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The Effect of Eight Weeks Aerobic Exercise on Visfatin Level in Non-Athletic Young Women, Southern Iran

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Abstract: Visfatin responses to exercise training have previously been reported which has adipocytokine exerting insulin-mimicking effects. This study evaluated the effect of eight weeks aerobic exercise on visfatin level in non-athletic young women, Southern Iran. Thirty non-athletic young women were randomly divided into two equal groups. Group 1 performed 8 weeks aerobic exercise on a fixed bicycle 3 times per week with an intensity of 60% in the first week for a period of 20 minutes. Two minutes were added ach week to the activity and the intensity increased 5% each two weeks to reach 75% of previous heart rate up to a period of 34 minutes. Visfatin level was measured before and after exercise session. Plasma visfatin level significantly decreased from 18.515±14.61 to 15.98±14.53 ng/ml before and after exercise training protocol, respectively. In control group, the changes were from 18.040±4.267 to 18.023±3.782 ng/ml at 1st week and after 8 weeks, respectively. We found that plasma visfatin level decreased after exercise training with weight loss suggesting that changes in visfatin concentration may be associated with beneficial effect of exercise. More studies seem necessary to elucidate the mechanisms responsible for the effects of exercise on plasma visfatin level.

Key words: Aerobic Exercise % Visfatin % Non-Athletic % Women

INTRODUCTION

Adipose tissue, in addition to the storage of lipids function for lipids, has an important role in normal metabolic homeostasis and in the development of several diseases, such as type 2 diabetes, dyslipemia and atherosclerosis [1]. It is not just a depot for surplus energy but rather, is an important organ modulating several biological functions and synthesizes and secretes many cytokines into the circulation including adiponectin and visfatin [2]. Nampt/pre-B-cell colony-enhancing factor/visfatin [visfatin] is released from adipocytes and is a nutrient responsive and is linked to systemic nicotinamide adenine dinucleotide biosynthesis and regulation of pancreatic beta-cell function [3].

It is also called nicotinamide phosphoribosyl transferase (NAMPT), is a 52 kDa protein originally established as pre-B cell colony-enhancing factor (PBEF) cytokine secreted from adipose tissue. It regulates immunity system and plays an important role in the NAD+ salvage pathway and facilitates regulation of lipid and glucose metabolism, especially in exercise-induced weight loss for obesity. It has adipocytokine exerting insulin-mimicking effects [4] and is predominantly secreted by visceral adipocytes with proinflammatory properties [5]. Recently, visfatin, as a pre-beta-cell colony-enhancing factor, was shown to be a highly expressed protein with insulin-like functions located predominantly in visceral adipose tissue [6-8]. Fukuhara *et al.* [6] reported that plasma visfatin level was strongly correlated with the

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quantity of visceral adipose tissue confirmed by computed tomography with a weaker correlation between the quantity of subcutaneous fat and visfatin. Pagano *et al.* [9] revealed that visfatin level was lower in subcutaneous fat regions and higher in visceral adipose tissue of obese subjects in comparison to lean individuals. In contrast, Berndt *et al.* [8] did not report any significant correlation between visfatin level and the amounts of visceral adipose tissue confirmed by CT measures.

Metabolic syndrome and obesity have been known worldwide as clinical markers in early detection of type 2 diabetes and cardiovascular disease [10, 11] suggesting the need to directly target obesity, rather than physical activity in an attempt to prevent the development of metabolic syndrome in the general population, however, the interaction of physical activity levels and obesity are obvious.

Visfatin was shown to be linked to obesity and increased health risks [12].

The insulin-mimetic activity of visfatin was reported to be mediated by its binding to insulin receptor but via a distinct binding site [6]. A recent study revealed an elevation in plasma visfatin level in type 2 diabetes patients [13]. Although the function of visfatin is not fully understood, it may have a dual role including an autocrine/paracrine function facilitating differentiation and fat deposition on visceral adipose tissue and an endocrine role modulating insulin sensitivity in peripheral organs [14].

So visfatin can facilitate glucose control and can promote the development of obesity [14]. Some authors suggested that visfatin may be related to the development of the metabolic syndrome [6] and others reported reverse findings that visfatin gene expression was not associated with the metabolic syndrome in diseased rats in comparison to lean controls [15].

Metabolic syndrome as a common disorder is caused due to a combination of unhealthy diet, sedentary lifestyle and genetic predisposition [16] and is considered as a risk factor for many chronic diseases, such as type 2 diabetes and cardiovascular diseases [2, 17]. Pagano et al. showed that plasma visfatin level reduced in obese subjects [9]. Berndt et al. found no significant correlation between visfatin plasma concentrations and various parameters of insulin sensitivity [8].

