Available online at www.pelagiaresearchlibrary.com

Pelagia Research Library

European Journal of Experimental Biology, 2011, 1 (4):216-220

ISSN: 2248 –9215
CODEN (USA): EJEBAU

Adiponectin responses to continues and intermittent training in non-athlete obese women

Sohaily Shahram, Kamyabnia Masoomeh, Imanzadeh Reza, Faraji Gholamreza

Department of Physical Education and Sport Science, Shahre - e - Qods Branch, Islamic Azad University, Iran

---

ABSTRACT

Adipose tissue-secreted adiponectin play a key role in systemic inflammation. The purpose of this research was to determine the effect of continues and intermittent training on Adiponectin in non-athlete obese women. For this purpose, 30 obeys women subject (BMI ≥ 26) of university Shahre Qods were randomly assigned to three groups (continues training, intermittent 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≤ 0.05). Results showed that continue and intermittent training caused a significant increase in Adiponectin and Vo2max, significant decrease in weight and body composition of the experimental group in comparison to control group. This data suggest that physical training have positive effects on serum Adiponectin.

Keywords: Continues and Intermittent Training, Adiponectin, Obese Women

---

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” [1]. Adipokines included adiponectin, leptin, resistin, tumour necrosis factor alpha and interleukin [2, 3]. Adiponectin plays a significant role in metabolic disorders such as obesity, type 2 diabetes, coronary heart disease and metabolic syndrome [4, 5]. Plasma concentration of adiponectin is lower in subjects with obesity, type-2 diabetes mellitus, and coronary heart disease as compared to healthy controls [4, 6]. 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 [7]. 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 [8, 9]. Adiponectin plays a protective role against the development of atherosclerosis by suppressing inflammatory processes on the vascular endothelium [10, 11]. Although less is known about the association of adiponectin with the beneficial effects of exercise, several studies have examined whether exercise training affects plasma adiponectin concentrations. And the results are controversial. Adiponectin levels increased, decreased in healthy humans [12, 13].

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 [14, 15, 16]. Therefore, the present study was designed to determine and compare the effects of continues and intermittent training on adiponectin concentration in non-athlete obese women.

MATERIALS 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 ≥ 26 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 (Continues training, intermittent 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:

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

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 [17]. The subjects were advised to keep up their usual diet during the research period.

Over 12 weeks the subjects exercised 3 time a week with a specific intensity and distance. Karvonen heart rate reserve formula was used to determine the exercise intensity. The exercise intensity was controlled using a heartbeat monitor (Polar, made in Finland). A session of training program in intermittent 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 80 to 95% of their maximum heart rate reserve with the work to rest ratio of one to three (Table 1). They cooled off for five minutes.

Table 1 - Intermittent training programs

Week	1	2	3	4	5	6	7	8	9	10	11	12
Target heartbeat (percentage)	70-75%	70-75%	70-75%	70-75%	75-80%	75-80%	75-80%	75-80%	80-85%	80-85%	80-85%	80-85%
Distance (meter)	8× 200	8× 200	9× 200	9× 200	12× 200	12× 200	14× 200	14× 200	15× 200	15× 200	16× 200	16× 200

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 2). They cooled off for five minutes.

Table 2 – 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

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 3. After 12 weeks of Intermittent and continuous training adiponectin level (p= 0.000) (Diagram 1) showed a significant increase. This increase was between Continues and intermittent 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 mass index, 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 3).

Table 3- Pre-and post-test physical, physiological and biochemical variables and one way analysis of variance test in the three groups

