Vitamin and Mineral Supplementation to Athletes

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Vitamin and mineral supplements are frequently used by competitive and recreational athletes. Dietary deficiencies of most vitamins are not very common among athletes except in those who restrict their food intake in order to maintain body weight. Vitamins most likely to be deficient in the diet are folate, B₁₂, and E. Biochemical evidence of vitamin deficiencies in some athletes have been reported for thiamine, riboflavin, and B₆. When the diet is deficient, vitamin supplements may improve performance but are not likely to be effective if the dietary intake is adequate. Some female athletes’ diets are low in calcium, iron, and zinc. Low calcium intake may reduce peak bone mass in young women. Iron deficiency may impair performance and needs to be corrected with an iron supplement. Zinc supplements that exceed the RDA interfere with the absorption of copper and lower HDL-cholesterol.

The use of vitamin and mineral supplements by athletes is widespread, especially in the United States. According to recent surveys of supplement use among athletes, more than 50% of elite women distance runners, nonelite women marathon runners and triathletes, and Ironman triathletes of both sexes consume vitamin and mineral supplements regularly (22, 32, 76, 109, 164). Thirty percent of nonelite men competing in marathons and triathlons report daily use of vitamin and mineral supplements (109, 164). Use of vitamin supplements is also prevalent among high school and college athletes—56% of males and 33% of females (118). Data from the National Health and Nutrition Examination Survey 1976–80 (NHANES II) indicate that one of every four women and one of every six men ages 25–44 take a vitamin and/or mineral supplement daily (89).

In competitive events where the difference between winning and losing is measured in seconds or even a fraction of a second, poor nutrition could have a negative effect. Many athletes take vitamin and mineral supplements to ensure adequate nutrient intake. Other athletes believe that certain vitamins or minerals have ergogenic properties that will improve performance. In this review we will examine whether athletes need additional vitamins and minerals. There are three primary reasons why supplementing the diet with specific vitamins and/or minerals might be beneficial to athletes: (a) The athlete’s diet is deficient in one or

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more of the vitamins and minerals. (b) Athletes have a greater need for a specific vitamin or mineral than the general population because they use or lose more of that nutrient. (c) The addition of certain vitamins or minerals improves performance. The evidence supporting or refuting each of these reasons will be examined first for vitamins and then minerals.

**Vitamins**

Vitamins are essential organic nutrients needed in very small quantities by the body. Thirteen nutrients have been officially recognized as vitamins. Four of these vitamins, A, D, E, and K, are fat soluble molecules and stored by the body with the fats. The remaining nine vitamins, those of the B complex and vitamin C, are water soluble. Because water soluble vitamins are distributed in the fluid compartments of the body, they are excreted on a daily basis.

Functions of vitamins include serving as coenzymes for energy producing reactions (thiamine, riboflavin, niacin, and pantothenic acid), protein metabolism (vitamin B₆), synthesis of new cells (folacin and vitamin B₁₂), serving as antioxidants and protecting the integrity of cell membranes (vitamin E), and helping to maintain blood calcium levels (vitamin D). Some of the vitamins, for example thiamine, riboflavin, and niacin, have essential roles in energy production. The exact role of some other vitamins (e.g., vitamin C) during exercise is not as well understood.

**Dietary Requirements and Vitamin Intake by Athletes**

The recommended dietary allowances (RDA) for individual vitamins are presented in Table 1. No RDA has been established for pantothenic acid or biotin at the present time. The RDAs for thiamine, riboflavin, and niacin are directly

**Table 1**

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related to energy intake. As energy intake increases, a greater intake of these vitamins is required. Estimated requirements for adults are 0.5 mg/1,000 kcal for thiamine, 0.6 mg/1,000 kcal for riboflavin, and 6.4 mg/1,000 kcal for niacin (45).

In the NHANES II survey of the U.S. population it was reported that the mean intake of thiamine by older adolescent and adult females falls below the RDA of 1.1 mg (18). Mean intake of preformed niacin by older adolescent girls also falls slightly below the RDA of 15 mg. Because niacin is formed from the amino acid tryptophan, low intakes of preformed niacin do not necessarily indicate poor niacin status, especially if protein intake is adequate. Most of the population surveyed in NHANES II (71% of the males, 90% of the females) had dietary intakes of vitamin B₆ that were below the 1980 RDA (71). Mean intake of vitamin C in the U.S. exceeds the RDA; however, more than 25% of the men and women consumed less than 2/3 of the RDA (18). Approximately 10% of the adult population consumed less than 2/3 RDA for vitamin A (18).

Numerous surveys of the nutrient intake of athletes competing in a variety of sports were published in the 1980s (10, 42, 64, 76, 100, 103, 143, 161, 164). In general, the vitamin intake of adult athletes was found to meet the RDA for most vitamins. There were some exceptions, however. The vitamin intake of many adolescent girls involved in ballet and gymnastics is low in comparison to the RDA (10, 90, 100). In particular, many girls had dietary intakes of vitamins E, B₆, and folate that were less than 2/3 of the RDA for these vitamins (10, 90). Welch and colleagues (161) found that the majority of college women athletes they studied were consuming diets deficient in vitamins B₆, B₁₂, and folate. In 1989 the RDA for folacin was lowered from 400 ug to 180 ug. Using the 1989 RDA, the proportion of female athletes with diets low in folacin is reduced considerably.

