Effects of resistance training on binge eating, body composition and blood variables in type II diabetics

Moisés Simão Santa Rosa de Sousa¹², Victor Manuel Machado dos Reis¹, Jefferson da Silva Novaes³, Josenaldo Mendes de Sousa¹² and Divaldo Martins de Souza¹²

¹Universidade de Trás os Montes e Alto Douro Quinta de Prados, 5001-801, Vila Real, Porto, Portugal. ²Universidade do Estado do Pará, Belém, Pará, Brazil. ³Universidade Federal do Rio de Janeiro, Rio de Janeiro, Rio de Janeiro, Brazil. *Author for Correspondence. E-mail: moisessantarosa@uepa.br

ABSTRACT. This study examined the effects of 12 weeks of resistance training (RT) on binge eating, body composition and blood variables and their correlations in 34 sedentary adults with type II diabetes. The participants aged 58.94 ± 10.66, had body weight of 71.62 ± 11.85 and BMI of 29.64 ± 4.27. Blood samples were collected for analysis of serum leptin, glucose, insulin, LDL, HDL, total cholesterol and triglyceride. The binge eating was assessed by the binge eating scale and the body composition by bioelectrical impedance. The training included three weekly sessions for 12 weeks, with three sets from 12 to 15 repetitions for the main muscle groups, and interval from 1 to 2 minutes between the sets. A significant decrease was found for the binge eating, body weight, BMI, fat percentage, and fat weight. As for the blood variables, there was a significant reduction in leptin; non-significant reductions in glucose, total cholesterol, LDL and triglycerides; as well a significant increase in HDL and non-significant increase in insulin. In conclusion the 12 weeks of RT proved to be enough to decrease the binge eating, to positively adjust the body composition and to modify the blood profile, demonstrating an association at a lower or higher level between these variables.

Keywords: diabetes, resistance training, blood variables, binge eating.

Introduction

At the World Congress on diabetes in Montreal in 2009, the International Diabetes Federation reported that the diabetes epidemic is developing more rapidly than previously thought. If this progression continues, in 2010 there will be 435 millions of diabetic people. Most are located in developing countries, and approximately 80-90% of individuals affected by the disease are overweight and/or obese, and the risk of diabetes is directly related to increased body mass index (BMI) (SARTORELLI; FRANCO, 2003). A large body of evidence suggests that there is an increase of psychopathology in patients with DM. Depression and eating disorders (ED) are among psychiatric disorders that arouse more interest in clinicians and researchers who deal with patients with DM (PAPELBAUM et al., 2007).
Binge eating (BE) refers to episodes of excessive food intake, characterized by the consumption of large amounts of food within short time intervals, followed by a feeling of loss of control over food (APO LINÁRIO, 2004). This unmanageability manifested in the binge eating disorder (BED) may be associated with hormonal disturbances that make difficult the identification of satiety (PAPELBAUM et al., 2005). The risk of the association between eating disorder (ED) and diabetes mellitus (DM) can be evidenced by worse glucose control and increased risks of acute and chronic complications (MELTZER et al., 2001).

Recent advances in the field of endocrinology and metabolism show that the adipocyte is capable of synthesizing and releasing several substances, being now considered an endocrine organ (FRÜHBECK et al., 2001). Among these substances leptin is a protein that acts in the hypothalamus informing about the organic nutritional status. Consequently, the hypothalamus increases the basal metabolism and reduces the appetite in order to decrease the body lipid content (GEBER, 2000). In human, the circulation of blood leptin is result of acute changes in the energy balance resulting from the increase or decrease in caloric intake (KERSHAW; FLIER, 2004). The fasting state or the loss of body mass results in low levels of leptin in the blood, which in turn, generates an enhanced expression of hypothalamic neuropeptide Y (NPY), stimulating thus the dietary intake (BLUNDELL; GILLET, 2001).

Considering the evidences of interaction between diabetes with obesity, binge eating and resistance to leptin as clinical manifestations commonly verified in type II diabetics, (PAPELBAUM et al., 2005; DONATTO et al., 2008), and the capacity of the resistance training to influence the body composition, it is necessary to study the possible chronic adaptations in these variables through this modality of exercise, as well as possible correlations between them. Such understandings try to fill the existing gaps as regards the need to find several ways for treating type II diabetes and its complications. In this sense, this research aimed to verify the effects of 12 weeks of resistance training on the binge eating, body composition and blood variables in type II diabetics and to verify the correlations among these variables.

**Material and methods**

The present research is characterized as almost experimental with sedentary diabetic individuals, of both sexes, with mean age of 58.94 ± 10.66, attended by the cardiovascular prevention program of the Diabetic House in Belém, state of Pará, Brazil, being the sample formed by 34 individuals chosen at random, who have not participated in programs of regular physical activity for the last six months. All the evaluations for data collection were performed before and after 12 weeks of resistance training (RT).

