Do Athletes Need More Dietary Protein and Amino Acids?

Peter W.R. Lemon

The current recommended daily allowance (RDA) for protein is based primarily on data derived from subjects whose lifestyles were essentially sedentary. More recent well-designed studies that have employed either the classic nitrogen balance approach or the more technically difficult metabolic tracer technique indicate that overall protein needs (as well as needs for some specific individual amino acids) are probably increased for those who exercise regularly. Although the roles of the additionally required dietary protein and amino acids are likely to be quite different for those who engage in endurance exercise (protein required as an auxiliary fuel source) as opposed to strength exercise (amino acids required as building blocks for muscle development), it appears that both groups likely will benefit from diets containing more protein than the current RDA of 0.8 g · kg⁻¹ · day⁻¹. Strength athletes probably need about 1.4–1.8 g · kg⁻¹ · day⁻¹ and endurance athletes about 1.2–1.4 g · kg⁻¹ · day⁻¹.

Whether large quantities of dietary protein are necessary for optimal athletic performance has been debated scientifically since at least the 1840s, when von Liebig proposed that protein was the major fuel used during physical exercise (79). Apparently, this view was widely accepted at the time by scientists and athletes alike. Although many athletes still believe that very large quantities of protein are essential for elite exercise performance (31), the opinion of most scientists changed dramatically during the late 1800s and early 1900s, when experimental data began to accumulate indicating that carbohydrate and fat, not protein, are the major fuels used during exercise. So complete was this turnabout in scientific understanding that for most of the 20th century investigators have concentrated on the effects of exercise on carbohydrate and fat metabolism and have largely ignored protein.

For example, in the second edition of their classic exercise physiology text, published in 1977, Åstrand and Rodahl, citing studies published in 1886, 1896, 1904, 1920, 1926, and 1957, concluded that “protein is not used as a fuel to any appreciable extent as long as energy supply is adequate” (1, p. 487). Further, based on studies reported no later than 1977, the section on protein requirements published in 1989 by the U.S. Food and Nutrition Board (77) contains a similar and perhaps slightly broader conclusion: “In view of the margin of safety in the RDA [recommended daily allowance] no increment is added for work or training” (p. 71). These conclusions are somewhat surprising given that as long ago as 1925 Cathcart, in a comprehensive review of this topic, concluded that “muscle activity does increase, if only in small degree, the metabolism of

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protein" (11, p. 239). Moreover, beginning in the 1970s and 1980s, experimental results from initial studies of endurance exercise (53) and later studies of strength exercise (45, 48) began to suggest that the importance of protein/amino acids in exercise metabolism may have been underestimated.

As a result, there has been a reexamination of much of the older data, and a considerable amount of new information (some of it utilizing techniques that were not available 100–125 years ago when this question was extensively studied) has been obtained during the past 15–20 years (49). This work indicates that active individuals can probably benefit from protein intakes as great as perhaps 50–125% above the current RDA. This does not mean that we should revert to the ideas of von Liebig; carbohydrates and fats are of critical importance during exercise, but we need to recognize the dynamic nature of protein metabolism during exercise. This paper examines some of the evidence regarding protein/amino acid needs for individuals who regularly engage in physical exercise.

Techniques to Assess Changes in Protein/Amino Acid Requirements

The major measurement techniques involve nitrogen balance and metabolic tracers. The nitrogen balance technique assumes that the dietary requirement for protein is that which provides enough nitrogen intake to balance nitrogen excretion in urine, feces, and sweat. To avoid deficiencies in a large portion (97.5%) of the population, the RDA is set at two standard deviations above this intake (22). Although considered a classic procedure, nitrogen balance is really a “black box” approach (Figure 1), and the valid interpretation of data derived from this technique is difficult. Problems with its use include the difficulty involved in accurately measuring all intake and excretion, study variability in length of any accommodation period to changes in protein intake, confounding effects of variable

Figure 1 — Overview of protein and amino acid metabolism. Arabic numerals indicate ways in which amino acids can enter the body’s free amino-acid pool (through which all amino acids flow). Roman numerals indicate ways that nitrogen can leave the free pool. Letters indicate ways that amino acid carbon can leave the free pool. C = carbon; NH₃ = ammonia; SYN = synthesis; DEG = degradation; CHO = carbohydrate.
energy intake, and the dissociation between highly positive nitrogen balance and muscle mass accretion while subjects are on high-protein diets (55).

