

Protein Requirements for Endurance Athletes

Mark Tarnopolsky

1. Introduction

This review will examine the literature regarding protein metabolism during endurance exercise and will attempt to provide some practical suggestions regarding protein needs for recreational sport enthusiasts as well as top sport athletes. Endurance activities can be broadly defined as those that utilize predominantly oxidative phosphorylation as the main energy source. There are a number of important aspects that will determine the impact of such activities on the physiological stress of acute and chronic 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, pre- and during-exercise nutrition and hydration status, 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 and not “blanket” recommendations. For example, a recreational athlete who is jogging 4 times a week at 45% of VO_{2max} for 1 hour represents a very different physiological scenario as compared to a top sport athlete who may be training and competing at intensities between 60–85% of VO_{2max} for a total of 8 to 40 hours per week. Even at the level of energy expenditure, the recreational athlete described above would metabolize about 2000 kcal/wk, whereas the competitive athlete would require anywhere from 5,600 to over 40,000 kcal of energy beyond basal needs per week (10). It is obvious that nutritional recommendations based upon 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 shall broadly divide the recommendations into only recreational, modest and top sport categories.

Clearly the predominant fuels during endurance exercise are carbohydrates and fats (2–4). Although there is no doubt that skeletal muscle oxidizes about 1–6% of total energy from amino acids during endurance exercise (49, 58, 71, 72), there is still some controversy as to whether this alters the dietary requirement for protein (5–7, 24, 29, 41, 42, 44, 49, 52, 58, 64, 66, 74). The purpose of this paper is to review the pathways and determinants of protein metabolism in skeletal muscle as they relate to endurance exercise. Special emphasis upon sex differences, carbohydrate and energy adequacy, and timing of nutrient delivery will be considered. The ultimate goal will be to consider whether dietary protein requirements for an endurance athlete ever exceed the suggested protein requirements for the general population.

The author is with the Department of Pediatrics and Medicine at McMaster University, Hamilton, Ontario, Canada L8N 3Z5.

2. Protein Metabolism in Skeletal Muscle

a. General Aspects

Proteins are important molecules that serve both structural and regulatory functions in the body. Structural proteins include cytoskeletal proteins such as dystrophin, connective tissue proteins such as collagen, and regulatory proteins, including enzymes such as hexokinase or carnitine palmitoyl transferase. Proteins are comprised of constituent amino acids (AA) that contain an amino ($-\text{NH}_2$), a carboxylic acid ($-\text{COOH}$), and a radical (different for each of the amino acids) group. Of the 20 amino acids, 9 are considered indispensable/essential (histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, valine; 70). 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 purpose of this constant flux is likely to allow for removal of damaged/dysfunctional proteins and to rapidly respond to altered cellular demands.

Protein synthesis is initiated following a signal (i.e., nutrient, hormone, mechanical) to the cell that 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'-poly-adenylation) 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 ATP). The translation process involves three steps called initiation, elongation, and termination. Following translation, the nascent protein can be further modified through processes such as glycosylation or degradation (post-translational modification). Following endurance exercise, there is an increase in mRNA for a number of proteins (46, 47, 55, 56, 62, 68). There is much more data regarding the effect of resistance exercise on muscle protein synthesis (27, 59, 60, 61, 76); however, the limited data suggest an increase in mixed muscle protein synthesis following endurance exercise (14, 16). Ultimately, endurance exercise results in a net increase in synthesis of a number of mitochondrial enzymes (17, 49). The duration of this response and the relationship to nutritional status has also not been explored in humans following endurance exercise. It is theoretically possible that endurance exercise training could impact upon 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 reutilization.

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; 70); however, during exercise, the branched-chain amino acids (BCAA: isoleucine, leucine, and valine) are preferentially oxidized (43, 49, 58, 70). The BCAA are transaminated to their keto-acids via branched-chain aminotransferase (BCAAT), with subsequent oxidation occurring via branched-chain oxo-acid dehydrogenase enzyme (BCOAD; 8, 9). 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 (15, 57). The BCOAD

enzyme is rate limiting in BCAA oxidation, with about 5 to 8% being active (dephosphorylated) at rest, and 20 to 25% being active during exercise (8, 49). BCOAD activation is related to a decrease in the ATP/ADP ratio, a decrease in pH, and a depletion of muscle glycogen (39, 40, 79, 80). The inverse correlation between BCOAD activation and muscle glycogen concentration (79, 80) supports the fact that strategies to ensure CHO availability during exercise should have a sparing effect upon 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 utilization (32, 33).

