A comparison of 8 weeks endurance, resistance and concurrent training on plasma visfatin in obese men

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Abstract

There is a lack of studies comparison exercise training type on visfatin in obese population. Therefore, our purpose was to comparison of 8 weeks endurance, resistance and concurrent training on visfatin in obese men. Forty four untrained obese student men were volunteered to participate in the current study. The subjects divided into four groups: Endurance (cycling on cycle ergometer for 40 min at 60% of HRmax), resistance (5 resistance exercises at 60% of 1RM) and concurrent (20 min endurance exercise at 60% of HRmax and 3 resistance exercises at 60% of 1RM) training and no training control group, then experimental groups participated in 8-week training program three times per week. Plasma samples were obtained pre training and within 24–48 h after the last exercise bout for analyses visfatin concentrations. Data was analyzed by using a 2-way ANOVA and LSD post-hoc test. The level of significance was set at p<0.05. After training program, visfatin levels were significantly decreased in endurance and concurrent training groups. Furthermore, body mass and fat mass of participants was decreased in these training groups. This study suggests that 8 weeks endurance and concurrent exercise training induced a significant reduction of plasma visfatin with body loss in obese men.

Key words: training type, obesity, adipokine, insulin-mimetic, adipose tissue

Introduction

The incidence and prevalence of obesity is increasing worldwide. Obesity as a major health problem is characterized by an excess of body fat that has been defined as a risk factor for the development of insulin resistance, type 2 diabetes, dyslipoproteinemia, hypertension, and cardiovascular disease morbidity/mortality from vascular diseases (Pi-Sunyer, 1993). Adipose tissue secretes some hormones and cytokines that are involved in the metabolic syndrome (Kershaw and Flier, 2004). Visfatin is an adipokine that is preferentially produced in visceral adipose tissue. It is a 52 kilo Dalton cytokine that both its expression and plasma concentration increase in obese people (Fukuhara et al., 2005). Furthermore, increasing concentrations of visfatin were independently and significantly associated with type 2 diabetes (Chen et al., 2006). The metabolic effects of visfatin are apparently mediated by the binding to and activation of the insulin receptor (Fukuhara et al., 2005). Indeed, visfatin has an
insulin-like function and insulin-mimetic effect of visfatin is dependent on its binding to the insulin receptor resulting in its tyrosine phosphorylation as well as phosphorylation of insulin receptor substrate-1 and -2 leading to enhanced glucose uptake in vitro and in vivo (Fukuhara et al., 2005; Haider et al., 2006b).

A positive correlation between visceral adipose tissue visfatin gene expression and body mass index (BMI) was noted (Varma et al., 2007; Seo et al., 2011), and plasma visfatin can be reduced after weight loss in obese subjects (Haider et al., 2006a). Also, increased plasma visfatin concentrations in morbidly obese subjects were reduced after gastric banding (Haider et al., 2006c). Previous acute exercise studies have examined changes in plasma visfatin responses after a single exercise bout (Ghanbari-Niaki et al., 2010; Shekholeslami et al., 2011). For example, we reported in our previous investigation that serum visfatin levels were reduced significantly following moderate exercise for 30-min in nine healthy male subjects (Shekholeslami et al., 2011). Ghanbari-Niaki et al. (2010) reported that high-intensity sprint exercise resulted in increased plasma visfatin levels. But, there are limited and controversial data regarding impact of chronic exercise training on plasma visfatin. However, regular physical activity confers many physiological and psychological benefits including an improved lipid profile and modified metabolic syndrome factors, enhanced insulin sensitivity, and an increased energy expenditure which has the potential to lower body fat and body weight (Jakicic et al., 2001; Seo et al., 2011). Multiple studies reported that aerobic exercise training tends to lower plasma visfatin levels. Often, this decrease is accompanied by a decrease in BMI (Choi et al., 2007; Hausel et al., 2009; Lee et al., 2010). However, some previous studies reported that plasma visfatin were not altered after exercise training (Fallah et al., 2011); even visfatin has been shown to increase after 12 weeks exercise training (Jorge et al., 2011). Collectively, there are limited data on the role of physical exercise on resting plasma visfatin levels. It has already been shown that the physiological and biochemical adaptations to exercise training are highly specific to the type of exercise performed (Sharkely, 1990; John, 2009). Therefore, our purpose was to investigate and comparison the possible effects of three different types of exercises include aerobic, resistance and concurrent exercise (combined aerobic and resistance exercise) on plasma visfatin levels following 8 weeks exercise training in obese men subjects.

