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The Effect of Acute Exercise on Serum Vaspin Level and Its Relation to Insulin Sensitivity in Overweight Elderly Men

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Abstract

***Background*:** Vaspin is a new discovered adipocytokine which is a member of serine protease inhibitor family secreted from adipose tissue and might play a role in insulin sensitivity. The purpose of this study was to investigate the effect of acute exercise on serum vaspin levels and its relation to insulin sensitivity in overweight elderly men.

***Materials and Methods*:** In this semi-experimental study, 12 healthy elderly men volunteers randomly selected and performed one session aerobic exercise including 30 minutes of cycling at 70-75% of HRmax, which was followed by 30 minutes of recovery. Three blood samples were taken before exercise, immediately after exercise and after 30 minutes of recovery. Data were analyzed by repeated measure ANOVA and Bonferroni test and Pearson's correlations were performed to identify possible relationship among the assessed variables. Statistical significance was set at $p \leq 0.05$.

***Results*:** There were no significant differences for vaspin across time. Insulin and glucose concentration and insulin resistance decreased immediately after exercise. However insulin concentration and insulin resistance returned to pre-exercise level at the end of recovery. Furthermore, no significant correlations were observed among the variables assessed except for the expected between insulin level and insulin resistance.

***Conclusion*:** These results indicate that a sub-maximal aerobic workout does not result in significant changes in vaspin levels in elderly men. Furthermore, we observed that vaspin is not associated with insulin sensitivity in this study.

Introduction

There is a global rise in obesity in industrialized countries and obesity is associated with insulin resistance, type 2 diabetes mellitus (noninsulin-dependent diabetes mellitus: NIDDM), dyslipidemia, and hypertension [1, 2]. When weight is gained, hyperplasia and hypertrophy of adipocytes within adipose tissue are found [1, 2]. The recent researches demonstrated convincingly that fat cells differentially secrete various proteins, so-called adipokines, which link obesity with components of the metabolic syndrome [1-3]. Recently visceral adipose tissue-derived serpin (vaspin) was identified as a member of serine protease inhibitor family in Otsuka Long-Evants Tokushima fatty rats.

Vaspin is a recently discovered adipocytokine predominantly secreted from visceral adipose tissue. Human vaspin mRNA was reported to be expressed in visceral and subcutaneous adipose tissue which might plays a role in insulin sensitivity [3-5] Hida et al [6]. reported that administration of recombinant human vaspin to a mouse model of diet-induced obesity improved glucose tolerance and insulin sensitivity. On the other side, physical inactivity is a well known risk factor for cardiovascular disorders, metabolic syndrome and NIDDM development [7-10] and aerobic training has been shown to reduce adiposity and insulin resistance in obese adults [8].

Physical exercise (chronic/acute) has been shown to influence insulin sensitivity. Moreover previous researches reported that acute and chronic exercise alters serum levels of adipokines and there is a relation between insulin sensitivity and adipokines such as adiponectin, chemerin and leptin [11-16].

Youn et al. reported that a 4-week training program increased vaspin concentration in normal glucose-tolerant, impaired glucose-tolerant and type 2 diabetic subjects [4]. While we know a lot about the mechanisms by which adiposity leads to insulin resistance [7] and how exercise increases insulin sensitivity [15-17], it hasn't been yet reported about the acute exercise-induced changes in vaspin concentrations, which may provide a link with insulin sensitivity. Whereas, some studies have reported that there is a correlation between vaspin levels and insulin sensitivity, only one study have studied the serum vaspin response to acute exercise and its relation to insulin sensitivity. Therefore, we aimed to study the effect of acute exercise on serum vaspin and its relation to insulin sensitivity in overweight men.

Materials and Methods

Twenty overweight male subjects volunteered to participate in this study. All subjects completed a medical questionnaire and a medical examination to ensure that they were not taking any medication, were free of cardiac, respiratory, renal, and metabolic diseases, were not using steroids, and were in good health. Also individuals who smoked were not allowed to participate in this study. Subjects were matched in terms of physical characteristics and activity levels.

All subjects were screened to assess physical activity level using a modified Minnesota leisure time physical activity questionnaire that correlates well with exercise testing. Then, twelve recreationally active subjects [age 63.41±2.53 years, height 173.17±6.27 cm, weight 76.90±7.37 kg, and body mass index 25.61±1.52 kg/m^2] were elected to perform exercise protocol. Only those subjects who reported previous history of recreational sport were selected. All participants were informed about the purpose and risks of the study before written informed consent was obtained. The exercise protocol was approved by the ethics committee of the Tabriz University of Medical Sciences, Iran.

