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ANTIDOTAL RECOVERY IN CARBOHYDRATE METABOLISM OF MERCURY INTOXICATED MICE, *MUS MUSCULUS* (LINN.)

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Penicillamine is also one of the antidote for heavy metal poisoning. The sub-lethal dose of mercuric chloride on carbohydrate metabolism in mice results in an initial increase and concomitant decrease of glycogen content in brain and liver tissues respectively. The glucose content shows decreased level in all tissues (brain, liver, muscle and kidney) to meet extra energy demand due to mercury toxicity. Antidote shows a remarkable recovery on carbohydrate metabolism in mercury toxicated mice.

INTRODUCTION

Nature, nowadays, facing a serious problem of environmental pollution. Environment pollution may be defined as the unfavourable alteration of our surroundings, wholly or largely as a byproduct of man's actions, through direct or indirect effects of changes in energy patterns, radiation levels, chemical and physical constitutions and abundance of organisms (Lu, 1996). These changes may effect human beings directly or indirectly through various routes (drinking water, consumption of food etc.). Heavy metals have a unique property of accumulation over a period of time, along a food chain and a very high level can be accumulated in an organism from very low concentration in water and sediments (Abbasi & Soni, 1986; Bose *et al.*, 1994).

The present study has been designed to find out the changes in carbohydrate constituents (glycogen and glucose) in sub-lethal dose of mercuric chloride exposure on mouse, *Mus musculus*. Another major objective of the present study has been to identify the with drawl effect of toxicant on carbohydrate constituents in various tissues through penicillamine treatment on mercury intoxicated mice with the aim to look into the possibilities of functional recoveries of the tissues.

MATERIALS AND METHODS

One hundred and eight laboratory bred white mice, *Mus musculus* (Linn.), 45 days old and weighing 25 ± 0.50g were divided at random into three groups (A, B and C), each of 36 mice. Each six mice were housed separately in a suitable cage, fed on a standard laboratory diet, supplied by Hindustan Lever Ltd., Mumbai and tap water *ad libitum*. Mice of group B and C were oral dosed on mercuric chloride at sub-lethal dose (0.1 g/kg body weight of the animal) on every day for 30 days. After 30 days, based on Klaasen *et al.* (1986) observations, the animal of group C were orally administered with penicillamine at the dosage of 5 mg/kg body weight respectively on each day for another 30 days. The mice of group A enjoyed the laboratory diet alone and tap water *ad libitum*. Total weight of the diet was kept constant throughout the experiment. After the scheduled treatment brain, liver muscle and kidney tissues were used for assay for determining the level of glycogen and glucose content (Kemp & Kits Van Heijhingen, 1954). The student's 't' test (Fisher, 1950) was used to calculate the statistical significance between control and experimental values.

RESULTS AND DISCUSSION

All cells and tissues require energy and almost all cells have carbohydrate to some degree. Some organs like brain, liver, muscle and kidney use carbohydrate in a different characteristic way. In the present study, an initial increase and concomitant decrease in the level of glycogen has been noticed in the brain and liver tissues and it suggests that it might be due to the conversion of glucose to glycogen initiated by mercuric chloride.

The disturbance in glycogen profile was considered as one of the most outstanding biological lesions due to action of heavy metal (De Bruin, 1976). The decrease in glycogen content in muscle and kidney may be due to glucose utilization to meet excess energy demand imposed by severe anaerobic stress of mercury intoxication. It also appears that the vigorous struggling may enhance the muscular activity which might have probably contributed significantly to the glycogen break

Table I : Changes in the level of glycogen content in different tissues of mice, *Mus musculus* treated to 30 days sublethal concentration of mercuric chloride followed by 30 days penicillamine treatment (values are expressed asμg/g wet weight of the tissue).

Nature of organs	Group A	Group B (days of exposure)					
		V	X	XV	XX	XXV	XXX
Brain	203.00	318.00	250.00	228.25	152.25	169.00	91.56
	± 0.31	± 0.85	± 1.06	± 1.19	± 1.53	± 2.04	± 1.12
% COC		+56.65*	+23.15*	+12.43*	-25.00*	-16.74*	-54.89*
Liver	218.64	229.00	229.50	152.50	112.25	96.25	60.75
	± 1.64	± 1.54	± 0.80	± 1.38	± 1.15	± 1.42	± 1.90
% COC		+4.73*	+4.96*	-30.25*	-48.65*	-72.21*	-72.21*
Muscle	649.50	324.25	306.50	251.00	219.00	136.25	108.00
	± 0.82	± 1.87	± 1.07	± 1.38	± 1.24	± 1.58	± 1.25
% COC		-50.07*	-52.80*	-61.35*	-66.28*	-79.02*	-83.37*
Kidney	208.75	208.00	214.25	157.75	142.50	126.50	83.75
	± 1.26	± 1.46	± 0.72	± 1.37	± 1.29	± 0.77	± 1.59
		-0.35	+2.63	-24.43*	31.73*	39.40*	-59.88*
		Group C (days of exposure)					
Brain	203.00	105.50	183.25	183.50	228.00	282.25	217.30
	± 0.31	± 1.05	± 1.46	± 1.64	± 1.47	± 1.15	± 1.28
% COC		-48.02*	-9.72*	-9.60*	+12.31*	+38.54*	+7.04*
Liver	218.64	495.25	392.50	120.02	302.75	347.00	215.00
	± 1.64	± 1.61	± 1.32	± 1.38	± 1.64	± 1.72	± 1.68
% COC		+126.51	+97.51*	-45.10*	+41.21*	+58.70*	-1.66*
Muscle	649.50	125.75	133.75	187.50	243.50	236.00	267.00
	± 0.82	± 1.87	± 1.38	± 1.15	± 1.15	± 1.85	± 1.37
% COC		-80.63*	-79.40*	-62.50*	-62.50*	-63.66*	-58.89*
Kidney	208.75	120.75	117.00	197.50	197.50	229.50	250.25
	± 1.26	± 1.55	± 1.41	± 1.46	± 1.64	± 1.46	± 1.30
		-42.15	-43.95*	-5.38*	-5.38*	+9.94*	19.88*

