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Effect of 12 Weeks Incremental Resistance Training on Serum Levels of Myostatin, Follistatin, and IGF-I in Sedentary Elderly Men

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ABSTRACT

Aims Numerous studies have established that resistance training is highly effective in preventing and addressing age-related muscle loss (sarcopenia) by enhancing the physiological function of skeletal muscle tissue. Thus, the objective of this study was to assess the impact of 12 weeks of incremental resistance training on the serum levels of myostatin, follistatin, and insulin-like growth factor-1 (IGF-I) in sedentary elderly men.

Materials & Methods Thirty sedentary elderly men voluntarily participated in this semi-experimental study and were randomly assigned to either a control group (15 men) with an average age of 62.1±3.7 years and weight of 85.1±7.7 kg, or a resistance training group (15 men) with an average age of 61.3±1.6 years and weight of 82.3±7.8 kg. The resistance training group undertook a 12-week training protocol, while the control group did not engage in any training program during this time. Blood samples and body composition measurements (using dual X-ray absorptiometry) were taken before the study commenced and 48 hours after the last training session concluded. Serum levels of myostatin, follistatin, and IGF-I were determined using the ELISA method. An independent t-test was employed to establish statistical significance between the groups, utilizing SPSS 21 software.

Findings After 12 weeks of resistance training, there was a significant decrease in serum myostatin levels and significant increases in serum follistatin and IGF-I levels in comparison to the control group ($p \leq 0.05$).

Conclusion Incremental resistance training proves to be an effective intervention for preventing sarcopenia in elderly individuals by decreasing serum levels of myostatin and increasing serum levels of follistatin and IGF-I.

Keywords Resistance Training; Myostatin; Follistatin; Insulin-Like Growth Factor I

CITATION LINKS

[1] Global prevalence of periodontal disease ... [2] Muscle disuse as a pivotal problem in sarcopenia-related ... [3] Epidemiology of muscle mass loss ... [4] Sarcopenia, dynapenia, and the impact of advancing ... [5] Sarcopenia: Origins and ... [6] Excitation-contraction coupling regulation in aging ... [7] Resistance training induced increase in muscle ... [8] Effects of exercise on muscle mass, strength, and physical ... [9] The age-related loss of skeletal muscle mass and function: ... [10] The central role of myostatin in skeletal muscle ... [11] Effects of upper-body, lower-body, or combined resistance ... [12] Regulation of skeletal muscle mass in mice by a new TGF-p ... [13] Human follistatin-related protein: A structural homologue of follistatin ... [14] The effect of resistance training in healthy adults on body fat percentage, ... [15] Exercise induces a marked increase in plasma follistatin: Evidence ... [16] Growth hormone(s), testosterone, insulin-like growth factors, and ... [17] Linkage of myostatin pathway genes with ... [18] The effects of muscle cell aging ... [19] The effect of resistance training and different sources of ... [20] Two weeks of reduced activity decreases leg lean mass and induces ... [21] Nutrition and physical activity for the prevention and ... [22] Resistance exercise-induced hormonal response promotes satellite ... [23] Effects of an alleged myostatin-binding supplement and heavy resistance training ... [24] A practical approach to strength ... [25] Exercise and physical activity ... [26] Regulation of muscle mass by growth ... [27] Aging of skeletal muscle: A 12-yr ... [28] Effects of progressive resistance training on cognition and IGF-1 levels in elder ... [29] Resistance training performed with single and multiple sets ... [30] Muscular strength adaptations and hormonal responses ... [31] The effect of 8 weeks resistance training with low load and high ... [32] Hormonal stress responses of growth hormone and ... [33] Selected contribution: Acute cellular and molecular responses ... [34] The effect of resistance training on serum insulin-like growth ... [35] Resistance training alters plasma myostatin but ... [36] Myostatin gene expression is reduced in humans ... [37] Effects of heavy resistance training on myostatin mRNA ... [38] Changes in lean mass and serum myostatin with habitual ... [39] The effects of concurrent training order on body ... [40] Analysis of the effects of androgens and training on myostatin propeptide ...

