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The effect of endurance and resistance training on adiponectin in sedentary young woman

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Abstract

Adiponectin may be a marker for coronary artery disease and seems to have protective metabolic and anti-inflammatory properties, which prevents atherosclerosis. The purpose of this research was to determine the effect of endurance and resistance training on Adiponectin in sedentary young women. In this regard, 30 sedentary young women subject (BMI ≥ 25) were randomly assigned to three groups (endurance training, resistance training and control). The experimental training programs were performed three days a week for 12 weeks at a definite intensity and distance. Before and after 12 weeks intervention, Adiponectin, weight and body composition, Vo2max was measured for all subjects. Data were analyzed by one way analysis of variance ($p \leq 0.05$). Results showed that endurance and resistance training caused a significant increase in Adiponectin and Vo2max, significant decrease in weight and body percent fat of the experimental group in comparison to control group. This data suggest that physical training have positive effects on serum Adiponectin.

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Introduction

Human adipose tissue is not merely a fat storage depot, but has been recognized as an endocrine organ capable of producing biologically active proteins termed "adipokines" (Kondo *et al.*, 2006). Adipokines included adiponectin, leptin, resistin, tumour necrosis factor alpha and interleukin (Rohti *et al.*, 2006; Gouzik *et al.*, 2006). One of these adipocytokines is adiponectin, which is reduced with obesity, increased insulin resistance, dyslipidaemia and diabetes. Adiponectin may be a marker for coronary artery disease (Hotta *et al.*, 2000) and seems to have protective metabolic and anti-inflammatory properties (Marcell *et al.*, 2005), which prevents atherosclerosis (Ahima *et al.*, 2006). Moreover, adiponectin has been found to increase with weight loss, and be negatively correlated with changes in body mass index (BMI), waist and hip circumference and plasma glucose levels (Yang *et al.*, 2001). Improvement in cardiovascular function by physical activity has been attributed to exercise-induced positive changes in metabolic abnormalities and risk factors that are associated with atherosclerosis (Laukkanen *et al.*, 2002; Thampson *et al* 2003). Adiponectin plays a protective role against the development of atherosclerosis by suppressing inflammatory processes on the vascular endothelium (Ouchi *et al.*, 1999; Ouchi *et al.*, 2001). Although less is known about the association of adiponectin with the beneficial effects of exercise, several studies have examined whether exercise training effects plasma adiponectin concentrations. And the results are controversial. Adiponectin levels increased, decreased in healthy humans (Kriketos *et al.*, 2004; Yatagai *et al.*, 2003). However, most of these studies indicate no significant changes in plasma adiponectin concentration after exercise training in spite of the variation in the subject's characteristics (healthy or diabetes), training protocols (single bout, intermittent or endurance) and intensities of the exercise (Hulver *et al.*, 2002; Boudou *et al.*, 2003; Kraemer *et al.*, 2003). Therefore, the present study was designed to determine and compare the effects of endurance and resistance training on adiponectin concentration in sedentary young women.

Material and methods

First of all call notices were posted in Azad University Shahre Qods Campus in which the researcher invited to identify overweight and obese individuals who were willing to run exercise for weight adjustment and improvement of their physiological conditions. In the next stage the candidates were invited for the purpose of the Initial assessments and from among them, at least 30 individuals with BMI ≥ 25 whose being overweight or obese was not associated with thyroid under-activity and did not have a history of exercise or caloric restriction diet were selected. After obtaining consent letters from the participants, they were asked to avoid rigorous physical activity 48 hours before the test and attend the pathobiology laboratory for blood sampling after 12 hours of fasting. The anthropometric measurements and maximal oxygen consumption of the subjects were done in the gym. The subjects were then divided randomly into three groups (Endurance training, Resistance training and control).

The height was measured using a medical height meter; weight and body composition were measured using a body composition monitor (OMRON, Finland). The maximum oxygen consumption of all the subjects was measured twice using the Cooper test; once before the test and once after the test. The subjects ran for 12 minutes at their maximum speed. The mileage was then placed in this formula:

$$\text{Vo2max} = \text{Mileage (M)} - \frac{504/9}{44/73}$$

The aerobic capacity of the subjects was calculated milliliters of oxygen for each kilogram of the body weight per minute. The amount of calories intake of the subjects was determined by data collection method using a three-day questionnaire, at the beginning, at the end and every fortnight during the exercise period (Foster *et al.*, 2005). The subjects were advised to keep up their usual diet during the research period.

