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ORGINAL ARTICLE

Effect of 12 Weeks Endurance Training on Plasma Visfatin in Men

Sheibani, S.H;[1] Hanachi, P[2]; Refahiat, M.A;[1] Khoshneshin, Z[3]

[1] Department of Physical Education, Fars Science and Research Branch, Islamic Azad University, Fars, Iran
[2] Department of Physical Education, Alzahra University, Tehran, Iran
[3] Department of Physical Education, Shiraz University, Shiraz, Iran

Abstract
Objective: Visfatin, is a novel adipokine, and an adipose tissue-derived hormone shown to correlate with visceral fat mass in patients with obesity. It is not well known whether endurance training – induced change in adipose tissue and blood lipids decreases plasma visfatin or not; thus, the purpose of this study was to examine the effect of 12 weeks endurance training on plasma visfatin in men. Methods: twenty five healthy men (aged 37.5 ± 4.8 years; height 17٤.7 ± 6.5 cm; BMI 25.2 ± 2.6 kg/m2; mean ± SD) participated as subjects in this study. The subjects were randomly assigned to endurance training group (n=15) or control group (n=10). Endurance training group underwent an 12-week intervention, with a frequency of 3 d/wk at an intensity corresponding to 65 – 80% maximum heart rate for 20 – 34 min. Results: results showed that body fat percent, WHR and plasma visfatin were decreased (P<0.05) after endurance training. Maximum oxygen consumption, on the other hand, increases significant (P<0.05) in the training group compared with the control group. Plasma visfatin levels at baseline were positively correlated (P<0.05) with body fat percent and triglyceride concentration. Conclusion: In conclusion, it seems that 12 weeks endurance training induced change in adipose tissue, decrease plasma visfatin in men.
Key words: Endurance training, Plasma visfatin, men.

Sheibani, SH (✉)
sh.sheybany@gmail.com

Introduction

Visfatin is a recently discovered adipokine, which is highly expressed in visceral fat. Adipocyte visfatin expression and plasma concentrations increase with obesity in animals (Fukuhara et al., 2005) and humans (Berndt et al., 2005). The metabolic effects of visfatin are apparently mediated by the binding to and activation of the insulin receptor (Davutoglu et al., 2009). Visfatin exerts insulin-mimetic effects and some studies suggest that visfatin is related to the development of the metabolic syndrome (Chen et al., 2006). Interestingly, visfatin treatment did not promote insulin resistance, but exhibited an insulin mimetic effect. Visfatin treatment in diabetic mice improved insulin sensitivity *in vivo* and

resulted in decreased glucose and insulin levels (Fukuhara et al., 2005). Studies demonstrated that pioglitazone reduces the visfatin expression (Lv et al., 2009), but visfatin unaffected from rosiglitazone and metformin (Kadoglou et al., 2010). Several studies exploring the effects of exercise on visfatin mRNA and plasma concentrations have results inconsistent findings. For example, Frydelund-Larsen et al demonstrated that visfatin mRNA expression increases after an exhaustion exercise and remained elevated 24 h after exercise (Frydelund-Larsen et al., 2007). On the other hand, Plasma visfatin not affected (Bo et al., 2009) or decreased (Haider et al., 2006; Haus et al., 2009) in response to exercise training. It is not well known whether exercise–induced change in adipose tissue – decreases plasma visfatin; therefore, we examined the effects of 12 weeks endurance training on plasma visfatin in men.

Methods

Subjects

Twenty five healthy men (mean ± SD: 37.5 ± 4.8 years), volunteered to participate in this study. Written informed consent was signed by all participants. The subjects were randomly assigned to a control group (n=10) or training group (n=15).

Exercise training

The subjects in the training group underwent a 12-week intervention, with a frequency of 3 d/wk at an intensity corresponding to 65-80% maximum heart rate for 20-34 min. Anthropometric and body composition measurements Height and body weight were measured, and body mass index (BMI; kg/m2) was calculated from height and weight of each subject. Waist circumference was determined by obtaining the minimum circumference (narrowest part of the torso, above the umbilicus) and the maximum hip circumference while standing with their heels together. The waist to hip ratio (WHR) was calculated by dividing waist by hip circumference (cm) (Williams & Wilkins., 2005).Subcutaneous body fat was measured at 3 sites (chest, abdominal, and thigh) with a Lafayette caliper. Body fat percent was calculated from the formula developed by Jackson and Pollock (Arner., 2006). All subjects fasted at least for 12 hours and a fasting blood sample was obtained by venipuncture. The plasma visfatin level was measured in duplicate using an enzyme-linked immunosorbent assay (ELISA) kits (Uscn Life cience Inc, Wuhan, China). Serum cholesterol triglycerides, HDL-c and LDL-c were assayed with automated techniques. Statistical analyses were performed with SPSS program (version 12, SPSS, Inc., Chicago, IL). Values were expressed as mean ± standard deviation (SD). Independent t-test and paired t-test were used to evaluate changes in variables. General linear regression analysis and Pearson's correlation were performed to calculate a correlation between variables in response to training. P-values less than 0.05 were considered statistically significant.