Introduction

Globally, the population of elderly individuals has significantly increased, rising from 382 million in 1980 to 962 million by 2017. Moreover, estimates suggest that this number will continue to grow, reaching 1.4 billion by 2030 [1]. Starting in their fifties, individuals begin to experience a gradual decline in lean mass, losing an average of approximately 1% per year [2, 3]. Longitudinal studies have shown that by around the age of 75, there is a noticeable reduction in muscle mass, with women experiencing a loss of about 0.64% to 0.7% per year, and men losing between 0.8% and 0.98% per year [4]. Consequently, by the age of 80, there is typically a substantial decrease of 30% to 50% in muscle mass [4]. The reduction in muscle mass and the accompanying weakness observed as individuals age is commonly referred to as "sarcopenia", a term initially coined by Rosenberg [5].

The onset of sarcopenia results in several changes within the muscles and nerves, including the loss of muscle mass, particularly in Type II muscle fibers, a decline in strength, an increase in fat mass, impaired coordination, elevated protein degradation (notably of contractile protein), and reduced satellite cell activity [6, 7]. The consequences of muscle atrophy and weakness lead to a variety of physiological and psychosocial effects, such as the inability to perform daily living tasks independently, increased frailty and fall risk, loss of independent living and associated depression/social isolation, a sedentary lifestyle due to physical inactivity, a heightened risk of chronic diseases, and an increased risk of all-cause mortality [8].

In recent years, extensive research has aimed to elucidate the cellular and molecular mechanisms underlying muscle hypertrophy and atrophy. The onset of age-related sarcopenia is believed to be due to one or more of the following factors: an increase in the basal-fasted rates of muscle protein breakdown, a reduction in basal muscle protein synthesis, or a combination of these factors [9].

Within the realm of signaling molecules, several myokines are recognized for their role in inhibiting the hypertrophic response in muscles [10]. A notable myokine in this context is myostatin (MSTN), identified as a key regulator of skeletal muscle mass. MSTN binds to Activin Type II receptors located in skeletal muscles, triggering the SMAD 2/3 intracellular pathway, which is responsible for inhibiting muscle growth [11]. In studies involving transgenic mice with a disrupted MSTN gene, a significant increase in muscle mass (2- to 3-fold) was observed without a corresponding rise in adipose tissue [12].

Follistatin (FLST) is another myokine that plays a critical role in muscle hypertrophy and atrophy. It functions as an antagonist to the MSTN receptor, exerting paracrine and autocrine effects [13]. By preventing MSTN from binding to its receptor, FLST decreases MSTN activity and facilitates an increase in muscle mass [14]. As a member of the TGF-β protein family, FLST is a glycoprotein that blocks MSTN's action by occupying its role [15].

Conversely, age-related changes in anabolic hormones and growth factors, such as IGF-1, are among the primary mechanisms influencing and exacerbating sarcopenia [16]. Consequently, levels of testosterone and insulin-like growth factor 1 (IGF-1) tend to decline, while catabolic factors, including cortisol and MSTN, often increase. Additionally, IGF-1 is a potent marker of satellite cell activity, leading to an increase in satellite cell numbers. It has been demonstrated that IGF-1 promotes the proliferation and differentiation of satellite cells by upregulating MSTN and downregulating P21 under various conditions [17]. Therefore, enhancing the expression of IGF-1 is considered crucial in the muscle hypertrophy process, particularly under mechanical loading [18].

Engaging in exercise training offers substantial benefits for older adults, including improved mobility and a lower risk of falls [19]. The considerable advantages of aerobic training in enhancing health, rehabilitation, well-being, and reducing cardiovascular disease risks among the elderly are well-documented [19]. Although aerobic exercise greatly benefits overall health, it does not directly contribute to an increase in muscle mass or musculoskeletal strength, both of which naturally decrease as part of the aging process. In contrast, resistance training (RT) uniquely addresses the age-related decline in muscle mass and strength, showing potential to reverse sarcopenia and, in some instances, prevent its onset [19]. Following a 2-week period of reduced physical activity, older adults exhibited anabolic resistance, characterized by diminished postprandial protein synthesis, decreased insulin sensitivity, and a reduction in leg muscle mass [20].

