Full Length Research Paper

Does regular aerobic training affect basal leptin level (difference between male and female)?

Salehzadeh Karim¹* and Agaziyev Afiq²

¹Department of Physical Education Maragheh Branch, Islamic Azad University, Maragheh, Iran.
²Azerbaijan National Academy of Science, Institute of Physiology, Baku, Azerbaijan.

Accepted 23 August, 2011

There is discrepancy about the effects of regular exercise training on basal leptin level and most of the existing data have been collected from obese/thin subjects. In this study, the effects of one year aerobic exercise was investigated in normal weight untrained healthy volunteer male and female subjects. The sample included 60 non-athletes, male and female university students. First, all samples were randomly divided into four groups. The practice program including selected aerobic exercises and volleyball drills with specific intensity was applied. The blood samples of subjects were taken once 24 h before the first section of the exercise program and 24 h after the last section of the exercise program. Resting plasma leptin level was determined using ELISA method in pre and posttest and in paired sample T test, whereas one way and two way (factorial) ANOVA was used to analyze the data. Studies show that basal plasma leptin levels in healthy young non-athlete individuals in response to regular exercise in the long term did not fluctuate. Aerobic exercise for one year showed no significant effect on plasma leptin levels (that is about the role of fat tissue as the most significant source of leptin), and it seems that it occurs because fat tissue in subjects did not change.

Key words: Aerobic exercise, leptin, long-term, non-athlete.

INTRODUCTION

Obesity has become a global epidemic in developed countries. More than 1.6 billion people are overweight and 0.4 billion people suffer obesity (Janghorbani et al., 2007). Most of the time, obesity leads to different health problems such as diabetes mellitus, hypertension, cardiovascular diseases and sudden deaths (Ren et al., 2004). Excess weight prepares the grounds for the overweight to come down with osteoarthritis and other respiratory system problems like sleep apnea. Thus, obesity increases the responsibilities and problems of the public health (Kraemer et al., 2002). The Great Britain has faced a tripled adiposity over the last two decades and nearly 60% of the people suffer obesity. It is quite certain that the main reasons for this problem are the decrease in physical activities and increase in eating foods of very high calorie as a part of modern lifestyles (Huuskonen et al., 2010). A study conducted in 2007 investigated the rate of adiposity in Iranian adults between 15-65 revealed that 42.8% of men and 57% of women were overweight (BMI≥25) and 11.1% of men and 25.2% of women were obese (BMI≥30). The researcher concluded that lack of descent physical activity is the main cause of adiposity in Iran (Henderson et al., 2006).

Leptin is the protein of obesity gene which is emitted in a pulsantory way to general blood movement by fat cells which transfers feedback signals between fat cells and central nerve system especially moving centers in hypothalamus and participating in hemostat balance of body weight (Pasman et al., 1998; Speakman et al., 2004). This protein hormone, excreted from fatty tissue, is released mainly from mass molecule 16 kDa and plays a key role in maintaining body weight. It is worth saying that leptin acts as a warning mechanism for maintaining fat content of the body. This hormone increases the level of consumed energy by enhancing the activity of sympathetic and lipolis nerves. Leptin controls appetite through affecting hypothalamus receivers (Hickey et al., 1997). Although several studies have failed to prove the pathologic role of leptin in obesity, this deficiency can be related to impotency of leptin in passing the brain-blood barrier as a cause of pathologic adiposity.
<table>
<thead>
<tr>
<th>Type of exercise</th>
<th>Stretches</th>
<th>Aerobic running</th>
<th>Joints warm-up</th>
<th>Practicing skills</th>
<th>Volleyball game</th>
<th>Recovery</th>
<th>Exercise program</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of exercise in each session</td>
<td>8 min.</td>
<td>12 min.</td>
<td>7 min.</td>
<td>18 min.</td>
<td>40 min.</td>
<td>5 min.</td>
<td>90 min.</td>
</tr>
<tr>
<td>Severity of exercise</td>
<td>70% $\text{HR}_{\text{MAX}}$</td>
<td>Competitive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

