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RESEARCH ARTICLE

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The Effect of Arginine Supplementation on Growth Hormone Response Following Resistance Exercise in Overweight and Obese Women

Shilan Rashidzadeh[1], Hassan Faraji [1], Mohammad Rahman Rahimi [2]

[1] *Department of Physical Education and Sport Sciences, Mari.C., Islamic Azad University, Marivan, Iran.*

[2] *Department of Physical Education and Sport Sciences, Faculty of Humanities and Social Sciences, University of Kurdistan, Sanandaj, Kurdistan, Iran.*

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Abstract

Introduction: Our aim was to investigate the effect of arginine supplementation on growth hormone response following acute resistance exercise in overweight and obese women.

Methods: The present study was an experimental, crossover, double-blind, randomized, and controlled study with a pretest-posttest design. The statistical population included inactive overweight and obese women (25 to 35 years old) without underlying diseases. 10 subjects selected based on inclusion and exclusion criteria, after completing the informed consent form, were randomly divided into two groups: arginine supplement (10 grams) and placebo (maltodextrin). After physiological measurements including height (163.5±4.6 cm), weight (95.3±9.1 kg), fat percentage (41.3±4.2), body mass index (35.6±2.8 kg/m2) and determination of one repetition maximum, the subjects performed one session of resistance exercise (three sets at an intensity of 75% of one repetition maximum). Blood samples were collected 2 hours after breakfast (as a pre-test) and immediately after the end of resistance exercise (as a post-test).

Results: Changes in growth hormone were not significant due to time ($F=1.8$, $p=0.214$), group ($F=1.28$, $p=0.314$), and the time × group interaction ($F=1.63$, $p=0.107$). Also, insulin ($F=0.83$, $p=0.118$) and glucose ($F=0.93$, $p=0.352$) did not show significant changes ($p>0.05$).

Conclusions: Taking arginine supplements before resistance exercise did not have a significant effect on growth hormone, insulin, and glucose levels in overweight and obese women. Therefore, arginine supplementation in these conditions is unlikely to significantly stimulate growth hormone-related hormonal responses.

Key Words: promoting lipolysis, GH, weight training, precursor of nitric oxide

Corresponding author:

Assistant Professor of Exercise Physiology, Department of Physical Education and Sport Sciences, Mari.C. Islamic Azad University, Marivan, Iran, faraji.hassan@iau.ac.ir .0989188763846

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1. Introduction.

Obesity significantly increases the risk of cardiovascular, gastrointestinal, renal, and psychological diseases, as well as cancer, type 2 diabetes, metabolic syndrome, respiratory problems, musculoskeletal disorders, and fertility issues (1). One of the strategies for the prevention and control of obesity is increasing physical activity, particularly regular exercise. Physical activity can reduce fat accumulation by increasing the levels of lipolytic factors and facilitating the utilization of stored lipids by muscles.

Growth hormone (GH), also known as somatotropin, is lipolytic, meaning that it promotes the breakdown of stored triglycerides into free fatty acids (FFAs) and glycerol. GH is secreted throughout life, with the highest levels recorded during puberty (2). Growth hormone not only contributes to fat reduction but also plays a key role in the redistribution of body fat, particularly by reducing visceral fat, which is strongly associated with increased health risks (3). Although GH may not always result in substantial weight loss, it can improve body composition by increasing lean body mass and reducing fat mass (4). Growth hormone stimulates protein synthesis and muscle growth, which can help maintain or increase lean body mass during weight loss and prevent muscle catabolism (5). GH also influences glucose metabolism. Activation of the growth hormone receptor generates signals that reduce glucose uptake into cells, particularly in the liver and adipose tissue, leading to increased blood glucose levels (6). Growth hormone reduces cellular glucose utilization and elevates blood glucose levels, which may contribute to insulin resistance. This condition is common in obesity and can predispose individuals to type 2 diabetes (7). These processes are considered among the mechanisms involved in the development of insulin resistance.

Research has shown that exercise stimuli significantly increase circulating growth hormone concentrations across all age groups, even in individuals who experience age-related declines in GH secretion (8, 9). GH secretion begins to increase approximately 10–20 minutes after the onset of acute exercise (10). The magnitude of this increase depends on the intensity and type of exercise, with higher intensities leading to greater stimulation of GH release (11). Both resistance and endurance exercises stimulate GH secretion; however, high-intensity resistance exercise generally induces a more acute increase in GH compared with endurance exercise (12). Nevertheless, it has been shown that in individuals with obesity, each unit increase in body mass index (BMI) is associated with an approximately 6% reduction in the normal GH secretory response to physiological stimuli (13). Under such conditions, investigating nutritional factors such as dietary supplements that may influence GH responses to resistance exercise appears necessary.

