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Adiponectin and insulin resistance responses to aerobic training in males with abdominal obesity

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Abstract

Accumulating evidence support the adiponectin role in genesis of obesity and insulin resistance. The objective of this study was to evaluate effect a chronic aerobic exercise on serum adiponectin and insulin resistance in obese men. For this purpose, at baseline and after 12 weeks of aerobic exercise training, blood samples were taken in order to measuring serum adiponectin, glucose, and insulin in twenty eight in sedentary obese healthy males that divided to exercise (60 min, 3 days/week for 12 weeks, n=14) and control (no training. n=14) groups matched for age and BMI by randomly. Insulin resistance was assessed using the homeostasis model assessment for insulin resistance formula derived from fasting insulin and glucose levels. Pre- and post exercise independent variables were compared using a paired-samples t-test. Significance was accepted at $P < 0.05$. No significant differences were found in serum adiponectin and insulin resistance at the end of the 3-month exercise program with compared to baseline ($p \geq 0.05$). Fasting glucose concentrations were significantly decreased by aerobic training program in exercise group ($P=0.01$). Despite to some previous study, in present study, we conclude that aerobic exercise training is not associated with improve in adiponectin or insulin resistance. Decreased glucose concentration is likely due to the other exercise induced hormonal changes.

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Introduction

In addition to a main source of triglyceride, adipose tissue functions as an endocrine organ (Pajvani *et al.,* 2003; Sell *et al.,* 2006), thermal insulation, and mechanical protection, releasing biologically active and diverse cytokines, termed adipokines (Pajvani *et al.,* 2003; Aldhahi *et al.,* 2003). Adiponectin, as anti-inflammatory cytokine, is a 30-kDa protein that is predominantly expressed in adipocytes (Maeda *et al.,* 1996; Scherer *et al.,* 1995) and circulating adiponectin concentrations are high in both humans and rodents varying from 2 to 30 mg/ml (Arita *et al.,* 1999). This anti-inflammatory cytokine, first identified in 1995 (Scherer *et al.,* 1995) and also known as AdipoQ and ACRP30, is a serum protein hormone secreted from white adipose tissue into the circulation, where it is found in high concentrations (Tsao *et al.,* 2002; Kim *et al.,* 2006). Review of research evidence shows that adiponectin plays a important role in metabolic disorders, such as obesity, type 2 diabetes, coronary heart disease, and metabolic syndrome (Salmenniemi *et al.,* 2004) due to its insulin sensitizing (Lara-Castro *et al.,* 2006), anti-infammatory, and anti-atherogenic properties (Ajuwon *et al*., 2005).

There is considerable evidence that Circulating levels of adiponectin, which promotes glucose uptake into skeletal muscle and increases fat oxidation rates, are decreased in obesity (Vivian *et al.,* 2007). Strategies to enhance the insulin-like and insulin-sensitizing actions of adiponectin have been shown to be effective in improving metabolic abnormalities associated with obesity and diabetes (Vivian *et al.,* 2007). Some previous study suggested that adiponectin is associated with insulin resistance in obesity or obesity related diseases (Meilleur *et al.,* 2010; Peti *et al.,* 2010).

Difference exercise protocols such as treadmill running and cycling have been used to measure the changes in adiponectin levels in healthy and diseases human subjects. Exercise training causes an increase in insulin sensitivity and some authors have hypothesized that this may be due to increased adiponectin secretion or sensitivity upon training (Vivian *et al.,* 2007). Of course, it was found that changes in total adiponectin levels were dependent on the intensity and duration of the training protocol (Zeng *et al.,* 2007). There is considerable evidence that exercise training is accompanied with increased serum adiponectin and decreased insulin resistance (Tang *et al.,* 2005; de Salles *et al.,* 2010). But, some previous studies showed no change in serum adiponectin in response to endurance exercise training in adolescent or middle-aged individuals (Hulver *et al.,* 2002; Kobayashi *et al.,* 2006; Rosenbaum *et al*., 2007). On the other hand, in recent study, a 6-week endurance-training program in non-obese healthy males decreased serum adiponectin and restored to basal levels 1-week following the last training session (Yatagai *et al.,* 2003). Reviewing the research findings indicates contradiction in the findings regarding adiponectin response to exercise training for short or long time and there is still no general consensus in these area. Therefore, the main objective of present study is to evaluate the effect of an aerobic exercise program on serum adiponectin as well as insulin resistance in adult obese men.