barriers, impotency in being matched with noropeptid Y and after all the change in the amount of food received, problems in sending massage to leptin receiver in brain (Pasman et al., 1998). It is known that the level of serum leptin correlates greatly with the fat content of the body, and as the body weight decreases, it decreases as well (Casimiro-Lopes et al., 2009; Ramazankhany et al., 2010). Leptin is one of the hormones controlling energy hemostas in normal conditions; however, presence of abnormal amounts of it in the blood causes severe problems (Olive and Miller, 2001). It is shown that the amount of energy received regulates leptin gene either positively or negatively and thus, the change in energy consumption through physical activity may change leptin level (Piri et al., 2009). Various reports on the effect of physical exercises in serum leptin are contradictory (Patrick et al., 2010; Neary et al., 2004; Martins et al., 2007; Hickey et al., 1997; Fatouros et al., 2005). Some indicate that the serum leptin does not change by running in a moderate speed (Magdalena et al., 2010; Fatouros et al., 2005; Bouassida et al., 2006) while others prove that hard practices are one of the reasons for the decrease in leptin level (Leila Maria et al., 2010; Piri et al., 2009; Reinehr and Andler, 2002).

On the contrary, there have been studies having revealed that plasma leptin level does not change by short term exercises (Esteghamati et al., 2010; Neary et al., 2004). On the other hand, there are clues that show different results in excretion of leptin and gerlin hormone in men and woman when exposed to special levels of exercises (Faruk et al., 2010; Haghighi and Hamedi-Nia, 2008).

Moreover, in most studies the effects of physical activity on the leptin level of obese and thin people is examined and there is not enough information about the weight of ordinary people (Unal et al., 2005). Thus, the present paper has attempted to study these effects in normal people that seem to be helpful in providing information on prevention of process of leptin change in these people.

This study measured the effect of a long-term (1-year) activity and in addition, comparison of the results tried to separate the effect of sex, exercise style, inactive lifestyle and their relation with the leptin release in long-term creativity in this regard and could definitely provide every exercise as well. Therefore, it seems that this study is of precise and comprehensive information for experts.

**METHODOLOGY**

This study is a quasi-experimental one in which 60 nonathletic healthy subjects, who has no experience of participation in any sport program or diet, contributed. They first signed an agreement of the subject voluntenerness. Subjects in this study neither used any medicine, cigarettes or anti- pregnancy drugs nor they were not pregnant. In addition, they did not have any kind of heart disease, respiratory, kidney, metabolic or menstrual disorders. However, it should be noted that in the 1-year time spent on conducting the research, the number of the subjects under study reduced, so we had to just analyze just 44 people (men 24 and women 20). The subject’s features are listed in Tables 1 and 2.

Exercise time was designed to be done in stipulated time of day (8 to 10:30 in the morning) to keep its full time rhythm. Subjects had their ordinary lifestyle during the study. Annual program included selected aerobic exercises and volleyball matches according to Table 1.

**Blood sampling and biochemical measurement**

Blood samples (10 ml) were taken from the vein after nocturnal breakfast for 12 h and before exercising began. After sampling, test tubes were fixed until clotting in order to keep blood samples. Then the serum was separated from the blood through centrifuge and was kept in normal room temperature. Then they were moved to -80 degrees centigrade for biochemical measurements. Leptin was measured using ELISA method (Japan IBL Inco).

**RESULTS**

In order to elicit the role of sex (male and female) and group (training situation/exercise) and their interaction with leptin changes in the intervals between pre and post tests in 4 groups, at first, the normal distribution of dual sex blocks (including male and female) and exercise situation (control and athlete) were assured. Then the effects of the dual blocks were studied through double variance analysis ($2^2$) test results are in Table 3, 4 and 5.

