Effect of 12 weeks of aerobic training on visfatin levels in obese women

Banaeifar Abdolali¹, Rahmanimoghadam Neda², Zafari Ardeshir³ and Kazemzade Yaser⁴

¹Department of Physical Education and Sport Science, South Tehran Branch, Islamic Azad University, Iran.
²Department of Physical Education and Sport Science, Science and Research Branch, Islamic Azad University, Iran.
³Department of Physical Education and Sport Science, Zanjan Branch, Islamic Azad University, Iran.
⁴Department of Physical Education and Sport Science, Islamshahr Branch, Islamic Azad University, Iran.

ABSTRACT

Visfatin is an adipocytokine that release from adipocytes. It is unknown whether training also influences concentrations of visfatin. The purpose of this study was to examine the effects of 12 weeks of aerobic training on visfatin levels in obese women. Thirty two obese women (age = 37.8 ± 13.2 years, body mass index = of 39.4 ± 6.4 kg/m²) volunteered to participate in a 12-wk exercise program. They were randomly assigned to either a training (n = 16) or control (n = 14) group. The training group exercised for 70 minutes per session, 3 days per week during the 12 week training program. The control group was asked to maintain their normal daily activities. Samples were obtained before and at the end of training program. We use t. paire and independent, test for data analyzes. Exercise training resulted in a decrease in body weight (p < 0.05), percent body fat (% fat) and BMI (p < 0.05), fasting glucose level and visfatin concentration decreased but wasn’t significant (p > 0.05). In conclusion, the 12 week aerobic training program used in this study was very effective for producing significant benefits to body composition but didn’t significant lowering visfatin levels in these obese women.

Key words: Aerobic training, visfatin, glucose, women.

INTRODUCTION

Obesity is defined to mean an increase in body fat, also it has been introduced as a risk factor for the development of lipid disorders, hypertension, cardiovascular disease and type 2 diabetes [1,2]. In recent years, Adipose tissue is now recognized as an endocrine organ that secretes many cytokines such as tumour necrosis factor alpha, interleukin 6, leptin, visfatin and resistin [3].

Bastard et al. States that at least part of the increase Risk of type 2 diabetes in obese is caused by a change in the adipose tissue function [4,5]. The classical conception about adipocytes which only as a storage site for increase lipid has changed over the last decade. This is attributed to the discovery that adipose tissue can function as an active endocrine organ, co-regulating whole-body metabolism[6]. Visfatin is a lately recognized adipocytokine in visceral adipose tissue and has insulin-like metabolic effects that may improve insulin sensitivity [7]. Visfatin is new adipokaine which is produced mainly in visceral adipose tissue, furthermore its plasma level correlates with the quantity of visceral fat in humans [7,8], and increased visceral body fat is closely linked to insulin resistance in
adults [9,10]. Visfatin can also be produced by cells, neutrophils and macrophages visceral adipose tissue [4]. Plasma levels of this adipocytokine have been shown to positively correlate with the amount of visceral adipose tissue determined [7]. Visfatin was originally thought to employ insulin mimetic effects by binding to and activating the insulin receptor [7]. Recently reported that visceral adipose tissue (VAT) VAT loss after aerobic exercise training improves glucose metabolism and is associated with the reversal of insulin resistance in older obese men and women [12,13]. Thus, it seems likely that visfatin would respond to exercise training. However, studies examining the effects of exercise on circulating visfatin are limited [8,13,14]. Research has shown that Plasma visfatin concentrations are elevated in patients with diabetes mellitus [15,17], and can be lowered in obese subjects by weight loss [18], and in patients with type 1 and type 2 diabetes mellitus by aerobic exercise programs [16,19]. The exercise program also had lowering effects on visfatin levels in non-diabetic women [19,20]. There are limited data on the role of physical exercise on visfatin. The results of visfatin studies conducted so far in obese individuals are, however, controversial and fail to unequivocally explain the relationship between this adipocytokine and obesity or glucose metabolism abnormalities. The aim of this study was to evaluate changes in visfatin levels following a 12-week training program in obese women.

MATERIALS AND METHODS

Subjects:
A total of 32 non-morbid obese (body mass index (BMI) inclusion criteria: 30e39.65 kg/m2) women from west Tehran city, aged between 30 and 45 years volunteered to participate in this study, and underwent a 12-week training program. Participants were health and non-athletic, they hadn’t cardiovascular disease or diabetes. All women received information about the nature and purpose of the survey, and all of them gave written consent for participation in the study.

Protocol:
Exercise training intensity commenced at a level prescribed between 50% and 55% of the HR maximum (HRmax), and gradually increased so that, by week 6, the subjects were exercising at 70%–75% HRmax. For the duration of the 12-wk period, subjects exercised 3 day per week, for 50–60 min with a 10-15 min warm-up and cool-down.

Collection of blood samples:
The fasting blood samples were collected in the morning between 8:00 AM and 10:00 AM following an overnight fast. Blood samples were centrifuged (3000 rpm) at room temperature for 20 minutes. The separated sera was then stored at –70 °C until the time of the assays for variables levels. Plasma visfatin was measured by a commercial visfatin kit (Biovendor- Laboratorial kit made by Biovendor Company, Czech).

