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# Vitamin and mineral supplementation and exercise performance

## Summary

Supplements of vitamins C and E have shown promise in reducing exercise-related symptoms (delayed muscle soreness) and biochemical indices of oxidative stress in both trained and untrained individuals. However, these antioxidant supplements appear to have no beneficial effect on performance and more research is needed to prove their long-term use is safe and effective. A prudent recommendation for athletes is to consume a diet rich in antioxidants. For female athletes, vegetarian athletes, and distance runners, daily consumption of foods rich in bioavailable iron together with periodic monitoring of iron status will minimize risk of iron deficiency. Iron supplementation is clearly indicated for cases of iron-deficiency anemia and may be beneficial in cases of low serum ferritin without anemia. The effect of magnesium (Mg) supplementation on exercise performance is equivocal. Overall, studies suggest Mg supplementation does not affect performance when serum Mg is within the range of normal values, but may improve performance when marginal or clinical Mg deficiency is present.

## Zusammenfassung

Vitamin-C- und -E-Supplemente scheinen sowohl bei Trainierten wie auch Untrainierten viel versprechend zu sein bezüglich der Reduktion von belastungsbedingten Symptomen («delayed muscle soreness») und biochemischen Indikatoren von oxidativem Stress. Diese antioxidativen Supplemente üben aber vermutlich keinen positiven Einfluss auf die Leistungsfähigkeit aus, und ihre Auswirkungen im Langzeiteinsatz müssen noch weiter erforscht werden. Eine sichere Empfehlung für Athletinnen und Athleten ist aber jedenfalls, auf eine Ernährung mit hohem Gehalt an antioxidativen Substanzen zu achten. Für weibliche oder vegetarische Athleten wie auch im Ausdauerbereich Sporttreibende wird die tägliche Zufuhr von Lebensmitteln, die reich an gut bioverfügbarem Eisen sind, und eine regelmässige Kontrolle des Eisenstatus das Risiko eines Eisenmangels minimieren. Eine Eisensupplementierung ist im Falle eines Eisenmangels angebracht und könnte auch in Fällen von tiefen Ferritingehalten ohne gleichzeitige Anämie von Vorteil sein. Der Einfluss von Magnesiumsupplementen auf die Leistungsfähigkeit ist nicht klar. Generell gesehen dürfte eine Supplementierung mit Magnesium die Leistung nicht beeinflussen, solange die Magnesiumgehalte im Serum im Normbereich sind. Bei marginalem oder klinischem Magnesiummangel hingegen dürfte eine Supplementierung eine Verbesserung der Leistungsfähigkeit nach sich ziehen.

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## Introduction

Vitamin, mineral and/or trace element supplements are beneficial if they supply a nutrient that is deficient in the diet. That is, when dietary intake is lower than the amount needed to provide maximum benefit as judged from all biological perspectives. It is difficult to accurately define nutrient «adequacy» in competitive athletes, for several reasons. First, requirements for vitamins and minerals vary: metabolic, environmental, and/or genetic factors can influence individual nutrient requirements. Second, physical activity and physical fitness are complex, involving multiple diverse components that are difficult to accurately and reliably measure. Third, a nutrient supplement that could improve performance by as little as 2–3% could provide a competitive edge; for example, reducing a 1500 m runner's time of 3 min 45 s by 6 s. In order to detect such small changes an intervention requires randomized, placebo-controlled, double-blind studies designed to maximize statistical power [1]. Inadequately designed studies may produce results that are unreliable and increase the likelihood a true effect will be missed. For these reasons, for many of the vitamins and minerals, it is difficult to argue forcefully for or against supplementation, and many competitive athletes choose to supplement.

However, there is general agreement that moderate physical activity does not adversely affect micronutrient status when diets pro-

vide recommended amounts of the vitamins, minerals and trace elements [2–5]. Moreover, most studies have found that, because athletes tend to consume higher amounts of food to balance increased energy needs, their diets contain the recommended dietary allowance (RDA) of the essential micronutrients [2–5, 6]. Exceptions to this general rule are athletes on weight-loss diets, those with restrictive diets, vegetarians, and those with eating disorders – dietary patterns often found in sports that promote unrealistically low body fat [7]. In such cases, a broad-spectrum vitamin/mineral supplement at the level of the RDA may be beneficial.

This short review focuses on several micronutrients – the vitamins E and C, iron, and magnesium – for which the available data, although incomplete, suggest supplementation may be beneficial for athletes. This review does not include a discussion of the electrolytes (Na, K, Cl), which are covered in a separate paper at this conference. For a more inclusive discussion of vitamin and mineral supplements in sport, the reader is referred to several detailed reviews [8–10].

