THE EFFECT OF ENDURANCE TRAINING ON ADIPONECTIN AND INSULIN RESISTANCE IN OVER WEIGHT FEMALE UNIVERSITY STUDENTS

SHAKERI NADER¹, SOHEILY SHAHRAM, YADEGAR ELHAM¹
¹Department of Physical Education and Sport Science, Science and Research Branch, Islamic Azad University, Tehran, Iran - ²Department of Physical Education and Sport Science, Shahr-e-Qods Branch, Islamic Azad University, Tehran, Iran - ³Department of Physical Education and Sport Science, South Tehran Branch, Islamic Azad University, Tehran, Iran

ABSTRACT

Aim: Adiponectin is an adiposity secreted hormone that plays a pivotal role in lipid and glucose metabolism that may also be a marker for coronary artery disease. The purpose of this study is to investigate the effect of endurance training on Adiponectin and insulin resistance in overweight female university students.

Methods: In this regard, 20 sedentary young women subject (BMI ≥ 25) have randomly been divided into to two similar groups (endurance training and control). The endurance training program was performed three days a week for 12 weeks at a definite intensity and distance. Before and after 12 weeks intervention, Adiponectin, weight and body composition, Vo2max were measured for all subjects. Data were analyzed by independent t-test (p≤ 0.05).

Results: the results illustrated that endurance training caused a significant difference in Adiponectin, Vo2max, body weight and fat percent of the body of the experimental group in comparison to control group.

Conclusion: endurance training leads to a significant increase of the levels of Adiponectin and significant decrease in insulin resistance. This suggests that the insulin resistance is affected by endurance training that may be mediated, even partially through increased Adiponectin in overweight female university students.

Key words: Endurance training, Adiponectin, Insulin Resistance, Sedentary Young Women.

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Introduction

In addition to its classic function of storing energy, adipose tissue is now recognized as an important and very active endocrine gland¹. Adipose tissue secretes multiple proteins are known as adipocytokines.

One of these adipocytokines is adiponectin, which is reduced with obesity, increased insulin resistance, dyslipidaemia and diabetes. Adiponectin may be a marker for coronary artery disease² and seems to have protective metabolic and anti-inflammatory properties³, which prevents atherosclerosis⁴. It has been suggested that physical exercise, enhanced physical fitness and obesity reduction are associated with improvements in the metabolic state, although concentrations of adiponectin have not changed after some experimental studies⁵,⁶. Although controversial, few studies⁷,⁸ have suggested a direct relationship between the levels of adiponectin and physical activity and, as pointed out by Bluher et al, physical training appears to increase the number of adiponectin receptors in subcutaneous fat⁹. Weight loss¹⁰ and exercise¹¹ are common clinical interventions for the treatment of insulin resistance. Two publications have reported significant increases in plasma adiponectin with weight loss¹²,¹³.
As another therapy to decrease insulin resistance along with weight reduction, exercise training might also affect adiposity metabolism and result in changes in adiponectinaemia. It has been established that endurance training increases insulin sensitivity and causes improvements in insulin sensitivity. However, there is still controversy with regard to the effects of endurance training on adiponectin. For example, different authors have demonstrated no change\(^{13, 5, 13}\), increase\(^{14, 15}\) or decrease\(^{16}\) in adiponectin following different training protocols. Therefore, the present study was designed to determine the effects of endurance training on adiponectin concentration and insulin resistance in overweight female university students.

Material and methods

The research was semi-experimental. Twenty girls with BMI ≥ 25 volunteered to participate in this study. All subjects were asked to complete a personal health and medical history questionnaire, which served as a screening tool. The university's ethics committee approved the experimental procedures and study protocols, which were fully explained to all subjects. A written consent form was signed by each subject after having read and understood the details of the experiments. The height was measured using a medical height meter; weight and body composition were measured using a body composition monitor (OMRON, Finland).

The maximum oxygen consumption of all the subjects was measured twice using the Cooper test; once before the test and once after the test. The subjects ran for 12 minutes at their maximum speed. The mileage was then placed in this formula: 

\[
\text{Vo2max} = \text{Mileage (M)} - (504.9 / 44.7).
\]

Blood samples were obtained from all subjects at 0800 h after an overnight fast before and after the training program. Fasting sera kept at -80 °C were used to measure serum adiponectin concentrations by ELISA (Biovendor, Germany). Insulin resistance in fasting state was determined using a homeostasis model assessment (HOMA-IR) and was calculated from fasting insulin (IF) and fasting glucose (GF) as follows: 

\[
\text{HOMA-IR} = \left(\frac{\text{IF} \times \text{GF}}{22.5}\right)^{(17)}.
\]

Endurance training consisted of continues raining 3 days a week, for 12 weeks (Table 1).

<table>
<thead>
<tr>
<th>Week</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target heartbeat, percentage</td>
<td>60-65%</td>
<td>60-65%</td>
<td>60-65%</td>
<td>60-65%</td>
<td>65-70%</td>
<td>65-70%</td>
<td>70-75%</td>
<td>70-75%</td>
<td>70-75%</td>
<td>70-75%</td>
<td>70-75%</td>
<td>70-75%</td>
</tr>
<tr>
<td>Distance, meter</td>
<td>1600</td>
<td>1600</td>
<td>1800</td>
<td>1800</td>
<td>2400</td>
<td>2400</td>
<td>2800</td>
<td>2800</td>
<td>3000</td>
<td>3000</td>
<td>3200</td>
<td>3200</td>
</tr>
</tbody>
</table>

Table 1: Endurance training program.

Statistical methods

All values are represented as mean ± SD. Independent t test was used for testing significance between groups. All the statistical operations were performed by spss software version 16 and significance level of tests was considered p ≤ 0.05.

