Effect of physical activity on thiamine, riboflavin, and vitamin B-6 requirements\textsuperscript{1–3}

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ABSTRACT Because exercise stresses metabolic pathways that depend on thiamine, riboflavin, and vitamin B-6, the requirements for these vitamins may be increased in athletes and active individuals. Theoretically, exercise could increase the need for these micronutrients in several ways: through decreased absorption of the nutrients; by increased turnover, metabolism, or loss of the nutrients; through biochemical adaptation as a result of training that increases nutrient needs; by an increase in mitochondrial enzymes that require the nutrients; or through an increased need for the nutrients for tissue maintenance and repair. Biochemical evidence of deficiencies in some of these vitamins in active individuals has been reported, but studies examining these issues are limited and equivocal. On the basis of metabolic studies, the riboflavin status of young and older women who exercise moderately (2.5–5 h/wk) appears to be poorer in periods of exercise, dieting, and dieting plus exercise than during control periods. Exercise also increases the loss of vitamin B-6 as 4-pyridoxic acid. These losses are small and concomitant decreases in blood vitamin B-6 measures have not been documented. There are no metabolic studies that have compared thiamine status in active and sedentary persons. Exercise appears to decrease nutrient status even further in active individuals with preexisting marginal vitamin intakes or marginal body stores. Thus, active individuals who restrict their energy intake or make poor dietary choices are at greatest risk for poor thiamine, riboflavin, and vitamin B-6 status. Am J Clin Nutr 2000;72(suppl):598S–606S.

KEY WORDS Thiamine, riboflavin, vitamin B-6, exercise, physical activity, nutrient requirements, dietary reference intakes, recommended dietary allowances, RDA, DRI

INTRODUCTION

Two questions are frequently asked by persons engaged in physical activity: 1) Does exercise increase the need for certain vitamins? and 2) Does vitamin supplementation improve exercise performance? Because thiamine, riboflavin, and vitamin B-6 (pyridoxine) are cofactors for many metabolic reactions that produce energy, these question are particularly relevant. Until recently, the recommended dietary allowances (RDAs) for these nutrients were determined on the basis of energy (thiamine and riboflavin) and protein (vitamin B-6) intakes (1). As a person becomes more physically active, it is logical to assume that both energy and protein intakes will increase along with the intakes of these vitamins. Unfortunately, this is not always true. In persons who make poor dietary choices, these micronutrients may not increase with increased energy and protein intakes. Conversely, if persons increase their physical activity and restrict their energy intake, the need for these vitamins may increase further.

This article reviews the current literature to determine whether physical activity increases the need for thiamine, riboflavin, and vitamin B-6 because of training-induced increases in the conversion or metabolism of these vitamins. First, the dietary sources for each of these vitamins is reviewed briefly. Second, exercise-related functions and requirements, measurements to assess status, and the theoretical rationale for increased need are briefly covered. Third, the effect of vitamin deficiency or marginal vitamin status on exercise performance and work is reviewed. Finally, each vitamin is considered relative to the nutrient intakes of and available data on the nutrient status and requirements of active individuals. Because research data are limited, this paper did not examine whether exercise increases the requirements of or alters the nutrient status of the other B-complex vitamins (niacin, folate, vitamin B-12, pantothenic acid, and biotin).

DIETARY SOURCES

Thiamine, riboflavin, and vitamin B-6 are water-soluble B-complex vitamins found in a variety of animal and vegetable products. Thiamine is found abundantly in lean pork, yeast, legumes, and enriched cereals and breads (1, 2). Riboflavin is found in eggs, lean meats, milk, milk products, broccoli, and enriched breads and cereals (1, 3). Vitamin B-6 is abundant in meats, especially chicken and tuna, and in plant foods such as beans, cereals, and brown rice (1, 4). In a mixed diet, all 3 nutrients have high bioavailability (75–95%) (5). These vitamins are also frequently added to commercially prepared foods at 25–100% of the RDA per serving. Thus, the consumption of fortified cereals, breakfast bars, sport bars and drinks, energy shakes or meal-replacement products will dramatically increase total dietary intakes. Frequently, individuals who “watch their

