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Progressive circuit resistance training improves inflammatory biomarkers and insulin resistance in obese men

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Abstract

Background: Circuit resistance training (CRT) is a time-efficient exercise modality for improving skeletal muscle and cardiovascular fitness. But the beneficial role of CRT in obese individuals is still not well understood. This study explores the reducing effects of progressive CRT on inflammatory biomarkers and cardiometabolic risk factors in obese young men. Methods: Thirty obese men (Body mass index (BMI): 30.67±3.06; age: 23±3.2 years) were divided into CRT and control groups. The CRT was performed for eight-weeks (3 times/week, 65-85% of 1 repetition maximum). Fasting blood samples were taken pre and post intervention for analyzing apelin, chemerin, serum amyloid A (SAA), C reactive protein concentrations (CRP), lipid profile, and insulin resistance index. The data were assessed by two-way repeated measures ANOVA. Results: Body mass, BMI and waist to hip ratio (WHR) were significantly decreased after training intervention ($P<.05$). Compared to the control group, the plasma concentrations of Chemrin ($P=.038$), SAA ($P=.004$), insulin ($P<.001$), insulin resistance index ($P<.001$), total cholesterol ($P=.033$), triglyceride ($P<.001$), and low-density lipoprotein ($P=.039$), were significantly mitigated in the CRT group, but high-density lipoprotein plasma levels increased in the CRT group compared to that of the control group ($P=.035$). There was no significant difference between two groups in apelin and CRP ($P>.05$). Moreover, insulin resistance was positively correlated with apelin ($r=.56$) and chemerin ($r=.51$). Also, chemerin had a positive correlation with SAA ($r=.49$), and WHR ($r=.54$). Conclusion: CRT caused an improvement in inflammation and cardiometabolic risk factors in young obese men, and this improvement was accompanied by decreased insulin resistance.

Keywords: Adipokine; Inflammation; Obesity; Exercise training.

1. Introduction

Obesity, a worldwide epidemic, is increasing throughout the world. It is accompanied by numerous health risks including cardiovascular diseases, hypertension, and Type 2 diabetes mellitus [1]. Adipose tissue, which plays a critical role in the storage of excess energy intake [2], is involved in obesity related health risk factors. Obesity is characterized by increases in the number and the size of adipocytes, as well as the lipid storage in mature adipocytes [3]. Adipose tissue produces a variety of cytokines or adipokines that are involved in the regulation of energy homeostasis [2, 3]. It has been suggested that any alteration in adipokine secretion mediates inflammation and cardiometabolic diseases associated with obesity [4].

Apelin plasma levels that play a substantial role in the regulation of cardiovascular and metabolic homeostasis, such as cardiovascular regulation and some anti-inflammatory properties, are produced principally by white adipose tissues [5]. Apelin influences glucose and insulin homeostasis and may be involved in the adipose tissue dysfunction in obesity [6]. Moreover, apelin plasma levels increase during obesity, and they are positively correlated with the body mass index (BMI) [3, 5-7].

Like apelin, chemerin is predominantly secreted by adipose tissues that play an important role in the differentiation and enhancement of glucose metabolism in adipocytes [8]. Some studies have shown that plasma concentrations of chemerin are positively associated with BMI and can rise in patients with abnormal blood glucose, high-density lipoprotein (HDL), triglycerides, and hypertension [8, 9]. The knockdown of chemerin resulted in a suppression of basal lipolysis and glucose uptake in adipocytes [10]. Obesity-related inflammation is characterized by an increase in lipid profile (especially low-density lipoprotein (LDL) and triglycerides) [11]. It has been suggested that acute phase inflammatory proteins, such as serum amyloid A (SAA) may affect the lipids and the apo-lipoproteins metabolism during inflammation [11]. Adipose tissue is the main source of SAA, a pro-inflammatory cytokine, that increases with obesity [12]. Likewise, another biomarker of low-grade inflammation, high sensitive C-reactive protein (hs-CRP), increases with obesity [13]. These inflammatory proteins, which have a positive correlation with the waist to hip ratio (WHR) and subcutaneous adipose tissue, reduce after weight loss [14].

