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The Effect of Resistance Training In Combination with L-Arginine Supplementation on the Expression of Myonectin and Myostatin Genes in the Soleus Muscle of Young Male Rats

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ABSTRACT

The myonectin and myostatin genes play crucial roles in the regulation of muscle growth. Myostatin acts as a negative regulator of muscle growth, while myonectin promotes muscle development. The aim of this study was to investigate the effects of resistance training in combination with L-arginine supplementation on the expression of myonectin and myostatin genes in the soleus muscle of young male rats. Thirty-two young male Wistar rats were randomly assigned to four groups: control, supplement (L-arginine), combined (resistance exercise + L-arginine supplementation), and exercise (resistance exercise only). Resistance training consisted of eight weeks of ladder climbing with moderate intensity (70% of maximal voluntary contraction, MVCC) for five days per week. Rats in the L-arginine supplementation group and the combined exercise + supplementation group received L-arginine supplementation post-exercise, five days per week for the duration of the 8-week protocol. The dosage of L-arginine was administered by gavage to the exercise and supplementation groups. The relative expression of myonectin and myostatin genes was measured using Real-Time PCR. Data were analyzed using two-way analysis of variance (ANOVA) followed by a post-hoc test. Results from the two-way ANOVA revealed that the expression of the myonectin gene in the soleus muscle was significantly higher ($P = 0.001$) in both the exercise and exercise + L-arginine supplementation groups compared to the untrained groups, with and without supplementation. In contrast, the expression of the myostatin gene was significantly lower ($P = 0.002$) in the trained groups compared to the untrained groups, regardless of supplementation. These molecular adaptations suggest an increased potential for muscle growth and a reduction in inhibitory signaling, creating a more favorable environment for muscle maintenance and development. Therefore, the combination of resistance exercise and L-arginine supplementation may be an effective strategy to promote muscle hypertrophy and prevent muscle atrophy in young skeletal muscle. Further studies are required to clarify the underlying mechanisms of these effects and to evaluate their clinical relevance.

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Introduction

Recovery is a critical component of athletic performance, influencing both training and dietary strategies. This process begins immediately after exercise and continues until the next session, facilitating faster recovery, reducing fatigue, and minimizing the risk of injury, as fatigue can impair coordination and control [1]. Key factors contributing to post-exercise fatigue include elevated body temperature, dehydration, depletion of energy stores—particularly muscle glycogen—metabolic by-products such as lactic acid, central nervous system fatigue, and exercise-induced muscle damage [1,2]. Therefore, effective recovery requires appropriate nutritional strategies and interventions to accelerate fatigue relief [3].

Athlete nutrition must be carefully balanced to meet the heightened energy and nutrient demands induced by both physical and psychological stress. These nutritional requirements vary depending on the type, intensity, and duration of the sport [3,4]. The increased exertion and energy expenditure during training and competition often raise the need for specific nutrients, such as protein, with some athletes finding it challenging to match energy intake with demand [5,6]. Skeletal muscle, now recognized as an endocrine organ, plays a key role in influencing metabolism and communicates with other tissues through myokines—hormones secreted by muscle that

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regulate metabolic and inflammatory processes [5]. Research has increasingly focused on how exercise modulates the secretion of myokines and their effects on metabolism, although the underlying molecular pathways remain incompletely understood [6].

Myostatin (GDF-8), produced by skeletal muscle and encoded by the MSTN gene, functions as a negative regulator, limiting muscle growth through myogenesis and signaling via activin type II receptors [7]. It inhibits muscle hypertrophy from embryonic development onwards. Lower myostatin levels have been associated with increased muscle fiber number and size in animal studies, emphasizing its critical role throughout life [8]. Physical activity, particularly resistance training, is recognized as a key intervention to counter age-related muscle atrophy in the elderly, often promoting muscle hypertrophy by enhancing protein synthesis and activating satellite cells [8]. In addition to exercise, nutritional interventions have also been explored as potential treatments for muscle-wasting disorders, such as sarcopenia [9].

Amino acids are widely used supplements among athletes, as protein—essential for growth and tissue repair—is derived from the breakdown of dietary sources [3]. Following exercise, amino acids facilitate muscle repair and rebuilding, underscoring the importance of post-exercise nutrition for recovery and performance [5].

Arginine, a non-essential amino acid, stimulates growth hormone release, enhances blood flow, and plays a significant role in cardiovascular health and tissue repair. It reduces platelet aggregation and, by promoting growth hormone secretion, contributes to muscle mass development, amino acid transport, and fat metabolism [4, 5]. Arginine supplementation, either alone or in combination with other compounds, may enhance athletic performance, partly by increasing nitric oxide-mediated vasodilation [4, 5].