Endurance training consisted of continues raining 3 days a week, for 12 weeks. A session of training program in Continues group included a ten-minute

warm-up with and stretching exercises. The subjects then continued with running a distance of 1600 to 3200 meters with the intensity of 60 to 75% of their maximum heart rate reserve (Table 1). They cooled off for five minutes.

Table 1. Continues training programs

Week	1	2	3	4	5	6	7	8	9	10	11	12
Target heartbeat (percentage)	60- 65%	60- 65%	60- 65%	60- 65%	65- 70%	65- 70%	65- 70%	65- 70%	70- 75%	70- 75%	70- 75%	70- 75%
Distance (meter)	1600	1600	1800	1800	2400	2400	2800	2800	3000	3000	3200	3200

Resistance training consisted of circuit weight training 3 days a week, for 12 weeks. This training was circularly performed in 11 stations and included four sets with 12 maximal repetitions at 50–60% of 1-RM in each station. The resting time between two stations was 30 second and the related time between the sets was 90 second. In order to determine the overload after a four - week training program, a test with one maximum repetition for each subject in each station will be carried out and the rat load will be determined based on it. General and specific warm-up was performed prior to each training session and each training session was followed by cool-down.

Five milliliter of blood was taken from each subject after 12 hours of fasting from the brachial vein and was reserved degrees by test time. Blood sampling in both phases was done between 8 and 9 AM of every subject. Biovendor kits were used accordingly to measure serum Adiponectin using ELISA method.

All values are represented as mean ± SD. As to the inferential statistics, first the Kolmogorov–Smirnov test was used for normal distribution Leven test was used for data homogeneity. Then one way analysis of variance test was used for testing significance between groups. All the statistical operations were performed by spss software and significance level of tests was considered $p \leq 0.05$.

Results

The descriptive profile of the groups in variables of age, height, weight, body mass index, body fat percentage and adiponectin as well as the one way analysis of variance are presented in the table 2. After 12 weeks of endurance and resistance training adiponectin level (p= 0.000) (Diagram 1) showed a significant increase. This increase was between endurance and resistance training whit control group and did not difference between two training groups. also the difference of measurements of variables of the three groups including Body weight, Body fat percentage, Maximum oxygen consumption was significant, that of this significant was between two training groups with control group and did not difference between two training groups ($p \leq 0.05$) (Table 2).

Table 2. Pre-and post-test physical, physiological and biochemical variables and one way analysis of variance test in the three groups

Group	Endurance		Resistance		Control		P
Index	Pre test	Pos test	Pre test	Pos test	Pre test	Pos test	-
Age (year)	22.4 ± 1.64	-	22.3 ± 1.41		22.77 ± 1.63	-	-
Height (cm)	160.40 ± 3.48	-	160.30 ± 5.41		159.60 ± 5.06	-	-
Weight (kg)	75.21 ± 2.86	72.62 ± 2.85	75.48 ± 1.63	73.53 ± 1.43	75.08 ± 1.40	75.37 ± 1.32	0.015
Body mass index (kg/m^2)	29.27 ± 1.70	28.78 ± 1.96	29.46 ± 2.04	28.78 ± 1.96	29.55 ± 1.92	29.66 ± 1.93	0.254
Fat percentage (%)	31.24 ± 1.40	27.75± 0.88	31.58 ± 1.94	28.60± 1.35	31.80 ± 1.57	31.96 ± 1.58	0.000
Vo_2max (ml/kg/min)	23.64 ± 2.24	29.83 ± 3.28	23.48 ± 2.30	27.56 ± 3.36	23.13 ± 2.49	23.03 ± 2.50	0.000
Adiponectin (µg/ml)	8.82 ± 0.17	10.81 ± 0.06	8.70 ± 0.18	10.31 ± 0.41	8.78 ± 0.19	8.75 ± 0.29	0.000

Data are expressed as mean and standard deviation

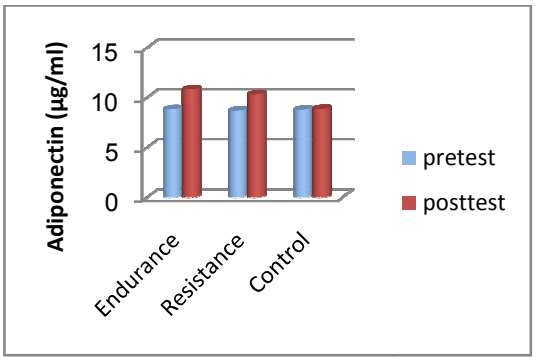

Fig 1. The pattern of changes in Adiponectin levels before and after 12 weeks exercise in there groups

Discussion

The results of the current study showed an increase in serum adiponectin levels among subjects who underwent 12 weeks of endurance and resistance training; with concurrent reduction in body weight and percent body fat, and that the high level of adiponectin is probably a proactive factor against the diseases related to adiponectin. The changes in the levels of adiponectin, body weight and percent body fat, as a result of adaptation to the endurance and resistance training in this study.