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weight” or engage in physical activity are users of such products. In addition, multivitamin or vitamin or mineral supplements usually contain ≥100% of the RDA for these nutrients. It is estimated that Americans spend > $5 billion/y on vitamin, mineral, herbal, and dietary supplements (6). Indeed, out-of-pocket expenditures for high-dose megavitamins in 1997 alone were estimated at $3.3 billion (7). Some of the highest supplement use is by persons engaged in physical activity, of whom it has been estimated that as many as 50% are taking supplements (8–10). Thus, the total intake of these vitamins by active Americans may be increasing regardless of dietary choices.

**EXERCISE-RELATED FUNCTIONS AND REQUIREMENTS**

Thiamine, as thiamine pyrophosphate, plays an important role in the metabolism of both carbohydrate and the branched-chain amino acids. It is a coenzyme for pyruvate dehydrogenase (lipooxinamide), which catalyzes the conversion of pyruvate to acetyl CoA. Thiamine is also a coenzyme for oxoglutarate dehydrogenase (lipooxinamide), an enzyme responsible for the formation of succinyl CoA in the tricarboxylic acid cycle, and for branched-chain decarboxylase, an enzyme responsible for the catabolism of the branched-chain amino acids. Physical activity stresses these energy-producing metabolic pathways (11–13). Because thiamine requirements are linked to energy metabolism, the 1989 RDA for thiamine was expressed in terms of energy intake (0.12 mg/MJ, or per 239 kcal) (1). The 1998 DRI for thiamine is 1.2 mg/d for men and 1.1 mg/d for women aged 19–50 y (5). An additional 0.3 and 0.4 mg/d are recommended during pregnancy and lactation, respectively (5).

Riboflavin is necessary for the synthesis of 2 important coenzymes—flavin mononucleotide and flavin adenine dinucleotide (FAD). These coenzymes are especially important in the metabolism of glucose, fatty acids, glycerol, and amino acids for energy. Paralleling its effect on thiamine, physical activity stresses the biochemical pathways involved in the metabolism of these substrates (3, 13, 14). Riboflavin is also involved in the conversion of vitamin B-6 to its functional coenzyme (1, 5, 15, 16). When the 1989 RDA was set, the dietary requirement for riboflavin was expressed in terms of energy intake (0.14 mg/MJ, or per 239 kcal) for people of all ages, with a minimum intake of 1.2 mg/d (1). The 1998 DRI for riboflavin is 1.3 mg/d for men and 1.1 mg/d for women aged 19–70 y (5). An additional 0.3 mg/d is recommended during pregnancy and an additional 0.5 mg/d is recommended during lactation (5).

A major function of vitamin B-6 is the metabolism of proteins and amino acids. The most biologically active form of vitamin B-6 is pyridoxal 5’-phosphate (PLP). PLP is a cofactor for transaminases, decarboxylases, and other enzymes used in the metabolic transformations of amino acids and nitrogen-containing compounds. During exercise, the gluconeogenic process involves the breakdown of amino acids for energy in the muscle and the conversion of lactic acid to glucose in the liver. Various PLP-containing enzymes are involved in this metabolically driven conversion.

Another function of vitamin B-6 directly related to energy production during exercise is the breakdown of muscle glycogen. Adequate vitamin B-6 must be present to release glucose-1-phosphate from muscle glycogen (4, 17, 18). Because vitamin B-6 is directly involved in amino acid metabolism, the requirements for vitamin B-6 are frequently expressed in terms of protein intake. In the past, a dietary intake of 0.016 mg vitamin B-6/g protein was considered adequate to ensure good vitamin B-6 status (4), but recent research suggests that 0.019 mg vitamin B-6/g protein may be required to maintain good status (19, 20). In sedentary lactoovovegetarian women, 1.9 mg vitamin B-6/d was required to maintain good vitamin B-6 status (19). The link between protein intake and vitamin B-6 requirements is especially important for athletes because they typically have a higher protein requirement than do sedentary individuals (21) and generally have higher protein intakes because of their higher energy intakes.

