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PRELIMINARY REVIEW ON: ZINC AND ITS IMPORTANCE FOR HUMAN HEALTH

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Abstract

Since its first discovery in an Iranian male in 1961, zinc deficiency in humans is now known to be an important malnutrition problem world-wide. It is more prevalent in areas of high cereal and low animal food consumption. The diet may not necessarily be low in zinc, but its bio-availability plays a major role in its absorption. Phytic acid is the main known inhibitor of zinc. Compared to adults, infants, children, adolescents, pregnant, and lactating women have increased requirements for zinc and thus, are at increased risk of zinc depletion. Zinc deficiency during growth periods results in growth failure.

Keywords: Deficiency, zinc, depletion, metabolism, nutrition

INTRODUCTION

Zinc essentiality was established in 1869 for plants, in 1934 for experimental animals and in 1961 for humans. A syndrome of anaemia, hypogonadism and dwarfism was reported in a 21-year-old Iranian farmer in 1961 who was subsisting on a diet of unrefined flat bread, potatoes, and milk. Shortly after, a similar syndrome was observed in Egyptian adolescents who had similar dietary history to that of the Iranians, mainly subsisting on bread and beans. Administration of supplemental zinc or diets containing adequate animal-protein foods improved growth and corrected the hypogonadism, while anaemia responded to oral iron treatment. Subsequent studies showed that the syndrome was primarily the result of low dietary zinc intake in the diet.

METABOLISM

Absorption

Zinc is absorbed in the small intestine by a carrier-mediated mechanism. Under normal physiologic conditions, transport processes of uptake are not saturated. The fraction of zinc absorbed is difficult to determine because zinc is also secreted into the gut. Zinc administered in aqueous solutions to fasting subjects is absorbed efficiently (60-70%), whereas absorption from solid diets is less efficient and varies depending on zinc content and diet

Zinc transporters (ZnTs)

There are at least 10 ZnTs and 15 zip transporters in human cells. They appear to have opposite roles in cellular zinc homeostasis. The expression and cellular distribution of the ZnTs is highly regulated by changes in zinc level. ZnTs reduce intracellular zinc availability by promoting zinc efflux from cells or into intracellular vesicles, while zip transporters increase intracellular zinc availability by promoting extracellular zinc uptake and perhaps, vesicular zinc release into the cytoplasm.

Homeostasis

Maintaining a constant state of cellular zinc, or homeostasis, is essential for survival. In animals and humans, adjustments in total zinc absorption and endogenous intestinal excretion are the primary means of maintaining zinc homeostasis. The adjustments in gastrointestinal zinc absorption and endogenous excretion are synergistic Shifts in the endogenous excretion appear to occur quickly with changes in intake just. Above or below optimal intake while the absorption of zinc responds more slowly, but it has the capacity to cope with large fluctuations in intake.

Excretion

Loss of zinc through gastrointestinal tract accounts for approximately half of all zinc eliminated from the body. Considerable amount of zinc is secreted through the biliary and intestinal secretions, but most of it is reabsorbed. This is an important process in the regulation of zinc balance.

HUMAN REQUIREMENTS

Since the mid-1990s, the WHO, the Food and Agriculture Organization, the International Atomic Energy Association and the Food and Nutrition Board (FNB) of the Institute of Medicine (IOM) have convened expert committees to develop estimates of human zinc requirements and dietary intakes needed to satisfy these requirements.

GROUPS AT HIGH RISK

Compared to adults, infants, children, adolescents, pregnant and lactating women have increased requirements for zinc and thus, are at increased risk of zinc depletion.

Infants and children

Young children are at greater risk of zinc deficiency because of increased zinc requirements during growth. Exclusively breast-fed infants of mothers with adequate zinc nutriture obtain sufficient zinc for the 1st 5-6 months of their life.

Adolescents

The physiological requirements for zinc peak during adolescence at the time of the pubertal growth spurt, which generally occurs in girls between 10 years and 15 years and in boys between 12 years and 15 years. Even after the growth spurt has ceased, adolescents may require additional zinc to replenish depleted tissue zinc pools.

Pregnant and lactating women

Increased nutritional demands during pregnancy and lactation predispose women to zinc deficiency. These demands are greater during lactation, although, physiological adjustments in zinc absorption help to meet the needs for lactation.

CONSEQUENCES AND CAUSES OF ZINC DEFICIENCY

Consequences of zinc deficiency

Due to the multitude of basic biochemical functions of zinc in the cells of human body, there is a broad range of physiological signs of zinc deficiency. These signs vary depending on the severity of the condition. Organ systems known to be affected clinically by zinc deficiency states include the epidermal, gastrointestinal, central nervous, immune, skeletal, and reproductive systems.

Growth and development

One of the most studied clinical features related to zinc deficiency is the impairment of physical growth and development. The mechanisms involved, however, are not well understood. This effect is of most significance during the periods of rapid growth such as pregnancy, infancy and puberty during which zinc requirements are highest.

Causes of zinc deficiency

The general causes of zinc deficiency include inadequate intake, increased requirements, malabsorption, increased losses and impaired utilization. Inadequate dietary intake of absorbable zinc is the primary cause of zinc deficiency in most situations. This may result from low dietary intake or heavy reliance on foods with little or poorly absorbable zinc. Inadequate dietary zinc intake is common in many parts of the world. It is often exacerbated by physiologic conditions associated with elevated zinc requirements.

PREVENTION OF ZINC DEFICIENCY (INTERVENTION STRATEGIES)

Numerous zinc supplementation trials have shown that a wide range of health benefits can be realized by increasing the intake of zinc where diets are inadequate in this micronutrient. The results of these trials strongly argue for the development of programs to improve zinc status in high-risk populations.