Despite the aging process, muscles maintain the ability to respond to increased activity levels, especially when subjected to RT. A meta-analysis in older adults has clearly shown the positive effects of incremental RT on muscle function. The improvements in muscle strength from RT are partly facilitated by proteins/hormones produced by skeletal muscle or associated tissues during exercise [21].

Therefore, while MSTN, FLST, and IGF-1 play essential roles in regulating skeletal muscle mass, the response of these growth factors to RT, particularly in the elderly, remains uncertain [22]. Previous studies have shown that three months of high-intensity RT leads to increased serum levels of FLST-like related gene [23]. In recent research, the FLST to MSTN (F:M) ratio has been identified as an important marker in assessing body composition and strength outcomes

following exercise training [11]. However, research on the response of myokines to exercise training is limited. For instance, a study on older males who underwent six weeks of high-intensity interval training reported no significant change in serum FLST concentrations [10].

Given the positive physiological outcomes observed in older adults through RT, it is clear that integrating such physical activities can help maintain or even increase muscle mass, improve muscle strength, and potentially reduce or prevent the onset of sarcopenia in this demographic. Nonetheless, the response of key factors in muscle hypertrophy and atrophy, such as IGF-1, MSTN, and FLST to resistance exercise, especially among the elderly, remains both sparse and inconsistent. Therefore, this study aimed to investigate the impact of incremental RT on the serum levels of IGF-I, MSTN, and FLST in sedentary elderly men.

Materials and Methods

Participants

This semi-experimental study, employing a pretest/posttest control group design, was carried out in the summer of 2022. The statistical research population included all employees (58 men aged 60 to 70 years) of the Razavi Khorasan Gas Company, Iran. From this population, 30 individuals were purposefully selected through available and voluntary sampling and were randomly divided into two groups, including experimental (15 men) and control (15 men). The sample size determination was estimated using G-Power software, setting the size of the intervention effect with 95% power at a significance level of 0.05 for 15 participants per group. Individuals with a history of cardiovascular and respiratory diseases (acute myocardial infarction, asthma), neurological disorders (stroke, paralysis, Parkinson's disease), spinal deformities, severe lower limb disabilities, skin diseases, smokers, those who were unmarried, and those regularly engaged in sports activities were excluded from the selection. Inclusion criteria included being at least 60 years old and sedentary, defined as not engaging in at least 30 minutes of moderate physical activity per day for three days a week.

Test Procedures

Body Composition Measurements

To evaluate body composition, the non-invasive and user-friendly electrical impedance analysis (BIA) method was utilized, employing the Olympia 3.3 Jawon device from Korea. This device provides an effective way to measure body composition with minimal intrusion. For the assessments, subjects in a fasting state and wearing minimal clothing had four electrodes attached to their body by the device. These electrodes were positioned under the feet and in the hands. After entering individual details, various parameters, such as total body water, intracellular water, extracellular water, fat percentage, and fat-free mass were measured. Like blood sampling, this test was carried out twice: once before starting the training protocol and once after its completion.

Determine One-Repetition Maximum (1-RM) at Each Station

Participants were asked to participate in a resistance exercise session to determine their 1-RM at each station. The measurement of one repetition maximum (1-RM) followed the protocol established by Brzycki *et al.* [24].

Table 1. Resistance training protocol

Exercise variables	Week 1-6			Week 7-12		
Weeks	1-2	3-4	5-6	7-8	9-10	11-12
Stations	8	8	10	10	12	12
Volume	1-2	1-2	2	2-3	3	3
Intensity	Voluntary	55% 1-RM	60% 1-RM	65% 1-RM	70% 1-RM	75% 1-RM
Repetitions	-	15-20	15	15	12	10
Frequency	2 training sessions/whole body	2 training sessions/whole body	3 training sessions/whole body	3 training sessions/whole body	2 training sessions upper body/2 training sessions lower body	2 training sessions upper body/2 training sessions lower body
Rest between each set	Voluntary	60-90 sec	90 sec	90 sec	90-120 sec	120 sec
Type	Familiarization	Endurance training	Muscular endurance training and hypertrophy	Muscular endurance training and hypertrophy	Hypertrophy and strength	Hypertrophy and strength
Exercise type	Body weight, pastural/stability, selective devices	Body weight, pastural/stability, selective devices	Pastural/stable, selective devices	Pastural/stability, selective devices, free weights	Selective machines, free weights	Selective machines, free weights