Subjects and methods

Subjects were aged 35–45 years, sedentary, obese (BMI 30–35 kg/m2) and divided randomly into exercise group (60 min, 3 days/week for 12 weeks, n=14) and control groups (no training, n=14). Main objective of this experimental study was to determine adiponectin and insulin resistance responses to an aerobic training program in obese males. Approval for the original study had been given in 2011 by the Ethics Committees of Islamic Azad University, Iran. Each participant received written and verbal explanations about the nature of the study before signing an informed consent form.

Inclusion or exclusion criteria

Participants were included if they had not been involved in regular physical activity in the previous 6

months. Exclusion criteria included medications that alter carbohydrate metabolism, diabetes, inability to exercise, and history of hypertension or heart disease. Subjects had neither used any medication 6 weeks prior to the study nor participated in any regular diet. Presence of previous coronary cardiac disease, chronic airway disease, and impaired hepatic dysfunction, diabetic and presence of any acute disease and having symptoms that may be indicative of ischemia in electrocardiography were determined as exclusion criteria.

Anthropometrical measurements

Weight and height of the participants were measured by the same person when the participant had thin clothes on and was wearing no shoes by using the standard hospital scales. Body composition monitor (BF508-Omron made in Finland) with a precision error of less than 100 g was used to measure weight and body fat percentage of the subjects. Body Mass index (BMI) was calculated using the formula body weight/height2 in terms of kg/m². Abdominal circumference was measured in the most condensed part using a non-elastic cloth meter.

Blood sampling and exercise program

After anthropometrical measurements the individuals in the experimental and control groups were asked to attend Hematology Lab following 12 hours of overnight fasting, between the hours of 8 to 9 am for blood sampling. Blood samples were taken after a rest of 20 minutes (pre-test). Exercise training program lasted 3 months (3 days/wk) 60 to 80 percent of maximum heart rate. Each session started by 15 min of flexibility exercises, 30-40 min of aerobic exercise and 5–10 min of cool down activity. Aerobic exercises in each session included walking on a treadmill and stationary cycling. Initially, subjects exercised at low intensity and the intensity of exercise was gradually increased to 80% of peak heart rate in next sessions. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US). In this 12-week period, participants in the control group were barred from participating in any exercise training. Finally, all measurements consist of fasting blood sampling, anthropometric measurements and Blood pressure repeated 48 h after last exercise session.

Assay

After sampling in ETDA- or serum-tubes, blood was immediately chilled on ice, centrifuged and aliquots were frozen at – 80°C until assayed. Blood samples were analyzed for glucose, insulin and serum adiponectin. Glucose was determined by the oxidase method (Pars Azmoon kit, Tehran) with COBAS MIRA from Roche (Lörrach, Germany). Plasma insulin was determined by ELISA method (Demedite, German). The Intra- assay coefficient of variation and sensitivity of the method were 2.6% and 2.88 µg/L, respectively. Adiponectin concentrations were measured by immunosorbent assay (ELISA; Biovendor, Czech) (Intra-assay CV: 5.9%; Inter-assay CV: 6.3%).

Statistical analysis

All values are represented as mean ± SD. Statistic analysis was done with SPSS 15.0 for Windows. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Baseline characteristics were compared by using independent t-tests. Student's paired 't' test was applied to compare the pre and post training values. All statistical tests were performed and considered significant at a $P < 0.05$.