Studies have revealed that regular exercise training plays a critical role in the prevention and treatment of chronic diseases such as obesity, Type 2 diabetes mellitus, and long-term cardiovascular complications [15, 16]. Regular exercise training also reduces levels of systemic concentration of pro-inflammatory cytokines [17, 18]. Among various modes of exercise training, circuit resistance training (CRT) improves the cardiorespiratory function, muscular strength, endurance, and power [19]. This is a type of combination training that incorporates both multi-joint resistance training and calisthenics exercises; it keeps the heart rate elevated during a training session. In addition, the metabolic cost of these types of exercise training is higher compared to traditional resistance training [20], even in compared to the combination of traditional resistance training and aerobic training [21]. During CRT, the individual moves from one exercise to another as quickly as possible with very little rest between exercises, which results in a short exercise session time. Progressive CRT is a unique mode of exercise that can reduce adiposity while increasing the skeletal muscle mass at the same time [21, 22]. Hence, it has also been shown the association between body composition changes and inflammation [23].

Most of the studies examined the effect of aerobic training and traditional resistance training on adipokines and circulating inflammatory indicators. However, with regard to superior advantages of CRT to traditional resistance and aerobic training [21], the influence of CRT on circulating adipokines related to cardiometabolic risk factors and inflammatory biomarkers in obese individuals is not yet fully understood. Therefore, the purpose of this study was twofold: first, to investigate the effects of eight weeks of CRT on acute phase protein inflammatory markers levels and body composition, and second, to examine the effects of CRT on cardiometabolic risk factors and insulin resistance in inactive obese young men. Our hypothesis was that the CRT, as a time efficient training strategy, can alleviate cardiometabolic risk factors and inflammation associated with obesity.

2. Materials and Methods

2.1. Participants and study design

Participants were healthy, obese (BMI ≥30 kg/m^2) men (Table 1). The participants were recruited after an announcement in a poster. The inclusion criteria were: men aged 20–30 years, inactive (no resistance training or aerobic training in the past six months), BMI (in kg/m^2) from 30 to 40, non-smoking and stable weight (< 5% weight change in the past six months). The exclusion criteria were: diabetes or evidence of clinical depression, cognitive disorder, heart disease, cancer, liver or renal disease, chronic pulmonary disease, uncontrolled hypertension, physical disability, or any contraindication for physical activities or weight loss (e.g. osteoporosis). Finally, after screening, 30 participants were selected out of a total of 100 men. Next, the participants were randomized into two equal groups of 15 people: the CRT group and the control group (Table 1). Following the full description of all risks, all participants gave their informed consent before participating in this study. All procedures performed in studies involving human participants were approved by the ethical committee of Islamic Azad university of Tehran (#1247/6) in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The control group was only assessed in the pre-test and post-test periods and did not do any regular exercise training during the period of study. They did not perform any heavy physical activity. The study lasted for a period of eight weeks. The described variables were evaluated twice—once at the beginning and once at the end of the eight-week period. One session was dedicated to explain the experiment phases relating to the participants. The participants were asked to maintain their eating patterns throughout the study. The dietary consumption was recorded during the intervention. Additional weight control diet instructions were not provided throughout the study and the subjects were asked not to alter their dietary habits. The ingredients and amounts of calories received on the days before blood sampling were the same. During the study, two participants in the exercise group and two participants in the control group were dropped out from continuing the experiment (for consuming medication or being absent for more than 1 sessions).

2.2. Training program

All participants attended the gym before the start of the eight-week intervention. The participants attended the gym for one session to get familiarized with the exercise equipment and the correct technique for all the exercises. The CRT involved an eight-week training program that the participants had to follow three times a week. The duration of each exercise session was approximately 60 minutes (Including a 15-minute warm-up period, and a 10-minute cool down period). In the first four weeks, the training intensity was 65%–75% of one repetition maximum (1-RM), and the subjects had to complete two circuits per session, with eight to 12 repetitions in each exercise. In the second four weeks, the training intensity was 75%–85% of 1-RM, and the participants had to complete four circuits per session, with six to eight repetitions in each exercise (Table 2). The rest between stations was the minimum time required for the subjects to move from one station to another (<15 seconds), and the rest between circuits was three minutes of active recovery. The training protocol consisted of the following eight exercises: squat, standing curls, bench press, leg extension, leg flexion, leg press, military press, and lat pull down. A 1-RM was calculated using the Brzycki regression equation, where 1 RM = weight lifted during an RM/(1.0278-.0278 (n) at the beginning of the study and each exercise block (week 1, 3, 6) as a measure of strength. To avoid injury, all exercises were performed on a strength training machine instead of free weight.