Arginine supplementation, whose chemical structure consists of a three-carbon aliphatic chain terminating in a guanidinium group, is primarily synthesized in the human body by the kidneys and the small intestine, which are considered the main sites of net arginine production (14). As a dietary supplement, arginine exerts several physiological effects. Its most well-known role is as a precursor of nitric oxide (NO), thereby improving blood flow and oxygen delivery to various tissues. This function underlies many of its reported benefits, including enhanced exercise performance, reduced blood pressure, and improvement of symptoms in vascular diseases such as angina pectoris and peripheral arterial disease. Arginine also plays roles in protein synthesis, immune system function, and growth hormone secretion (15). Although studies examining specific GH responses to exercise combined with arginine supplementation in obese individuals remain limited, meta-analyses suggest that chronic oral arginine intake in overweight or obese individuals can improve body composition. These improvements include reductions in body mass index (BMI), waist circumference, and fat mass, along with increases in lean body mass (muscle) over time. However, overall body weight typically remains unchanged (16).

At rest, oral L-arginine consumption at doses of 5–9 g can approximately double resting GH levels (about a 100% increase), whereas acute resistance exercise alone usually induces a much greater increase in GH levels (approximately 300–500%) (17). However, when arginine intake is combined with acute resistance exercise in normal individuals, the GH increase may be reduced compared with exercise alone (approximately a 200% increase). One study reported that exercise alone produced a greater

GH area under the curve (about 508 ng/mL), whereas arginine plus exercise resulted in approximately 260 ng/mL, which was not significantly different from arginine alone (18).
In obese individuals, evidence suggests that the GH response to arginine and exercise may differ. To the best of our knowledge, no study has specifically investigated GH responses to arginine supplementation combined with acute exercise in obese individuals. However, the well-documented GH resistance observed in obesity suggests that the typical increase in GH following arginine intake and resistance exercise may not occur fully or may be substantially attenuated in obese individuals (19). Therefore, the aim of the present study was to examine the effect of acute arginine supplementation on growth hormone levels following an acute bout of resistance exercise in overweight and obese women.

2. Study design

2.1. Research Participants

The statistical population of the present study consisted of inactive overweight and obese women aged 25–35 years. Physical inactivity was defined as not meeting the recommended levels of physical activity, namely at least 150–300 minutes of moderate-intensity aerobic activity, 75–150 minutes of vigorous-intensity aerobic activity, or an equivalent combination of moderate and vigorous activities per week (20). Participants were also required to be free from diseases (e.g., cardiovascular, neuromuscular disorders, diabetes, etc.) and to have no limitations for performing physical or exercise activities according to the Physical Activity Readiness Questionnaire (PAR-Q) and their medical history (Table 1) .
Because the menstrual cycle may influence growth hormone responses following arginine intake (21) or acute resistance exercise (22), all participants were selected during the early follicular phase of their menstrual cycle (days 1–10). The minimum required sample size was estimated to be nine participants per group based on previous studies (18, 19, 23) and the Borg and Gall formula. Among 21 eligible volunteers who met the inclusion criteria, 10 participants were ultimately selected through simple random sampling .
The inclusion criteria were: absence or history of any disease, no movement or exercise limitations, no use of sports supplements or participation in regular exercise during the six months prior to the study, no tobacco addiction, a body mass index (BMI) greater than 30, and an age range of 25–35 years.

