The effect moderate exercise on the plasma level of C-reactive protein, leptin, adiponectin and resistin and their relationship with body fat percentage in un-trained males

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Abstract

Purpose: aim of present study was investigating the effects of 16-weeks of aerobic training program on plasma levels of C-reactive protein (CRP), adiponectin, leptin and resistin in healthy un-trained men. Methods: for this purpose, twenty sexes healthy men were selected and depended on their body fat percentage were assigned in two equal groups. Experimental group performed 16-weeks aerobic training 3 days a week in alteration days with 65-85 percentages of maximum heart rate and control group remained sedentary. Blood samples were collected prior to and after training program for all subjects were measured. A P-value < 0.05 was considered to be statistically significant. Results: our results showed a significant increase in adiponectin and resistin concentrations, also a decrease in leptin concentrations and did not affect CRP levels following 16-weeks of aerobic training program (p<0.05). in addition, reduction in body fat percentage negatively correlated with adiponectin and resistin levels and positively correlated with leptin levels and non-correlation with CRP levels (p<0.05). Conclusion: in healthy un-trained men, 16-weeks of aerobic training reduced body fat percentage and improved plasma adipokines levels, so aerobic training can considered as major strategies for preventing obesity and associated diseases.

Key words: moderate exercise, plasma level, C-reactive protein, leptin, adiponectin, resistin

Introduction

Biologically, fat tissue is more than energy storage, it is rather an active tissue-releasing protein such as adiponectin and leptin (Elloumi et al., 2009), while other cytokines, such as CRP and resistin, are less released at fat tissues, and are mainly synthesized in immune cells (Reilly et al., 2005). These hormones have autocrine and paracrine effects on the brain, liver, and skeletal muscles. Also they play a crucial role in the diseasescaused by obesity through regulating metabolic and inflammatory processes (Kershaw et al., 2004).

C-reactive protein (CRP) is a nonspecific acute-phase protein secreted by the liver that is considered a surrogate marker of chronic low-grade systemic inflammation.

CRP is positively associated with body fat (Mora et al., 2006) and negatively associated with physical activity (Pitsavos et al., 2003) and cardiorespiratory fitness (Kuo et al., 2007) in observational studies. It is not clear whether improvements in cardiorespiratory fitness reduce CRP and whether such an effect is mediated entirely by a reduction in body fat.

One possible approach for improving levels of CRP maybe aerobic exercise, a low-cost, nonpharmacologic intervention that is available to most of the general public. For example, a recent qualitative systematic review that examined studies dealing with physical activity and CRP concluded that habitual physical activity results in lower levels of CRP (Kasapis and Thompson, 2005).

Adiponectin has anti-inflammation, anti-atherosclerosis and anti-insulin resistance effects (Hojlund et al., 2006). Adiponectin level has a negative correlation with body fat percentage, central fat distribution and leptin...
Patients suffering from diabetes, high blood pressure and ischemic heart disease have less adiponectin concentration compared to healthy people (Matsuzawa et al., 2004). Within four years of study, subjects with hypoadiponectinemia emaiadied because of heart diseases more than normal people. Hence, hypoadiponectinemia is introduced as the new cardiac risk factor (Zoccali et al., 2002).

Resistin is a hormone belonging to the family of cysteine-rich proteins identified as one of the symptoms of atherosclerosis and is one of the important predicting factors of cardiovascular diseases (Kadoglou et al., 2007). The increase in the level of resistin mostly happens in inflammatory conditions and it is shown that resistin stimulates the synthesis and the release of pre-inflammatory cytokines (Silswal et al., 2005). Reilly et al. have reported that in subjects suffering from metabolic syndrome, the level of resistin in plasma is predictor for coronary atherosclerosis (Reilly et al., 2005).

Leptin is identified to be of the first adipokines related to the body fat mass and the loss of weight and fat percentage is often accompanied by a decrease in the leptin levels (Koerner et al., 2005). An increase in the level of leptin is observed in cardiovascular, diabetes and even asthmatic patients (Sood et al., 2006).

Adipose tissue is the major source of leptin expression; however, other sites have been identified, including skeletal muscle, liver, stomach, heart, mammary epithelium, the placenta, and the brain (Bouassida et al., 2006). Leptin's actions is on energy balance (Ishii et al., 2001), and where of exercise is an effective way to reduce obesity (fat mass).