2.3. Measurement of anthropometric and Body Composition

BMI was calculated as weight in kilogram divided by height in meters squared. Waist-to-hip ratio (WHR) was calculated as Waist circumference divided by hip circumference.

2.4. Blood Sampling and Analysis

Blood samples were collected before and after the 8-week intervention program (24 hours before the first bout of exercise and 48 hours after the last bout of exercise) were taken from the antecubital vein. Blood was collected in the morning between 8 and 9 a.m. after an overnight fast. Blood was drawn from a forearm vein into a sterile tube containing EDTA (Klap activator model). Blood was centrifuged for 10 min at 1000 g

to separate plasma and stored at −80°C until analysis.

Plasma total cholesterol (TC), HDL, LDL and triglyceride concentrations were measured enzymatically on an Integra 400 Plus analyser (Roche Diagnostics GmbH). Fasting insulin concentration was measured in serum by ELISA (Mercodia, Uppsala, Sweden; inter-assay variability: 2.9 %; intra-assay variability: 3.4 %), which shows no cross-reactivity with proinsulin. In addition, fasting glucose levels were measured using Glucose oxidize. Insulin resistance was calculated with the homeostasis model assessment (HOMA-IR) by the following formula: fasting plasma glucose (mmol/L) x fasting serum insulin (μU/mL)/22.5.

Apelin concentrations in the plasma were measured by an enzyme-linked immunosorbent assay (Elisa; Phoenix Pharmaceuticals inc., Burlingame, CA, USA), according to the manufacturer's protocol. Plasma chemerin was measured by Elisa (Cusabio Biotech, Wuhan, china; sensitivity; 7.8ng/ml, inter-assay variability: <10 %; Intra-assay: <8 %). hs-CRP concentration was measured by Elisa (apDianv, Turnhout, Belgium; inter-assay variability: 6.1 %; intra-assay variability: 5.1 %). SAA concentrations were measured by Elisa (Cusabio Biotech, Wuhan, China, inter-assay variability: 7.2 %; Intra-assay: 5.6 %).

2.5. Statistical Analysis

Normality of the data was tested using a Shapiro-Wilk test. The two way repeated measure analysis of variance (ANOVA) was used to evaluate changes within and between groups (time*group interaction). A paired t-test was used to measure within group changes. The statistical significance level was set to $\alpha \leq .05$. Pearson correlation was used to determine the relationship between variables. SPSS version 19.0 was employed to analyse data.

3. Results

3.1. Body Composition

The statistical analysis using independent t-test showed that there was no significant difference in body composition between the two groups at pre-test ($P>.05$).

Intragroup changes: Compared to the baseline, body weight (P<.001), BMI (P=.002), and WHR (P=.044) significantly decreased in the CRT group following the intervention when compared to the pre-test. No changes were found in body weight (P=.458), BMI (P=.31), and WHR (P=.15) in the control group following the intervention period.

The two way repeated measure ANOVA showed that there was a significant decrease in body weight, BMI, and WHR in the CRT group compared to the control group (time*group interaction, P<.05) (Table 1).

3.2. Plasma parameters

The statistical analysis using independent t-test showed that the chemerin concentration was higher in the training group (P= .01), but there were no significant differences in other plasma markers at pre-test (P> .05) (Fig. 1B).

