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The Effect of exhaustive aerobic exercise on responses of serum visfatin and insulin resistance in overweight middle-aged men trained

Hiwa Ahmed*, Arsalan Damirchi, Hamid Mohebbi and Rastgar Hoseini

Exercise Physiology, Faculty of physical education & sport sciences, University of Guilan, Rasht, Iran

ABSTRACT

Visfatin produced by adipose tissue, skeletal muscle, liver and has insulin-mimetic actions. The studies shows, regular exercise enhances insulin sensitivity. Because the physiologic role of visfatin in exhaustive aerobic exercise responses are still unclear; The purpose of this study the effect of exhaustive aerobic exercise on responses of serum visfatin and insulin resistance in overweight middle-aged men trained. Eight healthy trained (age, 47.03 ± 6.69 years; Weight 78.92 ± 6.93 kg; height 170 ± 7.34 cm; Vo_{2max} 42.17 ± 7.30 ml.kg⁻¹.min⁻¹ and body mass index 26.05 ± 1.30 kg/m²); volunteers participated in the current study. Thus, subjects completed an informed consent form and health history questionnaire. The Exercise protocol included was Bulk test. Venous blood samples were collected before, immediately after exercise to assess serum visfatin, fasting glucose and insulin resistance responses. Data were analyzed by the Paired t-test and independent t- test. The results show that serum visfatin significant increased after exhaustive exercise (Pre 8.61 ± 0.61 vs Post 10.51 ± 1.57 ; $P=0.031$); while it remained unchanged in insulin resistance (Pre 1.19 ± 0.36 vs Post 1.10 ± 0.41 ; $P=0.58$). Therefore, promotion in physical activity level could be an effective way to increase visfatin and decrease overweight in middle-aged men trained.

Key Words: Serum Visfatin_ Exhaustive aerobic exercise_ Insulin resistance _ Middle-aged men

INTRODUCTION

Today obesity and overweight is one of important factors of mortality and chronic and fatal diseases. Obesity and overweight is defined as excessive accumulation of fat in the body. Fat tissue in addition to the storage and release of triglycerides can secrete many proteins which these proteins have role in cholesterol metabolism, immune system actions, regulation of energy cost, insulin action and nutrition [1]. Also fat tissue in addition to storage of fat has an important role in homeostasis of whole body as an active tissue by secretion of various hormones that called Adipocytokine. Adipokines have role in the physiological and pathophysiological routes by several mechanisms and in practice they could have protective or predisposing role in getting people to chronic diseases [2, 3]. Visfatin is one of the Adipokines that mostly secreted by visceral fat tissue and its gene expression and plasmatic levels reduces in obese humans. Furthermore, increasing concentrations of visfatin were independently and significantly associated with type 2 diabetes [4]. The metabolic effects of visfatin are apparently mediated by the binding to and activation of the insulin receptor [5]. 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 [5, 6]. Previous studies suggested that visfatin is a mediator of glucose homeostasis with a potential antidiabetic effect. Exercise is a metabolic and neuroendocrine stressor that mobilizes lipids for energy and is a cornerstone treatment for obesity and diabetes [7, 8]. Exercise also stimulates the secretion of proteins and cytokines from adipose tissues, including leptin, adiponectin and interleukins, all of which play an important role in metabolism [9, 10].

Exercise as the most important part of weight loss programs has a profound effect on nutrient balance and insulin sensitivity [8, 9]. O'Leary et al reported that reductions in visceral adipose tissue after aerobic exercise training results in glucose metabolism and is associated with the reversal of insulin resistance in older obese men and women (9). Previous acute exercise studies have examined changes in plasma visfatin responses after a single exercise bout [11, 12]. For example, Shekholeslami et al., (2011) reported in our previous investigation that serum visfatin levels were reduced significantly following moderate exercise for 30-min in nine healthy male subjects [12], 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 [11].

However, studies examining the effects of exercise on circulating visfatin are limited [13, 14]. Some studies have reported decrease [12] or increase [11] in visfatin levels after acute exercise. To our knowledge, no studies have yet investigated the effect of exhaustive aerobic exercise on serum visfatin concentrations. For this reason the purpose of this study was to effect of exhaustive aerobic exercise on responses of serum visfatin and insulin resistance in overweight middle-aged men trained.

MATERIALS AND METHODS

Subjects

We used a sample size consisting of 12 subjects to factor in the subjects who would be dropped from the analysis. The subjects were overweight middle-aged men trained aged 40–60 years, who had >25% body fat, and exercised at the Guilan University fitness center in Iran. However, 2 subject from was excluded because they attended only part of the exercise program, and 2 subject from was excluded because he did not participate in the test conducted at the end of the study. Thus, 8 subjects completed the pre- and post-study assessments. All the subjects submitted a written consent form, and all the study procedures were approved by the Human Care and Use Committee of the Society of Sport Research Institute at Guilan University. The characteristics of the subjects are shown in Table 1.