Results

Baseline and post training adiponectin levels, anthropometrical indexes and clinical characteristics of two groups are shown in Table 1. Data were expressed as individual values or the mean ± SD. At baseline, there were no differences in serum adiponectin between the two groups (p=0.211) (see Table 1). Fasting glucose, insulin and insulin resistance levels in exercise group were did not differ from control group (p>0.05). Also, significant differences were not found in body weight, body fat

percentage, BMI and abdominal circumference

Table 1. Mean and standard deviation of anthropometrical and biochemical variables of two studied groups in baseline and after intervention.

Variables	Exercise group		Control group	
	Pretest	post-test	Pretest	post-test
Age (year)	39 ± 6	39 ± 6	40 ± 5	40 ± 5
Height (cm)	177.5 ± 9.1	177.5 ± 9.1	176.4 ± 7.6	176.4 ± 7.6
Weight (kg)	102.25 ±11.2	96.5 ± 13*	103.2 ± 9.4	103.6 ± 11.3
Abdominal circumference (cm)	109.6 ± 11.5	104 ± 12.1*	108.9 ± 9.6	109 ± 10.3
Hip circumference (cm)	108.1 ± 9.7	104 ± 13*	107.5 ± 9.4	108 ± 11.2
Waist to hip ratio	1.01 ± 0.28	1 ± 0.19*	1.01 ± 0.12	1.01 ± 0.13
BMI (kg/m2)	32.46 ± 3.23	30.63 ± 2.44*	33.18 ± 2.36	33.31 ± 3.12
Body fat (%)	32.5 ± 3.11	27.3 ± 4.8*	33.1 ± 3.65	32.9 ± 2.65
Glucose (mg/dl)	101 ± 14	86 ± 11*	103 ± 12	105 ± 9
Insulin (µIU/ml)	8.01 ± 2.11	9.75 ± 2.23	9.01 ± 2.52	8.14 ± 2.65
Insulin Resistance	2.07 ± 0.33	1.94 ± 0.28	2.13 ± 0.36	2.06 ± 0.41
Adiponectin (ng/ml)	6.5 ± 1.34	7.01 ± 1.68	6.84 ± 1.65	6.94 ± 1.84

between two groups at baseline ($p>0.05$).

With aerobic exercise training, subjects in exercise group lost fat mass seen as a decrease in percent body fat, BMI, body weight and abdominal circumference ($p<0.05$). No differences in serum adiponectin concentrations were observed after aerobic exercise program compared to their respective baseline values in exercise group ($p=0.165$). Also, insulin resistance did not change with long-term exercise training in exercise group ($p=0.214$). Compared to pre-training, fasting glucose levels decreased significantly ($P=0.01$) after aerobic exercise program in the exercise subjects ($p=0.021$) but not in the control subjects ($p=0.235$). All variables remained without change in control group ($p<0.05$).

Discussion

The major finding of this investigation was that Fasting serum adiponectin and insulin resistance did not change with long-term exercise training while all anthropometrical indexes decreased significantly in all subjects in exercise group. It is generally accepted that adipocytes, particularly those located within the visceral fat, aremajor secretors of both pro and anti-inflammatory factors, often referred to as adipokines (Havel, 2002). Increased visceral fat affects insulin sensitivity and energy metabolism by releasing adipokines into the blood circulation. So with the absorption of these cytokines by target tissue, fat and carbohydrate metabolism is affected (Vivian *et al.,* 2007). Serum adiponectin as an anti-inflammatory adipokine decreases in the presence of obesity that is associated with decreased absorption of muscle glucose and fat oxidation (Vivian *et al.,* 2007). The insulin-like function of this 244-amino-acid peptide hormone (Maeda *et al.,* 1996; Havel, 2002) has shown that the reduction of its plasma concentration is effective on obesity and its related diseases like diabetes II (Vivian *et al.,* 2007). Decreased adiponectin concentration in obese individuals compared with people with normal weight as well as insulin resistant populations has been frequently observed (Hotta *et al.,* 2000; Weyer *et al.,* 2001).

