

Effect of an eight-week multi-joint circuit resistance training program on adiponectin levels and lipid profiles in overweight women from Absard City

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Abstract

Background: Circuit resistance training, beneficial to overweight individuals, combines aerobic and resistance exercises. This study aimed to investigate the effect of multi-joint circuit resistance training on serum adiponectin levels and lipid profiles in overweight women.

Methods: Thirty sedentary overweight women (Mean age of 34.48±4.304 years, mean weight of 81.17±9.137 kg, and mean BMI of 30.17±2.13 kg/m²) were randomly divided into two groups: circuit resistance training (n=15) and a control group (n=15). The experimental group trained at 65-85% of maximum heart rate for eight weeks, with three sessions per week (24 sessions total). The control group did not engage in regular exercise. Serum adiponectin levels and lipid profiles were assessed before and after the intervention. Data were analyzed using paired sample t-tests and dependent t-tests at a significance level of P-value<0.05.

Results: The results showed that adiponectin levels (P-value=0.001) and cholesterol levels (P-value=0.015) increased in the circuit training group, while LDL levels decreased (P-value=0.031). HDL levels increased but were not statistically significant (P-value=0.100). These variables did not change in the control group.

Conclusion: Finding ways to improve the state of adipokines is of vital importance for these patients. According to the results, circuit resistance training may be an effective method for addressing dyslipidemia and increasing adiponectin levels in overweight women.

Highlights

What is current knowledge?

- Obesity is one of the most serious health problems associated with adipokines.
- Circuit resistance training affected the adiponectin levels of obese women.
- Circuit resistance training had significant effects on the lipid profile of obese women.

What is new here?

Exploring the impact of multi-joint circuit resistance training on indicators associated with obesity is a novel aspect of this study.

Introduction

Obesity has increased significantly over the past few decades worldwide (1). Weight gain occurs due to an energy imbalance, with the body receiving more energy than needed (2). Adipose tissue dysfunction leads to conditions such as metabolic syndrome, cardiovascular disorders, and cancer in obese individuals (3). In general, metabolic syndrome is defined as the presence of at least three clinical risk factors, including abdominal (Visceral) obesity, hypertension, dyslipidemia, decreased high-density lipoproteins, high triglycerides (TGs), free fatty acids, low-density lipoproteins, and insulin resistance (4).

The adipose tissue was initially regarded as an energy storage organ; however, it has recently been identified as an active endocrine tissue (5). Adipose tissue produces and releases various pro-inflammatory and anti-inflammatory factors, including leptin, adiponectin, visfatin, resistin, and omentin, which function at autocrine, paracrine, and endocrine levels (6). Adiponectin is a protein composed of 244 amino acids and is found in the blood in three different molecular weights: low molecular weight, moderate molecular weight, and high molecular weight (7). High molecular weight is most significantly associated with obesity, cardiovascular diseases, and metabolic syndrome (8). Adiponectin activates adenosine monophosphate-activated protein kinase (AMPK) through the adaptor protein phosphotyrosine interacting with PH domain and leucine zipper 1. AMPK is primarily a signaling pathway that ultimately increases beta-oxidation, glucose transporter type 4, cluster of differentiation 36, and glucose and fatty acid intake by cells (8).

Adiponectin exerts its effects through two membrane receptors, AdipoR1 and AdipoR2; the former is primarily found in muscles, and the latter in the liver (9). In addition, adiponectin activates AMPK phosphorylation by binding to AdipoR1, thereby increasing glucose use and fatty acid oxidation in muscles and the liver (10). Adiponectin also activates PGC-1, a cell receptor that facilitates the release of mitochondrial proteins (11). PGC-1 regulates lipid metabolism and

long-chain fatty acid oxidation through the expression of several carboxylic acid cycle genes and the mitochondrial fatty acid pathway (12). Furthermore, PGC-1 increases the expression of fibronectin type III domain-containing protein 5 (13). This receptor also stimulates mitochondrial biogenesis and angiogenesis while reducing atrophy.

Adiponectin has antidiabetic properties due to its ability to increase insulin sensitivity (14). Studies indicate decreased adiponectin levels in obese individuals, which lead to increased susceptibility to health conditions such as metabolic syndrome, insulin resistance, and type II diabetes (15). A negative correlation has also been reported between this protein and the body mass index (BMI) (16). Conversely, increased adiponectin levels can significantly reduce total cholesterol, low-density lipoprotein (LDL), and TGs while increasing high-density lipoprotein (HDL) (17).

