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Zinc Status in Athletes

Relation to Diet and Exercise

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Abstract

Zinc is involved in the biochemical processes supporting life, such as cellular respiration, DNA reproduction, maintenance of cell membrane integrity and free radical scavenging. Zinc is required for the activity of more than 300 enzymes, covering all 6 classes of enzyme activity.

Zinc binding sites in proteins are often of distorted tetrahedral or trigonal bipyramidal geometry, made up of the sulphur of cysteine, the nitrogen of histidine or the oxygen of aspartate and glutamate, or a combination. Zinc in proteins can either participate directly in chemical catalysis or be important for maintaining protein structure and stability.

The nutritional habits of elite athletes during training and competition are quite different from the recommended diet in the majority of the population. Endurance athletes often adopt an unusual diet in an attempt to enhance performance: an excessive increase in carbohydrates and low intake of proteins and fat may lead to suboptimal zinc intake in 90% of athletes. Mild zinc deficiency is difficult to detect because of the lack of definitive indicators of zinc status. In athletes, zinc deficiency can lead to anorexia, significant loss in bodyweight, latent fatigue with decreased endurance and a risk of osteoporosis.

Zinc is one of the essential trace elements and, as such, a member of one of the major subgroups of the micronutrients that have attained such prominence in human nutrition and health.

The exceptional ability of the zinc atom to participate in strong but readily exchangeable ligand binding, together with the notable flexibility of the coordination geometry of this metal, has proved to be extraordinarily useful in biological systems. The incorporation of this trace element into mammalian biological systems has been further facilitated by the lack of redox properties of zinc atom, which, in contrast to iron and copper, allows its utilisation without the risk of oxidant damage.

Zinc is ubiquitous in subcellular metabolism, and is an essential component of the catalytic site or sites of at least one enzyme in all classes of enzymatic activity. Several hundred zinc metalloenzymes have been identified in the plant and animal kingdoms.[1]

Since the recognition in 1934 of zinc as an essential trace element in humans, there has been continuous progress in our knowledge of the role of zinc in biochemistry and medicine. Dietary zinc deficiency in humans was recognised in 1961, and the first study[2] identifying acrodermatitis enteropathica as a definite disease of zinc absorption was published in 1973. Today it is recognised that a

marginal deficiency of zinc is common throughout the world, not only among low income people but everywhere an unbalanced diet is consumed.[3]

During the past decade, more than 200 zinc-dependent transcription factors involved in the gene expression of various proteins have been recognised.[4] Also, studies on the mechanisms of low zinc absorption in acrodermatitis enteropathica have been carried out to identify physiological and pathological aspects in the absorption mechanism and distribution of zinc.[5]

1. Absorption and Homeostasis

Proteins, especially of animal origin, and fat are the most important sources of absorbable zinc. The relatively high amounts of phytates (inositol pentakis and hexakisphosphates) in cereal products, legumes and nuts can significantly decrease blood levels of zinc in vegetarians because intestinal absorption of zinc occurs through a specific process that is diminished by concomitant ingestion of phytates, phosphates, iron and copper.[6]

Calcium can be a potentiating factor because it reacts with phytates, forming a precipitating complex salt. Zinc can easily bind to the precipitate. The molar ratio of phytic acid to zinc in cereal products ranges from 18 to 37. Because of this high phytate content, the bioavailability of zinc in wholemeal cereal products is low compared with foods of animal origin.[7] Protein intake is important in this respect since proteins provide amino acids, some which are able to desorb zinc from the precipitate and improve bioavailability.

Cellular zinc transport is mediated by an anion carrier, probably from the family of cytochrome oxidase II, the protein involved in the genesis of acrodermatitis enteropathica.

The body maintains zinc homeostasis by changes in absorption and excretion. In good nutritional status, body loss of zinc correlates proportionally with total dietary intake. After periods of low zinc intake, daily zinc losses are substantially lower than at higher intakes. The fractional absorption does not differ between people consuming marginal or adequate quantities of zinc, but endogenous zinc is effectively conserved by the intestine in people whose habitual dietary zinc intake is marginal. Diminished intake of zinc leads to increased efficiency of absorption and decreased faecal excretion.[8]

Nutritional habits in elite athletes during training and competition can be quite different from the recommended diet for the general population. Endurance athletes often adopt unusual diets in an attempt to enhance performance: an excessive increase in carbohydrates and low intake of proteins and fat may lead to suboptimal zinc intake in 90% of athletes.[9]

A low fat diet could compromise health and performance because essential fatty acids and zinc may be too low, and zinc is an important determinant of the intestinal absorption of lipid-soluble vitamins. Even marginal zinc deficiency causes a marked decrease in the intestinal absorption of lipids in general and lowers significantly the lymphatic absorption of vitamin A and vitamin E.[10]

2. Requirements

Kinetic studies have shown that in humans there are multiple pools of zinc that fluctuate from minutes to years. Zinc can bind to different compounds in different concentrations. By comparing kinetics in healthy and various disease states, the role of zinc in disease may be elucidated through the identification of differences in metabolic processes.[11] Absorption and excretion are regulated by genetics, diet, environment and disease.[11] It may be that the maintenance of homeostasis is guaranteed by special adaptation mechanisms developed during ontogenesis.