**DISCUSSION AND CONCLUSIONS**

The main result of this study revealed that one year aerobic exercise with an average severity has no finding is in contrast to results of (Unal et al. 2005) who measured leptin concentration in athletic, young males trained in various sport fields and healthy inactive people. They observed that after exercising, leptin meaningfully

---

**Table 1. Exercise program of on session volleyball, 3 times in a week (1 year).**

<table>
<thead>
<tr>
<th>Exercise program</th>
<th>Stretches</th>
<th>Aerobic running</th>
<th>Joints warm-up</th>
<th>Practicing skills</th>
<th>Volleyball game</th>
<th>Recovery</th>
<th>Length of exercise in each session</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8 min.</td>
<td>12 min.</td>
<td>7 min.</td>
<td>18 min.</td>
<td>40 min.</td>
<td>5 min.</td>
<td>90 min.</td>
</tr>
</tbody>
</table>
Table 2. Features of the subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Index</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>BMI (Kg/m²)</th>
<th>Duration of Follicle Period (day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>Control (10)</td>
<td>26.25 ± 6.75</td>
<td>164.125 ± 9.14</td>
<td>23.21 ± 0.50</td>
<td>10/25 ± 0.99</td>
</tr>
<tr>
<td></td>
<td>Exercise (10)</td>
<td>24.98 ± 5.12</td>
<td>166.375 ± 7.19</td>
<td>24.597 ± 1.27</td>
<td>10.38 ± 1.03</td>
</tr>
<tr>
<td>Male</td>
<td>Control (12)</td>
<td>24 ± 2.65</td>
<td>174.375 ± 6.92</td>
<td>23.206 ± 0.53</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (12)</td>
<td>23.125 ± 1.93</td>
<td>175.775 ± 6.89</td>
<td>22.490 ± 0.377</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. Results of correlated T test on comparing each group's leptin in pre and post tests.

<table>
<thead>
<tr>
<th>Group</th>
<th>Average difference</th>
<th>T</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>male control</td>
<td>0.066 ± 6.83</td>
<td>0.034</td>
<td>0.974</td>
</tr>
<tr>
<td>female control</td>
<td>-0.54 ± 4.89</td>
<td>-0.349</td>
<td>0.753</td>
</tr>
<tr>
<td>male exercise</td>
<td>1.22 ± 7.24</td>
<td>0.568</td>
<td>0.570</td>
</tr>
<tr>
<td>female exercise</td>
<td>3.68 ± 8.12</td>
<td>1.43</td>
<td>0.186</td>
</tr>
</tbody>
</table>

*There is no significant difference (p<0.05).

Table 4. The Results of different leptin data between pre and post tests in 4 groups (ANOVA).

<table>
<thead>
<tr>
<th>Index</th>
<th>square average</th>
<th>F</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin</td>
<td>35.470</td>
<td>0.747</td>
<td>0.531</td>
</tr>
</tbody>
</table>

*There is no significant difference (p<0.05).

Table 5. The Results of comparison of leptin changes in four groups (in intervals between pre and post tests) by variance (2*2) analysis.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Studying the effects</th>
<th>loan F</th>
<th>loan Sig</th>
<th>Square of averages</th>
<th>F</th>
<th>Sig</th>
<th>Average difference</th>
<th>Freedom degree</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in leptin</td>
<td>Sex</td>
<td>0.076</td>
<td>0.973</td>
<td>9.317</td>
<td>0.196</td>
<td>0.660</td>
<td></td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Group</td>
<td>78.890</td>
<td>1.661</td>
<td>0.205</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Interaction sex/ group</td>
<td>25.565</td>
<td>0.538</td>
<td>0.467</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

decreases and concluded that regular exercise lessens blood leptin level by decreasing lipid percentage (Rahmani Nia et al., 2009). In another study, a 12-week aerobic exercise reduced serum leptin in females but not in males (Fatouros et al., 2005). Fatouros et al. (2005) also reported that elder women show a lower leptin level after exercises (Duclos et al., 1999). These results were confirmed by Ryan et al (2003) when they proved that exercise females had lower levels of leptin compared to control women (Witek et al., 2003; Otsuka et al., 2006). Hickey et al. (1997) indicated that sexes special response to exercise is based on different resistances to insulin in males and females. Males who had the most resistance to insulin possibly needed more time for exercising and more powerful stimulant for leptin level decrease (Fatouros et al., 2005). In addition, researchers indicated that low leptin concentration is an indirect result of exercise. Perhaps decrease of bodies' fat tissue formed by exercise is the main reason of leptin changes (Duclos et al., 1999). There seems to be obvious differences in subjects and exercise protocols could justify the observed differences in exercise- leptin relation by different studies (Wadden et al., 1998). However, some other researchers believe that the decrease in leptin after physical training is attributed to the increase in production of saturated hydrocarbons (Considine et al., 1996; Hickey et al., 1997) that is in contrast to present study. It seems as if the most important reason for this difference is the age of the subjects in this study in which older subjects went under different exercise protocols.