The procedure for determining 1-RM was as follows: To minimize the risk of injury, participants underwent both general and specific warm-up exercises before assessing 1-RM. The warm-up began with a general activity that involved 5 minutes of moderate-intensity jogging on a treadmill. This was followed by a specific warm-up, where participants performed 5 to 10 repetitions with a weight approximately 50% of their estimated maximum strength. After a one-minute rest with stretching

exercises, they then completed 3-5 repetitions with 60% to 80% of their estimated maximum strength. Following another 3 to 5 minutes of rest, the weight was incrementally increased until the final attempt to determine their 1-RM. If a participant successfully lifted the weight, it was increased for the next attempt. After a rest period of 3 to 5 minutes, the participant attempted the lift again. To avoid excessive fatigue, participants were advised to find their 1-RM within a maximum of five attempts. Adhering to the principle of progressive overload, 1-RM was reevaluated at each station during the 6th and 9th weeks to ensure gradual progression.

Training Intervention

The training protocol, which included training volume, intensity, frequency, and type per week, is detailed in Table 1. This protocol adheres to the recommendations provided by the American College of Sports Medicine for RT in the elderly population [25].

Blood Sampling and Analysis

The subjects attended an introductory session to become acquainted with the correct blood sampling procedure. To minimize potential interferences and confounding factors that could affect the research outcomes, and to limit the influence of food types on hormonal indicators, subjects were advised to avoid consuming fast food and beverages for at least 24 hours before blood sampling.

Blood sampling occurred in two stages: one day before the first training session (pre-test) and 24 hours after the final training session in the twelfth week. Both control and experimental groups were tested after fasting for 12-14 hours, specifically between 8-9 am. Before the initial blood sampling, subjects were instructed to avoid any strenuous physical activity for two days. Subsequently, 10 ml of blood was drawn from the brachial vein of the right arm while the subjects were seated and at rest. In the second stage, following the training period and a 48-hour break from the last session, blood collection was repeated under the same conditions as the first stage. After collection, the blood samples were centrifuged at 1500 rpm for 15 minutes. The separated serum was then stored at -20°C.

To measure concentrations of FLST and MSTN, enzyme immunoassay methods were used with ELISA kits from R and D Systems (Minneapolis, MN, USA). IGF-1 levels were determined using ELISA kits from Mediagnost (Reutlingen, BW, Germany). To ensure accuracy and consistency across the tests, coefficients of variance for both intra- and inter-assay measurements were calculated for all variables, consistently showing values below 10%.

Statistical Analysis

The data obtained were analyzed using SPSS version 20. After verifying the normality of data distribution with the Kolmogorov-Smirnov test and the homogeneity of variances with Levene's test, comparisons of means within and between groups were conducted using the paired sample t-test and ANCOVA test, respectively. A significance level of $p<0.05$ was established for all test results.

Findings

The Kolmogorov-Smirnov test results confirmed that the data for each variable were normally distributed. Table 2 lists the demographic characteristics, body composition, and biochemical variables of the subjects before and after the intervention.

Table 2. Characteristics and physiological variables measured pre-test and post-test in the experimental and control groups

Variable	Control group (n=15)		Training group (n=15)	
	Before intervention	After intervention	Before intervention	After intervention
Age (years)	62.1±3.7	.....	61.3±1.6	.....
Height (cm)	176±5.6	.....	175±7.3	.....
Weight (kg)	85.1±7.7	86.3±6.5	82.3±7.8	82.8±6.5
Fat mass (kg)	20.6±2.9	21.2±2.2	20.5±3.5	19.5±2.8
Fat-free mass (kg)	57.6±4.6	57.1±5.3	56.8±3.8	59.3±3.8

Values are expressed as mean±standard deviation.