The paired t-test revealed that SAA concentration significantly decreased after 8 weeks in the CRT group (P=.026) (Fig. 1A). The CRT tended to decrease apelin and chemerin concentrations, although the differences did not reach statistical significance (P=.073, P=.081; respectively) (Fig. 1B and 1C). Moreover, there was no significant difference in hs-CRP (P=.240) (Fig. 1D) following CRT. Further analysis of paired t-test showed that, plasma levels of TG (P<.001), TC (P=.001), LDL (P=.03) and insulin (P<.001), and also HOMA-IR (P<.001) significantly decreased in response to CRT in the post-test period when compared to the pre-test samples. Furthermore, HDL concentration increased significantly after 8 weeks CRT (P=.003) (Table 3), but glucose concentration (P=.235) did not change following the intervention period compared to the pre-test samples in the CRT group.

In the control group, the paired t-test results showed that no significant changes were found in plasma concentration of apelin (P=.394), chemerin (P=.804), hs-CRP (P=.493) and SAA (P=.061) in the post-test period compared to the pre-test samples, (Fig. 1). Also, there was no significant difference in plasma concentrations of LDL (P=.364), TG (P=.125), TC (.598), HDL (P=1.00) (Table 1), glucose (P=.277) and insulin (P=.763) as well as HOMA-IR (P=.405) in the post-test compared to the pre-test in the control group (Fig. 2).

The two way repeated measure ANOVA showed that Chemerin and SAA concentration significantly decreased in the training group compared to the control group (time*group interaction, P=.038, P=.004 receptively), But there were no significant differences in apelin, hs-CRP and glucose concentrations between two groups (time*group interaction, P>.05) (Fig. 1). In addition, Plasma levels of LDL, TC, TG and insulin significantly decreased in CRT compared to the control group (time*group interaction, P<.05). Moreover, HOAM-IR decreased, but HDL plasma levels increased in the CRT compared to that of the control group (time*group interaction, P=.035) (Table 3).

After adjusting for change in body weight and BMI, training effects on chemerin concentration attenuated and lost statistical significance (P=0.066, P=0.047 receptively), but SAA concentration remained statistically significant between groups (P=0.014, P=0.005 receptively),

3.3. Correlation between inflammatory variables and anthropometrical changes

The pearson correlation showed that there was no correlation between plasma apelin and anthropometrical characteristics (P>.05). But chemerin had a significant positive correlation with WHR and SAA (Table 4). Also, we found a positive correlation between apelin and acute phase inflammatory proteins (SAA and CRP; table 4). Chemerin and apelin changes were positively correlated with HOMA-IR (Table 4). Moreover, chemerin changes had a significant positive correlation with glucose changes (table 4). But apelin had no correlated with glucose and insulin changes (table 4). In addition, SAA had a strong positive correlation with insulin (r=.685, P=.002) and HOMA-1R changes (r=.748, P<.001), But there was no significant correlation between CRP and glucose homeostasis (P>.05). Plasma TG concentration was significantly correlated with CRP (r=-.471, P=.49) and SAA (r=.589, P=.01) changes.

4. Discussion 1

The present study evaluated the effects of CRT on some inflammatory markers, cardiometabolic risk factors, and insulin resistance in obese young men. The main findings were that the CRT improves cardiometaboilc risk factors (lipid profile and some adipokines related to cardiovascular disease), and glucose homeostasis, and alleviates acute inflammation (e.g. SAA) in obese young men. 2 3 4 5 6

Obesity causes low-grade inflammation, which leads to an imbalance in the secretion of adipokines. This, in turn, reduces insulin sensitivity and increases inflammation [2]. Chemerin is an essential adipokine, which plays a role in inflammation and adipocyte differentiation [8]. Similar to previous studies [4, 24], in this study, the levels of chemerin decreased about 11% after eight weeks of CRT. In contrast to chemerin, apelin concentration was reduced, although the reduction was not statistically significant. Plasma apelin levels are increased by obesity and reduced by diet-induced weight loss, and this change was significantly correlated with serum levels of triglyceride, glucose, and HOMA-IR [25]. A previous study showed that apelin was correlated with insulin sensitivity and it was reduced after 12 weeks of intense exercise in obese individuals [6]. In the present study, HOMA-IR significantly reduced and positively correlated with apelin. It has been shown that when insulin resistance is decreased, this leads to a decline in apelin levels compensatory mechanism to further increase insulin sensitivity [26]. This decline in apelin is likely to result in insulin sensitivity improvement and there was no compensatory advantage in more reduction in apelin concentration. 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22

