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Editorial

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Preventing Muscle Atrophy with Protein and Amino Acid Supplementation

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There is strong evidence supporting the role of protein and Amino Acid (AA) supplementation in the prevention of muscle protein breakdown and augmentation of muscle protein synthesis when ingested at both pre- and post-exercise [1-5]. Specifically, chronic protein and AA supplementation in conjunction with strength training has been linked to increased strength, power, myofribrillar protein, and myosin heavy chain I and IIa expression [5,6]. Acute protein and AA supplementation has also been shown to attenuate muscle proteolysis caused by endurance exercise [7]. Collectively, these data indicate that protein and AA supplementation may increase protein synthesis and decrease the protein degradation occurs with varying types of exercise. This evidence leads to the question of the potential effects of protein and AA supplementation in preserving skeletal muscle during times of injury, immobilization, and disuse.

In all forms of muscle atrophy there appears to be a shift in the balance between synthesis and degradation, but during unloading and disuse conditions, the decrease in protein synthesis appears to drive the loss of muscle mass, while the rate of protein degradation remains fairly constant [8,9]. However, damage to skeletal muscle caused by injury or damaging eccentric exercises has been shown to activate the ubiquitin proteolytic system to increase protein degradation [10,11]. So in the case of muscle atrophy caused by injury, both an increase in protein degradation and a decrease in protein synthesis occur. Hence, both injury and disuse results in lowered protein synthesis, so protein and AA supplementation may therefore be beneficial.

For injured and eccentric exercise-induced muscle damage, research has shown that the ingestion of protein and AAs induces an anabolic effect in the skeletal muscle resulting in an immediate increase in net protein balance as compared to a placebo group [1,3]. In the specific case of sarcopenia prevention, results showed that the ingestion of essential and nonessential AAs increased muscle protein synthesis, but there was no change in breakdown in both groups [12]. The authors concluded that the essential AAs are primarily responsible for AA-induced protein anabolism in the elderly. Building on this information, researchers evaluated if chronic ingestion of essential AAs for 28 days would help attenuate muscle atrophy and muscle protein breakdown during muscle disuse [13-15]. Protein fractional synthesis rate was higher in the essential AA supplemented group than the placebo group on the first and last day of 28-days of bedrest. Additionally, lean leg mass was maintained throughout bedrest in the essential AA group, but fell in the placebo group, and strength loss was more pronounced in the placebo group compared to the essential AA group. The authors concluded that essential AA supplementation may be a possible intervention in prevention of atrophy as a result of prolonged disuse, and my also play a large role in stimulating muscle protein synthesis. However, a recent study [16] showed that protein and AA supplementation with 28 days of lower leg immobilization was not effective in maintaining leg lean mass or strength when compared to the carbohydrate placebo group.

The aforementioned studies are important in indicating the potential role of protein and AA supplementation in preventing muscle atrophy. However, with the conflicting data, it appears that the effectiveness of supplementation is dependent upon the cause of the atrophy. Protein and AA supplementation appears to be more effective with injury or eccentric exercise-induced muscle damage, and ineffective in some instances of disuse. It may be that when an athlete sustains an injury, it is best to supplement with protein and AAs initially after the damage, prior to atrophy that is caused by disuse during recovery from the injury, but more research exploring the timing of the supplementation is necessary.

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