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Effect of Zinc Deficiency on Haematological Parameters and Mineral Contents of Selected Tissues in Albino Rats

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Abstract: The effect of zinc deficiency on tissue level of some minerals (Na, K, Ca, P, Cu, Fe, Mg, Zn, Mn) in growing rats was investigated. Two groups of rats were fed either a zinc adequate diet or zinc deficient diet (6 ppm) for six weeks. At the end of the feeding period, zinc deficiency decreased the final body weight of the rats by 70% compared to the control. There was no significant difference ($P < 0.05$) in the packed cell volume (PCV) and the white blood cell count (WBC) of the zinc deficient rats was significantly increased ($P < 0.05$) compared to the control. Tissue content of minerals measured in the liver and kidney of both group of rats showed that zinc deficiency significantly increased ($P < 0.05$) tissue concentration of some elements (Fe, Cu, Ma, Ca, K and Na) while Mg and phosphorus levels were lowered compared to the control. This result suggest that zinc deficiency in rats could have adverse effect on intermediary metabolism as a result of interplay in the interactions between these minerals.

Key words: Zinc deficiency, minerals, rat, metabolic function

Introduction

Minerals are inorganic nutrients that are required in small amounts for proper metabolic function in plant and animals. They are obtained through the diet or in combination with organic molecule (Seeley *et al*., 1996). Zinc is an essential element in the nutrition of man and animals and has been identified as an integral of numerous enzyme systems. Zinc status influence several aspects of cellular metabolism. Zinc deficiency has been associated with anaemia, skeletal defects demyelination and lesion in the cardiovascular system (Guthrie, 1989) especially during postnatal development Several studies have highlighted the importance of zinc in nutrition (Prasad, 1991; Adisa and Odutuga, 1998). In general zinc functions to regulate activity of many metalloenzymes (Ajayi and Odutuga, 2004), since it serves as a prosthetic group. The present study is aimed at acquisition of additional information about the effects of zinc deficiency on other minerals in selected tissues of albino rats.

Materials and Methods

Twenty male white albino rats *(Rattus Norvegicus*) were divided into two groups, each containing ten animals and housed in plastic cages of stainless steel wire top and bottom. The animals were acclimatized for 24hours before introducing the experimental diets.

The two groups were maintained on (a) Control diet containing adequate zinc and (b) Zinc deficient diet. The composition of the diet was as described by Odutuga and Ajayi (1998). All reagents were of analytical grade and products of BDH Limited and Sigma chemical Company, England. The feeding trial lasted six weeks.

Tissue preparation: The rats were anaesthetized and sacrificed by cervical dislocation. Blood was drawn from the heart by cardiac puncture using a sterile syringe and needle. Blood was drawn into heparinized capillary tube for PCV analysis. Blood serum was obtained and stored for haematological parameters. The liver and kidneys (decapsulated) were removed, drained of blood and weighed .

Each tissue was ashed and dissolved in 10% HCl and made up to 100ml standard flask with distilled water. The mineral content of each tissue were analyzed using Atomic Absorption Spectrophotometer (AOAC, 1990). Phosphorus was determined calorimetrically using vanadonomolybdate method.

Results and Discussion

Table 1 shows the mean initial and final body weight of rats in the control and zinc deficient rats. In the present study the average weight gained by the zinc deficient group of rats was 70% less than that of the control respectively. The overall growth pattern showed that zinc deficient rats grew less than the control. It may be due to the fact that zinc plays an essential role in the formation of DNA and RNA and hence synthesis of protein. (Prasad, 1991). Hence a low- zinc status may affect protein metabolism which can result in reduced growth rats. This observation agrees with previous reports (Odutuga and Ajayi, 1998). Iron and copper deficiencies had also been shown to affect growth of rats (Oloyede and Folayan, 1995: Ajayi, 2005) Also zinc deficiency had been associated with reduced blood growth hormone levels in animals and humans (King, 1990)

Table 2 shows the PCV and WBC count of the zinc deficient and control rats.

There was no significant difference between the packed

Table 1: Mean Initial and final body weight of animals

	A	B
Final Body weight (g)	88.84±2.32	63.10±1.20
Initial Body weight (g)	40.73±4.31	35.1±2.10

A: Control rats, B: Zinc deficient rats

Table 2: Haematological Parameters

	Packed cell volume (PCV) %	White blood cell WBC (count).
A	44.50 ± 5.6	2950 ± 70.71
B	46.50 ± 2.12	4150 ± 21.23

Table 3: Mineral contents of the Kidney

Minerals	Control (ppm)	Zinc deficient (ppm)
Iron	40.13±18.73	82.59±21.61
Copper	24.55±5.87	49.73±5.07
Zinc	229.42±1.39	225.93±1.89
Manganese	26.12±8.02	115.93±4.23
Magnesium	53.27±13.24	3.73±1.36
Calcium	41.47±13.58	80.80±28.82
Potassium	411.43±74.96	530.778±21.77
Sodium	497.76±76.85	862.10±148.85
Phosphorus	165.40±11.50	88.76±46.94.