Results

Anthropometric, physiological and metabolic characteristics of subjects are shown in Table 1. The results showed that body weight, body mass index, body fat percent and WHR were decreased ($P<0.05$) after endurance training. Maximum oxygen consumption, on the other hand, increases significant ($P<0.05$) in the training group compared with the control group. Plasma visfatin and LDL-c decreased ($P<0.05$) and HDL-c increased ($P<0.05$) after 12 weeks endurance training (Table 1). Pearson's correlation demonstrated a positive relationship between plasma visfatin levels at baseline ($P<0.05$) with body fat percent and triglyceride concentration.

Table 1. Anthropometric and metabolic characteristics of study subjects (mean ± SD)

Variables	Control		Training	
	Pre test	Post test	Pre test	Post test
Body weight (kg)	76.05 ± 10.8	76.3 ± 10.9	76.5 ± 4.3	74 ± 2.6*
BMI (kg/m2)	24.9 ± 3.3	25.2 ± 3.3	25.5 ± 1.7	25.00 ± 1.5*
%Body fat	18.3 ± 5.1	18.3 ± 5.2	17.9 ± 2.2	16.3 ± 2.2*
WHR	0.89 ± 0.05	0.9 ± 0.05	0.91 ± 0.04	0.89 ± 0.04*
VO2max (ml.kg-1.min-1)	39.5 ± 3.8	40.6 ± 4.6	36.8 ± 2.6	47.1 ± 3.1*†
Triglyceride (mg/dl)	148.3 ± 47.8	129.2 ± 45.4	157.6 ± 58.4	142.5 ± 86.9
Cholesterol (mg/dl)	200.2 ± 21.8	191.4 ± 26.04	200.4 ± 37.9	190.1 ± 45.1
LDL-c	126.6 ± 21.4	122.5 ± 26.6	132.5 ± 37.7	120.7 ± 39.2*
HDL-c	41 ± 8.1	42.7 ± 7.6	39.1 ± 6.9	45.1 ± 6.3*
Visfatin (ng/ml)	21.1 ± 6.2	20.9 ± 5.9	21.2 ± 5.9	14.1 ± 4.9*†

***** P<0.05 for between-group differences; † P<0.05, pretraining *vs.* posttraining values.

Discussion

The results showed that Plasma visfatin decreased (P<0.05, 29.6%) in response to 12 weeks endurance training compared to the control group. Recently, Fukuhara et al. identified visfatin as a new adipokine that is preferentially produced in the visceral adipose tissue of obese mice and humans (Fukuhara et al., 2005). Although the function of visfatin is not currently understood, visfatin may have a dual role: an autocrine/paracrine function that facilitates differentiation and fat deposition on visceral adipose tissue, and an endocrine role that modulate insulin sensitivity in peripheral organs (Jackson et al., 1980). Therefore, visfatin may facilitate glucose control; on the other hand, it may promote the development of obesity (Sethi & Vidal-Puig., 2005). Researchers suggested that improving body composition and adipose tissue may effective mechanisms for decrease circulating levels of visfatin (Haus et al., 2009). Waist-hip circumferences and abdominal adipose tissue are independent predictors for plasma visfatin (Bo et al., 2009; Seo et al., 2007). The results showed that body fat percent and WHR decreased after endurance training, thus exercise-induced changes in body fat, especially visceral adipose tissue, may attribute to plasma visfatin decrease. On the other hand, there was the positive relationship between plasma visfatin and body fat percent at baseline and after the training. In another study of Berndt et al found a positive correlation between plasma visfatin concentration and %BF measured by DXA. It is encouraging that only a modest loss of 5–10 percent of body fat percent, leading to simultaneous improvement in adipokines (Sun et al., 2005). Exercise without weight loss is effective in reducing visceral adipose tissue and preventing further increases in obesity (Freedland., 2004). Body fat percent decreased 8.8% after 12 weeks endurance training, thus it seems that the endurance training could offer a sufficient stimulus for plasma visfatin decreases. Although some studies suggest that visfatin is related to the development of the metabolic syndrome (Fukuhara et al., 2005), others reported that visfatin gene expression was not associated with the

metabolic syndrome in diseased rats when compared with lean controls (Ross et al., 2000). In the present study, Pearson's correlation demonstrated a positive relationship between plasma visfatin levels at baseline with triglyceride concentration. Sun et al demonstrated that triglyceride was the only significant predictor of baseline visfatin concentrations independent of age and body fat percent. Our data in confirm to another investigations suggest that visfatin may exert an independent role in regulating triglyceride metabolism in humans (Seo et al., 2007). Jian et al reported that a single nucleotide polymorphism at different loci of visfatin gene was associated with triglyceride and total cholesterol levels. These reports suggest that visfatin may play a role in lipid homeostasis; however, the underlying mechanism is currently unknown (Klöting & KlötingI., 2005). Generally, plasma visfatin may relate to the development of the metabolic syndrome. We have some limitation in this study. Visfatin exerts insulin-mimetic effects and some studies suggested that plasma visfatin may related to fasting glucose and insulin and inflammatory markers such as TNF-α and IL-6 **(**Jian et al., 2006**).** Therefore, additional researches are needed to determine the effects of exercise training on plasma visfatin, type 2 diabetes risk factors, inflammatory markers and their relationships.

Conclusions

In conclusion, endurance training induced change in adipose tissue, decrease plasma visfatin in men. These findings suggested that changes in visfatin levels may be associated with the beneficial effect of exercise. Further studies are needed to elucidate the mechanisms responsible for the effects of exercise on visfatin.

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