Metabolic tracers labeled with stable or radioactive isotopes (e.g., $^{14}$C, $^{13}$C, $^{15}$N, etc.) can be used to assess protein/amino acid requirements because, unlike carbohydrate and fat, protein/amino acids have no storage form in the body (body proteins have structural or enzymatic functions). Therefore, when dietary supply exceeds need, oxidative removal (labeled CO$_2$) should increase markedly (90). Moreover, with metabolic tracers, unlike the nitrogen balance technique, estimates of the component processes of protein turnover (protein synthesis or degradation, Figure 1) can be made after an isotopic steady state is established and labeled end products (in breath and urine) and/or labeled constituents (in plasma or tissue) (86) are measured. Unfortunately, this technique can be hazardous (radioisotopes), invasive (requiring blood or tissue sampling), and expensive (stable isotopes). Further, it relies on several assumptions that have been challenged (26, 59, 66).

Evidence That Exercise Affects Protein/Amino Acid Metabolism

Strength/Power Exercise

If strength/power athletes require additional dietary protein/amino acids in comparison to their more sedentary counterparts, the most probable cause for the increased need would be to provide supplemental raw materials to enhance muscle protein synthesis or to cover an increased amino acid loss as a consequence of amino acid oxidation during exercise. In theory, the enhanced availability of amino acids from increased dietary protein (61), in combination with the powerful anabolic stimulus known to accompany chronic strength exercise (27), could promote an accelerated rate of muscle protein synthesis or a decreased rate of protein breakdown, resulting in greater muscle development. The very high protein intakes (4.0–6.2 g · kg$^{-1}$ · day$^{-1}$) and the prevalence of amino acid supplementation common to strength athletes (35, 40, 70) clearly indicate that athletes readily accept this possibility. Although there are some recent scientific data consistent with this idea, considerable controversy exists, due in part to the limitations of the measurement techniques used (complete or partial nitrogen balance and radio/stable isotopic metabolic tracers) and the established tradition that exercise has minimal effects on protein metabolism.

Utilizing continuous L-[1-$^{13}$C]leucine infusion during a typical 60-min strength/power training session (nine exercises, three sets of 10 repetitions of each exercise at 70% of one repetition maximum), Tarnopolsky et al. (73) observed no change in whole-body leucine oxidation during the exercise or for 2 hr of recovery (Figure 2). Although studies replicating these findings are needed, these data support the conclusion of Astrand and Rodahl (1) that, at least with strength/power exercise, protein/amino acids contribute very little to exercise fuel.

In contrast, several experiments from a number of different laboratories indicate that the current RDA (0.8 g · kg$^{-1}$ · day$^{-1}$) is suboptimal for individuals involved in heavy resistance (strength/power) exercise. For example, when elderly, untrained men received a dietary supplement (2,345 kJ) containing protein amounting to 23 g · day$^{-1}$ (approximately 50% of the RDA for this group) in combination with a 12-week strength training program, they experienced greater gains in thigh muscle area (based on computerized axial tomography scans) and greater urinary creatinine (an index of muscle mass)
Figure 2 — Effect of strength exercise on whole-body leucine oxidation (adapted from Reference 73).

when compared to a matched group who trained without receiving the supplement (Table 1) (24). Despite these mass effects, strength gains were similar in both groups. Whether these data indicate that the observed muscle size gains were nonfunctional or rather that strength gains due to muscle changes (as opposed to adaptations in the nervous system) are more delayed in older subjects than in younger subjects requires more study.

Fern et al. (20) observed significantly greater gains in body mass with 4 weeks of resistance training (initial training status was not indicated) when protein intake was 3.3 versus 1.3 g · kg\(^{-1}\) · day\(^{-1}\) (Figure 3). Further, metabolic tracer data indicated that although the strength training increased protein synthesis with both diets, the increase was fivefold greater on the higher protein intake. This observation is of considerable importance because it appears to be the first documentation that a protein intake of approximately four times the RDA, in combination with strength training, can promote greater muscle size gains than the same training with a diet containing adequate protein. Unfortunately, no strength data were reported, and the observed 150% increase in amino acid oxidation on the higher intake indicates that the optimal protein intake had been exceeded.

