REVIEW ARTICLE

Vitamin and Mineral Status: Effects on Physical Performance

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Public health recommendations encourage the selection of a balanced diet and increasing physical activity to foster health and well-being. Whereas the adverse effects of restricted intakes of protein, fat, and carbohydrate on physical performance are well known, there is limited information about the impact of low intakes of vitamins and minerals on the exercise capacity and performance of humans. Physically active people generally consume amounts of vitamins and minerals consistent with the recommendations for the general public. However, when intakes are less than recommendations, some noticeable functional impairments occur. Acute or short-term marginal deficiencies, identified by blood biochemical measures of vitamin B status, had no impacts on performance measures. Severe deprivation of folate and vitamin B12 result in anemia and reduce endurance work performance. Evidence of vitamin A and E deficiencies in athletic individuals is lacking apparently because body storage is appreciable. In contrast to vitamins, marginal mineral deficiencies impair performance. Iron deficiency, with or without anemia, impairs muscle function and limits work capacity. Magnesium deprivation increases oxygen requirements to complete submaximal exercise and reduces endurance performance. Use of vitamin and mineral supplements does not improve measures of performance in people consuming adequate diets. Young girls and individuals participating in activities with weight classifications or aesthetic components are prone to nutrient deficiencies because they restrict food intake and specific micronutrient-rich foods. This information will be useful to professionals who counsel physically active people and scientific groups who make dietary recommendations to improve health and optimize genetic potential. *Nutrition* 2004;20: 632–644. ©Elsevier Inc. 2004

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INTRODUCTION

Individuals who seek to boost physical performance rely on proper diet and increased training. The growing awareness of the synergy between diet and physical activity has fueled an expanding interest in the valuable role that micronutrient nutrition can play in achieving one's genetic potential in physical performance.

Recreational and competitive athletes seek reliable and practical information describing the role that micronutrients play in fostering performance because of dubious perceptions and misinformation in the public press.[1] One impression is that most physically active individuals, as compared with their less active counterparts, fail to consume a diet that contains adequate amounts of vitamins and minerals, which leads to marginal nutrient deficiency and results in substandard training and impaired performance. Another perception is that physical activity promotes excessive losses of micronutrients because of increased catabolism and excretion. These opinions fuel the controversy for the use of vitamin and mineral supplements to improve physical performance, although scientific evidence to support the generalized use of nutritional supplements to augment work performance is lacking.[2–4]

Micronutrients differ from macronutrients in key characteristics. Water, proteins, carbohydrates, and fat are consumed in large amounts (>100 g/d), whereas vitamins and minerals are ingested in much smaller amounts (milligrams to micrograms per day). These differences in magnitude reflect turnover rates in the body and specific functions. Macronutrients provide sources of energy needed to fuel the body, maintain cellular hydration, and provide the body structure to perform work. Micronutrients enable the use of macronutrients for all physiologic processes. Despite their relative paucity in the diet and the body, vitamins and minerals are key regulators of health and function, including work performance.

This review describes the effects of impaired micronutrient status on human physiologic function during exercise and physical performance. It provides an operational model that integrates nutrient intake, metabolism, and status with measures of physical performance. The presentation summarizes the biological functions of vitamins and minerals, identifies specific biochemical measures of micronutrient nutritional status, describes intakes commensurate with optimal function, details physiologic impairments associated with micronutrient deficiencies, describes maximal intakes for health, and evaluates claims that micronutrient supplements improve physical performance.

MICRONUTRIENTS

Vitamins

Organic compounds, found in small amounts in foods, are designated as nutrients because they cannot be synthesized by the body

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TABLE I.

BIOLOGICAL FUNCTIONS OF SOME VITAMINS WITH REFERENCE TO EXERCISE

	Function	Deficiency sign or symptom
Water soluble		
Thiamin (B1)	Carbohydrate and amino acid metabolism	Weakness, decreased endurance, muscle wasting, weight loss
Riboflavin (B2)	Oxidative metabolism, electron transport system	Altered skin and mucous membrane and nervous system function
Niacin	Oxidative metabolism, electron transport system	Irritability, diarrhea
Pyridoxine (B6)	Gluconeogenesis	Dermatitis, convulsions
Cyanocobalamin (B12)	Hemoglobin formation	Anemia, neurologic symptoms
Folic acid	Hemoglobin and nucleic acid formation	Anemia, fatigue
Ascorbic acid (vitamin C)	Antioxidant	Fatigue, loss of appetite
Fat soluble		
Retinol (vitamin A)	Antioxidant	Appetite loss, prone to infections
Tocopherol (vitamin E)	Antioxidant	Nerve and muscle damage

and are required to support health and well being. Vitamins catalyze numerous biochemical reactions. They are not direct sources of energy; vitamins facilitate energy metabolism. Because the rates or activities of these metabolic processes increase during physical activity, an adequate supply of vitamins is needed to promote optimal physical performance. Vitamins are classified based on their solubility in water or fat.

Water-Soluble Vitamins

The B vitamins and vitamin C (ascorbic acid) are water-soluble vitamins (Table I). The B vitamins (thiamin, riboflavin, niacin, pyridoxine, folate, biotin, pantothenic acid, and choline) regulate energy metabolism by modulating the synthesis and degradation of carbohydrate, fat, protein, and bioactive compounds.[5] Vitamin B12 is required for hemoglobin synthesis and vitamin C acts as an antioxidant.[6]

Fat-Soluble Vitamins

Vitamins A, D, E, and K are stored in adipose tissue in substantial amounts.[6,7] These fat-soluble vitamins have no direct role in energy metabolism; they function in roles supportive of energy use. β-Carotene, a precursor of vitamin A, and vitamin E act as antioxidants in reducing muscle damage and enhancing recovery from exercise.[8] Vitamin D, which promotes calcium absorption and use in bone formation, and vitamin K, which functions in coagulation and bone formation, have not been shown to influence exercise performance.[9]

Minerals

Twelve minerals are designated as essential nutrients.[7] Magnesium, iron, zinc, copper, and chromium, whose essentiality is controversial, have biochemical functions with the potential to affect physical performance (Table II). These minerals serve as structural or catalytic components of enzymes and regulate cellular energy transduction, gas transport, antioxidant defense, membrane receptor functions, second-messenger systems, and integration of physiologic systems. Thus, mineral elements regulate macronutrient use.

TABLE II.

EXERCISE-RELATED FUNCTIONS OF SELECTED MINERALS

	Functions	Deficiency sign or symptom
Magnesium	Energy metabolism, nerve conduction, muscle contraction	Muscle weakness, nausea, irritability
Iron	Hemoglobin synthesis	Anemia, cognitive impairment, immune abnormalities
Zinc	Nucleic acid synthesis, glycolysis, carbon dioxide removal	Loss of appetite, growth retardation, immune abnormalities
Chromium	Glucose metabolism	Glucose intolerance

Dietary Reference Intakes

The dietary reference intakes are a set of reference values for intakes of nutrients for dietary assessment and planning. The goal of dietary reference intakes is to reach specific levels of nutritional adequacy.[7] The recommended dietary allowance (RDA) is the intake amount that is sufficient to meet the nutrient requirement for nearly all healthy individuals in a group. The RDA is used to guide healthy individuals to achieve adequate nutrient intake and represents a goal for average intake over time. The RDA is expressed as a single value set separately for each sex and specific age group. If an individual meets or exceeds the RDA for a nutrient, there is good assurance that the intake is adequate. If the intake is less than the RDA, it can be inferred that there is increased likelihood that intake is inadequate. The magnitude of the discrepancy from the RDA and the duration of the nutritional deficit determine whether adverse effects on health and well being occur.

