Protein Requirements for Endurance Athletes

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Acute endurance exercise results in the oxidation of several amino acids. The total amount of amino acid oxidation during endurance exercise amounts to only 1–6% of the total energy cost of exercise. The branched chain amino acid, leucine, has been most often studied in relation to endurance exercise. Leucine is oxidized by the enzyme, branched-chain oxo-acid dehydrogenase (BCOAD). BCOAD is relatively inactive at rest (~4–7%) and is activated at the onset of exercise by dephosphorylation (to about 25%). After a period of endurance exercise training, the activation of BCOAD and amino acid oxidation are attenuated, however the total amount of BCOAD enzyme is up-regulated. A low energy and/or carbohydrate intake will increase amino acid oxidation and total protein requirements. With adequate energy and carbohydrate intake, low to moderate intensity endurance activity has little impact on dietary protein requirements and 1.0 gPRO/kg/d is sufficient. The only situation where dietary protein requirements exceed those for relatively sedentary individuals is in top sport athletes where the maximal requirement is ~1.6 gPRO/kg/d. Although most endurance athletes get enough protein to support any increased requirements, those with low energy or carbohydrate intakes may require nutritional advice to optimize dietary protein intake. *Nutrition* 2004;20:662–668. ©Elsevier Inc. 2004

KEY WORDS: branched-chain oxo-acid dehydrogenase, low carbohydrate diet, oxygen consumption

INTRODUCTION

This review examines the literature on protein metabolism during endurance exercise and provides practical suggestions regarding protein needs for recreational sport enthusiasts and top sport athletes. Endurance activities can be broadly defined as those that use predominantly oxidative phosphorylation as the main energy source. There are several important aspects that determine the effect of such activities on the physiologic stress of short- and long-term exercise and the subsequent requirements for nutrients. There are issues regarding each specific exercise bout that will determine the metabolic and nutritive requirements including intensity and duration, nutrition and hydration status before and during exercise, and the background training status of the individual. When considering the nutritional needs of a person performing exercise, it is important to take all of these factors into consideration and make activity-specific, not “blanket,” recommendations. For example, a recreational athlete who is jogging four times a week at 45% of maximum oxygen consumption (V̇O₂max) for 1 h represents a very different physiologic scenario from a top sport athlete who may be training and competing at intensities 60% to 85% of V̇O₂max for 8 to 40 h/wk. Even at these levels of energy expenditure, the recreational athlete described above would metabolize about 2000 kcal/wk, whereas the competitive athlete would require anywhere from 5600 to more than 40 000 kcal/wk of energy beyond basal needs. It is obvious that nutritional recommendations based on modest physical activity should not be extrapolated to represent the needs of top sport or elite athletes. Given the current limited status of the literature regarding protein requirements in humans performing endurance exercise, I broadly divide the recommendations into only recreational, modest, and top sport categories.

Clearly the predominant fuels used during endurance exercise are carbohydrates and fats. Although there is no doubt that skeletal muscle oxidizes about 1% to 6% of total energy from amino acids during endurance exercise, there is some controversy as to whether this alters the dietary requirement for protein. The purpose of this report is to review the pathways and determinants of protein metabolism in skeletal muscle as they relate to endurance exercise. Special emphasis on sex differences, carbohydrate and energy adequacy, and timing of nutrient delivery are considered. The ultimate goal is to consider whether or not there are circumstances in which dietary protein requirements for an endurance athlete exceed that of the suggested protein requirements for the general population.

PROTEIN METABOLISM IN SKELETAL MUSCLE

General Aspects

Proteins are important molecules that serve structural and regulatory functions in the body. Structural proteins include cytoskeletal proteins such as dystrophin and connective tissue proteins such as collagen, and regulatory proteins include enzymes such as hexokinase or carnitine palmitoyl transferase. Proteins are comprised of constituent amino acids that contain an amino (-NH₂), a carboxylic acid (-COOH), and a radical (different for each amino acid) group. Of the 20 amino acids, 9 are considered indispensable or essential (histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine). The indispensable amino acids must come from the diet and/or endogenous protein breakdown. Proteins are in a constant state of metabolic flux with simultaneous synthesis and degradation. The likely purpose of this constant flux is to allow for removal of damaged or dysfunctional proteins and to rapidly respond to altered cellular demands.

