Serum leptin in response to acute running test in adult obese men

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Key words: Serum leptin, adult obese men, session running test.


Abstract

There is evidence that acute exercise have minor or no effects on leptin concentration. To determine of acute response of serum leptin to one session running test, twenty adult obese men were participated in study by randomly. Participants were included if they had not been involved in regular physical activity or diet in the previous 6 months. For this purpose, venous blood samples were taken before and immediately after single bout running test in order to measuring serum leptin and to compare its values between two samples. Student’s paired ‘t’ test was applied to compare the pre and post exercise values. Compared to pre-exercise, the leptin levels did not change significantly (P≥0.05) after acute exercise in studied subjects. Based on these data, we conclude acute long-time exercise is not associated with anti-inflammatory properties in obese with emphasis on serum leptin.

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**Introduction**

There is considerable evidence that adipose-secreted cytokine proteins, such as leptin are implicated in numerous biological functions (Fasshauer et al., 2003). This inflammatory cytokine represent a newly discovered adipose tissue derived hormone; that is associated with health status and glucose and free fatty acid metabolism (Bouassida et al., 2010). Its circulating concentration is very important for the control of food intake (appetite) and its plasmatic level is directly correlated with the adipose tissue mass (Pannacciulli et al., 2003).

On the other hand, Extensive studies have mentioned that central leptin resistance causes obesity and that obesity-induced leptin resistance injures many peripheral tissues, including the liver, pancreas, platelets, vessels, and myocardium (Martin et al., 2008).

According to the population studies, it has been indicated that Exercise training increases total energy expenditure by upregulating the direct energy cost of physical activity, which may lead to decreased body fat stores that ultimately will depress leptin secretion (Poehlman et al., 1991; Morley, 2001). It has been demonstrated that those exercise training which improve fitness levels and affect body composition could decrease leptin concentration (Bouassida et al., 2010). But the responses of this cytokine to short-time or one session exercise is controversial.

For example, some studies have been reported that circulating leptin levels remained unchanged after acute aerobic exercise (Kraemer et al., 2003; Ferguson et al., 2004), while another study was reported aerobic exercise can be decrease serum leptin (Nindl et al., 2002). Contradictory findings observed on the relationship between leptin levels and exercise in human studies can be attribute to the different training protocols used (intensity, volume, duration, subject’s initial conditioning status, and energy balance conditions). Therefore further study will be needed to clarify the mechanism of acute response of serum leptin to exercise.

**Materials and methods**

This study was approved by Ethics Committee of Islamic Azad University, Parand branch, Iran. Main objective of study was to determine acute response of serum leptin to a running exercise in non-trained middle-aged obese men. For this purpose, twenty adult obese men were participated in study by randomly. All participants were non-trained. In fact, Participants were included if they had not been involved in regular physical activity or diet in the previous 6 months. After the nature of the study was explained in detail, informed consent was obtained from all participants. Participants were reported to be non-smokers, not currently taking supplements of any kind, and having no major health problems (i.e., diabetes, cardiovascular disease, etc.). All subjects had a body mass index (BMI) of more than 30 kg/m^2^.

At first, all anthropometric measurements were made by the same trained general physician and under the supervision of the same pediatrician following standard protocols. Percentage body fat was measured using body composition monitor (OMRON, Finland). Body weight was measured in the morning following a 12-h fast. Height and body weight were measured twice, with subjects being barefoot and lightly dressed; the averages of these measurements were recorded. Waist circumference (WC) was measured with a non-elastic tape at a point midway between the lower border of the rib cage and the iliac crest at the end of normal expiration. BMI was calculated as weight (in kilograms) divided by the square of height (in meters).

In the next stage, Basal, fasting blood samples were taken after an overnight fast to determine serum leptin. Then, all participants were completed single bout modified tests consist of 45 min running at an intensity of 75(%) maximal heart rate. Blood sampling was repeated immediately after exercise test. Blood samples were dispensed into EDTA-coated tubes and centrifuged for 10 minutes in order to separate serum. Subjects were asked to avoid doing any heavy physical activity for 48 hours before blood sampling. Serum CRP was determined by ELISA method. The Intra-
Statistical analysis
All values are represented as mean ± SD. Statistical analysis was performed with the SPSS software version 15.0. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Student's paired ‘t’ test was applied to compare the pre and post training values. An alpha-error below 5% was considered as statistically significant.