The 1989 RDA for vitamin B-6 for adults aged ≥25 y was 2.0 mg/d for men and 1.6 mg/d for women (1). These recommendations were developed by multiplying the average protein intake of men (126 g) and women (100 g) by 0.016. Typical intakes of protein are higher than the 1989 RDA for protein of 63 g/d for men and 50 g/d for women (1). In 1998, the DRI for vitamin B-6 was decreased to 1.3 mg/d for men and women aged 19–50 y and 1.7 mg/d for men and 1.5 mg/d for women aged >51 y (5). As for thiamine and riboflavin, vitamin B-6 requirements increase slightly during pregnancy (by 0.6 mg/d) and lactation (by 0.7 mg/d) (5).

**STATUS ASSESSMENT**

Ideally, assessment of thiamine, riboflavin, and vitamin B-6 status should include various biochemical measures (both direct and indirect) along with dietary intake data (Table 1). Because some blood and urinary assessment indexes for these vitamins are influenced by recent nutrient intakes, studies examining nutrient status should consider the phase of the menstrual cycle. In premenopausal women studied over 4–6 ovulatory menstrual cycles, Martini et al (24) found that energy, protein, carbohydrate, fat, vitamin D, riboflavin, potassium, phosphorus, and magnesium intakes were all significantly higher in the midluteal phases than in the midfollicular phase.

The most widely used index for assessing thiamine is the measurement of the erythrocyte transketolase enzyme activity coefficient (ETKAC). Transketolase is a thiamine pyrophosphate–dependent enzyme. ETKAC is determined by first measuring the basal activity of the enzyme (without the added coenzyme) and then by measuring the stimulated enzyme activity (with the added coenzyme). The activity coefficient, or percentage of stimulation, is determined by dividing the stimulated enzyme activity by the basal enzyme activity. A high activity coefficient indicates poor or marginal nutritional status and reflects a decrease in thiamine availability in the erythrocyte (23). Criteria for interpreting ETKAC will depend on the laboratory and the method used. An activity coefficient of 1.00–1.15 is considered normal, a coefficient of 1.16–1.20 indicates marginal thiamine deficiency, and a coefficient >1.20 indicates severe deficiency (23). For the determination of the 1998 DRI for thiamine, an ETKAC value of 1.20–1.25 indicated marginal deficiency and a value >1.25 indicated deficiency (5). In addition to ETKAC, measurement of thiamine can be performed in the urine, whole blood, and plasma. Thiamine in the urine does not adequately reflect body stores but can be an index of dietary intake (23).

Riboflavin status is generally obtained by measuring the urinary excretion of riboflavin, determining erythrocyte riboflavin concentrations, and calculating the erythrocyte glutathione reductase activity coefficient (EGRAC). EGRAC has been found to be both useful and sensitive for determining impaired riboflavin status (23) and, as is ETKAC, it is determined by dividing the stimulated enzyme activity (with added FAD) by
For vitamin B-6, the most relevant direct measures are plasma PLP, total plasma vitamin B-6, and urinary 4-pyridoxic acid (4-PA). Indirect measures of vitamin B-6 status include the evaluation of either the erythrocyte alanine transaminase activity coefficient (EALTAC) or the erythrocyte aspartate transaminase activity coefficient (EASTAC) with stimulation by PLP (Table 1). These indexes indicate long-term vitamin B-6 status and provide a gross index of vitamin B-6 deficiency (23). As for thiamine and riboflavin, high activity coefficients (ie, > 2.00) indicate poor or marginal status. To assess vitamin B-6 status, ≥ 2 biochemical measures should be determined, and dietary intakes of vitamin B-6 and protein should be measured (4, 26).