Evidence supports the benefits of L-arginine supplementation during resistance training, particularly its positive effects on muscle mass and anabolic hormone levels [10]. Additionally, it appears to reduce central fatigue and muscle damage during exercise and may stimulate mitochondrial biogenesis in muscle tissue [11].

Given the critical role of muscle function in athletic health, resistance training combined with protein supplementation remains a key area of research. However, variations in the type, duration, and intensity of training across studies have led to inconsistent findings regarding the regulation of muscle growth. Notably, much of the existing research has focused on aerobic or endurance exercise, with fewer studies examining the effects of resistance training on key regulatory genes. Therefore, the aim of this study is to investigate the impact of resistance training and L-arginine supplementation on the expression of myonectin and myostatin genes in the soleus muscle of young male rats.

Materials and Methods

The present study employed an applied experimental design with a multi-group setup, including a control group. Thirty-two two-month-old male Wistar rats were obtained from the Pasteur Institute of Iran. After a one-month acclimation period to the new environment, the rats were randomly assigned to one of four groups: control, exercise, combined (exercise + supplement), and supplement. The study duration was two months. All procedures adhered to ethical guidelines outlined in the Helsinki Declaration and were approved by the Laboratory Animal Ethics Committee, ensuring the avoidance of unnecessary harm or distress to the animals. To minimize stress and physiological changes, the rats were housed under standard conditions for two weeks: temperature of 22±2°C, relative humidity of 50±5%, and a 12:12 hour light-dark cycle. The animals had unrestricted access to standard food and water, with daily food intake precisely measured and recorded. Rats were housed in polycarbonate cages measuring approximately 21×34×54 cm. Each rat received approximately 20 grams of pellet food daily, with amounts adjusted according to weight gain. The food was sourced from Pars Animal Feed Company.

Rats in the exercise groups underwent a familiarization period, which involved five days of ladder climbing without added weights. Following this period, the maximum voluntary carrying capacity (MVCC) was determined, defined as the heaviest load the rat could successfully carry [12]. Resistance training was then conducted five days per week for eight weeks, with MVCC reassessed every four weeks to adjust the training intensity.

The resistance training protocol involved climbing a 110 cm ladder inclined at 80°, with 26

steps spaced 2 cm apart. Rats climbed at 80% of their MVCC, performing 9-10 climbs per session, five days a week. To determine MVCC, a load equivalent to 75% of the rat's body weight was attached to its tail. For each successful climb, 30 grams were added to the load. A two-minute rest was given between climbs. This process continued until the rat successfully completed three consecutive full climbs [12].

Rats in the L-arginine supplement and combined groups received L-arginine supplementation (600 mg/kg) post-exercise, five days a week for eight weeks [13]. All animals were euthanized according to a predetermined protocol designed to minimize pain and distress. Forty-eight hours after the last training session, rats were anesthetized with an intraperitoneal injection of ketamine (90 mg/kg) and xylazine (10 mg/kg). Approximately 4-5 ml of blood was collected from the retro-orbital sinus using heparinized capillary tubes. The rats were then immediately euthanized and dissected by experienced personnel.

Gene expression levels were analyzed using RT-PCR, which involved RNA extraction, cDNA synthesis, and PCR with melting curve analysis to determine the threshold cycles for each gene. Relative gene expression was calculated using the 2^(-ΔΔCt) method, normalized to an internal control gene and the control group. Statistical hypotheses were tested using inferential methods at a significance level of $p < 0.05$. Normality and homogeneity of variance were assessed using the Shapiro–Wilk test. Data were analyzed by two-way ANOVA followed by Tukey's post hoc test to determine differences between groups. All analyses were performed using SPSS version 22.

Results

The results showed that the expression of the myonectin gene in the soleus muscle of young male rats was significantly higher in the exercise groups, both with and without L-arginine supplementation, compared to the non-exercise groups, regardless of supplementation. Conversely, the expression of the myostatin gene was significantly lower in the exercise groups compared to the non-exercise groups.

Table 1. Average of research variables

Measured indicators	Group	Number	Mean and standard deviation
Myonectin/actin gene expression	Control	8	2.0±20.23
	Supplement	8	2.0±10.32
	Exercise	8	6.0±32.12
	Exercise-supplement	8	6.0±12.13
Myostatin/actin gene expression	Control	8	4.0±56.32
	Supplement	8	4.1±48.88
	Exercise	8	2.0±12.47
	Exercise-supplement	8	2.0±20.51

No significant difference was observed between the supplement-only group and the control group, indicating that L-arginine supplementation without resistance training did not affect myonectin gene expression. However, when supplementation was combined with resistance training, the effect became significant.