Table 4: Mineral Contents of the Liver

Minerals	Control (ppm)	Zinc- deficient (ppm)
Iron	16.34±3.24	22-24±3.68
Copper	11.90±2.08	13.50±6.85
Zinc	90.07±21.32	90.96±33.12
Manganese	7.43±3.08	5.37±3.10
Magnesium	41.96±7.10	30.33±10.65
Calcium	6.03±3.98	19.13±7.66
Potassium	161.19±26.30	222.12±8.76
Sodium	183.93±27.32	246.22±17.77
Phosphorus	105.72±1.16	88.84±2.94

cell volume (PCV) of zinc deficient rats and the control. This may be due to interference of zinc with copper bioavailability. Relatively low levels of dictary zinc may interfere with copper absorption thereby increasing it and copper helps in the formation of red blood cells (Maurice *et al.*, 1997). The white blood cell count (WBC) for the zinc deficiency rats is significantly higher ($P<0.05$) than the control. This may probably be due to infection since considerable amount of copper facilitates Iron absorption, which favours the multiplicity of invading bacteria (Vander *et al.*, 1998). Also zinc deficiency is characterized by small thymus and spleen with resultant reduction in its capacity for T and β-lymphocyte production (Maurice *et al.*, 1997). Zinc deficiency resulted in accumulation of copper in the liver and kidney of rats. The copper level in these organs were significantly ($P<0.05$) higher than the control. This may be attributed to interference of zinc with copper absorption. Both zinc and copper compete for the same sites on the protein carrier therefore the zinc deficiency is accompanied with excess copper, this is similar to the observation made by (Pfeiffer and Lamola, 1983). The accumulation of copper also suggest the potential for a zinc-copper antagonism affecting metabolic phenomena. Its been reported that the intestinal mucosa is the site of competition between these closely related transition metals. The magnesium content of the zinc deficiency rats was significantly lower than the control. The reduction poses a threat to life because magnesium serves as a co- factor of many enzyme especially those that are energy yielding (ATPases) (Nelson and Cox, 2002). The low magnesium content herein observed in these organs may have adverse effect on intermediary metabolism.

The reason for the significant reduction ($P<0.05$) in manganese content of these organs cant be understood, because zinc deficiency had no known effect on the intestinal absorption of manganese in the rat. Nevertheless manganese serves as a cofactor to many enzymes (Wall Work *et al.*, 1982.). Magnesium is also needed for the O- methylation of catecholamines norepinephrine, epinephrine and dopamine to methylated derivatives.

The iron content of the liver and kidney were significantly increased ($P<0.05$) compared to the control. This may be due to the considerable amount of copper in the zinc deficient rats, since copper is needed to facilitate the absorption of iron (Maurice *et al.*, 1997).

The calcium content in these organs is significantly higher ($P<0.05$) than the control. This may likely affect the synthesis of parathyroid hormone which promotes intestinal absorption of calcium and demineralization of bone leading to increase in extracellular calcium. This may in part be responsible for the neuromuscular changes observed in zinc deficient rats (Prasad, 1991).

The sodium and potassium content of the zinc deficient group is significantly higher than that of the control. Sodium is found in soft tissues and extracellular fluids while potassium is the major constituent of intracellular fluids required for the maintenance of cell integrity. Both are concerned with maintenance of acid- base balance and osmotic regulation of body fluids (Willard *et al.*, 1989). The significant increase in the concentration of these two may probably affect the activity of enzymes involved in active transport in the kidney especially the sodium pump may be adversely affected.

The phosphorus content is zinc deficiently rats is significantly lower ($P<0.05$) in these organs. Phosphorus status may affect the level of the cellular currency (ATP) of the body. The finding in this study corroborates earlier reported that the less phosphate groups would be available for the phosphor of ethanolamine and choline needed for the synthesis of phosphatidyl ethanolamine and phosphatidyl choline due to reduced activity of Alkaline phosphatase in zinc deficiency (Odutuga and Ajayi, 1998).These two major phospholipids play vital roles in the maintenance of membrane fluidity. A change in membrane fluidity is an indication of membrane damage (Odutuga, 1977; Cunnane, 1988). The zinc content in the liver is not

significantly different from the control while a significant difference ($P<0.05$) was observed for the kidney this could be due to the fact that liver acts as zinc pool, (or as zinc stores), while the kidney turns over zinc rapidly during zinc deficiency. Its been reported that some organs bind zinc more strongly than others. (Kings, 1990). From the foregoing, zinc deficiency in rats could grossly affect intermediary metabolism since most of these minerals serve as activators to various enzymes involved in metabolic pathways.

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