Lemon et al. (55) compared protein intakes of 2.62 versus 0.99 g · kg\(^{-1}\) · day\(^{-1}\) in novice bodybuilders during the first month of training. Using nitrogen balance and linear regression methodology, they concluded that the protein requirement was 1.5 g · kg\(^{-1}\) · day\(^{-1}\) and the recommended allowance (requirement + 2 SD) should be 1.7 g · kg\(^{-1}\) · day\(^{-1}\) (Figure 4). Unexpectedly, gains in strength (both voluntary and electrically evoked) and size (based on computerized axial tomography scans and needle biopsies) were similar under the two dietary regimens, even though the lower protein intake produced a negative nitrogen balance. Confirmational studies are needed, but it may be that short-term negative nitrogen balance does not adversely affect muscle gains with
Table 1  Effect of Protein Supplementation (23 g · day⁻¹) During 12 Weeks of Strength Training (Reference 24)

<table>
<thead>
<tr>
<th>Group</th>
<th>Thigh muscle area (cm²)</th>
<th>M</th>
<th>SE</th>
<th>Urinary creatinine (g/day)</th>
<th>M</th>
<th>SE</th>
<th>1 RM  (kg)</th>
<th>M</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>+6.1</td>
<td>2.4</td>
<td></td>
<td>-0.05</td>
<td>0.07</td>
<td></td>
<td>+19.0</td>
<td>2.9</td>
<td></td>
</tr>
<tr>
<td>Supplement</td>
<td>+14.6*</td>
<td>1.5</td>
<td></td>
<td>+0.16*</td>
<td>0.05</td>
<td></td>
<td>+20.2</td>
<td>2.1</td>
<td></td>
</tr>
</tbody>
</table>

Note.  n = 5 or 6 per treatment.

*1 repetition maximum strength.

*Significantly (p < .02) greater than control.

![Figure 3](image.png)

Figure 3 — Effect of increased dietary protein on body weight gains with 4 weeks of strength training (adapted from Reference 20).

strength training because endogenous nitrogen reserves could provide an alternative protein supply (60, 83). If so, this would likely only be possible for a limited duration, after which a diet higher in protein might prove to be superior.

Walberg et al. (81) estimated nitrogen balance in two groups of bodybuilders consuming differing protein intakes (0.8 versus 1.6 g · kg⁻¹ · day⁻¹) and hypoenergy diets (75.3 kJ · kg⁻¹ · day⁻¹). Although nitrogen balance was quite variable across the 7-day experimental period, perhaps indicating a lack of dietary compliance, the diet lower in protein was
consistently associated with a negative nitrogen balance, whereas the higher intake produced values that were generally positive. Confirming previous findings on endurance athletes by Goranzon and Forsum (30), these data clearly demonstrate an inverse relationship between energy intake and protein need in strength-trained athletes, even though the absolute values may be incorrect due to the estimated measurements or problems with dietary compliance.

Utilizing both nitrogen balance and tracer methodology in the same experiment, Tarnopolsky et al. (72) showed that the optimal protein intake for strength athletes is between 1.4 and 2.4 g · kg⁻¹ · day⁻¹. Further, their nitrogen balance data indicated that the RDA should be 1.76 g · kg⁻¹ · day⁻¹ for the strength athletes and 0.89 g · kg⁻¹ · day⁻¹ for the sedentary controls. The results of the sedentary control group included in this study are noteworthy because the nitrogen balance values are in the expected range (22, 77), adding further credibility to the values obtained for the strength athletes. Finally, an RDA of 1.76 g · kg⁻¹ · day⁻¹ is higher than the 1.2 g · kg⁻¹ · day⁻¹ reported in their earlier study (74) but consistent with the value determined for novice bodybuilders in the study reported by Lemon et al. (55). More importantly, measures of whole-body protein synthesis were elevated in the strength athletes when their protein intake increased from 0.86 to 1.4 g · kg⁻¹ · day⁻¹, without an increase in amino acid oxidation. In contrast, the additional dietary protein produced no change in protein synthesis in the sedentary controls (Figure 5). Similar to the Fern et al. (20) data, these results suggest that, when combined with bodybuilding exercise, a protein intake above the RDA will enhance muscle development. Interestingly, when the strength athletes consumed 2.4 g · kg⁻¹ · day⁻¹, amino acid oxidation increased with no further increase in protein synthesis. Clearly, this indicates a protein overload at 2.4 g · kg⁻¹ · day⁻¹.
Taken together, these experiments provide strong evidence that increased dietary protein can enhance muscle development when combined with appropriate heavy-resistance exercise training. However, of critical importance is the observation from the tracer studies that the efficacy of dietary protein appears to plateau somewhere between 1.4 and 2.4 g · kg\(^{-1}\) · day\(^{-1}\). This is consistent with the recent nitrogen balance data suggesting that the protein intake of strength/power athletes should be about 1.7–1.8 g · kg\(^{-1}\) · day\(^{-1}\) (225% of the current RDA). Therefore, it appears that even when energy intake is adequate, strength/power athletes will benefit from a protein intake exceeding the current RDA, but there is no good evidence that the very high protein intakes (>2 g · kg\(^{-1}\) · day\(^{-1}\)) routinely consumed by strength/power athletes are either necessary or beneficial. For individuals on hypoenergy diets, optimal protein intakes are probably slightly higher due to the inverse relationship between energy intake and protein need.