One familiarization session was designed to habituate subjects with the testing procedures and laboratory environment. All basic measurements including body mass, height, body fat percent and waist-to-hip ratio (WHR) were done in this session. Body mass index (BMI) was calculated as body weight (kg) divided by height (m^2). The skin folds were obtained using Harpenden skin fold caliper (SlimGuioe, US) on the right side of the body at the following sites: triceps, abdominal, and suprailiac. All measurements were taken in triplicate and average values at each point were used to estimate body fat percent using Jackson-Pollack equation. After familiarization and body composition measurements, subjects were requested to attend the laboratory after an overnight fast, having abstained from exercise for 48 hours and caffeine for 24 hours preceding the trial. The exercise tests were conducted in the morning (from 8:00 am until 10:00 am). The laboratory had an ambient temperature of 20 to 22°C and a relative humidity of 45-50%. The exercise test commenced with a 5-minute warm-up period (riding a stationary bicycle together with some light stretching exercises). After warm-up subjects exercised continuously for 30-min at a power output corresponding to 75% HRmax (HRmax=220-age). Heart rate was monitored during the exercise using a polar heart rate monitor (PE3000, Polar Electro, Kemple, Finland) and pedaling frequency of 65±5 RPM was maintained. The exercise was followed by 30-min recovery, with the subjects remaining seated throughout. After reporting to the laboratory, subjects were requested to remain quietly in a seated position for 30 minutes, after which a venous blood sample was removed. Two further blood samples, each, were withdrawn immediately post-exercise and 30 minutes into recovery. Plasma was separated by centrifugation within 15 min of collection.

The samples were frozen and stored at -70°C for subsequent analyses performed within 4 weeks. Vaspin was analyzed by commercially available enzyme linked immunosorbent assay (ELISA kits, Biovender, Germany) with a sensitivity of 0.01 ng/ml and an intraassay coefficient of variation (CV) of 6.5%. Moreover glucose level was determined by enzymatic (GOD-PAP, Glucose oxidase-amino antipyrine) colorimetric method (Pars Azmoun, Tehran, Iran) and insulin level was measured by enzyme linked immunosorbent assay (ELISA kits, Monobind, USA) with a sensitivity of 0.182 pg/ml and an intraassay coefficient of variation (CV) of 4.3%. Likewise insulin resistance index was calculated regarding the homeostasis model assessment (HOMA-IR) according to the formula: HOMA-IR= [fasting glucose (mmol/l)×fasting insulin (mU/l)/22.5]. All data were first checked for normal distribution using Kolmogorov-Smirnov test. Then statistical analyses were performed using the repeated measures of ANOVA to determine the effect of exercise and recovery on responses of all parameters. When ANOVA indicated the presence of a significant difference, Bonferroni post-hoc test was used to determine the differences. Relationships between the parameters were examined with the Pearson's correlation test (r). All statistical analyses were performed using the SPSS-18 statistical software package. p-value≤0.05 was considered significant.

Results

Characteristics of the study participants are shown in table 1. Changes in assessed variables are shown in table 2. Glucose concentration decreased in response to 30-min cycling (p=0.001) and increased by the end of recovery. However insulin concentration and insulin resistance index decreased in response to 30-min cycling and returned to pre-exercise level at the end of recovery (p=0.001, p=0.0001). There were no significant changes in vaspin concentration in response to 30 min cycling and recovery. We observed a significant correlation between post-exercise insulin levels and insulin resistance (r=0.966). We didn't find any significant correlation among other variables at any measurement times.

Table1. Characteristics of the study participants (N=12)

Variable	Mean±SD
Age (year)	63.41±2.53
Height (cm)	173.17±6.27
Weight (kg)	76.90±7.37
BMI (kg/m^2)	25.61±1.52
Body fat (%)	27.5±2.7

Table 2. Changes in variables before exercise, after exercise and recovery (mean±SD) (N=12)

	Pre-exercise	Post- exercise	Recovery
Glucose (pg/mL)	112.50±11.51	100.00±9.84	105.83±12.08
Insulin (pg/mL)	8.30±3.56	3.52±3.39	6.35±2.60
HOMA-IR	2.26±0.94	0.86±0.81	1.64±0.64
Vaspin (ng/mL)	0.7±0.4	0.58±0.34	0.63±0.44