Protein synthesis is initiated after a signal (i.e., nutritional, hormonal, or mechanical) to the cell is “communicated” to the DNA to initiate gene expression (transcription). The primary RNA
transcript is processed (removal of introns, alternative splicing, 5’-cap and 3’-polyadenylation) and exported to the cytosol as mRNA for translation into proteins via ribosomes. The process of translation of the protein from mRNA involves a tRNA specific to each of the 20 amino acids, ribosomes, and tRNA synthetases (that require energy in the form of adenosine triphosphate). The translation process involves three steps: initiation, elongation and termination. After translation, the nascent protein can be further modified through processes such as glycosylation or degradation (post-translational modification). After endurance exercise, there is an increase in mRNA for a number of proteins. There are much more data regarding the effect of resistance exercise on muscle protein synthesis; however, limited data suggest that there is an increase in mixed muscle protein synthesis after endurance exercise. Ultimately, endurance exercise results in a net increase in the synthesis of a number of mitochondrial enzymes. The duration of this response and its relation to nutritional status also has not been explored in humans after endurance exercise. It is theoretically possible that endurance exercise training affects amino acid requirements through increased amounts of enzymes, capillaries, hemoglobin, and myoglobin. The amino acids for these processes may be derived from an increase in dietary protein intake and/or an increase in the efficiency of amino acid re-use.

A second fate of the intracellular amino acids is that of oxidation via the mitochondria. Human skeletal muscle can oxidize at least eight amino acids (alanine, asparagine, aspartate, glutamate, isoleucine, leucine, lysine, and valine), however, during exercise, the branched-chain amino acids (BCAAs; isoleucine, leucine, and valine) are preferentially oxidized. The BCAAs are transaminated to their keto-acids via branched-chain aminotransferase, with subsequent oxidation occurring via branched-chain oxo-acid dehydrogenase enzyme (BCOAD). The amino-N group is usually transaminated with α-ketoglutarate to form glutamate, which is then transaminated with pyruvate to form alanine or aminated via glutamine synthase to form glutamine. The BCOAD enzyme is rate limiting in BCAO oxidation, with about 5% to 8% being active (dephosphorylated) at rest and 20% to 25% being active during exercise. BCAO activation is related to a decrease in the ratio of adenosine triphosphate to adenosine diphosphate, a decrease in pH, and a depletion of muscle glycogen.

The inverse correlation between BCOAD activation and muscle glycogen concentration supports the fact that strategies to ensure carbohydrate (CHO) availability during exercise should have a sparing effect on BCOAD-mediated amino acid oxidation during endurance exercise. Amino acids may also be required for exchange reactions in the tricarboxylic acid cycle, which can also increase their net use.

In addition to dietary protein intake, protein degradation is the only other source of amino acid contribution to the intracellular free amino acid pool. The three main pathways for protein degradation in skeletal muscle are the lysosomal (cathepsin) and non-lysosomal (ubiquitin and calpain). The lysosomal pathway degrades endocytosed proteins, some cytosolic proteins, hormones, and immune modulators but does not appear to be a major factor in exercise-induced muscle breakdown. The two major non-lysosomal pathways in human skeletal muscle include the adenosine triphosphate–dependent ubiquitin pathway and the calcium-activated neutral protease (calpain) pathway. The calpain pathway does play a role in skeletal muscle proteolysis during exercise. The ubiquitin pathway is also activated during starvation, muscle atrophy, and exercise. It is not currently known whether endurance exercise training has an effect on the activation of any of the specific protein breakdown pathways.

Overall, the dietary protein requirements represent the amount of protein that is required to support net protein synthesis (growth, repair of damaged tissues, lactation, pregnancy, muscle hypertrophy, and enzyme synthesis), amino acid oxidation, and the efficiency inherent in the recycling of amino acids.

Amino Acid Oxidation During Endurance Exercise

Studies have used urea excretion as an indicator of protein oxidation (urea is a breakdown product formed in the liver after amino acid oxidation) and reported exercise-induced increases in men. This increase is missed if sweat is not collected for urea and other nitrogen compounds are contained in sweat. As a consequence, an athlete exercising in high ambient temperatures and/or humidity with a sweat rate of up to 2 L/h could have a substantial urea sweat loss. Urea excretion represents the in toto extent of amino acid oxidation but provides little information on the specifics of amino acid oxidation.

Many studies have demonstrated that endurance exercise results in increased leucine oxidation. With acute endurance exercise, there is an increase in the proportion of carbohydrate oxidation and a relative decrease in the proportion of leucine oxidized (1.5% to 5%). However, this may result in an absolute increase in amino acid oxidation because the total energy needs during intensive endurance exercise may increase 10-fold (depending on the VO2max of an athlete). If only one amino acid is oxidized during exercise, the predicted effect on protein requirements could be minimal; however, if an indispensable amino acid is oxidized (such as leucine or lysine), this could have a negative effect on protein requirements. An increase in lysine oxidation has also been observed during endurance exercise.

