

The Effect of 8-weeks of Aerobic Exercise Training on Plasma Visfatin and Lipid Profile of Overweight Women

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Abstract

Purpose: The wide prevalence of obesity and associated diseases around the world, makes identifying a method to reduce its risk factors and the complications in obese individuals an important area to research. The present study examined the effect of 8 weeks of aerobic exercise on the plasma visfatin and lipid profile of overweight women.

Material and Methods: 19 overweight females (mean age, 22±1.85 years, weight 77.4 ±10.35 kg, body mass index, 30.3±4 kilograms per square meter, waist to hip ratio 0.85±0.04 and fat percent 33.9±3.35) volunteered to participate in the study and were randomly divided into 2 groups. 9 subjects were assigned to the aerobic training group (training 5 sessions per week, for 8 weeks. The first week began with the 65 percent of maximum heart rate reserve (HRR) and reached 80 percent of HRR in the eighth week). Ten subjects were assigned to the control group. Blood samples were collected at the beginning and end of the research to determine the changes in plasma visfatin levels and the lipid profile of the participants. Dependent and independent T-tests were used to analyze the data ($P \leq 0.05$).

Results: The T-test results showed that plasma visfatin and HDL levels significantly decreased ($p=0.01$), while TG levels increased significantly in the experimental group ($p=0.03$). The weight and body mass index of the experimental group were significantly lower compared to the control group ($p \leq 0.05$). Other variables recorded did not change significantly in the groups.

Discussion and Conclusion: Training-induced weight loss and the eventual changes in the body composition of the experimental group had an important role in reducing the visfatin plasma levels.

Key words: Aerobic exercise training, Visfatin, Overweight women, Lipid profile

Introduction

Obesity, is one of the most common metabolic diseases is the most important threat to health, because it increases the risk of metabolic disorders, including type 2 diabetes, increased fat accumulation in different parts of the body, high blood pressure, cardiovascular diseases and cancer [1]. Adipose tissue actively secretes certain proteins called adipokine or adipocytokine. Indeed body fat is not only an energy source but behaves as an active endocrine organ producing biological materials called adipocytokine [2,3,4]. Visfatin is a new adipocytokine secreted mainly by visceral fat tissue and has an important role in obesity and insulin-resistance problems [5,6]. Although most of visfatin is produced in visceral adipose tissue, also seen in

the skeletal muscle, liver, bone marrow and lymphocytes[7]. Visfatin's biological properties are similar to some cytokines: both increase cell proliferation and appear to have an important role in regulating blood sugar. adipokine, is also a diabetogenic factor and plays a role in pathophysiology of insulin resistance in obesity, type 2 diabetes and changes in fetal growth [8]. This adipokine is mainly expressed in visceral fat of human and obese mice [7,9]. In fact, obesity leads to increased expression and plasma concentrations of visfatin in human and animals. Previous studies have shown that plasma concentrations of visfatin would increase in subjects with abdominal obesity or those who suffer from diabetes. A recent study has shown that with accurate control of blood glucose in diabetic patients, levels of plasma visfatin with glycosylated hemoglobin (HbA1C) would be reduced. Therefore, it seems possible to compensate

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insulin reduction that occurs as a result of pancreatic beta cells dysfunction, with changes in visfatin concentration [8].

