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B-Vitamins and Exercise: Does Exercise Alter Requirements?

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The B-vitamins (thiamin, riboflavin, vitamin B-6) are necessary in the energy-producing pathways of the body, while folate and vitamin B-12 are required for the synthesis of new cells, such as the red blood cells, and for the repair of damaged cells. Active individuals with poor or marginal nutritional status for a B-vitamin may have decreased ability to perform exercise at high intensities. This review focuses on the B-vitamins and their role in energy metabolism and cell regeneration. For each vitamin, function related to physical activity, requirement, and status measures are given. Research examining dietary intakes and nutritional status in active individuals is also presented. Current research suggests that exercise may increase the requirements for riboflavin and vitamin B-6, while data for folate and vitamin B-12 are limited. Athletes who have poor diets, especially those restricting energy intakes or eliminating food groups from the diet, should consider supplementing with a multivitamin/mineral supplement.

Key Words: thiamin, riboflavin, vitamin B-6, folate, vitamin B-12, athletes

Micronutrients play important roles in maintaining the health of physically active individuals (53, 54, 57, 59). For example, the B-vitamins are necessary in the energy-producing pathways of the body, while folate and vitamin B-12 are required for the synthesis of new cells, such as red blood cells, and for the repair of damaged cells. Other micronutrients are important for maintaining adequate immune function, protecting the tissues of the body from oxidative damage, maintaining bone health, and building and repairing muscle tissue.

Regular physical activity may alter the need or requirements for some micronutrients in several ways (53, 54, 57, 59). First, the metabolic pathways that produce energy are stressed during physical activity; thus, requirements for some of the nutrients used in these pathways may increase. Second, biochemical adaptations that occur with training in the tissues of the body may increase requirements. Third, strenuous exercise may also increase the turnover or loss of a particular micronutrient in sweat, urine, or feces. Finally, additional micronutrients may be required to repair and maintain the higher lean tissue mass of some athletes and active individuals.

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When the current Dietary Reference Intakes (DRI) for each of the B-vitamins were established for the US and Canada, there was inadequate information to calculate specific recommendations for active individuals (40, 41) (Table 1). If exercise increases the need for selected B-vitamins, then athletes and active individuals may have poorer status while consuming the Recommended Dietary Allowance (RDA) or Adequate Intake (AI) for these nutrients or have poorer status compared to sedentary controls with similar dietary intakes. Athletes at greatest risk of poor status are those who restrict energy intake for weight loss, those who are concerned about maintaining a low body weight (i.e., gymnasts, jockeys, wrestlers, figure skaters), or those who eliminate selected food groups, such as dairy or meat. Individuals with these types of eating behaviors may need to use a multivitamin supplement or use fortified foods to improve overall micronutrient status.

This review will focus on the B-vitamins (thiamin, riboflavin, vitamin B-6, folate, and vitamin B-12) and their role in energy metabolism and cell regeneration, especially the synthesis of red blood cells. For each nutrient, we will discuss their function related to physical activity, their requirements, and their status measures. We will also review the research literature examining the dietary intakes and nutritional status of these vitamins in active individuals. Studies published after 1980 examining nutritional status of athletes were identified using the PubMed search engine. Finally, we will discuss whether there are adequate data to suggest increased requirements for these vitamins in active individuals.

B-Complex Vitamins

B-complex vitamins have several important functions in the body, including energy production, hemoglobin synthesis, adequate immune function, and building and repair of muscle tissue. Research examining dietary intakes of the B-complex vitamins in athletes has reported adequate mean intakes (1, 4, 6, 20, 44, 63). However, when examining the dietary intake data for individual athletes, many athletes, especially women, do not consume the recommended levels for these nutrients (4, 38, 43, 45, 47, 55, 77). For example, folate, vitamin B-6, and riboflavin are B-complex vitamins that are frequently low in the diets of women who are physically active (32, 51-53, 57, 64), especially those with restricted energy intakes or disordered eating behaviors (4, 17, 96-98). In general, active males have better nutrient intakes, not because they select a more nutrient-dense diet, but because their energy intakes are typically higher than that of active females (32). Finally, it should be noted that many individuals under-report when recording diet records, which may contribute to some of the low nutrient intakes reported in the literature. Underreporting of food intake may result from incomplete recordkeeping, conscious misreporting to respond in a more socially desirable way, or the recording process causing a temporary change in eating behaviors (60, 87). Further research is needed to identify how to account for these discrepancies in studies of nutrition.

Thiamin

Thiamin is important for the metabolism of carbohydrates, fats, and proteins, especially the branched chain amino acids (BCAAs) (leucine, isoleucine, and valine) (52, 53, 57, 59). The active form of this micronutrient, thiamin pyrophos-

Table 1 Exercise-Related Function and the Dietary Reference Intakes (DRIs) for Individuals 19-50 y[a, b]

Nutrient	Function	Food sources	Males RDA	Females RDA	Males EAR	Females EAR	UL	Adverse effects of high doses
Thiamin (mg/d)	Serves as a coenzyme [TPP (thiamin pyrophosphate)] in reactions in the energy pathways	Whole grains and enriched grain products, pork, liver, dark green vegetables, nuts	1.2	1.1	1.0	0.9	ND[c]	Data are inadequate to assess risk
Riboflavin (mg/d)	Participates in electron transfer in energy metabolism	Milk and dairy products, organ meats, whole grains and enriched grain products, eggs, and nuts	1.3	1.1	1.1	0.9	ND[c]	Data are inadequate to assess risk
Vitamin B-6 (mg/d)	Involved in amino acid metabolism (such as transamination) and in glycogen breakdown	Animal foods, such as meats, fish, and poultry	1.3	1.3	1.1	1.1	100	No adverse effect of ↑ food B6; ↑ supplemental B6 can cause sensory neuropathy (difficulty walking)
Folate (µg/d as dietary folate equivalents [DFE])	Required for cell division and cell regeneration in cells such as the red blood cells	Enriched grains, plant sources, and organ meats	400	400	320	320	1000[d]	No adverse effect of fortified food folic acid; ↑ supplemental folic acid can mask vitamin B12 deficiency
Vitamin B-12 (µg/d)	Involved in the recycling of folate and in neural tissue	Only found in animal foods	2.4	2.4	2.0	2.0	ND[c]	Data are inadequate to assess risk; No adverse effect of ↑ food B12

[a] This table summarizes Recommended Dietary Allowances (RDA), Estimated Average Requirements (EAR), and Tolerable Upper Intake Levels (UL) for selected B-vitamins.

[b] Source: Institute of Medicine. Food and Nutrition Board (40-41). A complete table of RDAs and AIs for all life stages groups can be found at http://www.iom.edu.

[c] ND= none determinable due to lack of data.

[d] The UL for folate applies to synthetic forms obtained from supplements, fortified foods, or a combination of the two.

phate (TPP), serves as a coenzyme in many key reactions in the energy-producing pathways of the body, which are stressed during physical activity. For example, TPP is required by pyruvate dehydrogenase, the enzyme that converts pyruvate to acetyl CoA, and by α-keotglutarate dehydrogenase in the tricarboxylic acid (TCA) cycle. Thus, thiamin is critical to aerobically metabolize both carbohydrates and fats. In addition, thiamin is also needed for BCAA metabolism, which increases with physical activity, especially endurance exercise.

Food Sources and Recommended Intakes. Thiamin is found in a variety of foods, such as whole grains and enriched grain products, pork, liver, nuts, legumes, and green leafy vegetables. Athletes at risk for poor thiamin status are those who restrict dietary intake or who self-select diets with low thiamin foods. The RDA for thiamin is 1.1 and 1.2 mg/d for adult females and males, respectively (41). No Tolerable Upper Intake Level (UL) has been established.

