REVIEW ARTICLE

Protein Requirements and Supplementation in Strength Sports

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Daily requirements for protein are set by the amount of amino acids that is irreversibly lost in a given day. Different agencies have set requirement levels for daily protein intakes for the general population; however, the question of whether strength-trained athletes require more protein than the general population is one that is difficult to answer. At a cellular level, an increased requirement for protein in strength-trained athletes might arise due to the extra protein required to support muscle protein accretion through elevated protein synthesis. Alternatively, an increased requirement for protein may come about in this group of athletes due to increased catabolic loss of amino acids associated with strength-training activities. A review of studies that have examined the protein requirements of strength-trained athletes, using nitrogen balance methodology, has shown a modest increase in requirements in this group. At the same time, several studies have shown that strength training, consistent with the anabolic stimulus for protein synthesis it provides, actually increases the efficiency of use of protein, which reduces dietary protein requirements. Various studies have shown that strength-trained athletes habitually consume protein intakes higher than required. A positive energy balance is required for anabolism, so a requirement for "extra" protein over and above normal values also appears not to be a critical issue for competitive athletes because most would have to be in positive energy balance to compete effectively. At present there is no evidence to suggest that supplements are required for optimal muscle growth or strength gain. Strength-trained athletes should consume protein consistent with general population guidelines, or 12% to 15% of energy from protein. *Nutrition* 2004;20:689–695. ©Elsevier Inc. 2004

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INTRODUCTION

Body proteins are constantly and simultaneously being made (synthesized) and degraded. This constant turnover provides for a mechanism of steady maintenance of potentially damaged and dysfunctional proteins. In skeletal muscle, protein turnover is also ongoing and provides the basis for skeletal muscle's plasticity in response to the degree of imposed high-intensity loading (resistance exercise). A schematic representation of skeletal muscle protein turnover and other muscle-specific metabolic fates of amino acids is shown in Figure 1. The extent to which the amino acids, liberated as a result of muscle proteolysis, are reused is extensive. This intracellular recycling, however, is not 100% efficient and amino acids are lost from skeletal muscle, often in appreciable quantities. The amino acids that are lost from skeletal muscle have numerous fates, but generally speaking are oxidized or converted to glucose via gluconeogenesis, with the amino nitrogen yielding urea. Obviously, the lack of efficiency in reusing amino acids from proteolysis means that we have a daily requirement to ingest protein.

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RESISTANCE EXERCISE AND PROTEIN TURNOVER: MECHANISMS OF HYPERTROPHY

Proteins are constantly and simultaneously being synthesized and degraded (Figure 1). Repair of damaged proteins and remodeling of structural proteins appears to occur as a result of a resistance exercise stimulus.[1] However, in human muscle, the process of myofibrillar protein turnover, at least that induced by resistance exercise, is a relatively slow one.[2,3] This slow turnover of muscle protein means that resistance exercise, even though it can induce changes in muscle fiber type and increase fiber diameter, requires a repeated exercise stimulus and a relatively prolonged period (6 to 8 wk) before an outward change in phenotype, such as a change in fiber type and hypertrophy, is observed.[2,4,5] Because resistance exercise does not induce an acute increase in protein turnover or oxidation during exercise,[6] it is the postexercise period when changes in muscle protein turnover, more specifically an increase in muscle protein synthesis, occur; this assertion has been confirmed numerous times.[1,7–11]

PROTEIN SYNTHESIS

For an increase in fiber diameter to occur, there has to be synthesis of new muscle proteins, more than 70% of which are myofibrillar, mostly actin and myosin, in nature. During the period of fiber hypertrophy, there also needs to be a net positive protein balance: muscle protein synthesis must always exceed muscle protein breakdown. Different investigations have shown that resistance exercise stimulates mixed muscle protein synthesis[1,7–9] in trained and untrained subjects. The time course of protein synthesis after an isolated bout of resistance exercise appears to be somewhat

