The effect of acute exercise on vaspin and chemerin levels in obese men

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Abstract

Introduction: Vaspin and chemerin, secreted from adipose tissue, are associated with insulin resistance. vaspin and chemerin have been shown to increase insulin sensitivity, as well as decrease the risk of diabetes. The purpose of the research was to explain the effect of acute submaximal exercise on vaspin, chemerin and insulin resistance in obese men.
Materials and Methods: Nine obese subjects were randomly selected with age (22.33 ± 1.87) and body mass index (31.16 ± 2.55). The acute exercise was the astrand bicycle ergometer test. The blood samples were taken from subjects before, immediately after exercise, and 30 minutes after exercise. Repeated measures ANOVA with SPSS 24 software were used to analysis of all data.
Results: The results showed a significant decrease in vaspin level after acute exercise ($P<0.05$). There were no significant changes in insulin resistance and chemerin immediately after acute exercise and 30 minutes after exercise ($P>0.05$). There was a significant correlation, after exercise, between vaspin and chemerin ($P<0.05$).
Conclusion: It seems that acute submaximal exercise was effective in decreasing vaspin. But, response of chemerin to submaximal exercise was not significant. However, the exact effects of acute exercise on other adipocytokines are not clear yet.

Keywords: Vaspin, Chemerin, Obese, Acute exercis

Introduction

Increased adipose tissue is known as a risk factor for a variety of diseases (1). Obesity is known as a risk factor for type 2 diabetes, dyslipidemia, and cardiovascular disease. Nowadays, most researches are focused on endocrine function, the pathophysiology of obesity, and metabolic disorders (2). In 2016, more than 1.9 billion adults aged 18 years and older were overweight. Of these, over 650 million adults were obese. World health organization reported Globally, an estimated 422 million adults were living with diabetes in 2016, compared to 108 million in 1980. it was estimated that in 2017 there are 451 million (age 18–99 years) people with diabetes worldwide. These figures were expected to increase to 693 million) by 2045. It has been shown that this could be associated with an increased in overweight and obesity (3-5). Various studies introduce the main causes of metabolic syndrome are associated with insulin resistance and inflammatory biomarkers (6-8). Fat tissue Secretes Adipocytokine, which is called vaspin or serpin. The action of vaspin in obese mice can improve glucose tolerance, increase insulin sensitivity, and alter gene expression in genes that are candidates for insulin resistance (9-11). The pattern of

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Circadian rhythms secretion of serum vaspin is precisely the reverse of insulin and glucose. Vaspin has an inhibitory effect on (hK7) that has physiological effects on insulin sensitivity (12). Therefore, vaspin has been known as an adipocytokine to increase insulin sensitivity (13). Chemerin (RARRES2 or TIG2), also known as adipocytokine, is more expressed in the liver and adipose tissue (14). Chemerin has anti-inflammatory effects and plays a role in recalling macrophages to adipose tissue. In studies has been shown that treatment with chemerin shows insulin sensitivity and glucose uptake (by IRS-1 phosphorylation, protein kinase B and GSK3). In general, chemerin plays a vital role in recalling macrophages to adipose tissue and appears to be involved in the development of inflammation and insulin resistance(15,16).Therefore, adipocytokine vaspin and chemerin have different physiological functions in the body that can increase glucose uptake in various ways. Exercise training has beneficial effects on physical fitness, control of body weight, fasting blood sugar, and risk prevention kinds of diseases. It can be associated with an active lifestyle across the life span.. Exercise and exercise training can be preventing and treat atherosclerosis, insulin resistance in skeletal muscles (17, 18). Acute exercise gives different physiological responses to people in the community, especially obese people. It can be precious in designing exercise training with variable intensity. Many studies have shown that exercise alone has clinical benefits, such as insulin sensitivity and maximum oxygen consumption (19). The studies have been observed a relationship between lipid and vaspin plasma secretion and chemerin (20, 21). Also, there aren't various types of scientific studies such as acute submaximal exercise on Vaspin, chemerin, and insulin resistance. It seems, more studies are needed to provide more detailed information on the response obese people to acute exercise. On the other hand, researchers' interest in amount exercise intensity and the response Adipocytokines that related to insulin sensitivity. Therefore, in this study, we follow the answer to this question. Can submaximal acute exercise effect on vaspin, chemerin levels, and insulin resistance in obese men?