Assessment of Status. To assess status of a nutrient, dietary intake data and biochemical measures (blood and urine) are typically determined (28). For thiamin, biochemical status is determined by assessing the activity of an enzyme, erythrocyte transketolase, located in the red blood cells (28). Thiamin is needed at two steps of the pentose phosphate pathway, a pathway that generates ribose-5-phosphate needed in the production of ATP. One of these steps requires erythrocyte transketolase. The assessment of this activity is called erythrocyte transketolase activity coefficient or ETKAC. It is measured by determining the activity of erythrocyte transketolase before and after the coenzyme (thiamin) is added. If more enzyme activity is generated with additional thiamin, poor or marginal thiamin status is indicated (28, 41, 57). An ETKAC value > 1.25 suggests thiamin deficiency (28, 41).

Assessment of Dietary Intake in Active Individuals. Table 2 summarizes research examining thiamin intakes in adult athletes. Researchers have found that the dietary intakes of thiamin in active individuals meet recommended guidelines. For example, Niekamp and Baer (62) examined dietary intakes of several nutrients, including thiamin, in trained male cross-country runners. Using 4-d dietary records, they documented mean intakes of thiamin exceeding the 1989 RDA (1.5 mg) (61) for these male athletes. In a recent study, Beshgetoor and Nichols (10) measured thiamin intakes in the diets of supplementing and non-supplementing female athletes and found that mean intakes of thiamin exceeded the 1998 RDA for both groups. In addition, Rankinen et al. (68) compared nutrient intakes in Finnish elite male ski jumpers to age-matched controls. Despite a lower energy intake in the athletes, intake of thiamin was similar in both groups. Finally, Ziegler et al. (97) measured the dietary intakes of thiamin in male and female adolescent figure skaters and found mean intakes to be normal, with only 7% of males and 13% of the females to have thiamin intakes less than two-thirds the 1989 RDA for thiamin. Thus, thiamin intakes of most athletes, even those participating in sports that emphasize leanness, appear to be consistent with recommendations (5).

Assessment of Status in Active Individuals. Table 2 summarizes research examining thiamin status in adult athletes. Although dietary intakes for thiamin are typically above recommended guidelines, exercise or vigorous physical activity may alter nutrient requirements and result in marginal or poor status in some

Table 2 Summary of Studies Examining Thiamin Intake and/or Status in Adult Athletes[a,b]

Study	Subjects	Assessment index	Results
Thiamin intake			
Beals & Manore, 1998 (4)	24 female athletes with subclinical eating disorders; 24 female control athletes	7-d weighed food record	Athletes with subclinical eating disorders: 1.9 ± 0.6 mg/d Control athletes: 2.2 ± 0.6 mg/d
Beshgetoor & Nichols, 2003 (10)	25 female master cyclists and runners	4-d food record	2.0 ± 0.5 mg/d
Clark et al., 2003 (14)	13 female collegiate soccer players	2 3-d food records (pre-season and post-season)	Pre-season: 1.5 ± 0.6 mg/d Post-season: 1.0 ± 0.4 mg/d
DeBolt et al., 1988 (18)	267 male US Navy SEAL trainees	1-d food record	2.5 mg/d
Faber & Benade, 1991 (20)	30 field athletes (discus-, hammer-, javelin-throwers, and shotputters) (20 male; 10 female)	7-d food record	Males: 2.19 ± 0.73 mg/d Females: 1.31 ± 0.21 mg/d
Jensen et al., 1992 (42)	14 male endurance cyclists	5-d weighed food record while training; 3-d weighed food record while racing	Training: 4 ± 1 mg/d Racing: 5 ± 2 mg/d
Kaiserauer et al., 1989 (43)	17 female distance runners (8 with amenorrhea; 9 with eumenorrhea)	3-d food record	Amenorrheic distance runners: 0.8 mg/d Eumenorrheic distance runners: 1.4 mg/d
Keith et al., 1989 (45)	8 female highly trained cyclists	3-d weighed food record	1.7 ± 1.1 mg/d
Kopp-Woodroffe et al., 1999 (47)	4 female amenorrheic runners	7-d weighed food record	1.4 ± 0.3 mg/d
Leydon & Wall, 2002 (50)	20 jockeys (6 male; 14 female)	7-d weighed food record	Males: 0.91 ± 0.42 mg/d Females: 1.07 ± 1.08 mg/d
Niekamp & Baer, 1995 (62)	12 male runners	2 4-d food records	2.3 ± 0.6 mg/d
Nieman et al., 1989 (63)	347 marathon runners (291 male; 56 female)	3-d food record	Males: 1.95 mg/d Females: 1.39 mg/d

(continued)

Table 2 ***(continued)***

Study	Subjects	Assessment index	Results
Rankinen et al., 1998 (68)	21 male Finnish elite ski jumpers	4-d food record	1.6 ± 0.8 mg/d
Rico-Sanz et al., 1998 (70)	8 male elite soccer players	12-d food record	3.91 ± 0.88 mg/d
Saris et al., 1989 (78)	5 male cyclists during the Tour de France	22-d food record while racing	2.4 ± 0.7 mg/d
Worme et al., 1990 (94)	71 triathletes (50 male; 21 female)	3-d food record	Males: 2.1 mg/d Females: 1.7 mg/d
Ziegler et al., 1999 (97)	37 US National Figure Skaters (19 male; 18 female)	4-d diet record	Males: 2.2 ± 0.9 mg/d Females: 1.6 ± 1.4 mg/d
Thiamin status			
Fogelholm et al., 1992 (23)	17 Nordic skiers (9 male; 8 female)	4 7-d food records; ETKAC	Dietary Male: 3.0 mg/d Female: 2.2 mg/d ETKAC Males: 1.12 Females: 1.11
Fogelholm et al., 1993 (24)	42 physically active college students (24 male; 18 female)	Blood ETKAC	Males: 1.14 Females: 1.15
Frank et al., 2000 (25)	60 leisure athletes (55 male; 5 female)	Blood thiamin; ETKAC	Blood thiamin 16.2 ± 8.8 nmol/L ETKAC 1.08 ± 0.07
Guilland et al., 1989 (29)	55 male well-trained recreational athletes	7-d food record; ETKAC; erythrocyte TPP	Dietary 1.5 mg/d ETKAC 1.11 Erythrocyte TPP 0.33 µmol/L
Singh et al., 1993 (80)	17 ultramarathoners (15 male; 2 female)	4-d food record (usual intake); blood thiamin; urinary thiamin	Dietary 2.4 mg/d Blood 208.0 nmol/L Urine 5.83 µmol/L
Weight et al., 1988 (92)	30 well-trained male runners	5-d food record; blood transketolase	Dietary 1.51 ± 0.50 mg/d Blood transketolase 51.1 ± 10.6 U/L

[a]All values are means or means ± standard deviation.

[b]Reference values: 1989 RDA for males 19-50 y = 1.5 mg; 1989 RDA for females 19-50 y = 1.1 mg; 1998 RDA for males 19-50 y = 1.2 mg; 1998 RDA for females 19-50 y = 1.1 mg; 1998 EAR for males 19-50 y = 1.0 mg; 1998 EAR for females 19-50 y = 0.9 mg; Erythrocyte transketolase activity coefficient (ETKAC) = ≤ 1.25; Erythrocyte thiamin ≥ 70 nmol/L; Thiamin pyrophosphate effect < 25% (28, 41, 61).

active individuals. Fogelholm and colleagues have completed several cross-sectional studies examining thiamin status in active males and females compared to controls. In Finnish male athletes, Fogelholm et al. (22) examined whether physical activity alters biochemical indices of nutritional status, including thiamin status, using ETKAC. They found that the athletes did not have poor status of thiamin compared to the control subjects; ETKAC was actually statistically higher in the control subjects (1.17 ± 0.01) versus the athletes (1.14 ± 0.01 to 1.15 ± 0.01). This study was supported by another study examining thiamin status in elite Nordic skiers (23). Again they did not find that ETKAC differed between the elite Nordic skiers and controls. Seasonal variations in ETKAC were the same between skiers and controls despite the large changes by season in physical activity of the skiers. This research is supported by others who examined thiamin status in 1918 elite Polish athletes (males and females combined) from 1987 to 1992 and found little risk of thiamin deficiency, with only 2% of all athletes with poor status, ranging from 0 to 3% over a 6-y period (67). Conversely, another cross-sectional study by Fogelholm et al. (24) found that 12% ($n = 5$) of the participants (male and female recreational and competitive athletes) whom they examined had marginal thiamin status with an ETKAC > 1.20.

