

The Effect of Aerobic Exercise on Growth Hormone, Insulin and Blood Glucose Levels in Non-Athletes Middle Aged Women With High Fat Profile

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Abstract—Obestatin, an anti-hunger peptide, plays an important role in energy balance and body weight. Physiological changes caused by obesity, lack of exercise, and reduced fitness are the potential risk factors for metabolic syndrome, type 2 diabetes, cardiovascular diseases, cancer, and other diseases. The prevention and treatment of obesity are multifaceted problems that are not merely restricted to factors such as genetics, hormone levels, overindulgence in food or a sedentary lifestyle. The present study aimed to examine the effect of 12 weeks of aerobic exercise on growth hormone, insulin, BMI, insulin resistance and blood glucose levels in non-athletes women aged 35 to 50 years old with high fat profile. The results showed that after a 12-week treatment of aerobic exercises, weight, fat percentage, WHR, and BMI in the experimental group were significantly decreased ($P<0.05$). It was also revealed that insulin and insulin resistance in the experimental group was significant. The important point was that the glucose level and obestatin did not show any significant changes ($P<0.05$).

Keywords—*Aerobic Exercise; Growth Hormone; Insulin; Blood Glucose; Non-Athletes Women*

I. INTRODUCTION

Obesity is the biggest challenge of the present century posed to the public health; in most countries, healthcare has to deal with issues and complications arising from the increasing incidence of obesity. The prevention and treatment of obesity are multifaceted problems that are not merely restricted to factors such as genetics, hormone levels, overindulgence in food or a sedentary lifestyle. Energy balance is regulated by a complex mechanism consisting of central and peripheral factors. Ghrelin and obestatin peptides are two recognized environmental factors that appear to play a major role in regulating food intake and body weight [6].

Findings of few studies conducted on this subject suggest the likelihood of physical exercise affecting obestatin levels. Ghanbari, Niaki et al. examined the effect of 6 weeks of jogging on total obestatin levels of the intestine and the fundus. Results of their study demonstrated the significant reduction of obestatin levels in the small intestine and fundus of rats that had exercised, and the lack of a significant change in their total plasma obestatin levels. The reduction in obestatin levels was associated with a significant increase in

liver glycogen and plasma growth hormone and no changes in adenosine triphosphate levels in the small intestine and fundus. Researchers believe that the increased plasma growth hormone and glycogen content of the liver may inhibit the synthesis and secretion of obestatin in the small intestine and fundus. It thus seems that increased levels of the growth hormone inhibit obestatin production in the small intestine and fundus via a negative feedback [8].

Furthermore, evidence suggests that obestatin levels reduce in conditions of insulin-resistance [1]. Given the limited scope of findings, the important role of obestatin as an anti-appetite peptide involved in maintaining energy balance so as to prevent weight gain is still a mystery. Obestatin levels are most likely affected by exercise, causing a change in appetite and weight. Given the short time since its discovery as a peptide, few studies have investigated the effect of exercise on obestatin levels. The present study aimed to examine the effect of 12 weeks of aerobic exercise on serum obestatin levels in obese non-athletes women aged 35 to 50 years old.

II. MATERIALS AND METHODS

This quasi-experimental fundamental study with pretest-posttest and control group was conducted in 2014 in Isfahan. At the beginning of the research, there was a public call for obese non-athletes women aged 35 to 50 years old participants; after the initial screenings, 45 of them with a BMI above 30 were selected. Inclusion criteria were: absence of cardiovascular, respiratory, kidney or metabolic diseases as well as not being on any particular diets (low calorie, low fat or high protein). Participants with a history of exercise, diseases and smoking and also those trying to lose weight were excluded from the study. Prior [9] to the beginning of the exercise program, all participants submitted their written consent. A total of 20 volunteers qualified, who were then randomly divided into an experimental group (10 women) and a control group (10 women).

Study variables were measured, including age, height (using a SECA stadiometer made in Germany with the accuracy of 1mm), weight, fat percentage, BMI, waist to hip ratio (using In Body model 3 by BIOSPACE made in Korea), serum glucose levels (by means of a glucose measuring kit using an enzymatic staining method made by

Pars Azmoon Co. with a sensitivity of 1 mg/dl), serum insulin levels (using a Sandijii ELISA kit by Mercodia made in Uppsala Sweden with a sensitivity of 1 milliunit/liter), insulin resistance (using the Homeostasis model assessment formula based on insulin and glucose levels) and serum obestatin levels (using a special kit by Glory Co. made in the USA based on the ELISA method and used according to the manufacturer's guide).