The adequate intake is a value based on observed or experimentally determined approximations of nutrient intake by a group or groups of healthy people. The adequate intake is used only when an RDA can not be determined.

The tolerable upper intake level is the highest daily nutrient intake, food plus supplements, that is likely to pose no adverse health effects to almost all individuals. As intake increases above the tolerable upper intake level, the risk of adverse effects increases. At intakes less than the tolerable upper intake level, risk of adverse effects is minimal.

EVALUATING EFFECTS OF DIET ON PERFORMANCE

Faulty experimental designs hamper the determination of an effect of nutrient intake on performance. Failure to account for total intake, diet plus supplements, when evaluating performance is a common limitation of many studies. Changes in intake may induce homeostatic adaptations in absorption and use that redistribute nutrients without any effect on performance. Failure to assess biochemical indicators of nutritional status also limits any conclusions about influence of intake on biological function. A contemporary model that includes these key components and emphasizes

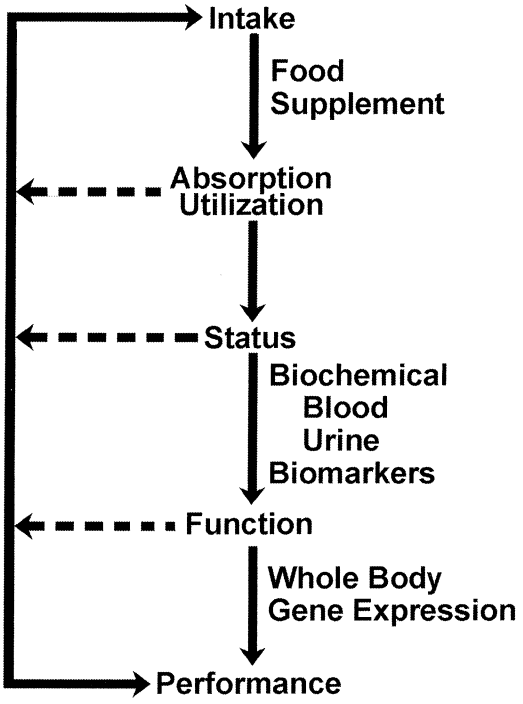

FIG. 1. Model to assess the interactions among nutrient intake, status, function, and performance.

feedback regulation among these components is presented (Figure 1). Future use of this model will enable rigorous research designs to test hypotheses relating nutrient intake, nutritional status, and human performance.

VITAMINS AND PERFORMANCE

Descriptions of the interactions between vitamin metabolism and physical performance are very limited because study designs have not been as comprehensive as necessary to form reasonable conclusions. Assessments of vitamin intake, biochemical measures of vitamin status, and determinations of physical performance are required. Very few studies have provided such information.[9]

This section describes the effects of graded intakes of some vitamins (thiamin, riboflavin, niacin, pyridoxine, folate, cobalamin, ascorbic acid, and vitamins A and E) on physical activity. Other vitamins (biotin, pantothenic acid, choline, and vitamins D and K) are not discussed because of the lack of information relating their intake with performance.

WATER-SOLUBLE VITAMINS

Vitamin B1 (Thiamin)

Thiamin plays a key role in carbohydrate and protein metabolism. As the compound thiamin pyrophosphate, it acts as a coenzyme in the conversion of pyruvate to acetyl coenzyme A and α-ketoglutarate to succinyl coenzyme A and participates in the decarboxylation of branched-chain amino acids. Thus, thiamin may be a potentially limiting nutrient in physical performance.

The suggested intakes for thiamin are 1.1 and 1.2 mg/d for women and men, respectively (Table III). Because the thiamin requirement depends on energy intake, the suggested RDA is 0.5 mg/1000 kcal.[5] Physically active individuals who consume large energy intakes should increase thiamin intakes proportionally. Studies of different groups of athletes have found adequate thiamin intakes.[10]

Thiamin intake, however, may be a problem for certain groups of athletes. Individuals who consume low-energy diets for participation in competitions that feature weight classes or physical appearance may have less than adequate thiamin intakes. Also, athletes who consume diets that emphasize low nutrient-dense foods (i.e., added-sugar beverages, candy, etc.) may be prone to low thiamin intakes. Athletes with low dietary thiamin include competitive female gymnasts[11,12] and collegiate wrestlers.[13,14]

Controlled human studies have found no significant effects of graded dietary thiamin intakes on physiologic function. Young men who consumed diets containing 0.23, 0.33, 0.53, and 0.63 mg of thiamin per 1000 kcal for 12-wk periods had no change in cardiovascular function, intermediary metabolism, muscle

TABLE III.

DIETARY REFERENCE INTAKES OF VITAMINS: RECOMMENDED INTAKES FOR INDIVIDUALS[5–7]*

	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin† (mg/d)	Vitamin B6 (mg/d)	Folate‡ (μg/d)	Vitamin B12 (μg/d)	Vitamin C (mg/d)	Vitamin A§ (μg/d)	Vitamin E (mg/d)
Females									
14–18 y	1.0	1.0	14	1.2	400	2.4	65	700	15
19–30 y	1.1	1.1	14	1.3	400	2.4	75	700	15
31–50 y	1.1	1.1	14	1.3	400	2.4	75	700	15
Males									
14–18 y	1.2	1.3	16	1.3	400	2.4	75	900	15
19–30 y	1.2	1.3	16	1.3	400	2.4	90	900	15
31–50 y	1.2	1.3	16	1.3	400	2.4	90	900	15
UL	NA	NA	35	100	1000‖	NA	2000	3000	1000

* Values are presented as recommended dietary allowances.
† As niacin equivalents; 1 mg of niacin = 60 mg of tryptophan.
‡ As dietary folate equivalents; 1 dietary folate equivalent = 1 μg of food folate = 0.6 μg of folic acid from fortified foods or as a supplement consumed with food = 0.5 μg of a supplement taken on an empty stomach.
§ As retinol activity equivalents; 1 retinol activity equivalent = 1 μg of retinol = 12 μg of β-carotene = 24 μg of α-carotene.
‖ From fortified foods or as a supplement.
NA, not available; UL, tolerable upper limit.

strength, or performance during treadmill exercise.[15] Urinary thiamin excretion, expressed as a percentage of total daily intake, decreased when dietary thiamin was reduced. In a double-blind study of thiamin depletion, young men fed 0.5 and 5 mg of thiamin for 4- to 5-wk periods in a crossover design experienced no change in endurance capacity or nerve conduction velocity in the presence of biochemical evidence of thiamin depletion (decreased urinary thiamin and erythrocyte transketolase activity and increased thiamin pyrophosphate).[16] Although short-duration thiamin restriction does not adversely affect physical performance, brief thiamin insufficiency can cause pyruvate accumulation and increase circulating lactate during work, which may promote fatigue, impair training, and thus reduce performance.[17]

Vitamin B2 (Riboflavin)

Riboflavin functions in the mitochondrial electron transport system as the coenzymes flavin mononucleotide and flavin adenine dinucleotide. Thus, riboflavin is required for oxidative energy production. Because riboflavin is found in a variety of foods, including dairy products, meat, vegetables, and cereals, riboflavin deficiency is uncommon in Western countries. However, there is evidence that riboflavin deficiency may be more common in athletes for whom food availability is limited.[17]

Recommendations for riboflavin intake are based on energy intake. It is suggested that an intake of 0.6 mg/1000 kcal will meet the needs of most healthy adults.[5] The current RDAs are 1.1 and 1.3 mg/d for women and men, respectively.[5]

Surveys have shown that most athletes consume adequate amounts of riboflavin.[18] Athletes with concerns about body weight may be at risk of riboflavin depletion. Female collegiate gymnasts have low self-reported dietary riboflavin.[19]

Biochemical assessment of riboflavin status involves the measurement of the activity of glutathione reductase, for which flavin adenine dinucleotide is a cofactor. Determination of glutathione reductase activity in erythrocytes (glutathione reductase receptor) is a reliable assessment tool for riboflavin status in humans. An activity coefficient (glutathione reductase receptor activity coefficient), or the ratio of enzyme activity with added flavin adenine dinucleotide to that without flavin adenine dinucleotide, is used to monitor the riboflavin nutritional status of humans.

