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The effect of eight weeks of aerobic training on hsCRP and resistin levels in menopause women

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ABSTRACT

CRP and resistin are two inflammatory markers of atherosclerosis. The aim of this study was to examine the effects of eight weeks aerobic training on concentrations of hsCRP and resistin in Menopause women. 30 volunteered Menopause women randomly assigned into experimental and control groups (each group 15 subjects). Subjects of experimental group accomplished eight weeks (3 sessions per week) aerobic exercise with intensity of 50 to 60 percent of their maximum heart rate. Before and after trainings, weight, body fat percent, hsCRP and resistin levels of subjects were determined. In compare with control group, significant reduction of weight, BMI, body fat percent and hsCRP ($P<0.001$) and significant elevation of resistin ($P<0.05$) in experimental group has been showed . Eight weeks regular aerobic training caused reduction of weight, body fat and hsCRP and elevation of resistin leads to reduction of risk of cardiovascular disease and improvement of health status in Menopause women.

Keywords: aerobic training, weight loss, hsCRP, body fat percentage, resistin.

INTRODUCTION

Low-mobility lifestyle is a problem facing both developed and developing countries. One of the side effects of this problem is increased cardiovascular diseases and premature mortality. In most cases, premature coronary artery disease has a direct relationship with the number and intensity of atherosclerosis risk factors [1].

It was observed that half of the heart attacks occur to the people without hyperlipidemia [2]. A prospective study conducted on American women, reported 77% of the future cardiovascular diseases among women with low LDL [3]. Also, according to a study conducted on 120000 patients suffering from coronary artery disease, 19% of men and 15% of women showed no symptom of hyperlipidemia, hypertension, diabetes and smoking, and that over 50% of the subject showed only one of these symptoms [4].

The animal-related, clinical and epidemiological research conducted within 10 to 15 years showed that inflammation and its cellular and molecular mechanisms play an important role in atherogenesis processes. Three important inflammatory markers are CRP, fibrinogen and resistin, which cause tissue damage and infection and atherosclerosis development [5-7].

CRP is an acute phase protein whose increase results in an increase in the possibility of the coronary artery diseases by 2 to 5 times. in the people with high fat, CRP levels are high and has a direct relationship with the insulin sensitivity and type 2 diabetes mellitus [8]. CRP causes the development of atherosclerosis through the following mechanisms: 1) by binding to the phospholipids of damaged cells and increasing the consumption of these cells by macrophages;

2) by activating endothelial cells for the gene expression of adhesive molecules; 3) by reducing the gene expression of endothelial nitric oxide synthase [9-11].

Resistin is a cysteine rich peptide hormone which has 108 amino acids. In the obese patients with diabetes, the level of this hormone is high. In human beings, this hormone is mainly made in fat and inflammatory cells and is directly related to hsCRP levels and atherosclerosis risk [6, 12].

Resistin increases atherosclerosis risk by impairing glucose and lipid metabolisms. It also increases the vulnerability of atherosclerosis plaques by stimulating proinflammatory cytokines [13]. Resistin causes lipid deposition by increasing the gene CD36 expression in macrophages and forming foam cells in the vascular walls [14]. Church et al (2002) studied the relationship between CRP and cardio respiratory fitness and found that these two variables are negatively related [15].

Little research has been done on the effect of exercise on resistin level. Jamurtas et al (2006) reported that one session of aerobic exercise less than maximum intensity by healthy and overweight men will not result in a meaningful change in adiponectin and resistin levels until 48 hours after the exercise [16]. Jones et al (2010) claimed that 8 months of regular aerobic exercise can reduce resistin levels in overweight teenagers [17].

Considering these contradictory findings, and since the positive effect of aerobic exercise on the prevention of cardiovascular diseases and insulin resistance has been proved, and that exercise can be used as a therapeutic approach for the treatment of diabetes and atherosclerosis, research on the effect of regular aerobic exercise on resistin levels can produce new and interesting results. On the other hand, there has been no research on the long-term effect of aerobic exercise on resistin and CRP levels in Menopause Iranian women.

MATERIALS AND METHODS

Training protocol

Written consent was obtained from 30 volunteer Menopause women, healthy, sedentary with overweight, who were divided randomly (using simple random sampling) into two groups: control and experimental (each consisting of 15 members). The exercise program consisted of eight weeks of aerobic training (3 sessions per week) with 50% to 60% maximum heart rate of the subjects. The maximum heart rate of each subject was measured using the formula: 220 minus age of the subject. Using the polar chest belt, the heart rate of the subjects was controlled. Each session included ten minutes of warm-up with joint rotations, stretching and jumping movements, 15 minutes aerobic exercise and running, taking in the first session, rising to 35 minutes in the last session, was followed with ten minutes of cooling down along with stretching activities.