Research has also examined the impact of training on thiamin status. In a prospective study, Fogelholm (21) assessed thiamin status in active and sedentary young women throughout a 24-wk fitness exercise program. ETKAC remained the same in both groups throughout the study; thus, exercise training has not been shown to alter thiamin status.

Although we have limited data on the prevalence of poor thiamin status in active individuals, thiamin deficiency may impair athletic performance (88, 89). Using a controlled metabolic feeding study, van der Beek et al. (89) depleted male athletes of thiamin, riboflavin, and vitamin B-6 over a 3-wk period by feeding commonly consumed processed foods. Although no control group was included in this study, the researchers found that exercise performance decreased significantly after the depletion of thiamin, riboflavin, and vitamin B-6, with significant increases in lactic acid production. However, this decrease in exercise performance was attributed to the deficiency of three B-vitamins, not just thiamin.

Thiamin functions in the conversion of pyruvate into acetyl CoA; thus, a deficiency of thiamin could impair metabolism of carbohydrates and result in the accumulation of lactic acid. Research examining whether supplementation with thiamin would improve exercise performance found that supplementation with a thiamin derivative did not influence high-intensity exercise performance, when thiamin status was adequate (91). Research by others supports these findings. Doyle et al. (19) found that thiamin did not improve performance in knee extension and flexion exercises, while Webster (90) found that blood lactic acid levels and exercise performance did not differ between the thiamin and placebo trials in trained cyclists. However, in a placebo-controlled crossover study, Suzuki and Itokawa (85) found that high-dose thiamin supplementation (100 mg/d) for 3 d significantly decreased the number of complaints after exercise in a subjective fatigue assessment. Thus, poor thiamin status may impact exercise performance; however, the prevalence of deficiency appears to be low in active individuals.

Riboflavin

Riboflavin is an essential component of two coenzymes, flavin adenine dinucleotide (FAD) and flavin mononucleotide (FMN). These coenzymes participate in the transfer of electrons in energy metabolism, amino acid metabolism, and steroid hormone production (52, 53, 57, 59). Thus, riboflavin is important for exercise because it assists with the transfer of electrons that come from the energy pathways to the electron transport chain for formation of ATP. Finally, riboflavin is involved in the conversion of vitamin B-6 to its active form (53).

Food Sources and Recommended Intakes. Adequate riboflavin intakes can easily be achieved by regularly consuming milk and dairy products, eggs, whole grains and cereals, lean meats, and broccoli. As with thiamin, those athletes at risk of poor riboflavin status are those that restrict dietary intake or select highly refined diets. The RDA for riboflavin is 1.1 and 1.3 mg for adult females and males, respectively, while a UL for riboflavin has not been established (41).

Assessment of Status. Riboflavin status is typically assessed by determining the extent to which enzyme activity of erythrocyte glutathione reductase increases in the presence of added riboflavin, termed the erythrocyte glutathione reductase activity coefficient (EGRAC) (28, 53). Activity of this enzyme is the most sensitive marker of riboflavin status and is the method used in most exercise studies examining riboflavin status. An EGRAC value ≥ 1.2 suggests riboflavin deficiency (28, 41).

Assessment of Dietary Intake in Active Individuals. Table 3 summarizes research examining riboflavin intakes in adult athletes. Most cross-sectional studies comparing dietary riboflavin intakes in active and sedentary individuals have found that riboflavin intake by athletes is adequate to meet dietary guidelines. For example, Niekamp and Baer (62) found that during the competitive season mean intakes of riboflavin for male cross-country runners were 158% of the 1989 RDA (1.7 mg) (61). In addition, Beshgetoor and Nichols (10) found that mean intakes for riboflavin for female master athletes (cyclists and runners) exceeded the 1998 RDA of 1.1 mg/d.

Assessment of Status in Active Individuals. Table 3 summarizes research examining riboflavin status in adult athletes. Like thiamin, riboflavin deficiency may impair athletic performance (88, 89). When van der Beek et al. (89) fed a diet low in thiamin, riboflavin, and vitamin B-6 until blood levels indicated deficiency, lactate threshold and exercise performance decreased. Thus, a combination of poor status of these B-vitamins (thiamin, riboflavin, and B-6) negatively impacted performance. In addition, supplemental riboflavin is beneficial for those individuals in poor riboflavin status. Suboticanec et al. examined the effects of riboflavin and vitamin B-6 supplements on physical fitness in male adolescents (age 12 to 14 y) attending three schools in a rural area of Central Croatia (84). After baseline data were completed, the schools were randomized to receive a riboflavin supplement (2 mg), a vitamin B-6 supplement, or a placebo supplement. Before supplementation, 19% of the participants had poor status of riboflavin as assessed by EGRAC. After supplementation, those participants with initial poor status of riboflavin significantly increased physical fitness.

Table 3 Summary of Studies Examining Riboflavin Intake and/or Status in Adult Athletes[a,b]

Study	Subjects	Assessment index	Results
Riboflavin intake			
Beals & Manore, 1998 (4)	24 female athletes with subclinical eating disorders; 24 female control athletes	7-d weighed food record	Athletes with subclinical eating disorders: 2.2 ± 0.6 mg/d Control athletes: 2.7 ± 0.7 mg/d
Beshgetoor & Nichols, 2003 (10)	25 female master cyclists and runners	4-d food record	2.0 ± 0.5 mg/d
Clark et al., 2003 (14)	13 female collegiate soccer players	2 3-d food records (pre-season and post-season)	Pre-season: 1.8 ± 0.7 mg/d Post-season: 1.2 ± 0.7 mg/d
DeBolt et al., 1988 (18)	267 male US Navy SEAL trainees	1-d food record	3.4 mg/d
Faber & Benade, 1991 (20)	30 field athletes (discus-, hammer-, javelin-throwers, and shotputters) (20 male; 10 female)	7-d food record	Males: 3.65 ± 2.18 mg/d Females: 1.67 ± 0.42 mg/d
Fogelholm et al., 1992 (23)	17 Nordic skiers (9 male; 8 female)	4 7-d food records	Male: 3.4 mg/d Female: 2.4 mg/d
Jensen et al., 1992 (42)	14 male endurance cyclists	5-d weighed food record while training; 3-d weighed food record while racing	Training: 5 ± 2 mg/d Racing: 5 ± 2 mg/d
Kaiserauer et al., 1989 (43)	17 female distance runners (8 with amenorrhea; 9 with eumenorrhea)	3-d food record	Amenorrheic distance runners: 1.2 mg/d Eumenorrheic distance runners: 2.0 mg/d
Keith et al., 1989 (45)	8 female highly trained cyclists	3-d weighed food record	1.6 ± 0.9 mg/d
Kopp-Woodroffe et al., 1999 (47)	4 female amenorrheic runners	7-d weighed food record	1.6 ± 0.3 mg/d

(continued)