Also, we found that other cardiometablic risk factors including LDL, TC, and triglyceride decreased after the training programme. On the other hand, HDL levels also significantly increased. In this line, Fett, Fett & Marchini (2009) showed a significant reduction in the total cholesterol and triglycerides levels following resistance training [27]. Owing to the nature of CRT, which increases the aerobic capacity [19], this type of training is likely to cause the compatibility of the oxidative pathway, thereby increasing lipid oxidation and reducing lipid profiles. Although the mechanisms of the effect of exercise training on the lipid profile remain unclear, exercise training appears to enhance the ability of muscles to oxidize lipid, which leads to a reduction in plasma 23 24 25 26 27 28 29 30 31

lipid concentrations. These mechanisms may include increases in lecithin–cholesterol acyltransferase (LCAT; the enzyme responsible for the HDL-cholesterol ester transfer) following exercise and increases in lipoprotein lipase activity involved in lipid metabolism, which increased the ability of muscle fibres in the oxidation of plasma lipids such as triglycerides and VLDL cholesterol [28].

One of the possible mechanisms by which exercise training affects adipokines may be due to the body composition changes. Resistance exercise has been shown to improve body composition by increases in lipolysis in abdominal adipose tissue, energy expenditure, and fat oxidation in obese subjects [29]. Moreover, CRT increases the maximum oxygen consumption (VO_2max) [19]. The VO_2max increases oxygen delivery to the working muscles, thereby increasing the fat oxidation [30] and reducing the fat cells' size and content [31]. In addition, it has been shown that the nM increase of circulating chemerin is accompanied by a decrease in the fat cell beta-adrenergic receptor function [10], lower the intracellular cyclic AMP [32], and a decrease in the function of catecholamines. It has been reported that the circulating levels of chemerin are positively correlated with the BF% [24]. Our results showed that the body weight, BMI, and WHR and were significantly lower in the training group compared to the control group, so it is likely that the decline in circulating chemerin would be caused by the improvement in the body composition.

Accordingly, a pervious study reported that the circulating levels of apelin were not necessarily correlated with weight and body fat mass, but it was strongly correlated with the levels of inflammatory parameters [33]. In our study, SAA fell following CRT, and a positive correlation was found between SAA and insulin or HOMA-IR. SAA has been shown to inhibit the genes involved in maintaining normal insulin sensitivity [34]. Thus, the reduction of SAA in this study is likely caused by an improvement in insulin sensitivity. In this regard, Ogawa and co-workers (2010) showed that the serum levels of SAA were reduced in response to 12 weeks of resistance training [35]. However, the serum levels of SAA did not change in older women in response to eight weeks of aerobic exercise. But, the levels of CRP decreased [36]. Moreover, higher levels of Chemerin in moderate and severe obesity were also found to be significantly associated with higher circulating levels of CRP [37]. In this study, CRP levels dropped in the

exercise group, but the reduction did not reach statistical significance and had no correlated with chemerin changes. In agreement with our study, Franklin and co-workers (2015) observed that CRT had no effect on h-CRP levels in obese women [22]. Nevertheless, chemerin changes had a positive correlation with SAA changes. Compared to CRP, SAA is known as a more sensitive biomarker of inflammation [14]. Thus, it seems that the response of these two acute phase proteins (SAA and h-CRP) is different. The proposed mechanisms for reducing the acute phase inflammatory proteins by exercise training may be due to the up-regulation of the levels of IL-6 and the suppression of TNF-α, which lead to the regulation of the CRP levels [38]. A study showed that 12 weeks of resistance training reduced the activity of the TNF-α messenger RNA and the protein expression [39]. Hence, perhaps the resistance training by regulating the cytokine levels reduced the CRP levels. Resistance training also increases the lean body mass by increasing protein synthesis [35]. It has been shown that TNF-α reduced protein synthesis in skeletal muscle and increased protein catabolism by reducing the translation initiation process [40]. Therefore, it may increase the protein synthesis induced by resistance training [39] which resulted in the suppression of inflammatory responses and significantly reduced the CRP levels.