Esposito et al. (2003) reported that aerobic exercise for two years in obese middle-aged women resulted in a decrease in body weight (BW) along with an increase in plasma adiponectin concentration. Contrary to these positive effects of exercise on adiponectin levels in the blood of obese individuals, Hara et al. (2005) suggested that complex exercise for eight weeks in obese adolescents did not induce changes in plasma adiponectin concentration, and Polak et al. (2006) reported that aerobic exercise for 12 weeks did not increase blood adiponectin concentration in obese individuals (18).

In a study, six weeks of acute aerobic training at an intensity of 40-50% VO₂max was reported to have no significant effect on adiponectin (19). Another study assessed the effect of aerobic training with varied intensities on adiponectin, indicating that eight weeks of low-intensity aerobic exercise had no significant effect on adiponectin. Meanwhile, moderate- and high-intensity aerobic training were shown to significantly increase adiponectin. In a related study, one session of aerobic training at an intensity of 65% VO₂max significantly reduced adiponectin (20).

In general, regular resistance training has been found to be effective in decreasing body fat in women and increasing adiponectin (21). High-intensity interval training significantly increases serum adiponectin (22). A study in this regard showed an increase in serum adiponectin levels following resistance and aerobic training, which improved inflammation and metabolic syndrome (23). Evidence suggests that regular physical activity also reduces TGs and LDL while increasing HDL (24). The most significant LDL reduction and HDL increase have been reported in individuals performing aerobic training at an intensity of 55-65% VO₂max (24). Furthermore, resistance training has been shown to significantly affect the lipid profile. A study indicated a significant decrease in TG and LDL and a significant increase in HDL following resistance training exercises (25).

Recently, combined aerobic and resistance training has been suggested to have a more favorable impact on cardiovascular and general health compared to

aerobic and resistance training performed separately (26). Circuit training, which is beneficial to overweight individuals, is a combination of aerobic and resistance training (27). These exercises increase muscle endurance and strength, as well as aerobic function (28). Previous studies have indicated that circuit training is associated with a significant decrease in blood pressure and an increase in muscle tissue, strength, and aerobic capacity (29). Moreover, circuit training has been reported to improve metabolism by increasing oxygen intake (29).

Considering these research findings, the effect of exercise type, intensity, and/or duration on blood adiponectin levels in obese individuals remains controversial. Therefore, the purpose of this study was to investigate the effect of eight weeks of resistance circuit training on plasma adiponectin levels and lipid profiles in overweight women.

Methods

This quasi-experimental research was performed with a pretest-posttest design and a control group. After the call, volunteers were invited to participate in the project through a public announcement. In addition, patients with medical records at the Research Institute were informed about and invited to the project through phone calls. The sample population included overweight women aged 25-45 years. Inclusion criteria were a BMI of 28-40 kg/m², absence of specific diseases, and no regular sports activity within the past six months. Individuals with hereditary hypolipoproteinemia and other chronic diseases, such as diabetes mellitus, or those who were drug users, were excluded from the study. In total, 30 women with a mean age of 34±4 years, mean height of 165.35±7.5 centimeters, mean weight of 81.17±9.13 kilograms, and mean BMI of 30.17±2.13 kg/m² were enrolled in the study. The participants were randomly divided into two groups: circuit resistance training (n=15) and control (n=15). The present research was conducted in Absard City in the winter of 2020.

In one session, the subjects were familiarized with the type of the study, its objectives, implementation methods, and possible risks, and informed consent was obtained. Data were collected using the Physical Activity Readiness Questionnaire prior to the research. Notably, 10 participants withdrew from the research for personal reasons. Finally, eight and 10 participants remained in the training and control groups, respectively. The study protocol was approved by the Ethics Committee of the Sports Sciences Research Institute (Code: IR.SSRC.REC.1398.114).

Anthropometric indices, including height, weight, fat percentage, and abdominal circumference, were measured. Height was measured using a tape measure with 0.5 cm precision, and the fat percentage of the participants was calculated using a skinfold caliper, which measures subcutaneous fat based on a three-site method (triceps, pelvis, and thigh) on the right side of the body, after applying the digits obtained by the Jackson and Pollock body density formula (30). Furthermore, maximum strength was measured using an indirect method involving leg and chest press movements to assess lower body and upper body strength, respectively (31).

Chemical variables were measured based on blood samples (5 cc) collected from the participants by a specialist at two stages: 48 hours before the first exercise session and 48 hours after the last exercise session. The samples were collected in a laboratory after 8-10 hours of fasting. After centrifuging the blood samples at 2,500 rpm/min, the samples were transferred to the Shahid Beheshti Endocrinology and Metabolism Research Institute in Tehran, Iran, in a special package at a specific temperature. The exercise protocol was performed by the training group in a multifunctional center, with three sessions per week for eight weeks. The control group was assigned no regular physical activity (Table 1).