It is not easy to establish a minimum zinc requirement because the human organism seems highly tolerant towards partial deficiencies over a period of time, and laboratory diagnosis of marginal zinc deficiency can be problematic because there is no reliable single measure that can demonstrate suboptimal zinc status.[12]

Assessment of dietary zinc status requires several steps, consisting of the measurement of zinc content in foods after different preparation methods

and considering inhibition of absorption by other compounds in the diet, and seasonal variation in content and in intake. The apparent zinc intake shows large day-to-day variations within individual eating patterns, suggesting that a reliable estimation of overall zinc intake can only be made after a long term follow-up.

3. Zinc Deficiency and Its Consequences

During infancy, even a moderate zinc deficiency can seriously impair human health, performance, reproductive systems and mental and physical development. In particular, zinc deficiency has been shown to adversely affect brain growth, learning and activity.[13] The pathological signs of zinc deficiency are related to impaired functioning of plasma membrane proteins, lowered plasma membrane level of thiols and reduced production of enzymes necessary for RNA and DNA synthesis.[14]

Temporary zinc deficiency is not a rare event: high physical activity, stress and dietary habits may be involved in a complex of factors which creates suboptimal zinc uptake. If not corrected, this shortage can remain for a long time or worsen without any apparent reason or act as a sustaining factor for abnormal eating behaviour.

Zinc is a determinant at several levels for the correct integration of the taste system. It is important for the synthesis of gustin, a parotid metalloprotein secreted into saliva and identified as an isoenzyme of carbonic anhydrase considered responsible for the maintenance of taste acuity.[15] Gustin is also a trophic factor that promotes the growth and development of taste buds through its action on taste bud stem cells. A consequence of zinc deficiency is decrease in taste intensity (hypogeusia) and selectivity (dysgeusia), since zinc is involved in the neurotransmission of the electrical stimulus generated in the bud cells and ending in the central nervous system.[16]

Generally, hypozincaemic individuals have a poor appetite, do not enjoy eating and complain of food, particularly protein, as being disagreeable. Reduced food consumption is a major consequence of these alterations in taste, but subchronic low protein intake worsens zinc availability. Furthermore, carbohydrate-rich foods have lower zinc content and reduced absorption capacity because of their phytate content. A diet particularly rich in starch could be the best way to incur zinc deficiency.[17] Hypogeusia and loss of appetite exacerbate zinc deficiency and in the exercising female can result in menstrual cycle irregularities, amenorrhoea and osteoporosis. Anorexia nervosa, frequently found in young females, especially in athletes, has a number of symptoms in common with zinc deficiency: bodyweight loss, depression and amenorrhoea.[18]

This aspect could be a normal biological response to stressors: when stressful stimuli are excessive, a large number of animal species reduce food intake and express a progressive wasting syndrome associated with hypozincaemia and susceptibility to infections.[19]

4. Effects of Zinc on Physical Activity

There are limited data available on the relationship between performance and zinc status, but physical activity seems to correlate positively with blood zinc level, perhaps through regulation of taste acuity and protein intake.[20] Zinc and energy could also be connected via leptin, an adipocyte-derived hormone that reflects the amount of energy stored in adipose tissue.[21]

Leptin travels through the bloodstream, is transported across the blood-brain barrier and produces effects in the brain after binding to specific leptin receptors located in the hypothalamus. Leptin was found to change neuropeptide Y (NPY) levels in the hypothalamus. High levels of leptin, reflecting high or adequate levels of body fat, were found to downregulate hypothalamic NPY mRNA and NPY levels, which in turn suggests a decrease in appetite in response to the signal of adequate body energy reserves.[22]

Dysregulation of leptin during zinc deficiency has the potential to affect both central and peripheral physiology, in that leptin receptors have been identified within reproductive tissues of the body. Leptin regulates energy intake and expenditure and its cir-

culating levels are influenced in equal measure by caloric imbalance and plasma zinc levels.[21]

Some data on the relationship between zinc deficiency and fatigue were obtained from patients 3 to 45 days after surgery. Post-operative fatigue, a well documented syndrome, appears to correlate with decreased serum zinc level in patients.[23] Zinc values returned to pre-operative values around 45 days and so did muscular efficiency.

The reason is perhaps a derangement in electrolytes. When healthy young volunteers had their energy intake reduced to less than 1600 kcal per day for 6 months, they experienced soreness, muscle cramps and fatigue.[24] In muscle, and in most other organs, when protein content is markedly reduced, so are the levels of potassium, magnesium and zinc.[25] Acute zinc depletion, tested in males, did not affect peak muscular force but altered the total work capacity of skeletal muscle.[26]

Performance of strenuous physical activity increases oxygen demand by 10- to 15-fold compared with resting conditions. The resulting elevated mitochondrial oxygen consumption and electron transport flux produces an 'oxidative stress' that leads to the generation of reactive oxygen species (ROS) and lipid peroxidation. Free radicals are mediators of muscle inflammation and damage. Zinc has an important role in antioxidant cellular defences, being a structural element of the non-mitochondrial form of the enzyme superoxide dismutase (SOD). Many stresses (ultraviolet radiation, physical exercise, hypoxia) can rapidly redistribute zinc-containing SOD to damaged tissues, temporarily decreasing its plasma levels.[27]