Changes in dietary intake including overfeeding, underfeeding, as well as exercise have important roles in adipose tissue metabolism [18].

It was shown that exercise training with weight loss resulted into a significant decrease in plasma visfatin level in non-diabetic Korean women [19]. Low levels of physical activity are related to several components of the metabolic syndrome [20]. Regular exercise was reported to reduce the risk of obesity, type 2 diabetes and cardiovascular disease [21]. Furthermore, it was shown that a change in lifestyle, specifically a decrease in physical activity, caused an increase in asthma prevalence and severity [22].

Many reports have supported the beneficial effects and safety of exercise in asthmatic subjects [23]. Changes in adipokine levels have been an important clue to understand the benefits of exercise. Endurance exercise training was demonstrated to decrease plasma leptin levels independently of changes in plasma insulin levels and body fat percentage [24].

Aerobic exercise training was shown to have many beneficial physiological responses in obese people such as improvement in glucose tolerance and insulin sensitivity that are linked with changes of visfatin concentration [19, 25]. The 12 week together with resistance and aerobic training program was shown to be significantly beneficial to body composition and metabolic syndrome factors, as well as lowering visfatin levels in obese middle-aged women [12].

Visfatin responses to exercise training have previouly been reported [19]. An increase in physical activity can manage metabolic syndrome to sustain a healthy weight [26]. It was suggested that combined resistance and aerobic exercise training is better than aerobic exercise alone to improve metabolic indicators of metabolic syndrome [12]. Data from Strasser and Schobersberger, (2011) study have revealed that resistance exercise training can be an alternative to aerobic training to improve body composition and reduce percent body fat in obese patients. It should also be noted that resistance training can mobilize visceral and subcutaneous adipose tissue in the abdominal region [27].

Therefore, any strategy to prevent the risk of obesity, type 2 diabetes, metabolic syndrome and cardiovascular diseases would be of great importance to reduce the therapeutic costs of the patients [28-31]. In this relation, the biological activity and regulation of the novel adipokine visfatin are still largely unknown and there are conflicting results [32]. This study was undertaken to determine the effect of eight weeks aerobic exercise on visfatin level in non-athletic young women, Southern Iran.

MATERIALS AND METHODS

Twenty-eight young non-athletic female students of Shiraz Islamic Azad University aged between 20 and 30 years were voluntarily enrolled in this study. All subjects were healthy with BMI less than 23.0 kg/m^2. They had a stable body weight (less than 2 kg/6 months weight change) and a sedentary life style (less than 30 min exercise once per month). For all subjects, a medical history was obtained, they underwent physical examination and a fasting blood profile was provided to ensure that they were non-diabetic and with no background of cardiovascular, metabolic and other major diseases or were taking medications that could affect laboratory test results.

We also excluded participants who smoked. An informed consent was provided from each participant before the start of the study and the ethical committee of Shiraz University in accordance with the Declaration of Helsinki of the World Medical Association, approved the study.

The candidates were randomly divided into two equal control and experimental groups. The control group did not undergo any exercise training session and just followed for 8 weeks. In experimental group, subjects were encouraged to have aerobic exercise training for an 8 weeks period, three times per week, for 20 minutes consisting of a brief warm-up at an intensity of 60%. The exercise trainings sessions were supervised by an exercise physiologist.

The training program started at 60% of observed maximal heart rate and gradually increased to 75% of maximal heart rate by week 8. Two minutes were added ach week to the activity and the intensity increased 5% each two weeks to reach 75% of previous heart rate up to a period of 34 minutes (Table 1). Aerobic exercise training included treadmill walking/running (M901T, Motus Co., Seoul, Korea).

Blood samples were taken after a 12-h overnight fast and were kept at -80EC for further assay. Blood samples were collected two times including the beginning (1st week: Before) and the end of the study (8 thweek: After). The glucose oxidase method was employed to measure plasma glucose. Plasma visfatin level was measured using a visfatin ELISA kit (Raybiotech, PM82/6, China).

SPSS software (Version 11.5, Chicago, IL, USA) was applied for statistical analysis. Data are presented as means±SD. Differences between groups were tested using the Pearson and Student's t-test. A p value equal or less than 0.05 was statistically considered significant.

Table 1: Aerobic exercise training protocol among non-athletic young women.

