



## Moderate exercise test is not associated with acute or recovery response of serum leptin in obese individuals

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**ABSTRACT:** Accumulating evidence suggests that leptin plays an important role in the obesity and metabolic syndrome, although the molecular mechanisms for this are less understood. This study aimed to determine the acute and recovery response of serum leptin to a moderate exercise test in adult obese men. For this purpose, fifteen sedentary adult obese men aged 35-43 year and body mass index (BMI) 30-34 kg/m<sup>2</sup> were completed a moderate exercise test involved 45 min run on smooth surface without slope at 70 % of maximal heart rate. Blood samples were collected prior to exercise, at the end of exercise, and at 60 min recovery. Given normal distribution of the data, which was analyzed by -Smirnov test, subsequent analysis was performed by Student's t-test or repeated measures ANOVA. No significant difference found in serum leptin between 3 samples. On the other hand, exercise test was not associated with acute or recovery response in serum leptin in studied subjects. Based on this data, it seems that one moderate exercise test can not affect serum leptin for short time recovery in obese individuals. Further studies are necessary to elucidate the significance of acute exercise on peptide mediators in obese or related diseases.

**Keywords:** Leptin, Obesity, Exercise test, Acute response

### INTRODUCTION

Sedentary lifestyle is one of the most important factors causing obesity and its related diseases, as obese people follow a sedentary lifestyle and it has been reported that there is a causal relationship between obesity and sedentary lifestyle in many cases. However, it doesn't mean that obesity and increased level of body fat cause some diseases only because of inactivity [1, 2], but hormonal changes caused by increased levels of body fat are of special importance in emergence and increased severity of obesity-related diseases.

Disorder in systemic levels of hormones secreted from adipose tissue and other endocrine organs of body is of factors contributing to obesity and related diseases such as atherosclerosis, diabetes, and cardiovascular or respiratory diseases [3, 4]. Previous studies have shown that these peptide mediators regulate both the energy balance and body weight by interfering with the mechanisms of metabolism, resting energy expenditure, food absorption rate, and appetite control [1]. For instance, leptin, which is known as a hormone involved in reducing hunger and appetite control and also as one of the factors influencing the relationship between obesity and its associated chronic disease, decreases during fasting and increases after feeding [5, 2]. These concepts can be differently applied to any of other

peptides secreted by adipose tissue like Adiponectin, resistin or some of interleukins. Most studies have somehow stressed the role of these above-mentioned mediators in lipid disorders, insulin resistance, and obesity-related abnormalities [6, 7]. Hence, it seems that innovating and proposing appropriate solutions, besides medication, can play a potential role in preventing or reducing such diseases.

The role of exercise, as a non-medication solution, in establishing a balance in the level of these variables in healthy and patient populations has been extensively and frequently studied. Although there are different findings about peptide mediators response to various types of exercise, some studies have reported the positive effect of exercise [8, 9] and some others have denied any impact of exercise on these elements [10, 11]. For example, some studies reported a significant decrease in leptin [12] and some others observed no change in the levels of leptin or other peptide mediators [13] in response to exercise. Researchers have attributed these inconsistencies to the difference in the intensity and duration of exercise, difference in blood sampling time, and difference in the studied population. According to these inconsistent pieces of evidence, the present study aims to determine the acute and delayed (after 60 minutes) response of leptin to a session of running at a moderate intensity in sedentary obese men.

## MATERIAL AND METHODS

### A. Human subjects

In this study, acute and recovery (60 min) response of serum leptin to one moderate exercise were measured in sedentary adult obese men. Subjects were fifteen non-trained adult obese men aged 35-43 year and BMI 30-34 kg/m<sup>2</sup> and were completed exercise test. The study protocol was approved by the institutional ethics committee of Islamic Azad University, Iran.

