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Zinc Homeostasis in Exercise: Implications for Physical Performance

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Editorial

Zinc is involved in numerous metabolic functions, including energy metabolism, immunity and antioxidant activity [1]. The majority of zinc is found within the musculoskeletal system as part of protein complexes. In addition to providing structural stability for proteins, zinc also acts as a cofactor for metalloenzymes, including lactate dehydrogenase (LDH), superoxide dismutase (SOD) and carbonic anhydrase (CA). At the muscle tissue level, exercise can disrupt cellular structures [2] which leads to the release of proteins and ions, such as zinc, from myocytes. In the initial stages of muscle repair, monocytes and leukocytes infiltrate muscle cells, initiating cytokine production and the subsequent inflammatory response [3]. Inflammatory cytokines have been shown to regulate the expression of cellular zinc transporters in a number of tissues and thereby alter zinc homeostasis [4]. In the present review we examine the interactions between exercise and zinc status in humans.

Acute Effects of Exercise on Zinc Metabolism

Zinc loss, in particular through sweat during exercise, is well documented [5,6]. The magnitude of zinc loss in sweat appears to be dependent on training status, duration of exercise and ambient temperature. In prolonged exercise, conservation of sweat zinc is evident after an hour of aerobic activity and this adaptation is enhanced further in heat-acclimatised individuals [7]. Similarly, the magnitude of urinary zinc excretion is confounded by the differences in exercise test conditions resulting in inconsistent reports for urinary zinc loss post exercise [8,9].

Conflicting results have also been reported for plasma zinc concentrations immediately after maximal physical exertion [8,10,11]. There appears to be negligible effects on plasma zinc immediately after submaximal exercise [12,13]. In exercise recovery, a decrease in plasma zinc concentrations is observed, especially in studies that report higher plasma zinc immediately after exercise [8,14]. The individual's training status is implicated in regulating zinc homeostasis during exercise. Endurance trained individuals, who have higher aerobic thresholds, have smaller fluctuations in serum zinc during exercise when compared to inactive individuals [9]. In inactive subjects, lower levels of zinc and CA-I in erythrocytes were found immediately after high intensity cycling, which returned to baseline values after 30 minutes of rest [15]. The concomitant reduction in plasma zinc suggests a shift of zinc from plasma to erythrocytes. Taken together, the redistribution of zinc between different compartments highlights the rapid flux of zinc when challenged by exercise.

A number of mechanisms have been proposed to account for the flux of zinc observed during exercise recovery, namely localised exercise-induced muscle inflammation and its sequel. In a study where ^{70}Zn was infused into subjects after a maximal aerobic exercise bout, zinc flux shifted from plasma into the interstitial fluid and the liver, possibly due to the acute phase response and/or changes in oncotic pressure with exercise [16]. The acute stress of exercise induces the production of cytokines, such as interleukin-6 (IL-6), which can sequester zinc in the liver through hepatic metallothionein (MT) and differential regulation of zinc transporters [17].

Effect of Chronic Exercise Training on Zinc Status

Additional zinc losses and transfer between body compartments as a result of repeated exercise bouts are hypothesised to compromise zinc status. In previously inactive individuals who were subjected to an aerobic training program, there was a decline in serum zinc concentration after several weeks of training [18]. In addition, Ohno et al. reported a reduction in the circulating pool of exchangeable zinc in men after a 10-week running program [19]. Collectively, these observations suggest an increased requirement of zinc in the presence of chronic exercise stress.

Longitudinal studies which followed athletes over a competitive season report contradictory changes to blood zinc concentrations [20,21]. However, the failure of some studies to assess dietary zinc intake during the study period limits the interpretation of the results. In cross-sectional studies, there appears to be no significant differences in plasma zinc levels between athletes and the general population [22]. High impact sports which result in increased level of muscle damage may lead to higher amounts of zinc released from muscle cells. Athletes in aerobic disciplines, such as triathletes or long distance runners, are more likely to display signs of zinc redistribution from plasma to erythrocytes when compared to their anaerobically-trained counterparts [23]. In addition, erythrocyte-SOD appears to be upregulated as a result of exercise adaptation. Correlations between erythrocyte-zinc, -MT and -SOD activity in elite athletes further emphasize the requirement for zinc in the development of antioxidative adaptation in erythrocytes [23].