This study investigated the effects of a 16-weeks aerobic exercise intervention on CRP, adiponectin, leptin and resistin in 26 sedentary adults. In order to find out the levels of the hormones of CRP, adiponectin, leptin and resistin were measured prior to and after executing exercise program, and their relationship with fat percentage was calculated.

Materials and Methods

Subjects

Twenty four healthy-untrained males volunteered to participate in this investigation. Before initiation of the study, subjects were asked to sign a written, informed consent. All subjects completed a medical questionnaire to ensure that they were not taking any medication, were free of cardiac, respiratory, renal, or metabolic diseases, and were not using steroids.

Methods

All subjects randomly divided into 2 equal groups: Group 1: Control group (n=13), Group 2: Experimental group (n=13). 48 h before starting the aerobic training program Weight, Height, body fat, Blood samples and BMI were taken from all the subjects using proper devices/methods: weight by scale, height by measuring-tape, body mass index (BMI) by the ratio of weight square root of height (Table 1). Then, experimental group performed the aerobic training program included running with 65-85% of individual maximum heart rate on treadmill for 3 sessions per week, 30 minute persession for 16 consecutive weeks. Then another Weight, body fat, Blood sample and BMI was taken in the of the training period. Serum levels of CRP, adiponectin, leptin and resistin were measured prior to and after the training period were measured using standard biochemical methods from all the subjects in both groups again. Fat percentage of the subjects was measured using caliper device by the method of three skinfold thickness (subcapular, abdominal, and triceps) (Wagner, 1996).

<table>
<thead>
<tr>
<th>variable</th>
<th>Control group (n=13)</th>
<th>experimental (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age(y)</td>
<td>34.6±4.7</td>
<td>37.9±5.2</td>
</tr>
<tr>
<td>Height(cm)</td>
<td>178.7±6.2</td>
<td>177.3±5.9</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>77.3±6.7</td>
<td>78.2±6.3</td>
</tr>
<tr>
<td>BMI(kg/m²)</td>
<td>24.44±2.3</td>
<td>25.32±2.16</td>
</tr>
<tr>
<td>Body fat percentage (%)</td>
<td>20.19±2.17</td>
<td>21.31±2.42</td>
</tr>
</tbody>
</table>

Data are expressed as mean±SD.

Statistical analysis

All values are reported as Means ±SE. Differences between exercise-induced changes in plasma samples concentrations and body fat before and after exercise protocol were evaluated using a Students t-test for paired samples. A P-value < 0.05 was considered to be statistically significant.
Results

As expected, after 16 weeks of aerobic exercises, the experiment group showed a significant decrease in weight and fat percentage. The plasma levels of adiponectin and resistin showed an increase and the leptin level showed a decrease and not affects CRP level in the experimental group, comparing to the control group (Fig. 1 and Table 2). Also, there was no significant relationship between fat percentage and plasma levels of the three hormones of adiponectin, leptin and resistin and CRP was correlated with percent body fat (Table 3). However, after studying the percentages of the changes in these variables, it was showed that the decrease in fat percentage had a negative correlation with the increase of two hormones of adiponectin and resistin, and a positive correlation with leptin and non-correlation with CRP.

**Table 2: anthropometric and hormonal indicators of the subjects, before and after the training program**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group</th>
<th>Experiment group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-test</td>
<td>Post-test</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77.3±6.7</td>
<td>78.1±5.5</td>
</tr>
<tr>
<td>BMI</td>
<td>24.4±2.3</td>
<td>24.3±3.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>20.19±2.17</td>
<td>20.31±1.93</td>
</tr>
<tr>
<td>CRP (mg/l)</td>
<td>4.37±2.8</td>
<td>4.36±2.9</td>
</tr>
<tr>
<td>Adiponectin (µg/ml)</td>
<td>15.73±1.27</td>
<td>16.37±1.38</td>
</tr>
<tr>
<td>Leptin (ng/ml)</td>
<td>3.33±0.59</td>
<td>3.41±0.63</td>
</tr>
<tr>
<td>Resistin (ng/ml)</td>
<td>9.21±1.51</td>
<td>9.39±1.41</td>
</tr>
</tbody>
</table>

Significance of the changes at the level of 0.05 the results are showed in the form of mean±SD.