Table 3. Mean MSTN, FLST and IGF-I levels in the pre-test and post-test

Variable	Groups	Pre test	Post test	Within-group		Between-group		
				t	p-value (paired t-test)	F	p-value (analysis of covariance)	Effect size
IGF-I (ng/ml)	Training	126.9±17.4	133.6±14.40*	-5.11	0.001	1.4	0.004	0.65
	Control	128.3±21.7	127.3±19.7	7.23	0.057			
Follistatin (ng/ml)	Training	11.75±1.8	15.88±2.6*	-3.89	0.004	3.1	0.001	0.26
	Control	12.31±3.13	12.12±2.8	-2.10	0.067			
Myostatin (ng/ml)	Training	19.59±5.06	15.92±4.11*	3.93	0.001	3.03	0.001	0.38
	Control	19.28±6.5	19.63±5.78	-7.11	0.075			

The independent t-test revealed no significant differences between the average values of age, weight, height, fat mass, and fat-free mass of the training and control groups prior to the intervention, indicating homogeneity in these characteristics between both groups, as depicted in Table 2. At the start of the intervention, no differences in the assessed variables were noted between the groups.

Table 3 showcases the outcomes of the independent t-test, highlighting the differences in the mean serum indices of the training and control groups before and after RT. Serum levels of IGF-1 and FLST experienced

a significant increase of 17% (p=0.004) and 20% (p=0.001), respectively, after 12 weeks of RT with progressively increasing intensity. In contrast, serum levels of MSTN saw a significant decrease of 14% (p=0.001). Notably, these changes were significant in comparison to the control group.

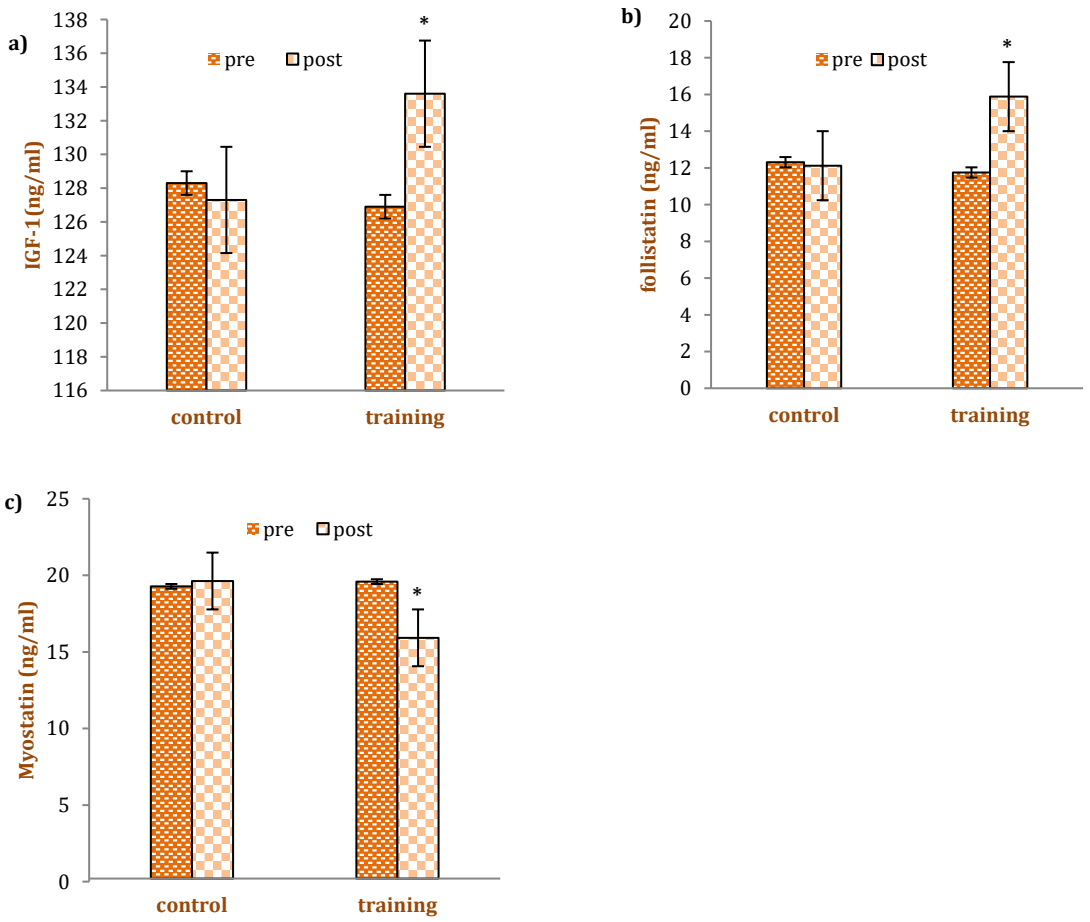

Figure 1. Mean IGF-I, a) Follistatin, b) Myostatin, c) levels in the pre-test and post-test in each group. The * denotes statistical significance (p<0.05) in respective pre- to post-comparisons.

