

ROLE OF ZINC IN FAILURE TO THRIVE

THESIS

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TO MY PARENTS

AND MY COUNTRY



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CONTENTS

	<u>Page</u>
1. INTRODUCTION.....	1
2. AIM OF THE WORK.....	3
3. REVIEW OF LITERATURE.....	4
A- Importance of zinc.....	6
B- Requirements of zinc.....	20
C- Sources of zinc.....	23
D- Zinc metabolism.....	27
- Absorption.....	27
- Distribution.....	35
- Excretion.....	42
E- Zinc deficiency.....	44
- Aetiology.....	44
- Clinical manifestations.....	48
- Biochemical criteria.....	56
F- Growth and factors affecting it.....	61
4. MATERIAL AND METHODS.....	65
5. RESULTS.....	75
6. DISCUSSION.....	115
7. SUMMARY AND CONCLUSION.....	124
8. REFERENCES.....	127
9. ARABIC SUMMARY.....	

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INTRODUCTION

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INTRODUCTION

Zinc has an important role in protein, carbohydrate and fat metabolism. It is also responsible for activation of various enzymes such as D.N.A. and R.N.A. polymerase, alkaline phosphatase, aldolase, lactate dehydrogenase and phospholactate.

Symptoms arising from zinc deficiency include growth retardation, anorexia and hypogonadism (Prasad et al., 1963 a).

Growth retardation is the earliest and cardinal feature of zinc deficiency in all young animal species. Zinc deficiency in humans has been recognized only for the last decade or so. Prasad et al., (1961) and 1963 a) were able to show that a condition described nearly fifty years before and which was characterized by hypogonadal dwarfism in young males of Middle East was largely attributable to zinc deficiency.

Evidence of zinc deficiency in Egyptian children and infants suffering from kwashiorkor was reported by many authors (Sandstead et al., 1965, 1967 and Salem 1978). The interaction between multiple nutrient deficiencies and/or parasitism is important, with the increasing supplementation of diet with cereals and vegetables (a diet rich in phytates and fibre) which

AIMS OF THE WORK

17

AIM OF THE WORK

- (1) To study the contribution of zinc deficiency in children who fail to thrive.
- (2) To compare children infested with common parasites in Egypt namely Bilharziasis, Ascariasis, Ankylostomiasis and Giardiasis, to those deparasitised.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Many elements occur constantly in living tissues in such small amounts that the early workers, who were unable to measure their concentrations, referred to them as occurring in "traces". By popular usage they were then referred to as "trace elements". Other terms such as "Minor elements", "Oligo elements and the more recent one which is perhaps more acceptable, "Micronutrient elements"(Prasad et al., 1970). These elements (Underwood 1977), are iron, zinc, copper, manganese, nickel, cobalt, molybdenum, selenium, chromium, iodine, fluorine, tin, silicon, vanadium and arsenic. In addition, boron is essential for the higher plants but has not yet been shown to be necessary for animals. According to Mertz (1970) an element is considered to be essential if its deficiency consistently results in impairment of a function from optimal to suboptimal. Cotzias (1967) states the position more completely. He maintains that a trace element can be considered essential if it meets the following criteria:-

- It is present in all healthy tissues of all living things.
- Its concentration from one animal to the next is fairly constant.

- Its withdrawal from the body induces reproducibly of the same physiological and structural abnormalities regardless of the species studied.
- Its addition either reverses or prevents these abnormalities.
- The abnormalities induced by deficiency are always accompanied by pertinent, specific biochemical changes.
- These biochemical changes can be prevented or cured when the deficiency is prevented or cured.

Thus zinc is one of the essential trace elements.

Importance of Zinc:

(1) Effects on growth, appetite and taste:

Growth retardation was observed in the original demonstration of zinc deficiency in rats (Elvehjem 1934) and has been a feature of this deficiency in all subsequent studies with all species. It probably arises primarily from a decreased activity of thymidine kinase and hence impaired D.N.A. synthesis and cell division. Lambs and calves fed a severely Zinc deficient diet (1.2 p.p.m. zinc) ceased weight gain abruptly, and growth arrest occurred within two weeks (Mills et al., 1967). In pregnant rats a maternal dietary zinc deficiency severely impairs fetal growth (Hurley and Swenerton 1966), while such a diet fed during lactation impairs growth in the suckling pups. Nutritional dwarfism and hypogonadism were the main clinical manifestations of the conditioned zinc deficiency first demonstrated by Prasad and co-workers in young males in Iran (Prasad et al., 1961) and Egypt (Prasad et al., 1963 a). When adequate zinc supplements were given to these individuals growth increased.

It was found that the growth inhibition of zinc deficiency results partly from impaired appetite (Miller et al., 1968).

Chesters and Quarterman (1970) found that the voluntary food intake of Zinc-deficient rats falls to 70% of that of the controls. The zinc deficient rats responded to a zinc supplemented diet within 1-2 hrs by an increase in food intake.

The physiological role for zinc in normal taste sensation was first established by Henkin and associates, (1969), together with the further important observation that the hypogeusia and dysgeusia that commonly occur in adults can respond to oral zinc therapy.

(2) Zinc and Keratogenesis:

Alopecia and gross skin lesions were observed in the early investigations of zinc deficiency in rats and mice (Underwood 1977). Histological studies disclosed a condition of parakeratosis i.e., a thickening and hyper keratinization, with failure of complete nuclear degeneration, of the epithelial cells of the skin and oesophagus (Follis et al., 1941). In more severe zinc deficiency scaling and cracking of the paws with deep fissures develop, in addition to loss of hair and dermatitis (Forbes 1960). The healing response of such parakeratotic lesions to supplemental zinc is rapid and dramatic. An association between chronic skin ulceration from different causes in man and subnormal plasma or serum zinc concentrations has been observed in several studies e.g., Halstead and Smith