Initiation of increased physical activity has an adverse effect on riboflavin status. Sedentary young women consuming 2.15 mg/d of riboflavin experienced a significant increase in the glutathione reductase receptor activity coefficient (e.g., decreased riboflavin status) within 4 d of starting an endurance exercise program with a concomitant increase in urinary riboflavin excretion.[20] Women, ages 50 to 67 y, fed low (90 μg/1000 kcal) versus adequate (600 μg/1000 kcal) dietary riboflavin had decreased riboflavin status (significantly increased glutathione reductase receptor activity coefficient and decreased urinary riboflavin) after 4 wk of endurance training.[21] Differences in dietary riboflavin, however, did not affect the magnitude of the gains in peak oxygen uptake and anaerobic threshold after training. Men with mild riboflavin depletion had a significant increase in glutathione reductase receptor activity coefficient and a decrease in urinary riboflavin with the start of physical training, but no change in the energy cost of treadmill walking.[22] Thus, biochemical evidence of altered riboflavin status reflects changes in riboflavin retention or use in response to exercise training without adverse effects on physical performance. These findings suggest that functional consequences of decreased riboflavin nutriture likely occur after decreases in biochemical indices of riboflavin status.

Niacin

Niacin refers to two compounds, nicotinic acid and nicotinamide. Nicotinamide is a precursor of nicotinamide adenine nucleotide and nicotinamide adenine dinucleotide phosphate, which serve as electron and proton acceptors, respectively. Nicotinamide adenine nucleotide is an electron carrier in many oxidative reactions, and reduced nicotinamide adenine dinucleotide phosphate is a hydrogen donor in the pentose phosphate shunt.

Niacin intake by various groups of athletes has been shown to be adequate.[18] Athletes who generally restrict food intake have decreased niacin intakes.[18]

Biochemical assessment of niacin status is hampered by the lack of a reliable marker. The most reliable and sensitive measures of niacin status are urinary excretion of two methylated metabolites, N^1-methyl-nicotinamide and N^1-methyl-2-pyridone-5-carboxamide. No reliable data for comparisons of niacin nutriture between the two groups of athletes or between the control and athletic groups are available.

Assessments of the role of niacin or nicotinic acid on metabolic responses during acute exercise or physical training provide limited information. The rationale to evaluate the role of niacin on performance is based on the potentially adverse effect of niacin on fatty acid use and its indirect effect on accelerating depletion of glycogen stores and performance measures.[9] Heath et al.[23] used pharmacologic doses of nicotinic acid (1 g before submaximal exercise and then 3 g/d for 21 d). Compared with tests without nicotinic acid supplements, acute ingestion of 1 g of nicotinic acid decreased circulating free fatty acid concentration and increased the respiratory exchange ratio, which suggest a reduction in fat use. The effect on respiratory exchange ratio was diminished after 11 d of treatment, which implies that an adaptation to nicotinic acid supplementation had occurred.

Murray et al.[24] determined the effect of supplemental nicotinic acid (330 and 521 mg for women and men, respectively) and a glucose-electrolyte beverage on performance. It was hypothesized that the mixture of nicotinic acid and glucose would reduce free fatty acid concentrations and increase dependence on glycogen stores and glucose from the beverage, which hypothetically might attenuate the use of muscle glycogen and increase performance. The supplemental nicotinic acid diminished the anticipated increase in free fatty acid concentration. Performance in a time trial, however, was not affected by the carbohydrate-electrolyte beverage with nicotinic acid as compared with placebo.

Vitamin B6

Vitamin B6 refers to all biologically active forms of vitamin B6. The forms include pyridoxine, pyridoxal, pyridoxamine, pyridoxine phosphate, pyridoxal phosphate, and pyridoxamine phosphate.[25] Pyridoxine, pyridoxal and pyridoxamine are the forms most common in foods. Vitamin B6 in the form of pyridoxal phosphate acts as a cofactor for transferases, transaminases, decarboxylases, and cleavage enzymes used in transformations of amino acids. During exercise, pyridoxal phosphate is needed for gluconeogenesis and for glycogenolysis in which it serves as a cofactor for glycogen phosphorylase.

The RDAs for vitamin B6 are 1.5 and 1.7 mg/d for women and men, respectively.[5] Male and female athletes generally consume adequate amounts of this vitamin.[26] When athletes fail to ingest adequate vitamin B6, it is explained by low-energy intakes and poor food choices.[26]

Few studies have examined the vitamin B6 status of physically active people. Three surveys reported that 40% to 60% of athletes have reduced vitamin B6 based on the enzyme stimulation test.[27–29] Only one study[27] reported more than one indicator of B6 nutriture, and it found less (17% to 35%) deficiency among the athletes than when only one indicator of status was used, indicating the need to use independent biochemical measures of B6 nutritional status and dietary B6 intakes to assess the interaction between diet and performance.

Coburn et al.[30] examined the influence of dietary B6 on circulating vitamers of B6 and muscle B6 in men. The men received a

B6-depletion diet (1.8 μM of pyridoxine HCl) for 6 wk followed by a self-selected diet supplemented with 980 μM/d of pyridoxine HCl for 6 wk. Depletion resulted in a significant decrease in plasma pyridoxal and pyridoxal phosphate concentrations and a significant increase in the enzyme stimulation test. Dietary B6 did not affect muscle pyridoxal phosphate. Despite alterations in dietary B6, skeletal muscle concentrations of B6 are recalcitrant to depletion and plasma B6 concentrations are a rapidly responding pool that may not reflect tissue stores. Unfortunately, dynamic muscle function was not assessed.

Folate

Folate serves as a coenzyme in single-carbon transfers in the metabolism of nucleic and amino acids. It is required for synthesis of purines and pyrimidines that are needed for DNA production and erythropoiesis. A deficiency of folate causes abnormal cell replication, particularly in the erythropoietic system, and results in megaloblastic anemia. This type of anemia also is caused by vitamin B12 deficiency. Supplemental folate in the presence of B12 deficiency can correct megaloblastic anemia but not the B12 deficiency.[5]

The RDA for folate is 400 μg/d for women and men; it increases to 600 and 500 μg/d during pregnancy and lactation, respectively.[5] Surveys of athletes before 1989 indicated that a significant percentage of female athletes and adolescent ballet dancers and gymnasts did not consume at least two-thirds of the RDA (180 μg/d) for folate.[31] Studies after 1989 did not find a high proportion of athletes consuming low dietary folate. These differences may be attributed to the change in RDA for folate from 180 to 400 μg/d.[5] Unfortunately, the more recent surveys did not include athletic groups at risk for folate deficiency. With the recent increase in the RDA for folate to 400 μg/d,[5] nutritional surveys of athletes may find dietary folate inadequacy.

Inadequate folate intake first is seen as decreased serum folate concentration, then as decreased erythrocyte folate concentration and increased homocysteine concentration, followed by megaloblastic anemia. Increased mean erythrocyte volume is an early sign of folate deficiency.

There are limited data assessing blood biochemical measures of folate status in physically active individuals. Singh et al.[32] found adequate serum folate concentrations in ultra-marathoners.