Participants took part in 12 weeks of aerobic exercise on a treadmill, 3 sessions per week. Each session began with 60-65% of the maximum heart rate after a 10-minute warm-up by brisk walking, stretching and jogging. The first session contained 15-20 minutes of jogging and reaching 60-75% of the maximum heart rate and maintaining it for 25-30 minutes. The third week's exercise started with reaching 75-80% of the maximum heart rate and maintaining it for 35-40 minutes – a pattern that continued until the sixth week. The seventh week's exercise started with reaching 80-85% of the maximum heart rate and maintaining it for 45-50 minutes – continuing until the twelfth week. Every session ended with 10 minutes of slow cooling down through stretching. In order to control exercise intensity, a Polar stethoscope was used along with the equation for determining maximum heart rate based on age and the Karvonen formula (Maximum Heart Rate = 220 - Age). The control group was only monitored by a diet questionnaire.

Dietary intake data were obtained using 24-hour dietary recalls to determine the approximate amount of nutrients received. Participants were asked to list every food and drink they had consumed over the last 24 hours. This questionnaire was filled out by all participants on three non-consecutive occasions once a month over a 12-week period. The questionnaire used was the 24-hour Dietary Intake Assessment by the Department of Clinical Nutrition and Dietetics of Shahid Beheshti University of Medical Sciences.

Blood samples were collected twice; first, 24 hours before starting the first session of the exercises (pretest), and second, 24 hours after the last session of the exercises had ended (end of the twelfth week); at 8 o'clock in the morning following an overnight of fasting and resting, blood was collected in the amount of 10 ml from the anterior vein of the participants' left arm while in a seated position. Samples taken were then poured into sterile tubes and incubated at room temperature for 10 minutes. Their blood serum was then separated from the blood clot by centrifuging at 3000 rpm for 10 minutes and was kept frozen at -70 °C till the measuring stage.

In both groups, descriptive statistics were used to examine participants' characteristics, including age, height, weight, BMI, fat percentage, waist to hip ratio and serum glucose, insulin, growth hormone and obestatin levels. After ensuring the normal distribution of the data using the Kolmogorov-Smirnov test, a dependent t-test was used to make internal comparisons between group members and an independent t-test to compare the two groups with one another. SPSS-19 software was used for analysis of the data and the significance level for computational purposes was decided to be equal to or lower than 0.05.

TABLE I. CHANGES TO THE STUDY VARIABLES FROM THE PRETEST TO THE POSTTEST STAGE IN THE CONTROL AND THE EXPERIMENTAL GROUP (WITHIN GROUPS) (MEAN ± STANDARD DEVIATION)

Variable	Group	Pretest	Posttest	Paired T	Significance Level
Weight (kg)	Control	86.8±6.21	86.32±5.73	1.475	0.17
	Experimental	94.11±5.35	90.59±5.64	4.53	0.001*
Fat Percentage	Control	42.2±2.53	41.9±2.43	1.571	0.15
	Experimental	44.87±4.39	43.29±4.53	3.53	0.006*
BMI (kg/m ²)	Control	33.02±0.98	33.04±1.30	-0.16	0.87
	Experimental	37.21±3.73	35.6±3.64	3.48	0.007*
Waist to Hip Ratio	Control	1.01±0.03	1.00±0.02	0.93	0.37
	Experimental	1.04±0.06	1.03±0.05	4.14	0.002*
Glucose (mg/dl)	Control	6.74±1.77	5.9±1.00	1.72	0.11
	Experimental	15.82±4.03	11.77±2.85	2.99	0.01*
Insulin (mIU/L)	Control	6.74±1.77	5.9±1.00	1.72	0.11
	Experimental	15.82±4.03	11.77±2.85	2.99	0.01*
Obestatin (ng/ml)	Control	0.40±0.09	0.42±0.12	-0.557	0.59
	Experimental	0.46±0.17	0.54±0.21	-1.01	0.33
Growth hormone (ng/ml)	Control	2.04±0.7	1.93±0.7	1.76	0.18
	Experimental	2.18±0.7	1.94±0.5	1.23	0.04*
Insulin Resistance	Control	1.38±0.27	1.23±0.19	1.64	0.13
	Experimental	3.76±1.79	2.57±0.59	2.50	0.03*

* Significance Level $\alpha < 0.05$

TABLE II. RESULTS OF THE INDEPENDENT T-TEST IN THE CONTROL AND THE EXPERIMENTAL GROUPS

Variable	Mean difference	Independent t	Significance Level
Weight (kg)	3.04	3.61	0.004*
Fat Percentage	1.32	2.76	0.013*
BMI (kg/m ²)	1.63	3.40	0.006*
Waist to Hip Ratio	0.01	2.39	0.028*
Glucose (mg/dl)	4.9	1.71	0.115
Insulin (mIU/L)	3.21	2.23	0.04*
Obestatin (ng/ml)	-0.06	-0.69	0.49
Growth hormone (ng/ml)	1.06	1.12	0.064
Insulin Resistance	1.04	2.15	0.057*

* Significance Level $\alpha < 0.05$.