2.2. Research Procedure

The present study employed an experimental, randomized, double-blind, crossover, placebo-controlled design with a pretest–posttest approach. The study protocol was approved and registered with the ethics code IR.IAU.SDJ.REC.1401.090. Participants were randomly assigned into two equal groups: supplement and placebo. All participants were informed about the study procedures, potential benefits, and possible risks, and subsequently signed an informed consent form prior to participation.
During familiarization sessions, physiological measurements (height, weight, and body fat percentage) and performance measures were obtained. One-repetition maximum (1RM) was estimated using the Brzycki formula in the following exercise stations: squat, knee extension, knee flexion, leg press, chest press, lat pulldown machine, shoulder press, biceps curl, and triceps extension (24). Body fat percentage was measured using a bioelectrical impedance analyzer (Gaia 359 Plus, South Korea).
Participants then attended a testing session that included supplement consumption and an acute resistance exercise protocol. After a 14-day washout period (25), the testing session was repeated under the same conditions, with the supplement and placebo groups switched.
On the testing day, participants performed one session of resistance exercise consisting of three sets at each exercise station at an intensity of 75% of 1RM. Rest intervals were set at 60 seconds between sets and 120 seconds between stations (26). Participants were encouraged to perform up to 10 repetitions per set.
Participants in the supplement group consumed 10 g of arginine dissolved in 250 mL of water 60 minutes before the start of the exercise session (27), while the placebo group simultaneously received an equivalent amount of maltodextrin. To improve taste and ensure uniformity, 3 mL of natural lemon juice was added to each drink in both the supplement and placebo groups.
Dietary intake during the 24 hours prior to the first testing session was recorded using a dietary recall form, and participants were instructed to consume the same foods during the 24 hours preceding the subsequent session. They were also asked to refrain from any moderate-to-vigorous physical activity (Borg scale score >13) and from using any medication or dietary supplements during the study period.

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On each testing day, participants arrived at the gym at 8:00 a.m. after at least 10 hours of overnight fasting. They consumed a standardized breakfast within 15 minutes. The standardized breakfast provided 4 kcal per kilogram of body weight (28) and consisted of lavash bread (approximately 70 g), cheese (approximately 15 g), and vegetable butter (approximately 8 g), providing an average of 294.56 ± 56.84 kcal with a macronutrient distribution of 45–65% carbohydrates, 10–35% protein, and 20–35% fat. After breakfast, participants were allowed to drink only water.

Blood samples (approximately 7 mL) were collected from the antecubital vein of the right arm two hours after breakfast (pretest) and immediately after the resistance exercise session (posttest).

Laboratory Analysis

Blood samples were centrifuged at 3000 rpm for 15 minutes to separate serum and were then stored at −20 °C for subsequent analyses. Biochemical analyses and measurements of growth hormone concentrations were performed using the enzyme-linked immunosorbent assay (ELISA) method with a ZellBio kit (Italy), with an accuracy of 0.02 ng/L and a sensitivity of 0.01. Blood insulin levels were measured using the ELISA method with a human Monobind kit (Iran). Glucose levels were assessed using an automated biochemical analyzer based on enzymatic colorimetric reactions.

2.3. Statistical analysis

The Shapiro–Wilk test and Levene's test were used to assess the normality of data distribution and the homogeneity of variances, respectively. A two-way (2×2) repeated-measures analysis of variance (ANOVA) was applied to examine the main effects of time, group, and the interaction between time and group. All statistical analyses were performed using SPSS software (version 21), and the level of statistical significance was set at $p < 0.05$.

3. Results

The data were normally distributed and exhibited homogeneity of variances. The levels of growth hormone, insulin, and glucose are presented in Figures 1–3.

Analysis of growth hormone levels showed that in the supplement group, the effects of time ($F = 1.8$, $p = .214$, $\eta^2 = .18$), group ($F = 1.28$, $p = .314$, $\eta^2 = .19$), and the interaction between time and group ($F = 1.63$, $p = .107$, $\eta^2 = .22$) were not statistically significant. Similarly, in the placebo group, the effects of time ($F = 0.93$, $p = .227$, $\eta^2 = .09$), group ($F = 0.82$, $p = .361$, $\eta^2 = .14$), and the interaction between time and group ($F = 1.14$, $p = .150$, $\eta^2 = .20$) were also not significant.

The analysis of insulin levels indicated that in the supplement group, the effects of time ($F = 0.83$, $p = .118$, $\eta^2 = .10$), group ($F = 1.73$, $p = .296$, $\eta^2 = .13$), and the interaction between time and group ($F = 1.36$, $p = .147$, $\eta^2 = .14$) were not statistically significant. Likewise, in the placebo group, the effects of time ($F = 0.86$, $p = .411$, $\eta^2 = .08$), group ($F = 1.22$, $p = .289$, $\eta^2 = .09$), and the interaction between time and group ($F = 1.19$, $p = .207$, $\eta^2 = .17$) were not significant.