Folate supplementation of folate-deficient, but not anemic, athletes did not improve physical performance. Female marathoners with low serum folate concentrations (<4.5 ng/mL) supplemented with folate (5 mg/d for 10 wk) did not experience a significant improvement in treadmill performance, cardiorespiratory function, or metabolic response during exercise as compared with placebo-treated, folate-depleted controls.[33] The lack of a physiologic benefit of folate supplementation contrasted with a significant increase in serum folate. Thus, changes in circulating folate may not reflect changes in tissue or cellular folate status.

Vitamin B12 (Cobalamin)

Cobalamin is a general term that describes a group of cobalt-containing compounds, the corrinoids. This vitamin contains cobalamin with or without a cyanide group, both of which are biologically active for humans. Vitamin B12 functions as a coenzyme for the methyl transfer reaction that converts homocysteine to methionine and another reaction that converts L-methyl-malonyl coenzyme A to succinyl coenzyme A. It also is required for normal erythrocyte production and neurologic function.[5] Biochemical assessment of vitamin B12 status includes hematologic indices (hematocrit, hemoglobin, and erythrocyte indices), serum or plasma vitamin B12, and serum methylmalonic acid concentrations.[5]

The RDA for vitamin B12 is 2.4 μg/d for adults.[5] Surveys of nutrient intakes by groups of athletes suggest that certain groups may not be consuming adequate amounts of this vitamin based on the previous RDA of 2 μg/d. Athletes on energy-restrictive diets[11,12,34] and strict vegetarians[35] are at risk for vitamin B12 depletion. Interest in vitamin B12 among physically active individuals stems from its vital role in erythropoiesis.

General vitamin B12 supplementation apparently has no beneficial effect on performance. Adolescent boys provided 50 μg/d of cyanocobalamin had no improvement in the half-mile run or Harvard step test scores after 7 wk as compared with the non-supplemented control group.[36] Similarly, parenteral B12 administered to non-anemic men failed to elicit improvements in muscle strength and endurance.[37] Thus, supplemental B12 does not benefit performance unless a nutritional deficit is present.

Vitamin C (Ascorbic Acid)

Vitamin C has certain biological functions that can influence physical performance.[38] It is needed for synthesis of carnitine, which transports long-chain fatty acids into mitochondria, and the catecholamines, epinephrine, and norepinephrine. Ascorbic acid facilitates the transport and uptake of non-heme iron at the mucosa, the reduction of folic acid intermediates, and the synthesis of cortisol. Vitamin C is a potent antioxidant that serves to regenerate vitamin E from its oxidized byproduct.[39]

Vitamin C depletion may adversely affect various aspects of physical performance.[40] These detrimental effects range from non-specific responses such as fatigue and muscle weakness to anemia. Vitamin C depletion also decreases training because of recurrent injuries to connective tissues and causes decrements in endurance performance as a result of anemia. Historical reports indicate that sailors and soldiers with scurvy (vitamin C deficiency) experienced shortness of breath during physical exertion, and reduced energy and endurance during work.[41]

The recommendation for vitamin C intake is 60 mg/d to maintain tissue concentrations and prevent scurvy in most individuals.[6] Physiologic stressors, such as infection, cigarette smoking, altitude, and extreme environmental temperatures, increase vitamin C requirements.[6] Exercise is another physiologic stressor that may increase vitamin C needs.

Physically active adults generally consume adequate amounts of vitamin C.[40] Male athletes have a range of vitamin C intake of 95 to 520 mg/d, whereas females have intakes ranging from 55 to 230 mg/d, as compared with the RDA of 60 mg/d. Some athletic groups, however, have suboptimal intakes. Among male athletes, 23% of wrestlers[14] and 20% of football players[41] consumed less than 70% of the RDA. Female athletes, 13% of basketball players and 22% of gymnasts,[41] 25% of cyclists,[9] and 10% of collegiate gymnasts,[12] ingest less than 70% of the RDA. Increased dietary vitamin C intake may be indicated to meet the RDA in some groups of athletes.

Athletes generally display plasma vitamin C concentrations in the range of normal values.[40] Some surveys, however, report a small fraction (~15%) of athletes with values less than the lower limit[4] and many values at the lower end of the range of normal values.[42] Caution should be shown when interpreting plasma ascorbic acid values because physical activity within the previous 24 h can increase plasma vitamin C concentrations.[42]

Vitamin C may indirectly benefit physical performance by enhancing physiologic functions. Men supplemented daily with 250 or 500 mg of vitamin C or placebo for 10 d and exposed to controlled exercise in the heat had decreased body temperature as compared with men treated with placebo.[43] The mechanism of this apparent adaptation was not described.

Vitamin C may enhance immune function. Male runners consuming 500 mg/d of vitamin C received 600 mg of vitamin C or placebo for 21 d and then ran in a marathon race. During the 14 d after the race, the runners supplemented with vitamin C had fewer upper respiratory tract infections than did men receiving the pla-

cebo (33% versus 68%).[44] Additional studies are needed to define the immune function that may be beneficially affected by vitamin C.

Clear evidence of a benefit of vitamin C supplementation on physical performance is lacking.[40] Vitamin C, however, may exert permissive effects on physiologic functions (i.e., antioxidant, immunocompetence, and collagen repair) that facilitate recovery from intense training and, thus, promote performance.

FAT-SOLUBLE VITAMINS

Vitamin A

Vitamin A refers to a group of compounds including retinol, retinaldehyde, and retinoic acid.[7] Carotenoids, principally β-carotene, are precursors of vitamin A and another source of vitamin A. Vitamin A is important for normal vision, gene expression, growth, and immune function by its maintenance of epithelial cell functions.

The requirement for vitamin A is expressed as retinol equivalents. One retinol equivalent equals 1 μg of retinol or 12 μg of β-carotene. The RDAs for vitamin A are 700 retinol equivalents for women and 900 retinol equivalents for men.[7] In contrast to the water-soluble vitamins, very large intakes of vitamin A are toxic and can result in significant metabolic perturbations including nausea, anorexia, hair loss, and kidney and liver damage.[45] Carotenoids, however, even at large intakes apparently are not dangerous to health.[6] High doses of supplemental β-carotene resulted in an unexpectedly increased incidence of lung cancer and heart disease among men who were at increased risk because of a history of smoking, drinking, alcohol use, or working with asbestos.[46]

Vitamin A intakes of athletes are variable. Surveys of endurance runners,[44] professional ballet dancers,[47] female collegiate athletes,[34] male collegiate athletes,[27] female collegiate heavy weight rowers,[48] and male cross-country runners[49] indicate adequate dietary vitamin A intakes. Adolescents and young adults participating in wrestling,[14] ballet,[11] and gymnastics[12] tend to consume less than 70% of the RDA for vitamin A.

Whereas food restriction may explain the diminished vitamin A intake among these athletes, another factor may be causal. Young women beginning an exercise program[50] reported a decrease in vitamin A intake unrelated to any decrease in dietary energy. This decrease was related to a relatively high-fat intake (>40%) and very low consumption of fruits and vegetables. Therefore, food selection and reduced food intake may contribute to decreased vitamin A intakes among certain groups of athletes.

Vitamin A status can be assessed by measuring retinol in the blood, which provides an index of body stores. For example, when liver stores are low, plasma retinol decreases.[51] Guilland et al.[27] found that, despite low self-reported intakes of vitamin A among athletes, biochemical measures of retinol were normal. Similarly, Weight et al.[52] did not identify vitamin A deficiencies among highly trained athletes.