In addition to dietary protein intake, protein degradation is the only other source of amino acid contribution to the intra-cellular free amino acid pool. The three main pathways for protein degradation in skeletal muscle are the lysosomal (cathepsin) and non-lysosomal (ubiquitin and calpain) pathways. 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 (45), unless there is significant inflammatory cell infiltration following rather severe damage (75). The two major non-lysosomal pathways in human skeletal muscle include the ATP-dependent ubiquitin pathway (45), and the calcium-activated neutral protease (calpain) pathway (2, 3, 26, 34). The calpain pathway does play a role in skeletal muscle proteolysis during exercise (2, 3). The ubiquitin pathway is also activated during starvation, muscle atrophy, and exercise (45). 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 daily protein requirements represent the amount of protein that is required to support net protein synthesis (growth, repair of damaged tissues, lactation, pregnancy, muscle hypertrophy, enzyme synthesis), amino acid oxidation, and the inefficiency inherent in the amino acid recycling process.

b. 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 following amino acid oxidation) and reported exercise induced increases in men (58, 74). This increase is missed if sweat is not collected for urea and other nitrogen compounds are contained in sweat (24, 74). Consequently, 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 resulted in increased leucine oxidation (7, 24, 42–44, 49, 58). With acute endurance exercise, there is an increase in the proportion of carbohydrate oxidation and a *relative* decrease in the proportion of leucine oxidized (1.5–5%; 7, 24, 42–44, 49, 58). However, this may result in an *absolute* increase in amino acid oxidation, for the total energy needs during intensive endurance exercise may increase 10 fold (depending on an athlete's VO_{2max}). If only one of the amino acids 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 (42). Leucine oxidation is greater with exercise at higher

intensity, glycogen depletion, and longer exercise duration (49, 58, 79). Following endurance exercise, there is a prompt return towards baseline leucine oxidation levels (58), although there appears to be a slight increase in leucine oxidation following eccentric exercise that may persist for up to 10 days (28).

Since proteins serve either a structural or functional role within the cell, basic physiological 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 had a negative nitrogen balance at the start of an exercise program, yet this returned to baseline with continued training (35). The animal data are conflicting, with some studies showing that training increased amino acid oxidation (22, 36), yet another finding a reduction in leucine oxidation (37). In humans, there is also a greater proportion of leucine flux diverted towards oxidation in the untrained versus trained athlete at rest (44), yet these differences disappear when the data are expressed relative to lean mass (43). Our group examined both leucine oxidation and BCOAD activation during 90 min of exercise at 65% of $\text{VO}_{2\text{max}}$, before and after 28 days of endurance exercise training in men and women (49). Leucine oxidation during exercise was lower after training, as was BCOAD activation (49). In contrast, we found that total BCOAD activity was higher after training, which indicated that the absolute *capacity* for BCAA oxidation increased (49). Together, the above data suggest that chronic endurance training results in a sparing of protein oxidation due to a reduced activation of BCOAD, yet 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, very intensive training) stress, the daily amount of amino acid oxidation could exceed that of a sedentary person or recreational athlete.

c. Influence of Gender on Protein Metabolism

A number of studies have examined the influence of gender on metabolic fuel selection during endurance activity (30, 38, 42, 49, 50, 58, 73). Overall, women appear to oxidize proportionately more lipid and less carbohydrate as compared to men during endurance exercise (30, 38, 42, 49, 50, 58, 73). The lower contribution from carbohydrate in exercising females would imply that amino acid oxidation should also be lower as compared to men. In an earlier study using 24-h urinary urea excretion as a marker of total amino acid 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 to a rest day (71). Using a stable isotopic tracer (L-[1- ^{13}C]-leucine), our group (58) and others (41, 42, 58) found that women oxidized proportionately less leucine as compared to men during endurance cycling. The lesser leucine oxidation observed for women during endurance exercise was apparent prior to and following 31 days of endurance exercise training (2). In the latter study, we did not find that the gender difference could be explained based upon either the total or active proportion of skeletal muscle BCOAD (2). This finding suggests that the locus of the gender 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 1.