Table 1: Characteristics of the subjects (Mean $\pm$ SD)

Variables	Age, years	Height, cm	Weight, kg	BMI, kg/m ² *	Vo2max
Mean $\pm$ SD	49.75 $\pm$ 7.26	170. $\pm$ 7.34	78.92 $\pm$ 6.97	26.65 $\pm$ 1.30	45.13 $\pm$ 6.89

* BMI; Body Mass Index

Exercise program

All the subjects were asked to stretch their entire body before (warm up, 10 min) and after (cool down, 10 min) test. They performed a Bulk test; which consisted of participants started walking on treadmill, from the speed of 03.5 km (3.3 miles per hour) and the slope of zero percent. The slope is set to 2% (1.2°) after 1 minute and then every minute thereafter, the slope is increased by 1% (0.6°), and continues until the participants were unable to maintain. Exercise intensity was monitored during the training sessions by using a Polar real time system (Polar S610, Finland).

Analytical Methods

Blood samples were obtained from all subjects, after they had fasted overnight for 12 h, before exercise and immediately following exercise. The aliquots were frozen and stored at -80°C for subsequent analysis (within 2–3 weeks). The samples were analyzed for visfatin, insulin and glucose. Serum visfatin concentration was determined by an enzyme immunoassay [Serum Visfatin C-Terminal (Human); Phoenix Pharmaceuticals, Burlingame, Calif., USA]. Assay sensitivity was 2.2 ng/ml, and intra-assay and inter-assay coefficient of variation of <10%, and <15%. Serum glucose was determined by an enzymatic, colorimetric method (glucose oxidase-amino antipyrine; Pars Azmoun, Tehran, Iran), and the intra-assay coefficient of variation and sensitivity were 1.3% and 1.0 mg/dl, respectively. Serum insulin Was determined by an enzyme-linked immunosorbent assay (Monobind Inc, USA); the intra-assay coefficient of variation and sensitivity were 4.1% and 0.07_ g/l, respectively. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the following formula [24]. Fasting Insulin ($\mu\text{U} / \text{ml}$). Fasting Glucose (mmole /L) / 22.5

Statistical analysis

All the descriptive data were expressed in terms of mean $\pm$ standard deviation. Paired t-test was used to examine the differences in subject characteristics between the groups at pre and post test. All the analyses were performed using SPSS version 16.0. The statistical significance level was set at $P < 0.05$.

RESULTS AND DISCUSSION

The results show that serum visfatin significant increased after exhaustive exercise (Pre 8.61 ± 0.61 vs Post 10.51 ± 1.57 ; $P = 0.031$); while it remained unchanged in insulin resistance (Pre 1.19 ± 0.36 vs Post 1.10 ± 0.41 ; $P = 0.58$).

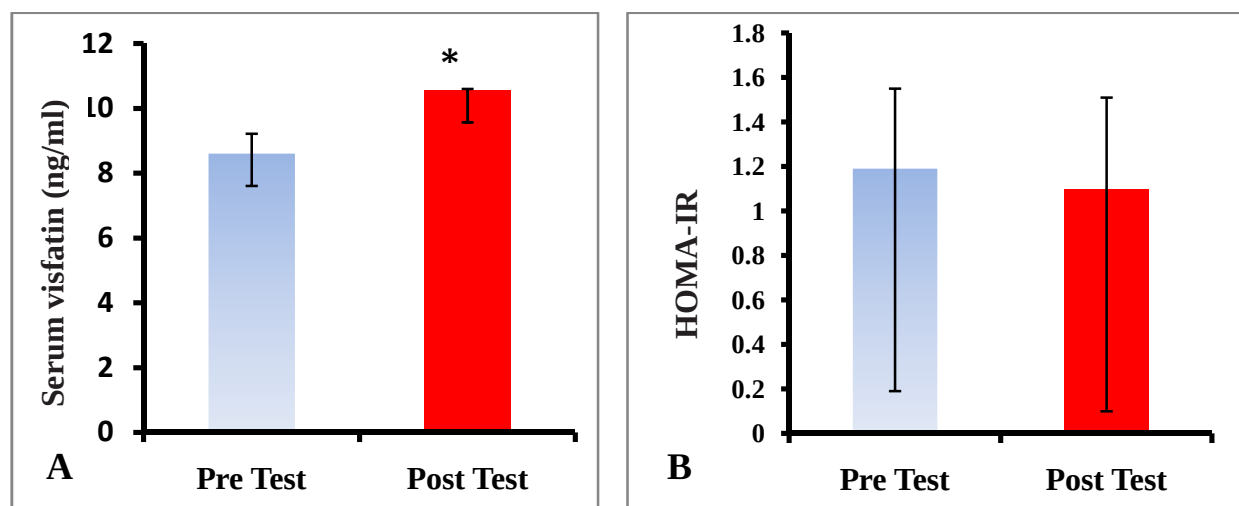


Fig. 1. Serum visfatin (A) and HOMA-IR (B) concentrations in overweight middle-aged men trained Pre and Post exhaustive aerobic exercise.