Results
Baseline and post exercise leptin levels, anthropometrical indexes and the other variables are shown in Table 1. All values are given as mean and standard deviation. The Kolmogorov-Smirnov test showed a normal distribution in all variables. Anthropometrical indexes showed that all participants have obese. As mentioned above, main objective of study was to investigation of acute response of leptin to exercise test in obese men. The study finding showed no significant response of leptin of post-test when compared to baseline (p ≥ 0.05) (see Fig 1). On the other hand, one session running test with relatively long-time did not affect serum leptin immediately after finished test (13.82 ± 1.40 at baseline to 14.16 ± 1.40 ng/ml, p ≥ 0.05).

Discussion
This study shows that a relatively moderate-intensity running session does not lead to significant changes in serum leptin levels in obese non-athletic men. During the past two decades, numerous studies have been conducted to determine the effects of different types of exercises on cytokine levels in healthy populations or patients which have brought about different answers depending on the exercise protocol. In some studies, the response of certain inflammatory or non-inflammatory to exercise even in the presence of increased cardiovascular fitness was not statistically significant (Pischon et al., 2004). Besides, there are also studies that despite a cytokine remaining unchanged, others cytokines’ response to exercises are significant. Such an inconsistency in the findings is seen in most previous studies.

Similar to other cytokines, a contradiction in acute or delayed responses to different exercise tests are also frequently observed on serum leptin levels. For example, the findings of a recent study show that 30 minutes of rowing exercise leads to a meaningful acute and delayed reduction in serum leptin (Jurimae, 2005). In another study too, a relatively short-term exercise significantly decreased serum leptin levels immediately after stopping the test but not one hour after the test (Legakis et al., 2004). It has been suggested that the response of leptin to short-term intense exercise is significant only when all large muscles are involved (Bouassida et al., 2010). Moreover, in another study, a significant reduction in serum leptin was observed at only 24 hours after relatively prolonged exercise (Olive et al., 2001). Some other studies report leptin significant response only after those exercise tests associated with negative energy balance (Essig et al., 2000).

Despite the said findings, the findings of this study showed that a single session of exercise in the form of a 45 minute run with 70% of maximum heart rate does not change levels of leptin immediately after the test. Similar to the findings of our study, no
significant change in serum leptin levels was observed in adults after a relatively moderate-intensity cycling exercise (Ferguson et al., 2004). In another study, too, no change in serum leptin concentrations has been reported within 30 minutes after resistance exercise (Zafeiridis et al., 2003). According to their findings, some researchers have concluded that the decrease in leptin levels after a single-session exercise occurs only when the exercise takes more than 60 minutes so it can stimulate release of free fatty acids; or when physical activity is associated with energy expenditure of more than 800 kcal (Hqjbjerre et al., 2007; Kraemer et al., 2000). Citing this material, absence of acute and delayed response of leptin in the present study may be attributed to lower periods of time and energy expenditure than the values reported by the researchers in this exercise test.

Table 1. Mean and standard deviation (SD) of anthropometric and metabolic characteristics of studied subjects.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean</th>
<th>SD</th>
</tr>
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<tbody>
<tr>
<td>Age (year)</td>
<td>38.2</td>
<td>2.11</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172</td>
<td>4.8</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>93.6</td>
<td>7.90</td>
</tr>
<tr>
<td>Abdominal circumference (cm)</td>
<td>104.4</td>
<td>4.87</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>105.9</td>
<td>4.65</td>
</tr>
<tr>
<td>WHO</td>
<td>0.99</td>
<td>0.02</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>31.64</td>
<td>1.58</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>32.47</td>
<td>2.01</td>
</tr>
<tr>
<td>Visceral fat</td>
<td>13.20</td>
<td>2.18</td>
</tr>
<tr>
<td>Leptin (ng/ml)</td>
<td>13.82</td>
<td>1.40</td>
</tr>
</tbody>
</table>

Although the main reasons for inconsistent findings on the responses of these mediators to a single-session exercise test are yet unclear, differences in age, initial fitness level, the phenotype of obesity and other environmental factors that affect the effect of exercise on metabolism can all explain these inconsistent findings. However, some researchers attribute the slight changes of peptide mediators’ levels in response to exercise to changes in plasma volume but not to the response to exercise (Kraemer et al., 2003).

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