Higher visfatin levels in diabetic and obese patients as compared to healthy and thin subjects, confirm the compensatory role of visfatin in improving insulin sensitivity. Furthermore, visfatin as an adipokine secreted by adipose tissue is able to improve the lipid abnormalities associated with diabetes via affecting lipids metabolism. Catalan et al (2009) noted the relationship between plasma concentration and gene expression of visfatin with lipid metabolism in mortal obesity [10]. Sun and colleagues also listed triglyceride as the only predictor of baseline levels of age-independent visfatin [11]. Several studies have been carried out on the response of visfatin to exercise in humans [12, 13]. In the study by George et al (2011), 12 weeks of three different training protocols: aerobic, resistance and a combination of both (3 times a week, 60 minutes each session) in patients with type 2 diabetes, increased visfatin levels, but there was no significant differences between the training groups in this regard. These researchers assumed visfatin as a beneficial adipokine with an increasing effect on insulin sensitivity. Their findings showed that insulin receptor expression in resistance group was 65 percent and in the combination group was 90 percent [14]. Header et al (2006) reported that aerobic exercise for 2 and 4 months significantly decreased plasma levels of visfatin in diabetic patients [12]. The training protocol included 4 months of exercise training on an ergometer with 60-70 percent of heart rate reserve (HRR)¹. In another study the effect of aerobic, resistance and combined training (12 weeks of aerobic training, 45 minutes per session, with an energy cost of 300 kilocalories per session, and resistance training, 20 minutes per session, with an energy cost of 100 kilocalories per session, 5 times a week) were examined on plasma levels of 50 to 55-year old, overweight, non-diabetic women, and the results showed that visfatin levels decreased significantly after 12 weeks of training [5]. In general, aerobic and resistance exercises have beneficial physiological changes including weight loss, and improvements in glucose tolerance, insulin sensitivity, and energy metabolism. Because visfatin is recently discovered, there is a little information regarding the effects of exercise on the alterations of

this protein in overweight human subjects. Therefore understanding the interaction between possible changes in visfatin levels and the weight of the participants can shed more light on the role of visfatin in obesity and weight loss. So this study examined the effects of 8 weeks of aerobic exercise on Visfatin's levels and plasma lipid of overweight women. It seems that exercise offers a useful strategy for weight control, improvement in insulin sensitivity and thus prevention from diabetes incidence.

Material and Methods

The present research was a quasi-experimental one, including a pre- and a post-test and an experimental and a control group. The statistical population of the present study included female students who lived in the dormitory of... university who were selected through a call for volunteers to participate in this project. None of the participants were smokers, took supplementations or suffered from cardiovascular diseases, hypertension, diabetes, irregular menstrual cycles or diseases affecting the biochemical parameters. They were not on any special diets (low calorie, low fat, high protein) and did not have regular physical activity for last 6 months. Among the students with the mentioned criteria, 19 overweight volunteers were selected and were randomly divided into the experimental (n = 9; mean age: 22.5±1.5 years, mean height: 162.2±3.81 cm) and control groups (n = 10; mean age: 21.5±2.2 years, mean height: 157.5±6.61 cm). First, the weights of the subjects were measured using a digital scale with a precision of 0.1 g, and their heights were measured and recorded using a wall height meter. BMI was calculated by dividing weight (in kilograms) to square of height (in meters). Body fat and waist to hip ratio² (WHR) were also measured at the beginning and at end of the study using the body composition measuring device and the Bioelectrical Impedance Analysis method.

A training session started with 10 minutes of warm-up including running and stretching. In the first week, Subjects walked with 65% of (HRR) for 16 minutes and in the eighth week it reached to 60 minutes, with 80 percent of maximal heart rate reserve. And at the end of each session a cool-down was performed which included slow running and stretching for 5 minutes. During exercise, HR was monitored continuously (Polar Electro).

Exercise intensity based on maximum heart rate

1. Heart Rate Reserve

reserve was calculated using the Karvonen formula as follows:

$$220 - \text{Age} = \text{Maximum HR}$$

$$\text{Max HR} - \text{Resting HR} = \text{HRR (heart rate reserve)}$$

$$\text{HRR} \times \text{Training Intensity \%} + \text{Rest HR} = \text{Training HR}$$

Note: To control the intensity of exercise the perceived stress scale (RPE)¹ was used.

Blood samples were collected from the brachial vein of the experimental group, 24 hours before and 48 hours after the last training session in the luteal phase of the menstrual cycle following 12 hours of fasting from 7 pm to 8 am (in the morning). The samples were kept in tubes containing anticoagulant (EDTA)² and were quickly centrifuged, at 2000 rpm for 10 minutes. The obtained plasma was maintained at a temperature of - 70 ° C until assay. Plasma visfatin levels were measured through ELISA method, using human Kits (Human Visfatin, ELISA, CUSABIO BIOTECH, Wuhan, China). The sensitivity of the mentioned method was 0.16 ng per ml and the intra-assay variation coefficient was 9.6 percent. Total cholesterol, HDL and triglycerides were measured with the enzymatic CHOD-PAP and GPO-PAP Enzymatic method using Pars Azmoon kits respectively. The following formula was used in order to measure the LDL concentration:

$$\text{LDL-c} = \text{TC} - (\text{HDL-c} + \text{TG}/5)$$

Statistical Analysis

The normality of data distribution was confirmed by the Kolmogorov – Smirnov test.