The antioxidant properties of zinc have been linked partly to its role as an integral component of SOD, as a stabiliser of cell membranes, as a protector of the thiol groups of proteins against oxidation and as a competitor with copper and iron for binding to oxygen ligands, reducing the potential for hydroxyl radical ($OH^{\bullet}$) production from membrane phospholipids. In particular, training appears to upregulate the mRNA abundance of SOD in aerobic tissues such as liver, heart and the deep portion of vastus lateralis muscle.[28]

Unbalanced diet could be the main reason for the zinc deficiencies frequently found in athletes, although in certain cases strenuous exercise could contribute to the deficiency by increasing sweat loss and zinc redistribution between plasma and erythrocytes.[29] Significant decreases in the erythrocyte content of zinc represent the acute effects of prolonged exercise, whereas hepatic zinc increases after daily physical training and the change is more elevated at the end of a competitive season. These differences suggest that long term exercise may induce redistribution of zinc. In athletes studied at the beginning of the season there were no deficiencies of zinc, and plasma levels in sportsmen involved in anaerobic training were significantly higher compared to those undertaking aerobic activity.[30]

Urinary zinc excretion decreased during the first month of resistance training and then returned to baseline values in the next 4 weeks, suggesting an adaptation in mineral excretion in response to heavy training.[29]

In general, hypozincaemic athletes have a higher viscometric erythrocyte rigidity index.[31] Their power output during performance is lower and they have a higher increase in blood lactate during exercise, resulting in a lower lactate threshold.[32]

Strenuous exercise can also have an adverse effect on bone tissue [bodyweight, bone mineral content and bone mineral density (BMD)] and a consistent negative association can be observed between changes in BMD and urinary zinc excretion.[33]

Sazawal et al.[34] suggested that zinc deficiency may be an important cause of the lower motor activity levels associated with hyponutrition. There was a causal relationship between delayed linear growth and reduced motor activity in malnourished pre-school children. In this population, zinc supplementation can positively affect growth and motor activity, probably because zinc, particularly abundant in the brain, is important for both brain function and development.

5. Therapy

There is a minimal daily zinc intake that cannot be disregarded over a long period. When protein

and energy intakes meet the dietary reference values for sex and age, there is no zinc deficiency and differences observed in different physical conditions depend on modified distribution of intra- and extracellular pools.

Zinc is used in pharmacological doses to treat diseases such as acrodermatitis enteropathica, Wilson's disease and in cases of overt nutritional deficiency. Exogenous oral zinc treatment is also effective in patients with decreased levels of carbonic anhydrase and in cancer patients with taste alterations caused by head or neck irradiation.[35]

Zinc supplementation can be a therapeutic option in addition to nutritional approaches in many other conditions in which a pathological or iatrogenic condition can modify zinc disposability, such as gastrointestinal malabsorption, acute or chronic blood losses and in dialysed patients.

Development of methods to investigate zinc status and identify people at risk of zinc deficiency and to determine the need for initiation of zinc fortification of the food supply or zinc supplementation is needed. Before considering the appropriateness of zinc supplementation, it is important to assess the type of the deficiency (acute, subacute or chronic) and the degree (subclinical, mild or severe).

There is a widespread idea that mineral supplements can help athletes. This assumption originates from the belief that the athlete has a higher than normal requirement for minerals and that even a marginal deficiency can have a negative effect on performance. Similar to iron, dietary zinc supplements can improve athletic performance in individuals deficient in this element[36] and possibly in athletes who do not consume well-planned and varied diets. The quality of physical performance *per se* is not associated with the nature of the foods consumed, except in case of diets exclusively vegetarian, with a low protein and elevated phytate content.[37]

Reduction in dietary zinc beyond the capacity to maintain homeostasis leads to utilisation of zinc from rapidly turning over pools located in the bone and liver. Even partial depletion of these important zinc stores can lead to the rapid onset of both biochemical and clinical signs of zinc deficiency. In this case it can be useful to give athletes zinc supplements, preferably in form of a single soluble salt. Zinc acetate or zinc gluconate are better than zinc sulphate because they are less acidic and cause less gastric mucosal injury. If zinc supplements are used, it is important that they are not excessive, as excess zinc in the diet can result in a secondary copper deficiency.[38]

6. Conclusion

Zinc is a transition metal essential for plant and animal nutrition. It is contained to some extent in all vegetable material but it is not necessarily abundant in edible components. When Prasad[4] documented for the first time that the Iranian and Egyptian dwarfs were zinc deficient, it was commonly believed that zinc deficiency could never occur in humans. Today, there is a growing interest in micronutrient malnutrition, and endurance athletes are at risk of zinc deficiency if they have an unbalanced diet. Given the reality of nutrient-nutrient interactions, it seems to be time to give up the ancient concepts of deficiency and excess to a global perspective of 'balanced diet'.

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