Leucine oxidation is greater with exercise at higher intensity, glycogen depletion, and longer exercise duration. After endurance exercise, there is a prompt return toward baseline leucine oxidation levels; although there appears to be a slight increase in leucine oxidation after eccentric exercise that may persist for up to 10 days.

Because proteins serve a structural or functional role within the cell, basic physiologic principles would predict that the repeated stress of endurance exercise should result in an adaptive down-regulation of amino acid oxidation during endurance exercise. Initial work showed that untrained men have a negative nitrogen balance at the start of an exercise program, but this balance returned to baseline with continued training. The animal data are conflicting, with some showing that training increases amino acid oxidation and another finding a decrease in leucine oxidation.

In humans, there is also a greater proportion of leucine flux diverted toward oxidation in the untrained versus trained athlete at rest, but these differences disappeared when the data were expressed relative to lean mass. My colleagues and I examined leucine oxidation and BCOAD activation during 90 min of exercise at 65% of VO2max before and after 28 d of endurance exercise training in men and women. Leucine oxidation during exercise was lower after training, as was BCOAD activation. In contrast, total BCOAD activity was higher after training, which indicated that the absolute capacity for BCAA oxidation increased. Together, these data suggested that chronic endurance training results in a sparing of protein oxidation due to a reduced activation of BCOAD, even though the total capacity for BCAA oxidation increased. Under most circumstances, these adaptations would predict that endurance exercise training would decrease the relative contribution of amino acids to total fuel oxidation; however, under periods of nutritional (i.e., low energy or CHO intake) or metabolic (i.e., ultra sports or very intensive training) stress, the daily amount of amino acid oxidation could exceed that of a sedentary person or recreational athlete.

Influence of Sex on Protein Metabolism

Many studies have examined the influence of sex on metabolic fuel selection during endurance activity. Overall, women appear to oxidize proportionately more lipid and less CHO than do men during endurance exercise. The lower contribution from CHO in exercising females implies that amino acid oxidation should also be lower as compared with men. In a previous study using 24-h urinary urea excretion as a marker of total amino acid oxidation,
oxidation, we found that men, but not women, showed increases during a day in which they completed a 15.5-km treadmill run as compared with a rest day. Using a stable isotopic tracer (L-[1-13C]-leucine), we found that women oxidize proportionately less leucine than do men during endurance cycling. The lesser leucine oxidation observed for women during endurance exercise was apparent before and after 31 d of endurance exercise training. In the latter study, we did not find that the sex difference could be explained based on the total or active proportion of skeletal muscle BCOAD. This finding suggests that the locus of the sex difference in amino acid oxidation cannot be explained at the skeletal muscle level and may be at the hepatic level. A summary of the effects of exercise on protein metabolism in men and women is found in Table I.

**Other Factors Influencing Protein Metabolism During Exercise**

It has been known for many years that CHO intake has a significant sparing effect on amino acid oxidation and protein balance. The dietary interaction between protein and CHO may have implications for those athletes who habitually consume fast foods that stress a very low CHO intake. Given that carbohydrates are the predominant fuel used during endurance exercise and that they can be depleted during prolonged endurance exercise, it is important for amino acid metabolism to be considered in light of the CHO intake and storage (i.e., glycogen) status of the athlete. We recently reported that men and women who performed endurance exercise on a daily basis were apparent before and after 31 d of endurance exercise. Using a stable isotopic tracer (L-[1-13C]-leucine), we did not find that the sex difference could be explained based on the total or active proportion of skeletal muscle BCOAD. This finding suggests that the locus of the sex difference in amino acid oxidation cannot be explained at the skeletal muscle level and may be at the hepatic level. A summary of the effects of exercise on protein metabolism in men and women is found in Table I.