Vitamin A and β-carotene can be measured in blood. A survey of elite German athletes found plasma retinol concentrations within the range of normal values (49 to 93 μg/dL) with considerable variability in β-carotene (14 to 123 μg/dL).[53] Athletes in four sports (marathon running, weight lifting, swimming, and cycling) had values similar to those obtained from blood donors for retinol (60 ± 14 μg/dL) and β-carotene (38 ± 25 μg/dL). These data indicate an adequate intake of vitamin A but a varied, although not extremely low, intake of β-carotene.

The importance of diet in maintaining blood content of vitamin A and β-carotene is emphasized among groups of active adults. Physically active, older Dutch women (60 to 80 y) had dietary intakes of vitamin A (1.1 versus 1.0 mg/d) similar to those of sedentary controls.[54] The active women consumed more fruits and vegetables, whereas the sedentary women had greater intakes of meat and milk. This dietary pattern resulted in increased serum β-carotene concentrations (0.99 ± 0.59 versus 0.67 ± 0.22 mM/L), whereas serum retinol concentrations were similar (1.99 ± 0.41 versus 2.02 ± 0.39 mM/L) among the active and sedentary women. Similar findings have been reported for older men in the United States.[55] Thus, circulating vitamin A reflects body stores that apparently are stable and maintained because of large body stores, whereas β-carotene is more variable probably because of fluctuations in carotenoid intake.

The role of supplemental β-carotene as a facilitator of work metabolism has been examined. Compared with men administered a single dose of β-carotene 2 h before a bout of exercise to exhaustion, placebo-treated men experienced significantly increased plasma concentrations of corticotropin-releasing hormone, adrenocorticotropic hormone, epinephrine, and norepinephrine.[56] This stress hormone response was progressively diminished with increasing doses of β-carotene, and completely suppressed in the men who ingested 30 mg. The rapid biological response to a single dose of β-carotene was surprising and should be confirmed in studies in which subjects serve as their own controls.

There is limited information describing the effects of dietary vitamin A on performance. Running performance did not change in men maintained on a diet deficient in vitamin A for 6 mo and then repleted for another 6 wk.[57] The researchers suggested that the large vitamin A intake (75 000 IU) before the start of the depletion phase probably increased body vitamin A stores.

Although a small percentage of athletes selects diets that are inadequate in vitamin A, there is no evidence that these athletes have biochemical deficiencies or that exercise increases vitamin A needs. Whether β-carotene provides any beneficial effect on performance remains to be determined.

Vitamin E

Vitamin E is a generic term for eight naturally occurring compounds in two classes designated as α-tocopherols and γ-tocopherols.[58] The most active and well known of these compounds is RRR-α -tocopherol, formerly known as *d*-α-tocopherol. Vitamin E supplements contain the natural or synthetic form of α-tocopherol. The natural form of vitamin E, RRR-α-tocopherol or its esterified form with acetate or succinate, is more bioavailable than the synthetic forms.[59] The principal sources of dietary vitamin E include vegetables, seed oils, sunflower seeds, nuts, and whole grains; animal products are generally poor sources of vitamin E.[6]

Dietary vitamin E is primarily composed of α- and γ-tocopherol, of which 20% to 50% is normally absorbed. Vitamin E is absorbed similarly as dietary fat.[60] Thus, any impairment in bile production and secretion, pancreatic function, and chylomicron metabolism will impair vitamin E absorption and use. As polyunsaturated fatty acid intake increases, the vitamin E requirement increases.[61] Among common vegetable oils in the United States, the vitamin E content parallels the polyunsaturated fat content of the oil.

Vitamin E serves as an antioxidant of polyunsaturated fatty acid in cell membranes and subcellular structures.[62] It influences cellular response to oxidative stress through signal transduction pathways.[63] Vitamin E deficiency is associated with neurologic damage and erythrocyte hemolysis.

Vitamin E may play a differential role in oxidative metabolism of different muscle fibers. Human skeletal muscle consists of two major fiber types, type I (red, slow twitch) and type II (white, fast twitch). Type I fibers are plentiful in myoglobin and mitochondrial enzymes and replenish phosphocreatine more efficiently via oxidative phosphorylation than do type II fibers.[64] Type I, as compared to type II, fibers also have greater catalase activity to eliminate reactive oxygen species produced from an increased oxidative metabolism. Thus, type I fibers may also use more vitamin E than type II fibers. Muscles composed of mainly type I

fiber have been reported to contain greater α-tocopherol concentrations than muscles composed of principally type II fibers.[65] Thus, it may be hypothesized that vitamin E is a key nutrient in supporting physical performance.

Vitamin E deficiency adversely affects skeletal muscle; it can lead to muscle degradation in humans.[66] Vitamin E deficiency may promote transformation of type I to type II fibers.[66,67] The mechanism by which these actions occur is unknown but is apparently related to free radical damage. Thus, severe vitamin E deficiency increases oxidative stress in skeletal muscles, alters muscle fiber types, and causes degradation and inflammatory processes that lead to dystrophic conditions.

The RDA for vitamin E is 15 mg of α-tocopherol for women and men.[6] This estimate is based on maintenance of normal physiologic function and protection of tissues from extensive lipid peroxidation. Factors such as increasing dietary polyunsaturated fatty acid and the degree of unsaturation of fatty acids increase the vitamin E requirement. Vitamin E is relatively non-toxic at intakes up to 800 mg.

According to the National Health and Nutrition Examination Survey III (NHANES III), the vitamin E content of the diets of most of the US population is slightly less than the RDA, and the diets of 20% of men and 32% of women contain less than 50% of the RDA.[6] However, when corrections are applied for underreporting of food intake, mean intakes of α-tocopherol meet and probably exceed the RDA of 15 mg/d.[6] Although a summary of 22 dietary intake surveys of endurance and power athletes indicated that they consumed vitamin E in amounts equal to the RDA, most athletes ingested nutritional supplements.[68] In contrast, other surveys reported that 53% of college athletes,[27] 50% of adolescent gymnasts,[12] and 38% of ballerinas[11] consumed less than 70% of the RDA. The mean intake of vitamin E for athletes was 77% of the RDA as compared with 60% for sedentary controls.[27] Thus, vitamin E intake is greater among physically active than among inactive individuals.

Because exercise causes a dramatic increase in oxidative metabolism, there is growing interest in the effect of exercise and vitamin E on metabolism and performance. Vitamin E supplementation did not affect performance in standard tests of strength and endurance among male adolescent swimmers supplemented with 400 mg of α-tocopherol daily for 6 wk,[69] work capacity and muscle strength of collegiate male swimmers given 1200 IU of vitamin E for 85 d,[70] peak oxygen uptake in ice hockey players given 1200 IU/d for 50 d,[71] swimming endurance and blood lactate response in competitive swimmers given 900 IU/d for 6 mo,[72] motor fitness tests, cardiorespiratory function during ergocycle tests, and 400-m swim times in male and female trained swimmers given 400 mg/d for 6 wk.[73] Thus, generalized supplementation with vitamin E apparently does not enhance performance.

Supplemental vitamin E may be beneficial against free radical assault during work. Young men fed 300 mg/d of α-tocopherol for 4 wk had decreased leakage of enzymes and a lower malonaldehyde concentration after strenuous exercise as compared with non-supplemented controls.[74] Men supplemented with 800 IU/d of vitamin E for 4 wk had decreased lipid peroxides, chemical markers of lipid peroxidation, and malonaldehyde in blood.[75] Young and old men supplemented with 800 mg of α-tocopherol, compared with non-supplemented controls, had decreased urinary output of lipid peroxides 12 d after the exercise bout.[76]

Vitamin E deficiency probably is rare among athletes consuming balanced diets. Evidence for a beneficial effect of vitamin E supplementation on physical performance is lacking. Although vitamin E apparently acts in the prevention of damage associated with lipid peroxidation, results are equivocal with regard to the use of vitamin E in the reduction of exercise-induced muscle damage in humans. Thus, the generalized use of large doses of supplemental vitamin E is not encouraged because of the need for protein degradation, the extent of which is not known, to stimulate postexercise muscle protein synthesis.[77] Additional human studies are needed to examine the relations between vitamin E intake, objective measures of exercise-induced muscle soreness and damage, and skeletal muscle protein turnover.