## Vitamin E

Exercise increases the production of oxygen free radicals. Free radicals in muscle during or after exercise can arise from: 1) the mitochondrial respiratory chain; 2) the capillary endothelium;

and/or 3) inflammatory cells mobilized because of muscle damage. If production overwhelms the cellular antioxidant defense system, membrane lipid peroxidation occurs. This can disrupt the membrane bilayer and impair enzyme and/or receptor function. Strenuous exercise, particularly in unconditioned individuals, can produce oxidative damage and muscle injury. In vivo and in vitro animal and human studies have reported free radical generation during and after exercise [11, 12]. Prolonged submaximal exercise increases whole-body [13] and skeletal muscle [14] lipid peroxidation byproducts.

Multiple enzymatic and nonenzymatic cellular antioxidant defense systems reduce damage to membranes and other cell structures from free radicals, and aerobic exercise training strengthens these antioxidant defenses [15, 16]. Membrane-bound vitamin E is abundant in the inner mitochondria, the site of the electron transport system [17]. In rats, acute submaximal exercise reduces vitamin E concentrations in skeletal muscle and increases requirements for the vitamin [18]. In exercised rats, vitamin E deficiency increases susceptibility to oxidative damage of lysosomal membranes, and is associated with earlier exhaustion and a ~40% decline in endurance capacity [14].

Meydani et al. [19] reported that vitamin E supplementation (800 mg *all-rac*- $\alpha$ -tocopherol-d<sup>-1</sup> for 30 d) increased skeletal muscle  $\alpha$ -tocopherol by 53%. Vitamin E supplementation in humans reduces oxidative stress, lipid peroxidation and muscle soreness after exercise in some [20–23], but not all [24, 25] studies. Dillard et al. [20] gave subjects 1200 IU  $\alpha$ -tocopherol-d<sup>-1</sup> for 2 wk and found a significant reduction in expired pentane at rest and during exercise. Sumida et al. [21] reported that vitamin E decreased exercise-induced increases in circulating glutamic-oxaloacetic transaminase (aspartate transaminase),  $\beta$ -glucuronidase, and rate of lipid peroxidation, but because there was no placebo group, the results may have been at least partially due to an adaptation effect. Supplementation with 800 mg  $\alpha$ -tocopherol-d<sup>-1</sup> for 48 d reduced exercise-induced oxidative injury, as indicated by a sparing of muscle fatty acids, reduced muscle lipid-conjugated dienes and decreased excretion of urinary thiobarbituric acid adducts [22]. Rokitzki et al. [23] gave competitive cyclists 300 mg  $\alpha$ -tocopherol-d<sup>-1</sup> or placebo for 5 months and then tested their response to strenuous exercise. In the treatment group, the increase in serum malondialdehyde (MDA) and creatine kinase (an intramuscular protein released if membranes are damaged) was significantly reduced compared to placebo. In subjects given 300 mg vitamin E-d<sup>-1</sup> for 6 wk or placebo, the activity of muscle enzymes (e.g., creatine kinase and lactate dehydrogenase) in blood after strenuous exercise were similar [24]. Francis and Hoobler [25] gave subjects 600 mg vitamin E-d<sup>-1</sup> for 2 d before and 2 d after strenuous eccentric exercise, but found that muscle soreness was not reduced by supplementation. Although vitamin E supplementation may enhance performance at high altitude [26, 27], most well-controlled studies have not found an ergogenic effect of vitamin E supplementation, either on performance during standard exercise tests or cardiorespiratory fitness tests [28].

### Vitamin C

Water-soluble vitamin C in the muscle cytosol can serve as an electron donor to vitamin E radicals generated in membranes during oxidative stress [29]. In human subjects, supplementation with 400 mg ascorbic acid-d<sup>-1</sup> for 3 wk increased blood ascorbic acid concentrations but did not significantly reduce plasma MDA after a bench-stepping exercise [30]. However, using the same supplementation schedule and exercise protocol, Jakeman and Maxwell [31] reported the ascorbic acid group showed reduced strength loss in the triceps surae after exercise and faster recovery, suggesting vitamin C supplementation reduced muscle damage. Delayed-onset muscle soreness may be an indicator of muscle damage induced by exercise. Staton [32] gave men 200 mg ascorbic acid-d<sup>-1</sup> or placebo for 30 d, and then had them perform sit-up exercises to induce soreness. When they repeated the exercise 24 h

later, the supplemented group was able to perform significantly more sit-ups than the placebo group. Kaminski and Boal [33] gave subjects 3 g ascorbic acid-d<sup>-1</sup> or placebo for 3 d before eccentric exercise of the calf muscles. Compared to placebo, in half of the treated subjects there was a >33% reduction in soreness. However, well-controlled studies have found no benefit of ascorbic acid supplementation on either endurance or strength performance [34–38].