**PROTEIN OXIDATION DURING ENDURANCE EXERCISE**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subjects</th>
<th>Exercise</th>
<th>Protein (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tarnopolsky</td>
<td>6 F, T</td>
<td>15.5-km run at ~65%</td>
<td>F = 0.3</td>
</tr>
<tr>
<td>et al.15</td>
<td>6 M, T</td>
<td>VO2max</td>
<td>M = 9.1</td>
</tr>
<tr>
<td>Phillips et al.</td>
<td>6 F, T</td>
<td>90-min cycle at 65%</td>
<td>F = 2.0</td>
</tr>
<tr>
<td>Tarnopolsky</td>
<td>6 M, T</td>
<td>VO2max</td>
<td>M = 3.3</td>
</tr>
<tr>
<td>et al.15</td>
<td>8 F, T</td>
<td>60-min cycle at 75%</td>
<td>F = 1.6</td>
</tr>
<tr>
<td>Tarnopolsky</td>
<td>7 M, T</td>
<td>VO2max</td>
<td>M = 6.3</td>
</tr>
<tr>
<td>et al.15</td>
<td>8 F, T</td>
<td>90-min cycle at 65%</td>
<td>F = 2.0</td>
</tr>
<tr>
<td>et al.15</td>
<td>8 M, T</td>
<td>VO2max</td>
<td>M = 3.0</td>
</tr>
<tr>
<td>McKenzie</td>
<td>6 F, T</td>
<td>VO2max</td>
<td>M = 4.8</td>
</tr>
<tr>
<td>et al.15</td>
<td>6 M, T</td>
<td>VO2max</td>
<td>M = 8.4</td>
</tr>
<tr>
<td>Lamont</td>
<td>7 F, T</td>
<td>VO2max</td>
<td>M = 2.0</td>
</tr>
<tr>
<td>et al.15</td>
<td>7 M, T</td>
<td>VO2max</td>
<td>M = 3.0</td>
</tr>
<tr>
<td>Mean</td>
<td>41, F</td>
<td>VO2max</td>
<td>M = 2.1 (1.6)</td>
</tr>
<tr>
<td>Mean</td>
<td>40, M</td>
<td>VO2max</td>
<td>M = 5.5 (3.2)</td>
</tr>
</tbody>
</table>

* Data based on urinary urea excretion.
† Data derived from L-[1-13C]-leucine oxidation.
‡ Mean (standard deviation).
F, female; M, male; T, trained; UT, untrained; VO2max, maximum oxygen capacity.

Nitrogen balance (NBAL) is a classic method used to determine the protein requirements of humans. The technique involves quantifying all the protein that enters the body (diet, intravenous, etc.) and all the nitrogen that is excreted. Because the body excretes nitrogenous compounds rather than whole proteins and proteins are approximately 16% nitrogen (w/w), NBAL involves measurement of total nitrogen intake (NIN) and total nitrogen excretion (NOUT, where NOUT = urine + feces + sweat + miscellaneous, i.e., menstrual loss, hair, semen, and skin). NBAL is positive during net anabolism and negative if a person is losing more protein than he or she is taking in. The estimated safe protein intake for a given physiological state (e.g., exercise, pregnancy, or lactation) is determined by feeding different protein intakes and calculating NBAL at each level of dietary intake. From these data, a regression analysis can be used to estimate the intake required for zero balance and a "safety factor" (often +2 standard deviations) added to account for interindividual variability. From these calculations, a "safe" protein intake level is estimated to cover 97% of the population. Another important determinant of the safe intake level is the biological value of the dietary protein. For example, a protein requirement of 1.0 g · kg⁻¹ · d⁻¹ calculated from egg white and milk protein sources would have to be higher for a diet based on lower biological value proteins such as grains. Dietary protein intake recommendations are often based on the biological value estimated to be the mean for the population.
One issue of concern with NBAL experiments is that derived protein requirement estimates may underestimate what is required for optimal function, because as protein intake decreases, there is an increase in the efficiency of amino acid re-use and a lower overall amino acid flux.\textsuperscript{4} As a consequence, NBAL may be attained with a compromise in some physiologically relevant processes such as lesser enzyme activity upregulation or capillarization and allowing for injury, infection, and other stressors over a period of several years. As outlined above, this method would have an inordinate expense, such studies are not likely to ever be completed, and the best evidence as to the impact of exercise on protein requirements will have to continue to rely on surrogate markers of protein adequacy and established techniques such as tracer turnover and NBAL.

### Studies in Endurance Athletes

There is no doubt that to make any suggestion regarding protein requirements without considering the issues of training status, daily intensity, and duration of the workout, sex, and dietary energy and CHO intake is not valid. Based on the data published to date, it is possible to provide some general guidelines for endurance athletes in three categories, namely recreational athletes (low to moderate intensity), modestly trained athletes, and top sport endurance athletes. Strong suggestions that endurance exercise has no effect whatsoever on protein requirements without considering these factors\textsuperscript{6,9} are unjustified. The fact that most athletes get enough protein in their diet (see below) is insufficient justification to discount that endurance exercise per se has an influence on protein requirements.