**Materials and Methods**

**Subjects**

A total of 44 men subjects who were untrained and obese voluntarily participated in this study (Table 1). Exclusion criteria included evidence of overt diabetes (Type 1 or Type 2), smoking, medication affecting metabolism, acute or chronic disease (cardiovascular, cerebrovascular, liver, renal, hematological, thyroid, or cancer), eating disorders and any condition that prevent them from partaking in physical activity. The Institutional Review Board of the University approved the research protocol. The subjects refrained from any additional nutrition supplementation and exercise during this study and were encouraged to adhere to their usual dietary patterns. The subjects gave a written informed consent after receiving an explanation of the procedures and risk involved.

Body composition was determined by skinfold method (Lohman et al., 1988) using a Lange skinfold caliper and standard techniques from seven sites (triceps, subscapular, midaxillary, chest, suprailliac, abdomen, and thigh). Skinfold measurement were based on the average of two trials and obtained on the right side. Body density was estimated using the age-adjusted equation of Pollock and Jackson (1984). The three-compartment Siri equation was used for body fat percent (Siri, 1961). Height and body mass were assessed by digital scale (Camry, EB 9003) and height rod (Iran). After formalization sessions and 1RM test, participants were randomly divided to four groups as control (C), aerobic exercise (AE), resistance exercise (RE) and concurrent exercise (CE) to participate in a 8-wk supervised exercise training program.

Exercise training program. All exercise training sessions were supervised by an exercise physiologist. For the duration of the 8-wk period, subjects in experimental groups (AE, RE and CE) exercised 3 days/wk for 40–45 min. The subjects in AE group performed cycling on cycle ergometer for 40 minutes at 60% of the maximum heart rate calculated from 220–age equation. Participations of resistance group performed 3 sets of 10 repetitions of the 5 exercises (bench press, parallel squat, lat pull-down, standing two-arm curl, hamstring curl), with a workload corresponding to 60% of 1RM, and an interval of 30s between the sets and 60s between the exercises.
Exercise for CE group were include 3 sets of 10 repetitions of the 3 exercises (bench press, parallel squat, lat pull-down), with a workload corresponding to 60% of 1RM, then cycling on cycle ergometer for 20 minutes at 60% of the maximum heart rate calculated from 220–age equation. Participations of C group remained without any exercise training in throughout the study.

Exercise training intensity gradually increased so that, by week 4, the subjects were exercising at 65–70% of heart rate maximum or 1RM until end of study. Prior to each exercise trial a 10 minute warm-up was performed and exercise trials were concluded by performing 10 minutes cool-down. Target heart rates were monitored via a heart rate telemetry strap (Polar Fitwatch, Polar Electro Oy, Finland). All tests were conducted at the same time of day (16:00–17:30 h) to minimize potential diurnal variations in related hormones. Ambient temperature was controlled between 23 and 26°C.

Blood samples were obtained after a 12-h overnight fast and were kept at -20°C for subsequent assay. We collected blood samples from an antecubital vein before exercise training and within 24–48 h after the last exercise bout.

The concentrations of serum insulin were determined by Insulin Human ELISA kit (Demeditec Company, Germany). The plasma visfatin was measured using enzyme immunoassay ELISA kit (Cusabio, Biotech, Wuhan, China). The intra-assay for visfatin was less than 5.9%.

Statistical analyzes

Data are reported as mean ± SEM. Baseline levels in different groups were analyzed by one-way analysis of variance (ANOVA). Visfatin responses after exercise training were evaluated by two-way repeated measures ANOVA (group × time). In the case of a significant F value, a Fisher’s least significant difference (LSD) post hoc test was used between means. Statistical significance was set at P<0.05. Data were analyzed using SPSS for Windows (version 19.0).