Ambiguous and contradictory evidence prevents the current understanding of resistance training capacity to reduce the systemic markers of chronic inflammation. Some evidence shows that resistance training does not have any effect on CRP or inflammatory cytokines [4]. Otherwise, another study reported that resistance training causes a noticeable reduction in inflammatory cytokines [41]. The inconsistency of these results may be attributed to the subjects' characteristics (e.g. age, basal inflammation, training status, and body composition) and/or the differences in methodology (e.g. training volume, intensity, and training period time). The physiological mechanism of the effectiveness of resistance training on inflammation levels may be contributed to several factors. Based on the main role of adipose tissue in the secretion of several inflammatory cytokines, the reduction in the BMI and WHR in our study may affect the adipose tissue secretion of inflammatory cytokines. Furthermore, resistance training stimulates protein synthesis rates in the skeletal muscle, so this stimulation may induce the secretion of anti-inflammatory myokines in such a

way that it can affect inflammatory cytokines [41]. The lack of any significant change in hs-CRP levels in the present study could be owing to the short duration of exercise training. This is because the study showed that a longer duration of exercise training needs to reduce the acute phase inflammation [35].

In the current study, the body composition (body weight, BMI, waist circumference, and hip circumference) significantly improved via CRT, which is consistent with a previous study [42]. It seems that resistance training by increasing the resting metabolic rate and the fat-free mass reduces body fat [43], which results in a negative energy balance. Thus, these results suggest that a decrease in the body weight and BMI lead to a parallel reduction in the circulating levels of SAA. In the current study, SAA had a positive correlation with plasma triglyceride changes. Moreover, it seems that SAA has a role in lipid transfer through the reverse cholesterol transport to the peripheral cells, and it facilitates the removal of free cholesterol at the sites of inflammation [44]. Lipid profile (especially very low-density lipoprotein (VLDL) and triglyceride) has a tendency to increase during inflammation [11]. Furthermore, it has been suggested that SAA may affect the lipids and the apo-lipoproteins metabolism during inflammation [11]. Consequently, it is plausible that decrease in SAA caused by the CRT can improve lipid profile in obese individuals.

A possible limitation of this study was that only obese young men were investigated. It is well documented that increasing adipose tissue mass with age throughout the body leads to low-grade inflammation by producing high levels of inflammatory adipokines [45]. In addition, it has been shown that the key mediators of inflammatory cytokines are up-regulated by the aging process [46]. In this regard, Bruunsgaard and Pedersen (2003) reported that inflammatory cytokines (e.g. hsCRP and SAA), even in the absence of chronic diseases, increased two to four-fold in elderly compared to young adults [47]. Accordingly, this limitation should be considered in generalizing these findings to all age groups.

5. Conclusions

To the best of our knowledge, it is the first study that showed that eight weeks of CRT could improve circulating levels of inflammatory proteins and some adipokines in obese

young men. These improvements were associated with insulin resistance reduction. Thus, these results confirm that CRT can ameliorate cardiometabilc risk factors in part through improving body composition in obese young men. Overall, although CRT may be useful as an exercise training therapy for chronic diseases involving higher cardiometabolic risk factors in young obese individuals, further studies are needed to elucidate the beneficial effects of CRT on inflammation and metabolic syndrome in other populations (e.g. obese children, women, and elderly).

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Table 1. Anthropometrical characteristics of the participants. 6

	Control group		Training group	
	Pre-test (n=15)	Post-test (n=13)	Pre-test (n=15)	Post-test (n=13)
Age (yrs)	24.0±4.8	-	23.0±3.8	-
Weight (kg)	101.6±13.49	102.4±13.48	99.1±12.3	96.6±12.08*$
BMI	31.1±3.2	31.4±3.2	30.12±2.99	28.06±3.00*$
WHR	.98±0.97	1.00±0.12	.90±.08	.87±.05*$

Data are given as Means ± SD. Differences between groups for baselines were tested with paired t- test. Also, Differences 7
between groups for changes were tested with ANOVA repeat measure. The*significant difference compared to pre-test. The $ 8
Significant difference compared to the control group (time*group interaction). 9

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Table 2. Training programme. 13

Training sessions	Percentage of 1-RM	Repetition
1-3	65	12-14
4-7	70	10-12
8-12	75	8-10
13-18	80	8-10
19-end	85	6-8

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Table 3. Lipid profile changes in the resistance training and control groups.

