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Enzymological changes in the kidney of rat due to higher intake of cobalt, manganese and arsenic

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Abstract: The effects of cobalt and manganese ions on some key enzymes in the kidney of rat were recorded. Cobalt, manganese and Arsenic invariably inhibited the activity of alkaline phosphatase, acid phosphatase, glucose-6-phosphatase and cholinesterase. However, manganese exposed rats showed an elevation in the activity of lipase while cobalt decreased the activity. After a combined treatment with cobalt manganese and Arsenic the activity of enzymes returns towards normal status indicating antagonistic behavior of these essential trace elements. However Arsenic is a cumulative poison and considered as very toxic metalloid that causes oxidative stress.

Keywords- Cobalt, Manganese, Arsenic, Kidney, Poison

INTRODUCTION

The cobalt manganese and Arsenic are elements and play vital role in numerous biological reactions, they are known to initiate and to control certain biological processes. But in higher quantities they are hazardous to plants and animals health.

Manganese poisoning is characterized by a severe psychiatric disorder known as *Locura Manganica* which is similar to a neurological disorder the "Parkinson's disease". Cobalt although less toxic, causes true polycythaemia, hyperplasia of bone marrow, reticulocytosis and congestive heart failure, arsenic toxicity cause oxidative damage of organs chromosomal aberrations and apoptosis. (Berlin, 1950; Herich, 1965; Dickson, 1974; Rana *et al*., 1985; Prakash, 1991, 1993 and ATSDR, 2000)[1-7]. Frequent use of cobalt and manganese both in glass, steel, paints and ceramic industries has increased the risk of their individual and combined effects. Since the enzymological changes determine the nature, the extent of metabolism and the action and fate of the chemicals in the animal, the present work on alkaline phosphatase (3.1), acid phosphatase (EC 3.1.3.2). Glucose-6-phosphatase (EC 3.1.3.9), cholinesterase (EC 3.1.1.7) and lipase (EC 3.1.1.3) and arsenic supplemented diets and arsenic and melatonin supplemented diets was undertaken in the kidney of rats to candidly assess the position.

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MATERIALS & METHODS

Fourty albino rats (*Rattus rattus* albino) of both sexes and same age and weight (100+10 g) were selected from laboratory rat colony. They were allowed for laboratory conditions for one week and then were randomly allocated into four groups each having ten rats. Each rat was housed in separate galvanized case fed on a standard diet and provided tap water *ad libitum*. They were maintained under standard conditions as described earlier (Rana and Prakash, 1986)[8]. The rats of group A were exposed with sub lethal dose of cobalt as acetate (50mg/kg body weight/day/rat), those in group B received a sub lethal dose of manganese

as manganese chloride (250 mg/kg body weight/day/rat) and rats in group C were exposed a sub lethal dose of the equimolar mixture of cobalt manganese and arsenic (150mg/kg body weight/day/rat). The rats of group D were fed on laboratory diet alone and were taken as controls. The solutions of metals were administered to the rats by gavages daily for thirty days.

The rats were starved for 24 hours and then killed by decapitation. Both the kidneys were removed carefully and quickly excised and frozen. Tissue homogenates were prepared in 0.25 M ice cold sucrose solutions (10%W/v). During homogenation the temperature was maintained near 0°C. The homogenates were centrifuged for 20 minutes at 500g and respective clear supernanent fluids were used as enzyme source. The activities of alkaline phosphatase and acid phosphatase (Bodonsky. 1933)[9], glucosce-6-phosphatase (Swanson. 1965)[10]. Cholinicstcrase (Rappaport *et.al.*, 1959)[11] and lipase (Bier, 1955)[12] were determined spectrophotometrically. For each enzyme triplicate samples were analyzed and the incubations were repeated three times. Student's "t" test (Fisher, 1950)[13] was applied to calculate statistical significance. Total protein content was determined following the method of Lowry *et. al.* (1951)[14].

RESULTS

Results are shown in Table 1. Cobalt manganese both invariably inhibited the activity of all the enzymes selected for the present study except the lipase which was elevated by manganese. The results on their reciprocal effects are shown in Table 1. Approximated control values favoring a physiological antagonistic mechanism between these metals.

Table 1 : Mean Values of Enzymes in the Kidney of rat treated with Cobalt, Manganese and arsenic.

Enzymes	Control	Treatment		
		Cobalt	Manganese	Cobalt + Manganese +Arsenic
Alkaline Phosphatase[a]	0.48 ± 0.016	0.10 ± 0.042 (79.16)[c]	0.12 ± 0.0180 (75.0)[c]	0.32 ± 0.026 (33.33)[c]
Acid Phosphatase[a]	0.32 ± 0.018	0.11± 0.024	0.18 ± 0.021	0.24 ± 0.020
Glucose-6 Phosphatase[a]	0.56 ± 0.24	0.49 ± 0.018 (12.50)[c]	0.50 ± 0.029 (21.42)[c]	0.52 ± 0.016 [C] (7.14)[c]
Cholinesterase unit	28.00 ± 3.020	20.0 ± 20.86 (28.57)[c]	8.00 ± 2.850 (71.42)[c]	18.00 ± 2.160 (35.71)[c]
Lipase units	10.00 ± 1.020	6.00 ± 0.890 (40.0)[c]	14.00 ± 1.018 (40.0)[b]	9.00 ± 0.080 (10.0)[c]
Arsenic and Melatonin	3.31 ± 0.10	3.25 ± 0.12	2.98 ± 0.07	3.07 ± 0.12

All values expressed as mean S.E. (5-observations).