Another exception was wrestlers during the competitive season when they were trying to maintain lower body weight (143, 148). Steen and McKinney (148) reported that many college wrestlers have diets deficient in vitamins A and B₆. There is also some evidence of low vitamin B₆ intake in young male athletes involved in other sports as well (53). In a recent study of recreational triathletes, the majority of both men and women were found to have low intakes of vitamin E and folate (164). In the cases previously mentioned, a supplement containing the deficient vitamins might be beneficial in helping the individual athletes meet the RDA. However, the vast majority of athletes consume adequate quantities of each vitamin in foods to meet the RDA.

**Vitamin Needs During Exercise**

Biochemical indices are very useful as indicators of vitamin deficiencies. These include the direct measurement of vitamins in plasma or urine (e.g., nicotinic acid), their coenzymes (e.g., thiamin pyrophosphate), and the activation of enzymes by a vitamin coenzyme (e.g., erythrocyte glutathione reductase activation by FAD). Using biochemical indices of vitamin deficiency, Guilland and colleagues (53) found that 17% of the male athletes they examined were deficient in thiamine and 35% were deficient in vitamin B₆. Haralambie (56) reported that 44% of the male athletes he examined were riboflavin deficient and 39% were deficient in both thiamine and riboflavin.
Adequacy of the riboflavin requirement (0.6 mg/1,000 kcal) was examined in a group of young women exercising 20–50 minutes/day (7). Using an erythrocyte glutathione reductase activation coefficient (EGRAC) >1.25 as evidence of riboflavin deficiency, Belko and colleagues (7) found that physically active young women needed between 0.63 and 1.4 mg/1,000 kcal. When overweight women on a caloric restricted diet were examined, it was found that 0.8 mg riboflavin/1,000 kcal was not adequate during periods of exercise or nonexercise (8). Subsequent work with overweight women demonstrated that a riboflavin intake of 1.16 mg/1,000 kcal (1.4 mg/day) was adequate during a weight reduction program that included exercise (6).

Theoretically, an increased need for those B vitamins (thiamine, riboflavin, niacin, B₆, and pantothenic acid) involved in glycolysis, gluconeogenesis, and the aerobic metabolism of carbohydrates and fats might be expected during training. Since increases in energy expenditure are usually accompanied by increased energy intake, the average individual is likely to increase the intake of B vitamins as well. One method of determining whether vitamin intake meets the needs of the athlete is to feed him or her a fixed amount of the vitamin (e.g., riboflavin or vitamin B₆) and measure the amount of the vitamin excreted in the urine. If more of the vitamin is needed during training, a smaller percentage of the vitamin or its metabolite will be excreted.

Using this technique, Dreon and Butterfield (37) found that male distance runners excreted less 4-pyridoxic acid (4-PA), a vitamin B₆ metabolite, than sedentary men who consumed the same amount of B₆. However, following a methionine challenge test the runners increased their excretion of 4-PA while the sedentary men decreased 4-PA excretion. If the runners were deficient in vitamin B₆, they should have decreased their 4-PA excretion following the methionine challenge. Dreon and Butterfield (37) suggested that during exercise training extra pyridoxine (B₆) is retained by the body in a readily mobilized pool. Such a pool of pyridoxine could explain the increased 4-PA excretion in runners following methionine challenge as well as increased plasma pyridoxal phosphate (PLP) observed after distance runs (87).

The best indicators of vitamin C status are the serum ascorbic acid concentration and the leukocyte ascorbic acid content. Low levels of serum and leukocyte ascorbic acid are indicators of a biochemical deficiency. Approximately 30% of Yugoslavian adolescent boys had a biochemical deficiency of vitamin C (15, 151). On the other hand, Guilland and colleagues (53) found no evidence of low serum ascorbic acid in young male athletes. Training may increase ascorbic acid levels. With similar dietary intakes of vitamin C, serum ascorbic acid concentration was greater in trained runners than in sedentary men (44). Increased plasma and lymphocyte ascorbic acid concentrations were found immediately following long-distance runs (50). Gleeson and colleagues (50) hypothesized that ascorbic acid was released from the adrenal glands during exercise. The ascorbic acid content of the adrenal glands is reduced following exhaustive exercise (157).

In humans, the primary function of vitamin E is thought to be as an antioxidant. Vitamin E protects polyunsaturated fatty acids (PUFA) in cell membranes from being oxidized, a process known as lipid peroxidation (152). In the presence of a peroxyl free radical (LOO•), vitamin E will be oxidized instead of the PUFA. Tappel and Dillard (152) presented evidence that peroxidation of PUFA leads to
increased pentane production which can be measured in exhaled air. Elevated levels of pentane have been found in exhaled air during moderate exercise (36). Significant increases in plasma and red blood cell tocopherol (vitamin E) have been observed during heavy exercise (123). The source of the increased tocopherol is not known, but it could be released from the adipose tissue with fatty acids (123).