### RATIONALE FOR INCREASED NEED

Because exercise stresses metabolic pathways that use thiamine, riboflavin, and vitamin B-6, the requirements for these vitamins may be elevated in athletes and active individuals. Theoretically, exercise increases the need for these nutrients because of a decreased absorption of nutrients; an increased turnover, metabolism, or loss of nutrients; biochemical adaptations associated with training; increased concentrations of mitochondrial enzymes that require the nutrient as a cofactor; and the need for tissue maintenance and repair (11–14, 17, 18, 27–30). In addition, there is some biochemical evidence of poor vitamin status in active persons (12, 13, 30–33) (Table 2); however, studies examining these issues have been limited and equivocal. One reason for poor nutritional status in active persons may be long-term marginal dietary intakes associated either with poor dietary choices or reduced energy intake (12, 39–41).

Differences in experimental design may also help to explain why studies of the vitamin status of active individuals have yielded inconsistent results. For example, variation has been seen in the degree of dietary control, the type and intensity of exercise, the type and number of status indexes measured, the amount of regular physical activity engaged in, the type of subjects used, and in whether a control group was included.

Exercise is known to be capable of increasing both energy and protein needs, and thus it could increase the total daily needs of thiamine, riboflavin, and vitamin B-6 in active individuals. If such persons consume adequate energy to maintain body weight and cover exercise energy expenditure, their dietary intakes of these vitamins should be adequate unless their food choices are poor. Conversely, if active persons restrict their energy intake or eliminate food groups, their intakes of these vitamins will probably be low.

### VITAMIN DEFICIENCY AND PHYSICAL ACTIVITY

Because of the roles that thiamine, riboflavin, and vitamin B-6 play in producing energy during exercise, it is generally assumed that individuals with poor status have a reduced ability to perform physical activity. Several studies have examined the effect of vitamin deficiency on work performance (13, 32, 42–44). For example, van der Beek et al (43), who depleted 24 healthy men of thiamine, riboflavin, and vitamin B-6 over an 11-wk metabolic feeding period, found that vitamin depletion significantly decreased maximal work capacity (VO, max) by 12%, onset of blood lactate accumulation by 7%, oxygen consumption at onset of blood lactate accumulation by 12%, peak power by 9%, and mean power by 7%. This study supports earlier research in which individuals with subclinical vitamin deficiencies were identified and the ability to do work was assessed before and
after vitamin supplementation (32, 44). In one of these studies, Suboticanec et al (32) measured the vitamin B-6 and riboflavin status of 124 boys aged 12–14 y and found that 24% had poor vitamin B-6 status (EASTAC > 2.00) and 19% had poor riboflavin status (EGRAC > 1.20). One subgroup (n = 37) of the original sample pool was given 2 mg vitamin B-6 for 6 d/wk for 2 mo and a second subgroup (n = 38) was given 2 mg riboflavin for 6 d/wk for 2 mo. At the beginning and end of the respective treatment periods, physical work capacity was measured on a bicycle ergometer; the researchers reported a significant negative correlation (P = 0.036) between \( \dot{V}O_2 \) max and EASTAC values. Thus, as vitamin B-6 status improved (ie, EASTAC values decreased), work capacity improved. For riboflavin, the results were similar to those observed with vitamin B-6, except that the negative correlation was not significant. In general, there was a linear relation between EGRAC and maximal work capacity until EGRAC values reached 0.90–1.00. Thus, as EGRAC improved (ie, decreased), so did work capacity. These data suggest that subclinical deficiencies of riboflavin and vitamin B-6 negatively affect aerobic capacity in young boys and that correction of the deficiency improves work capacity. In both situations, supplementation significantly improved vitamin status (P = 0.001). In summary, deficiencies of thiamine, riboflavin, and vitamin B-6 due to poor dietary intakes of these vitamins may decrease the ability to do work, especially maximal work.