With regard to the biochemical indices, adiponectin was measured using the ELISA assay with a sensitivity of 0.1 ng/l and an intragroup change coefficient of 7.2%, using the Zellbio GmbH kit (Made in Germany). TGs were measured using a colorimetric enzymatic method with a sensitivity of 1 mg/dl and an intragroup change coefficient of 2.3%, using the Pars test kit. Cholesterol was measured using an enzymatic photometric method with a sensitivity of 3 mg/dl and an intragroup change coefficient of 2.1%, using the Pars test kit. HDL was measured using an enzymatic photometric method with a sensitivity of 1 mg/dl and an intragroup change coefficient of 3.4%, using the Pars test kit. LDL was measured using the Friedewald formula (32).

Data analysis was performed in SPSS version 24 using the Shapiro-Wilk test to determine the normal distribution of data among the groups. In addition, Levene's test was used to assess the homogeneity of variance between the groups. One-way analysis of variance (ANOVA) and dependent t-test were also applied at the significance level of P-value ≤ 0.05.

Exercise protocol

The training protocol consisted of a warm-up (10 minutes of jogging and light physical activity), the main exercises, and a cool-down (Five minutes of stretching). The circuit resistance training protocol was implemented three sessions a week, with each session involving 60 minutes of resistance bodyweight training (Table 2). The exercises were performed in three rounds, with 2-3 minutes of rest intervals between each round of running and the desired stations, at an intensity of 55-65% VO₂max.

Results

According to the results of the paired t-test, eight weeks of circuit resistance training significantly increased adiponectin levels (P-value=0.001) and significantly decreased LDL (P-value=0.031) and cholesterol (P-value =0.001). In addition, TG levels decreased, and HDL levels increased, although these changes were not considered significant (Table 2).

According to the obtained results, weight, fat percentage, and BMI significantly decreased in the training group, while no significant changes were observed in the control group. The results of the independent t-test also indicated significant differences between the study groups regarding changes in adiponectin levels, LDL, cholesterol, weight, fat percentage, and BMI (Table 2).

Table 1. Circuit resistance training protocol

Exercise	Week 1	Week 2	Week 3	Week 4
	Repetition	Repetition	Repetition	Repetition
Burpees with no jumps	10	15	20	25
Crunches	10	10	15	20
Squats	10	15	20	25
Reverse crunches	10	10	15	20
Push-ups	10	10	12	12
Step	15	20	25	25
Hyperextension	15	15	20	20
Agility ladder	15	20	20	25
Agility shuffles	15	20	20	25
Planks (On forearms)	30 sec	40 sec	50 sec	60 sec
Exercise	Week 5	Week 6	Week 7	Week 8
	Repetition	Repetition	Repetition	Repetition
Burpees	15	15	20	25
Crunches and Reverse crunches	25	30	30	35
Squats, jump, and lunges	20	20	25	30
Side plank	20	25	30	30
Lunges	20	20	25	30
Step	30	30	35	35
Bridge	20	25	25	30
Agility ladder	25	25	30	35
Dolphins	30	30	35	35
Planks (On forearms)	60 sec	70 sec	80 sec	90 sec

Table 2. Changes in biochemical variables at pre-test and post-test

Indices	Circuit resistance training group		Control group		P-Value between groups (Post-Test)
	Pre-test Mean±SD	Post-test Mean±SD	Pre-test Mean±SD	Post-test Mean±SD	
Adiponectin (ng/l)	6.17±0.54	7.13±0.47	6.21±0.82	6.38±0.78	0.001
Total Cholesterol (mg/dl)	204.75±32.49	172.12±35.56	200.7±18.52	198.9±19.45	0.002
LDL (mg/dl)	135±29.39	102.97±33.17	135.2±18.05	136±16.35	0.040
HDL (mg/dl)	33.92±6.38	37.32±12.05	36.71±12.11	36±13.07	1.000
TG (mg/dl)	156.62±32.34	140.5±18.38	156.7±60.68	185.3±64.48	1.000
Weight (kg)	63.48±9.01	61.50±9.10	64.24±11.84	65.47±12.82	0.001

The variables evaluated in the pre-test did not have significant differences between the groups, and the post-test values were compared.