Table 1 Protein Oxidation During Endurance Exercise

| Reference | Subjects | Exercise | Protein (%) |
|---------------------------|----------------------|--|------------------|
| Tarnopolsky et al., 1990* | 6 F, T; 6 M, T | 15.5-km run @ ~65% VO _{2max} | F = 0.3, M = 9.1 |
| Phillips et al., 1993†,* | 6 F, T; 6 M, T | 90-min cycle @ 65% VO _{2max} | F = 2.0, M = 3.3 |
| Tarnopolsky et al., 1995* | 8 F, T; 7 M, T | 60-min cycle @ 75% VO _{2max} | F = 1.6, M = 6.3 |
| Tarnopolsky et al., 1997* | 8 F, T; 8 M, T | 90-min cycle @ 65% VO _{2max} | F = 2.0, M = 3.0 |
| McKenzie et al., 2000† | 6 F, UT→T; 6 M, UT→T | 90-min cycle @ 65% VO _{2max} | F = 4.8, M = 8.4 |
| Lamont et al., 2001† | 7 F, T; 7 M, T | 90-min cycle @ 65% VO _{2max} | F = 2.0, M = 3.0 |
| Mean values, females | 41, F | 85-min cycle @ 67% VO _{2max} | F = 2.1 (1.6) |
| Mean values, males | 40, M | | M = 5.5 (3.2) |

Note. *Data based upon urinary urea excretion; †data derived from L-[1³C]-leucine oxidation. F=female, M = male, UT = untrained, T = trained. Values are Mean (SD).

d. Other Factors Influencing Protein Metabolism During Exercise

It has been known for many years that carbohydrate (CHO) intake has a significant sparing effect upon amino acid oxidation and protein balance (19, 25). The dietary interaction between protein and CHO may have implications for those athletes who habitually consume fad diets that stress a very low CHO intake. Given that carbohydrates are the predominant fuel utilized during endurance exercise (49, 58) and that they can become depleted during prolonged endurance exercise (49), it is important for amino acid metabolism to be considered in light of the carbohydrate intake and storage (i.e., glycogen) status of the athlete. We have recently reported that both men and women show attenuated total amino acid oxidation (serial urinary urea excretion) during endurance exercise when carbohydrate supplements are consumed during exercise (65). This latter study (65) emphasized the fact that carbohydrate consumption during exercise is an effective strategy to attenuate potential exercise-induced increases in amino acid oxidation. We have also found that urea excretion in urine was lower during a period of intensive endurance exercise training when women consumed a post-exercise supplement containing carbohydrate with a small amount of protein, as compared to consuming the same supplement at a time unrelated to the exercise (67). Glucose consumption during endurance exercise appears to reduce leucine oxidation (~20%) only when dietary protein intakes are rather high (1.8 g/kg/d) but not when they are low (0.7 g/kg/d; 6). It appears that a high protein intake leads to an expected increase in amino acid oxidation at rest and during endurance exercise (6, 7).

Energy is a second “classical” determinant of protein metabolism, with sub-optimal energy intake leading to a relative increase in protein oxidation (13, 19). An increase in total energy intake was also associated with an improvement in protein balance in young women who were performing endurance exercise on a daily basis (67). As I will articulate below, most men and women consume enough energy and protein to accommodate any possible increase in protein requirements; however, the goal of the sport nutritionist is to identify and work with athletes who have unique and special needs. Over the years, our group and others have expressed concern that a varying number of female athletes appear to report very low energy intakes (20, 21, 23, 48, 54, 58, 69, 71, 72). Unfortunately, energy restriction is on a continuum from “dieting” to severe cases of anorexia, and these disordered eating patterns are not uncommon among various types of female athletes (53). These athletes require the most attention with regards to energy and protein balance. Fortunately, strategies such as changing the timing of nutritional delivery can have beneficial effects on protein balance and performance, without altering total energy intake (67).