**DIetary Intakes of Active Individuals**

Only limited research has been conducted on the thiamine, riboflavin, and vitamin B-6 intake of active adult individuals other than athletes. Accordingly, this discussion will primarily include studies examining dietary intakes (collecting at least 3-d diet records) of athletes published within the past 10 y but will also review the limited data on active individuals. In general, studies of active males report adequate dietary intakes of thiamine (12, 45–54), riboflavin (12, 36, 45–48, 50–54), and vitamin B-6 (33, 37, 45, 46, 49–53, 55, 56), which can be attributed to the relatively high energy intakes in these subjects. Only Guil-land et al (12) reported low mean intakes of vitamin B-6 in young (20 y) male athletes; 67% of their subjects were consum-

### Table 2

Incidence of low or marginal vitamin B-6, riboflavin, and thiamine status in studies of nonsupplemented active individuals

<table>
<thead>
<tr>
<th>Study and assessment index used</th>
<th>Type of subjects</th>
<th>Low status</th>
<th>Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vitamin B-6</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fogelholm et al (34)</td>
<td>Active subjects [42]</td>
<td>43</td>
<td>—</td>
</tr>
<tr>
<td>Guillard et al (12)</td>
<td>Male athletes [55]</td>
<td>35</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>Telford et al (35)</td>
<td>EASTAC</td>
<td>60</td>
<td>—</td>
</tr>
<tr>
<td>Rokitzki et al (36)</td>
<td>Athletes [57]</td>
<td>5</td>
<td>1.36–5.40</td>
</tr>
<tr>
<td>Weight et al (37)</td>
<td>Female athletes [30]</td>
<td>0</td>
<td>1.7 ± 0.6</td>
</tr>
<tr>
<td><strong>Riboflavin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fogelholm et al (34)</td>
<td>Active subjects [42]</td>
<td>57</td>
<td>—</td>
</tr>
<tr>
<td>Guillard et al (12)</td>
<td>Male athletes [55]</td>
<td>4</td>
<td>2.1 ± 0.2</td>
</tr>
<tr>
<td>Keith and Alt (38)</td>
<td>EGRAC &gt; 1.20 and urinary riboflavin</td>
<td>0</td>
<td>1.9 ± 0.9</td>
</tr>
<tr>
<td>Weight et al (37)</td>
<td>EGRAC &gt; 1.50, whole blood, and urinary riboflavin</td>
<td>0</td>
<td>1.4–2.5</td>
</tr>
<tr>
<td><strong>Thiamine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fogelholm et al (34)</td>
<td>Active subjects [42]</td>
<td>12</td>
<td>—</td>
</tr>
<tr>
<td>Guillard et al (12)</td>
<td>Male athletes [55]</td>
<td>17</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>Weight et al (37)</td>
<td>TPP stimulation &gt; 25%</td>
<td>0</td>
<td>1.5 ± 0.5</td>
</tr>
</tbody>
</table>

1. EASTAC, erythrocyte aspartate transaminase activity coefficient; PLP, pyridoxal 5’-phosphate; 4-PA, 4-pyridoxic acid; EGRAC, erythrocyte glutathione reductase activity coefficient; ETKAC, erythrocyte transketolase enzyme activity coefficient.
2. n values in brackets.
3. ± SD or range.
4. Includes both males and females.
5. Based on 7-d weighed food records.
ing <100% of the 1989 RDA and the group as a whole had a ratio of vitamin B-6: to protein of 0.013. No studies reported low mean thiamine or riboflavin intakes in active males, and only 2 (46, 49) reported that some of their subjects (5–18%) were consuming less than the 1989 RDA of these 2 vitamins. In addition, no studies reported low intakes of thiamine or riboflavin expressed as mg per 4184 kJ (1000 kcal)/d. It appears that the high energy intake of active males keeps the dietary intakes of all these vitamins high, usually 1.5–2 times the DRI.