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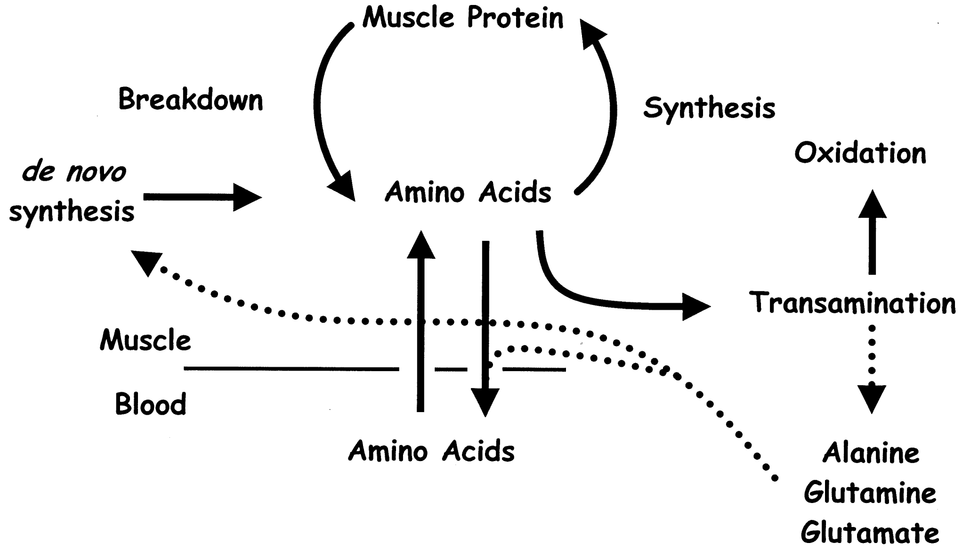

FIG. 1. Schematic of protein turnover and various metabolic fates of amino acids in skeletal muscle.

different in untrained subjects, for whom changes in the mixed muscle protein fractional synthetic rate persist for up to 48 h postexercise.[8] Results from cross-sectional comparisons have shown that prolonged resistance training actually attenuates the acute immediate response of muscle protein synthesis to an isolated bout of resistance exercise,[10] which one might expect as a general adaptation response to training. I and my colleagues recently confirmed these cross-sectional findings[10] by using a longitudinal design.[12] The implications of these findings[10,12] are that trained persons would likely require less protein after training to support the maximal protein synthetic response to a given workout.

PROTEIN BREAKDOWN

Resistance exercise stimulates an increase in the synthetic rate of muscle proteins[1,7–9] and there is a concomitant increase in the rate of muscle protein breakdown.[1,8,10] The tight relation between muscle protein synthesis and breakdown has been observed in a number of studies in which the two variables have been measured simultaneously.[1,8,10]

By using a surrogate marker of muscle myofibrillar protein degradation, urinary 3-methylhistidine, others have observed increases,[13–15] or no change[8,9,16] in this variable after resistance exercise. Why there is such disparity in the results from studies using 3-methylhistidine as an indicator of muscle proteolysis is likely related to the unknown contribution of gut tissue to whole-body proteolysis, which contains significant quantities of actin.[17] In studies where protein degradation has been directly measured after resistance exercise, it has been shown consistently that resistance exercise stimulates muscle protein degradation.[1,8,10] Recent evidence using microdialysis has shown that 3-methylhistidine release into the interstitium after resistance exercise also is not increased.[18] This finding[18] and the observed lack of change in 3-methylhistidine release with hyperinsulinemia that markedly reduces overall amino acid release[19] suggest that myofibrillar proteins are remarkably refractory to being degraded. It is likely that almost solely non-myofibrillar proteins are being degraded and are contributing to amino acid release after resistance exercise.[1,8,10]

PROTEIN BALANCE

Every study that has measured muscle protein balance (synthesis minus breakdown) after resistance exercise has found that, while synthesis is markedly elevated (in some cases >150% above baseline levels), muscle balance is negative[1,8,10] until amino acids are provided intravenously (to simulate postprandial concentrations) or orally.[20–22] This feeding-induced stimulation of muscle protein synthesis[20–24] has been shown to be independent of insulin[25] and is likely reflective of an increased delivery of amino acids to the muscle.[26,27] The effects of feeding and resistance exercise are also independent and additive, due mostly to a feeding- and exercise-induced stimulation of muscle protein synthesis (Figure 2A). Hence, it appears that feeding and resistance exercise combine in the fed state to increase protein synthesis above normal and, thus, protein balance to a greater extent than feeding or resistance exercise alone (Figure 2B). In addition, in the fasted state, muscle protein balance is less negative due to a stimulation of protein synthesis.[1,8,10] Therefore, hypertrophy is the result of the accumulation of successive periods of positive protein balance after exercise when protein is consumed. A lesser contributor to resistance exercise-induced muscle protein gains would be the reduction in fasted negative protein balance brought about by exercise (Figure 3).[1,8,10]