Variable	Control group		Training group	
	Pre-test	Post-test	Pre-test	Post-test
TG (mg/dL)	164.3±13.5	166.3±14.3	168.6 ±7.68	148.25±10.1*$
TC (mg/dL)	195.9±17.4	193.8±16.9	198.8±16.7	185.6±14.2*$
HDL (mg/dL)	43.9±6.53	43.9±5.85	45.3±7.3	48.3±6.9*$
LDL (mg/dL)	108.6±11.7	111.1±15.5	110.2±9.5	105.1±9.9*$

Data are given as Means ± SD. Differences between groups for baselines were tested with paired t- test. Also, Differences between groups for changes were tested with the two way ANOVA repeat measure. The*significant difference compared to pre-test. The $ Significant difference compared to the control group (time*group interaction).

Table 4. Pearson correlation between variable changes

	Apeiln		Chemerin	
	R	P	r	P
Weight	.249	.319	.109	.312
BMI	.178	.480	.121	.633
WHR	.245	.067	.542	.034*
TG	.123	.435	.032	.901
LDL	.098	.701	.035	.881
HDL	-.021	.935	-.149	.554
TC	.029	.910	.320	.902
Glucose	.404	.096	.547	.019*
Insulin	.407	.093	.377	.123
HOMA-IR	.561	.014*	.506	.032*
SAA	.467	.033*	.490	.039*
CRP	.543	.028*	.294	.236