Animal models have also been used to study vitamin E and lipid peroxidation during exercise. Endurance training lowers the vitamin E content of muscular tissue in both vitamin E deficient and normal rats (1). Aikawa and colleagues (1) hypothesized that during exercise vitamin E is oxidized. Exhaustive exercise more than doubles the concentration of free radicals in muscle (32). Similar levels of free radicals and evidence of damage to mitochondria and sarcoplasmic reticulum were found in normal rats exercised to exhaustion and in sedentary vitamin E deficient rats (32). Although vitamin E deficient rats have less endurance than normal rats, the difference in performance could not be explained by differences in muscle mitochondrial activity (51).

**Vitamin Supplements and Performance**

_B Complex Vitamins._ Investigators of B vitamin supplementation in subjects who were already receiving adequate amounts of these vitamins in the diet have consistently found that additional vitamins had no beneficial effect on performance (75, 86, 101, 104, 126, 155, 159). However, there are two exceptions where improved performance was observed following supplementation. Early and Carlson (40) found that men receiving a supplement containing large quantities of most of the B vitamins for 1 week had less fatigue during training in a warm environment. Bonke (13) examined the effects of supplementation with large amounts of thiamine and vitamins B₆ and B₉ on shooting accuracy. Accuracy improved during the latter part of the 8-week experiment in the subjects receiving the supplement. Because all three vitamins are associated with nervous system functions, Bonke (13) suggested that the improvement was due to an improvement in motor control.

Supplementing the diet with B vitamins will be of greatest value when the diet is deficient in those vitamins. If an individual is deficient in one or more of the B vitamins, supplements will improve the biochemical status (53, 99, 156). Some studies have also reported that deficient subjects improved in performance following supplementation with one or more of the B vitamins (12, 156), while others found no improvement in performance (99, 151). In a group of Yugoslav schoolchildren, many of whom had biochemical deficiencies of riboflavin, B₆, and C, supplementing the diet with these vitamins improved performance on a physical work capacity test (15). A follow-up study from this same laboratory reported that supplementation with vitamin B₆ and riboflavin had no significant effect on physical work capacity of adolescent boys who were deficient in these vitamins (151). Restricting the intake of thiamine, riboflavin, B₆, and C to 1/3 or less of the RDA for 8 weeks in a group of young men produced significant reductions in the maximal oxygen uptake and anaerobic threshold (156). When these vitamins were added to the diet, performance improved significantly.

Large amounts of any vitamin should be used with caution. Although most of the water soluble B vitamins will be excreted, large quantities of niacin and
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Vitamin B<sub>6</sub> can have toxic side effects. Nicotinic acid (niacin) is a potent vasodilator and will cause flushing of the skin. Dosages of 1,000 mg or more will inhibit fatty acid mobilization during exercise (11, 17). The result is a greater depletion of the glycogen stores during the exercise bout than normal (11). Pernow and Saltin (122) demonstrated that if the muscle glycogen stores are already depleted, taking nicotinic acid prior to exercise will reduce endurance.

**Vitamin C.** Previous studies of the effect of vitamin C supplements on exercise have produced equivocal results. Significant reductions in energy expenditure and increases in mechanical efficiency during submaximal exercise have been found following an injection of 0.5 g vitamin C (146) or ingestion of 1.0 g/day for 5 days (66). Increased physical work capacity and lower heart rates were observed after 1 g/day ascorbic acid supplement for 2 weeks (68). In adolescent boys with low plasma ascorbic acid levels, a 70-mg supplement for 2 months significantly increased predicted maximal oxygen uptake (151). On the other hand, no significant differences in submaximal or maximal oxygen uptake were found when subjects consumed 2 g/day for 5 days or a single 250-mg vitamin C dose (4, 97). Vitamin C supplementation (1 g/day) during training had no significant effect on endurance, predicted maximal oxygen uptake, or anaerobic capacity (49, 74).

Howald and associates (68) found higher levels of catecholamine metabolite excretion following exercise, and lower blood glucose and higher free fatty acid concentrations during exercise, when subjects were taking ascorbic acid supplements. These investigators attributed the fat mobilization to increased catecholamine secretion.

Vitamin C supplements have been found to have no effect on muscular strength and endurance (73) or to decrease muscular strength (157). Because of the role ascorbic acid plays in collagen synthesis, the effects of supplements on muscle soreness and athletic injuries have also been investigated. Staton (147) found no significant difference in muscle soreness 24 hours after exhaustive muscle exercise between vitamin C supplemented and placebo groups. Gey and colleagues (49) found no difference in the frequency or duration of injuries between ascorbic acid and placebo groups.

Ascorbic acid’s potential to improve heat tolerance and reduce heat illness has attracted the attention of several investigators. No difference in the frequency of heat exhaustion was observed between men with restricted ascorbic acid intakes and men receiving 500 mg supplements (63). During 10 days of heat acclimatization, men receiving 250 and 500 mg of vitamin C had significantly lower rectal temperatures (an indication of a faster rate of acclimatization) than men who received a placebo (150). In a subsequent study, these same investigators found an inverse relationship between plasma ascorbic acid concentration and rectal temperature during heat exposure (79). Sweat rate was also inversely related to ascorbic acid concentration. A reduction in sweat rate would conserve body water and reduce the risk of dehydration.