Chesley et al. (12), Rennie et al. (65), and Yarasheski et al. (89) measured muscle protein synthesis directly by quantifying L-[\(1^{-13}\)C]leucine incorporation into skeletal muscle (Figure 6). Use of this technique to confirm the increases in whole-body protein synthesis observed by Fern et al. (20) and Tarnopolsky et al. (72) with strength training and high protein intakes would be the definitive experiment to determine the optimal protein intake to maximize muscle development.

**Endurance Exercise**

If endurance athletes require more dietary protein/amino acids in comparison to their more sedentary counterparts, the most probable causes for the increased need would be to cover an increased loss of amino acids oxidized during exercise and to provide additional raw materials to replace any exercise-induced muscle damage, especially when the exercise has a large eccentric component (18). Unlike strength/power athletes,
endurance athletes do not have the long history of high protein/amino acid intake; however, according to anecdotal reports, many of these athletes have also recently begun to increase their protein/amino acid intake in an attempt to improve their endurance performances.

It is clear that the amino acid alanine is produced in skeletal muscle via transamination of NH$_3$ groups from the branched-chain amino acids to pyruvate (Figure 7) in an exercise intensity-dependent manner (Figure 8) (19, 62). These observations are of considerable importance relative to dietary protein/amino acid needs for endurance athletes, because they suggest that carbon skeletons from amino acids in either muscle or liver (the two major sources in the body) could be irreversibly lost via oxidation. This possibility has been documented in oxidation studies using tracers labeled with radio- and stable isotopes (50, 54, 65, 85). Moreover, this amino acid oxidation phenomenon has been shown to be dependent on exercise intensity (Figure 9) (2). The most likely explanation for this increased oxidation is the intensity-dependent activation of the limiting enzyme (branched-chain ketoacid dehydrogenase) in the oxidation pathway (39). The magnitude of this response can be significant; 2 hr of moderate-intensity exercise (55% VO$_{2}$max) can result in oxidation equivalent to 86% of the daily requirement for at least one of the branched-chain amino acids (Table 2) (17).

In addition to this dehydrogenase activity, the availability of the branched-chain amino acids is also important because increased dietary protein (23 versus 8% dietary energy as protein) elevates whole-body leucine oxidation in rodents both at rest and during endurance exercise (40 min at 24 m·min$^{-1}$ up a 10% grade), although the exercise-induced increase in leucine oxidation is not additive with the increase caused

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**Figure 6** — Muscle protein synthesis 4 hr and 24 hr after a strength training session (adapted from Reference 12).
Figure 7 — Metabolic pathway in liver and muscle showing alanine and urea formation as well as branched-chain amino acid oxidation (adapted from References 19 and 62).

Figure 8 — Effect of exercise intensity on alanine production from active muscle (adapted from Reference 19).
Whole Body Leucine Flux Oxidized (%)

Figure 9 — Effect of endurance exercise intensity on whole-body leucine oxidation in four subjects (adapted from Reference 2).