*significant difference at α <.05

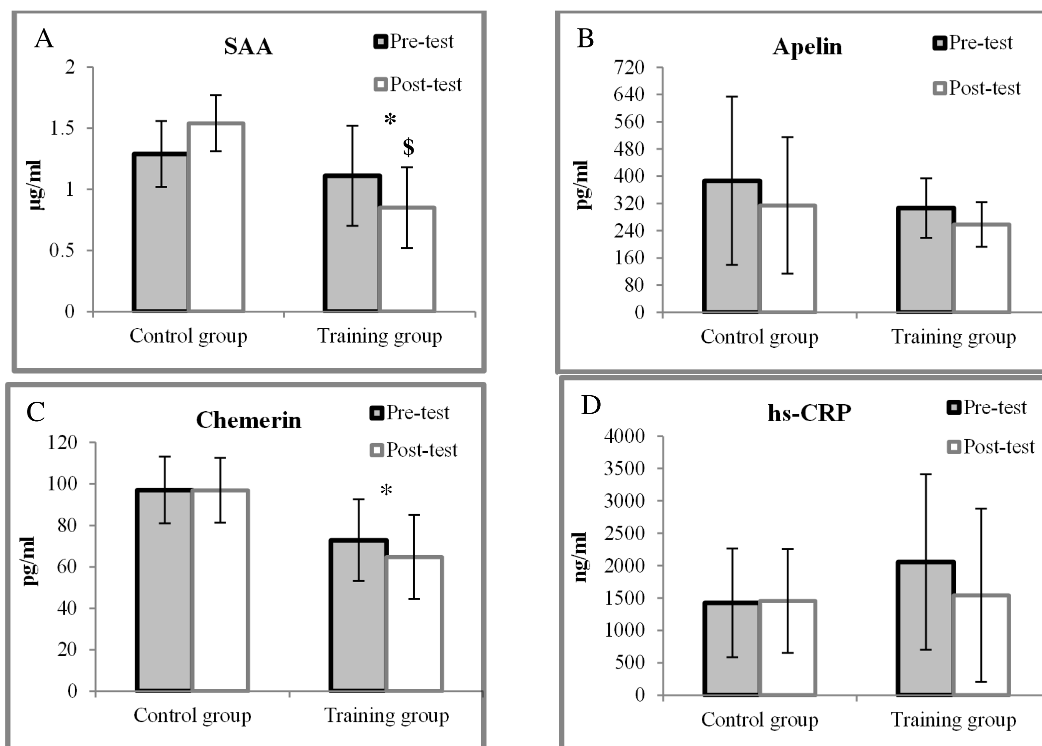

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Figure 1. Effects of progressive circuit resistance training on inflammatory biomarkers. Values are in mean ± SD. A) Plasma apelin concentration; B) Plasma chemerin concentration, *P=.038 compared to the control group (time*group interaction); C) Plasma SAA concentration, *P=.004 compared to the control group (time*group interaction), $P=.026 compared to the baseline; D) Plasma hs-CRP concentration. SAA: Serum amyloid A; hs-CRP: high-sensitivity C-reactive protein.

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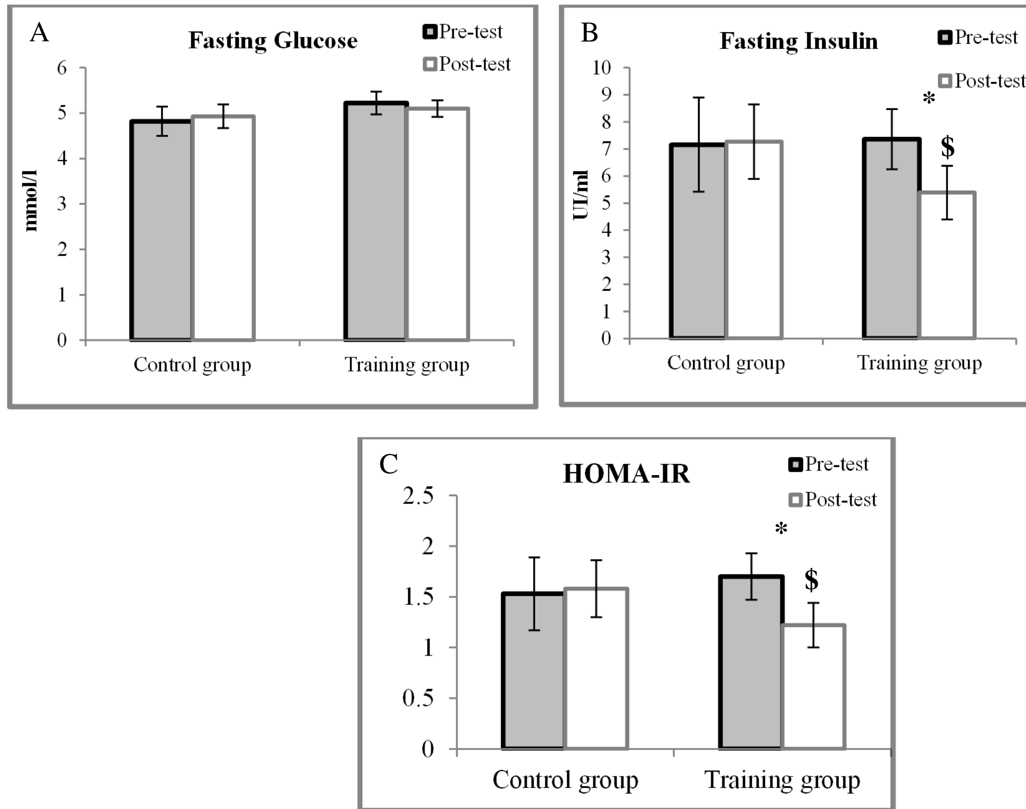

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Figure 2. Effects of progressive circuit resistance training on Glucose, Insulin, and HOMA-IR. Values are in mean ± SD. A) Fasting Glucose concentration; B) Fasting Insulin concentration, *P <.001 compared to the control group (time*group interaction), $P< .001 compared to the baseline; C) HOMA-IR , *P< .001 compared to the control group (time*group interaction), $P< .001 compared to the baseline. HOMA-IR: Homeostatic model assessment of insulin resistance.

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