During aerobic training, adaptation to reduce oxidative damage may involve neutrophil monocyte accumulation in exercised muscle and secretion of cytokines, including IL-1, IL-1 $\beta$  and tumor necrosis factor (TNF) [39, 40]. In animal studies, IL-1 and TNF increase muscle proteolysis and release of amino acids [41]. In humans, circulating IL-1 increases acutely after eccentric exercise [42], and downhill running significantly increases IL-1 $\beta$  in the vastus lateralis [43]. Supplementation with vitamins C and E may improve adaptive response to training by modifying circulating and muscle cytokines [44]. After muscle-damaging exercise, supplementation with 1 g ascorbic acid given together with 400 mg *all-rac*- $\alpha$ -tocopherol has a greater effect on stimulating IL-1 $\beta$  and TNF- $\alpha$  than does each vitamin alone [44].

### Iron

Dietary iron (Fe) intake is marginal or inadequate in many females who engage in regular physical exercise [45–47]. Basal obligatory losses in adults are ~1 mg Fe-d<sup>-1</sup> and must be replaced by absorbed Fe to maintain balance. In many athletes, poor food choice and/or energy restriction to reduce body mass contributes to negative Fe balance [45]. The Fe density of a typical meat-containing Western diet is 6 mg Fe/1000 kcal. Heme Fe (from meat, fish and poultry) is an important dietary source as it is better absorbed (5–35%) than nonheme Fe (2–20%) [48]. Because of lower dietary Fe density and reduced Fe bioavailability from plant foods, vegetarian athletes are at greater risk for Fe deficiency [45].

Increased Fe turnover and Fe losses also contribute to negative Fe balance during aerobic training. Fe losses may be due to covert gastrointestinal blood loss, increased Fe losses in sweat and/or erythrocyte hemolysis within the foot due to impact during running [49–51]. In Fe-depleted rats, trained animals had higher erythrocyte Fe turnover than did non-exercise-trained animals [52]. In humans, Ehn et al. [53] found increased erythrocyte turnover in athletes and demonstrated that whole-body loss of Fe occurred ~20% faster in female athletes than in non-athletes.

The prevalence of Fe-deficiency anemia (IDA) in adults in Western countries (both athletes and non-athletes) is ~5–6%. The prevalence is higher in younger female athletes, because of needs for growth, menstrual losses, and, for some, energy restriction [54]. Fe depletion, indicated by a low serum ferritin concentration, is common among athletes, with estimates of 35–50% among male and female endurance athletes [54, 56–59]. The prevalence of IDA may be lower among elite athletes: Wijn et al. [55] measured Fe status in selected top-level athletes and found only 2% of male and 2.5% of female athletes were Fe-deficient anemic.

Depletion of the body's Fe stores decreases hemoglobin concentration and reduces oxygen transport capacity [45]. In addition, reduced levels of myoglobin, mitochondrial cytochromes and other Fe-containing proteins in muscle may further lower aerobic capacity [46]. During Fe depletion, these biochemical and physiological changes in muscle presage the decrease in hemoglobin concentration [46]. IDA impairs oxygen delivery to tissues and reduces VO<sub>2</sub>max, performance and endurance [45]. Increasing dietary Fe intake and/or Fe supplementation can improve performance [56]. Less clear are the effects of Fe supplementation on the athletic performance of those without reduced hemoglobin concentrations but with Fe depletion as evidenced by low serum ferritin. Limited evidence suggests Fe deficiency without anemia in women may reduce VO<sub>2</sub>max [60], and that supplementation may be beneficial [56]. Dietary changes, such as daily consumption of a single meat-containing meal, may help prevent decreases

in ferritin associated with exercise [61]. Although  $\geq 60$  mg supplemental Fe per day is recommended for treatment of IDA [62], moderate-level supplementation (40 mg Fe-d<sup>-1</sup> as ferrous sulfate) prevented a decrease in serum ferritin in competitive swimmers [63].

## Magnesium

Magnesium (Mg), a cofactor in >300 enzymatic reactions in cells, plays a fundamental role in energy metabolism [64]. Mg is essential to many reactions, including glycogen breakdown, fat oxidation, protein synthesis, and ATP synthesis, particularly important during physical activity. Mg also serves as a physiologic regulator of membrane stability and is important in neuromuscular and cardiovascular function [65].