Blood samples

Before and after the first and last sessions of training, the percent of body fat, resistin and CRP levels of the subjects were measured. The body fat percentage was measured using Dale Wagner three skin fold thickness. Blood samples were taken 48 hours prior to first session and after last session of training from the left brachial veins and was placed in two tubes containing sodium citrate and EDTA, then sent to the laboratory for analysis, where after plasma and serum separation, they were kept at the -20 centigrade. Before blood sampling, subjects were recommended to avoid any intense physical activity as well as eating and smoking During eight weeks of training. Serum resistin was measured using Human Resistin Elisa Kit with Sandwich Elisa method. The method used for measuring hsCRP was strengthened immune-boturbidimetric for measuring two points with a photometer. In this method, the CRP existing in the sample of the subject forms a complex with the antibody of the sensitized polyclonal against human CRP coded on the latex particles and creates opacity. The amount of opacity created is related to the amount of CRP existing in the sample of the subject. The kit used was made by the Iranian Pars Azmoun Company. The kit has been designed to measure CRP within 0.1 to 20 milligram per liter and the minimum hsCRP it can measure is 0.1 milligram per liter.

Statistical analysis

The statistical analysis included descriptive statistics (measuring standard deviation and mean) and inferential statistics. One sample Colmogorov Smirnov test was used to measure the normality of the distribution of the data.

After it was made sure that the distributions of all the data were normal, paired samples T test was used to compare intra-group results and independent samples T test was used to examine the intergroup results. The significant level was P<0.05.

RESULTS

The subjects were homogeneoused in age, height, weight, BMI and percentage of body fat (See Table 1).

Table 1. descriptive characteristics of the subjects and differences among them before trainings

Age	54.4± 3.5	52.4± 4.2	0.44
Weight	76.08 ± 6.7	78.1 ± 6.1	0.55
BMI	27.39 ± 2.3	28.9 ± 3.3	0.16
Body Fat Percent	31.17 ± 2.8	31.19 ± 4.2	0.10

Data are expressed as mean ±SD.

The results of the independent T test showed that in the experimental group compare with control group, weight, BMI and percentage of body fat have been reduced significantly . Also, the amount of hsCRP of plasma showed a significant reduction while the amount of resistin of the serum showed a significant elevation. None of the mentioned variables in the control group showed a significant change (p<0.05) (See Table 2).

Table 2. changes of dependent variables in response to 8 weeks aerobic training

	Pre test	Post test	P-value	Pre test	Post test	P-value	
Weight	76.08±6.7	75.01±6.6	0.001	78.1±6	78.2±6.1	0.37	0.001
BMI	27.3±2.3	27.01±2.2	0.001	28.91±3.3	28.94±3.3	0.36	0.001
BFP	31.1±2.8	29.2±2.5	0.001	31.19±4	31.39±4.1	0.33	0.001
hsCRP	2.68±0.3	2.27±0.3	0.047	2.77±0.2	2.74±0.2	0.28	0.001
Resistin	5.75±0.3	5.98±0.3	0.092	5.76±0.4	5.78±0.3	0.63	0.041

Data are expressed as mean ±SD.

The independent T-test showed that the reduction of weight, BMI and percentage of body fat in the subjects of the experimental group was significantly higher than those at the control group. Also, the reduction of hsCRP and elevation of resistin in the subjects of the experimental group was significantly higher than those in the subjects of the control group (See Table 2).

DISCUSSION

The results of the study showed a significant reduction of hsCRP in experimental group. This finding was similar to that of Hamedinia et al (2009). They reported a significant reduction of CRP levels in 24 healthy and sedentary middle-aged men [8]. Salesi et al (2007) studied the effect of the type of exercise and estrogen on CRP levels and blood lipids. They showed an insignificant drop in the CRP levels [18].

Gaeini et al (2008) chose 64 female Wistar rats of 21 months old and divided them into four groups of young, fat, young thin and old fat, to examine the effect of 12 weeks of interval aerobic exercise on their hsCRP levels. They results showed reduction of hsCRP [19]. Dabidi Roshan et al (2009) examined the effect of 12 weeks of interval aerobic exercise, on the hsCRP level of rats. They believed that Physical training have a great role in reducing inflammation and preventing the cardiovascular disease [20].