Table 2 Leucine Oxidation in Humans During 2 hr of Endurance Exercise at 55% VO$_2$max (Reference 17)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Rate (µmol · kg$^{-1}$ · hr$^{-1}$)</th>
<th>% of daily requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>SE</td>
</tr>
<tr>
<td>Rest</td>
<td>14.8</td>
<td>1.3</td>
</tr>
<tr>
<td>Exercise</td>
<td>46.1*</td>
<td>9.7</td>
</tr>
</tbody>
</table>

Note. $n = 8$ subjects/treatment.

*Significantly (p < .05) greater than rest.

by greater intake of dietary protein (Figure 10). Further, fasting for 14 or 84 hr (Figure 11) by human subjects increases whole-body leucine oxidation and decreases leucine incorporation into protein, both at rest (compare Figure 11 with Table 2) and during 2 hr of endurance exercise (45% VO$_2$max) relative to exercise without fasting (41).

Chronic exercise (training) may further elevate branched-chain amino acid oxidation, but not all the obtained data are consistent. Using incubated muscle slices from rodents that had been trained for 12 weeks (final training was 5 days · week$^{-1}$ for 2 hr · day$^{-1}$ at 29.5 m · min$^{-1}$ up an 8% grade, with 30-s sprints every 10 min), Dohm et al. (14) observed an increase in amino acid oxidation that was apparent by Week 8 (Figure 12). Henderson et al. (33) confirmed this training effect on leucine oxidation using in vivo measures (Figure 13). Probably these adaptations are due to an increased amount
Whole Body Leucine Oxidation
(nmol alpha-ketoisocaproate oxidized \cdot g^{-1} \cdot min^{-1})

Unlike letters P<0.05

23% Dietary Protein

Layman et al (1994)

Figure 10 — Effect of endurance exercise and protein intake on whole-body leucine oxidation (adapted from Reference 44).

Whole Body Leucine Metabolism (umol \cdot kg^{-1} \cdot h^{-1})

Rest * P<0.05 vs Rest
Exercise + P<0.05 vs 14 h Fast

84 h Fast

14 h Fast

Figure 11 — Effect of endurance exercise and fasting on whole-body leucine metabolism (adapted from Reference 41).
**Muscle Leucine Oxidation (nmol \cdot g^{-1} \cdot h^{-1})**

![Graph showing muscle leucine oxidation over training time.](image)

Figure 12 — Effect of endurance training on the in vitro leucine oxidation capacity of muscle (adapted from Reference 14).

**Whole Body Leucine Oxidation (umol \cdot kg^{-1} \cdot min^{-1})**

![Graph showing whole body leucine oxidation over V\text{O2}.](image)

Figure 13 — Effect of endurance training on whole-body leucine oxidation at rest (first data point for each group) and during exercise (adapted from Reference 33).
of the branched-chain ketoacid dehydrogenase in the active form, because training-induced increases of the enzyme have been measured in both muscle and liver (Figure 14) (44). Unless a compensatory decrease (below baseline) in oxidation occurs between training sessions, these data indicate that chronic endurance exercise is likely to elevate daily requirements for leucine. However, the training response may not be so straightforward, because muscle leucine oxidation in situ does not seem to be affected by training (9 weeks, 60 min · day\(^{-1}\), 30 m · min\(^{-1}\), 15% grade) (36).

The magnitude of total amino acid oxidation must wait for the study of other individual amino acids because oxidation rates may vary (88). Nevertheless, the exercise-induced increases in blood urea, the major end-product of amino acid metabolism, and the increased postexercise urea excretion observed in a variety of studies (47) suggest that overall protein needs could be affected. This is likely to be the case only for exercise that exceeds a certain intensity (perhaps 55–60% \(VO_2\)max, Figure 15); several studies that utilized less intense exercise did not observe increased protein use (10, 76, 87).

Using data from several prolonged exercise studies, Haralambie and Berg (32) observed that blood urea increased exponentially beginning at about 100 min of continuous exercise at 60–70% \(VO_2\)max (Figure 16). They interpreted this to mean that total amino acid oxidation must be significant at this time because any reduction in urea removal from the blood by the kidneys could not account for the observed increases in blood urea. It appears that reduced glycogen availability is responsible for this increased amino acid oxidation, because glycogen stores are known to be substantially reduced by 100 min of exercise at this intensity (34). In support of this suggestion, it has been shown that when initial glycogen stores are low prior to prolonged exercise, increases in blood urea occur earlier, increases in urea excretion (especially via sweat [52]) are greater, and there is greater activation of the branched-chain ketoacid dehydrogenase (82). Although indirect, these results also suggest that total amino acid oxidation can be substantial when exercise is prolonged.
Figure 15 — Effect of endurance exercise intensity on excretion of urea nitrogen (N) in urine (Lemon, Dolny, and Yarasheski, unpublished data).