### B. Inclusion and exclusion criteria

Subjects were asked to complete questionnaires on anthropometric characteristics, general health, smoking, alcohol consumption, and present medications. Participants were non-smokers and non-alcoholics. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. None of the subjects used drugs or therapies for obesity, and none had a past history of disease or injury that would prevent daily exercise. Exclusion criteria included having history of known hyperlipidemia, hypertension, coronary artery disease, cerebrovascular disease, peripheral artery disease and type 2 diabetes.

### C. Anthropometric measurements

After the nature of the study was explained in detail, informed consent was obtained from all participants. Anthropometric measurements were performed in all study participants before breakfast, with the subject wearing light clothing without shoes. Weight was measured to the nearest 100 g using digital scales. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. Waist circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Percentage of body fat was estimated by bioelectrical impedance method (Omron Body Fat Analyzer, Finland). Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m<sup>2</sup>). All anthropometric measurements were made by the same trained general physician.

### D. Blood samples and exercise test

Venous blood samples were obtained before, immediately after, and after 60 minutes of recovery. Serums were immediately separated in order to measure leptin by ELISA method (Biovendor-Laboratoria medicina a.s. Czech). Concentrations of leptin were measured and compared with each other. Exercise test lasted 45 min at 70 % of maximal heart rate. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US).

### E. Data analyses

After calculation of the mean and the standard deviation, the statistical analysis was conducted using the SPSS software version 15.0. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Results are presented as means  $\pm$  SD. Data were analyzed by two-way repeated-measures ANOVA. A P-value of  $< 0.05$  was considered to be statistically significant.

## RESULTS

In this study, acute and recovery response of serum leptin to a moderate exercise test were determined. Table 1 show the descriptive anthropometric and biochemical features of the study groups. All subjects were obese. Data by repeated measure method showed no significant differences in serum leptin between pre-test, immediately and 60 min recovery. On the other hand, we did not observe acute or recovery response of serum leptin to exercise test compared to baseline in studied obese subjects (Table 2 and Fig. 1).

## DISCUSSION

No significant change in serum leptin levels immediately and one hour after the exercise is one of the main findings of the present study. According to the findings of this study, it can be concluded that serum leptin levels in obese men do not show a significant acute and delayed (after 60 minutes) to a relatively long running session at a moderate intensity. This is both consistent and inconsistent with the findings of some previous studies that will be mentioned below.

It has been well documented that excessive increase in the volume of adipose tissue is associated with obesity-related health problems. Inflammatory characteristics of obesity are complex in the pathogenesis of obesity-related mortality. Given its role in regulating the secretion and release of certain hormones and hormone receptors [14, 15], adipose tissue is known as an important endocrine organ that secretes some biological activators called adipokines [16, 17]. Regulation of body weight is a complex behavior that is influenced by physiological and genetic behaviors and other environmental interventions and energy balance is one of the most important factors affecting it [18]. Leptin is one of the peptide mediators related to obesity, as increased volume of adipose tissue, especially abdominal obesity is followed by higher levels of leptin [18, 19]. In fact, leptin is known as an anti-obesity or anti-hunger hormone which acts as an afferent signal in the negative feedback regulation of body weight through controlling the food intake and energy expenditure by affecting the cycle of hypothalamus-pituitary-gonads [20].

