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Glucose post exposure recovery from lead intoxicated freshwater fish *Anabas testudineus*

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Abstract : *Glucose are important amongst the several molecules available in the cells, Carbohydrates play an important role in the cellular process Under extreme stress conditions, carbohydrate metabolite such as glucose have been known to act as the energy supplier in metabolic pathways and biochemical reactions. In the present investigation fish treated with an equitoxic dose of 10 ppm of lead nitrate and lead acetate intoxicated fish After a period of 15 days of exposure a batch from lead nitrate exposed fish and a batch from lead acetate exposed fish were transferred to lead-freewater. Fishes were scarified on 1, 4, 8, 12 and 15 days for the analysis of recovery pattern in tissues viz. liver, muscle, kidney, gill and brain .It is found that lead toxicated fishes were recovered after 15 days depends upon physical condition of fish.*

Key Words : Carbohydrate, Lead, Anabas

Introduction

Now a days industrialization is increasing rapidly in our country. The modern industries are making use of various heavy metals such as iron, steel, copper, nickel, platinum and lead. Among the different types of pollutions, chemical pollution appears to be the major type which threatens the living systems very extensively. Among the different habitats aquatic environment is the major target of pollution. Most of the heavy metals are natural constituents of the aquatic environment. Some of them are biologically essential, but some metals like cadmium, lead and mercury are highly hazardous to aquatic biota and normally occur in low concentration (Mali, 2002).It is clearly known the common forms of lead poisoning result from the mining, processing and commercial dissemination of lead (Hammond, 1969).The primary source of lead exposure to animals are contaminated soils, lead paints that remain on older structures, water from plumbing systems that contain lead, and lead based products, especially batteries, used crankcase oil, and linoleum (Waldner *et al.*, 2002). The lead containing gasoline fumes from automobile exhausts constitute the chief and wide spread source of lead contamination in urban environments. A major source of lead to waterfowl and other wildlife is spent lead shot, bullets, cartridge, and lead sinkers used in sport fishing (De francisco *et al.*, 2003)

Materials and Methods

Anabas testudineus which is selected as test species in the typical representative of Anabantoid fishes in South India. It is fresh water, euryhaline and eurythermal teleost. Biochemical assays were made in different tissues from both experimental (exposed to toxicant) and Normal (toxicant free) fishes. Fish approximately of same size and weight were selected and grouped into 6 batches. 2 batch of fish served as controls, 2 batches of fish were exposed to lead nitrate and the remaining two batches were exposed to lead acetate for a period of 15 days. After a period of 15 days

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of exposure a batch from lead nitrate exposed fish and a batch from lead acetate exposed fish were transferred to lead-free water and scarified at the same intervals to observe the recovery responses. In all the experiments, a minimum of six individual observations were made. The values of different parameters were expressed as mean with their standard error. Significance of the values obtained were tested using student 't' test. The glycogen and glucose content in the tissues were estimated by the method of Nicholas *et al.*, (1956).

Results and Discussion

The glucose content was reduced progressively throughout the exposure span indicting its utilization during lead toxicity. The depletion of glucose was time-dependent and tissue-specific. After 24 hours of exposure, maximum amount of reduction was found in liver (-9.48%), $P < 0.001$for lead nitrate and -39.53%, $P< 0.001$ for lead acetate) followed by kidney (-33.95% for lead nitrate and -37.65% for lead acetate both significant at $P < 0.05$), gill (-34.38% for lead nitrate, -35.82% for lead acetate, both arte significant at $P < 0.001$), muscle (-26.56 % for lead nitrate and -29.19 % for lead acetate significant at $P < 0.001$) and brain (9.86% for lead nitrate and -14.55% for lead acetate, the values are statistically insignificant).

After 4th day of exposure the depletion was statistically significant in all the tissues at ($P < 0.001$; $P < 0.05$). Maximum amount of reduction was recorded in the liver (-50.12% for lead nitrate and -51.61% for lead acetate) followed by kidney (-49.28% lead nitrate and -51.01% for lead acetate) gill (-44.55% lead nitrate, -45.51% for lead acetate) muscle (-34.25% for lead nitrate, -35.80% for lead acetate) and brain (-16.09% lead nitrate, -21.46% for lead acetate).