**Table 3: therelationship between fat percentage and the hormones of CRP, adiponectin, leptin and resistin**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre training</th>
<th>Post training</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r value</td>
<td>r value</td>
</tr>
<tr>
<td>CRP</td>
<td>0.54</td>
<td>0.52</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>0.22</td>
<td>0.03</td>
</tr>
<tr>
<td>Leptin</td>
<td>0.13</td>
<td>0.25</td>
</tr>
<tr>
<td>Resistin</td>
<td>0.35</td>
<td>0.05</td>
</tr>
</tbody>
</table>

**Figure 1: changes in the fat percentage and hormones of CRP, adiponectin, leptin and resistin in experimental and control groups.**

**Discussion and Conclusion**

The purpose of this study was to examine the effects of aerobic exercise on CRP, adiponectin, leptin and resistin in adults. Despite statistically significant improvements in body weight, percentage of body fat, changes in CRP as a result of aerobic exercise were not statistically significant.
The results for CRP from our meta-analysis as well as the individual studies included are in sharp contrast to the findings from a recent qualitative systematic review on this topic that correctly concluded, based on the evidence reviewed; that both observational and interventional studies suggested that exercise training improved CRP levels (Kasapis and Thompson, 2005). One of the reasons for the discrepant findings may have to do with the different types of study designs included.

Alternatively, it may be that one has to be exposed to regular exercise over a longer period than the training protocols included in our studies to experience any CRP-lowering benefits.

Although the focus of our study was on the independent effects of aerobic exercise on CRP, it may be that exercise in combination with other lifestyle interventions may yield the greatest benefit. For example, Milani et al. (2004) found statistically significant reductions in CRP as a result of exercise training and diet in both men and women with coronary heart disease. Given these findings, it may be that the greatest reductions in CRP may be derived from a combination of exercise and other lifestyle modifications such as diet.

The fact that we did not find any statistically significant decreases in CRP as a result of aerobic exercise may be related to the inability to isolate those subjects with elevated CRP levels. To support this possibility, a recent no-control study in sedentary black and white women and men found that 20 weeks of aerobic exercise did not significantly reduce CRP levels for those in the low (b 1.0 mg/L) and moderate (1.0-3.0 mg/L) risk categories, but did significantly reduce CRP levels for those in the high risk category (N3.0 mg/L) (Lakka et al., 2005).

Many studies have shown, Leptin is an adipocyte-secreted hormone that seems to play an important role in the body weight regulation in humans (Bouassida et al., 2009).

The result of present study demonstrate that moderate-intensity exercise for 16 weeks and three session per week, significant decrease in body weight, BMI and serum leptin level. However, further studies are necessary to clarify this relationship. Our data consistent with those reported exercise training resulted in decreased leptin concentration (Nammi et al., 2004; Weltman et al., 2000). However, these decreases were related mainly to negative energy balance and/or to loss of adipose tissue, and suggested that exercise training does not have an independent effect on circulating leptin. Kohrt et al (1996) for example, who found that long-term exercise training, can decrease plasma leptin concentrations in adult men by reducing fat mass. Also, Ishii et al (2001) proved that serum leptin level decreased after 6 weeks of aerobic training exercise in type 2 diabetics’ patients, but there was not any relationship between the decrease in leptin concentration and changes in adipose tissue. Reseland et al (2001) concluded that long-term diet and exercise may have direct effects on plasma leptin concentration beyond the effect expected due to changes in fat mass. The results of this study contrast to the results of Weltman et al (2000), who reported that 30 min of exercise at various intensities and caloric expenditure (from 150 ± 11 to 529 ± 45kcals) in 7 healthy young men did not cause modifications in leptin levels during the exercise and during the recovery (3.5 hours).

What is known is that leptin act through changes in obgene expression in adipose tissue. Changes in leptin correlated with the changes in body weight. Some researchers believe that aerobic exercise is the best way to reduce body fat and ultimately in the regulation of leptin level. They are trying to be able to leptin treatment by way easier to find for the treatment of obesity.

The results showed that body fat and plasma leptin decreases after 8 weeks of aerobic exercises. This change in the level of leptin has been reported by a great range of previous studies.

The level of leptin in the blood has reduced after 2 weeks of skiing exercises in a group of men (Eriksson et al., 2008). Also, the similar result was found on serum level of leptin, after a one-year training program in a group of overweight men (Miyatake et al., 2004).

The leptin level in a group of old men (65-75 years) divided into three groups of light, moderate and heavy training was decreased after a year of resistance training. Fat percentage and BMI decreased in the three experimental groups and this decrease was more at heavier intensities. The changes in the level of leptin had a direct relationship with the changes in the level of fat, which are in agreement with the results of this study (Fatouros et al., 2005).