Results

All subjects completed the 8-wk exercise training program. All parameters of interest were similar between the two groups at baseline (p>0.05). Table 2 displays the changes in visfatin, insulin and body composition to aerobic, resistance and concurrent exercise protocols for all groups at baseline (pre training) and following the 8 week intervention (post training).

Table 1: Baseline physical characteristics, Data are means (±SD)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Aerobic group (AE)</th>
<th>Resistance group (RE)</th>
<th>Concurrent group (CE)</th>
<th>Control group (C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>29.52±3.08</td>
<td>29.62±3.8</td>
<td>28.75±5.61</td>
<td>28.61±3.9</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>92.13±7.61</td>
<td>90.87±7.89</td>
<td>91.82±8.12</td>
<td>91.8±8.25</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171.12±6.8</td>
<td>173.42±5.3</td>
<td>172.21±5.2</td>
<td>171.48±3.4</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>31.5±2.1</td>
<td>30.4±1.8</td>
<td>31.1±1.7</td>
<td>31.4±2.1</td>
</tr>
</tbody>
</table>
Table 2: Physiological parameters of study subjects before and after the exercise training program

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Aerobic group (AE)</th>
<th>Resistance group (RE)</th>
<th>Concurrent group (CE)</th>
<th>Control group (C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pre</td>
<td>post (p)</td>
<td>pre</td>
<td>post(p)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>92.13±7.61</td>
<td>90.22±3.63* (0.013)</td>
<td>90.87±7.89</td>
<td>91.72±3.18* (0.024)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>31.5±2.1</td>
<td>30.4±1.2* (0.008)</td>
<td>30.4±1.8</td>
<td>30.6±1.3</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>28.11±5.83</td>
<td>26.82±2.69* (0.001)</td>
<td>29.32±6.4</td>
<td>28.93±3.94</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>25.79±7.42</td>
<td>23.82±5.41* (0.011)</td>
<td>26.35±7.82</td>
<td>26.1±3.74</td>
</tr>
<tr>
<td>Fat free mass (kg)</td>
<td>66.34±5.32</td>
<td>66.40±3.71</td>
<td>64.52±5.12</td>
<td>65.62±3.11* (0.037)</td>
</tr>
<tr>
<td>Visfatin (ng/ml)</td>
<td>14.72±8.24</td>
<td>13.18±7.73</td>
<td>14.24±7.55</td>
<td>8.83±3.78* (0.000)</td>
</tr>
</tbody>
</table>

* P<0.05 vs. pre-training. † P<0.05 vs. RE

The results showed that plasma visfatin, body mass, fat mass, and body fat percent were decreased after training in both AE and CE (P<0.05). Body mass significantly increased in RE and BMI significantly decreased in AE. Measures of fat free mass significantly increased in RE and CE but not in AE.

Discussion and conclusion

The purpose of present investigation was to study the effects of three different type exercises include aerobic, resistance and concurrent exercise training on plasma visfatin levels following 8 weeks exercise training in obese men subjects. The results demonstrated that aerobic (AE) and concurrent exercise (CE) training can reduce plasma visfatin levels in obese men. The significant improvements observed in visfatin levels for the training groups (AE and CE) can provide an effective approach for combating this factor associated with obesity.

Adipocyte visfatin (as a new adipokine) expression and plasma concentrations increase with obesity in animals (Berndt et al., 2005) and humans (Fukuhara et al., 2005). Visfatin may have a dual function: an autocrine/paracrine role that facilitates differentiation and fat deposition on visceral adipose tissue, and an endocrine role that modulate insulin sensitivity in peripheral organs (Sethi and Vidal-Puig, 2005). Acute administration of visfatin to mice lowered glucose levels (Fukuhara et al., 2005) and visfatin may facilitate glucose control; on the other hand, it may promote the development of obesity (Arner, 2006). In the present study, plasma visfatin was significantly reduced by aerobic exercise training. Brema et al. (2008) found a similar visfatin response by approximately 80 and 50% after 12 weeks of endurance exercise training in obese young subjects with type 2 diabetes or normal glucose tolerance, respectively. Haider et al. (2006a) reported that circulating visfatin concentrations are reduced by chronic exercise. They also noted that this effect is sustained after training cessation. Haus et al. (2009) investigated the impact of exercise training on plasma visfatin by studying non-diabetic obese men. Aerobic exercise was done at 60% of HRmax at the start of the study and increased up to 85% of HRmax by the end of the 12-week training program. Training elicited a significant decrease in overall body fat, body weight, BMI, resting blood glucose, and a decrease in plasma visfatin.