Many studies and reviews have correctly demonstrated and concluded that low- to moderate-intensity endurance exercise does not negatively affect protein or amino acid balance and that, if energy intake is adequate, there is an increase in use of amino acids.\textsuperscript{9,63,76–78} A 24-h leucine tracer study found that NBAL was achieved with a protein intake of 1.0 g · kg\textsuperscript{−1} · d\textsuperscript{−1} in young men performing low to moderate physical activity (2 bouts × 90 min at 50% of VO\textsubscript{2peak}/d).\textsuperscript{13} The provision of extra protein beyond requirement (nutrient excess) resulted in increased leucine oxidation in men performing moderate endurance exercise.\textsuperscript{14,17} In my estimation, the most comprehensive study in men at the low to moderate level of endurance exercise intensity was completed by Forsslund et al.\textsuperscript{14} This study examined leucine oxidation, protein, CHO, fat, and energy balance over a 24-h period in men performing low- to moderate-intensity exercise (90 min at 45% to 50% of VO\textsubscript{2peak}) while consuming a higher (2.5 g · kg\textsuperscript{−1} · d\textsuperscript{−1}) and lower (1.0 g · kg\textsuperscript{−1} · d\textsuperscript{−1}) protein intake.\textsuperscript{14} They found that protein balance was slightly negative on the 1.0 g · kg\textsuperscript{−1} · d\textsuperscript{−1} diet and positive on the 2.5 g · kg\textsuperscript{−1} · d\textsuperscript{−1} diet and that fat oxidation and CHO storage were greater on the higher protein diet.\textsuperscript{14}

There have been three studies that examined protein requirements in moderately to well-trained endurance athletes using NBAL.\textsuperscript{5,12,78} One study measured NBAL in young (27 y, VO\textsubscript{2peak} = 65 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}) and middle-age (52 y, VO\textsubscript{2peak} = 55 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}) male athletes who consumed three protein intakes (0.61, 0.92, and 1.21 g · kg\textsuperscript{−1} · d\textsuperscript{−1}) and found no appreciable age effect with an overall mean protein intake for a zero NBAL of 0.94 g · kg\textsuperscript{−1} · d\textsuperscript{−1} and a safe intake of 1.26 g · kg\textsuperscript{−1} · d\textsuperscript{−1}.\textsuperscript{78} Phillips et al.\textsuperscript{80,81} examined NBAL in endurance-trained men (VO\textsubscript{2peak} = 59 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}) and women (VO\textsubscript{2peak} = 55 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}) who were adapted to the then Canadian recommended daily protein intake of approximately 0.86 g · kg\textsuperscript{−1} · d\textsuperscript{−1} for a 10-d period and found that subjects were in negative NBAL.\textsuperscript{5} This latter study included direct stool and sweat measurements in addition to the usual urinary measurements, and subjects were in energy balance.\textsuperscript{5} A final study found that moderately trained male and female endurance athletes consuming protein at 1.0 g · kg\textsuperscript{−1} · d\textsuperscript{−1} were in slightly negative NBAL (women, ~0.22 g/d; men, ~3.95 g/d).\textsuperscript{12} Together, these studies showed that the protein intakes at or just below 1.0 g · kg\textsuperscript{−1} · d\textsuperscript{−1} were not adequate to meet the needs of men and women at this level of exercise volume and intensity. Both studies that included women and men found a more negative NBAL in the men.\textsuperscript{5,12} The sex difference is consistent with tracer studies in men and women athletes.\textsuperscript{2,5,11,12}

There have been three studies that investigated the protein requirements for top sport endurance athletes using NBAL.\textsuperscript{80,82} Only one measured all routes of nitrogen excretion (including feces) and included a sedentary control group.\textsuperscript{6} Our group completed an NBAL experiment in six top sport male endurance athletes (mean VO\textsubscript{2peak} = 76.2 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}, ~12 h/wk training volume) and calculated a safe protein intake of 1.6 g · kg\textsuperscript{−1} · d\textsuperscript{−1}.\textsuperscript{5} In the latter study, the protein intake estimate for a group of six sedentary men was 0.86 g · kg\textsuperscript{−1} · d\textsuperscript{−1}, which was identical to Canadian daily recommended intake at that time.\textsuperscript{6} Friedman and Lemon\textsuperscript{82} measured NBAL in five well-trained endurance runners and determined a safe protein intake to be 1.49 g · kg\textsuperscript{−1} · d\textsuperscript{−1}. In a Tour de France cycling simulation with well-trained cyclists (VO\textsubscript{2peak} = 65.1 mL · kg\textsuperscript{−1} · min\textsuperscript{−1}), Bruins et al.\textsuperscript{80,81} estimated protein requirements of 1.5 to 1.8 g/kg/min to maintain NBAL.