MINERAL METABOLISM AND PERFORMANCE

In contrast to the data on vitamins, there are burgeoning data reporting the interaction between mineral nutritional status and physical activity. The roles of iron, magnesium, zinc, and chromium in performance are described.

Iron

Iron is a key trace element that is required for the delivery of oxygen to tissues and the use of oxygen at the cellular and subcellular levels. It serves as a functional component of iron-containing proteins including hemoglobin, myoglobin, cytochromes, and specific iron-containing enzymes. Iron plays a critical role in energy use during work.

The RDAs for iron are 18 and 8 mg/d for women and men, respectively.[7] Sources of iron in the diet are classified into two groups, heme and non-heme. Heme iron is found in flesh foods as myoglobin. Non-heme iron comes from vegetables and grains. The ability of the body to absorb and use dietary iron is influenced by its chemical form. Heme iron is absorbed with greater efficiency (5% to 35%) than is non-heme iron (2% to 20%) from single meals.[7] Men absorb an average of 6% of total dietary iron and women of child-bearing age absorb an average of 13%.[78]

The adequacy of dietary iron among various groups of physically active people, including competitive athletes, has been reviewed recently.[79] Male athletes generally consume at least the RDA for iron, but female athletes tend to consume somewhat less than the recommended 18 mg/d. A primary cause of iron deficiency among women is inadequate iron intake. It is estimated that about half of all girls and women older than 12 y in the United States consume 10 mg/d or less of iron and only 25% of women of child-bearing age consume the RDA for iron.[7,80] Women runners consume about 12 to 15 mg/d of iron.[81–83] These runners have low intakes of meat and high fiber intakes that reduce the bioavailability of iron to about 10%.[79] When this low amount of available iron (i.e., 1.5 mg) is contrasted with menstrual iron losses of about 0.9 mg and postulated sweat iron of about 0.3 to 0.5 mg, iron balance may be compromised.

Alterations in iron status among various groups of athletes have been summarized.[79] Low ferritin concentrations (<12 μg/L) have been reported in male and female runners. Iron deficiency, defined as ferritin concentration less than 12 μg/L and transferrin saturation less than 16%, have been observed among women competing in field hockey, cross-country skiing, crew, basketball, and softball. Anemia also has been found principally in female runners and skiers. The prevalence of these disturbances is similar to that found in the general population. Depleted tissue iron is seen in about 20% to 25% of girls and women in the United States,[80] which is consistent with most surveys of female runners.[79] However, some youth and adult female runners have a greater prevalence of tissue iron depletion (25% to 44%). Male runners and skiers have greater tissue iron depletion (4% to 13%) than is expected from population standards (2%). Iron deficiency also is increased among some athletic groups, although iron deficiency is increased among girl runners (20% to 30%). Anemia, however, is not more prevalent among male and female athletes than in the general population.

Studies in humans verify the importance of iron to work performance. Iron-deficient workers treated with iron supplements had decreased blood lactate concentrations and increased oxygen uptake during standardized testing compared with pretreatment values.[84,85] The workers with the lowest hemoglobin concentrations also had the shortest time to exhaustion. Work decrements in iron-deficient anemic workers reflected the level of anemia rather

than biochemical changes related to non-hemoglobin.[86] After transfusion, the work capacity of the transfused workers was the same as that measured in other workers with the same (normal) hemoglobin concentrations. These findings illustrate the importance of hemoglobin-associated iron to aerobic work performance in which delivery of oxygen to working tissues is limiting at high-intensity work.

Iron supplementation improves blood biochemical measures of iron status. Among iron-deficient anemic women, iron supplementation not only improves iron status but also increases work capacity and reduces exercise heart rate and lactate concentration.[84]

Several studies have examined changes in the iron nutriture of athletes supplemented with various doses of iron during training. When iron supplements were 18 mg/d and the athletes were not anemic, no significant changes in iron status were noted.[81] When supplements equalling or exceeding 50 mg/d were given to iron-deficient, non-anemic athletes, significant improvements in iron status were observed.

Iron supplementation of iron-deficient, non-anemic athletes yields equivocal performance effects. Matter et al.[33] noted changes in serum ferritin without any effect on peak oxygen uptake or blood lactate concentrations in iron-deficient female marathoners. Similar results were reported in other iron-deficient, non-anemic athletes supplemented with iron.[87,88] In contrast, Rowland et al.[89] found an improvement in endurance time in adolescent, non-anemic female runners supplemented with iron. Two other studies[90,91] reported significant decreases in blood lactate concentrations without a change in peak oxygen uptake in iron-deficient, non-anemic women when serum ferritin concentrations increased in excess of 12 μg/L. These findings emphasize an increased reliance on glycolytic metabolism when tissue iron stores are depleted in the absence of anemia.

Recent findings provide additional support for a beneficial effect of iron supplementation on human physical performance in iron deficiency without anemia. Time to complete a 15-km ergocycle bicycle test was significantly decreased in iron-deficient (serum ferritin < 16 μg/L), non-anemic (hemoglobin > 120 g/L) women (18 to 33 y) randomized to receive 100 mg/d of iron ($FeSO_4$) for 6 wk compared with controls who received placebo.[92] Investigators hypothesized that low tissue iron, characterized by significantly decreased ferritin concentrations, may explain the beneficial effects on exercise performance after concurrent iron supplementation and endurance exercise training. Adaptation to endurance training in untrained women with iron depletion, low transferrin saturation without anemia, was enhanced after 4 wk of iron supplementation (50 mg of iron as $FeSO_4$ given twice daily) compared with age-matched female, placebo-treated controls.[93] Also, iron supplementation (100 mg/d for 6 wk) of women with depleted iron status but not anemia significantly enhanced muscle function compared with non-supplemented controls.[94] Thus, iron deficiency without anemia impairs work performance by promoting skeletal muscle fatigue.

Magnesium

Magnesium is required in a wide variety of fundamental cellular activities that support diverse physiologic systems. Magnesium is involved in more than 300 enzymatic reactions in which food is metabolized and new products are formed.[95] Some of these activities include glycolysis, fat and protein metabolism, adenosine triphosphate hydrolysis, and the second-messenger system. Magnesium also serves as a physiologic regulator of membrane stability and neuromuscular, cardiovascular, immune, and hormonal functions. Thus, magnesium may be considered a potentially limiting element for human performance.

The RDAs for magnesium are 320 and 420 mg/d for women and men, respectively.[96] Dietary sources of magnesium include green, leafy vegetables and unprocessed grains. Some sources of water also may be good sources of magnesium.[97]

Dietary surveys of athletes found an important pattern of magnesium intakes. In general, magnesium intakes for male athletes equaled or exceeded the previous RDA of 350 mg/d, but those of female athletes tended to be 60% to 65% of the current recommendation of 280 mg/d.[97] Regardless of sex, athletes participating in sports that require weight classifications (i.e., wrestling) or have an esthetic component included in the competition (i.e., ballet or gymnastics) tended to consume inadequate amounts of dietary magnesium, 30% to 55% of the 1989 RDA.[41]

Not all cross-sectional studies of athletes have found inadequate dietary magnesium. Comparisons of magnesium intakes among male and female cross-country skiers and control subjects observed magnesium intakes of 170% to 185% of the 1989 RDA (280 and 350 mg/d for women and men, respectively) for athletes, as compared with 108% to 116% for age- and sex-matched control subjects.[98]

Magnesium deficiency impairs physical performance. Clear evidence was provided when muscle spasms in a tennis player were associated with decreased serum magnesium concentration (serum magnesium, 0.65 mM/L; normal range, 0.8–1.2 mM/L).[99] Treatment with 500 mg/d of magnesium gluconate relieved the muscle spasms within a few days.