There is no question that dehydration can significantly alter exercise performance and ultimately lead to more severe medical disorders such as heat stress and heat stroke (1, 18). Hydration is also a determinant of amino acid oxidation, with cellular dehydration inducing an increase in leucine oxidation and cellular hyperhydration showing the opposite in resting young men (4). Although dehydration during exercise should undoubtedly increase amino acid oxidation, no study has yet been completed during endurance exercise in men or women to explore this concept. Although most athletes strive to attain optimal hydration, there are a number of factors that can limit oral intake of fluids during exercise, and every athlete in the world has undoubtedly experienced some degree of dehydration during training

and/or competition. The varying and episodic dehydration that all athletes experience will have some impact on protein requirements.

3. Protein Requirements During Endurance Exercise

a. Methods of Determining Protein Requirements

Nitrogen balance (NBAL) is a classical method used to determine the protein requirements of humans. The technique involves quantifying all of the protein that enters the body (diet, intravenous, etc.) and all of the nitrogen that is excreted (63, 81). Because the body excretes nitrogenous compounds rather than whole proteins and that proteins are ~16% nitrogen (w/w), NBAL involves measurement of the total nitrogen intake (N_{IN}), and the total nitrogen excretion [N_{OUT} = 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 they are taking in. The estimated safe protein intake for a given physiological state (e.g., exercise, pregnancy, and lactation) is determined by feeding varying 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 *SD*) added in order to account for inter-individual variability. From these calculations, a “safe” protein intake level is estimated to cover 97% of the population (63, 81). 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/d 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 upon the biological value estimated to be the mean for the population (63, 81).

One issue of concern with NBAL experiments is that derived protein requirement estimates may underestimate what is required for optimal function for, as protein intake decreases, there is an increase in the efficiency of amino acid reutilization and a lower overall amino acid flux (74). Consequently, NBAL may be attained with a compromise in some physiologically relevant processes such as lesser enzyme activity up-regulation or capillarization after endurance exercise training.

Since NBAL methods do not give specific details on the dynamic processes of protein turnover, an interest in the use of amino acid tracer methodology has been employed for decades (57, 58, 64, 81). A conceptual framework has been proposed by Young and Bier to use amino acid turnover to determine amino acid and protein requirements (81). They proposed the terms: nutrient deficiency, accommodation, adaptation, and nutrient excess. A state of protein deficiency could be reflected by a reduction in amino acid oxidation and protein synthesis to all but the essential organs (e.g., brain), ultimately resulting in muscle wasting. A state of accommodation could be defined as a state where NBAL is achieved through a reduction in a physiologically relevant process such as attenuated aerobic enzyme induction. A state of adaptation would reflect the dietary intake that would provide for optimal enzyme or capillary up-regulation during endurance training while supporting immune surveillance and a safety margin for times of stress. A protein excess would be defined as that intake where amino acids show an exponential increase in oxidation for energy and/or where protein synthesis is not further stimulated by a further

increase in intake. Stable isotope tracers can also be used to look at 24-h amino acid balance and determine the influence of exercise (24, 29).

In an ideal world, the ultimate method for determining dietary protein requirements for endurance athletes would be to provide a large group of sedentary individuals with a variety of randomly assigned protein intakes over a prolonged period of endurance training and determine which was the optimal intake to achieve maximal improvements in physiological adaptation (i.e., $\text{VO}_{2\text{max}}$), while also allowing for injury, infection, and other stressors over a several-year period. As outlined above, this method would have an infinite number of variables that would influence the requirements including: genetic capacity, exercise intensity and duration, state of training, energy balance, CHO proportion, gender, and type of protein. Given the inordinate amount of 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 upon surrogate markers of protein adequacy and established techniques such as tracer turnover and NBAL.

b. 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 work out, gender, and dietary energy and CHO intake is not valid. Based upon the data published to date, it is possible to provide some general guidelines for endurance athletes in three categories, namely, recreational athletes (low-modest intensity), modestly trained athletes, and top sport endurance athletes. Strong suggestions that endurance exercise has no impact whatsoever upon protein requirements without considering these factors (52, 64) 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.