Materials and Methods

Nine participants were selected randomly from between forty people students at Islamic Azad University, East Tehran Branch. Inclusion criteria were male, age: 20–25, did not have one-year history of regular exercise, body mass index: ≥ 30 $kg.m^{-2}$, and obese. Exclusion criteria were smoking, infectious disease within past 6 weeks, cardiac, respiratory, renal, and metabolic diseases use drugs for weigh loss or medication. The study was approved by the East Tehran Branch, Islamic Azad University, Tehran, Iran. Written informed consent was obtained from all participants prior to study enrolment.

The method of the present study was Quasi-experimental conditions. The research design included a pretest and posttest plan. Before starting acute exercise, participants have performed the initial assessment. Participant's blood samples were taken before exercise, immediately after exercise, and after 30 minutes of recovery.

In this study, we used the Astrand Cycle Test. Astrand is the submaximal aerobic test that uses in exercise physiology labs. This test estimate maximal oxygen consumption by heart rate on a Cycle ergometer for 6 minutes (22). We used it twice. The first time to estimate the maximum oxygen consumption of participants and the second time for the response of acute submaximal exercise on vaspin and chemerin.

All measurements were performed for body mass index, VO_2max, and fat percentage in the laboratory on the other day before start the acute exercise protocol. Three days later, at four separate sessions, at 8:00 am, participants were performed Astrand acute submaximal exercise, then the blood samples were taken before, immediately,

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and 30 minutes after exercise. exercise intensity was based instruction on the standard astrand cycle test. All participants did not eat food 12 hours before the exercise protocol. They did not have any exercise activity for 48 hours before start the exercise protocol. Furthermore, before the initial blood test, they did not eat caffeinated foods in the last 24 hours. The laboratory had an ambient temperature of 20 to 24°C and relative humidity of 40-50%. Blood samples were taken from the anterior vein of the arm. After blood sampling, samples were centrifuged within 15 minutes at 4 °C. then, serum was separated and frozen at -80 °C. Serum vaspin was measured by Sandwich ELISA kit with a sensitivity of 0.01 ng/ml and intra-assay: cv < 8%, inter-assay: cv < 10%. serum chemerin was measured by Sandwich ELISA kit with a sensitivity of 4.99 ng /l and intra-assay: cv < 8%, inter-assay: cv < 10%. (Bioassay technology laboratory Shanghai Chinese). Glucose was measured by autoanalyzer colorimetric method with a sensitivity of 0.1 mg/dL. (Biorex, England) and Insulin was measured by electrochemiluminescence assay method with a sensitivity of 0.01 µIU/mL (Roche, Germany). The HOMA-IR was calculated using the Matthews et formula (23) and used polar F11 watch for the Astrand test.

Statistical Analysis

The Shapiro wilk test was conducted for the normality of the data. Then, normal distributions were shown for all data. We used parametric statistics. To measure comparisons of the means were used, Repeated measures analysis of variance to determine the effect of acute exercise on all parameters' responses. Then the Bonferroni post-hoc test was used to determine the differences. And the Pearson correlation coefficient was used for the correlation relationship. All statistical analyses were performed using the SPSS 20 statistical software package. A P value < 0.05 was considered significant.

Results

Descriptive characteristics for the 9 participants is With mean age (22.33±1.87), Hight (176.61±7.26 cm), Bodyweight (99.22±5.03 kg), Body mass index (31.16±2.55 kg/m2), fat percent (30.43±2.07) and maximum oxygen consumption (Vo2max) (43.45± 2.55 ml.kg-1.min-1). There were significantly different on vaspin result of repeated measures analysis of variance. The test of within-subjects effects with sphericity assumed (effect exercise on vaspin) (P = 0.004, df = 2 F (1,16) = 8.187) with effect size η= 0.506. according to the significant difference, the Bonferroni test was used to compare means. The results showed a significant difference between the two groups before and immediately after acute exercise (Table 2). there were not significantly different on chemerin result of repeated measures analysis of variance. Test of within-subjects effects with sphericity assumed (effect exercise on chemerin) (P = 0.237, df = 2 F (2,16) = 1.57) with effect size η= 0.165. There were no significant differences in the homeostatic model assessment 1 (HOMA1) result of repeated measures analysis of variance. Test of within-subjects effects with Greenhouse-Geisser epsilon (effect exercise on HOMA1) (P = 0.152, df = 1.2 F (1.2,16) = 2.497) with effect size η= 0.238. Also, there were significantly different glucose within-subjects effects (P = 0.000), but There were no significantly different insulin levels before and immediately after and 30 min after exercise (P = 0.603). We observed a significant correlation between before and immediately after exercise vaspin and chemerin serum levels (P <0.05).