**Fat Soluble Vitamins.** The presence of free radicals and evidence of damage to muscle cells in normal rats suggest that lipid peroxidation might be prevented by increasing the vitamin E content of the diet. Dillard and colleagues (36) found that vitamin E supplements for 2 weeks decreased pentane production during moderate exercise. The concentration of vitamin E in the blood more than doubled following supplementation. Because exercising untrained muscles is ac-
companied by the release of muscle enzymes (e.g., creatine kinase) into the blood, increasing the vitamin E content of the tissues might reduce enzyme leakage through the muscle cell membrane. However, no difference in the plasma concentrations of muscle enzymes was observed following exercise between vitamin E supplemented and placebo groups (62).

There is very little evidence that vitamin E supplements actually improve performance. Most of the studies that have examined vitamin E supplements and performance have used relatively large supplements, 400 mg alpha tocopherol or more per day, for several weeks. No significant improvements in maximal oxygen uptake (141, 158), swimming performance (85, 139, 140), postexercise plasma lactate (85, 141), or muscular strength and endurance (139, 140, 141) were found in vitamin E supplemented subjects compared to subjects receiving placebos. There appears to be some benefit to using vitamin E in hypoxic environments such as mountain climbing at high altitudes (up to 8,000 m). Vitamin E supplements (400 mg alpha tocopherol/day) given to mountain climbers prevented both a significant reduction in anaerobic threshold and a significant increase in pentane exhaled at 5,000 m (144).

Supplementing the diet with fat soluble vitamins involves some risk because these vitamins are stored by the body. Vitamins A and D are likely to produce toxic reactions if taken in large enough quantities. Vitamin A toxicity results from the consumption of large amounts of retinol, but not carotenoids. Fumich and Essig (47) reported a case of vitamin A toxicity in a young soccer player who had consumed more than 100,000 IU/day for 2 months. Vitamin D can be toxic when the intake is five times the RDA, 50 ug/day (45). Symptoms of toxicity are elevated blood calcium levels and deposition of calcium in soft tissues. Individuals who are exposed to the sun or who consume two or more glasses of milk daily should not take vitamin D supplements. There is little evidence at the present time that large doses of vitamin E produce any toxic side effects.

Minerals

Twenty-one minerals have been identified as being essential to the functioning of the human body. RDAs have been established for only seven minerals: calcium, phosphorus, magnesium, iron, zinc, iodine, and selenium. Previous research has focused on the needs of the exercising individual for the first five of these minerals. Although no RDA has been established for sodium and potassium, there has been considerable interest in the need to replace both minerals during exercise.

Calcium

Calcium is the mineral found in the largest quantity, making up 2% of the total body weight. Bone is the major reservoir for calcium and stores 99% of the total. Calcium is required for the formation of bone during growth, with the greatest need occurring during the adolescent growth spurt. Once growth has stopped, bone remodeling will continue. Maximal bone mineral content, and therefore calcium storage, is achieved at age 30 and will be greater in males than in females. Loss of bone mass begins in most women before the age of 40.

The remaining 1% of the calcium is distributed among a number of tissues and is involved in several important functions. Calcium ions (Ca$^{2+}$) stored in the
sarcoplasmic reticulum of muscle fibers are involved in the linkage of actin and myosin in the contractile process. The release of neurotransmitters at the synapse is stimulated by the influx of Ca\(^{2+}\) into the axon. Ca\(^{2+}\) found in the plasma is one of the catalysts needed for blood clotting.

**Dietary Requirements and Intake.** Calcium is required in the greatest quantity during the adolescent growth spurt. In the 1989 revision of the RDA for calcium, the recommended 1,200 mg/day for males and females ages 11–18 years was extended to include men and women 19–24 years as well (45). The RDA for children 1–10 years and men and women over 24 is 800 mg calcium/day. There is some evidence that women may need 1,500 mg/day after menopause if not treated with estrogen replacement therapy (61). Most women in the U.S. do not consume 800 mg calcium/day. The average calcium intake of women ages 25–30 and 45–74 years is less than 600 mg/day (18, 121). Average calcium intake for men ages 18–64 years is more than 800 mg/day (18).

Although most nutritional surveys of male and female athletes reported that the mean intake of calcium exceeded the RDA of 800 mg, many female athletes consumed less than the RDA. This was especially true of gymnasts and dancers (10, 90, 100), college athletes (161), and elite marathon runners (34). Using the new RDA of 1,200 mg for ages 19–24 years to reevaluate data on college athletes reported by Short and Short (143) suggests that many of the women athletes would fall below the new RDA for calcium. The proportion of athletes who consume calcium supplements has been reported to range from 7% of nonelite marathon runners (109) to 33% of elite women distance runners (22).

**Calcium Intake and Bone Mass.** Low calcium intake during growth could have an adverse effect on bone growth and peak bone mass. One study has reported that supplements containing calcium added to diets deficient in calcium increased the thickness of the cortical bone in the metacarpals and femur of children ages 2–6 years to a greater extent than in children not receiving calcium supplements (125). After linear growth has stopped, calcium intake during the early adult years may have an impact on the peak bone mass achieved by age 30. Young women who consumed less than 800 mg of calcium had lower vertebral and radial bone densities than women who consumed more than 800 mg (70). Men and women with a life history of low calcium intakes had smaller cortical bone widths than those with adequate dietary calcium (98). Several recent studies have reported that a high lifetime calcium intake is associated with a greater bone density of the radius in women (21, 55, 137). There may be a threshold of calcium intake above which no additional increase in bone density is observed. Using the current dietary intake, postmenopausal women with low calcium intakes (50% or less of the RDA) lost vertebral bone density while women who consumed 800 mg calcium or more per day did not lose bone density over 7 months (33).