As expected, dietary intakes of these vitamins are generally lower in active females than in males; yet, most studies have found that mean intakes for thiamine and riboflavin were adequate (38, 40, 46, 47, 51, 57–59). Only Kaiserauer et al (60) reported mean thiamine and riboflavin intakes to be low (in their amenorheic runners). Dietary vitamin B-6 intakes in active females are more variable; yet, all of these studies reported mean intakes >66% of the 1989 RDA (40, 46, 51, 57–61). In general, those studies reporting lower dietary intakes of vitamin B-6 also found energy intakes <7843 kJ/d (1874 kcal/d) (60, 62). All of the studies reviewed, except for that by DeBolt et al (49) and Fogelholm et al (34), used 3- to 7-d food records to determine nutrient intakes. Thus, it appears that unless an individual restricts energy intake or consumes a diet high in refined foods, nutrient intakes of thiamine, riboflavin, and vitamin B-6 are adequate.

PHYSICAL ACTIVITY AND VITAMIN REQUIREMENTS

To determine whether exercise increases the need for thiamine, riboflavin, or vitamin B-6, researchers must feed a known amount of the vitamin to both sedentary and active individuals and determine whether their nutrient statuses differ. If the vitamin status is poorer in the active individuals than in the sedentary individuals or if the active individuals require more of the vitamin than do the sedentary subjects, the active individuals are considered to need more of the vitamin. This section will evaluate the metabolic studies that have examined riboflavin and vitamin B-6 status of both sedentary and active individuals under controlled conditions; to date, there have been no metabolic studies of thiamine status in active and sedentary individuals.

On the basis of results from a series of metabolic studies in active women, exercise, dieting for weight loss, and dieting plus exercise all appear to increase the need for riboflavin above both the 1989 RDA and the 1998 DRI (14, 33, 63, 64). In a study published in 1983, Belko et al (14) fed young women various amounts of riboflavin over a 10-wk period and determined their EGRACs and urinary riboflavin excretion. During the first 2 wk, when 0.14 mg riboflavin/MJ was fed, EGRACs were above the cutoff of 1.25, indicating poor status. During the second 2-wk period, 0.24 mg riboflavin/MJ was fed and mean EGRAC values improved to within the normal range. For the next 3 wk, the riboflavin intake was held at 0.24 mg/MJ, but subjects exercised for 20–50 min 6 d/wk. The introduction of exercise increased mean EGRAC values above the cutoff. During the last 3 wk, subjects continued to exercise while the riboflavin intake was increased to 0.33 mg/MJ (0.33 mg/239 kcal). At this riboflavin intake, mean EGRAC values were normal (Figure 1). Throughout this study, urinary riboflavin excretion was significantly and negatively correlated with EGRAC (r = −0.23, P < 0.01).

Two other metabolic studies, also by Belko et al (63, 64), examined the effect of dieting and dieting plus exercise on riboflavin status. In these studies, overweight women consumed a metabolic diet providing 5000–5300 kJ (1195–1266 kcal/d) and various amounts of riboflavin (0.14–0.19 mg/MJ, or per 239 kcal). Dieting increased the amount of riboflavin required to maintain good status and even more riboflavin was required when dieting was combined with exercise. The researchers concluded that 0.38 mg riboflavin/MJ is required to keep EGRAC values in the normal range when subjects are dieting for weight loss and exercising 3–4 h/wk at 75–85% of the maximal heart rate. Winters et al (33) repeated the first (14) of Belko et al’s 3 studies in 50–67-y-old active women. The subjects were fed a metabolic diet providing adequate energy for weight maintenance (7500–8300 kJ/d, or 1793–1983 kcal/d) and 2 different amounts of riboflavin (0.14 and 0.22 mg/MJ, or per 239 kcal) for 5 wk. They found that during the weeks that subjects exercised (2.5 h/wk), EGRAC increased significantly over the period in which no exercise was performed. In addition, 0.22 mg riboflavin/MJ was required to maintain mean EGRAC values within the normal range while subjects were exercising. These data indicate that dieting alone or exercise alone may increase riboflavin requirements above the RDA and that dieting (5200 kJ/d, or 1243 kcal/d) plus exercise (2.5–5 h/wk) increases the requirement even more (0.38 mg/MJ, or 2 mg/d).