A recent study by Bohé et al.[28] demonstrated that extracellular rather than intracellular amino acid concentration is the controlling parameter in stimulating muscle protein synthesis and that the relation between the two is hyperbolic. These findings[28] demonstrated a plateau in muscle protein synthesis with increasing delivery of amino acids, implying that consumption of larger protein meals would stimulate muscle protein synthesis only up to a point. Protein consumed over and above a level that stimulates protein synthesis would result in only increased urea production. Presumably, the same relation would hold true for the postexercise period. The metabolic "machinery" responsible for muscle anabolism within skeletal muscle does, however, have the capacity to respond to repeated doses (3 to 6 g) of amino acids given only hours apart.[29,30] Further, the difference in the dose of essential amino acids given was two-fold (3 g[30] and 6 g[29]) and response was scaled to dose, implying that these small protein doses do not "top out" the synthetic response. In addition, Tipton et al.[31] showed that, after resistance exercise, consumption of two 15-g boluses of essential amino acids before and 1 h after resistance exercise elicited similar anabolic responses. Exactly which oral dosage of protein or amino acids would elicit a plateau is not known, but the findings of Bohe et al.[28] showed that synthesis does plateau; hence,

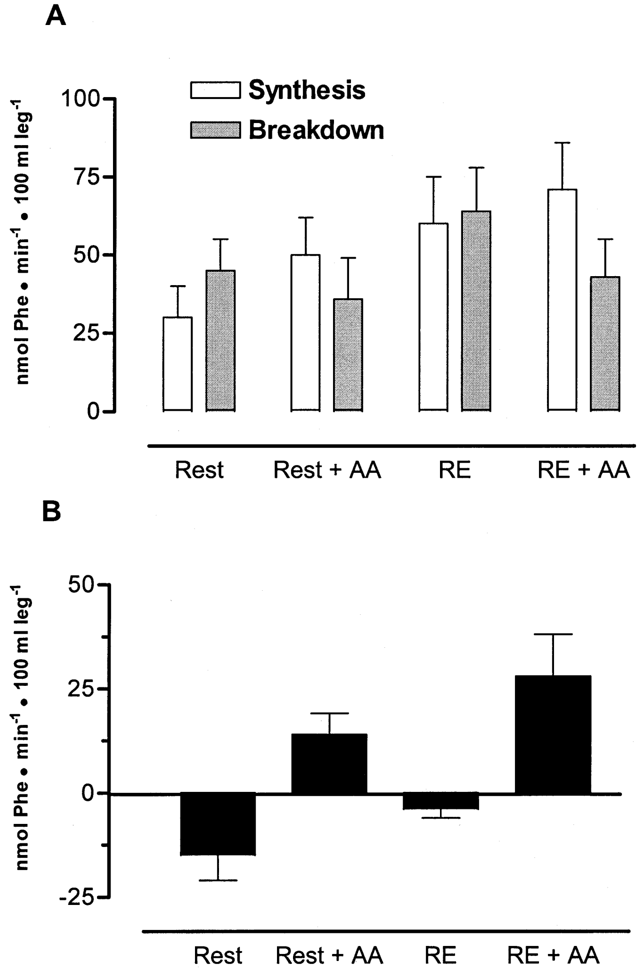

FIG. 2. (A) Influences of AA consumption at rest, performance of RE, and AA consumption after RE on muscle protein synthesis and breakdown. (B) Net protein balance (synthesis minus breakdown) under the same conditions. Data are redrawn from Biolo et al.[1,20] Values are means ± standard deviation. AA, amino acid; RE, resistance exercise.