Discussion

According to the results of the present study, eight weeks of circuit resistance training significantly decreased BW, BMI, fat percentage, LDL, and cholesterol, while significantly increasing adiponectin levels in the training group compared to the control group. However, no significant changes were observed in TG and HDL levels. Consistently, Ricci et al. reported increased adiponectin levels and improved insulin resistance after eight weeks of resistance training (33). According to Zamani et al., eight weeks of resistance training significantly increased adiponectin and testosterone levels in healthy male participants (34). Furthermore, Dehrashid et al. reported a significant increase in adiponectin levels after 12 weeks of circuit resistance training (35). Similarly, Dehrashid et al. observed a significant increase in adiponectin levels following 10 weeks of resistance training. In the mentioned research, the reduction in TG, LDL, and cholesterol was not considered significant (35).

According to the literature, long-term sports activities increase adiponectin levels (36). While all the studies in this regard involve eight weeks of training, the interventions range from high-intensity interval training to endurance training (36). Moreover, the duration of these interventions varies, with the number of weekly sessions ranging from a minimum of three per week (21,37) to a maximum of seven per week (38-40).

In a study conducted by Vardar et al., two training protocols commonly used in community training programs were compared. The proper exercise volume (Six sessions per week) was highlighted as a strength of the exercise protocol in the mentioned study, while the inadequate adiponectin measurement (Gene expression alone without assessing plasma adiponectin levels) was noted as a limitation (41). Studies conducted by Karajbani et al. (2018), Rostamizadeh et al. (2018), Hosseini et al. (2018), Mosaf and Abedi (2018), Ghaleno et al. (2017), Bettiol et al. (2017), Albashi et al. (2017), Piri and Zamani (2016), Nader et al. (2016), Bouri and Piri (2015), and Wang et al. (2015) were similar to the current research in terms of objectives and design. The common positive aspects of these studies included the number and type of participants (Obese and overweight individuals and patients) and the appropriate length of the interventions (8-20 weeks). However, the limitations of these studies included the use of the maximum heart rate index instead of maximum oxygen consumption in most cases, which might have reduced the accuracy of controlling training intensity and resulted in the relatively low volume of training (Three sessions per week).

Adiponectin is a protein hormone secreted by adipocytes that aids in insulin sensitivity and inflammation (18), regulating glucose metabolism in skeletal muscle and adipocytes through the activation of phosphorylated AMP-activated protein kinase and proliferator-activated receptors. Obese individuals have a lower plasma concentration of adiponectin compared to those with normal weight, and a reduction in BW and BFM increases adiponectin levels in the blood (18).

Controlling environmental confounding factors is one of the key strengths of the animal studies conducted by Vardar et al (2018) and Racil et al. (2013). In these studies, less attention was given to the type of diet, which could be considered a limitation. Meanwhile, high training intensity and the use of the maximum oxygen consumption index instead of the maximum heart rate increased the accuracy of these studies. The relatively small number of participants and training sessions might have also affected the results of the aforementioned studies (41,42).

Voss et al. (2016) conducted research on individuals with athletic bodies, reporting that exercise background could affect the adiponectin response to sports activities (43). Therefore, performing similar studies on non-athletic individuals could yield interesting results. In another study, Saunders et al. (2012) evaluated the effect of moderate-intensity running (Close to the anaerobic threshold) on obese individuals. One of the strengths of the mentioned study was its appropriate number of participants (n=38) and their condition (Obesity). Meanwhile, their exercise protocol (i.e., running) burns a considerable number of calories, which might have altered the adiponectin levels of the subjects more significantly. In addition, VO₂max is considered a more accurate index for controlling exercise intensity (44).

Given the involvement of large muscles, multi-joint bodyweight resistance exercises increase energy consumption, which is essential for overweight individuals (45). Therefore, it seems that eight weeks of circuit training could increase adiponectin levels while saving time. Furthermore, these exercises could decrease LDL, cholesterol, body fat percentage, and weight by increasing energy consumption. It is suggested that multi-joint and aerobic exercises be incorporated into circuit training protocols for overweight individuals.

Conclusion

According to the findings of this study, an 8-week circuit resistance training program can be effective in improving adiponectin levels and lipid profile in obese women.

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Ethical statement

The study protocol was approved by the Ethics Committee of the Sports Sciences Research Institute (Code: IR.SSRC.REC.1398.114).

Conflicts of interest

The authors have no conflicts of interest that are directly relevant to the content of this original research paper.

Author contributions

Sara Valikhani contributed to data collection, statistical analysis. Mehdi Hedayati contributed to laboratory experiments. Seyed Mohsen Avandi conceived and supervised the study. All authors read and approved the final manuscript.

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