Supplementation of diets of competitive athletes with magnesium salts has been reported to improve cellular function.[100] Among competitive female athletes with plasma magnesium concentrations at the low end of the range of normal values, serum total creatine kinase and creatine kinase isoenzyme MB decreased after training in the women supplemented with 360 mg of magnesium as magnesium aspartate daily for 3 wk, as compared with the women receiving placebo. Serum lactate concentration and oxygen uptake during an exhaustive rowing ergometer test decreased in male competitive rowers supplemented with magnesium (360 mg/d) for 4 wk, as compared with rowers receiving placebo.[100]

Supplemental magnesium also has been reported to increase muscle strength and power.[101] Young men participating in a strength-training program received a placebo or a magnesium supplement to reach a daily magnesium intake of 8 mg/kg of body weight for 7 wk. Daily magnesium intakes were estimated as 250 and 507 mg/d for the placebo and supplement groups, respectively. Peak knee-extension torque increased significantly (20%) in the magnesium-supplemented men. This observation indicates that strength gain occurs with dietary magnesium intakes of 250 mg/d, but further increases in strength are achieved with intakes of 500 mg/d, which exceed the RDA of 420 mg/d.[96] It also suggests a key role for magnesium in activities that require glycolytic metabolism.

Dietary restriction of magnesium results in reduced magnesium status, impaired physiologic function, and performance in untrained adults. Erythrocyte and skeletal muscle magnesium concentrations and magnesium balance decreased significantly and heart rate and oxygen consumption increased significantly during submaximal exercise in postmenopausal women fed 180 versus 320 mg/d of magnesium.[102] These results indicate that dietary magnesium deprivation induces magnesium loss from the body, depletes cellular magnesium stores, and decreases muscular efficiency during submaximal work in women. Similarly, physically active collegians experienced significant increases in endurance performance and decreased oxygen use during submaximal exercise after magnesium supplementation ($8\ mg \cdot kg^{-1} \cdot d^{-1}$).[103] The findings confirm the observation of increased oxygen requirement during steady-state submaximal work when magnesium status is low and indicate that the adverse effects of magnesium depletion are independent of training status.[100] The biological mechanism of decreased energy efficiency during magnesium restriction relates to increased recruitment of antagonistic muscles to accommodate the increasing work during the exercise tests.[104]

Marathon runners with adequate magnesium status did not benefit from supplemental magnesium. Trained runners, supple-

mented with 365 mg/d of magnesium as magnesium aspartate hydrochloride for 6 wk exhibited no improvement in running performance, no increase in resistance to muscle damage, no change in muscle magnesium concentration, and no improvement in skeletal muscle function.[105] These findings confirm that magnesium supplementation per se does not elicit a beneficial effect on physiologic function or performance when magnesium status is normal.

Zinc

Zinc is required for the structure and activity of more than 300 enzymes from many species. The importance of zinc is reflected by the numerous functions and activities over which it exerts a regulatory role.[106] Zinc is required for nucleic acid and protein synthesis, cellular differentiation and replication, and glucose use and insulin secretion. Zinc exerts regulatory actions in various aspects of hormone metabolism including the production, storage, and secretion of hormones and regulating interactions between hormones and receptors and end-organ responsiveness. Adequate zinc is needed for the integration of many physiologic systems such as immunity, reproduction, taste, wound healing, skeletal development, behavior, and gastrointestinal function. This array of zinc-dependent functions suggests that zinc status should regulate work performance.

Athletes generally consume less zinc than recommended, 8 and 11 mg/d for women and men, respectively.[107] However, a significant proportion of participants in some activities, including long-distance running and gymnastics, consume less than 10 mg/d. Marginal intake is more prevalent among female than among male athletes who restrict food intake.[11,12,108]

Attention to zinc status as a potential factor affecting physical performance came with the report of significantly decreased serum zinc concentrations among some endurance runners.[109] About 25% of competitive male runners had serum zinc values less than the lower limit designated for the range of normal values. Importantly, serum zinc was significantly correlated with weekly training distance. The investigators postulated that dietary habits, specifically the avoidance of animal products and consumption of low-zinc, high-carbohydrate foods and increased zinc losses, contributed to the low serum zinc concentrations in some of the runners.

Low circulating zinc concentrations have been reported in physically active adults. Elite German athletes had similar serum zinc values as sex-matched non-athletes.[110] Low serum zinc, however, was found in about 25% of the athletes as compared with only 13% of the non-athletes. Serum zinc values also were low in a group of female marathon runners, with 22% having values that would be considered low.[108,111] In contrast, no differences in plasma zinc concentrations were found in comparisons of female and male collegiate athletes and sex-matched controls.[112,113] One explanation of these findings is that dietary zinc may have been inadequate in the athletes with the decreased circulating zinc. The athletes with clinically depressed circulating zinc had zinc intakes less than the RDA.[108,113] The relation between dietary zinc and plasma zinc concentration indicates that, when athletes consume at least 70% of the RDA, plasma zinc concentrations are within the range of normal values.[107]

There are limited data to imply that zinc status may affect physical performance. Preliminary observations have indicated that zinc enhances in vitro muscle contraction.[114] Muscle strength and endurance were improved in middle-age women supplemented with zinc (30 mg/d) and a placebo in a double-blind, cross-over designed study with a washout period.[115] Because these muscle functions rely on recruitment of fast-twitch glycolytic muscle fibers, it may be hypothesized that zinc supplementation enhanced the activity of lactate dehydrogenase, a zinc-dependent enzyme. Interpretation of the significance of these findings is confounded by the large dose of the zinc supplement, which suggests that any biological effect on performance was a pharmacologic response. Further, the limited duration of the zinc supplementation (14 d) and the ability of the body to use the administered zinc raise other concerns.

Carbonic anhydrase, a zinc metalloprotein[106] that regulates the elimination of carbon dioxide from cells, may play a role in facilitating performance. Preliminary data have indicated that the activity of this enzyme in red blood cells responds to dietary zinc.[116] Oxygen use and carbon dioxide elimination during constant workload decreased significantly when dietary zinc was 4 as opposed to 18 mg/d in physically active men.[117] Total erythrocyte carbonic anhydrase activity decreased significantly with low zinc intake. However, because respiratory quotient increased with zinc restriction, it may be speculated that lactate dehydrogenase activity also was adversely affected. These findings provide the first evidence of impaired metabolic response during work when dietary zinc is suboptimal.

Chromium

Chromium is a provisionally essential mineral that functions broadly in the regulation of glucose, lipid, and protein metabolism by potentiating the action of insulin at the cellular level.[118] The mechanism has been proposed to involve several enzymatic changes including insulin binding to its membrane receptor, resulting in an intermolecular phosphorylation cascade leading to increased insulin sensitivity.[119] Physiologically active forms of chromium facilitate insulin action, which results in a decreased need for insulin.[120,121] This fundamental role of chromium and the knowledge that the general public may consume diets low in chromium[122] have prompted physically active individuals to consider chromium as a potentially limiting nutrient for promotion of physical fitness and health.