The primary concern for achieving as high a peak bone mass as possible during early adulthood is the possibility that it could delay or even prevent non-traumatic fractures associated with osteoporosis from occurring later in life. Low bone mass increases the risk of fractures. An increased incidence of hip fractures was found among adults with a low lifetime calcium intake and smaller metacarpal bone widths (98). In two recent studies the relative risk of hip fracture in elderly adults was examined. In Chinese men and women, who have relatively low calcium intakes, the risk of hip fracture decreased with increased calcium
intake (84). Cooper and colleagues (24) found that British men with calcium intakes above 1,000 mg/day had a lower risk of hip fracture, but there was no relationship between calcium intake and risk of hip fracture in British women.

Use of calcium supplements to prevent bone loss in postmenopausal women is controversial. Calcium carbonate supplements (1,500 mg or more/day) have been found to reduce the loss of finger bone mass (127) and the risk of vertebral fracture (130) in postmenopausal women. On the other hand, calcium supplements ranging from 500 to 2,000 mg/day alone were not found to reduce the rate of bone loss in postmenopausal women (43, 110, 131). Although calcium supplements are not as effective as estrogen replacement therapy in preventing bone loss (131), the combination of a calcium supplement and estrogen may be beneficial during menopause. Ettinger and associates (43) found that combining a calcium supplement (1,000 mg/day) with 0.3 mg estrogen slightly increased bone mineral content. Calcium supplements are more likely to be beneficial if a woman is consuming less than the RDA.

**Phosphorus**

Next to calcium, phosphorus is the most abundant mineral found in the body. Most of the phosphorus (85%) is located in bone as the calcium salts. Phosphorus in muscle plays a critical role in the production of energy as part of the high energy phosphate compounds ATP and creatine phosphate, cyclic AMP, and the phosphate ions involved in glycolysis.

**Phosphorus Requirements and Intake.** The RDA for phosphorus is the same as that for calcium: 800 mg for men and women age 25 and older; 1,200 mg for ages 11 to 24 years; 800 mg for children ages 1 to 10 years. Mean phosphorus intake for adults under the age of 35 in the U.S. is greater than 1,500 mg for men and 900 mg for women (18). Most of the studies that have examined dietary phosphorus intake of athletes found adequate levels for both males and females (76, 108, 160). Approximately 15-23% of adolescent female dancers and gymnasts consumed less than 2/3 of the RDA for phosphorus (10, 90, 100).

Nutritional deficiencies of phosphorus are very rare. There is a recent report of low plasma phosphate concentrations (hypophosphatemia) in runners who collapsed during a half marathon (29). Although the cause of low plasma phosphate is unknown, rapid uptake by skeletal muscle to replenish creatine phosphate may be responsible (162). Normal or elevated plasma phosphate concentrations are observed in highly trained runners following exercise (30).

**Phosphate Supplements and Exercise.** When phosphate depletion has been observed, the concentrations of ATP and 2,3-diphosphoglycerate (2,3-DPG) in red blood cells were decreased. Unloading of oxygen from hemoglobin at the tissues is reduced when the concentration of 2,3-DPG in the red blood cell is low. Several recent studies have reported conflicting results on the effect of phosphate supplements on 2,3-DPG concentration during exercise and maximal oxygen uptake. Ingestion of 4 grams of phosphate/day for 3 days prior to exercise significantly increased resting serum phosphate and 2,3-DPG (16). Maximal oxygen uptake was significantly related to the 2,3-DPG concentration, which suggested that the phosphate supplement increased the unloading of oxygen at the tissues. Kreider and colleagues (80) found higher maximal oxygen uptakes after 4 g phosphate/day but a decrease in 2,3-DPG concentration. No change in 2,3-DPG or improvement in maximal oxygen uptake was observed after 5.7 g
phosphate/day; however, an increase in the arteriovenous oxygen difference was found (14). Duffy and Conlee (39) reported no significant effect of phosphate supplements on endurance, leg power, or oxygen uptake.

Cade et al. (16) suggest caution in the use of phosphate supplements because increased plasma phosphate stimulates parathyroid hormone secretion and excretion of phosphate in the urine. Parathyroid hormone secretion will also stimulate bone resorption. Normal plasma phosphate was restored without using supplemental phosphate in the hypophosphatemic runners who collapsed (29). Phosphate supplements are not recommended for use by athletes at the present time.

Magnesium

Magnesium is one of the most important intracellular minerals. It is an essential ion (Mg$^{2+}$) for many metabolic reactions including glycolysis, citric acid cycle, fatty acid oxidation, and amino acid metabolism. The human body contains approximately 300–400 mg magnesium per kg of body weight. Muscle contains about 25% of the total magnesium and more than 50% is found in bone (142).

**Magnesium Intake and Requirement.** The RDA for magnesium is 350 mg for men and 280 mg for women. Average intake of magnesium by women and men (ages 25–30 years) in the U.S. is 190 mg and 295 mg, respectively (116). Male and female athletes are reported to have mean magnesium intakes that meet the RDA (34, 76, 108, 160, 164). However, about 40% of adolescent dancers and gymnasts consumed less than 2/3 of the RDA for magnesium (10, 90).