In establishing the 1998 DRI for riboflavin (5), the committee considered data from the 3 studies by Belko et al (14, 63, 64) and the 1 by Winter et al (33). The committee noted that riboflavin requirements might be higher in active individuals but indicated that adequate data were not available from which to quantify the adjustment that should be made (5). All of the subjects in the 4 referenced studies performed moderate exercise (2.5–5 h/wk) for fitness; no metabolic data are available on individuals who exercise more strenuously or participate in competitive sports. However, data are available indicating that even marginal riboflavin deficiencies can decrease work performance (42, 43).

Metabolic studies performed in active and sedentary individuals consuming known amounts of vitamin B-6 indicate that ≈1.5–2.3 mg vitamin B-6/d is required to maintain plasma PLP concentrations above the cutoff value of 30 nmol/L (26). A meta-
1 mg vitamin B-6 during a marathon (42.18 km). Runners lost 10 mg vitamin B-6/d during prolonged exercise (4–5 h/wk) and all subjects (including the sedentary women) were fed a metabolic diet providing 2.3–2.4 or 10 mg vitamin B-6/d for 7 wk. Throughout the study, the active women continued to exercise 4–5 times during the study. Plasma PLP concentrations measured during the metabolic-diet period are given in Table 3 and are compared with measurements made during consumption of a free-living diet. As shown, some of the women had poor plasma PLP concentrations while consuming their free-living diet. Mean plasma PLP concentrations improved in all 3 groups during the metabolic-diet period.

In the study by Manore et al (65), plasma PLP concentrations increased significantly in all 3 groups during exercise (Figure 2) but returned to baseline within 60 min after exercise stopped. A phenomenon that was documented in several other studies (27, 66, 67). The metabolic rationale for this increase in plasma PLP concentration during exercise is not known, but several hypotheses have been proposed (16, 17, 27, 65–67). Because plasma PLP concentrations increase within the first 5 min of exercise and stay elevated during exercise, the probability increases that PLP will be metabolized to 4-PA and lost in the urine (27). Thus, exercise can increase the turnover and loss of vitamin B-6. Indeed, researchers have documented higher 4-PA losses in active individuals compared with sedentary control subjects or during nonexercise periods (65, 68) and after a strenuous bout of exercise (27). Rokitkzi et al (69) calculated from measures of 4-PA excretion that marathon runners lost 1 mg vitamin B-6 per 42.18 km (26.2 miles). However, no research has documented a decrease in plasma PLP concentrations because of exercise-induced 4-PA losses. In general, any loss of vitamin B-6 due to exercise is small and could easily be replaced by eating 1–2 servings of a food high in vitamin B-6. However, as shown in Table 3, some individuals, especially the elderly, have poor plasma PLP concentrations while consuming self-selected, free-living diets. In addition, several studies have documented poor vitamin B-6 status in the elderly (70–73) and in individuals with such chronic diseases as arthritis (74) and diabetes (75). For example, in a 6-wk metabolic study by Manore et al (75), the vitamin B-6 status of diabetic, hypertensive, and diabetic and hypertensive white men fed 2.1–2.2 mg vitamin B-6/d was compared with that of healthy control subjects. Over the 6-wk period, plasma PLP concentrations declined gradually in the diabetic and hypertensive and diabetic subjects until all of the nonhypertensive, diabetic men and 63% of the hypertensive, diabetic men had values < 30 nmol/L (Figure 3). To date, no studies have determined whether exercise or dieting plus exercise increases the need for vitamin B-6 in these individuals.