Discussion

Vaspin is known as adipokine with effects of insulin sensitivity (17). Studies have been done on the effects of exercise on vaspin levels, especially regular exercises, followed by losing weight on vaspin (24).

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Table1. Changes in the variables of participants before, after and 30 min after exercise (n=9).

Variables	Mean and standard deviation			Within subjects effects			
	Before exercise	Immediately after exercise	30 min after exercise	F	Sig	Size effect	Observed power
Vaspin (ng/ml)	1.85±1.30	2.30±1.38	2.02±1.20	8.187	0.004	0.506	0.918
Chemerin (ng/l)	317.16±208.88	377.94±199.10	453.72±237.15	1.57	0.237	0.165	0.285
HOMA1	4.21±1.89	3.01±1.16	3.55±1.43	2.498	0.152	0.238	0.289
Glucose (mg/dl)	95±10.10	76.44±5.29	79.44±5.74	19.58	0.000	0.710	1.000
Insulin (µIU/m)	17.51±6.40	15.31±6.28	17.02±6.86	0.522	0.603	0.061	0.121

Data are shown as mean ± SD. HOMA1, homeostatic model assessment 1.

This study showed an increase vaspin levels immediately after exercise and a decrease in 30 min after the recovery time. Therefore, there were significant differences in vaspin levels response to acute submaximal exercise. There was a significant correlation between serum Vaspin levels before and immediately after exercise and 30 min recovery time. Researchers have reported contradictory results on subjects in Vaspin (24-29). The results of this study were similar to Ouberbach, Safarzadeh, and Soori. However, in a study by Ouberbach, the acute boat of exercise on Vaspin was examined after four training weeks. In a study by Bashiri & et al., the study effects acute bout of exercise (on a cycle ergometer) for 30 minutes with 70-75% of maximum heart rate on vaspin levels associated with insulin sensitivity in overweight men; their results contradict those found in the present research in which vaspin levels declined significantly. The probable reason for this difference can be the participants' initial status in the study that participants in our study were all obese. According to Han & et al., obese people have higher levels of vaspin (21), which differences which could be rleated to this problem. The reasons for all these contradictory results are not known. They may be related to differences in age and maturity or changes in other hormones such as human growth hormone (GH) between the groups of participants. Differences in visceral fat could cause these abnormal metabolic responses. Visceral fat releases pro-inflammatory adipocytokines, including TNFα, IL-6, resistin, etc., that promote insulin resistance and lead to diabetes (30). Has been suggested that vaspin could modify insulin function only in the presence of the protease targets in white adipose tissue. related these changes to upregulation of defense mechanisms against insulin resistance (31). Contradictions can result from responses recorded in animal and human subjects, assuming that animal subjects in research processes are placed under more controlled conditions or the exercise protocol's type and intensity. The noteworthy point in these studies could be that people with high initial vaspin levels had insulin resistance in general. Moreover, general metabolic compatibility occurred in response to the exercise training due to improved insulin sensitivity. Vaspin secretion in adipose tissue may decrease because the body responds to insulin resistance development by increasing the vaspin level. Therefore, compared to studies in which the participants do not have insulin resistance or to studies that investigate the response to a single exercise session, it is expected that studies conducted on these people will show a greater significant reduction in vaspin response to exercise. In the present research, all of the participants were obese but none of them had insulin resistance. The significant difference reduction of vaspin in response to exercise in the present research can be attributed to the initial vaspin level or the effect of other adipocytokines related to insulin sensitivity, which were not considered. In the present research, the participants did not have a history of engaging in exercise. The response of vaspin differed in the research by Soori et al. because the participants were in the age range of 48 to 60 years, and vaspin response

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to exercise may differ in different age groups. Gender differences may also result in differences in vaspin response to exercise, considering that females' body fat percentage is higher than in males and can influence initial vaspin levels. Kloting et al showed that there was a significant relationship between expression of vaspin in visceral adipose tissue and body mass index (BMI) and body fat percentage. However, this relationship did not exist in slim participants, which conforms to the findings of the present research. Yuan reported a positive correlation between serum vaspin level and BMI in healthy people but not in diabetes patients (32). No accurate findings have been reported yet regarding the factors influencing vaspin levels in studies into the effects of exercise and exercise training on vaspin levels. As previously mentioned, some researchers consider initial vaspin levels based on body fat percentage, weight loss, and changes in calorie intake (diet) to be the main factors involved in the reduction of vaspin levels caused by exercise and exercise training (29, 32). Still, another group of scientists has suggested that visceral adipose tissue changes are the reason for vaspin reduction because visceral adipose tissue is one of the main sources for vaspin production and the creation of insulin resistance. An opposing group of researchers has not reported any changes in vaspin level in response to exercise and have proposed that vaspin is not expressed due to exercise and different responses are exhibited at rest and after exercise and exercise training (28, 33). It seems that exercise alone is a factor in changing vaspin levels after the exercise and can also influence other metabolic indicators related to vaspin. Factors including age, hormones and adipocytokines, insulin resistance, inflammation,intensity/frequency/and duration of exercise, gender, and biological clock of vaspin may explain why different results have been obtained in various studies.