Zinc and Exercise Performance

A number of zinc depletion studies have investigated the effect of zinc on measures of exercise performance in humans. In a randomised cross-over trial in men, low dietary zinc intake (3.8 mg/day for 9 weeks) was shown to impair cardiorespiratory function and lower levels of erythrocyte zinc and CA activity [24]. Muscle endurance of the shoulder complex and knee extensor have been shown to decline significantly after 33-40 days of zinc depletion [25]. Peak force, however, was unaffected by low dietary zinc intake. The authors attributed these effects to changes in lactic acid metabolism as a result of zinc depletion, possibly through reduced activity of LDH within the muscles. A decline in cardiovascular function and total work capacity of skeletal muscles as a result of acute zinc depletion further emphasise zinc's role in exercise performance.

Marginal zinc deficiency in athlete groups, induced by inadequate zinc intake and additional zinc loss, could contribute to early fatigue. Zinc supplementation has been shown to increase the count and deformability of erythrocytes, thereby improving blood rheology during exercise [26]. Although the effect of zinc on exercise performance is unclear, there appears to be a reduction in athletes' ratings of perceived exertion at submaximal intensities during zinc supplementation. Figure 1 summarises the potential interactions between exercise-induced metabolic stress and zinc homeostasis. Inflammation may serve as a key mediator by influencing cellular zinc transport. Following exercise, a cocktail of pro- and anti-inflammatory cytokines, such as IL-1 receptor antagonist, IL-6, IL-8 and IL-10, are elevated [27] and are capable of impacting zinc homeostasis by altering the expression of cellular zinc transporters [4].

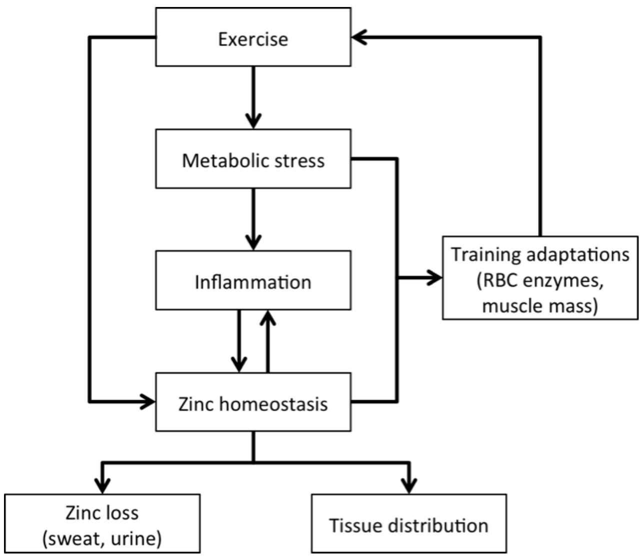

Figure 1: Potential relationships between exercise, inflammation and zinc homeostasis. Exercise-induced metabolic stress and inflammation can alter zinc homeostasis by promoting zinc loss and zinc redistribution to other tissue. This may be contributed by the link between inflammatory cytokines and regulation of cellular zinc transporters. In exercise training, metabolic stress and zinc homeostasis may affect exercise adaptations such as increased RBC enzymes and muscle mass.

Implications for Research and Practice

Although zinc status is implicated in exercise performance, further evidence is required to establish dietary zinc requirement for the athletic population. As such, the current recommendation for athletes and those who regularly participate in strenuous activities is to consume the level of dietary zinc proposed for the general population (14 mg/day for men; 8 mg/day for women) [28]. Zinc supplementation at levels below the Upper Limit may be appropriate for athletes who have suboptimal dietary zinc consumption despite strategies to incorporate additional zinc through diet. This is especially relevant for those on energy restricted or high carbohydrate diets, where bioavailable zinc may be insufficient. Further research is required to elucidate the mechanisms of zinc metabolism during exercise, and to consider specific challenges in measurements under exercise conditions, such as changes in blood volume [12].

Conclusion

Exercise has been shown to alter zinc metabolism and cause the redistribution of zinc within the body. Although the mechanisms of zinc homeostatic response during exercise are not clear, there is an indication of increased zinc requirement with strenuous activity due to additional zinc losses through sweat and urine. Failure to meet the requirement for zinc may contribute to suboptimal performance in some athletic population.

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