**Magnesium and Exercise.** During prolonged exercise, serum magnesium concentration decreases (9, 88, 116, 128, 134) or does not change significantly (20). Erythrocyte magnesium has been reported to increase during exercise, which suggests there is a shift of the Mg$^{2+}$ from extracellular to intracellular fluids (20, 128). Uptake of Mg$^{2+}$ is thought to increase 2,3-DPG formation, which would facilitate the unloading of oxygen from the blood. Reduced erythrocyte magnesium concentration following a marathon has also been reported (88). It is possible that Mg$^{2+}$ is taken up by the muscle (153) or the adipose tissue (88) during exercise.

There has been some concern that low serum magnesium during exercise could be due to excessive loss through sweating. Sweat magnesium concentration has been reported to be as high as 4.5 mEq/L, but falls with progressive dehydration (26). Costill and associates (26) estimated that a 6% loss in body fluids produced a 1% decrease in total magnesium content.

Evidence that magnesium is beneficial to performance is limited at the present time. In trained athletes a significant relationship was observed between plasma magnesium concentration and maximal oxygen uptake (91). Rats fed diets low in magnesium have less endurance than rats fed a higher magnesium diet or mineral water with a high magnesium content (72). Studies are needed on low and high dietary magnesium levels in humans to determine whether magnesium enhances performance.

Sodium

Almost all of the 65 grams of sodium found in the body is located in the extracellular fluids, with very little inside the cells. The osmotically active sodium ions (Na$^{+}$) draw fluid from inside the cells to the extracellular fluid (ECF) and help
to maintain balance between intracellular fluid (ICF) and ECF volumes. If the concentration of Na⁺ in the ECF decreases, more fluid will move into the ICF, causing the cell to swell and the ECF to decrease. Sodium on the outside of nerve and muscle cells is important because it can generate an action potential by entering the cell. Sodium salts help maintain homeostasis by buffering acids such as lactic acid produced during exercise.

**Dietary Needs and Intake.** There is no RDA for sodium; however, the Food and Nutrition Board of the National Research Council has estimated the safe and adequate intake (ESAI) for sodium. For adolescents and adults the ESAI is 500–2,400 mg sodium/day (45). Since most of our sodium intake is in the form of salt (NaCl), this would be the equivalent of 1.3–6.1 g NaCl/day.

The average dietary intake of sodium for males in the U.S. is 2,922 mg/day, and for females it is 2,060 mg/day (18). However, this does not include salt that is added to food in cooking or at the table. It has been estimated that the total NaCl intake averages 10 g/day, approximately 4,000 mg Na (45). Mean intake of sodium from food by male and female distance runners and triathletes has been reported to range between 2,260 and 4,425 mg/day (76, 108, 160, 164).

**Exercise and Sodium Needs.** There has been some concern about excessive sodium loss through sweating when exercising in warm environments. Sweat sodium concentration averages 55–60 mEq/L (126–138 mg/dL) in unacclimatized persons (26, 114). If a person had a 3-liter sweat loss, the amount of sodium excreted would be about 4,000 mg. Excessive loss of fluid and sodium will stimulate the renin-angiotensin-aldosterone mechanism, resulting in sodium conservation by the kidneys and sweat glands and a more dilute sweat. During acclimatization to heat, sodium conservation by the kidneys becomes maximal in 3–5 days and by the sweat glands in 5–10 days (3). If the sodium intake is high (about 9,000 mg/day), the amount of sodium lost in the sweat and urine is high and increases during heat acclimatization instead of decreasing (2).

It has been recommended that when the sweat loss exceeds 5 liters per day, the sodium intake should be greater than the ESAI upper limit of 2,400 mg/day. Soldiers who consumed 2,300 mg Na/day had higher rectal temperatures and heart rates when exercising in the heat and were more prone to develop heat illness than men receiving 5,100–6,600 mg Na/day (154). The lower salt intake resulted in an inadequate water intake due to a depressed thirst mechanism. Increasing the sodium intake to 10 g or more per day produced no further improvement in heat tolerance (154). More recent studies have reported no differences in rectal temperature or heart rate during 60 to 90 minutes of exercise in the heat with lower (1,150–2,300 mg) sodium intakes (2, 58).

The addition of sodium to fluid replacement drinks appears to enhance recovery from exercise. When 4.5 g NaCl per liter was added to water consumed following exercise, fluid intake was greater and the rate of plasma volume restoration was faster than with water (113). Consumption of a carbohydrate-electrolyte drink containing 9.2 mM Na following exercise also resulted in greater fluid intake and increase in plasma volume than when water was consumed (19). Electrolyte drinks may also be beneficial in maintaining plasma volume during prolonged exercise in the heat (19).

Consumption of large quantities of water or dilute solutions containing little sodium may be detrimental in prolonged exercise such as ultramarathons. There are several reports of hyponatremia (low blood sodium) in ultramarathons, triathlons, and even a marathon lasting more than 5 hours (46, 65, 102, 111,
In some cases the competitors had consumed a large amount of fluid during the race but very little sodium. Prolonged hydration with water or dilute solutions may lower the plasma sodium concentration. In events lasting more than 4 hours, foods and fluids containing sodium should be consumed (65).