In a study conducted on the effects of a period of aerobic exercise on serum leptin, cortisol and...
testosterone level of obese and thin men, meaningful
decrease of leptin level in both groups of obese and thin
men was observed that the body mass index and weight
of obese subjects decreased meaningfully (Sprung et al.,
2008). In addition, a meaningful increase in cortisol, BMI
and weight of thin group (Martins et al., 2008) was seen.
On the other hand, results of inter-group comparison
show a significant difference in leptin, testosterone, BM
level, and weight in obese and thin groups (Blandine
et al., 2006). According to these findings, aerobic exercise
plays an important role in regulating body weight by
changing some hormones like leptin, cortisol and
testosterone (Zaferiridis et al., 2003).

Also leptin levels of blood are closely related to the
amount of human and animal fat tissues, and their
consistency against bodies’ fat tissues is more than body
weight. Leptin central and environmental injection in
rodents leads to a decrease in food intake and body
weight. Leptin increase in rodents happens a few minutes
after having a meal and increase after a few days of over-
indulgence in humans. In contrast, starvation decreases
leptin level. The rate of these responses is adjusted to
changes (Kraemer et al., 2002). Researchers usually
think of leptin as a stabilizer before significant changes in
weight (Haghighi and Hamedi-Nia, 2008; Marco et al.,
2010; Sarigianni et al. 2010).

Blandine et al. (2006) reported that short and long term
regular physical exercises meaningfully decrease BMI
and plasma leptin level and increase testosterone which
in that research decomposed fat and was believed to be
related to increase in beta adrenergic, Adina sickles,
protein kinase A and lipase receptors. It is certain that
males encountered low level of free testosterone with
decrease in decomposition of fat tissues in response to
catecholamine and possibly these metabolic changes
decrease decomposition. Besides, transfer of tri glyceride
and compilation of fat tissue in body increase (Blandine
et al., 2006).

Previous researches about males demonstrated that
there is a strong negative relationship between leptin
level and free plasma testosterone that is independent
from insulin plasma level and other metabolic factors
(Sprung et al. 2008). Nevertheless, in this research
plasma leptin level did not have a meaningful effect (that
is about the role of fat tissue as the most significant
source of leptin) and it seems that it occurs because fat
tissue in subjects did not change. It must be noted that
lack of control in subjects' diet is the main reason for this.

It may be possibly females’ regular physical exercises
that causes changes in their reproduction system and
shortness of leptin phase of subjects that kept leptin
unchanged (Hickey et al., 1997; Okazaki et al., 1999;
Thong et al., 2000), so intermittent studies in female
encounter more problems. Considering the effects of sex
hormones on related blood lipid even leptons to exercise
effects seems a little difficult.

In general, results of the present study failed to show
any significant differences between plasma leptin level of
normal weight untrained healthy volunteer male subjects
and also there were not significant effect of the sex,
group (exercise/control) or their interaction upon the
changes of leptin level between pre and post tests
(Figure 1). The need for further study on the relationship
between fat tissue in response to leptin and exercise
consistencies is felt. With regard to the young subjects
studied, type of exercise, university life, and also the
controversial reports in the existing literature about obese
and thin people, it seems that other factors such as
lifestyle or daily energy expenditure, and relation of
aerobic exercise with diet or without it and considering
endocrine factors which adjust leptin, should be certainly
investigated to draw more obvious conclusions.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Table of subjects Leptin level in pre and post test.}
\end{figure}