Table 3 ***(continued)***

Study	Subjects	Assessment index	Results
Leydon & Wall, 2002 (50)	20 jockeys (6 male; 14 female)	7-d weighed food record	Males: 1.11 ± 0.26 mg/d Females: 0.92 ± 0.34 mg/d
Niekamp & Baer, 1995 (62)	12 male runners	2 4-d food records	2.7 ± 0.7 mg/d
Nieman et al., 1989 (63)	347 marathon runners (291 male; 56 female)	3-d food record	Males: 2.28 mg/d Females: 1.63 mg/d
Rankinen et al., 1998 (68)	21 Finnish elite male ski jumpers	4-d food record	2.1 ± 1.0 mg/d
Rico-Sanz et al., 1998 (70)	8 male elite soccer players	12-d food record	2.48 ± 0.56 mg/d
Saris et al., 1989 (78)	5 male cyclists during the Tour de France	22-d food record while racing	Dietary 5.0 ± 1.6 mg/d
Worme et al., 1990 (94)	71 triathletes (50 male; 21 female)	3-d food record	Males: 2.6 mg/d Females: 2.2 mg/d
Riboflavin status			
Fogelholm et al., 1993 (24)	42 physically active college students (24 male; 18 female)	Blood EGRAC	Males: 1.34 Females: 1.38
Frank et al., 2000 (25)	60 leisure athletes (55 male; 5 female)	Plasma riboflavin; EGRAC	Plasma riboflavin 69.6 ± 18.9 nmol/L EGRAC 1.25 ± 0.12
Guilland et al., 1989 (29)	55 male well trained recreational athletes	7-d food record; EGRAC	2.1 mg/d 1.06
Keith & Alt, 1991 (44)	13 female athletes	3-d weighed food record; EGRAC; Urinary riboflavin	Dietary 1.9 ± 0.9 mg/d EGRAC 1.06 ± 0.06 Urine 98 ± 51 μg/24 h

Table 3 ***(continued)***

Study	Subjects	Assessment index	Results
Rokitzki et al., 1994 (73)	62 athletes (50 male; 12 female)	7-d weighed food record; EGRAC; Whole blood riboflavin; Urinary riboflavin	Dietary Male: marathon 2.2 mg/d wrestling 2.2 mg/d soccer 2.1 mg/d basketball 2.5 mg/d Female: handball 1.4 mg/d EGRAC Male: marathon 1.29 wrestling 1.15 basketball 1.27 Female: handball 1.32 Whole blood riboflavin Male: marathon 372 nmol/L wrestling 303 nmol/L soccer 359 nmol/L basketball 356 nmol/L Female: handball 353 nmol/L Urine riboflavin Male: marathon 2.67 µmol/g Cr wrestling 2.68 µmol/g Cr soccer 2.68 µmol/g Cr basketball 2.95 µmol/g Cr Female: handball 1.78 µmol/g Cr
Weight et al., 1988 (92)	30 well-trained male runners	5-d food records; EGRAC	Dietary 1.79 ± 1.80 mg/d EGRAC 1.02 ± 0.10

[a]All values are means or means ± standard deviation.

[b]Reference values: 1989 RDA for males 19-50 y = 1.7 mg; 1989 RDA for females 19-50 y = 1.3 mg; 1998 RDA for males 19-50 y = 1.3 mg; 1998 RDA for females 19-50 y = 1.1 mg; 1998 EAR for males 19-50 y = 1.1 mg; 1998 EAR for females 19-50 y = 0.9 mg; Erythrocyte glutathione reductase activity coefficient (EGRAC) = ≤ 1.2; Erythrocyte riboflavin ≥ 400 nmol/L; Urinary riboflavin ≥ 120 µg/d (28, 41, 61).

Keith and Alt (44) found that non-supplementing female athletes can maintain good riboflavin status from dietary intakes. Fogelholm (21) assessed riboflavin status in active and sedentary young women throughout a 24-wk fitness exercise program; EGRAC remained the same throughout the study. However, other research suggests that athletes may need more riboflavin than the general population. For example, using a cross-sectional design, Fogelhom et al. (24) found that 55% of their athletes (males and females combined) had poor status as determined by EGRAC. These data are supported by metabolic studies examining the effects of exercise, dieting, or dieting plus exercise on riboflavin requirements (7-9, 93); overall results have indicated that riboflavin needs are higher in females engaging in exercise for fitness compared to sedentary controls. In the 1980s, Belko et al. (7-9) conducted a series of metabolic studies examining the riboflavin status of active young women. In the first metabolic study, riboflavin requirements were assessed during a 12-wk feeding study (8). The study was divided into 6 wk of no exercise followed by 6 wk of moderate physical activity (20 to 50 min/d, 6 d/wk). Normal-weight women were fed a diet containing 0.6 mg riboflavin per 1000 kcal. Throughout the study, riboflavin intake was increased by 0.2 mg riboflavin per 1000 kcal. Riboflavin requirements were defined as the intake of riboflavin needed to keep the EGRAC < 1.25. Using linear regression analysis, riboflavin requirements increased to as high as 1.4 mg/1000 kcal (1998 RDA = 1.1 mg/d for adult women) during the physical activity phase of the study (41). Two subsequent metabolic studies also found that dieting (1200 to 1250 kcal/d) and dieting combined with fitness exercise increased riboflavin requirements in active women (7, 9). In the first study, the effect of exercise and dieting on riboflavin status was examined in moderately overweight, untrained women (9). Using a crossover design, participants were initially assigned into either an exercise or non-exercise group. The exercise consisted of 50 min of dance for 5 d/wk. The women consumed only 1200 kcal per day with a riboflavin concentration of 0.8 mg per 1000 kcal. Riboflavin depletion, assessed by increased EGRAC and decreased urinary excretion of riboflavin, occurred during both the non-exercise and exercise periods. Thus, both exercise and dieting increased riboflavin requirements in these women. In a follow-up study, estimates for riboflavin requirements were determined during non-exercise and exercise for dieting, overweight women (7). The women consumed either 0.96 mg riboflavin per 1000 kcal or 1.16 mg riboflavin per 1000 kcal. The study was a crossover design (exercise, non-exercise) at each level of riboflavin intake. At the lower riboflavin intake, the erythrocyte glutathione reductase activity coefficient increased in both the exercise and non-exercise groups. When subjects were both dieting for weight loss and exercising, 1.4 mg of riboflavin/d was required to maintain good riboflavin status. Even active older women (2.5 h exercise/wk) eating a weight-maintenance diet (1800 to 2000 kcal/d) required 1.8 mg/d of riboflavin to maintain good status (EGRAC < 1.20) (93). Based on these studies, it appears that exercise, dieting, and dieting plus exercise increase the need for riboflavin above the 1998 RDA for active women (7-9, 41, 93). However, it should be noted that these subjects performed moderate exercise (2.5 to 5 h/wk) for fitness. Unfortunately, there are no metabolic studies available examining riboflavin status in female athletes who participate in strenuous exercise and competitive sports; however, if moderately active females have an increased need for riboflavin, then female athletes who are even more active, would have just as great a need, if not greater. In another study,

riboflavin status declined during a short period of increased physical activity in adult men whose riboflavin status was marginal (81). Thus, based on the research to date, athletes may need more riboflavin than the general population and more than the current RDA. However, athletes can maintain good status for riboflavin when adequate energy is consumed.

Vitamin B-6

Vitamin B-6 plays an important role in the metabolic pathways required for exercise, principally amino acid metabolism and glycogen breakdown (51-53, 57, 59). The active form of vitamin B-6, pyridoxal phosphate (PLP), is required as a cofactor for enzymes involved in protein transamination and deamination reactions, thus more protein breakdown or increased protein consumption may increase the need for vitamin B-6. The breakdown of glycogen is regulated through glycogen phosphorylase, which also requires PLP.

Food Sources and Recommended Intakes. Rich sources of vitamin B-6 include animal foods, such as meat, fish, and poultry, and plant foods, such as bananas, navy beans, whole grains, and walnuts. The RDA for vitamin B-6 for men and women age 19 to 50 y is 1.3 mg while the UL for vitamin B-6 is 100 mg (41).

Assessment of Status. Several blood and urine indicators are used in the assessment of vitamin B-6 status, including plasma PLP and urinary concentrations of a B-6 metabolite, 4-pyridoxic acid (28). Individuals with adequate status of vitamin B-6 will have a PLP concentration ≥ 20 nmol/L and a urinary 4-pyridoxic acid concentration > 3.0 μmol/d (28, 41). Activity of erythrocyte alanine or aspartate amino transferase (EALT or EAST) is also used to determine status. Values suggesting good status of vitamin B-6 for EALT and EAST are < 1.25 and < 1.7, respectively (28, 41).