For 8th day of exposure all the tissues except gill exhibited a reduction statistically significant at $P < 0.001$). Maximum amount of depletion was recorded in kidney (-56.45% for lead nitrate, - 52.96% for lead acetate) followed by liver (-50.08% for lead nitrate, 48.74% for lead acetate), gill (-49.47% for lead nitrate and -49.73% for lead acetate), muscle (-44.26% for lead nitrate and -49.59% for lead acetate) and brain (-25.48% for lead nitrate, -28.85% for lead acetate).

On 12th day of exposure the depletory response was significant at ($P < 0.01$ and $P < 0.001$). Maximum depletion was found in liver (-59.79% for lead nitrate, -62.08% lead acetate) followed by gill (-59.80% for lead nitrate, -60.80% for lead acetate), muscle (-52.67 for lead nitrate, -51.56% for lead acetate), kidney (-48.22% for lead nitrate, -52.66 for lead acetate) and brain (-30.52% for lead nitrate, -29.72% for lead acetate).

On 15th day of exposure glucose content was found depleted maximum in all the tissues when compared to preceeding exposure periods. Maximum depletion was found in kidney (-9.89% lead nitrate, significant at $P < 0.01$, and -61.93% for lead acetate and significant at $P < 0.001$) followed by liver (-59.74% for lead nitrate and -60.47% for lead acetate, significant at $P < 0.001$), gill (-58.73 % for lead nitrate,-60.32% for lead acetate significant at $P < 0.01$) ,muscle (-51.53% for lead nitrate; -52.81% for lead acetate), and brain (-37.41% for lead nitrate and -40.48% for lead acetate).

During recovery period, depletion in glucose content was gradually reduced in all the tissues maximum recovery was achieved on 15th day of recovery period. The percent reduction over controls were statistically insignificant at the end of the 15th day for liver, muscle, kidney and gill while in the brain % difference between control and experimental were statistically insignificant on 12th day of recovery period (Table and Fig.1).

In the present investigation the tissue glucose level was found depleted throughout the exposure period. The depletion was progressive

Table 1. Glucose content in the tissues of *Anabas testudineus* during exposure and recovery periods after lead nitrate and lead acetate intoxication