Furthermore, changes in glucose levels were not statistically significant. In the supplement group, the effects of time ($F = 0.93$, $p = .352$, $\eta^2 = .07$), group ($F = 2.1$, $p = .138$, $\eta^2 = .19$), and the interaction between time and group ($F = 1.52$, $p = .145$, $\eta^2 = .11$) were not significant. Similarly, in the placebo group, the effects of time ($F = 0.96$, $p = .208$, $\eta^2 = .79$), group ($F = 2.01$, $p = .254$, $\eta^2 = .11$), and the interaction between time and group ($F = 1.9$, $p = .118$, $\eta^2 = .19$) were not statistically significant.

Table 1 Physiological characteristics of the subjects.

Variable	mean ± SD
Age (years)	33.8 ± 9.3
Height (cm)	163.5 ± 4.6
Weight (kg)	95.3 ± 9.1
BMI (kg/m^2)	35.6 ± 2.8
Body fat (%)	41.3 ± 4.2

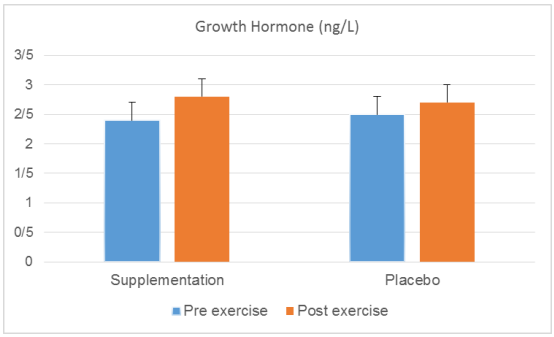

Figure 1: Mean growth hormone levels in different groups

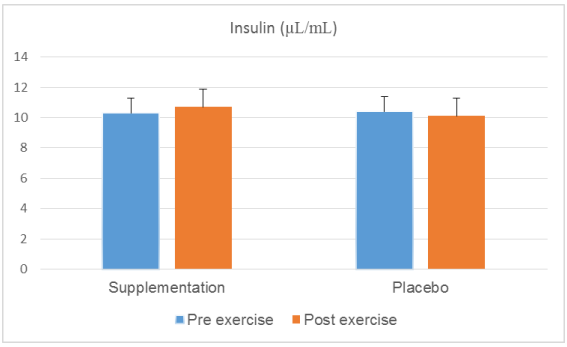

Figure 2: Mean insulin levels in different groups

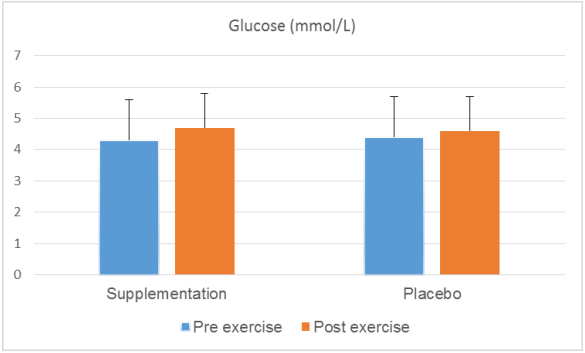

Figure 3: Mean glucose levels in different groups

4. Discussion

The main finding of the present study showed that acute oral arginine supplementation (10 g), consumed 60 minutes before a resistance exercise session performed at an intensity of 75% of one-repetition maximum (1RM), did not significantly increase the growth hormone response in overweight and obese women compared with the placebo group (maltodextrin). Moreover, no significant time × group interaction was observed. This finding confirms that oral arginine does not stimulate growth hormone secretion under acute conditions in this specific population. It has been established that high-intensity resistance exercise leads to a significant increase in plasma growth hormone concentrations (29). Studies have shown that, in addition to

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stimulating muscle protein synthesis, increased growth hormone levels may enhance lipolysis in subcutaneous adipose tissue and reduce visceral fat tissue (30).

Overweight and obese individuals exhibit growth hormone resistance, which leads to impaired secretion and action of this hormone. This resistance involves reduced pituitary growth hormone secretion, decreased sensitivity of cellular receptors, and increased anti-growth-hormone effects mediated by inhibitory factors such as free fatty acids and somatostatin (16, 31). Elevated free fatty acids in obesity increase somatostatin activity and reduce the growth hormone secretory response to stimuli. In addition, visceral fat is responsible for the secretion of inflammatory cytokines, which disrupt the hypothalamic–pituitary axis and reduce growth hormone secretion (31).