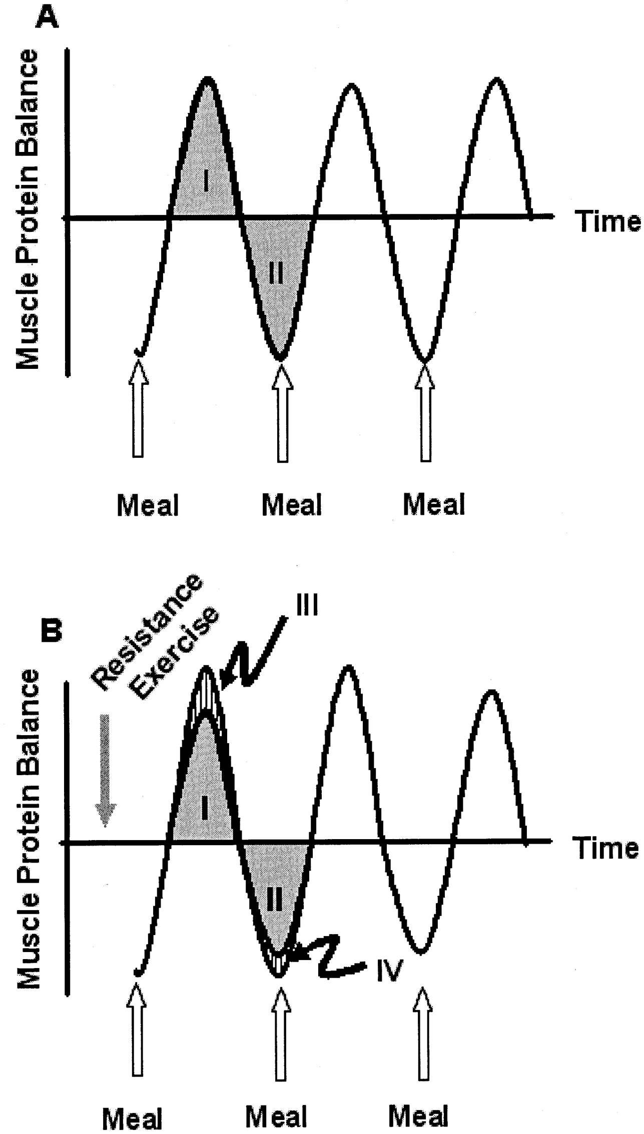

FIG. 3. (A) Normal fed-state gains and fasted-state losses in skeletal muscle protein balance (synthesis minus breakdown). The area under the curve in the fed state (I) would be equivalent to the fasted loss area under the curve (II); hence, skeletal muscle mass is maintained by feeding. (B) Fed-state gains and fasted-state losses in skeletal muscle protein balance with performance of resistance exercise. In this scenario, fasted-state gains are enhanced by an amount equivalent to the stimulation of protein synthesis brought about by exercise (III). In addition, fasted-state losses appear to be less (IV) due to persistent stimulation of protein synthesis in the fasted state.[10]

at some point the system would become unresponsive to increasing amino acid delivery.

The effect of the timing of delivery of amino acids relative to exercise has been examined acutely[21,32] and long term[33] after resistance exercise. Insofar as timing of postexercise consumption of protein supplements (6 g of amino acids plus 35 g of sucrose) is concerned, it appeared to make little difference as to whether a protein plus carbohydrate supplement was consumed 1 h or 3 h postexercise because the same positive net protein balance resulted at both times.[21] In another investigation by Tipton et al.,[32] pre-exercise consumption of the same protein plus carbohydrate supplement used previously[21] did augment muscle protein balance. The long-term practice of pre-exercise protein consumption would, according to these results,[32] result in improved gains in muscle protein mass as a result of resistance exercise. This has not been tested in a long-term setting.

Esmark et al.[33] used a long-term design to examine the influence of timing of protein supplementation in supporting hypertrophy in elderly males. They found that delaying delivery of a supplement by 2 h after resistance exercise and the delivery of protein (10 g) and carbohydrate (7 g; 420 kJ total energy) do not result in muscle hypertrophy after 12 wk of resistance training (three sessions per week). More importantly, the 2-h delayed supplement group had inferior strength gains versus a group that received the same supplement immediately postexercise, in whom muscle hypertrophy (25% increase in mean muscle fiber area) was observed. These findings[33] are striking given the small amount of protein (10 g) that was ingested by both groups and that a delay of only 2 h in ingesting that protein had such profound physiologic effects, such as absence of hypertrophy and lesser strength gains. Obviously, one major difference between the acute[21] and long-term[33] studies was the age of the subjects. However, even in the absence of food intake, resistance exercise has been shown to stimulate muscle protein synthesis at 24 h[7,8] and up to 48 h postexercise in young persons.[8] That fasted protein synthesis is stimulated for so long after resistance exercise in the young[7,8] and that feeding and resistance exercise synergistically add to each other to produce an enhanced synthetic response (Figure 2B) imply that the elderly may have a markedly shorter synthetic response to exercise or an insensitivity to amino acid feeding, assuming the data of Esmark et al.[33] can be generalized. Alternatively, as suggested by the results of Volpi et al.,[34] the elderly subjects studied by Esmark et al.[33] may have had an age-related resistance to insulin, which might have blunted their anabolic response to consumption of an amino acid and protein supplement.