The response of visfatin to resistance exercise training in this study is similar to that found in previous study. Mohammadi and Khajehlandi (2010) were examined the effect of resistance training on plasma visfatin in non-obese middle-aged men. Resistance exercise training was performed for 3 days a
week at an intensity corresponding to 65–80% of one-repetition maximum, 8–12 repetitions, 2–4 sets for 8 weeks. The results showed that body fat percent, WHR and plasma visfatin were decreased in the training group. However, visfatin was found to be increased after resistance training consisted of 7-exercise circuits involving major muscle groups done 3 times per week at high intensity (Jorge et al., 2011). This conflict results is unclear, however a possible explanation for the contrasting findings may be related to differ in employed exercise intensity.

The reduced plasma visfatin after concurrent exercise training in current study is consistent with some reports. For example, Seo et al. (2011) examined the effects of 12 weeks of combined exercise training on visfatin and metabolic syndrome factors in obese middle-aged women. They reported that combined resistance and aerobic training program can reduce visfatin levels in obese middle-aged women. In another study, the effect of exercise training on plasma visfatin and eotaxin levels in non-diabetic women was investigated. The researchers found that plasma visfatin and eotaxin levels were decreased after exercise training with weight loss (Choi et al., 2007). In contrast, Seo et al. (2007) after measurements in fasting plasma visfatin levels in 10 women who contributed in a supervised combined exercise program (60 min/day, 60–70% HR-reserve, 400 kcal, 10RM/3 set, 3 days/week) for three months, reported that plasma visfatin levels were not significantly changed after combined exercise training in exercise group. Collectively, these results demonstrated that aerobic exercise training tends to lower plasma visfatin levels and this has been shown to be true in multiple studies. Further work, however, is needed to better clarify exercise training types effects on plasma visfatin levels.

Comparisons between AE and RE groups in the current study suggest that AE decreases both body weight and fat mass than does RE. These changes were driven by different mechanisms, where RE increased fat free mass and AE decreased fat mass. These data are supported by other findings from this trial that indicate AE significantly reduced visceral adipose tissue more than RT and trended toward the same result in liver fat change (Slentzet al., 2011). We also examined whether the addition of AE to an RE program provided benefits for reduction in fat mass. Our results showed that combined AE and RE as CE produced similar change with AE in body and fat mass. Moreover, although CE increased fat free mass, CE type decreased body weight, thus CE via decrease in fat mass provided beneficial effect on body weight. In our study, similar to some previous studies (Choi et al., 2007; Hauser et al., 2009; Lee et al., 2010), reduced plasma visfatin levels were associated with decreased body mass, fat mass, body fat percent in AE and RE group and BMI in AE group. Although possible mechanism/mechanisms responsible for decrease circulating levels of visfatin by exercise training are not well understood, some previous results suggested that improving body composition and adipose tissue may effective mechanisms for decrease concentration of plasma visfatin (Hauet al., 2009). It has been reported that each 1 cm increase in waist circumference of subjects associated with 4/2 ng/mL increase in plasma visfatin level (Bo et al., 2009). Berndt et al. (2005) found a positive correlation between plasma visfatin concentration and body fat percent measured by DXA. Moreover, visfatin can be lowered in obese subjects by weight loss (Haider et al., 2006c). Therefore, because the decrease of plasma visfatin were associated with decrease in body mass, fat mass and percentage body fat after AE and CE, it seems that the aerobic and concurrent exercise training could offer a sufficient stimulus for plasma visfatin decreases. Further studies, however, is necessary to determine this expectancy and explanation the mechanisms responsible for the effects of exercise training on visfatin.

Although more work is warranted to accurately describe the characteristics of visfatin, however, we found that plasma visfatin levels were reduced after aerobic and concurrent exercise training and this exercise-induced reduction of plasma visfatin is most likely the result of weight loss and body composition changes. These results suggest that changes in visfatin levels may be associated with the beneficial effect of AE and CE exercise training in obese men.

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**References**