Dependent and independent t-tests were used to compare the results of pre- and post- tests , and the experimental and the control groups respectively. data analysis was done using SPSS software (version 15) and the significance level was set at (P≤0.05).

Results

Table 1 shows the demographic characteristics of the control and experimental groups. Based on the results of the dependent t-test, the weight of the experimental group decreased significantly after aerobic exercise (p=0.01).

Also, the results of the independent t-test showed significant weight changes between experimental

and control groups (p=0.01); Body mass index and body fat percent of the aerobic exercise group significantly decreased following aerobic exercise (P≤0.05). The results of independent t-test also showed that the changes in body mass index between aerobic and control groups were significant (P≤0.05). Waist to hip ratio decreased in the control group, but this change was not significant (P≤0.05).

The independent t-test results also showed that the ratio of waist-to-hip in aerobic and control groups (P≤0.05) was significantly different. Net body mass of the control group decreased significantly (p=0.01) and there was also a difference in net reduction of body mass between the aerobic and control groups (p≤0.05).

Table 1: Individual characteristics of the experimental and control groups (mean ± SD)

group	experimental		Control	
	Pre test	Post test	Pretest	post test
Weight (kg)	75.7±10.8	69.9±10.3*	79.1±9.9	79.2±10.2
BMI (kg/m ²)	28.8±4.5	26.6±4.4*	31.8±3.5	31.9±3.6
WHR	0.84±0.05	0.82±0.02*	0.86±0.04	0.83±0.03
lean body mass (kg)	40.8±2.8	41.6±2.9*	42.2±4.9	41.8±5.1**
Body fat %	31.1±4	27.8±4.8*	36.7±2.7	36.7±2.7

*Significant compared to the pre-test, **significant compared to the control group in the same test level

Based on the data presented in Table 2, cholesterol level increased at the end of the study and the related t-test results showed that this increase was statistically significant in the control group (p=0.008) while it was not significant in the experimental group (p=0.06). The results of the independent t-test also showed that differences in the total cholesterol between the experimental and the control groups were not significant (P=0.14) however at the end of the study, triglyceride levels decreased in both groups. Dependent t-test results showed that this decrease was not statistically significant in the control group (P=0.48), but it was significant in the experimental group (p=0.001). results of the independent t-test also showed that differences in triglycerides' changes between the experimental and the control group were not significant (p=0.002). HDL levels decreased in the

1. Rate of Perceived Exertion

2. Ethylene Diamine Tetra Acetic Acid

control group and increased in the exercise group at the end of the study. Findings of the dependent t-test showed that this reduction was statistically insignificant in the control group ($p=0.58$) and significant in the experimental group ($p=0.001$). The results of the independent t-test also showed significant changes in HDL levels between experimental and control groups ($p=0.002$). While at the end of the study LDL levels increased in both experimental and control groups, the results of the dependent t-test showed that this increase was

statistically significant in the control group ($p=0.01$) and insignificant in experimental group ($p=0.36$). Results from the independent t-test also showed significant changes in LDL levels between the experimental group and the control group ($p=0.008$). Based on the results presented in Table 2, changes in visfatin levels were statistically significant in the control group ($p=0.01$), and insignificant in the experimental group ($p=0.07$). It also revealed significant changes in visfatin levels between experimental and control groups ($p=0.02$).