**Table 1: Descriptive characteristics of anthropometrical markers and serum leptin in studied subjects**

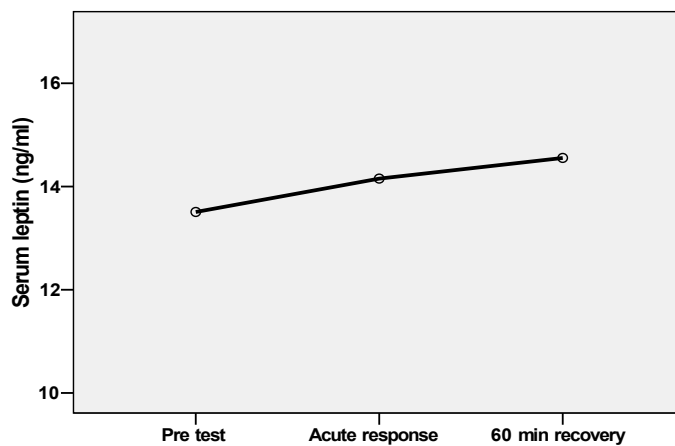
	Minimum	Maximum	Mean	Std. Deviation
Age (year)	35	43	39.07	2.086
Height (cm)	163	176	172.93	4.148
Weight (kg)	80	105	94.60	6.311
Abdominal (cm)	95	112	104.27	4.847
Hip (cm)	96	114	105.07	5.325
WHO	.97	1.03	.9928	.01967
BMI (kg/m <sup>2</sup> )	30.1	34.4	31.606	1.2994
Body fat (%)	30.3	36.8	32.560	2.0138
Visceral Fat	12	17	13.60	1.724
Leptin (pre test)	4.6	20.1	13.507	4.8305
Leptin (acute response)	4.8	21.3	14.153	5.2025
Leptin (60 min recovery)	8.9	19.6	14.553	3.9130

**Table 2: Acute and recovery response of serum leptin to exercise test in obese subjects: Data by Repeated measure Anova**

Measure: MEASURE\_1

(I) leptin	(J) leptin	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval for Difference	
					Lower Bound	Upper Bound
1	2	-.647	.878	.473	-2.529	1.236
	3	-1.047	1.083	.350	-3.369	1.276
2	1	.647	.878	.473	-1.236	2.529
	3	-.400	1.197	.743	-2.968	2.168
3	1	1.047	1.083	.350	-1.276	3.369
	2	.400	1.197	.743	-2.168	2.968

Based on estimated marginal means



**Fig. 1:** The changes pattern of serum leptin in pre test, acute and recovery response to exercise test.

The question is that why obese people show higher levels of leptin compared with ordinary people. As an answer to this question, researchers have pointed to leptin resistance in obese individuals [21]. They argue that internal leptin resistance is the cause of obesity and leptin resistance resulting from obesity would cause damages to some peripheral tissues such as liver, pancreas, platelets, blood vessels, and myocardium [21].

Although most studies have emphasized on the beneficial effects of sport activities and especially long-term exercises on serum or plasma levels of leptin or other peptide mediators involved in obesity or obesity-associated diseases [12, 22, 23], there are few contradictory findings about immediate and delayed response of leptin to short-term exercise tests or one-session tests. On the other hand, differences in the response of peptide mediators and inflammatory and anti-inflammatory cytokines to exercise tests are of the subjects that have attracted the attention of sport sciences researchers. It has been repeatedly observed that although some pro-inflammatory or anti-inflammatory cytokines do not change significantly after a specific exercise, some others considerably change compared with the primary levels. For example, in a recent study, despite a significant increase in Adiponectin, serum leptin levels did not change significantly in obese men in response to a short-term cycling session [24]. Another study showed that 45 minutes of running with an intensity of 65%  $VO_{2max}$  did not lead to any significant change in Adiponectin concentration immediately, 24 hours, and 48 hours after an exercise test [25].

However, in another study, it was reported that maximum rowing exercise for 30 minutes led to a decrease in leptin levels immediately and 30 minutes after the exercise [26]. Also, another finding indicates that 20 minutes of intense jogging by middle-aged men and women caused a significant decrease in leptin concentration but it returned to baseline levels after one hour of rest (27). Based on these findings, researchers have concluded that leptin response to an intense short exercise would be significant only when all the large muscles of body are involved [28]. Although the large muscles of body were involved in the exercise test performed in the present study, duration and intensity of exercise and the amount of calorie consumption also seem to have a major contribution to leptin response to exercise, because most of the studies that have reported the reduction in leptin levels, had used intense sport exercises [26, 27]. Also, some other studies have reported a significant decrease in serum leptin levels after a one-session physical activity with an energy expenditure of 1500 kcal followed by a negative energy balance [217].

With regard to body weight and duration of exercise test in the present study, it does not seem that exercise test has been associated with negative energy balance in the subjects.

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