Values are significant at P<O.05, P<0.02 P<0.01 P<0.001

a. activity is expressed in mg of inorganic phosphate librating/mg protein/hr at 37°

b. Indicates % stimulation and c. indicates % inhibition.

values in parentheses indicate per cent alterations.

DISCUSSION

The earlier records have shown that manganese is involved in a wide range of enzyme activities of tissues and that mitochondrial structure and function are particularly affected in manganese deficiency (Rosenstock *et al.*, 1971)[15]. Furthermore, a dietary deficiency of manganese in mice was found to cause alterations in the integrity of cell membranes. Endoplasmic reticulum was swollen and irregular mitochondria were found with elongated stocked cristae in the liver, heart and kidney cells and there was an over abundance of lipid in liver parenchymal cells (Bell and Hurley, 1974)[16]. However, several reports on the effects of manganese have nevertheless been limited to brain only (Graham, 1984; Bonilla, 1984)[17-18]. Chandra and Shukla (1978)[19] have reported that manganese inhibits the activity of succinate-dehydrogerase (SDH) and alkaline phosphatase whereas it the activity of monoamine oxidase (MAO). Alto oxidases although no comparative data on kidney enzymes are available, present results are primarily the manifestations of manganese accumulation in the kindly.

Enzymological effects of cobalt have not been studied however cobalt salts are known to decrease cytochrome P450 (Tephly and Hibbeln, 1971)[20] and nicotinamide adenine dinucleotide phosphatehydratase (NADPH) in the liver. An impairment of the drug metabolizing enzyme system (DMES) has also been reported (Mitchell *et.al.*, 1974)[21]. A significant inhibition by cobalt of acid phosphatase, alkaline phosphatase, glucose-6-phosphatase, and cholinesterase was noted by Rana *et al*. (1985)[4] in the liver of rat.

The interaction between cobalt and manganese which may beat the absorption or past absorption level clearly shows antagonism. The inhibition of cation competition for absorption may be speculated upon, but nothing is certain until the biological significance of the gut compartment for cobalt and manganese is established. Cobalt is the interim part of vitamin B_{12} and may modify manganese toxicity in combination. It is important that the complex inter relationship among essential minerals are as significant as those among heavy metals.

The amount of enzymes may be controlled by factors affecting their rate of synthesis or their rate of breakdown. Although a large number of compounds of different molecular types may affect the same enzyme system, there may be a number of enzymes which are affected by one class of compounds and not by another. Variation in enzyme activity are further known to be the result of change caused by allostecric interaction of the toxicophore with the enzyme or of differences in the content of enzyme protein induced by a change in the synthesis of degradation of the proteins.

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REFERENCES

1. **Berlin, I.N. 1950.** *J. Biol. Chem*. **187:** 41
2. **Herich, R. 1965.** *Chromosoma*. **17:** 194.
3. **Dickson, J. 1974.** *Aust. Vet. J.* **50:** 236.
4. **Rana, S.V.S.. Prakash, R. and Agrawal, V.P. 1985.** *Arh. hig rada. toksikil*. **36:** 365-370.
5. **Prakash, R. 1991.** *J. Tissue Research*. **1:**21-27
6. **Prakash, R. 1993.** *J. Tissue Research*. **3:** 53-60
7. **ATSDR 1990.** Case studies in environmental medicine, Agency for toxic substance and disease registry. Atlanta G.A. U.S.A.
8. **Rana, S.V.S. and Prakash. R. 1986.** *Exptl. Pathology*. **29:** 1930-1935.
9. **Bodonsky, A. 1933.** In Howk's Physiological Chemistry. Oser. B.L. (Ed.). *Tata McGraw-Hill*, Bombay. 1118.
10. **Swanson., M.A. 1965.** Methods in Enzymology, Colowick, S.P. and Kaplon, N.O. (Eds.). Academic Press, New York, 542p.
11. **Rappaport, F., Fischl, J. and Pinto, N. 1959.** *Clin.Chim Acta*, **4:**227.
12. **Bier, M. 1955.** Lipase. In: Methods in Enzymology, Colowick. S.P. and Kalpan N.O. (Eds.), Academic Press, New York. pp. 627-642.
13. **Fisher, R.A. 1950.** Statistical Methods for Research Workers, 11th ed., Oliver and Boy, London.
14. **Lowry, O.H. Rosebrough, N.J., Farr, A.L. and Randall, J.R. 1951.** *J.Biol-Chem*. **193:** 265.
15. **Rosenstotock, H.A., Simons, D.G. and Meyer, J.S. 1971**. *J. Amer. Med. Chem*. **217:**1354.
16. **Bell, L.T. and Hurley, L.S. 1974.** *Pro. Soc. Exp. Biol and Med*. **145:**1321.
17. **Graham, D.G. 1984.** *Neurotoxicity*. **5:** 83-96.
18. **Bonilla, E. 1984.** *Experimental Neurology*. **84**: 696-700.
19. **Chandra, S.V., Shukla, G.S. 1978.** Manganese encephalopathy in growing rats. *Environ Res*. **15:**28-37.
20. **Tephly, T.R. and Hibbeln, P. 1971.** Biochemical Mechanism of Liver Injury, StatusT.F. (Ed.), Academic Press, London.
21. **Mitchell, J.R., Jollow, D.J., Gillete, J.R. 1974.** Int. Symp. on hepatotoxicity, Eliakim, J. Eshdion and Zimmerman, A.J. (Eds.), Academic Press, New York, 45p.

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