PROTEIN REQUIREMENTS IN STRENGTH-TRAINED ATHLETES

Resistance exercise is followed by a period lasting as long as 48 h[8] when rates of muscle protein synthesis are elevated above resting levels.[1,7,9,10,20] The observation that protein synthesis rates are elevated after acute bouts of resistance exercise and that infusion or consumption of amino acids (i.e., protein) synergistically adds to the exercise response[20,21,29–31] provide the underlying basis for skeletal muscle growth. Observations of increases in lean body mass and muscle hypertrophy after long-term resistance training[5,35–37] are obviously the result of periods in which net protein balance (synthesis minus breakdown) has been positive (Figure 1)[31]; this occurs only when feeding and resistance exercise are superimposed (Figures 2B and 3). Hence, an additional requirement for protein in a group of individuals engaging in strength training theoretically may come about due to an increased requirement for protein to support protein synthetic gains (Figure 2B). In addition, protein needed to repair any ultrastructural damage in muscle tissue occurring as a result of some eccentric component to the activity[38,39] may lead to an increased requirement for dietary protein in athletes wishing to increase their lean body mass.[40,41] Studies have been conducted in which the protein requirements of resistance-trained athletes have been directly examined and the protein requirements of these habitually exercising persons have been determined to be greater than those of comparable sedentary persons.[40–42]

Despite the preceding proposed rationale for why a strength-trained athlete might have an increased requirement for dietary protein, in addition to experimental evidence,[40–42] there is no consensus, at least in reviewed scientific literature, as to whether habitual resistance exercise increases protein requirements.[43–45] The current dietary reference intakes (DRIs) set protein intake at 0.8 $g \cdot kg^{-1} \cdot d^{-1}$, and there is no recommendation for consumption of extra protein with exercise.[46] That there is no general concurrence on the issue of elevated dietary protein requirements for athletes likely arises from a number of confounding issues; more importantly, it is less than clear what the best method is when it comes to estimating protein requirements.[47] It has been proposed that there are inherent problems in conducting studies of protein requirements in habitually active persons,[44] which have led to a flawed interpretation of data from studies in which the dietary protein requirements in athletes have been found to be elevated.[40–42]

Tarnopolsky et al.[40] conducted a study using the nitrogen balance approach to examine the protein requirements of a group of resistance-trained athletes and a group of sedentary controls. Tarnopolsky et al.[6] previously demonstrated that an isolated bout of resistance exercise does not increase leucine oxidation or perturb whole-body protein turnover. It would appear that any extra protein required by strength-trained individuals is directed toward muscular hypertrophy in the earlier phases of training, when muscle mass is still increasing. In contrast, in highly trained powerlifters and bodybuilders, in whom muscle mass is high but stable, it is unlikely that their dietary protein requirements are elevated much more than those of a sedentary person. In fact, any increase in protein requirements for such a highly trained group of individuals is likely due to an increased rate of resting protein turnover. In support of the idea that training might induce an increase in resting muscle protein turnover, protein requirements of highly trained bodybuilders were found to be only 12% greater than those of sedentary controls who had a protein requirement of 0.84 $g \cdot kg^{-1} \cdot d^{-1}$.[40] The results of this study[40] highlight a consistent yet puzzling result. When consuming a protein intake (actually equivalent to the habitual protein requirement of the bodybuilders) of approximately 2.8 $g \cdot kg^{-1} \cdot d^{-1}$, all bodybuilders were in highly positive nitrogen balance (~12 to 20 g of nitrogen per day). When extrapolated back to actual body protein, this means that the bodybuilders should have gained approximately 300 to 500 g of lean mass per day (assuming a tissue water content of 75%), which obviously did not occur.[40] The increasingly positive nitrogen balance associated with higher protein intakes that was observed in this[40] and other[41,42] studies is often incorrectly used to justify the high protein intakes for resistance-trained athletes. Such shortcomings of nitrogen balance have long been recognized and have led to the recommendation of combining tracer and nitrogen balance approaches to determining protein requirements.[48] Despite the criticism of nitrogen balance approaches to determining protein requirements, it is still the approach that underlies the establishment of the dietary reference intake for protein in sedentary persons,[46] so the same flaws would be inherent in determining protein requirements for the athletic and sedentary populations.[49]