Dietary diversification/modification

Dietary diversification or modification is a sustainable long-term approach to improving the intake of several nutrients simultaneously. Dietary diversification or modification strategies at the community or household level have the potential to increase the intake of bio-available zinc. Such strategies include (1) Agricultural interventions (2) Production and promotion of animal-source foods through animal husbandry or aquaculture (3) Processing strategies at the commercial or household level to enhance zinc absorption from plant-based diets. Agricultural interventions focused on plant-based foods may have little impact on intake of bio-available zinc.

Supplementation

Supplementation programs are useful for targeting vulnerable population subgroups, which are at a particular high-risk of micronutrient deficiencies. The easiest way to supplement zinc could be to include it in programs already delivering daily or weekly nutrient supplements for the prevention of iron deficiency anaemia and other micronutrient deficiencies.

Fortification

Food fortification is a more cost-effective and sustainable strategy to overcome micronutrient malnutrition than supplementation. Fortification programs can also be specifically targeted to increase the intake of zinc in groups of high-risk such as infants and young children who consume particular type of food. In many countries, infant formulas and complementary foods are currently fortified with zinc and other micronutrients.

Bio-fortification

Bio-fortification differs from ordinary fortification because it focuses on intrinsic enrichment of micronutrients in plant parts that are used for food while the plants are still growing, rather than having nutrients from external resources added to the foods when they are being processed. This is an improvement on ordinary fortification when it comes to providing nutrients for the rural poor, who rarely have access to commercially fortified foods.

REFERENCES

- 1. King JC, Cousins RJ. Zinc. In: Shils ME, Shike M, Ross AC, Caballero B, Cousins RJ, editors. Modern Nutrition in Health and Disease. 10th ed. Baltimore: Lippincott Williams and Wilkins; 2006. pp. 271– 85.
- 2. Prasad AS, Miale A, Jr, Farid Z, Sandstead HH, Schulert AR. Zinc metabolism in patients with the syndrome of iron deficiency anaemia, hepatosplenomegaly, dwarfism, and hypognadism. J Lab Clin Med. 1963; 61:537-49.
- 3. Sandstead HH, Prasad AS, Schulert AR, Farid Z, Miale A, Jr, Bassilly S, et al. Human zinc deficiency, endocrine manifestations and response to treatment. Am J Clin Nutr. 1967; 20:422–42.
- 4. Cousins RJ. Absorption, transport, and hepatic metabolism of copper and zinc: Special reference to metallothionein and ceruloplasmin. *Physiol Rev.* 1985; 65:238–309.
- 5. Cousins RJ, Liuzzi JP, Lichten LA. Mammalian zinc transport, trafficking, and signals. J Biol Chem. 2006; 281:24085–9.
- 6. Devergnas S, Chimienti F, Naud N, Pennequin A, Coquerel Y, Chantegrel J, et al. Differential regulation of zinc efflux transporters ZnT-1, ZnT-5 and ZnT-7 gene expression by zinc levels: A realtime RT-PCR study. Biochem Pharmacol. 2004; 68:699–709.
- 7. Hambidge M, Krebs NF. Interrelationships of key variables of human zinc homeostasis: Relevance to dietary zinc requirements. Annu Rev Nutr. 2001; 21:429–52.
- Maret W, Sandstead HH. Zinc requirements and the risks and benefits of zinc supplementation. J *Trace Elem Med Biol.* 2006; 20:3–18.
- King JC. Determinants of maternal zinc status during pregnancy. Am J Clin Nutr. 2000; 71:1334S— 43S.
- 10. Hambidge KM, Walravens PA. Disorders of mineral metabolism. Clin Gastroenterol. 1982; 11:87–
- 11. Brown KH, Peerson JM, Rivera J, Allen LH. Effect of supplemental zinc on the growth and serum zinc concentrations of prepubertal children: A meta-analysis of randomized controlled trials. Am J Clin Nutr. 2002; 75:1062-71.
- 12. Anderson JJ. Minerals. In: Mahan LK, Escott-stump S, editors. Krause's Food, Nutrition and Diet Therapy. USA: WB Saunders Co; 2004. pp. 120–63.
- 13. Devergnas S, Chimienti F, Naud N, Pennequin A, Coquerel Y, Chantegrel J, et al. Differential regulation of zinc efflux transporters ZnT-1, ZnT-5 and ZnT-7 gene expression by zinc levels: A realtime RT-PCR study. Biochem Pharmacol. 2004; 68:699-709.
- **14.** Lonnerdal B. Dietary factors influencing zinc absorption. *J Nutr.* 2000;130: S1378–83.
- 15. Andriollo-Sanchez M, Hininger-Favier I, Meunier N, Toti E, Zaccaria M, Brandolini-Bunlon M, et al. Zinc intake and status in middle-aged and older European subjects: The ZENITH study. Eur J Clin Nutr. 2005;59: S37-41.
- 16. Maret W, Sandstead HH. Zinc requirements and the risks and benefits of zinc supplementation. J Trace Elem Med Biol. 2006; 20:3-18.
- 17. Gibson RS, Anderson VP. A review of interventions based on dietary diversification or modification strategies with the potential to enhance intakes of total and absorbable zinc. Food Nutr Bull. 2009;30: S108-43.
- 18. Banuelos G, Lin ZQ. Florida: CRC Press, Boca Raton; 2009. Phytoremediation of seleniumcontaminated soil and water produces biofortified products and new agricultural byproducts. Development and Uses of Biofortified Agricultural Products; pp. 57–70.