Assessment of Dietary Intake in Active Individuals. Table 4 summarizes studies examining dietary intake of vitamin B-6 in athletes. Some studies report vitamin B-6 intakes by athletes that are less than recommended guidelines, especially female athletes with low energy intakes (51, 75). For example, when dietary intakes of trained female cyclists were examined, more than a third consumed less than the RDA for vitamin B-6 (45). In addition, Steen et al. found that only 70% of female heavyweight rowers met the RDA for vitamin B-6 (83). A study of strength and speedpower German athletes found that more than 30% did not meet the dietary guidelines for vitamin B-6 for that country (1.8 mg/d male, 1.6 mg/d female) (75). However, Niekamp and Baer (62) documented better vitamin B-6 intakes in male cross-country runners with mean intake levels greater than the 1989 RDA. Because vitamin B-6 is concentrated in certain foods, elimination of meats, whole grains, legumes, and bananas will dramatically reduce the intake of vitamin B-6 in the diet. Thus, the degree to which an athlete is low in vitamin B-6 may depend on food preferences and selection.

Assessment of Status in Active Individuals. Table 4 summarizes studies examining vitamin B-6 status in athletes. Poor status in athletes has been documented in the research literature; studies examining vitamin B-6 status in male and female athletes report poor vitamin B-6 status ranging from 5 to 60% of the athletes (24,

Table 4 Summary of Studies Examining Vitamin B-6 Intake and/or Status in Adult Athletes[a,b]

Study	Subjects	Assessment index	Results
Vitamin B-6 intake			
Beals & Manore, 1998 (4)	24 female athletes with subclinical eating disorders; 24 female control athletes	7-d weighed food record	Athletes with subclinical eating disorders: 2.1 ± 0.8 mg/d Control athletes: 2.5 ± 0.8 mg/d
Beshgetoor & Nichols, 2003 (10)	25 female master cyclists and runners	4-d food record	3 ± 1 mg/d
Clark et al., 2003 (14)	13 female collegiate soccer players	2 3-d food records (preseason and post-season)	Pre-season: 1.8 ± 0.6 mg/d Post-season: 1.1 ± 0.6 mg/d
DeBolt et al., 1988 (18)	267 male US Navy SEAL trainees	1-d food record	2.7 mg/d
Faber & Benade, 1991 (20)	30 field athletes (discus-, hammer-, javelin-throwers, and shotputters) (20 male; 10 female)	7-d food record	Males: 2.84 ± 1.08 mg/d Females: 1.62 ± 0.42 mg/d
Jensen et al., 1992 (42)	14 male endurance cyclists	5-d weighed food record while training; 3-d weighed food record while racing	Training: 5 ± 2 mg/d Racing: 6 ± 3 mg/d
Kaiserauer et al., 1989 (43)	17 female distance runners (8 with amenorrhea; 9 with eumenorrhea)	3-d food record	Amenorrheic distance runners: 1.5 mg/d Eumenorrheic distance runners: 2.1 mg/d
Keith et al., 1989 (45)	8 female highly trained cyclists	3-d weighed food record	1.8 ± 1.1 mg/d
Kopp-Woodroffe et al., 1999 (47)	4 female amenorrheic runners	7-d weighed food record	1.7 ± 0.5 mg/d
Leydon & Wall, 2002 (50)	20 jockeys (6 male; 14 female)	7-d weighed food record	Males: 1.06 ± 0.30 mg/d Females: 0.90 ± 0.49 mg/d

Table 4 ***(continued)***

Study	Subjects	Assessment index	Results
Manore et al., 1989 (55)	10 female long distance runners	3-d food record	1.8 ± 0.9 mg/d
Niekamp & Baer, 1995 (62)	12 male runners	2 4-d food record	2.5 ± 2.0 mg/d
Nieman et al., 1989 (63)	347 marathon runners (291 male; 56 female)	3-d food record	Males: 2.58 mg/d Females: 1.64 mg/d
Rankinen et al., 1998 (68)	21 Finnish elite male ski jumpers	4-d food record	2.1 ± 0.9 mg/d
Rico-Sanz et al., 1998 (70)	8 male elite soccer players	12-d food record	3.30 ± 0.74 mg/d
Saris et al., 1989 (78)	5 male cyclists during the Tour de France	22-d food record while racing	Dietary 2.4 ± 0.7 mg/d
Worme et al., 1990 (94)	71 triathletes (50 male; 21 female)	3-d food record	Males: 2.6 mg/d Females: 2.0 mg/d
Vitamin B-6 status			
Fogelholm et al., 1993 (24)	42 physically active college students (24 male; 18 female)	Blood EASTAC	Males: 2.08 Females: 1.92
Gaume et al., 2005 (27)	12 trained male athletes	Quantitative food frequency questionnaire; Plasma homocysteine	Dietary 2.05 ± 0.10 mg/d Blood (homocysteine) 7.48 ± 0.4 μmol/L
Guilland et al., 1989 (29)	55 male well trained recreational athletes	7-d food records; EASTAC; Plasma PLP	Dietary 1.5 mg/d Blood EASTAC: 1.95 PLP: 66.5 nmol/L
Leonard & Leklem, 2000 (49)	11 ultramarathoners (8 male; 3 female)	Plasma PLP; Plasma 4-pyridoxic acid	PLP 41.1 ± 14.2 nmol/L 4-pyridoxic acid 25.5 ± 7.6 nmol/L

(continued)

Table 4 ***(continued)***

Study	Subjects	Assessment index	Results
Rokitzki et al., 1994 (74)	13 male endurance athletes	7-d weighed food record; Whole blood; serum B-6; EASTAC; Urinary 4-pyridoxic acid	Dietary 2.13 mg/d Whole blood vitamin B-6 0.05 nmol/mL Serum vitamin B-6 0.07 nmol/mL EASTAC 1.38 Urinary B-6 4.48 µmol/g Cr
Rokitzki et al., 1994 (75)	57 athletes (45 male; 12 female)	7-d weighed food record; EASTAC; Whole blood vitamin B-6; Urinary 4-pyridoxic acid	Dietary Male: body building 5.40 mg/d wrestling 1.72 mg/d soccer 2.10 mg/d basketball 2.12 mg/d Female: handball 1.36 mg/d EASTAC Male: body building 1.00 wrestling 1.38 basketball 1.43 Female: handball 1.43 Whole blood vitamin B-6 Male: body building 0.404 nmol/mL wrestling 0.043 nmol/mL soccer 0.046 nmol/mL basketball 0.053 nmol/mL Female: handball 0.037 nmol/mL Urine Male: body building 10.43 µmol/g Cr wrestling 4.15 µmol/g Cr soccer 3.49 µmol/g Cr basketball 3.59 µmol/g Cr Female: handball 3.22 µmol/g Cr

Table 4 ***(continued)***

Study	Subjects	Assessment index	Results
Rousseau et al., 2005 (76)	74 competitive male athletes	7-d food record; Plasma homocysteine	Dietary Athletes performing intermittent activity: 2.28 ± 0.73 mg/d Athletes performing anaerobic activity: 2.53 ± 0.99 mg/d Athletes performing aerobic activity: 3.09 ± 1.66 mg/d Blood (homocysteine) Athletes performing intermittent activity: 10.6 ± 2.6 μmol/L Athletes performing anaerobic activity: 9.6 ± 1.7 μmol/L Athletes performing aerobic activity: 9.2 ± 2.0 μmol/L

[a]All values are means or means ± standard deviation.

[b]Reference values: 1989 RDA for males 19-50 y = 2.0 mg; 1989 RDA for females 19-50 y = 1.6 mg; 1998 RDA for males and females 19-50 y = 1.3 mg; 1998 EAR for males and females 19-50 y = 1.1 mg; 1998 UL for males and females 19-50 y = 100 mg; Pyridoxal phosphate (PLP) ≥ 20 nmol/L; Erythrocyte aspartate aminotransferase (EAST) = < 1.6; Erythrocyte alanine aminotransferase (EALT) < 1.25; Plasma homocysteine < 16 μmol/L; Urinary 4-pyridoxic acid > 3.0 μmol/d (28, 41, 61).