Figure 16 — Effect of exercise duration on changes in serum urea concentration (adapted from Reference 32).
Based on nitrogen balance methodology (Figure 17), it appears that a protein intake of 1.0 g · kg\(^{-1}\) · day\(^{-1}\) is insufficient for individuals who begin an endurance exercise program (28). It has been suggested that this increased protein need is only transient, because it seems to disappear over a few weeks of accommodation to the exercise regime (Figure 18) (29). However, if this were correct, one would expect the RDA (0.8 g · kg\(^{-1}\) · day\(^{-1}\)) to be adequate for individuals who have trained regularly for years. Yet several studies from different laboratories utilizing either the nitrogen balance or the metabolic tracer technique have determined that the RDA should be about 1.2–1.4 g · kg\(^{-1}\) · day\(^{-1}\) (150–175% of the current RDA) for endurance athletes (7, 23, 58, 74). As mentioned previously, exercise intensity appears to affect protein utilization during endurance exercise. This could explain the less negative nitrogen balance observed with time in the Gontzea et al. (29) study, because the same exercise regime would represent a less intense stimulus as these previously untrained individuals experienced training adaptations.

Finally, there are other types of evidence from exercise studies indicating that regular endurance exercise increases dietary protein/amino acid needs. Examples include exercise-induced increases in the following: muscle ammonia (an end-product of amino acid metabolism); total and essential amino acids (ones that cannot be produced by the body) in muscle, without increases in branched-chain amino acids (56) (Table 3); the excretion of 3-methylhistidine (an amino acid that is quantitatively excreted in the urine following contractile protein degradation) (15); skeletal muscle urea content (51, 78) (Figure 19); and urinary urea excretion (16, 64, 75). These data from several different experimental approaches suggest that dietary protein needs of individuals who regularly engage in endurance exercise are greater than those of their more sedentary counterparts.

![Nitrogen Balance (g · d\(^{-1}\))](image)

Figure 17 — Effect of endurance exercise and protein intake on nitrogen balance (adapted from Reference 28).
Figure 18 — Effect of several weeks of accommodation to an endurance exercise session on nitrogen balance (adapted from Reference 29).

Table 3  Effects of Cycling to Exhaustion at 75% VO₂max on the Concentrations of Ammonia and Amino Acids in Muscle and Blood (Reference 56)

<table>
<thead>
<tr>
<th></th>
<th>Plasma</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonia</td>
<td>Increase</td>
<td>Increase</td>
</tr>
<tr>
<td>Total amino acids</td>
<td>No change</td>
<td>Increase</td>
</tr>
<tr>
<td>Essential amino acids</td>
<td>No change</td>
<td>Increase</td>
</tr>
<tr>
<td>Branched-chain amino acids</td>
<td>No change</td>
<td>No change</td>
</tr>
</tbody>
</table>

Drawing on the nitrogen balance data and using conventional linear regression techniques (22), Lemon (47) concluded that the protein RDA for individuals who regularly engage in endurance exercise should be about 1.2–1.4 g · kg⁻¹ · day⁻¹ (150–175% RDA). Unfortunately, most of these data were obtained on male subjects aged 20–40 years. Future studies need to concentrate on females (63, 71, 75) as well as on individuals from differing age groups (67), because it is possible that protein/amino acid needs with exercise differ in these populations.

**Adverse Effects of High Protein/Amino Acid Intake**

Many believe that excessive protein intakes are hazardous, but most of the support for such a contention has been extrapolated from studies on patients with compromised kidney function (5). There is no published evidence that the high protein intakes routinely
Urea N Content (ug · g body weight⁻¹)

Figure 19 — Effect in rats of endurance exercise (60 min at 25 m · min⁻¹, 0% grade) on tissue urea content (Lemon and Dolny, unpublished data).

consumed by strength/power athletes lead to kidney disease. Further, minimal problems have been observed in studies where rodents have been fed massive protein intakes (80% of energy intake) for more than 50% of their life spans (91). Apparently, the health concern regarding high protein intakes has been overstated, at least in individuals with normal kidney function. At present, there is certainly no reason to believe that protein intakes in the range of those recommended (<2.0 g · kg⁻¹ · day⁻¹) would cause any health concerns.