Although growth hormone increases in response to high-intensity resistance exercise, this increase has been reported to be attenuated in obese individuals (32). Accordingly, we sought to investigate factors such as supplementation that might prevent this attenuation and thereby promote the anabolic and lipolytic effects associated with increased growth hormone. Exogenous arginine administration, particularly when delivered as an intravenous bolus, elicits a pronounced growth hormone response (23). However, few studies have examined the effects of oral arginine on growth hormone concentrations, and no study has established a relationship between oral arginine intake and the growth hormone response in overweight and obese individuals. The present study examined the effect of acute arginine intake on the growth hormone response to resistance exercise in overweight and obese women. The results showed that consuming arginine 60 minutes before resistance exercise at 75% of 1RM did not produce a significant increase in growth hormone compared with placebo, and no significant time × group interaction was observed. In addition, changes in insulin and glucose were not significant.

Our findings regarding the lack of stimulation of growth hormone or insulin by arginine are consistent with several previous studies. For example, da Silva et al. (2014) found that arginine supplementation did not stimulate the production of growth hormone, insulin, or IGF-1 and therefore had no effect on hormonal responses in their study population (33). Similarly, Nieves et al. (2022) reported that 6 g/day of arginine for four weeks in physically active participants did not alter hormonal or metabolic parameters beyond the changes induced by exercise alone (34). Gannon et al. (2002) also observed that orally ingested arginine, unlike intravenous administration, did not stimulate insulin secretion (35). An animal study conducted in 2022 examining arginine supplementation showed a significant increase in pituitary growth hormone gene expression and related reproductive hormones at higher doses of arginine. This suggests that arginine may modulate growth hormone at the transcriptional level; however, this may not always translate into detectable increases in circulating plasma growth hormone, particularly under acute conditions or at lower doses. The authors suggested that this finding reflects complex regulatory mechanisms and dose dependency in the effect of arginine on growth hormone (36).

However, our finding is not consistent with some studies that have reported the efficacy of arginine. In this regard, one notable study showed that oral arginine (7 g) alone could produce a moderate increase in growth hormone; however, when combined with resistance exercise, the growth hormone response was actually reduced compared with exercise alone. This may reflect negative autofeedback mechanisms that limit growth hormone secretion during simultaneous stimuli (18). A comprehensive review in 2022 emphasized that the effectiveness of arginine supplementation depends on dose, timing, and combination with other secretagogues such as growth hormone-releasing hormone (GHRH), while isolated arginine alone has shown variable effects on growth hormone secretion across studies. The acute effects of arginine on prolactin and insulin are generally absent or minimal in healthy adults (37). Another study in 2020 reported a moderate increase in growth hormone following acute arginine supplementation, but no significant effects on prolactin or insulin. This supports the conclusion that arginine may selectively affect the growth hormone axis without causing broad short-term changes in other pituitary hormones or metabolic markers (38).

Nevertheless, a systematic review and meta-analysis by Ghigo et al. (2022) showed that arginine alone had a significant effect on growth hormone release, with a mean difference of approximately 10 ng/mL, and that even larger effects were observed when arginine was combined with GHRH (37). For example, GHRH plus arginine, or arginine combined with other amino acids and sometimes exercise, tends to elicit larger growth hormone responses. Arginine alone appears to be less reliable, a conclusion supported by meta-analytic evidence (37).

Mechanistically, the lack of a significant effect of arginine on circulating growth hormone levels may be explained by its role in inhibiting somatostatin release, which may increase growth hormone pulse frequency or amplitude without producing a clearly detectable increase in basal growth hormone concentrations during the study period. In addition, arginine-induced nitric oxide (NO) production may affect hypothalamic–pituitary signaling; however, downstream hormonal responses are governed by

feedback loops and individual physiological variability (36). Obese individuals characteristically show reduced growth hormone secretion in response to stimuli such as exercise or GHRH, which has been attributed to impaired pituitary growth hormone reserve and altered hypothalamic regulation. Studies have shown that although arginine enhances growth hormone secretion by inhibiting somatostatin, the growth hormone response in obese individuals is not only reduced compared with lean individuals but also shows a greater time delay. This finding has been observed even when arginine is combined with GHRH or acute exercise (39).