Group	Intermittent		Continues		Control		P
Index	Pre test	Pos test	Pre test	Pos test	Pre test	Pos test	-
Age (year)	22.2 ± 1.68	-	22.4 ± 1.64		22.77 ± 3.06	-	-
Height (cm)	159 ± 1.88	-	160.80 ± 3.43		158.80 ± 3.99	-	-
Weight (kg)	75.21 ± 2.86	72.92 ± 2.44	75.01 ± 6.32	72.80 ± 5.85	75.08 ± 2.52	75.20 ± 2.49	0.007
Body mass index (kg/m^2)	29.8 ± 1.21	28.89 ± 1.22	29.13 ± 1.99	28.12 ± 0.89	30.17 ± 1.85	30.39 ± 1.74	0.004
Fat percentage (%)	30.92 ± 1.48	29.01± 1.03	31.26 ± 1.40	29.36± 1.02	31.80 ± 1.57	31.96 ± 1.65	0.000
Vo_2max (ml/kg/min)	23.64 ± 2.24	29.83 ± 3.28	23.48 ± 2.30	29.56 ± 3.36	23.13 ± 2.49	23.03 ± 2.50	0.000
Adiponectin (µg/ml)	8.86 ± 0.19	11.02 ± 24.45	8.90 ± 0.16	10.98 ± 0.24	8.78 ± 0.19	8.85 ± 0.29	0.000

Data are expressed as mean and standard deviation

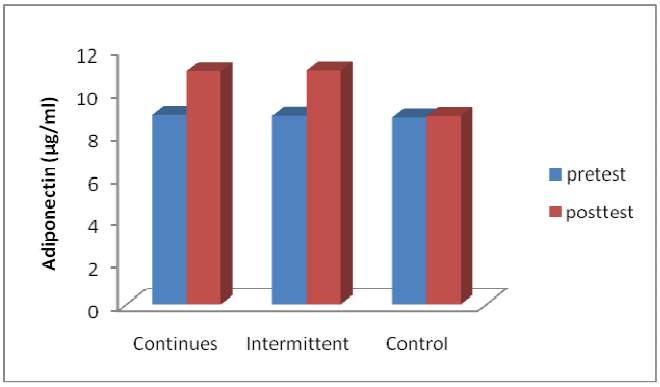

Diagram 1: The pattern of changes in Adiponectin levels before and after 12 weeks exercise in there groups

DISCUSSION

The results of the current study showed an increase in serum adiponectin levels among subjects who underwent 12 weeks of continuous and intermittent training, with concurrent reduction in body weight, percent body fat and BMI, 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, percent body fat and BMI, as a result of adaptation to the continuous and intermittent training in this study.

Previous studies examining the effects of exercise training on adiponectin levels have reported conflicting results. Some have reported increased [1, 13, 18, 19] and others have reported no changes in adiponectin levels after exercise training [15, 20, 21, 22]. Most studies that reported increased adiponectin levels after exercise training also observed significant weight loss [1, 19, 23, 24]. Esposito et al, observed a 48% increase in adiponectin levels after 2 years of a combined low-energy Mediterranean diet and increased physical activity [24]. 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 [19]. 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 [18].

It seems that modifications in body weight or body composition might be responsible for alterations in adiponectin levels [24, 25]. 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 [26]. 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 [21]. 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 [15]. 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 [26, 27] or using athlete subjects who have higher adiponectin level in baseline or other unknown factors [28].

CONCLUSION

This study demonstrates that 12 weeks of continuous and intermittent training improved body composition, Vo2 max, and adiponectin levels in non-athlete obese women. Ideal levels of adiponectin can play an outstanding role in preventing metabolic and cardiovascular diseases. Therefore, suggest that overweight and obese women should be encouraged to increase their physical activity levels to prevent early development of chronic diseases related to obesity.