III. RESULTS

Table 1 shows the rate of changes to the various variables within the groups. Results showed a significant reduction in weight, BMI and fat percentage of both groups as a result of

performing aerobic exercises ($P < 0.05$). Results of the comparison made between the two groups are shown in table II. Significant changes were observed in both groups' insulin level and insulin resistance (for the control group, pretest, 1.38 and posttest, 1.23; for the experimental group, pretest 3.76, and posttest, 2.57); however, no significant changes were observed in their obestatin and plasma glucose levels after 12 weeks of aerobic exercise (for the control group, pretest, 0.4 and posttest, 0.42; for the experimental group, pretest, 0.46 and posttest, 0.54) ($P < 0.05$) (table II).

IV. DISCUSSION

Results of the present study showed that 12 weeks of aerobic exercise had caused a significant reduction in weight, fat percentage, BMI, waist to hip ratio, insulin levels and insulin resistance; however, changes in glucose and obestatin levels were insignificant.

Exercise and weight loss work in parallel through separate but interrelated mechanisms to improve metabolic and cardiovascular risk factors. An aerobics course is expected to reduce weight, BMI, fat percentage and waist to hip ratio, which is confirmed by results of the present study. Although mechanisms of spending energy in the exercise of sports and controlling the appetite are effective for weight loss, their method of operation is still unclear. Various hormones playing key roles in regulating the appetite and body weight have been identified, but mechanisms through which obestatin can help regulate food absorption are still unknown. Nevertheless, the appetite suppressing effects of obestatin after an intracerebroventricular injection are indicative of a certain activity taking place in the central nervous system.

Some studies have shown that ghrelin and obestatin play key roles in controlling energy balance and weight [11] [5]. It appears that these peptides perform reverse activities in food intake, weight gain and adipocytes. Studies have demonstrated the sensitivity of ghrelin to conditions of negative energy and its significant role in short-term and long-term energy balance and glucose homeostasis. Vicennati [12] observed that, compared to women of normal weight, obese women had higher levels of obestatin, lower levels of ghrelin and a lower ghrelin to obestatin ratio.

Researchers suggested that increased obestatin levels following weight loss might be the required mechanism for maintaining weight loss [10]. In the present study, while a significant reduction was achieved in participants' weight, their plasma obestatin levels remained unchanged after 12 weeks of aerobic exercise. Plasma obestatin level variations might therefore be related to participants' age and nutritional status.

As the main regulator of blood glucose level, lipid synthesis, adipose protein and glycogen, insulin stimulates muscular and liver cells and inhibits the disintegration of glycogen and lipids and the destruction of proteins. In a study conducted by Granata [4] on rats fattened through 8 weeks of high-fat diet, obestatin caused a reduction in insulin resistance and increased its secretion from the pancreatic Langerhans islets.

The present investigation did not demonstrate a significant difference in growth hormones as a result of 12 weeks of aerobic training which was in line with the findings of some other studies [7]. The results of the studies showed that physical activity is a stimulus or releasing factor for the growth hormone. Some studies have investigated factors like intensity, duration and type of activity and environmental conditions and have concluded that these factors may influence the effect of physical activities on the secretion of growth hormone [7] [10]. Therefore, the data of this study revealed that probably one of the effective factors on the changes of growth hormone was the intensity of the applied activities and obesity percent of the participants. So, it is likely that decrease in obesity and increase in the intensity of exercises and change in the type of activities increase the response of growth hormone to the exercises.

Results of the present study demonstrate that obestatin contributes to the functioning of lipid cells and glucose metabolism; it is thus suggested that it be used for the treatment of insulin resistance. The insulin resistance drop observed in the present study has likely caused increased hypothalamic obestatin levels and has kept plasma obestatin levels unchanged. Overall it seems that the type and intensity of exercises and the age of participants have affected the responses and compatibilities. In addition, the fasting or non-fasting status of participants, their weight and BMI, type of exercise performed and even the timing of post-activity sampling must have affected findings of the study. It is therefore advised that these factors be taken into consideration in future studies. Moreover, since studies demonstrate that high-intensity interval exercise increases fat oxidation and mitochondrial enzymatic activities, investigating the effect of interval exercise on variables examined in the present study is encouraged.

V. CONCLUSION

Overall, results of the present study demonstrated that a 12-week aerobic exercise program does not cause a significant change in plasma obestatin levels while it significantly reduces body compounds and insulin resistance. It thus appears that plasma obestatin levels are not associated with changes of weight and insulin resistance.

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