**Potassium**

The amount of potassium in an adult male is approximately 175 g. Potassium is found almost exclusively inside the cells. Its concentration in the intracellular water helps to maintain the balance between the ICF and ECF. Potassium is also involved in the conduction of nerve impulses and skeletal muscle contractions. Because very little potassium is in the ECF, the potassium concentration of plasma and sweat is much lower than for sodium. Most of the potassium excretion is through the kidneys and gastrointestinal tract.

**Dietary Intake and Need.** The estimated safe and adequate intake (ESAI) for potassium is 2,000–3,500 mg/day (45). Mean potassium intake in the U.S. is 3,087 mg/day for men and 1,851 mg/day for women (18). More than 50% of young women ages 18–24 years consume less than 2,000 mg/day. The potassium intake of most male and female athletes meets or exceeds the ESAI (76, 108, 143, 160, 164).

**Exercise in Hot Environments.** Acclimatization to the heat has little effect on the amount of potassium lost in the urine or sweat (2, 94). When aldosterone stimulates reabsorption of Na⁺ into the blood, potassium ions (K⁺) are secreted into the urine. Theoretically, acclimatized persons losing large amounts of sweat per day who are exercising or working in the heat would be in negative potassium balance if the dietary intake was low. Costill and colleagues (27) found that a potassium intake of 3,000 mg/day maintained a slightly positive potassium balance in subjects losing 3 L of sweat per day. Reducing potassium intake to less than 2,000 mg/day significantly reduces the amount of potassium lost in the urine but has little effect on sweat and fecal potassium loss (27, 95). Adding potassium supplements to a normal dietary intake increases the amount of urinary potassium but appears to have no effect on sweat potassium (83). Carbohydrate-electrolyte drinks that are high in potassium also increase the loss of potassium in the urine (107).

There is some evidence that young men training in the heat for several weeks develop a potassium deficiency since the amount of exchangeable $^{42}$K in the body is decreased (77, 78). Potassium deficiency may increase the risk of heat illness. Animals that are potassium deficient are more prone to exercise-induced heat stroke than animals with normal potassium stores (69). Malhotra et al. (95) observed no difference in exercise tolerance, heart rate, or core temperature in a hot environment after men had consumed a potassium deficient diet for 1 week. Because acclimatization does not reduce sweat potassium concentration, prolonged exposure to heat for several weeks could progressively deplete potassium stores if the dietary intake is low. However, the most likely cause of potassium depletion are excessive loss through the gastrointestinal tract from vomiting and diarrhea and through the kidney when diuretics are used.

**Iron**

Iron is the trace mineral with the greatest potential to directly influence performance during exercise. Only 3–5 g are normally found in the body. Approximately 2/3 of the iron is incorporated into hemoglobin molecules and 3–5% is in
myoglobin within skeletal and cardiac muscle. About 1% of the iron is distributed among a number of mitochondrial enzymes including the cytochromes. The remaining iron is stored as ferritin and hemosiderin. Men have approximately 1,000 mg and women 300 mg of iron stores.

Iron Requirements and Intake. The RDA for iron is 15 mg/day for adolescent and adult females until menopause. Young adolescent males have an RDA of 12 mg/day to meet their needs for growth and expanded blood volume (45). Older adolescent and adult males need 10 mg/day. Approximately 50% of all females over 11 years of age in the U.S. consume less than 10 mg iron/day (18). Low dietary iron intakes have been reported in some studies of female athletes (10, 34, 90, 109, 161, 164) but not in others (42, 76). Low dietary iron intakes by male athletes are extremely rare but have been reported in college wrestlers (148). Use of iron supplements is fairly common among athletes, especially distance runners and triathletes (22, 34, 109, 164).

Under normal circumstances only about 10% of the dietary iron is absorbed. The absorption of iron is affected by the form of iron in the diet and the presence of enhancing factors in the same meal. Heme iron found in meat, fish, and poultry is more highly absorbable than nonheme iron. It has recently been reported that many women distance runners have low heme iron intakes because they consume little meat (60, 96, 145). Reduced iron absorption may explain why many of these same women runners were iron depleted.

Biochemical Evidence of Iron Deficiency. Evidence of depleted iron stores includes absence of bone marrow iron and plasma ferritin concentrations of 12 ug/L or less. Low ferritin concentrations and depleted iron stores have been found in both men and women runners (5, 41, 106, 163). Poor iron status has also been observed in women athletes competing in many other sports (35, 59, 117, 132, 136, 138). Although the incidence of iron depletion in athletes appears to be high, it may not be any greater in athletes than in age-matched nonathletic controls, according to two recent studies (5, 132). Low plasma ferritin concentrations in some athletes may also be due to plasma volume expansion. However, Magnusson and colleagues (93) found that a 15% larger plasma volume could not explain a 78% reduction in serum ferritin in male distance runners.

Iron depletion in athletes may also be due to excessive iron loss through sweating and gastrointestinal blood loss. Sweat iron concentrations of 0.13–0.42 mg/L have been reported for males and females during exercise (82, 120). Iron loss through gastrointestinal bleeding has also been reported in distance runners (133, 149).