A number of papers and reviews have correctly demonstrated and concluded that low-modest intensity endurance exercise does not negatively impact upon protein or amino acid balance and that, if energy intake is adequate, there is an increase in utilization of amino acids (13, 52, 77, 78, 82). A 24-h leucine tracer study found that NBAL was achieved with a protein intake of 1.0 g/kg/d in young men performing low-modest physical activity (2 bouts \times 90 min @ 50% of $\text{VO}_{2\text{peak}}$ /d; 24). The provision of extra protein beyond requirement (nutrient excess) resulted in increased leucine oxidation in men performing modest endurance exercise (7, 29). In my estimation, the most comprehensive study in men at the low-moderate level of endurance exercise intensity was completed by Forslund, Hambraeus, and Young (29). This study looked at leucine oxidation, protein, carbohydrate fat, and energy balance over a 24-h period in men performing low-moderate intensity exercise (90 min @ 45–50% $\text{VO}_{2\text{peak}}$) while consuming a higher (2.5 g PRO/kg/d) and lower (1.0 g PRO/kg/d) protein intake (29). They found that protein balance was slightly negative on the 1.0 g/kg/d diet and positive on the 2.5 g/kg/d diet, and that fat oxidation and CHO storage were higher on the higher protein diet (29).

There have been three studies that have examined protein requirements in moderate to well trained endurance athletes using NBAL (44, 51, 58). One study measured NBAL in young (27 y, $\text{VO}_{2\text{peak}} = 65$ ml/kg/min) and middle aged (52 y, $\text{VO}_{2\text{peak}} = 55$ ml/kg/min) male athletes while consuming three protein intakes (0.61, 0.92, and 1.21 g PRO/kg/d), and found no appreciable age effect, with an overall

mean protein intake for zero nitrogen balance of 0.94 g PRO/kg/d and a safe intake of 1.26 g PRO/kg/d (51). Phillips and colleagues examined NBAL in endurance trained men ($\text{VO}_{2\text{peak}} = 59 \text{ ml/kg/min}$) and women ($\text{VO}_{2\text{peak}} = 55 \text{ ml/kg/min}$) who were adapted to the then Canadian Recommended Daily Intake of $\sim 0.86 \text{ g PRO/kg/d}$ for a 10-day period and found that they were in negative NBAL (58). This latter study included direct stool and sweat measurements in addition to the usual urinary measurements, and subjects were in energy balance (58). A final study found that moderately trained male and female endurance athletes consuming 1.0 g PRO/kg/d were in slightly negative NBAL (women = -0.22 ; men = -3.95 g/d ; 44). Together, these studies showed that the PRO intakes at or just below 1.0 g/kg/d were not adequate to meet the needs of the majority of men and women at this level of exercise volume and intensity. Both of the studies that included women as well as men found a more negative NBAL in the men (44, 58). The gender difference is consistent with tracer studies in men and women athletes (42, 44, 49, 58).

There have been three studies that have looked at the PRO requirements for top sport endurance athletes using NBAL (11, 12, 31, 74). Only one of these has measured all routes of N excretion (including feces) and has included a sedentary control group (74). Our group completed an NBAL experiment in 6 top sport male endurance athletes (mean $\text{VO}_{2\text{peak}} = 76.2 \text{ ml/kg/min}$, $\sim 12 \text{ h/wk}$ training volume) and calculated a safe protein intake of 1.6 g PRO/kg/d (74). In the latter study, the PRO intake estimate for a group of 6 sedentary men was 0.86 g PRO/kg/d , which was identical to Canadian Daily Recommended Intake at that time (74). Friedman and Lemon (31), measured NBAL in 5 well-trained endurance runners and determined a safe intake to be 1.49 g PRO/kg/d . In a simulated Tour de France cycling simulation with well trained cyclists ($\text{VO}_{2\text{peak}} = 65.1 \text{ ml/kg/min}$), Brouns and colleagues (11, 12) estimated protein requirements of 1.5 to 1.8 g PRO/kg/min to maintain NBAL.

My colleague, Stuart Phillips, recently used the retrospective data from the four studies with moderate to top sport athletes where NBAL data was obtainable and performed a regression analysis using a total of 46 data points (31, 51, 58, 74). With the inclusion of a safety margin to account for inter-individual differences, his estimated intake was 1.11 g PRO/kg/d (S. Phillips, Personal Communication, 2003).