Tissues		Exposure Period (In days)					Recovery period (in days)				
		1	4	8	12	15	1	4	8	12	15
Liver	C	22.39±0.15	24.90±0.42	26.24±0.24	25.82±0.39	23.40±0.89	23.75±0.66	21.52±0.49	23.90±0.72	20.92±0.35	24.35±0.75
	A	15.79±0.27*	12.42±0.50*	13.10±0.35*	10.38±0.78	9.42±0.75*	10.79±0.72*	12.60±0.82*	15.31±0.49	17.75±0.25*	23.14±0.39NS
	%V	-29.48	-50.12	-50.08	-59.79	-59.74	+54.57	-41.45	-35.94	-15.15	-4.97
	B	13.54±0.55*	12.05±0.57*	13.45±0.57*	9.79±0.52*	9.25±0.72*	10.34±0.45*	12.02±0.23*	16.49±0.49*	17.15±0.49*	22.92±0.17NS
	%V	-39.53	-51.61	-48.74	-60.08	-62.47	-56.46	-44.14	-35.19	-18.02	-5.87
Muscle	C	8.63±0.021	7.29±0.20	8.45±0.27	8.98±0.72	7.84±0.95	7.05±0.021	8.32±0.15	6.98±0.04	6.94±0.09	7.39±0.79
	A	6.14±0.08*	4.72±0.45*	4.71±0.41*	4.25±0.84**	3.80±0.47**	3.61±0.25*	5.03±0.22*	4.94±0.18*	5.94±0.06*	7.21±0.85NS
	%V	-26.56	-35.25	-44.26	-52.67	-51.53	-48.79	-39.54	-29.23	-14.41	-2.43
	B	5.92±0.15*	4.68±0.92***	4.26±0.56*	4.35±0.85**	3.70±0.7**	3.57±0.24*	5.00±0.52*	4.98±0.15*	5.90±0.04*	7.14±0.75NS
	%V	-29.19	-35.80	-49.59	-51.66	-52.81	-49.36	-39.90	-28.65	-14.99	-3.38
Kidney	C	3.24±0.23	3.45±0.19	3.72±0.08	3.38±0.11	3.94±0.32	3.17±0.23	2.95±0.26	2.89±0.11	3.64±0.044	3.51±0.45
	A	2.14±0.36***	1.75±0.14*	1.62±0.06*	1.75±0.09*	1.58±0.48**	1.45±0.22*	1.62±0.33**	1.85±0.04*	3.05±0.069	3.28±0.75NS
	%V	-33.95	-49.28	-56.45	-48.22	-59.89	-54.27	-45.08	-35.99	-16.21	-6.55
	B	2.02±0.32***	1.69±0.29*	1.75±0.14*	1.62±0.12*	1.50±0.25*	1.39±0.29*	1.67±0.12**	1.82±0.05*	2.99±0.081*	3.25±0.38NS
	%V	-37.65	-51.01	-52.96	-52.66	-61.93	-56.15	-43.39	-37.02	-17.86	-7.41
Gil	C	3.49±0.037	3.12±0.045	3.74±0.38	3.98±0.43	3.15±0.33	3.28±0.3	2.85±0.09	2.63±0.045	2.74±0.075	2.98±0.09
	A	2.29±0.045*	1.73±0.027	1.89±0.42***	1.60±0.40**	1.30±0.2*	1.52±0.29**	1.84±0.08*	2.02±0.024*	2.35±0.052**	2.90±0.014NS
	%V	-34.38	-44.55	-49.47	-59.80	-58.73	-53.66	-35.44	-23.19	-14.23	-2.68
	B	2.24±0.045	1.70±0.039*	1.88±0.15*	1.56±0.36**	1.25±0.14*	1.49±0.21*	1.78±0.0.*	2.05±0.075*	2.32±0.039*	2.87±0.07NS
	%V	-35.82	-45.51	-49.73	-60.80	-60.32	-54.57	-37.54	-22.05	-15.33	-3.69
Brain	C	2.13±0.439	2.05±0.11	2.08±0.037	2.49±0.034	2.94±0.039	2.75±0.022	2.15±0.022	2.43±0.071	2.85±0.073	2.71±0.11
	A	1.92±0.75NS	1.72±0.05***	1.55±0.052*	1.73±0.025*	1.84±0.018*	1.9±0.05	1.84±0.04*	2.22±0.045NS	2.69±0.045NS	2.69±0.14NS
	%V	-9.86	-16.09	-25.48	-30.52	-37.41	-30.91	-14.42	-8.64	-5.61	-0.74
	B	1.82±0.3 NS	1.61±0.06**	1.48±0.045*	1.75±0.046*	1.75±0.05*	1.87±0.05*	1.81±0.042*	2.19±0.061***	2.70±0.048NS	2.67±0.14NS
	%V	-14.55	-21.46	-28.85	-29.72	-40.48	-32.00	-15.81	-9.88	-5.26	-1.48

Values are mean ± S.E. of six observations and are expressed as mg glucose / gm wet weight of tissues. C= control, A= lead nitrate, B = Lead acetate, %V= percent variation from control. Values are significant at *P < 0.001, **P < 0.01, ***P < 0.05, NS = not significant.