Iron Supplements and Performance. There is little question that iron supplements are beneficial in increasing hemoglobin concentration and physical work capacity and reducing blood lactate in persons with iron deficiency anemia (48, 115). However, only a small percentage of athletes have iron deficiency anemia (5, 132, 136). The question is whether iron supplements are beneficial to athletes or physically active persons who are not anemic. Supplements containing low doses of iron (18–50 mg) given to athletes who were not iron deficient did not significantly improve hemoglobin or iron status (25, 59, 119). When supplements containing larger dosages of iron (100 mg or more) were given to iron deficient athletes, significant improvements in hemoglobin and iron status were observed (23, 105, 124, 135, 138). Although iron supplements are not beneficial in improving iron status in persons with normal iron stores, they may
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prevent iron stores from becoming depleted during training. Women runners who took iron supplements had significantly higher serum ferritin concentrations than women runners who did not take supplements (5).

Iron supplements given to athletes who were not anemic did not increase maximal oxygen uptake (138). However, postexercise blood lactate was significantly lower in the subjects who received iron supplements. Several studies have shown an improvement in endurance when iron deficient rats receive iron (31). Recently, Rowland and colleagues (135) found that adolescent female runners who received a large iron supplement for 4 weeks had more endurance than runners who received a placebo. Improvements in performance following iron supplementation have not been observed in other studies (99, 103).

Use of iron supplements with more than 50 mg of iron by athletes who are not iron depleted is not advised because of the possibility of iron toxicity. Some individuals have a genetic disorder called hemochromatosis that causes them to absorb and store large amounts of iron, which can damage the liver. Intake of iron supplements may also interfere with the absorption of zinc. It is preferable to screen athletes for iron deficiency at the annual physical examination. Those athletes with poor iron status should be given iron supplements.

Zinc

Zinc is an important trace mineral in many metabolic pathways because it is an essential part of many key enzymes. Included among the enzymes containing zinc are carbonic anhydrase, alkaline phosphatase, alcohol dehydrogenase, and lactate dehydrogenase. Most of the zinc in the body is located in the muscles and bones. Zinc in blood is found primarily in erythrocytes, with much smaller amounts found in plasma.

Zinc Intake and Evidence of Deficiency. The RDA for zinc is 15 mg/day for men and 12 mg/day for women (45). Average daily intake is 16 mg for young men and 9.7 mg for young women (121). Zinc intake of some athletes is reported to be less than the RDA (10, 34, 90, 108, 164).

Low zinc intake may contribute to zinc depletion in some athletes. Plasma zinc concentration declines when dietary zinc is less than 4 mg/day (92). It is thought that plasma zinc is a part of the exchangeable zinc pool which can be mobilized during stress and is useful as an index of zinc deficiency (92). There are several reports that plasma zinc concentration is below the normal range (80–130 ug/dl) in many athletes. Dressendorfer and Sockolov (38) found that 23% of the men runners they examined had less than 65 ug zinc/dl serum. Haralambie (57) also found that 23% of the men and 43% of the women athletes examined had serum zinc concentrations below the normal range. Women marathon runners have plasma zinc concentrations that are near the lower limit of the normal range (34). Although no significant difference in plasma zinc was found between varsity athletes and nonathletes, both groups were at the lower end of the normal range (91).

Zinc Supplementation. Because zinc is a part of many enzymes, there is some concern that low plasma zinc concentrations could have a detrimental effect on exercise. There is some evidence that zinc supplements increase muscular endurance in both rats and humans (81, 129). However, the statistical analysis used in the rat study (129) is questionable. The increase in muscular endurance
in humans was significant only for isometric endurance and not for isokinetic endurance (81). The amount of zinc in the human supplement (135 mg/day) was nine times the RDA. Large quantities of zinc can inhibit the absorption of copper and result in a copper deficiency. Even supplements with less zinc (22.5 mg/day) will increase serum zinc in runners but also reduce serum copper (54).

It appears that zinc supplements containing large amounts of zinc (160 mg/day) lower the HDL-cholesterol concentration (67). Use of a smaller supplement (29 mg/day) did not lower HDL-cholesterol (28). More recent evidence suggests that zinc supplements may negate the positive effect of exercise on the HDL-cholesterol (52). Unless a zinc deficiency has been diagnosed, use of zinc supplements larger than the RDA (15 mg/day) is not advisable.

Summary

Vitamin and mineral supplement use by competitive and recreational athletes is very common in the U.S. Most athletes consume adequate amounts of vitamins in the diet and have little need for vitamin supplements. However, some adolescent and adult athletes have dietary intakes that are below the RDA for B₆, B₁₂, folate, and E. There is also some evidence of biochemical deficiency of thiamine, riboflavin, and B₆. When the vitamin intake is adequate, there is little evidence that vitamin supplements improve performance.

Although the diets of most male athletes contain enough calcium and iron to meet the RDA, some female athletes have low dietary intakes of one or both minerals. Evidence of a biochemical deficiency of iron is also common among women athletes. Female athletes who are iron depleted may need an iron supplement to correct the deficiency. In order to maximize peak bone mass, females under the age of 25 should increase their calcium intake to 1,200 mg/day. Inclusion of sodium in food or drink during recovery from exercise is beneficial in restoring plasma volume and fluid balance. Additional sodium is necessary in exercise bouts lasting more than 4 hours. Some athletes have zinc intakes that are below the RDA. Zinc supplements that exceed the RDA interfere with copper absorption and may lower HDL-cholesterol.

References


