyruvate levels came down to normal level within 8 days after transferring to normal water (+1.75% lead nitrate, -1.75% lead acetate) the difference between control and experimental values were found insignificant indicating the recovery in all the tissues. Muscle and gill recovered after 12 days of transfer to fresh water. Liver and Kidney recovered the pyruvate content on 15th day by exhibiting insignificant percent variation(Fig.1., a,b,c,d,e).

The pyruvate levels exhibited a tissue specific and time-dependent changes in the tissues. The pyruvate content was found depleted upto 4th day of exposure in all the tissues, however, an enhancement in the pyruvate levels were recorded from 8th day onwards. The depletion of pyruvate suggests its utilization during early stages of toxic manifestation, while the accumulation suggests an impairment of its utilization. The maximum responses in liver and kidney indicates the hepatotoxic and nephrotoxic nature of the lead ions.

The magnitude of responses was found more in the organic form of lead comparison to the inorganic form. The differences in the responses between these two lead forms may be due to the differences in the rate of absorption, elimination and retention of these two salts of lead in the tissues. The tissues-specific variation in responses could be attributed to concentration factors of the lead in the tissues.

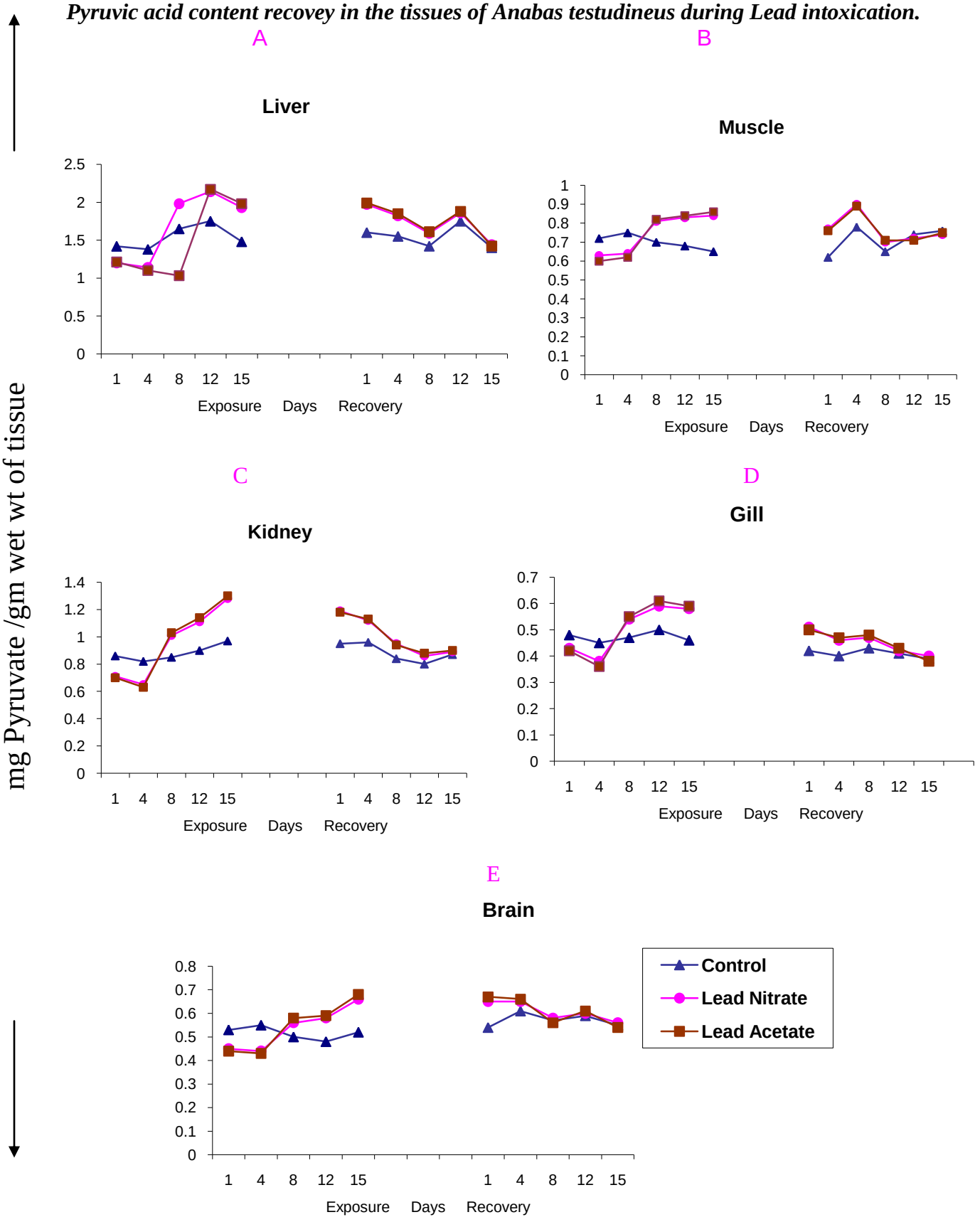
The decrease in the pyruvate content during early stages of exposure could be attributed to its oxidative decarboxylation (Tokaski et al., 1978) by pyruvate dehydrogenase (PDH) to yield an acetyl CoA essential for the commencement of the krebs cycle. Pyruvic acid is important metabolite in the metabolic pathway of carbohydrate. It is convertible in to lactic acid or acetyl Co A depending upon the absence or presence of molecular oxygen in the tissues and also NAD(Raj kumar et.al, 2008) .Decrease in pyruvate level in the organs of mussel and fish exposed to cadmium could be due to the speedy reduction of pyruvate to lactate(Venkata chandrudu et.al,2008).

An increase in the PDH activity in muscle of *Channa punctatus* during chromium toxicity (Sastry & Sunitha, 1982a) lends support for the depletion of pyruvate content. Accumulation of pyruvic acid in the tissues during the subsequent exposures to lead may be due to the impairment of PDH. Inpairment of PDH in tissues of *Channa punctatus* during chronic exposure to chromium (Sastry & Sunitha, 1983b) suggests that, the duration of exposure plays an important role in the pyruvate accumulation in the tissues.

The pyruvate utilization depends on the aerobic and anaerobic state of an animal. Under aerobic situations the pyruvate may be oxidized by PDH to acetyl CoA to meet the excess of energy demands posed by toxic manifestations of lead. The accumulation of pyruvate during the later stages of exposure suggests the arrival of anoxic or hypoxic conditions in the fishes due to the less availability of oxygen.

Figure - 1

Pyruvic acid content recovey in the tissues of Anabas testudineus during Lead intoxication.



The depletion in the pyruvate levels could also be attributed to its utilization in the biosynthesis of amino acid like alanine , through transamination reactio. The studies on glutamate pyruvic transaminase would present a correct picture of pyruvate utilization. Further, the chances of pyruvate utilization in the gluconeogenic pathway through oxaloacetate to provide excess of glucose during metal induced anaerobic situations cannot be ruled out in the present study.

Studies on the pyruvate carboxylase would confirm the pyruvate utilization in gluconeogenisis. Accumulation of pyruvate levels may be due to decrease oxidative decarboxylation of the pyruvate. (Tokarski et al., 1978) or may be due to the transamination of alanine amino acid. In evidence to this ALAT activity in all the tissues of the present study was found elevated. The level of pyruvic acid was found to be depleted after exposure to phosalone toxicity in the tissue of freshwater fish *Channa punctatus* (Raj kumar et.al, 2008).

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