51, 53, 75, 86). For example, Telford et al. (86) studied 86 male and female athletes before and after an 8-month training period. At study entry, they found that 60% of the athletes had poor baseline vitamin B-6 status while consuming their typical diets. Roughly half of the subjects ($n = 42$) then consumed a multivitamin/mineral supplement and half took a placebo ($n = 44$). At the end of the 8-month study, 41% of the athletes assigned to the placebo still had poor vitamin B-6 status while only 10% of those athletes assigned to the multivitamin/mineral supplement were still in poor vitamin B-6 status. Raczynski and Szczepanska (67) also examined vitamin B-6 status in 1753 elite male and female Polish athletes from the years 1987 to 1992. The risk of vitamin deficiency averaged 9% in all athletes over the 6-y period, with an annual range of 4 to 15%. The highest prevalence of vitamin B-6 deficiency was seen in endurance athletes (13%) and in those athletes participating in team sports (10%). They also observed that the risk of vitamin B-6 deficiency was highest in pre-Olympic years (16%) and lowest in Olympic years (3%), suggesting that some athletes had poor vitamin B-6 status while consuming their typical diets.

Metabolic research studies have documented that approximately 1.5 to 2.3 mg/d of vitamin B-6 is required to maintain good vitamin B-6 status in sedentary

individuals (30, 31, 39, 48, 56). This level of vitamin B-6 is higher than the current RDA of 1.3 mg/d for men and women (41). Research has also documented that acute exercise alters vitamin B-6 metabolism by increasing blood concentrations of PLP (16, 37, 48, 56, 57), which in turn increases the probability that PLP will be converted to 4-pyridoxic acid and lost in the urine during exercise (16, 37, 48, 56). Thus, exercise may increase the turnover and loss of vitamin B-6 from the body (16, 56). At least one study has documented higher 4-pyridoxic acid losses in active individuals compared to sedentary controls or periods of inactivity (56); higher 4-pyridoxic acid losses have also been documented after strenuous physical activity (16). Using 4-pyridoxic acid excretion concentrations, Rokitzki et al. (74) calculated that marathon runners lost approximately 1 mg of vitamin B-6 during a race (26.2 miles). Thus, based on the current research literature, some active individuals, depending on training level, may require 1.5 to 2.5 times the current RDA (approximately 2.0 to 3.0 mg/d) for vitamin B-6 to maintain good B-6 status.

Folate

Folate is required for a number of enzymes that are critical for DNA synthesis and amino acid metabolism (57, 59). In addition, folate's role in assisting with cell division makes it a critical nutrient for growth, the synthesis of new cells, such as red blood cells, and for the repair of damaged cells and tissues. Thus, folate requirements might be higher with exercise, since damaged muscle tissue needs to be repaired. Finally, folate, vitamin B-12, and vitamin B-6 are closely interrelated in the metabolism of methionine, an essential amino acid. If these key B-vitamins are not available, homocysteine, an intermediate metabolite in methionine metabolism, increases. High blood concentrations of homocysteine have been associated with an increased risk of cardiovascular disease (12, 13, 15, 71). However, recent large clinical trials have found that supplementing with folic acid, vitamin B-6, and vitamin B-12 did not reduce the risk of cardiovascular events (11, 33). Thus, other relationships in the intermediates in this metabolic pathway should be explored.

Food Sources and Recommended Intakes. Rich sources of dietary folate include leafy green vegetables, fortified cereals and grains, nuts, legumes, liver, and brewer's yeast. If these types of foods are not consumed in the diet, dietary supplementation may be required. The RDA for folate for adults is 400 µg/d of dietary folate equivalents (DFE) while the UL for folate is 1000 µg/d of synthetic folic acid (41). Because of folate's critical role during the first few weeks of pregnancy and because many women of child-bearing age do not consume adequate folate, the fortification of breads and cereals with folic acid was initiated. Thus, ready-to-eat cereals, bread, and other grain products are among the primary sources of folate in the US.

Assessment of Status. Folate status is assessed by measuring serum folate and red blood cell folate concentrations (28). Normal concentrations of serum and erythrocyte folate are ≥ 7 nmol/L and ≥ 305 nmol/L, respectively (28, 41). With a folate deficiency, the ability to make red blood cells becomes impaired and can eventually lead to megaloblastic anemia, as the megaloblasts fail to replicate into functional red blood cells. This results in large red blood cells that cannot effectively transport oxygen. Another measure of folate status is mean corpuscular volume (MCV), which is an indication of red blood cell size (28). Fortunately, folic acid fortification has markedly improved folate status in the US (26).

Assessment of Dietary Intake in Active Individuals. Table 5 summarizes studies examining folate intakes in adult athletes. Active men typically have adequate intakes of folate due to high energy intakes (57). Conversely, dietary intakes of folate in active women are consistently low, ranging from 126 to 364 µg/d (1, 4, 20, 45, 63, 94). For example, using 7-d weighed food records, Beals and Manore (4) reported mean folate intakes for these female athletes (n = 48) to be less than 400 µg/d. Ziegler et al. (97) found poor mean dietary intakes of folate for male (234 µg/d) and female (275 µg/d) figure skaters, which can be attributed to lower energy intakes. However, Niekamp and Baer (62) found that the mean intake of male distance runners was 454 µg/d. Studies completed after the FDA mandatory fortification of folic acid should report an improvement in dietary intakes for folate. Unfortunately, Clark et al. (14) reported that female soccer players have mean folate intakes less than the 1998 EAR during pre- and post-season play.

Table 5 Summary of Studies Examining Folate Intake and/or Status in Adult Athletes[a,b]

Study	Subjects	Assessment index	Results
Folate intake			
Beshgetoor & Nichols, 2003[c] (10)	25 female master cyclists and runners	4-d food record	402 ± 115 µg/d
Clark et al., 2003[c] (14)	13 female collegiate soccer players	2 3-d food records (pre-season and post-season)	Pre-season: 271 ± 130 µg/d Post-season: 186 ± 113 µg/d
DeBolt et al., 1988 (18)	267 male US Navy SEAL trainees	1-d food record	420 µg/d
Faber & Benade, 1991 (20)	30 field athletes (discus-, hammer-, javelin-throwers, and shotputters) (20 male; 10 female)	7-d food record	Males: 407 ± 207 µg/d Females: 230 ± 64 µg/d
Jensen et al., 1992 (42)	14 male endurance cyclists	5-d weighed food record while training; 3-d weighed food record while racing	Training: 683 ± 230 µg/d Racing: 925 ± 386 µg/d
Kaiserauer et al., 1989 (43)	17 female distance runners (8 with amenorrhea; 9 with eumenorrhea)	3-d food record	Amenorrheic distance runners: 198.5 µg/d Eumenorrheic distance runners: 276.4 µg/d

(continued)

Table 5 ***(continued)***

Study	Subjects	Assessment index	Results
Keith et al., 1989 (45)	8 female highly trained cyclists	3-d weighed food record	303 ± 305 µg/d
Kopp-Woodroffe et al., 1999[c] (47)	4 female amenorrheic runners	7-d weighed food record	250 ± 105 µg/d
Leydon & Wall, 2002 (50)	20 jockeys (6 male; 14 female)	7-d weighed food record	Males: 146 ± 60 µg/d Females: 132 ± 52 µg/d
Manore et al., 1989 (55)	10 female long distance runners	3-d food record	252.7 ± 111.4 µg/d
Niekamp & Baer, 1995 (62)	12 male runners	2 4-d food records	452.0 ± 181.8 µg/d
Nieman et al., 1989 (63)	347 marathon runners (291 male; 56 female)	3-d food record	Males: 367 µg/d Females: 266 µg/d
Rankinen et al., 1998[c] (68)	21 Finnish elite male ski jumpers	4-d food record	286 ± 107 µg/d
Rico-Sanz et al., 1998[c] (70)	8 male elite soccer players	12-d food record	905 ± 286 µg/d
Spodaryk et al., 1996 (82)	40 trained female athletes	7-d food record	189.9 ± 33 µg/d
Worme et al., 1990 (94)	71 triathletes (50 male; 21 female)	3-d food record	Males: 386 µg/d Females: 302 µg/d
Folate status			
Beals & Manore, 1998[c] (4)	24 female athletes with subclinical eating disorders; 24 female control athletes	7-d weighed food record; Serum folate	Dietary Athletes with subclinical eating disorders: 306 ± 157 µg/d Control athletes: 364 ± 99 µg/d Blood Athletes with subclinical eating disorders: 20.2 ± 7.3 nmol/L Control athletes: 19.3 ± 8.4 nmol/L
Gaume et al., 2005[c] (27)	12 trained male athletes	Quantitative food frequency questionnaire; Plasma homocysteine	Dietary 287 ± 15 µg/d Blood (homocysteine) 7.48 ± 0.4 µmol/L