Daray (2009) studied the effect of endurance and concurrent exercise (endurance + resistance) on the CRP level. They selected 58 men and women aged between 18 to 24, and showed a significant reduction in the CRP level of the concurrent exercise group [21]. CRP is directly related to obesity, BMI, WHR and waist circumference. Weight loss is an effective way to reduce CRP. Every kilogram of weight loss reduces 13% milligram/liter of CRP. Fat cells produce IL-6, which stimulates the liver to produce CRP. Therefore, weight loss coupled with fat loss and IL-6 reduction result in a reduction of CRP [22]. The study also showed that the BMI in the experimental group reduced significantly, which could have been responsible for the reduction of hsCRP.

In obese people with insulin resistance, the CRP concentration is high. In such people, the CRP concentration lowers as they lose weight [23] .physical exercise is directly related to insulin sensitivity, a factor reversely related to CRP. In sum, regular physical activities reduces CRP by reducing the production of cytokines from fat tissues, skeletal muscles, endothelium and blood mononuclear cells, improving endothelial function, increasing antioxidant effects, reducing fat and leptin and increasing adiponectin expression [24].

The resistin levels of the subjects of the study showed a significant increase at the end of the experiment. Even though no experiment was found on Menopause women, the findings of this study are consistent with those of Monzillo et al (2003), Kelly et al (2007), Camera et al (2010), Perseghin et al (2006) [25-28] But they were inconsistent with the findings of Balducci et al (2010), Kadoglou et al (2007), Elloumi et al (2009), Jones et al (2009), Zelber-Sagi et al (2008) [17, 29, 30-32].

Jones et al (2009) examined the effect of 8 months of aerobic exercise on serum lipid levels, leptin, adiponectin, resistin, peptide YY and ghrelin in overweight youngsters. They reported a significant reduction of resistin [17]. Kadoglou et al (2007) examined the effect of 16 weeks of aerobic exercise with an intensity of 50 to 70% Vo2max on the resistin levels in overweight type 2 diabetes patients and showed a significant reduction of this hormone in them [29]. Zelber-Sagi et al (2008) showed that in the patients with nonalcoholic fatty liver disease, resistance exercise (at least one session per week) and resistin levels are significantly and negatively correlated [30]. Elloumi et al (2009) studied obese youngsters and reported that their subjects showed a significant reduction of resistin levels after two months of exercise [32]. Balducci et al (2010) reported a reduction of resistin in the patients with diabetes and overweight after 12 months of physical activity [31]. Monzillo et al (2003) showed that 6 months of physical activity with an average intensity causes resistin elevation in healthy people and type 2 diabetes patients [27]. Kelly et al (2007) studied overweight children in an eight-week exercise program of 50 to 60 percent of Vo_2max. Resistin levels showed an insignificant elevation [28]. Camera et al (2010) showed that ten days of duration exercise increase resistin gene expression in sedentary young men [26]. Perseghin et al (2010) showed that endurance athletes had higher resistin levels than sedentary people [25].

Thus, none of the studies mentioned above had any resemblance to this study in terms of type, intensity, exercise period or subjects. Therefore, no comparison can be made between the findings of this study with those of others, even though it can be inferred that regular duration exercise with an average intensity can cause elevation of resistin levels.

The mechanisms of resistin increase in the subjects of the study are not exactly clear, but a review of the studies can suggests a few possible mechanisms. One possible mechanism is that Since resistin is directly linked with adiponectin [33] , adiponectin elevation as a result of exercises [27] can increase resistin. In this study, the most important mechanism explaining resistin elevation after aerobic exercise is the role of this hormone in the anti-oxidant defense of the body, since resistin is negatively correlated with nitrotyrosin [34].

Reactive nitrogen species are among important regulators of inflammation in the body. In response to inflammatory stimulant, resistin acts as antioxidant, a meaningful interaction has been discovered between polymorphism of a single nucleotide in the promoter of human resistin gene and an oxidant marker and insulin resistance. Mononuclear cells of blood produce resistin in response to low grade inflammation, which can have antioxidant properties. Bo et al (2005) found no significant relationship between CRP and NT, which indicates that there is a complicated interaction between oxidant markers and inflammatory markers [34].

Thus the hsCRP reduction and resistin elevation of in the subjects of this study can be the result of the anti-inflammatory and anti-oxidant compatibility. Generally, it can be deduced that regular aerobic training coupled with weight loss by reducing adipocytes such as leptin and IL-6 reduces hsCRP levels while it increases antioxidant defense of the body by stimulating the synthesis of resistin in mononuclear cells.

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