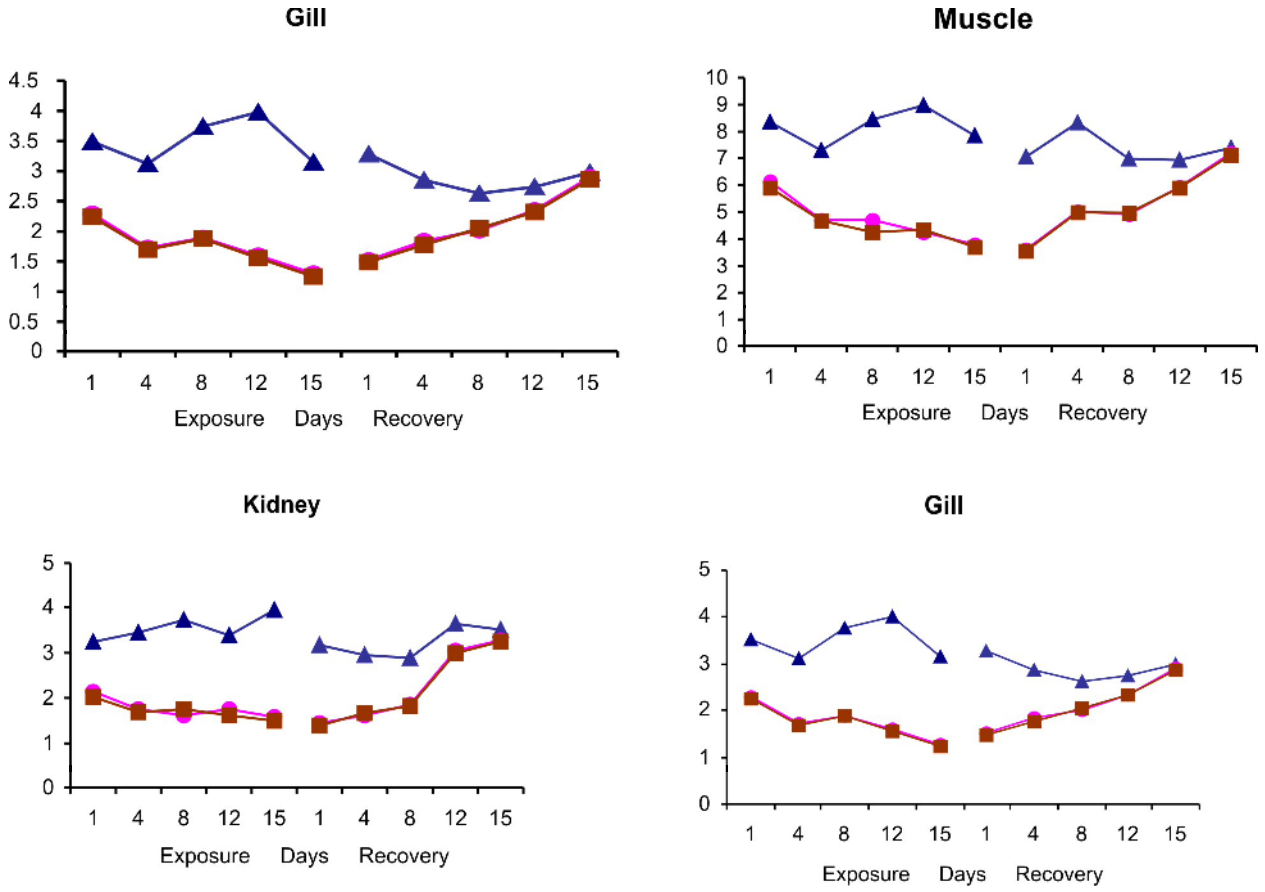

Fig 1. Glucose content in the tissues of *Anabas testudineus* during exposure and recovery period after Lead nitrate and Lead acetate intoxication

in all the tissues during entire exposure period, indicating persistent and cumulative inhibitory effects of lead (Harrison *et al*., 1971). The magnitude of depletion was more in fish treated with lead acetate. The inorganic lead i.e. lead nitrate is not greatly soluble in cell membranes and owing to its reduced mobility, accumulates in lesser amounts when compared to organometallic form, and therefore produces less toxic effects. The investigations of Hodson (1979) and Bondy (1984) also reveal that organic lead is more toxic than inorganic form. Tissue-specific depletion of glucose as observed in the present study may be due to its rapid utilization to meet the energy demands under toxic manifestations. The tissues-specific responses may be due to differential accumulation and movement of lead in tissues which depends on various factors like age, temperature, perfusion, vascularity and residual blood volume (Villarreal Trevino and Vilegas-Navorro 1987). Some observations revealed that liver and kidney retain more amount of lead incomparision to heart muscle and brain (Olojo *et al*., 2005; Gbem *et al*., 2001; Sulthana and Rajan, 2007). Hence it can be speculated that excess retention of this metal in these organs was responsible for pronounced decrease in the glucose and glycogen content. Another possible reason for the observed depletion of glucose content be due to imbalanced release of hormones as observed in some fish models under heavy metal intoxication (Mathan *et al*., 2009).

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