Table 5 ***(continued)***

Study	Subjects	Assessment index	Results
Konig et al., 2003[c] (46)	42 male triathletes	Plasma folate; Plasma homocysteine	Plasma folate: 6.4 ± 2.3 nmol/L Plasma homocysteine: 12.3 ± 2.0 µmol/L
Real et al., 2005[c] (69)	22 male marathon runners	Plasma folate; plasma homocysteine	Plasma folate 27.01 ± 10.21 nmol/L Plasma homocysteine 8.6 ± 1.8 µmol/L
Rousseau et al., 2005[c] (76)	74 competitive male athletes	7-d food record; plasma homocysteine	Dietary Athletes performing intermittent activity: 295 ± 105 µg/d Athletes performing anaerobic activity: 298 ± 100 µg/d Athletes performing aerobic activity: 434 ± 220 µg/d Plasma homocysteine Athletes performing intermittent activity: 10.6 ± 2.6 µmol/L Athletes performing anaerobic activity: 9.6 ± 1.7 µmol/L Athletes performing aerobic activity: 9.2 ± 2.0 µmol/L
Telford et al., 1992 (86)	86 athletes (50 male; 36 female)	Blood folate	Males: 11.1 ± 1.6 nmol/L Females: 14.2 ± 1.8 nmoL/L
Weight et al., 1988 (92)	30 well-trained male runners	5-d food records; Serum folate; RBC folate	Dietary 264.6 ± 100.1 µg/d Serum folate 11.1 ± 4.3 nmol/L RBC folate 630 ± 148 nmol/L

[a]All values are means or means ± standard deviation.

[b]Reference values: 1989 RDA for males 19-50 y = 200 µg; 1989 RDA for females 19-50 y = 180 µg; 1998 RDA for males and females 19-50 y = 400 µg of Dietary Folate Equivalents (DFE); 1998 EAR for males and females 19-50 y = 320 µg of DFE; 1998 UL for males and females 19-50 y = 1000 µg of synthetic folic acid; Erythrocyte folate = ≥ 305 nmol/L; Serum folate = ≥ 7 nmol/L; Plasma homocysteine ≤ 16 µmol/L (28, 41, 61).

[c]Paper published after the 1998 FDA mandatory fortification of grain products with folic acid.

Assessment of Status in Active Individuals. Table 5 summarizes studies examining folate status in adult athletes. Research examining folate status in athletes is limited. Matter et al. (58) examined folate status in non-supplementing female marathon runners and found that 33% ($n = 85$) had poor folate status; however, no assessment of dietary folate was completed in this study. When Beals and Manore (4) examined folate status in their female athletes (~ 50% reported some type of supplementation or use of fortified food), 8% of their athletes had poor folate status (plasma folate ≤ 6.8 nmol/L). In this study, supplementation may have contributed to the adequate folate status of some athletes, because mean dietary folate intakes were low. Recently, Herrmann et al. (34) examined folate status in 72 recreational endurance athletes (64 men and 8 women) and 46 inactive controls (34 men and 12 women); no differences were seen between groups for blood folate concentrations. Unfortunately, the researchers did not examine dietary intakes of folate in this study. Based on the limited research, it is recommended that all athletes, both males and females, increase their daily intake of folate to the current RDA of 400 μg/d. For some individuals, this may mean that folic acid supplements need to be consumed.

Research shows that athletes, especially women, have poor intakes and status of folate. However, it has not been clearly documented that supplementing with folate will improve exercise performance. Matter et al. supplemented female marathon runners in poor folate status (low serum folate concentrations) with 5 mg of folic acid for 10 wk (58). Although hematological parameters for folate improved, exercise performance on a treadmill did not. Thus, more research is needed to determine the relationship between folate and exercise performance.

Vitamin B-12

Vitamin B-12 is one of a number of coenzymes that assist with DNA synthesis, which is necessary for the formation of red blood cells (57). A deficiency in vitamin B-12 can lead to megaloblastic anemia, which is characterized by large red blood cells similar to that seen with folate deficiency (57). Most vitamin B-12 deficiencies reflect inadequate absorption, rather than poor intakes. The inadequate absorption typically occurs due to a lack of hydrochloric acid or intrinsic factor (28). Hydrochloric acid is required to release the vitamin from dietary proteins so that it can bind with intrinsic factor before absorption. Vitamin B-12 deficiency caused by a lack of intrinsic factor is known as pernicious anemia. Vitamin B-12 is also essential for the nervous system because it helps maintain the sheath that coats nerve fibers. If these nerve fibers are damaged or altered, conduction of nervous signals is interrupted causing numerous neurological problems. As mentioned earlier, adequate levels of vitamin B-12, folate, and vitamin B-6 are also necessary for the metabolism of methionine and keeping blood levels of homocysteine low (57).

Food Sources and Recommended Intakes. The best dietary sources include meats, fish, poultry, shellfish, eggs, milk, and milk products. Because vitamin B-12 is found almost exclusively in animal products, an athlete following a vegetarian diet needs to include food sources that will provide vitamin B-12 (2). Fortunately, many vegetarian food products are fortified with vitamin B-12. The risk of poor vitamin B-12 status is low in active individuals unless animal products are avoided and no supplementation is occurring. However, many older adults may malabsorb vitamin B-12 due to low gastric acidity. Thus, it is recommended that older adults

obtain the majority of their vitamin B-12 from fortified foods or a supplement containing vitamin B-12. The RDA for vitamin B-12 for adults is 2.4 µg/d while no UL for vitamin B-12 has been determined (41).

Assessment of Status. Assessment of vitamin B-12 status includes measuring the vitamin B-12 transport proteins, transcobalamin II and haptocorrins, and measuring the concentration of vitamin B-12 bound to the transport protein (holotranscobalamin II and holohaptocorrins) (28). In addition, serum vitamin B-12 and MCV can also be used. Normal concentrations of serum vitamin B-12 are > 120 pmol/L (41).

Assessment of Dietary Intake in Active Individuals. Table 6 summarizes studies examining vitamin B-12 intakes in adult athletes. Limited research has examined vitamin B-12 intakes and status in active individuals. Keith et al. (45) found that more than 33% of highly trained female cyclists did not consume the recommended intake for vitamin B-12, while Ziegler et al. (97) found the vitamin B-12 intakes in their male and female figure skaters were less than that reported by the Third National Health and Nutrition Examination Survey (NHANES III). Steen et al. (83) found that 80% of female collegiate heavyweight rowers met the RDA for vitamin B-12. As mentioned above, those athletes that eliminate animal products from the diet will be at greatest risk for poor vitamin B-12 intakes.