Dietary Mg intakes of physically active individuals generally are satisfactory; most surveys have reported intakes  $\geq 70\%$  of the DRI [64, 65]. Comparing Mg intakes among physically active and age-matched control subjects in Finland, Fogelholm et al. [66] found greater Mg intakes among athletes than controls. Similarly, dietary Mg intake was higher among Nordic skiers than in their age- and sex-matched, nontraining counterparts [67]. These differences appeared to be due to the athletes' higher energy intake and the greater Mg density of their diet. Longitudinal dietary monitoring of competitive female swimmers during training also reported adequate dietary Mg intakes [68]. These studies do not suggest that physical activity per se increases risk of insufficient Mg intake.

In response to acute bouts of exercise, there is substantial redistribution of Mg within body compartments, as well as increased losses of Mg [69, 70]. During exercise, Mg shifts from the plasma into red blood cells, and urinary excretion of Mg significantly increases [69, 70]. Sweat losses of Mg also contribute: men exercising for 8 h on ergocycles lost 15–18 mg Mg-d<sup>-1</sup> in sweat that accounted for 4–5% of daily Mg intake and 10–15% of total Mg excretion [71]. Recently, Lukaski and Nielsen [72] examined the effects of dietary Mg restriction on physiologic responses during submaximal exercise. Peak oxygen uptake, total and cumulative net oxygen uptake, and peak heart rate increased during submaximal work when dietary Mg was restricted, suggesting Mg depletion adversely affected cardiovascular function [72].

Mg supplementation trials in athletes have produced mixed results [73–76]. A limitation to studies of Mg supplementation in athletes is the lack of a sensitive indicator of status. Serum Mg concentration, although commonly used to evaluate Mg status in surveys, is a relatively insensitive index of marginal Mg status [65]. In an anecdotal report, low serum Mg in a female tennis player with muscle spasms associated with prolonged outdoor exercise resolved with Mg supplementation (500 mg-d<sup>-1</sup>) [77]. Brilla and Haley [73] gave men participating in a 7-wk strength-training program and matched for quadriceps strength either a placebo or Mg supplements to obtain a total daily Mg intake of 8 mg/kg body mass. Total daily Mg intakes were 507 and 250 mg-d<sup>-1</sup> for the men receiving the Mg supplement and placebo, respectively. Peak knee-extension torque increased more in the Mg-supplemented than in the placebo-treated men [73]. Golf et al. [74] gave competitive rowers a Mg supplement (360 mg-d<sup>-1</sup>) for 4 wk and reported lower serum lactate concentrations and ~10% lower oxygen uptake during controlled submaximal exercise. In another supplementation trial, female athletes with marginally low plasma Mg received 360 mg-d<sup>-1</sup> as Mg aspartate or placebo for 3 wk [74]. Compared to placebo, the treated group had significantly reduced total serum creatine kinase and creatine kinase isoenzyme from skeletal muscle after training. Trained adults given 250 mg Mg-d<sup>-1</sup> showed improved cardiorespiratory function during a 30-min submaximal exercise test compared to placebo [75]. However, in a placebo-controlled trial, Mg supplementation (250 mg-d<sup>-1</sup>) of men in a 12-wk program of mainly aerobic or a combination of aerobic and anaerobic activities did not increase peak oxygen uptake, affect urinary Mg loss, or improve performance [76]. A review of 12 well-controlled studies of Mg supplementation in humans and

exercise performance concluded that the evidence is equivocal, regardless of whether the performance outcome was strength, anaerobic, or aerobic [78]. Overall, studies suggest Mg supplementation does not affect performance when serum Mg is within the range of normal values, but may improve performance when marginal or clinical Mg deficiency is present [79]. Trained subjects appear to benefit less than untrained subjects, and there is a lack of research in physically active females who may be at the highest risk for Mg deficiency [78].

## Practical consequences

- In both trained and untrained individuals, supplements of vitamins C and E may reduce exercise-related symptoms (delayed muscle soreness) and biochemical indices of oxidative stress.
- Supplements of vitamin C and E appear to have no beneficial effect on performance.
- More research is needed to prove long-term vitamin C and E supplementation is safe and effective.
- Athletes should consume a diet rich in antioxidants.
- Female athletes, vegetarian athletes, and distance runners should consume foods rich in bioavailable iron daily.
- Periodic monitoring of iron status and prompt treatment will minimize risk of iron deficiency.
- Iron supplementation is clearly indicated for cases of iron-deficiency anemia and may be beneficial in cases of low serum ferritin without anemia.
- The effect of magnesium supplementation on exercise performance is equivocal.
- Studies suggest magnesium supplementation does not affect performance when serum magnesium is within the range of normal values, but may improve performance when marginal or clinical magnesium deficiency is present.

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