In summary, the above data suggest that low to modest intensity recreational endurance exercise does not require dietary PRO requirements in excess of current general population recommendations. Modest intensity endurance athletes require only marginal ($\sim 1.1 \text{ g PRO/kg/d}$, $\sim 25\%$ 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/d . Although more work is required, it appears that the dietary protein requirements for female athletes are $\sim 15\text{--}20\%$ 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 PRO/kg/d) to a high (2.5 g PRO/kg/d) protein diet (29).

c. Habitual Protein Intakes in Athletes (Doesn't Everyone Get Enough in the Diet?)

In the aforementioned studies, it is apparent that the majority of the participants were consuming enough PRO to meet even these modestly elevated requirements (Table 2). This latter observation does not justify discounting an effect of physical

Table 2 Habitual Protein Intakes in Male and Female Endurance Athletes

| Reference | Subjects | Protein (g/kg/d) | %E _{IN} |
|--------------------------|----------------------|---------------------|------------------|
| Tarnopolsky et al., 1997 | <i>n</i> = 8 male | 1.9 | 17 |
| | <i>n</i> = 8 female | 1.2 | 14 |
| Tarnopolsky et al., 1995 | <i>n</i> = 7 male | 1.8 | 15 |
| | <i>n</i> = 8 female | 1.0 | 12 |
| Tarnopolsky et al., 1988 | <i>N</i> = 6 male | 1.5 | 11 |
| Phillips et al., 1993 | <i>n</i> = 6 male | 1.9 | 15 |
| | <i>n</i> = 6 female | 1.0 | 13 |
| Schultz et al., 1992 | <i>N</i> = 9 female | 1.4 | 13 |
| Tarnopolsky et al., 1990 | <i>n</i> = 6 male | 1.2 | 12 |
| | <i>n</i> = 6 female | 1.7 | 13 |
| Saris et al., 1989 | <i>N</i> = 5 male | 2.2 | 15 |
| Deuster et al., 1986 | <i>N</i> = 51 female | 1.6 | 13 |
| Hellsworth et al., 1985 | <i>N</i> = 13 male | 2.1 | 14 |
| Nelson et al., 1986 | <i>n</i> = 17 EUM* | 1.0 | 15 |
| | <i>n</i> = 11 AMEN* | 0.7 | 15 |
| Marcus et al., 1985 | <i>n</i> = 6 EUM* | 1.3 | 17 |
| | <i>n</i> = 11 AMEN* | 1.0 | 15 |
| Drinkwater et al., 1984 | <i>n</i> = 13 EUM* | 1.1 | 13 |
| | <i>n</i> = 14 AMEN* | 1.2 | 16 |
| Approximate mean | Male | 1.8 (0.4) | 14 (2) |
| | Female | 1.2 (0.3) | 14 (2) |

Note. EUM = eumenorrheic, AMEN = amenorrheic females, % E_{IN} = % energy intake. Values are mean (SD).

activity upon PRO requirements (52, 64). Although the mean protein and energy intake in most studies is adequate even to meet a modest increase in PRO requirements, the range of intakes indicates that there are some individuals who were not habitually consuming adequate levels (20, 21, 49, 58, 71, 72). For example, in some of our studies we have reported adequate mean PRO and energy intakes in male and female endurance athletes, yet ~10% of men and ~20% of women had intakes below sedentary recommendations (49, 58, 71, 72). The refining of PRO requirements for athletes is important, not for the majority of athletes who already consume ample amounts of protein, but for those with special nutritional needs (i.e., low energy intake, increased training demands, training camp).

4. Conclusions

It appears that low- and moderate-intensity endurance exercise does not impact upon dietary protein requirements. At the initiation of an endurance exercise program,

or during a ramp increase in training demands (such as a 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 days per week for > 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 PRO/kg/d. In spite of these elevated requirements in top sport athletes, there is no need for supplementation, with a mixed diet providing adequate energy, and 10–15% coming from dietary protein. For example, an energy intake of about 3,500 kcal/d would amount to about 125 g PRO/d or ~1.7 g/kg/d.

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 . . .) (1)

As mentioned, dietary protein intake is not a concern for athletes who are consuming 10–15% PRO and adequate energy to meet the needs of their activity; however, a sport nutritionist/dietician is often called upon to deal with the special cases where one or more of these guidelines are not met and knowledge of a requirement is helpful.

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