My colleague, Stuart Phillips, used the retrospective data from the four studies with moderate to top sport athletes where NBAL information was obtainable and performed regression analysis using 46 data points.\textsuperscript{5,6,7,9,82} With the inclusion of a safety margin to account for interindividual differences, his estimated protein intake was 1.11 g · kg\textsuperscript{−1} · d\textsuperscript{−1} (Phillips S, personal communication, 2003).

In summary, these data suggest that low- to moderate-intensity recreational endurance exercise does not require dietary protein requirements in excess of current general population recommendations. Moderate-intensity endurance athletes require only marginal (~1.1 g · kg\textsuperscript{−1} · d\textsuperscript{−1}, ~25% increase) increases in dietary protein over that recommended for the general population and the maximal protein requirement attainable by only a minority of all endurance athletes (top sport or elite) is not likely to exceed...
1.6 g · kg⁻¹ · d⁻¹. Although more work is required, it appears that the dietary protein requirements for female athletes are approximately 15% to 20% lower than for males. Further research should be conducted to follow-up on the apparent CHO-sparing and increased fat oxidation seen in the study comparing a modest (1.0 g · kg⁻¹ · d⁻¹) with a high (2.5 g · kg⁻¹ · d⁻¹) protein diet.²

Habitual Protein Intakes in Athletes (Doesn’t Everyone Get Enough in the Diet?)

In the aforementioned studies, it is apparent that most participants were consuming enough protein to meet even these modestly elevated requirements (Table II). This latter observation does not justify discounting an effect of physical activity on protein requirements.³⁴ Although the mean protein and energy intakes in most studies are adequate even to meet a modest increase in protein requirements, the range of intakes indicates that there are some individuals who habitually do not consume adequate levels.²⁻⁵,⁶,⁷,⁸ For example, in some of our studies, we reported adequate mean protein and energy intakes in male and female endurance athletes, but approximately 10% of men and approximately 20% of women had intakes below sedentary recommendations.²⁻⁵ The reining of protein requirements for athletes is important not for the majority of athletes who already consume ample amounts of protein; rather, it is important for those athletes with special nutritional needs (i.e., low energy intake or increased training demands such as training camp).

CONCLUSIONS AND PRACTICAL SUGGESTIONS

It appears that low- and moderate-intensity endurance exercise does not affect dietary protein requirements. At the initiation of an endurance exercise program or during a ramp increase in training demands (such as training camp), there is a transient increase in dietary protein needs; however, the body rapidly adapts to the increase in need through an increase in efficiency. For the well-trained endurance athlete training 4 to 5 d/wk for longer than 60 min, there appears to be a very modest increase in dietary protein requirements of only 20% to 25%. For the top sport elite endurance athlete, the increase in dietary protein intake may be up to 1.6 g · kg⁻¹ · d⁻¹. Despite these elevated requirements in top sport athletes, there is no need for supplementation with a mixed diet providing adequate energy and 10% to 15% coming from dietary protein. For example, an energy intake of about 3,500 kcal/d would amount to about 125 g/d or protein, or approximately 1.7 g · kg⁻¹ · d⁻¹.

The guidelines that I have suggested are modest and in line with a recent recommendation from the American College of Sports Medicine, the American Dietetic Association, and the Dietitians of Canada regarding nutrition and sports performance: “Data are not presently available . . . to suggest that athletes need a diet substantially different from that recommended in the Dietary Guidelines for Americans or the Nutrition Recommendations for Canadians ( . . .12% to 15% of energy from protein . . .). . . When energy intake is 4,000 to 5,000 kcal per day . . . if protein intake in such a diet was even as low as 10% of energy intake, absolute protein intake (100 to 125 g per day) would exceed the recommendations for protein intake for athletes (1.2 to 1.7 g per day or 84 to 119 g in a 70 kg athlete) . . .”⁹ Dietary protein intake is not a concern for athletes who are consuming 10% to 15% protein and adequate energy to meet the needs of their activity; however, a sport nutritionist or dietitian is often called upon to deal with the special cases in which one or more of these guidelines is not met and knowledge of a requirement is helpful.

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