Exercise (week)	Intensity (Max heart rate/min)	Duration (min)
1st	60-55	20
2nd	60-55	22
3rd	65-60	24
4th	65-60	26
5th	70-65	28
6th	70-65	30
7th	75-70	32
8th	75-70	34

RESULTS

The mean age of study subjects was 25±5 years. After 8 weeks of the exercise training program, all subjects had lost weight. Furthermore, plasma visfatin level also significantly decreased from 18.515±14.61 to 15.98±14.53 ng/ml before and after exercise training protocol, respectively. In control group, the visfatin concentration was 18.040±4.267 ng/ml at the beginning of the study and was 18.023±3.782 ng/ml after 8 weeks (Table 2).

DISCUSSION

Our study revealed that exercise training was associated with weight loss and could reduce visfatin level in non-athletic young women. Visfatin as a new adipokine is produced in the visceral adipose tissue of obese mice and humans [6] and has autocrine/paracrine function facilitating differentiation and fat deposition on visceral adipose tissue and laso has an endocrine role modulating insulin sensitivity in peripheral organs [33]. It can facilitate glucose control and promote the development of obesity [14].

Some authors indicated to the visfatin role for development of the metabolic syndrome [6] and some reported opposite findings [15]. Regular exercise was demonstrated to be associated with a decreased risk of obesity, type 2 diabetes and cardiovascular disease [34]. Lucas *et al.* [22] reported that frequent moderately intense activity resulted into protective effects against asthma.

In our study including non-athletic young women, visfatin level reduced after eight weeks aerobic exercise training. Changes in adipokine levels were shown to be an important clue to detect the beneficial effects of exercise. Similar findings confirming our results were reported by researchers. Pagano *et al.* [9] showed that plasma visfatin concentration decreased in obese subjects. Aggloussi *et al.* [35] on working on 42 obese children who performed swimming training showed that exercise training could reduce visfatin levels in these children.

Table 2: Comparison between control and experimental groups regarding visfatin plasma level after aerobic exercise training.

Group		No. of candidates	Mean visfatin level (ng/ml)	Standard deviation	*P* value
Control	Before	15	18.040	4.267	0.98
	After	13	18.023	3.782	
Experimental	Before	14	18.515	14.61	0.01*
	After	14	15.98	14.53	

Blood samples were collected two times including the beginning
(1st week: Before) and end of the study (8th week: After).

Straburzyñska-Migaj *et al.* [36] who studied on 28 males with systolic heart failure referred for cardiopulmonary exercise revealed that serum visfatin concentration in these subjects were significantly lower in comparison to healthy controls. Roupas *et al.* [37] also reported that visfatin was downregulated after a chronic intensive exercise plan. Choi *et al.* [19] also showed that plasma visfatin level decreased after exercise training with weight loss. Identical results were noticed in Kadoglou *et al.*'s study [11] on 247 men and women denoting that moderate intensity physical activity could ameliorate visfatin level.

Lowering visfatin levels were seen in obese middle-aged women exercised for 1 hour, 3 days per week too [12]. The plasma visfatin level also significantly reduced in obese female adolescents after 12-week aerobic exercise-training program [38]. Exercise-induced reduction of plasma visfatin among sixteen obese men and women who performed a 12-wk supervised exercise program was previously noticed [3]. It was shown that aerobic exercise training with weight loss could result into a significant decrease in plasma visfatin level in non-diabetic Korean women [19].

Fasting plasma visfatin concentrations were measured in 18 subjects who participated in a supervised aerobic exercise program for 4 months. Plasma visfatin level was shown to be lowered by regular physical exercise [17]. Similar results were observed in Iranian studies reporting a decline in visfatin concentration after an exercise training program [39, 40].

There are some reports in literature showing opposite results of increase in visfatin level after exercise. Rudwill *et al.* [41] in their study on 23 men showed that physical activity itself could increase visfatin concentrations. Sliwicka *et al.* [42] on 10 young male triathletes undergoing incremental running exercise demonstrated a significant increase in visfatin concentration. Plasma visfatin level increased significantly after a 3-month period moderate aerobic exercise in fifty healthy young female professional basketball and handball players [43]. A significant increase in plasma visfatin was also found immediately after a single session of a running-based anaerobic sprint exercise in six young, physically fit men [7].

Even most of studies demonstrated a decrease in visfatin concentration after physical trainings but the conflicting results of few studies mentioned above regarding increase in visfatin concentration after exercise may be due to differences in candidate selection, the exercise training protocol and the weight loss and body composition changes of participants after the training programs [3]. Our findings have reached to the point that exercise training was associated with weight loss and reduction in visfatin level among non-athletic young women showing that changes in visfatin concentration was associated with beneficial effects of exercise. More studies seem to be necessary for building up the mechanisms responsible for the effects of exercise on plasma visfatin level.

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