There is no RDA for chromium. Adequate intakes are 25 and 35 μg/d for women and men, respectively,[7] and is based on the mean daily intake of men and women consuming usual diets.[122]

Routine assessment of dietary chromium is problematic.[118] The chromium concentration of food is very low and contamination of samples can be a difficulty. Estimates of the chromium concentration in foods of the typical Western diet range from 5 to 24 μg/1000 kcal with an average intake of 15 μg/1000 kcal.[121] Fruits, whole grains, and vegetables are primary contributors of chromium to the diet.[123]

Chromium excretion increases after exercise. Acute exercise in trained male runners resulted in a significant increase in urinary chromium excretion from 0.09 to 0.36 ng/mg of creatinine.[124] Chromium excretion immediately after a 6-mile run was minimal, about 2% of total daily excretion. It almost doubled, however, on the day of the run as compared with the urinary losses on the day before the run (5.7 versus 3.1 nM/d). Serum chromium concentration increased significantly at the end of a timed run (3.3 nM/L) and remained elevated for 2 h after the completion of the run (3.7 nM/L), as compared with pre-exercise values (2 nM/L).[125] Urinary chromium losses also doubled on the day of the run (7.1 versus 3.8 nM/d).

Because dietary chromium can affect circulating and urinary chromium levels, additional studies were initiated. Trained and untrained men consumed a controlled diet containing 9 μg of chromium per 1000 kcal for 7 d during which a standardized exercise test to exhaustion was performed.[126] Serum chromium was similar between groups and did not change immediately after the exercise test. Two hours after the test, however, serum chromium concentration almost doubled in the trained men, whereas the untrained men showed no apparent change; these changes were not statistically significant. The trained men excreted significantly more chromium in urine than did the untrained men who had no significant change in urinary chromium loss. These findings indicate that high-intensity exercise can increase urinary loss of chromium, although the loss is small.

Because dietary chromium may be limiting and exercise may promote chromium losses, chromium supplementation as chromium picolinate, a highly bioavailable form, has been proposed to enhance physical performance because of its purported anabolic actions.[127] The effect of supplemental chromium (200 μg/d as chromium picolinate) and resistance training on body composition was studied in young men.[127] In one trial, 10 young men received the chromium supplement or a placebo for 40 d while participating in a weight-training program. Chromium supplementation was associated with significant gains in weight (2.2 kg) and lean body mass (1.6 kg) and fat as determined with anthropometry. A second study involved male football players who received the chromium supplement and participated in a more intensive strength-training regimen for 42 d. After 14 d, body fatness decreased 2.7% and lean body mass increased 1.8 kg; no significant changes were seen in the men receiving the placebo. After 42 d, the men receiving the chromium lost body weight (1.2 kg) and fat (3.4 kg) and gained lean body weight (2.6 kg), but the placebo-treated men also lost fat (1.8 kg) and gained lean body weight (1.8 kg). It was concluded that chromium supplementation promotes gains in lean body mass.

Kaats et al.[128] evaluated the effects of graded chromium supplementation (0, 100, and 200 μg) on body composition changes of overweight adults during a 72-d study. Consumption of 200 or 400 μg/d of chromium significantly improved an indirect indicator, not a measurement, of body composition change (gain of fat-free mass and loss of body fat) compared with placebo.

Other studies have not confirmed these findings. Hasten et al.[129] found that chromium supplementation (200 μg/d as chromium picolinate) of male and female beginning weight-training students had no effect on anthropometric measurements or strength gain. Body weight gain was significantly greater in the women than in the men. Also, compared with placebo, 400 μg/d of supplemental chromium for 16 wk did not enhance body weight or body fat reduction, fat-free mass gain, or physical fitness of US Navy personnel.[130]

Other studies of chromium supplementation and resistance training have found no beneficial effects of chromium. Compared with placebo-treated men, collegiate football players[131] and untrained men[132] supplemented with 200 μg/d of chromium for 9 and 12 wk, respectively, had no significant increase in fat-free mass or a decrease in body fat or a significant strength gain. Chromium picolinate (200 μg/d of chromium) also did not significantly increase muscle mass or strength or decrease body fat in young men participating in strength training.[133]

The effect of chromium supplementation on weight loss and glucose metabolism in mildly obese women was examined.[134] Young women were randomly assigned to receive chromium picolinate without exercise training, placebo with exercise, chromium picolinate with exercise, or chromium nicotinate with exercise training for 9 wk. The women consumed two capsules containing 200 μg/d of chromium per capsule. Regardless of chemical form, chromium supplementation had no significant effect on body fat loss.

Although most diets may not provide adequate chromium, the use of chromium supplements provides no propitious effect on body composition or physical performance.[118,135] More research is needed to identify individuals who would benefit from supplemental chromium to enhance function.

USE OF NUTRITIONAL SUPPLEMENTS

Experimental findings have indicated that clinical vitamin and mineral deficiencies are rare in the general population when a balanced diet is consumed.[7] However, some individuals who seek to enhance performance may opt for nutritional supplements. Professional organizations provide advice to physically active individuals who seek to use nutritional supplements. The American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine have stated that the proper nutritional strategy to promote optimal health and performance is to obtain adequate nutrients from a wide variety of foods. Vitamin and mineral supplementation is appropriate when scientific evidence supports safety and effectiveness. Because some groups of physically active people may be at increased risk of nutrient depletion, the use of a vitamin and mineral supplement, not exceeding the recommended daily intake, may be consumed as a preventive measure.[136] Recommendations for maximal intakes of vitamins and minerals are presented in Tables III and IV. These estimates are the sum of diet and supplement sources, except for folate, whose tolerable upper intake level pertains to fortified foods and supplements.

TABLE IV.

DIETARY REFERENCE INTAKES OF MINERALS: RECOMMENDED INTAKES FOR INDIVIDUALS[7,96]*

	Chromium (μ/d)	Iron (mg/d)	Magnesium (mg/d)	Zinc (mg/d)
Females				
14–18 y	24†	15	360	9
19–30 y	25†	18	310	8
31–50 y	25†	18	320	8
Males				
14–18 y	35†	11	410	11
19–30 y	35†	8	400	11
31–50 y	35†	8	420	11
UL	NA	45	350‡	40

* Values are presented as recommended dietary allowances.
† Values are adequate intakes.
‡ From supplemental magnesium.
NA, not available; UL, tolerable upper limit.

The use of generalized vitamin and mineral supplementation has not enhanced nutritional status and physical performance of trained athletes. Studies of elite male and female athletes supplemented with a balanced vitamin and mineral preparation while training for participation in a wide variety of competitive sports have not shown any propitious effects on biochemical measures of nutritional status or sports performance.[3,4,28,52,137]

SUMMARY

The following points may be gleaned from the previous discussion:

- Evidence of inadequate dietary vitamin and mineral intakes among physically active individuals is generally lacking. Some athletes, such as adolescent ballerinas, gymnasts, long-distance runners, and wrestlers, may not consume adequate amounts of micronutrients because they limit food intake to meet weight restrictions for esthetic requirements or competition.
- Physically active individuals should consume a diet that contains a variety of foods to maximize vitamin and mineral and other nutrient intakes and thus to eliminate the need for nutritional supplements.
- Performance will not be improved if individuals consuming nutritionally adequate diets use nutritional supplements.
- Only individuals with a defined nutrient deficiency or deficiencies will benefit from supplementation of the limiting nutrient(s).
- An athlete should direct questions and concerns about the quality of a diet to a licensed registered dietitian who is experienced in counseling physically active people.

- Athletes who intermittently ingest a vitamin and mineral supplement should not exceed the recommended intakes for nutrients.
- There is a need to evaluate the validity of nutrient recommendations for adolescents in the promotion of physical activity.

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