Table 2: Plasma levels of study variables in pre-and post-tests (mean \pm SD)

Variable		Control (n=10)	Experimental (n=9)	P value		
				Within the experimental group	Within the control group	inter group
Visfatin (ng/ml)	Pre	9.65 \pm 4.06	9.05 \pm 4.71	0.709	#0.01	*0.02
	Post	9.52 \pm 4.37	7.50 \pm 4.16			
HDL (mg/dl)	Pre	47.57 \pm 7.57	46.33 \pm 6.94	#0.03	0.58	*0.02
	Post	46.66 \pm 10.13	52.50 \pm 7.22			
LDL (mg/dl)	Pre	25.57 \pm 0.98	18.68 \pm 1.03	0.36	0.010	*0.008
	Post	34.79 \pm 1.25	17.69 \pm 1.21			
TC (mg/dl)	Pre	26.50 \pm 1.75	20.18 \pm 1.17	0.64	0.07	0.14
	Post	38.90 \pm 1.93	16.81 \pm 1.78			
TG (mg/dl)	Pre	24.17 \pm 1.45	18.64 \pm 1.78	#0.001	0.48	*0.002
	Post	22.34 \pm 1.42	38.71 \pm 1.23			

#Significant difference between pre-test and post-test

*Significant in pre-and post-test between experimental and control group

Discussion and Conclusion

Visfatin levels were significantly lower in the experimental group ($p=0.01$) as compared to the control group. Also, the results showed significant differences in these values between experimental and control groups ($p=0.02$). Although visfatin functions are not clear yet but it may have a dual role; its first role is probably an autocrine/paracrine function that facilitates distinction and subsidence of fat cells in visceral adipose tissue, and its other role is an endocrine role to moderate insulin sensitivity in peripheral organs. Therefore, visfatin may facilitate glucose control and lead to the development of obesity [5]. Some studies have reported that circulating visfatin increases in obesity and type 2 diabetes while some of them did not show a relationship between obesity and metabolic variables. Exercise has also been successful in improving insulin sensitivity and reducing the levels of visfatin. In addition, it has been shown that significant reduction in fat mass, insulin resistance and improvement of inflammation may account for the reduction of

visfatin as a result of interventions [15]. Visfatin affects insulin signaling and connects to insulin receptors. It has been shown that the expression of visfatin, affects plasma glucose and lipid concentrations [16].

Lee et al (2010) reported that 12 weeks of aerobic training, 4 sessions per week, 45 to 50 minutes a day, with energy costs equivalent to 400 to 300 calories, led to a significant decrease in plasma visfatin levels in obese adolescents and women [17]. In this regard, Domyh et al (2010) reported that 8 weeks of aerobic exercise, three days a week, at 65-80 percent of maximum heart rate, decreased plasma visfatin levels in middle-aged men. A positive correlation was also observed between visfatin and plasma triglyceride levels and body fat percent [18].

Arsvy et al also examined the relationship between visfatin and parameters of obesity, including BMI and waist circumference and insulin resistance in healthy female subjects. There was no relationship between serum visfatin levels, metabolic parameters and obesity and they stated

that the distribution of fat does not affect visfatin levels in healthy female subjects. Since adipose tissue is associated with lipid metabolism and is responsible for the secretion of adipokines including visfatin, some studies have examined the relationship between levels of visfatin and plasma lipids [6].

The present study showed that HDL and triglyceride levels changed significantly after 8 weeks of aerobic exercise and that changes in LDL and total cholesterol levels were insignificant. The relationship between visfatin and lipid profile could be explained through cytosolic function of visfatin as a phosphoribosyl transferase (NAMPT). Observations have shown that maintaining low HDL plasma triglycerides may be related to NAD metabolism. The relationship between visfatin levels and lipid profile indicated that visfatin may be a link between these two processes. In mice circulating visfatin plays a physiological role in plasma glucose levels. Visfatin's positive correlation with HDL and its negative correlation with triglyceride levels showed that circulating visfatin is a useful marker of lipid metabolism that is probably related to NAD metabolism [19]. Further studies in this field may be helpful in understanding the physiological roles of circulating visfatin.

Based on our results, weight loss, body mass index, improved lipid profiles, and insignificant reduction of visfatin after 8 weeks of aerobic training represents physical activity as an effective strategy in dealing with obesity, overweight and their related complications. Overall, our study showed that 8 weeks of endurance training decreased visfatin levels in overweight women, although this change was not significant. It also seems that in overweight and normal women lipid profile improvement is associated with changes in visfatin levels. However in our study, visfatin levels decreased. So it may be possible to consider changes of these parameters, especially visfatin levels, independent of regular physical activity.

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