Regarding insulin and glucose, the well-documented insulinotropic effect of arginine usually requires specific glycemic challenges, which may not be present under fasting or resting conditions; this may explain the non-significant changes observed in the present study. Moreover, arginine may stimulate glucagon release, balance glucose homeostasis, and prevent large fluctuations after supplementation (37). Miekus et al. (2015) showed that although long-term L-arginine supplementation improved insulin sensitivity in mice fed a high-fat diet, acute or short-term effects on insulin and glucose were often small or non-significant in the absence of dietary or exercise interventions (40). Mousavi et al. (2021), in a meta-analysis, found that short-term arginine supplementation did not significantly reduce body weight or BMI, factors closely related to insulin and glucose metabolism. This supports the idea that metabolic improvements require longer intervention periods (41). Forzano et al. (2023), in a clinical review, concluded that acute arginine intake has limited effects on fasting blood glucose and insulin levels. Instead, long-term supplementation appears necessary to produce clinically meaningful effects on glycemic control (42). Salgueiro et al. (2017) found that chronic arginine supplementation may induce insulin resistance when used alone, whereas exercise training reverses these effects, highlighting the complex interactions between arginine metabolism and insulin function in obese individuals (43).

In obese populations, because of complex metabolic adaptations, a single dose of arginine is often insufficient to induce immediate and measurable changes in insulin or glucose levels (44). Arginine is known as the main precursor of NO, a compound responsible for vasodilatory effects and improved insulin sensitivity. However, in obesity, NO production and bioavailability may be impaired, reducing the acute efficacy of arginine in improving insulin function. Furthermore, the effect of arginine on insulin signaling pathways, particularly PI3K/Akt, may involve time-dependent changes in gene and protein expression that are not detectable under acute conditions (45). Resistance exercise alone can temporarily improve insulin sensitivity, but adding arginine does not necessarily produce additive effects in the short term. Some studies suggest overlapping pathways, such as NO signaling and GLUT4 translocation, through which arginine supplementation may not substantially enhance the acute effects of exercise on insulin and glucose (46).

One of the major limitations of the present study was the small sample size (10 participants in each group), which may have limited the statistical power to detect significant differences. In addition, participants included only women aged 25–35 years with a BMI greater than 30, which limits the generalizability of our conclusions to this specific population. Another limitation was the lack of assessment of other hormones such as IGF-1 and metabolic regulatory hormones. Future studies are recommended to include larger samples, more diverse age and sex groups, longer intervention periods, and more precise evaluation of the GH–IGF-1 axis and other hormones involved in this physiological pathway. Moreover, investigating the molecular mechanisms underlying growth hormone resistance in obese individuals may help improve understanding of this issue.

5. Conclusion

Considering the present findings and their comparison with the existing scientific literature, it can be concluded that acute consumption of 10 g of arginine 60 minutes prior to resistance exercise does not lead to a significant increase in growth hormone levels in overweight and obese women. Furthermore, the exercise-induced growth hormone response appears to be attenuated in this population due to the presence of physiological growth hormone resistance. In addition, changes in insulin and glucose levels were not significant following arginine supplementation and resistance exercise under the conditions of the present study. These results highlight the complexity of growth hormone regulation in obesity and indicate the need for more specific strategies to improve this hormonal response.

6. Highlights

Taking 10 grams of oral arginine 60 minutes before a resistance workout does not increase growth hormone levels in women who are overweight or obese. Arginine supplementation does not provide additional benefits for managing blood glucose or insulin levels in this population when combined with a single resistance exercise session.

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Coaches and practitioners should be aware that acute arginine supplementation may be ineffective for boosting hormonal responses in individuals with obesity, likely due to existing physiological resistance.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

The first author was responsible for the study design, practical implementation of the research protocol, participant coordination, data collection, and drafting the initial version of the manuscript. The second author supervised the implementation of the study, contributed to data analysis and interpretation of the results, and reviewed the manuscript critically. The third author supervised the execution of the research protocol, assisted in manuscript preparation, and contributed to the revision and finalization of the article. All authors read and approved the final version of the manuscript.

Supplementary Materials

Supplementary materials are not available for this study.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board/Ethics Committee of Islamic Azad University, Sanandaj Branch, Iran, with the ethics approval code IR.IAU.SDJ.REC.1401.090.

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