Table 2. Results of two-way ANOVA test of myonectin and myostatin gene expression

index		Sum of squares	DF	Mean of squares	F	P	Squared Eta
Myonectin Gene Expression	Resistance training	8.62	1	6.65	4.20	0.215	0.93
	L-arginine supplementation	6.59	1	3.32	3.38	0.328	0.88
	Resistance training + supplementation	8.32	1	10.28	5.74	0.001*	0.90
	Error	32.20	30	1.32			
	Total	20.32	32				
Myostatin Gene Expression	Resistance training	25.10	1	25.10	5.82	0.321	0.107
	L-arginine supplementation	45.94	1	85.94	18.33	0.274	0.201
	Resistance training + supplementation	52.89	1	92.89	11.96	0.002*	0.901
	Error	59.26	30	6.34			
	Total	74.78	32				

Similarly, a significant difference was observed between the exercise groups with and without L-arginine supplementation. Therefore, resistance training, whether alone or combined with L-arginine supplementation, significantly increased the expression of both myonectin and myostatin genes in the soleus muscle of young male rats.

Table 3. Tukey's post-hoc test for different groups

Index	Group	Group	P
Myonectin Gene Expression	Exercise-supplement	Control	0.001*
		Supplement	0.001*
		Exercise	0.122
	Exercise	Control	0.001*
		Supplement	0.001*
	supplement	Control	0.258
Myostatin Gene Expression	Exercise-supplement	Control	0.002*
		Supplement	0.001*
		Exercise	0.621
	Exercise	Control	0.457
		Supplement	0.001*
	supplement	Control	0.231

The expression of the myonectin gene in the soleus muscle of young male rats was significantly higher in the exercise groups, both with and without L-arginine supplementation, compared to the untrained groups, regardless of supplementation. The results of the present study showed that myonectin gene expression in the soleus muscle of rats in the resistance training groups, with and without L-arginine supplementation, was higher than in the control and supplementation-only groups. There was no significant difference between the supplement and control groups. In fact, L-arginine supplementation alone, without resistance training, had no effect on myonectin gene expression. However, when supplementation was combined with resistance training, this effect became significant.

There was no significant difference between the two training groups, with and without L-arginine supplementation. Therefore, resistance training, regardless of L-arginine supplementation, resulted in a significant increase in myonectin gene expression in the soleus muscle of young male rats. Conversely, the expression of the myostatin gene in the soleus muscle was significantly lower in both exercise groups (with and without L-arginine supplementation) compared to the untrained groups, regardless of supplementation.

The results of this study showed that myostatin gene expression levels were lower in the resistance training groups, both with and without L-arginine supplementation, compared to the control and supplementation-only groups. There was no significant difference between the supplement and control groups, and in fact, L-arginine supplementation without resistance training had no effect on myonectin gene expression. However, when L-arginine supplementation was combined with resistance training, a significant effect on myonectin gene expression was observed. Similarly, resistance training, whether with or without L-arginine supplementation, significantly reduced myostatin gene expression levels in the soleus muscle of young male rats.

Discussion and Conclusion

Recent advances in sports science and exercise physiology have significantly enhanced our understanding of the physiological characteristics and performance capacities of athletes [14]. Exercise physiology explores how the body responds and adapts to various physical activities and environmental conditions. Athletes undergoing prolonged and intensive training often experience chronic fatigue, primarily due to reduced muscle fiber activity and depletion of glycogen stores [15]. This can result in energy deficits, muscle mass reduction, and increased psychological stress. Adequate recovery after training sessions is therefore crucial for restoring muscle energy and structure; insufficient rest may lead to a reduction in muscle fiber quantity [16].

The results of this study indicate that resistance training, with or without L-arginine supplementation, significantly reduced the expression of the myostatin gene in the soleus

muscle, while supplementation with L-arginine alone had no significant effect. This finding is consistent with the work of Ahnbach et al. (2018), who reported a significant negative correlation between myostatin gene expression and muscle glycogen content during exercise. Their study suggested that higher exercise intensity and greater glycogen depletion reduce myostatin expression and enhance β-catenin activity [17]. However, Amin et al. (2017) observed no significant changes in myostatin expression in the skeletal muscle of rats after short-term resistance exercise, suggesting that the response may depend on both the duration and modality of the exercise [17].