Protein/Amino Acid Supplementation Versus Dietary Sources

Clearly, many athletes believe that large dietary intakes of protein and amino acids are necessary for elite performance; the accumulating recent scientific data have further fueled what has become a very large commercial protein supplementation market (milk, egg, or soy protein powder, protein hydrolysates; free-form amino acids, etc.). However, even though dietary protein/amino acid needs for active individuals probably exceed the current RDA, it does not necessarily follow that dietary supplementation is necessary; in most cases, sufficient protein can be obtained from dietary sources. Before undertaking a program of supplementation an athlete should assess current intake, because many individuals consume adequate protein to cover the additional requirements of an active lifestyle. For example, dietary protein typically amounts to 10–15% of energy intake. If energy intake is adequate, this diet would contain sufficient protein (10% of a daily 10,500-kJ [2,500-kcal] intake would represent 63 g of protein/day or 0.9 g · kg⁻¹ · day⁻¹) for a 70-kg sedentary individual. Further, as one becomes more physically active, the resulting increasing energy intake would still result in sufficient protein intake (10% of a daily 21,000-kJ [5,000-kcal] intake would represent 126 g/day or 1.8 g · kg⁻¹ · day⁻¹).

Despite this typical scenario, use of protein supplements might be advantageous in some situations because the protein supplements are very low in fat, purines, and
cholesterol. As a result, partial substitution of supplements for protein-containing foods could reduce the atherogenic nature of diets high in animal protein that are frequently consumed by many athletes (especially those in strength/power activities). Moreover, it is possible that some groups of individuals may benefit from specialized supplementation. Examples include those whose diets are not well mixed or are low in protein quality (adolescents, vegetarians, or individuals living alone), those who consume low total energy (dieters or participants in weight-restricted activities, e.g., dancers, wrestlers, etc.), and those whose energy output is excessive (athletes involved in prolonged, heavy, multiday activities). More study is needed to ascertain the value of supplementation for these select groups.

Finally, although far from conclusive, some data suggest that individual amino acid supplementation may be beneficial for some types of exercise performance. Examples include branched-chain amino acids (which may affect central fatigue inhibition) (4, 42), tryptophan (69), aspartates (57, 84), potential growth-hormone-stimulating amino acids (8, 21, 37, 38, 43), and glutamate (6). This is an area where additional study is definitely needed, because few actual performance-enhancing effects have been documented (13, 25, 80), despite claims to the contrary, and because adverse effects of excess amino acid intake are known to occur (3).

Summary

Dietary protein requirements for individuals involved in regular exercise programs have been debated for many years. Current recommendations are based primarily on data derived from subjects whose lifestyles were essentially sedentary. As such, these recommendations may have little relevance for active individuals. More recent, well-designed studies using either the classic nitrogen balance approach or the more technically difficult metabolic tracer technique indicate that overall protein needs (as well as needs for some specific individual amino acids) are increased for those who exercise regularly. Strength athletes probably need about 1.4–1.8 g · kg⁻¹ · day⁻¹ and endurance athletes about 1.2–1.4 g · kg⁻¹ · day⁻¹. Although these intakes exceed the current RDA, there is no indication that they will cause any adverse side effects in healthy individuals. Further, they can be obtained without any special supplementation as long as a variety of foods are consumed and total energy intake is adequate. In fact, because most athletes consume very high dietary energy, it is possible to obtain these protein intakes with as little as 10% of total energy as protein intake. Further, in contrast to the belief of many that if a little more protein is good, a lot more must be better, there is essentially no valid scientific evidence that protein intakes exceeding about 1.8–2.0 g · kg⁻¹ · day⁻¹ will provide an additional advantage.

Despite the recent resurgence of investigation into the effects of exercise on protein metabolism, existing knowledge is embarrassingly rudimentary compared to that for carbohydrate and fat metabolism. There is still a tremendous amount of work to be done, especially in the area of actual exercise performance effects, before we can feel comfortable about our understanding of exercise and protein/amino acid needs.

References


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