The effect of dieting plus exercise on vitamin B-6 status has been investigated to a limited extent. Fogelholm et al (76) examined the effect of a 3-wk diet (=7000 kJ, or 1673 kcal/d) on male, elite wrestlers and found a significant increase in EASTAC (P < 0.01) but no changes in thiamine and riboflavin status. No dietary intake data for vitamin B-6 were given, however. Thus, the possibility that poor dietary intake (either alone or combined with high activity) contributed to the increased EASTAC must be considered. In another study, van Dale et al (77) examined the effect of a 14-wk diet (=3700 kJ, or 884 kcal/d) or diet plus exercise on the vitamin status of 12 obese men (mean age: 40 y). These researchers found that plasma PLP concentrations decreased significantly in the diet-plus-exercise group (from 54.5 to 40.0 nmol/L) compared with the diet-only group (from 49.8 to 48.7 nmol/L). Riboflavin and thiamine status decreased in both groups, but the only significant change was for riboflavin status in the diet-plus-exercise group. However, the dietary intake of vitamin B-6 and thiamine were below the 1989 RDA for the last 9 wk of the study, whereas riboflavin intake was at the 1989 RDA.

![FIGURE 2](https://example.com/figure2.jpg)

**FIGURE 2.** Changes in pyridoxal 5'-phosphate (PLP) over a 20-min exercise period at 80% of maximal work capacity in women. Blood samples were drawn before exercise (baseline), immediately after the exercise session ended, and 30 and 60 min after the exercise session ended. *Significantly different from baseline, P < 0.05. **Significantly decreased from after exercise to 60 min after exercise (P < 0.05). Adapted from Manore et al (65).
SUMMARY

Research examining the micronutrient needs of active individuals is still limited, with most of the work performed in competitive athletes. On the basis of the available research, riboflavin requirements appear to increase with exercise, dieting, and dieting plus exercise in both young and older women engaged in moderate activity. No data for riboflavin are available in individuals who exercise strenuously. Exercise also appears to increase the loss of vitamin B-6 through urinary 4-PA excretion. However, for both of these nutrients, the amount of additional vitamin needed to cover losses or increased needs is small and can be met easily through good food choices. The data on changes in thiamine status with exercise are also limited, but some cross-sectional studies suggest that a small percentage of active individuals may have poor status.

NUTRITIONAL STATUS OF ACTIVE INDIVIDUALS

If exercise increases the need for thiamine, riboflavin, or vitamin B-6, active individuals should theoretically have poor status if they consume the RDA for these vitamins. Much of the research that has examined whether exercise increases the need for vitamins was conducted in athletes (Table 2). Presented in the table are studies that examined the nutritional status of active individuals consuming free-living diets with no supplemental intakes of thiamine, riboflavin, or vitamin B6. The number of assessment indexes used varied, but in most studies poor status was based on more than one measurement as well as on the dietary intake of the nutrient. For vitamin B-6, the proportion of active individuals with poor status ranged from 0% to 60% in 5 studies, with the highest percentage reported by Telford et al (35). In their study, three-fifths of a group of 86 male and female athletes had poor vitamin B-6 status. Studies that examined the status of active individuals consuming a free-living diet showed fewer incidences of poor riboflavin and thiamine status. However, Fogelholm et al (37), who examined vitamin B-6, riboflavin, and thiamine status in 42 physically active college students (18–32 y) before and after 5 wk of supplementation with vitamin B complex, found poor vitamin B-6 status at baseline in 43%, poor riboflavin status in 57%, and poor thiamine status in 12% of the students. Supplementation significantly increased (P < 0.0001) the erythrocyte activity coefficients for all vitamins. These data suggest that some active individuals have poor or marginal vitamin status while consuming their free-living diets and that exercise will diminish the vitamin status of individuals who already have poor or marginal vitamin status (13).

REFERENCES

B VITAMINS AND EXERCISE