One study combined nitrogen balance and kinetic measurements of whole-body protein turnover and showed that protein requirements for American football and rugby players were almost 100% greater than those of a sedentary control group.[41] Consumption of a "low" protein diet (0.86 $g \cdot kg^{-1} \cdot d^{-1}$) by the strength-training group resulted in an accommodated state in which whole-body protein synthesis was reduced as compared with "medium" (1.4 $g \cdot kg^{-1} \cdot d^{-1}$) and high (2.4 $g \cdot kg^{-1} \cdot d^{-1}$) protein diets. In contrast to the results of Tarnopolsky et al.,[40,41] nitrogen balance studies conducted in the elderly have shown that a program of strength training results in reduced protein requirements due to the anabolic stimulus of the resistance exercise.[50,51] The results of Campbell et al.[50,51] are similar to those reported by Torun et al.[52] in showing that low-intensity isometric exercise routines improve protein use. Support for the possibility that more intense resistance exercise can improve nitrogen economy at the muscle tissue level can be found in the results of Phillips et al.[8,12] who showed that, in the fasted state, an isolated bout of resistance exercise increases muscle net protein balance, implying an improved intracellular reutilization of amino acids. Others have observed that exercise per se as opposed to the creation of an energy deficit via diet improves dietary protein retention.[45,52,53] A reason for the discrepancy may be that the athletes studied by Tarnopolsky et al.[40,41] were well trained and were performing exercise that was more intense than that described in the studies that showed a reduction in protein requirements.[45,51,54] In addition, nitrogen balance, particularly in long term studies,[51,54] may be less reflective of requirements but rather of mechanisms that result in an accommodated state.[49] At issue is whether the accommodation by athletes to lower protein intakes would result in a reduced level of synthesis of some proteins that might compromise performance; however, to test such a hypothesis would be very difficult. Millward et al.[43,44] detailed some of the reasons protein requirements are hard to determine for athletes.

An analysis of nitrogen balance data[40–42] from persons who were in a steady state of strength training or performing structured rigorous training involving resistance exercise is shown in Figure 4 and demonstrates that nitrogen balance for these athletes is achieved at a protein intake almost 49% greater than the current dietary reference intake (1.19 $g \cdot kg^{-1} \cdot d^{-1}$). Inclusion of a 95% confidence interval to the regression line to achieve zero balance yields a protein intake of 1.33 $g \cdot kg^{-1} \cdot d^{-1}$, or almost 66% greater than the current dietary reference intake (Figure 4). The data presented in Figure 4 represent those from a variety of studies in athletes completing resistance exercise at different intensities

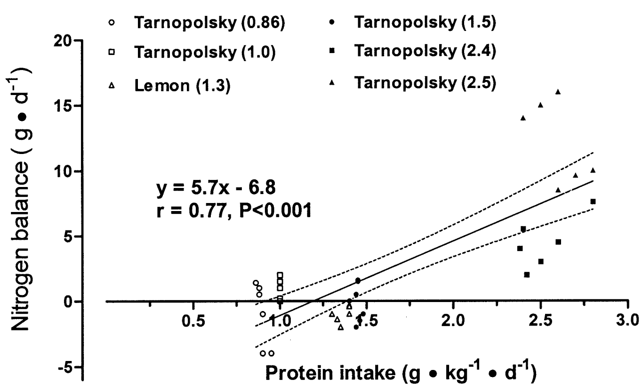

FIG. 4. Retrospective analysis of nitrogen balance data[40–42] in males engaging in resistance exercise. Numbers in parentheses are approximate protein intakes.

