USANA Technical Bulletin

Zinc

Technical Background

- Zinc is a cofactor for more than 300 enzymes needed for cell function in the eyes, kidneys, muscles, skin, and bones.[1] Its functions in the body are broad and numerous.
- Zinc serves as a necessary structural component of DNA-binding proteins that affect gene expression.
- Zinc is a component of alcohol dehyrdogenase, which is important in the conversion of retinol to retinal (required for vision in humans).
- The function of cell membranes is also dependent on zinc. Zinc affects the activity of enzymes attached to plasma membranes, and directly affects cell membranes by stabilizing compounds that prevent peroxidative damage.
- Zinc also frees the vitamin folate (so it can move across cell membranes), aids in the manufacture of heme, aids in essential fatty acid metabolism, and helps release vitamin A from its storage place in the liver.[2]
- Zinc is essential for normal salt-taste perception, wound healing, immune function, and general growth and development.[3,4]
- Signs and symptoms of zinc deficiency include growth retardation, skeletal abnormalities, defective collagen synthesis and/or cross linking, poor wound healing, night-blindness, impaired immune function, and impaired protein synthesis. [2]
- Excessive intakes of zinc can cause toxicity. Intakes of 1 to 2 grams can cause a metallic taste, nausea, vomiting, epigastric pain, and abdominal cramps. Ingestion of doses of 100 to 300 mg zinc in individuals with sickle cell anemia can induce copper deficiency (because zinc and copper compete for absorption).[5]

Sources and Recommended Intakes

- Foods rich in zinc include protein sources like shellfish, meat, and liver. Eggs and whole grain products are also high in zinc, but the phytates in grains can influence the absorption of zinc. The amount of zinc in vegetables will vary depending on the amount of zinc in the soil in which they are grown.
- The Recommended Dietary Allowance (RDA) for zinc is 5 mg/day for infants, 10mg/day for children, 15 mg/day for adult males and 12 mg/day for adult females. It is recommended that pregnant women take 15 mg/day and lactating women take 19 mg/day.[6]

Abstracts

Prasad AS. Zinc: an overview. Nutrition 1995;11:93-9. Zn deficiency in humans is widespread throughout the world. It is more prevalent in areas where the population subsists on cereal proteins. Conditioned Zn deficiency is seen in many disease states. Its deficiency during growth periods results in growth failure and lack of gonadal development in males. Other effects of Zn deficiency include skin changes, poor appetite, mental lethargy, delayed wound healing, neurosensory disorders, and cell-mediated immune disorders. Severe Zn deficiency, as seen in acrodermatitis enteropathica (a genetic disorder), is fatal if Zn is not administered to these patients. A clinical diagnosis of marginal Zn deficiency in humans remains problematic. Assays of Zn in granulocytes and lymphocytes provide better diagnostic criteria for marginal Zn deficiency than plasma Zn. Approximately 300 enzymes are known to require Zn for their activities. Zn is required for DNA synthesis, cell division, and protein synthesis. Recently, we learned that Zn-finger proteins are involved in genetic expression of various growth factors and steroid receptors. We suspect that several hundred Zn-containing nucleoproteins are probably involved in gene expression of various proteins. Zn deficiency adversely affects lymphocyte proliferation. This may be related to the enzymatic role of Zn in DNA synthesis and cell division. Thymulin, a thymic hormone involved in T-lymphocyte maturation, is known to be Zn dependent and is adversely affected by Zn deficiency. Thus, an adverse effect of Zn deficiency may also be in lymphocyte differentiation and maturity. Zn deficiency is known to decrease interleukin 2 production by helper T lymphocytes, and abnormalities in T-lymphocyte subpopulations have been observed in Zn-deficient humans.

Prasad AS. Zinc deficiency in human subjects. Prog Clin Biol Res 1983;129:1-33. During the past two decades, the essentiality of zinc for man has been established. Deficiency of zinc in man due to nutritional factors and several diseased states has been recognized. High phytate content of cereal proteins decreases availability of zinc; thus the prevalence of zinc deficiency is likely to be high in a population subsisting mainly on cereal proteins. Alcoholism is known to cause hyperzincuria and thus may play a role in producing zinc deficiency in man. Malabsorption, cirrhosis of the liver, chronic renal disease and other chronically debilitating diseases may similarly induce zinc deficiency in human subjects. A severe deficiency of zinc has recently been recognized to occur in patients with sickle cell anemia and a beneficial effect of zinc therapy in such patients has been reported. Growth retardation, male hypogonadism, skin changes, poor appetite, mental lethargy and delayed wound healing are some of the manifestations of chronically zinc-deficient human subjects. Taste abnormalities, correctable with zinc supplementation, have been observed in uremic subjects. Recently, abnormal dark adaptation related to zinc deficiency in patients with cirrhosis of the liver and sickle cell disease has been reported. In severely zinc-deficient patients, dermatological manifestations, diarrhea, alopecia, mental disturbances and intercurrent infections predominate and if untreated the condition becomes fatal. Zinc deficiency is known to affect testicular functions adversely in man and animals. This effect of zinc is at the end organ level and it appears that zinc is essential for spermatogenesis and testosterone steroidogenesis. Zinc is involved in many biochemical functions. Several zinc metalloenzymes have been recognized in the past decade. Zinc is required for each step of cell cycle in microorganisms and is essential for DNA synthesis. Thymidine kinase, RNA polymerase, DNA-polymerase from various sources and RNA-dependent DNA polymerase from viruses have been shown to be zinc-dependent enzymes. Zinc also regulates the activity of RNase; thus the catabolism of RNA appears to be zinc-dependent. The effect of zinc on protein synthesis may be attributable to its vital role in nucleic acid metabolism. The activities of many zinc-dependent enzymes have been shown to be affected adversely in zinc-deficient tissues. Three enzymes, alkaline phosphatase, carboxypeptidase and thymidine kinase, appear to be most sensitive to zinc restriction in that their activities are affected adversely within three to six days of institution of a zinc-deficient diet to experimental animals.

References

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