REFERENCES

[1] Kondo T, Kobayashi I, Murakami M, *Endocr J*, **2006**, 53,189.
[2] Ronti T, Lupattelli G, Mannariono E, *Clin Endocrinol*, **2006**, 64, 355.
[3] Guzik T, Mangalat D, Korbut R, *J Physiol Pharmacol*, **2006**, 57, 505.
[4] Hotta T, Funahashi T, Arita Y, et al, *Arterioscler Thromb Vasc Biol*, **2000**, 20,1595.
[5] Ouchi N, Kihara S, Arita Y, et al, *Circulation*, **1999**, 100, 2473.
[6] Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley R, Tataranni P, *J Clin. Endocrinol Metab*, **2001**, 86, 1930.
[7] Yang W, Lee W, Funahashi T, Tanaka S, Matsuzawa Y, Chao C, Chen C, Tai T, Chuang L, *J Clin Endocrinol Metab*, **2001**, 86, 3815.
[8] Laukkanen J, Kurl S, Salonen J, *Current Atherosclerosis Reports*, **2002**, 4, 468.
[9] Thompson PD, Buchner D, Pina IL, et al, *Circulation*. **2003**, 107, 3109.
[10] Ouchi N, Kihara S, Arita Y, Maeda K, Kuriyama H, Okamoto Y, Hotta K, Nishida M, Takahashi M, Nakamura T, Yamashita S, Funahashi T, Matsuzawa Y, *Circulation*, **1999**, 100, 2473.
[11] Ouchi N, Kihara S, Arita Y, Nishida M, Matsuyama A, Okamoto Y, Ishigami M, Kuriyama H, Kishida K, Nishizawa H, Hotta K, Muraguchi M, Ohmoto Y, Yamashita S, Funahashi T, Matsuzawa Y, *Circulation*, **2001**, 103, 1057.
[12] Kriketos A, Gan S, Pounten A, Furler S, Chisholm D, Campbell L, *Diabetes Care*, **2004**, 27, 629.
[13] Yatagai T, Nishida Y, Nagasaka S, Nakamura T, Tokuyama K, Shindo M, Tanaka H, Ishibashi S, **2003**, *Endocrine J*, 50, 233.
[14] Hulver M, Zheng D, Tanner C, Houmard J, Kraus W, Slentz C, Sinha M, Pories W, MacDonald K, Dohm G, *American Journal of Physiology, Endocrinology and Metabolism*, **2002**, 283, 861.
[15] Boudou P, Sobngwi E, Mauvais-Jarvis F, Vexiau P, Gautier JF, *European Journal of Endocrinology*, **2003**, 149, 421.
[16] Kraemer R, Aboudehen K, Carruth A, Durand R, Acevedo E, Hebert E, Johnson L, Castracane V, *Medical Science and Sports Medicine*, **2003**, 35, 1320.
[17] Foster-Schubert K, McTiernan A, Frayo R, Schwartz R, Rajan K, Yasui Y, Tworoger S, Cummings D, *J Clin Endocrinol Metab*, **2005**, 90, 820.
[18] Balagopal P, George D, Yarandi H, et al, *J Clin Endocrinol Metab*, **2005**, 90, 6192.
[19] Bluher M, Bullen J, Lee J, et al. *J Clin Endocrinol Metab*, **2006**, 91, 2310.
[20] Nassis GP, Papantakou K, Skenderi K, et al, *Metabolism*, **2005**, 54, 1472.
[21] Yokoyama H, Emoto M, Araki T, et al, *Diabetes Care*, **2004**, 27, 1756.
[22] Ryan A, Nicklas B, Berman D, et al, *Int J Obes Relat Metab Disord*, **2003**, 27, 1066.
[23] Oberbach A, Tonjes A, Kloting N, et al, *Eur J Endocrinol*, **2006**, 154, 577.
[24] Esposito K, Pontillo A, Di Palo C, et al, *JAMA*, **2003**, 289, 1799.

[25] Monzillo L, Hamdy O, Horton E, Ledbury S, Mullooly C, Jarema C, Porter S, Ovalle K, Moussa A, Mantzoros C, *Obes Res*, **2003**, 11, 1048.
[26] Hara T, Fujiwara H, Nakao H, Mimura T, Yoshikawa T, Fujimoto S, *Eur J Appl Physiol*, **2005**, 94, 520.
[27] Klimcakova E, Polak J, Moro C, Hejnova J, Majercik M, Viguerie N, et al, *J Clin Endocrinol Metab*, **2006**, 91, 5107.
[28] Zelber-Sagi S, Nitzan-Kaluski D, Goldsmith R, Webb M, Zvibel I, Goldiner I, et al, *Hepatology*, **2008**, 48, 1791.