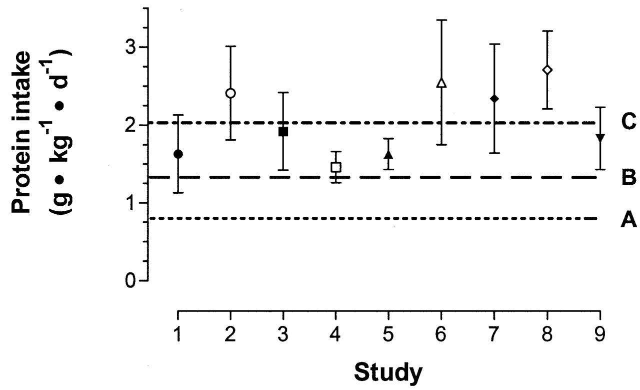

FIG. 6. Reported habitual protein intakes in resistance-trained athletes in studies 1,[7] 2,[63] 3,[43] 4,[42] 5,[64] 6,[65] 7,[65] 8,[40] and 9.[41] Dietary reference protein intake (0.8 g · kg^{-1} · d^{-1}) is shown by line A, an estimated "safe" protein requirement (1.33 g · kg^{-1} · d^{-1}) extrapolated from Figure 5 is indicated by line B, and the mean reported mean protein intake (2.05 g · kg^{-1} · d^{-1}) is indicated by line C. Values are means ± standard deviation.

with different levels of experience. Exercise intensity, duration, frequency, and training status may influence whether someone requires more protein. The data of Gontzea et al.[54] (Figure 5) highlight the fact that unaccustomed endurance exercise can induce a negative nitrogen balance, albeit transiently, but that consuming 1.0 g · kg^{-1} · d^{-1} is sufficient and results in zero balance after approximately 12 d of exercise (6 × 20 min/d of intense cycling). Lemon et al.[42] showed that novice weightlifters require more dietary protein (1.4 to 1.5 g · kg^{-1} · d^{-1}) than do more experienced weightlifters (1.05 g · kg^{-1} · d^{-1}), as reported in a previous study.[40] In addition, when intense weightlifting is combined with training for sports with power and aerobic bases (rugby and football), protein requirements have been reported to be as high as 1.76 g · kg^{-1} · d^{-1}.[41] After an initiation phase of any resistance training program and the initial adaptation to the performance of exercise are over (Figure 5), it is hard to reconcile that resistance-trained athletes would have markedly elevated protein requirements.

An informative and revealing point in any examination of the adequacy of protein to support lean mass gains in strength-training athletes, or to support existing lean mass in well-trained strength athletes, would be to examine their habitual protein intakes. A compilation of studies that have reported habitual protein intakes of strength-training athletes is shown in Figure 6. The mean habitual protein intake observed in these studies was 2.05 g · kg^{-1} · d^{-1}. Clearly, at these levels of protein intake, assuming that reported protein intakes from diet records are accurate; these athletes are meeting their protein requirements by any standard. Hence, any discussion of "safe" or adequate protein intakes for strength-training athletes appears to be dubious. Most, if not all, strength-training athletes are getting the protein intake required for gaining and/or maintaining any amount of skeletal muscle mass! Protein supplements, although convenient, are quite obviously (Figure 6) not necessary for most resistance-trained athletes.

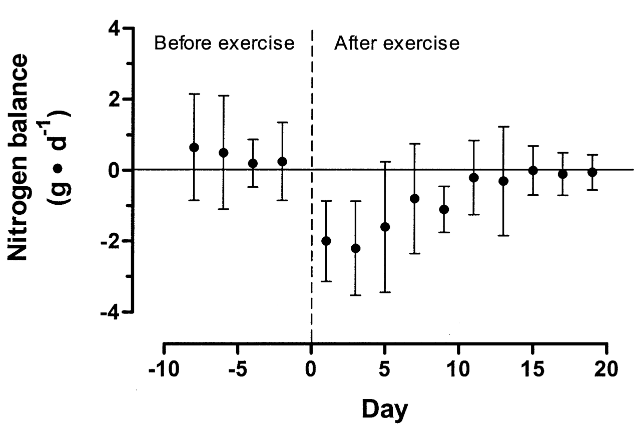

FIG. 5. Time course of adaptation in nitrogen balance after the initiation of exercise training in novices (adapted from Gontzea et al.[54]). All subjects consumed protein at the rate of 1 g · kg^{-1} · d^{-1} throughout the experiment and energy 10% greater than their calculated requirement. Values are mean ± standard deviation.