Discussion

The main goal of this study was to assess the effects of a 12-week incremental RT program on the serum levels of MSTN, FLST, and IGF-1 in sedentary elderly men.

IGF-1, known for enhancing muscle development, acts as a positive regulator. Activation of the muscle IGF-1 receptor initiates a cascade of signaling pathways that lead to mitogenic and myogenic responses [26]. Increased levels of IGF-1 have been associated with numerous health benefits, including improved muscle, bone, tendon, body composition, and cognitive functions [16]. In the context of sarcopenia, IGF-1 plays a vital role in maintaining lean body mass through its effects on skeletal muscles [17]. As individuals age, they experience not only a decline in musculoskeletal system integrity, resulting in reduced muscle and bone mass but also a decrease in circulating IGF-1 levels [16]. The level of circulating IGF-1 can potentially increase the vulnerability of older adults to a higher risk of sarcopenia, functional decline, and loss of lean body mass [27].

The findings of this research demonstrated that 12 weeks of incremental RT significantly increased IGF-1 levels in sedentary elderly individuals (p=0.004). While some studies have observed chronic increases in circulating IGF-1 and muscle hypertrophy following resistance exercise training [28, 29], other studies have reported no change or even decreases in serum IGF-1 levels resulting from RT [30, 31]. For instance, the study by Rashidi *et al.* [31] involved an 8-week training protocol, whereas, in the current study, the participants engaged in RT for 12 weeks. This suggests that the length of the training period is crucial in determining the degree of increase in IGF-1 expression.

In some studies, the response of IGF-I to RT is characterized by two distinct phases. The first phase, which lasts about 5 to 6 weeks, is often termed the catabolic phase, during which serum IGF-I levels may remain unchanged or even decrease in response to

training. This phase is followed by an anabolic phase, beginning after 7 weeks or more, where serum IGF-I levels start to rise [32]. Therefore, the lack of changes in IGF-I observed in the study by Rashidi *et al.* underscores the two-stage adaptation process of IGF-I in response to exercise [31]. In essence, during the initial period, IGF-I is in a catabolic phase, playing a limited role in muscle adaptations during RT. Additionally, the study by Haddad *et al.* found that the expression of Mechano growth factor (MGF) precedes that of IGF-IEa in response to exercise [33], indicating that following mechanical strain and/or muscle damage, the IGF-I gene first shifts towards MGF and later towards IGF-IEa in rodent muscles. Jiang *et al.* further showed that an extended duration of RT is required to increase the expression of hepatic IGF-I [34].

Conversely, the lack of change in serum IGF-I concentration after RT, as noted in the study by Walker *et al.*, might relate to the initial IGF-I levels of the subjects in the current study [35]. Interestingly, our study's subjects had significantly lower initial IGF-I levels than those in Walker's study. Therefore, according to the research findings, numerous confounding factors could influence the observed results. These factors include inadequate dietary control, differences in subject characteristics, changes in plasma volume, dynamics of synthesis, release and receptor uptake, variations in exercise modes and intensities, limitations regarding the timing of post-exercise sampling, types of exercise protocols, study durations, training frequencies, sample sizes, and the age, sex, and baseline IGF-1 levels of participants.

MSTN acts as an inhibitor of growth and differentiation, specifically expressed in both developing and mature skeletal muscle tissues [11]. Disruption or mutation of the gene responsible for MSTN leads to a significant increase in skeletal muscle mass during development [12]. Additionally, serum MSTN levels are inversely related to total body muscle mass relative to height in older compared to younger adults, both males and females. Higher MSTN levels in the bloodstream are associated with muscle atrophy resulting from prolonged bed rest and thyroxine administration, underscoring its role in muscle changes during immobilization and hormonal interventions [11].