On the other hand, 6 weeks of strength training (Lau et al., 2010) and 6 weeks of resistance training (Ara et al., 2006) did not change the level of leptin of obese subjects. It should be mentioned that the subjects of those study did not show a significant loss of weight after executing this program. In a temporal-phenomenal historical study on the patients suffering from fatty liver and on healthy people, it was indicated that people, who did aerobic exercises, at least one a week, had lower serum leptin (Zelber et al., 2008).

Current study showed that the plasma adiponectin increases following 8 weeks of aerobic training, and it absolute amounts after increasing are almost two times higher than the patients in the other studies (Urawska et al., 2009) and this high level of adiponectin is probably a preventing factor against diseases related to adiponectin. The changes in the level of adiponectin because of adaptation with the aerobic exercises in most of the previous studies have shown the same results as of this study and such agreement is also observed in resistance exercises (De salles et al., 2010).

In a study on young and middle-aged women, doing ten weeks of aerobic training resulted in an increase in the plasma levels of adiponectin alongside with a decrease in insulin resistance (Lim et al., 2008).
Also, eight young fat women (BMI≥25) showed a significant decrease in the level of fat mass and leptin, also an increase in the level of adiponectin after 7 months of aerobic training program (Kondo et al., 2006).

In another study, a group of fat subjects with insulin resistance were given a program of 19 weeks of aerobic training alongside with diet. The results showed that visceral fat was decreased and adiponectin was increased. And also it is reported that adiponectin level is directly related to fat distribution (Shadid et al., 2006), which was not found in present study.

There are also studies, which show exercises have no effect on the level of adiponectin. That maybe because of using a combination of endurance-strength exercises (De Salles et al., 2010) or using athlete subjects who have higher adiponectin level in baseline (Jurimae et al., 2006) or other unknown factors (Balducci et al., 2010).

Kadoglou et al. found that 16 weeks of aerobic training resulted in the reduction in plasma levels of resistin, CRP, and interleukin-6 in the patients suffering from type 2 diabetes (Kadoglou et al., 2007). Patients suffering from type 2 diabetes and metabolic syndrome after 12 months of aerobic training showed an increase in their serum level of adiponectin, and also showed decreases in serum levels of resistin and leptin (Balducci et al., 2010).

As seen, researches which have studied the effect of exercising on people suffering from diseases, mostly has found the resistin to be reduced, while in this study which is done on healthy people who are not overweight, exercising has increased plasma level of resistin. In agreement with the results of this study, Perseghin et al. showed that elite endurance athletes comparing to patients with type 2 diabetes and also healthy subjects with no physical activity have higher levels of resistin and insulin sensitivity (Perseghin et al., 2006). This challenges the direct relationship between resistin level and obesity and associated diseases (Huang et al., 2005).

Elloumi et al (2009) divided 21 fat teenagers (BMI= 30.8±3.2) into the three groups of low-calorie diet, exercising, and a combination of both. Following 2 months of the protocol of weight loss, the three groups showed an increase in the level of adiponectin and decrease in the level of leptin, and that was more significant in the group with the combination of diet and exercise. Also the level of resistin showed a significant increase in the two groups of exercising and combination of exercising and diet.

In order to clarify the effect of exercising on the level of resistin in different subjects, a comparative study on ill people, healthy people and athletes seems required. It should be mentioned that in this study, the mean value of increased serum levels of resistin in the experimental group after 8 weeks of practicing was not comparable with the absolute amount of resistin measured in the other patients of the other researches, and it was much lower. It seems that increases in the levels of resistin, which happen in healthy people because of exercising, they are originated by the inflammation of training. The most important mechanism which can explain the increase in the level of resistin after aerobic exercises in this study, is the role of this hormone in anti-oxidation defences of the body, as Boet al (2005) have reported in their study that resistin functions as an antioxidant in response to inflammatory stimulant.

In conclusion, the results of our study suggest that aerobic exercise does not lower CRP levels, but , most important findings were to show that plasma adiponectin, leptin and resistin concentrations will improve followed by 16 weeks of aerobic training that was associated with improvement of body composition. Ideal levels of such adipokines can play an outstanding role in preventing metabolic and cardiovascular disease.

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Conflict of interest
The authors declare no conflict of interest

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