Group A = Control; Group B = Mercury treated; Group C = Penicillamine treated; Mean ± S.E. (Mean of six individual observation); (-) Indicates the percentage over the control; * = Significance at 5% level of 't' test; % COC = Percentage change over control.

down. A similar suggestion was made by Vatal & Aiyar (1988) for accumulation of lactic acid in rat muscle when exposed to lithium. Another possible reason for glycogen depletion in the tissues may be due to impairment of glycogen synthesis.

In the present investigation, mercury intoxicated mice showed decrease in their liver glucose level from that of control (Table II). Mobilization and utilization of glucose level were observed by Sampath *et al.* (1992) in *Rana tigrina* exposed to sevin.

Table II : Changes in the level of glucose content in different tissues of mice, *Mus musculus* treated to 30 days sublethal concentration of Mercuric chloride followed by 30 days penicillamine treatment (values are expressed as μg/g wet weight of the tissue).

Nature of organs	Group A	Group B (days of exposure)					
		V	X	XV	XX	XXV	XXX
Brain	139.53	72.40	42.25	48.40	74.13	108.90	155.91
	± 0.24	± 1.51	± 0.56	± 0.79	± 0.98	± 0.67	± 1.37
% COC		-48.14*	-69.74*	-65.33*	-46.90*	-22.00*	+11.65*
Liver	371.48	113.89	61.07	90.65	143.85	179.58	194.74
	± 2.94	± 1.24	± 0.65	± 0.82	± 0.82	± 0.78	± 0.85
% COC		-69.34*	-83.56*	-75.59*	-69.27*	51.65*	-47.57*
Muscle	156.53	113.51	56.65	83.35	115.81	146.92	79.00
	± 0.78	± 1.08	± 0.54	± 1.00	± 0.82	± 0.78	± 1.11
% COC		-27.48*	-63.80*	-46.75*	-26.01*	-61.30*	-49.53*
Kidney	165.38	147.50	54.16	74.32	102.37	160.18	189.95
	± 0.40	± 1.12	± 0.68	± 0.78	± 1.42	± 1.18	± 0.61
		-10.81*	-67.25*	-55.06*	-38.10*	-3.14*	+14.85*
		Group C (days of exposure)					
Brain	139.53	149.04	236.62	197.05	141.74	144.43	115.04
	± 0.24	±0.85	±1.05	±0.96	±1.20	±1.02	±0.85
% COC		+6.73	69.46*	+41.12*	1.51	+3.43*	-17.61*
Liver	371.48	129.64	340.91	271.38	150.19	173.24	112.54
	± 2.94	±0.81	±0.78	±0.92	±1.12	±0.92	±1.22
% COC		-65.10*	-8.22	-26.94*	59.56*	-53.36*	-69.70*
Muscle	156.53	62.42	461.32	133.84	96.22	140.97	160.75
	± 0.78	±0.81	±1.41	±0.85	±0.89	±1.08	±1.08
% COC		-60.12	+194.71*	-14.49*	-38.52*	-9.94*	+2.69*
Kidney	165.38	355.89	189.56	135.40	103.13	43.21	162.29
	± 0.40	±0.99	±1.08	±1.35	±1.76	±0.81	±1.06
		+115.19*	+14.62*	-18.12*	-3764*	73.87*	-1.86*

Group A = Control; Group B = Mercury treated; Group C = Penicillamine treated; Mean ± S.E. (Mean of six individual observation); (-) = Indicates the percentage change over the control; * = Significance at 5% level of 't' test; % COC = Percentage change over control.

The response of glucose profiles exhibited a similarity with those of glucose patterns. Under hypoxic conditions, the mice derive the energy by anaerobic breakdown of glucose which is available to the cells with the increased glycogenolysis. The observed depletion is due to hypoxia which is evident in this study. The higher rate of glycogen and glucose depletion in present study explain the increased demand for these molecules to provide energy for the cellular biochemical

process under toxic manifestations.

In the present investigations, *Mus musculus* show a remarkable recovery from mercury toxicity. When mice were exposed to mercury followed by penicillamine treatment, they show a fluctuation in the level of carbohydrate metabolism in all the tissues, when compared to mercury treated respective tissues (Table I & II). The fluctuation in glucose and glycogen profile suggest that the tissues have recovered from the mercury toxin. It has been pointed out (Mary Chandravathi & Reddy, 1996) that metal elimination from endocrine glands might restore the normal hormones may help the glycogen spilitting enzymes (phosphorylase) and glycogen synthesizing enzymes during recovery period to bring back the glycogen and glucose level to near normal/a little over untreated controls.

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