In this study, it was observed that sedentary elderly men exhibited a significant decrease in serum MSTN levels after participating in 12 weeks of incremental RT. Roth *et al.* were among the first to report a decrease in MSTN mRNA expression in both young and older individuals, including women and men, following 9 weeks of RT [36]. Conversely, Willoughby *et al.* noted an increase in MSTN mRNA expression, despite a rise in muscle mass, after 12 weeks of RT [37].

The inconsistency in findings could be due to differences in sampling times, methods, exercise intensity and duration, or methods of measuring MSTN. Bagheri *et al.* reported a significant decrease in serum MSTN concentrations in middle-aged men after 8 weeks of combined upper and lower body RT at 50 to 80% of 1-RM. However, serum MSTN concentration did not significantly decrease after 12 weeks of non-periodized upper and lower body RT at 85 to 90% of 1-RM in untrained males [11]. Additionally, Binns *et al.* observed a trend toward reduced serum MSTN concentration in community-dwelling older adults after 20 weeks of high-velocity whole-body RT at 70% of 1-RM [38].

The evidence suggests that exercise leads to decreased serum MSTN concentrations in middle-aged and elderly individuals, but not in young healthy men [11]. Although our data does not clarify the precise mechanism behind the reduction in plasma MSTN, the existing literature indicates that the decreased levels might be due to diminished production, processing, and/or secretion of the protein into the circulation. Alternatively, the observed lower MSTN levels could result from reduced stability, increased distribution in the circulatory system, enhanced disposal or reuptake of the protein, or a combination of these factors, potentially influenced by exercise training [11].

FLST is another myokine that plays a crucial role in muscle hypertrophy and atrophy, particularly in the elderly [13]. As a single-chain polypeptide, FLST belongs to the transforming growth factor (TGF) superfamily [14] and is ubiquitously expressed in various human tissues, including skeletal muscle, exerting both paracrine and autocrine effects [13]. Previous research has shown that FLST has the capacity to promote both anabolic and catabolic effects on skeletal muscle by binding to and neutralizing MSTN [14].

The results of this study reveal a significant increase in serum FLST levels among sedentary elderly men following 12 weeks of incremental RT. These findings are in line with those from other studies, which reported that serum FLST concentrations increased in sedentary females and middle-aged men after 8 weeks of high-intensity RT [11, 39]. An elevated FLST:MSTN ratio, indicative of a more favorable anabolic environment, resulted from decreased MSTN concentrations and increased FLST levels [11]. However, these results contrast with those of Diel *et al.*, which found no effect of 12 weeks of RT on serum FLST levels in participants [40].

It's crucial to consider the limitations and various factors that could account for these differing outcomes. The inconsistency may arise from differences in the age of the subjects and the training protocol intensities across studies, likely leading to the varied results reported in the literature. The consistent studies involved middle-aged and elderly participants, whereas Diel *et al.*'s study focused on young, healthy males. Additionally, it should be noted that the exercise protocol intensity in Diel *et al.*'s

study was lower compared to that used in the present study.

To summarize, a 12-week resistance exercise training program in elderly men led to significant changes: a 14% reduction in plasma MSTN, a 20% increase in plasma FLST, and a 17% increase in IGF-1.

Normally, the maintenance of muscle fiber size is dependent on a delicate balance between positive regulators (such as FLST and IGF-1) and negative regulators (such as MSTN) of muscle growth. However, in situations of muscle atrophy, such as that experienced by older individuals, this equilibrium is disrupted, leading to a dominance of negative influences. Consequently, the introduction of RT, which involves muscle loading, can potentially tip the balance in favor of positive regulators. While the exact mechanism of interaction between these regulators is still not fully understood, it seems to involve a complex negative feedback loop.

Conclusion

From the results of this study, it can be cautiously inferred that RT, through its dual role of enhancing muscle growth factors and reducing atrophy factors, has the potential to alleviate the effects of sarcopenia in elderly individuals. This, in turn, could address various sarcopenia-associated conditions, including metabolic syndrome, inflammation, glucose intolerance, reduced arterial elasticity, and obesity.

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