Statistical analysis
All data sets were tested for normal distribution using the Kolmogorov-Smirnov test. Baseline measurements were compared between groups using the independent test. The effects of training were assessed by t.pair test. All calculations were performed using the spss ver 19. Values are expressed as means_sd.

RESULTS

All parameters of interest were similar between the two groups at baseline (p > 0.05) (Tables 1). Table 2 also displays anthropometric and biochemical indicators for both groups at baseline (pre training) and following the 12 week intervention (post training).

<table>
<thead>
<tr>
<th>variables</th>
<th>Pre.T</th>
<th>Post.T</th>
<th>Pv</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age(year)</td>
<td>37±5.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height(cm)</td>
<td>159.43±7.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.38±7.77</td>
<td>80.63±7.93</td>
<td>.000</td>
</tr>
<tr>
<td>Body mass index (kg/m2)</td>
<td>32.36±1.11</td>
<td>31.29±1.36</td>
<td>.000</td>
</tr>
<tr>
<td>circumference hip</td>
<td>114.75±6.60</td>
<td>110.50±6.85</td>
<td>.005</td>
</tr>
<tr>
<td>Visceral fat(level)</td>
<td>8.46±2.85</td>
<td>8.44±2.91</td>
<td>.939</td>
</tr>
<tr>
<td>Percent body fat (%)</td>
<td>46.52±3.88</td>
<td>44.5±3.67</td>
<td>.001</td>
</tr>
<tr>
<td>Fasting blood glucose (mmol/l)</td>
<td>92.25±9.88</td>
<td>82.81±12.32</td>
<td>.001</td>
</tr>
<tr>
<td>Visfatin (ng/ml)</td>
<td>2.91±1.64</td>
<td>2.71±1.17</td>
<td>.292</td>
</tr>
</tbody>
</table>

The training group had significant decreases in body weight, % fat and circumstance hip (p < 0.05), while the control group remained unchanged. fasting glucose levels, and visfatin concentration and visceral adipose tissue weren’t
significant decreases (p > 0.05). As shown in figure 1, the mean visfatin decreased but this decrease wasn’t significant.

**figure 1** Fasting plasma visfatin concentration (ng/mL) before and after the 12-wk exercise training program.

**DISCUSSION**

In this study we investigated visfatin response to 12 week aerobic exercise program in obese women. The comparison of base quantity of weight, fat percent, hip circumference, visceral fat, visfatin and glucose in experimental and control groups showed, no significant difference between the two groups (P>0.05). The results showed exercise program induced insignificant decrease in visfatin concentration, visceral fat and glucose levels (P>0.05). However body weight, BF%, percent body fat, BMI and HC reducing significantly in exercise groups. (P<0.05). There are limited data about effects of exercise training on visfatin levels. The decrease of visfatin due to effect of training report in many studies. [16, 20, 21]. Haider et al. demonstrated that exercise training could lowers plasma visfatin in patients with type 1 diabetes mellitus [16]. Further it has been reported exercise training that resulted in weight loss induced significant reductions in plasma visfatin in young, overweight Korean women [20]. Also Büyükayazi and et al [21], reported the concentration of visfatin (p<0.05) reduced in obese women (30-49 years) in response to high-intensity walking program (twelve weeks, five days per week from 30-60min/day). Brema et al. [14] showed plasma visfatin concentration was significantly reduced in young, obese patients with type 2 diabetes. On the other hand in Büyükayazi and et al studies, they found no significant change in visfatin concentration in obese women (30-49 years) in moderate-intensity walking group after twelve weeks, walking program [21]. Saghebjo et al reported that 8 week aerobic exercise weren’t induced significant decrease in visfatin levels in obese women [22].

Limited studies that have been done on the effect of exercise on visfatin levels indicate that Visfatin response to exercise have been inconsistent. The results of this study related to changes of visfatin due to effect of exercise showed, Visfatin after 12 weeks of aerobic exercise training hasn’t decreased compared to the before exercise training. This finding is inconsistent with the results of the studies determined reductions [14, 16, 20, 21] in visfatin levels of the participants due to exercise training. However the visfatin levels was deceased but wasn’t significant. Likewise the finding of this study is consistent with the results of many studies [22, 21].

The reduced plasma visfatin after exercise training in the Choi et al study explained by benefits of exercise. Overall, Most research is done about effects of training on visfatin in some groups (obese, diabetic), show decrease visfatin levels. The lack of significant reduction in our study, may indicate that intensity exercise is not enough to cause reductions in visfatin levels. On the other hand Fukohara explain that visfatin is preferentially produced by human visceral adipose tissue (VAT). Also it has been reported that visfatin levels associated with visceral fat [7]. The survey results of this study showed that most of the changes in anthropometric variables such as weight, body mass index and hip circumference decreased. However, visceral fat in this study were not significant (p>0.05). Furthermore, because of central obesity is closely related to visceral adipose Tissue. It seems that the reason lack of significant change in visfatin levels in this study was due to absence of change in visceral adipose tissue. It also can be result in low intensity or type of exercise protocol. According to the results obtained, It seems that more research is needed on the effect of exercise on visfatin levels.
Acknowledgement
The authors would also like to thank the study participants cooperation. This research was supported by Islamic Azad university Tehran south branch.

REFERENCES