Discussion

The aim of this study was to investigate the effect of acute exercise on vaspin levels and its relation to insulin sensitivity in overweight elderly men. In the present study glucose concentration decreased significantly in response to 30 minutes cycling and remained elevated at the end of recovery. These findings support results of previous studies which showed significant reduction in glucose concentration. Brouns et al. reported that levels of plasma glucose were decreased in response to exercise [18]. The mechanisms by which a single session of endurance exercise could change plasma glucose levels are known. Glucose must travel the path from blood to interstitium to intracellular space and then be phosphorylated to glucose 6-phosphate (G6P). Movement of glucose from blood to interstitium is determined by skeletal muscle blood flow, capillary recruitment and the endothelial permeability to glucose. The influx of glucose from the interstitium to intracellular space is determined by the number of glucose transporters in the sarcolemma and the glucose gradient across the sarcolemma. Any change in glucose uptake occurs due to an alteration in one or more of these steps. Exercise increases blood flow and both the movement of glucose from blood to sarcolemma and the permeability of the sarcolemma to glucose [19]. In agreement with the literature insulin concentration decreased in response to 30-min cycling. However insulin concentration returned to pre-exercise level at the end of recovery [20-22]. Previous data suggest that exercise-induced norepinephrine secretion inhibits the release of insulin from pancreatic beta cells [23]. However we observed that insulin resistance index significantly decreased in response to 30-min cycling and returned to pre-exercise level at the end of recovery. Majority of studies have reported significant decrease in insulin resistance index after both acute and chronic endurance exercise [21, 22, 24, 25]. Previous studies indicated that the mechanism by which insulin resistance index decreases, is independent from insulin concentration. Exercise increases the number of glucose transporters (GLUT4) in the sarcolemma that leads to decrease in insulin resistance index and glucose uptake during exercise [19]. Furthermore, there were a positive correlation between insulin and insulin resistance index which is in accordance with previous studies [12, 26].

The main finding of the present study is that acute exercise did not affect the levels of vaspin as well as there were no significant correlations between vaspin and insulin resistance (HOMA-IR). Vaspin is an adipocyte-released hormone that has been reported to be affected with obesity and impaired insulin sensitivity and may play a role in insulin sensitivity [3, 4, 6]. Previous studies have shown that lifestyle modification, calorie restriction, weight-loss and physical activity might influence serum vaspin concentration [4, 27, 28]. Youn et al. reported that physical training for 4 weeks in untrained individuals causes increased serum vaspin concentrations with weight loss [4]. On the other hand Oberbach et al. reported a significant reduction of serum vaspin concentrations after 4 weeks of training in healthy young men [29]. Lee et al. also reported that vaspin levels significantly decreased in obese children after seven days lifestyle modification including physical activity, dietary intensive modification [28]. Ghahremani et al. investigated the effect of acute resistance exercise on serum vaspin concentration in overweight women.

They reported that one bout of resistance exercise has no significant effect on serum vaspin levels [30]. To our knowledge, only Oberbach et al. has investigated the effect of acute bout of aerobic exercise on serum vaspin concentration. In contrast to the results of our study, they reported significant reduction in serum vaspin concentrations after a 1-hour acute aerobic exercise bout in healthy young men. These authors concluded that vaspin serum concentrations decreased by exercise-induced oxidative stress [29]. Based on previous studies, the data about response of vaspin to acute bout of exercise is still scarce and it is difficult to make comparisons. It seems that the differences between the results found in our study and those reported by Oberbach et al. may be related to the exercise protocol, and subject's characteristics (overweight elderly men vs. healthy young men). More studies are required to understand the responses of vaspin to acute exercise. It has been reported that obesity and insulin resistance increase vaspin concentration and it might represent a compensatory response against obesity and insulin resistance [3, 4, 6]. Therefore vaspin might represent a defense mechanism against insulin resistance [31]. Some studies have reported that physical training and weight loss are associated with a reduction in vaspin levels, insulin and insulin resistance (HOMA-IR) [4, 27, 28]. In our study, serum vaspin levels were not correlated with insulin resistance at any measurement times. Oberbach et al. suggested that vaspin serum concentrations are decreased by exercise-induced oxidative stress in response to acute exercise, but not by exercise-associated improvement in insulin sensitivity [29]. In contrast, Youn et al. suggested that vaspin improves insulin sensitivity and represents a new biomarker for obesity and impaired insulin sensitivity [4]. More studies are needed to identify correlation between vaspin and insulin resistance. In conclusion, it appears that acute aerobic exercise doesn't result in significant alteration in vaspin levels in healthy overweight individuals. Therefore, this adipokine may not be responsible for the well-known effect of acute exercise on insulin resistance. One limitation for present study is that all subjects employed were healthy. Diabeteic patients and obese subjects may show different results and further studies on patients are warranted. Furthermore we couldn't control subject's physical activity and diet during the study period. We just limited participant's physical activity and diet a day before reporting to laboratory.

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Authors' Contributions

All authors had equal role in design, work, statistical analysis and manuscript writing.

Conflict of Interest

The authors declare no conflict of interest.

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