The influence of energy intake cannot be ignored in determining protein requirements.[55] Landmark studies[45,53,56] have clearly demonstrated that energy intake may be as, if not more, important than protein intake in determining nitrogen balance. What these early, elegantly designed studies showed was that, even when no protein is consumed, increasing energy intake improves nitrogen balance. Conversely, even when consuming relatively high protein intakes, positive nitrogen balance was not possible until energy balance was positive; however, exercise modified this relation and actually increased nitrogen balance, even in the face of a deficit in energy balance.[45,53] Hence, athletes who are trying to "make weight" for a particular competition by decreasing their caloric intake to deficit levels may lose some lean mass.[57,58] Importantly for this review, however, is the knowledge that performance of resistance exercise during hypoenergetic periods appears to attenuate, if not completely ablate, the loss of lean body mass.[59] In addition, consumption of a moderately higher amount of protein than normal (27% of energy intake, which was ~6 MJ/d, or ~100 g of protein) during energy restriction may attenuate and perhaps even completely prevent lean mass losses[60]; however, this effect seems to be more pronounced in females. In terms of macronutrients that support protein retention, isoenergetic substitutions of fat for carbohydrate have clearly shown that carbohydrate is protein sparing.[61] Hence, a recommendation for athletes attempting to spare protein (i.e., muscle mass) during hypoenergetic periods would include the advice to perform resistance exercise,[57–59] consume a higher than average amount of energy intake as protein (say 20% to 25% versus 15%),[60] and adequate quantities of carbohydrate to try and keep muscle glycogen relatively high (for performance), and support protein retention.[61]

SUMMARY

Muscle anabolism occurs when protein is consumed but is stimulated to a greater degree when resistance exercise is performed

(Figure 2B). Hypertrophy of muscle requires that a period of net positive protein balance occur and, consistent with the rate of turnover of muscle proteins, takes a relatively long time to be observed. The summative effect of acute periods of positive balance resulting from protein consumption and performance of resistance exercise are what ultimately lead to hypertrophy (Figure 3).

Although more studies need to be carried out to make a definitive statement regarding timing of protein intake relative to exercise and its effect on muscle mass and/or strength gains, it is likely that an athlete who consumes protein (plus carbohydrate) sooner and more often after exercise would provide a better environment for anabolism based on other evidence showing that the rate of synthesis of new muscle proteins has a ceiling and that consumption of protein above a certain level would not stimulate protein synthesis further. How much protein would have to be consumed to maximally stimulate muscle protein synthesis is not known; however, large protein meals, in excess of the protein required to maximally stimulate muscle protein synthesis, would not likely offer any benefit to athletes if consumed after resistance exercise. In this situation, amino acids in excess of those used to support protein synthesis would be directed toward oxidation and ultimately lead to increased urea production (Figure 1).[28]

Several studies have shown that protein requirements for strength-trained or training athletes are elevated above those of sedentary individuals (Figure 4). In contrast, several other reports have suggested that exercise results in a more economic use of protein and may actually reduce protein requirements.[45] Retrospective analysis of available data (Figure 4) has indicated that a "safe" level of protein intake for strength-trained athletes is 1.33 $g \cdot kg^{-1} \cdot d^{-1}$; however, this estimate is based on nitrogen balance, which is at best a badly flawed approach for examining protein requirements. All things considered, it is abundantly clear that any protein requirement set for strength-training athletes is of little relevance, considering that these athletes habitually consume protein far in excess of any recommended level, even the pseudo-recommendation based on Figure 4, in their normal diet. In other sports of which strength and power are components, e.g., wrestling, rugby, ice hockey, or American football, a requirement for dietary protein would be easily met when the athlete is consuming adequate energy, which may have a much greater influence on protein requirements than protein itself. Therefore, as a guide, I believe that the joint position statement of the American College of Sports Medicine, the American Dietetic Association, and the Dietitians of Canada[62] is the best guide that can be given: "Data are not presently available, however, to suggest that athletes need a diet substantially different from that recommended in the Dietary Guidelines for Americans or the Nutrition Recommendations for Canadians (55% to 58% of energy from carbohydrate, 12% to 15% of energy from protein, and 25% to 30% of energy from fat)." There is no evidence to suggest that protein supplements are more effective than consumption of high-quality protein from standard dietary sources.

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