Studies have revealed that treatment with chemerin results in sending messages of insulin sensitivity and in glucose uptake, and it seems that chemerin is involved in the development of inflammation and insulin resistance (14, 16). In the same vein, researchers became interested in conducting studies on chemerin response (together with the response of other adipocytokines) to exercise and exercise training. However, these studies have yielded different results concerning chemerin response to exercise (34-39). The present study indicated that a single session of submaximal exercise had no significant effect on chemerin levels immediately following and 30 minutes after the exercise. Chemerin levels increased immediately after the exercise, and they showed increases 30 minutes after the exercise, but these increases were not statistically significant. Moreover, there was a significant positive correlation between serum chemerin levels before and immediately following the exercise. Jessie Lloyd et al. studied the effect of an acute bout of aerobic exercise on chemerin levels in obese adults. Chemerin levels did not show statistically significant differences immediately after the exercise and 1-2 hours later. Nevertheless, they increased immediately following the exercise but declined by 12% two hours after the exercise (39). This research agreed with the present study that in the present research, chemerin levels increased 30 minutes after the exercise. In contrast, its levels decreased one and two hours after the exercise in the study by Lloyd et al. This difference was probably caused by when blood samples were taken because chemerin levels return to the initial ones 1-2 hours after the exercise. It must be mentioned that chemerin levels increased immediately after the exercise, but the increase was not statistically significant, and, therefore, the results must be interpreted with greater caution. Rima Chakaroun et al. demonstrated that chemerin mRNA expression increased

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significantly in adipose tissue of patients with type 2 diabetes and was correlated with circulating chemerin levels, percentage body fat, and insulin resistance. The reduction in chemerin expression and in its serum concentration may be related to improved insulin sensitivity and to disease-unrelated inflammation (34). Stefanov et al. studied the reduced levels of circulating chemerin in response to combined resistance and endurance training. Results indicated that reduced chemerin levels were accompanied by a significant decrease in the levels of total cholesterol, TG, and fasting insulin, as well as insulin resistance index, systolic blood pressure, high-sensitivity CRP, leukocyte count, and leptin levels. Therefore, after the six-month training program, chemerin levels declined significantly in the experimental group (35). These results were probably the response of chemerin to exercise training. So, Hun Kim et al. investigated the effects of lifestyle modification on serum chemerin concentration and its association with insulin sensitivity in overweight or obese type 2 diabetic patients. The 12-week lifestyle modification significantly reduced chemerin level compared to the control group. The reduced chemerin level was accompanied by improved insulin sensitivity (36). Neuparth et al studied the positive effect of moderate walking exercise on Portuguese type 2 diabetic patients. Results indicated that BMI and chemerin concentration decreased significantly inactive diabetic patients compared to the inactive ones(38). Of course, it is worth mentioning that baseline chemerin levels in diabetic patients are higher compared to healthy people. More of these studies investigated the effect of exercise training but not to an acute bout of exercise. In general, the differences between these studies can arise from differences in age, gender, sample volume, measurement method, type and duration of the exercise and exercise training, and in the initial condition of the healthy or sick participants. The differences may also result from differences in visceral fat tissue and from the way that other adipocytokines change. However, considering the results of the present research and of other studies, it seems that an acute bout of exercise cannot lower chemerin levels. Nevertheless, these results must be interpreted with greater caution.

Conclusion

Acute bout of submaximal exercise increased vaspin levels immediately following the exercise but reduced it 30 minutes later. However, the chemerin level and the insulin resistance did not change significantly immediately following and 30 minutes after the exercise. Considering the changes made in the present study, it seems that the acute bout of submaximal exercise changes vaspin levels but not chemerin levels. However, the exact effects of acute exercise on other adipocytokines are not clear yet.

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Conflict of interest

The authors declare that no conflict of interest exists.

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