Table 6 Summary of Studies Examining Vitamin B-12 Intake and/or Status in Adult Athletes[a,b]

Study	Subjects	Assessment index	Results
Vitamin B-12 intake			
Beshgetoor & Nichols, 2003 (10)	25 female master cyclists and runners	4-d food record	6 ± 2 µg/d
Clark et al., 2003 (14)	13 female collegiate soccer players	2 3-d food records (pre-season and post-season)	Pre-season: 4.5 ± 1.9 µg/d Post-season: 2.1 ± 1.7 µg/d
Faber & Benade, 1991 (20)	30 field athletes (discus-, hammer-, javelin-throwers, and shotputters) (20 male; 10 female)	7-d food record	Males: 26.9 ± 42.0 µg/d Females: 5.26 ± 2.29 µg/d
Jensen et al., 1992 (42)	14 male endurance cyclists	5-d weighed food record while training; 3-d weighed food record while racing	Training: 8 ± 6 µg/d Racing: 11 ± 7 µg/d

(continued)

Table 6 ***(continued)***

Study	Subjects	Assessment index	Results
Kaiserauer et al., 1989 (43)	17 female distance runners (8 with amenorrhea; 9 with eumenorrhea)	3-d food record	Amenorrheic distance runners: 2.8 µg/d Eumenorrheic distance runners: 4.1 µg/d
Keith et al., 1989 (45)	8 female highly trained cyclists	3-d weighed food record	3.3 ± 4.7 µg/d
Kopp-Woodroffe et al., 1999 (47)	4 female amenorrheic runners	7-d weighed food record	3.2 ± 1.1 µg/d
Leydon & Wall, 2002 (50)	20 jockeys (6 male; 14 female)	7-d weighed food record	Males: 2.90 ± 0.90 µg/d Females: 2.15 ± 1.07 µg/d
Manore et al., 1989 (55)	10 female long distance runners	3-d food record	2.5 ± 1.3 µg/d
Nieman et al., 1989 (63)	347 marathon runners (291 male; 56 female)	3-d food record	Males: 5.98 µg/d Females: 2.98 µg/d
Rico-Sanz et al., 1998 (70)	8 male elite soccer players	12-d food record	10.7 ± 9.2 µg/d
Weight et al., 1988 (92)	30 well-trained male runners	5-d food record	4.95 ± 3.2 µg/d
Worme et al., 1990 (94)	71 triathletes (50 male; 21 female)	3-d food record	Males: 5.1 µg/d Females: 4.9 µg/d
Vitamin B-12 status			
Beals & Manore, 1998 (4)	24 female athletes with subclinical eating disorders; 24 female control athletes	7-d weighed food record; Serum B-12	Dietary Athletes with subclinical eating disorders: 3.9 ± 2.6 µg/d Control athletes: 4.3 ± 1.9 µg/d Blood Athletes with subclinical eating disorders: 254 ± 77 pmol/L Control athletes: 331 ± 172 pmol/L
Gaume et al., 2005 (27)	12 trained male athletes	Quantitative food frequency questionnaire; plasma homocysteine	Dietary 7.5 ± 0.4 mg/d Blood (homocysteine) 7.48 ± 0.4 µmol/L

Table 6 ***(continued)***

Study	Subjects	Assessment index	Results
Konig et al., 2003 (46)	42 male triathletes	Plasma B-12; plasma homocysteine	Plasma B-12: 362 ± 104 pmol/L Plasma homocysteine: 12.3 ± 2.0 µmol/L
Real et al., 2005 (69)	22 male marathon runners	Plasma vitamin B-12; plasma homocysteine	Plasma vitamin B-12 409.6 ± 106 pmol/L Plasma homocysteine 8.6 ± 1.8 µmol/L
Rousseau et al., 2005 (76)	74 competitive male athletes	7-d food record; plasma homocysteine	Dietary Athletes performing intermittent activity: 5.87 ± 3.63 µg/d Athletes performing anaerobic activity: 4.91 ± 1.37 µg/d Athletes performing aerobic activity: 8.06 ± 6.34 µg/d Blood (homocysteine) Athletes performing intermittent activity: 10.6 ± 2.6 µmol/L Athletes performing anaerobic activity: 9.6 ± 1.7 µmol/L Athletes performing aerobic activity: 9.2 ± 2.0 µmol/L
Telford et al., 1992 (86)	86 athletes (50 male; 36 female)	Plasma B-12	Males: 299 ± 32 pmol/L Females: 299 ± 35 pmol/L

[a]All values are means or means ± standard deviation.

[b]Reference values: 1989 RDA for males and females 19-50 y = 2.0 µg; 1998 RDA for males and females 19-50 y = 2.4 µg; 1998 EAR for males and females 19-50 y = 2.0 µg; Serum vitamin B-12 = > 120 pmol/L; Serum methylmalonic acid = 73 to 271 nmol/L; Plasma homocysteine < 16 µmol/L (28, 41, 61).

Assessment of Status in Active Individuals. Table 6 summarizes studies examining vitamin B-12 status in adult athletes. As with assessment of dietary intakes of vitamin B-12 in athletes, there are few reports assessing vitamin B-12 status. Beals and Manore (4) examined vitamin B-12 status in their female athletes and found no athlete with poor vitamin B-12 status; however, about half of the athletes reported taking a multivitamin supplement, which would contain vitamin B-12. In an earlier study, Telford et al. (86) reported that 5% of their athletes had poor vitamin B-12 status, but they did not report vitamin B-12 intakes. Recently, Herrmann et al. (34) reported altered vitamin B-12 metabolism in recreational athletes compared

to sedentary controls. Thus, more research is needed to understand vitamin B-12 status and metabolism in athletes, especially older active individuals who may have decreased ability to absorb vitamin B-12.

As mentioned earlier, elevated blood homocysteine concentrations are considered another risk factor for cardiovascular disease (12, 13, 15, 71). There is a strong inverse relationship between blood homocysteine concentrations and dietary intake and/or blood measures of folate, vitamin B-12, and vitamin B-6 (3, 13, 66, 72, 79); however, little is known about the effects of exercise on blood homocysteine concentrations. Research examining the effect of chronic exercise on homocysteine found that highly active individuals (men and women) had significantly lower homocysteine concentrations than their sedentary counterparts (65); however, this study did not report B-vitamin intake or B-vitamin status. Conversely, recent research has documented the effects of acute strenuous endurance exercise on blood homocysteine concentrations and found that running a marathon significantly increased homocysteine concentrations, while mountain biking (120 km) or running (100 km) had no effect (35). These same researchers also found that 3 wk of strenuous swimming increased blood homocysteine (36); thus, changes in training volume may also impact homocysteine. Other research suggests that acute exercise does not seem to have any impact on plasma homocysteine in healthy young men (95). Konig et al. (46) also examined the influence of the training volume on plasma homocysteine, folate, and vitamin B-12. They found that intense exercise acutely increased plasma homocysteine concentrations; however, athletes with the highest training volumes exhibited a significant decrease in plasma homocysteine concentrations. Other research suggests that the combined effects of chronic physical exercise and a high folate and vitamin B-12 intake could help reduce plasma homocysteine concentrations and possibly prevent many chronic diseases (27). More research is needed to determine the impact of physical activity on homocysteine concentrations and B-vitamin status for athletes.

Summary

The B-vitamins play important roles in maintaining the health of the active individual and assuring that energy can be produced for physical activity. Athletes who have poor or marginal nutritional status for a B-vitamin may have decreased ability to perform exercise at high intensities. However, the studies examining these issues in athletes, while controlling for diet and confounding factors, are limited. More controlled studies, especially metabolic studies, are needed in this area using highly active individuals. In addition, the assessment of status for many micronutrients is expensive and time consuming, making it unrealistic to screen athletes on a regular basis. If better methods were available for assessment, we could more quickly see which athletes had poor status and recommend changes in diet and/or supplementation.

Many athletes, especially young athletes, do not appreciate the importance of diet for performance, maintenance of a healthy immune response, and for recovery from injury and illness. These same athletes typically do not know how to buy and prepare healthy foods for themselves, especially if they live away from home. Long periods of poor nutrition status can have serious consequences for the athlete and

active individual. To obtain adequate status of the B-vitamins, nutrient-rich foods, such as enriched and whole grains, fruits, vegetables, and lean meats, should be selected and energy intake should be adequate to maintain weight. Athletes who have poor diets, especially those restricting energy intakes or eliminating food groups from the diet, should consider supplementing with a multivitamin/mineral supplement. Current research suggests that exercise may increase the requirements for riboflavin and vitamin B-6; however, more research is needed to further determine if exercise increases the need for folate and vitamin B-12.

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