The reduction in myostatin expression observed with resistance training, independent of L-arginine supplementation, suggests that these interventions may regulate myostatin through distinct mechanisms. While tendon morphological adaptations following resistance training are well documented, the underlying molecular mechanisms remain incompletely understood. The TGF-β1 pathway is considered crucial for tendon adaptation, with myostatin, as a member of this family, playing a role in negatively regulating muscle growth and stimulating tendon fibroblast activity [18]. Although TGF-β1 and myostatin are known to enhance fibroblast proliferation and type I collagen synthesis in vitro, their specific roles in mature muscle adaptation remain unclear.

In this study, resistance training resulted in reduced basal myostatin mRNA levels in the soleus muscle. While most studies confirm a reduction in myostatin following exercise, research focusing on the combined effects of resistance training and L-arginine supplementation remains limited. One study found no significant change in myostatin mRNA in the gastrocnemius following short-term resistance training with L-arginine, suggesting that differences in exercise duration and protocol may account for these discrepancies [19].

Variation in myostatin response may also be related to muscle fiber type. Studies have shown higher myostatin protein concentrations in fast-twitch fibers compared to slow-twitch fibers, which could explain the substantial reduction (over 34%) in myostatin expression observed in the predominantly slow-twitch soleus muscle in this study [20-23].

Beyond its role in regulating muscle size, myostatin also influences the muscle and tendon extracellular matrix, particularly through the expression of type I collagen. Mendias et al. (2015) demonstrated that genetic inactivation of myostatin in rats increased muscle mass without negatively affecting tendon mechanical properties, suggesting that TGF-β1 signaling plays a more critical role in tendon adaptation to resistance training, with myostatin serving a secondary role [24]. It is important to note that tissue samples in this study were collected 48 hours after the final training session, meaning that earlier post-exercise changes in myostatin expression may not have been captured, which represents a limitation of the study. The present study also found that resistance training, with or without L-arginine supplementation, significantly increased myonectin gene expression in the soleus muscle, while supplementation alone had no significant effect. Other studies have reported increased myonectin expression following exercise. Although not all metabolic markers were assessed here, it is likely that resistance training may downregulate the Akt pathway, thereby increasing myonectin gene expression.

Insulin activates PI3K, which leads to the activation of Akt by kinases such as PDK-1, PDK-2, and ILK, subsequently inactivating GSK3-β. In the absence of insulin signaling, increased myonectin gene expression is associated with enhanced β-catenin phosphorylation [25].

Exercise increases metabolism, requiring enhanced oxygen delivery and waste removal, processes that depend on cardiovascular adaptations like angiogenesis and capillary expansion [26]. These vascular changes improve substrate exchange and endurance [9-27]. Additionally, vascular endothelial growth factor promotes glucose uptake, insulin sensitivity, and fat metabolism [8-12].

Amino acid availability, particularly essential amino acids, is critical for muscle repair following exercise, as the body cannot synthesize these amino acids and must obtain them from the diet [28]. The balance of energy substrates utilized during prolonged or intense exercise also influences muscle protein turnover [6, 9, 27]. L-arginine supplementation has been shown to improve strength, endurance, and recovery, particularly when combined with exercise [27, 29]. Arginine intake becomes increasingly

important with age to maintain hormonal balance and prevent degenerative changes [9]. As a nitric oxide precursor, L-arginine promotes vasodilation, enhances immune function, and regulates metabolism, potentially reducing exercise-induced oxidative stress [29-31].

In summary, eight weeks of resistance training, with or without L-arginine supplementation, resulted in significantly increased myonectin expression and decreased myostatin expression in the soleus muscle of young male rats. These molecular responses suggest an enhanced potential for muscle growth and maintenance, indicating that the combination of resistance exercise and L-arginine may be an effective strategy for stimulating muscle hypertrophy and preventing atrophy. Future studies should address the limitations discussed and further explore the underlying mechanisms.

Conclusion

Overall, it appears that two months of resistance training, with or without L-arginine supplementation, has a direct and significant effect on the expression of myonectin and myostatin genes. This creates a favorable environment for the survival of soleus muscle cells, promotes growth, prevents muscle atrophy, and may inhibit apoptosis. Furthermore, given the significant synergistic effect of combining resistance training with L-arginine supplementation on myonectin and myostatin gene expression in rats, it is likely that this combination enhances mitochondrial membrane integrity and reduces its susceptibility to apoptotic damage.

Declarations

Conflicts of Interest

The authors declare no conflict of interest.

Ethical Considerations

All procedures adhered to ethical guidelines outlined in the Helsinki Declaration and were approved by the Laboratory Animal Ethics Committee, ensuring the avoidance of unnecessary harm or distress to the animals.

Authors' Contributions

All authors contributed to the study conception and design.

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