Results

After 12 weeks of endurance training, adiponectin level (p=0.000) and Vo2max (p=0.000) increased significantly. However, Insulin resistance (p=0.000), Body weight (p=0.041), Body fat percentage (p=0.000), Glucose (p=0.000) and Insulin (p=0.000) decreased significantly (p ≤ 0.05) (Table 2).

<table>
<thead>
<tr>
<th>Group Index</th>
<th>Endurance</th>
<th>Control</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre test</td>
<td>Post test</td>
<td>Pre test</td>
</tr>
<tr>
<td>Age, year</td>
<td>22.4 ± 1.64</td>
<td>-</td>
<td>22.77 ± 3.06</td>
</tr>
<tr>
<td>Height, cm</td>
<td>160.80 ± 3.43</td>
<td>-</td>
<td>158.80 ± 3.99</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>75.01 ± 6.32</td>
<td>72.80 ± 5.85</td>
<td>75.08 ± 2.52</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>29.13 ± 1.99</td>
<td>28.27 ± 1.83</td>
<td>30.12 ± 1.83</td>
</tr>
<tr>
<td>Fat percentage, %</td>
<td>31.26 ± 1.40</td>
<td>27.75 ± 0.88</td>
<td>31.80 ± 1.57</td>
</tr>
<tr>
<td>Vo2max, ml/kg/min</td>
<td>23.48 ± 2.30</td>
<td>23.96 ± 3.36</td>
<td>23.13 ± 2.49</td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>4.53 ± 0.27</td>
<td>3.68 ± 0.24</td>
<td>4.75 ± 0.26</td>
</tr>
<tr>
<td>Insulin, µIU/ml</td>
<td>12.64 ± 0.29</td>
<td>8.39 ± 0.19</td>
<td>12.60 ± 0.32</td>
</tr>
<tr>
<td>Insulin Resistance Index</td>
<td>2.54 ± 0.17</td>
<td>1.37 ± 0.11</td>
<td>2.66 ± 0.16</td>
</tr>
<tr>
<td>Adiponectin, µg/ml</td>
<td>8.90 ± 0.16</td>
<td>10.81 ± 0.26</td>
<td>8.78 ± 0.19</td>
</tr>
</tbody>
</table>

Table 2: Pre-and post-test physical, physiological and biochemical variables and independent t test in the two groups.

Discussion

In the present study, 12 weeks endurance training decreased insulin resistance. These findings con-
firm those of previous studies that found improvement in insulin sensitivity after endurance training in obese and healthy individuals\(^{(3,5,16)}\). Several mechanisms have been proposed to be responsible for the increases in insulin sensitivity after exercise training\(^{(17-20)}\). These include increased post-receptor insulin signaling\(^{(18)}\), increased glucose transporter protein and mRNA\(^{(19)}\), decreased release and increased clearance of free fatty acids\(^{(17)}\), increased muscle glucose delivery and changes in muscle composition\(^{(20)}\). Accompanied by the above mentioned studies in this paper, the results showed a meaningful decrease in the insulin, blood glucose, body weight and the fat percentage. The weight loss- and exercise-related improvements in insulin sensitivity reported in the present study are in agreement with results from others\(^{(13,17)}\).

In contrast, the results of some studies indicated that the improvement in the insulin sensitivity has nothing to do with the adiponectin and body composition\(^{(21,22)}\).

The results of the current study showed an increase in serum adiponectin levels among subjects who underwent 12 weeks of endurance training; with concurrent reduction in body weight and percent body fat. The current study showed concurrent improvement in adiponectin levels and insulin resistance measured by HOMA-IR and fasting insulin levels. However, there are a few studies that did not observe an increase in adiponectin levels with significant improvement in insulin resistance\(^{(13,17)}\).

These results suggest that changes in adiponectin levels during/after exercise training are not necessary for improvement in exercise-associated improvement in insulin sensitivity.

Excess fat accumulation causes insulin resistance by two different main pathways, including adipocyte-derived cytokine\(^{(23)}\) and lipotoxicity-related altered insulin signaling\(^{(20)}\). However, long-term exercise training that reduces the levels of fat accumulation may also cause further changes in adipokine levels and result in further improvement in insulin sensitivity. Previous studies examining the effects of exercise training on adiponectin levels have reported conflicting results. Some have reported increased\(^{(16,25)}\) and others have reported no changes in adiponectin levels after exercise training\(^{(13,26)}\). Most studies that reported increased adiponectin levels after exercise training also observed significant weight loss\(^{(14,25,27)}\). Esposito et al, observed a 48% increase in adiponectin levels after 2 years of a combined low-energy Mediterranean diet and increased physical activity\(^{(27)}\). It seems that modifications in body weight or body composition might be responsible for alterations in adiponectin levels\(^{(27)}\). From these previous studies, we can speculate that weight loss, more specifically body fat loss, is necessary for the exercise training effects on adiponectin to be revealed.

In addition, Hulver et al also reported no changes in adiponectin levels despite significant increased insulin action and no changes in body weight or fat mass\(^{(13)}\). There are also studies that show exercises to have no effect on the level of adiponectin. That may be because of using a combination of endurance and strength exercises\(^{(28)}\) or using athlete subjects who have higher adiponectin level in baseline or other unknown factors\(^{(29)}\). Based on the negative correlation between adiponectin and body fat percentage found in the present study and to reduce the risks of atherosclerosis by reducing body fat, it could be suggested that patients and normal sedentary individuals need to participate in endurance training programmed that lead to loss of body weight and fat reduction.

References


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Corresponding author
nsprofsport@mail.com