Adiponectin has known direct and indirect functions primarily related to endothelial function, the

promotion of insulin sensitivity and inhibition of inflammatory mediators (Berg *et al.,* 2005). Full-length adiponectin acts with insulin to inhibit hepatic glucose production whereas the globular domain stimulates fatty acid oxidation in skeletal muscle (Fruebis *et al.,* 2001), with receptor expression up-regulated in obesity, impaired glucose tolerance, and type 2 diabetes (Bluher *et al.,* 2006).
It was reported that plasma adiponectin levels of 5–25 μg/ml had significant inhibitory effects on tumor necrosis factor-α–induced monocyte adhesion and adhesion molecule expression, suggesting an increased risk of adverse health effects at serum concentrations below this level (Ouchi *et al.,* 1999). In accordance with these observations, circulating adiponectin levels in normal subjects has been reported as 5–20 μg/ml (Matsuzawa, 2005). Te primary characteristic found to be closely related with circulating adiponectin concentration in humans is level of obesity. Other adipokines are generally secreted at above normal levels in obesity (Arita *et al.,* 1999).

Despite this information, there is conflicting evidence about adiponectin response to exercise training. Review of research findings show that adiponectin levels increased (Kriketos *et al.,* 2004), decreased (Yatagai *et al.,* 2003), or remained unaltered (Hulver *et al.,* 2002) in healthy humans, whereas they increased in patients with cardiovascular or metabolic diseases after aerobic exercise training (Hotta *et al.,* 2000; Ishii *et al.,* 2002). Our study clearly showed that serum adiponectin did not affect by 12 week aerobic exercise in obese men. In the light of these observations, Boudou et al. and Yokoyama et al. found no signigcant changes in total adiponectin levels with 8- and 3-weeks (respectively) of endurance training that facilitated fat loss in T2DM subjects (Yokoyama *et al.,* 2004). Additionally, in a recent study, no signigcant changes in total and individual multimeric adiponectin levels were observed in 21 subjects who lost weight during 12-weeks of aerobic exercise training with and without caloric restriction (O'Leary *et al.,* 2007).

Although exercise in combination with weight loss diets has been shown to increase adiponectin levels (Bruun *et al.,* 2006), it is not clear whether exercise in the absence of weight loss or dietary interventions can modify adiponectin levels. Therefore, it is most likely that a lack increase in adiponectin levels or insulin resistance in our study to be due to Lack of Control diet during the training program.

On the other hand, although serum adiponectin did not change by chronic aerobic training in our study, it is likely that the anti-inflammatory effect of exercise is associated with increased gene expression and adiponectin receptors. In line with this hypothesis, in Huang study, although exercise did not significantly change plasma adiponectin concentration at 2 hours or 18 hours after the exercise, the expression levels of adiponectin receptors (AdipoR1) significantly increased in both skeletal muscle and liver compared with control group (Huang *et al.,* 2007). In this area, it seems that the measuring of inflammation cytokines alone does not directly account for the beneficial effects of physical activity.

Another possible explanation for lack increase in adiponectin levels during exercise may be the result of concomitant increases in other cytokines and hormones that are known to reduce adiponectin secretion. For example, glucocorticoids, TNF-alpha, and IL-6 can down-regulate the expression of adiponectin (Fasshauer *et al.,* 2003) and all of these may be elevated during prolonged exercise (Pedersen *et al.,* 2001). It has been shown that glucocortocoids (GC) inhibit adiponectin expression in vitro (Shi *et al.,* 2010).

Fasting glucose concentration was significantly decreased by aerobic exercise program in exercise group when compared with baseline levels. Although we did not observe any change in serum adiponectin and

insulin resistance, recent findings also suggest improvements in insulin sensitivity may occur acutely and independently of changes in adiponectin concentrations (Simpson *et al.,* 2008). In this area, there are studies in the literature reporting an unchanged adiponectin response despite a training-induced enhancement of insulin action (Hulver *et al.,* 2002; Boudou *et al.,* 2003). It seems that decreased glucose level by exercise training in our study to be due to improved insulin sensitivity. It should also be noted that lack measuring of insulin sensitivity is a main limitation of our study.

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