Previous studies examining the effects of exercise training on adiponectin levels have reported conflicting results. Some have reported increased (Kondo *et al*., 2006; Yatagai *et al*.,2003; Balagopal *et al*., 2005; Bluher rt al., 2006) and others have reported no changes in adiponectin levels after exercise training (Boudou *et al*., 2003; Nassis *et al*., 2005; Yokoyama *et al*., 2004; Ryan *et al*., 2003). Most studies that reported increased adiponectin levels after exercise training also observed significant weight loss (Kondo *et al*., 2006; Bluher *et al*., 2006; Oberbach *et al*., 2006; Esposito *et al*., 2003; Monziliio *et al*., 2003). Esposito *et al*, observed a 48% increase in adiponectin levels after 2 years of a combined low-energy Mediterranean diet and increased physical activity. A study also reported increased adiponectin levels in subject groups with normal glucose tolerance, impaired glucose tolerance, and type 2 diabetes after only 4 weeks of aerobic exercise intervention, which induced 2.0%, 3.7%, and 1.7% weight reduction, respectively (Bluher *et al*., 2006).

In addition, one of the recent studies showed that 3 months of aerobic exercise increased plasma adiponectin levels from 4.44 ± 0.47 to 5.95 ± 0.49 µg/mL, with a significant reduction in body fat mass without changes in body weight (Balagopal *et al*., 2005).

It seems that modifications in body weight or body composition might be responsible for alterations in adiponectin levels (Esposito *et al*., 2003; Monziliio *et al*., 2003). Recent reports indicate that in young obese men, adiponectin levels are increased following an improvement of the body composition and this is more important than the way training is performed (Hara *et al*., 2005). From these previous studies, we can speculate that weight loss, more specifically body fat loss, is necessary for the exercise training effects on adiponectin to be revealed. The present study examined the effects of continuous and intermittent training where there was evidence of body weight or body composition change and this could explain of modifications in adiponectin levels.

On the other hand, Yokoyama and corporation reported no changes in adiponectin levels after 3 weeks of combined intervention of diet and exercise, which induced slight weight loss among 40 patients with type 2 diabetes (Yokohama *et al*., 2004). In addition, Hulver *et al* also reported no changes in adiponectin levels despite significant increased insulin action and no changes in body weight or fat mass (Boudou *et al* 2003). There are also studies that show exercises to have no effect on the level of adiponectin. That may be because of using a combination of endurance and strength exercises (Hara *et al*., 2005; Klimcakova *et al*., 2006) or using athlete subjects who have higher adiponectin level in baseline or other unknown factors (Zelber *et al*., 2008).

Few resistance-training studies that have investigated changes in adiponectin have also reported conflicting results (Fatouros *et al*., 2005; Klimcakova *et al*., 2006; Brooks *et al*., 2006). Klimcakova *et al* reported no changes in adiponectin concentration in response to 3 months of resistance training performed three a week at 60–70% of 1-RM with 12–15 repetitions for each exercise (30–45 min per session).

In an interesting study, Fatouros *et al*, investigated the effects of 6 months of resistance training at different intensities (low, moderate and high intensities) on adiponectin concentration in elderly individuals. They reported significant increases in adiponectin after moderate- and high-intensity training but not after low-intensity training, which were accompanied by weight reductions. Recently, Brooks *et. a.l* demonstrated significant increases in adiponectin concentration after 14 weeks of high-intensity resistance training. From these previous studies, we can speculate that weight loss, more specifically body fat loss, is necessary for the exercise training effects on adiponectin to be revealed.

Conclusion

This study demonstrates that 12 weeks of endurance and resistance training improved body composition, Vo2 max, and adiponectin levels in sedentary young women. Increase levels of adiponectin can play an outstanding role in preventing metabolic and cardiovascular diseases. Therefore, suggest that sedentary young women should be encouraged to increase their physical activity levels to prevent early development of chronic diseases.

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