$P < 0.05$ * Tested by Paired t-test

This study focused on the effectiveness of an exhaustive aerobic exercise on responses serum visfatin and insulin resistance in overweight middle-aged men trained. Results of present study showed that exhaustive aerobic exercise has significant effect on increase of visfatin. To our knowledge, this is the first study to report a significant increase in serum visfatin concentrations as a result of acute exhaustive aerobic exercise. Visfatin was recently identified as a new adipocytokine that was highly enriched in the visceral fat of both humans and mice [15]. While plasma visfatin levels are increased in obesity and insulin resistant states, the specific role of visfatin in insulin resistance is unclear because of difficulties in replicating data showing that visfatin expressed insulin-like activity and could bind to the insulin receptor, thereby lowering blood glucose levels [5, 15]. Frydeland-Larson (2006), showed that exercise induces increase in expression of visfatin mRNA in subcutaneous fat tissue of healthy males [16]. In our study, contrast to some previous studies [17, 18], reduced plasma visfatin levels were associated with a significantly decreased body mass, fat mass, body fat percent. Although possible 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 [17]. 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 [19]. 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 [20]. More recent studies by Revollo et al (2007) have shown that visfatin is homologous to the enzyme nicotinamide phosphoribosyltransferase that is responsible for intracellular and extracellular nicotinamide adenine dinucleotide biosynthesis, and is involved in insulin secretion from pancreatic islets. In this latter context, the increase in visfatin and insulin immediately after intense anaerobic exercise illustrates the parallel interplay between the bioenergetic demands of exercise and the target organs that serve to regulate glucose homeostasis [21]. It seems that the reason increase significant in visfatin levels in this study was due to absence of change in visceral adipose tissue. It also can be result in high intensity or type of exercise protocol. Further studies, however, is necessary to determine this expectancy and explanation the mechanisms responsible for the effects of exercise training on visfatin. It is well established that acute physical activity and endurance exercise training lead to enhancements of insulin-mediated glucose metabolism in healthy individuals and in normal rodent models [22, 23]. In normal rodent models, moderate- or high-intensity exercise training can improve glucose tolerance [24, 25], whole body insulin sensitivity [26], and insulin action on skeletal muscle glucose transport [27, 28]. The protein expression of GLUT-4 appears to play an important role in the capacity of a skeletal muscle for insulin stimulation of glucose transport [29]. The increased insulin action on skeletal muscle glucose transport after exercise training is associated with increased GLUT-4 protein expression [22, 27, 28], as well as adaptive responses of enzymes involved in glucose phosphorylation and oxidation [23]. On the bases of these observations, exercise represents an important potential intervention for improving the metabolic status of insulin resistant individuals.

Results of present study showed that was not significant insulin resistant after exhaustive exercise (Pre 1.19 ± 0.36 vs Post 1.10 ± 0.41 ; $P = 0.58$). An acute bout of exercise (30 min at $\sim 70\%$ of $\dot{V}O_2$ max) by untrained rats does not improve intravenous glucose tolerance [25]. Likewise, a single bout of prolonged aerobic exercise (30–60 min at ~ 60 – 70% of $\dot{V}O_2$ max) will normally not improve the diminished glucose tolerance of insulin-resistant Type 2 diabetic subjects, as evaluated with a standard oral glucose tolerance test (95). In contrast, a prior bout of prolonged

moderate-intensity exercise (45 min of cycle ergometry at 45% of VO₂ max) performed by Type 2 diabetic subjects can reduce the glycemic excursions and elevated plasma insulin levels during the 4-h period after a breakfast meal containing 56% carbohydrate, 30% fat, and 14% protein [30]. Several mechanisms have been proposed to be responsible for the increases in insulin sensitivity after exercise training [31, 32]. These include increased post-receptor insulin signaling (Dela et al. 1993) [31], increased glucose transporter protein and mRNA (Dela et al. 1994) [32], increased activity of glycogen syntheses and hexokinase (Ebeling et al. 1993) and increased muscle glucose delivery.

Although insulin levels have been suggested to play a role in acute changes in adiponectin levels [33, 34]; it seem unchanged in insulin resistance; possibly was due to no change in insulin and glucose in the present study. Finally, mechanisms linking adiponectin to insulin are less unclear and further studies are necessary to clarify why in certain case adiponectin remains stable in spite of hypoinsulinemia. Further studies are needed to determine the mechanisms which intervene in the regulation of the synthesis and the release of adiponectin after the exercise and why this response is delayed.

CONCLUSION

The goal of the present study was to determine effect of the effect of exhaustive aerobic exercise on responses of serum visfatin and insulin resistance in overweight middle-aged men trained. The results of the study showed that exhaustive aerobic exercise of visfatin seed significantly increased visfatin levels and no chinchos on insulin resistance. Therefore, one can say that exhaustive aerobic exercise seed can be regarded as a treatment or preventive method for diabetes. More studies are definitely needed since there are limited information and few studies in this field.

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