**Binge eating evaluation:** It was evaluated through the questionnaire of the Binge-Eating scale of Freitas et al. (2001), version translated and validated by the department of epidemiology at the Institute of Social Medicine of the Federal University of Rio de Janeiro.

**Body Composition:** It was evaluated through the equipment Byodinamics Body Composition Analyzer – Model 310, with the patient fasting for 12h and after the bladder emptying. The placement of electrodes was tetrapolar: on the dorsal surface of the right hand at the metacarpal joint and between the prominences distal and proximal of the radius and ulna in the fist; on the dorsal surface of the right foot between the medial and lateral malleolus in the ankle, with the patients in the dorsal decubitus position, with the upper and lower limbs apart.

**Evaluation of BMI:** It was obtained through the relationship between the measures of weight (kg) and height (m), expressed in the equation below (ACSM, 2010). The body mass was obtained by weighing in a Fillizola scale, with the capacity between 0 and 100 kg, accurate to 100 g, and the height verified using a Sanny stadiometer (model ES 2020), with measures between 0.40 and 2.20 m height and tolerance of two millimeters.

\[
\text{BMI} = \frac{\text{BM(kg)}}{\text{heigth(m)}^2}
\]

**Blood Evaluation:** The collection of fasting blood for the analysis of leptin, glucose and insulin was made at the laboratory of the Hospital da Beneficente Portuguesa. Samples of venous blood were obtained by puncture of the basilic median cubital vein with disposable hypodermic syringe. The samples were placed in tubes and centrifuged for 15 minutes at 3000 rpm for plasma separation. The analyses were subsequently performed using the following methods: Leptin (Elisa method); Glucose (Enzymatic Colorimetric method); Insulin (Chemiluminescence).

**Assessment of the Muscular Strength:** The initial intensity of training before the protocol of resistance training (RT) was determined at the Resistance Training Laboratory (RTL) of the University of the State of Pará (UEPA).
Considering the sedentary lifestyle and the inexperience with the training, it was opted for the load test through maximum repetitions (RM), which consisted in the execution of a specific number of repetitions of a load to which the person was able to move / raise, for 12 to 15 maximum repetitions, and no more than that, being the load determined through the mathematical model:

**Resistance Training:** It consisted of 10 exercises for the main muscle groups, constituted by 3 series from 12 to 15 RM, with interval from 1 to 2 minutes between series, performed three times a week, in alternate days, during 12 weeks. The period of adaptation of 4 weeks was composed of 6 exercises, leg press (LP), seated supine (SS), leg curl (LC), seated row (SR), high-pulley (HP) and Abdominal (AB), adding in the other weeks four more exercises; leg extensions (LE), presses (PR), dumbbell arm abductions (AA) and chest exercises (CE). The progression of load was given with the increase from 5% to 10% in the load, when was reached 15 repetitions with appropriate technique (ACSM, 2009). As for the movement execution it was considered the individual total functional amplitude, limited by the manifestation of pain or discomfort, being the exercises alternated by body segments in the following sequence: Leg Extension, Press, Leg Curl, Seated Row, Chest, Leg Press, Seated Supine, Abdominal, Dumbbell Arm Abduction, and Seated Pulley.

**Data analysis:** The sample normality was analyzed using Shapiro-Wilk test, in order to verify normality in data distribution. The data received statistical treatment through the program SPSS 16.0, adopting the descriptive statistics to characterize the sample; the Student’s t-test to compare the differences between two moments of testing; the Pearson’s linear correlation to verify the association between variables. It was adopted for the analysis \( p \leq 0.05 \).

The present study was carried out based on the resolution 196/96 of the National Health Council of the Brazilian Ministry of Health, and the project was approved by the Research Ethics Committee, under protocol CAAE – 0013.0.412000-08, being the data used exclusively for the present research.

**Results and discussion**

The Table 1 lists the sample descriptive characteristics, as well as the differences in the studied variables before and after training.

Several studies have shown a high prevalence of psychiatric disorders among individuals with diabetes. However, few have specifically evaluated the prevalence of eating disorders (ED) eating among patients with type 2 diabetes mellitus (T2DM). Most authors who have studied ED in subjects with T2DM have reported high rates of these disorders (PAPELBAUM, 2005). Melo et al. (2009) verified high prevalence of suggestive diagnosis of BED, which is prevalent among women with DM; indicating that the presence of BED in this population favors the increase of the body weight and it negatively influences the metabolic control, contributing to the appearance of early complications related to the disease. Papelbaum et al. (2005) found in the subjects with type II DM a 7.7% prevalence of eating disorder among patients with normal BMI; 18.2% among those with overweight and 25.7% among obese, being the obesity higher amongst women than in men (57.4% against 25.0%; \( p = 0.023 \)) and the (BE) was associated with anxiety disorders.

Table 1. Mean values, standard deviation, Student’s t-test and \( p \) value for age, anthropometric, of body composition and of binge eating variables, before and after training.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Mean ± SD</th>
<th>After Mean ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>58.94 ± 10.66</td>
<td>58.94 ± 10.66</td>
<td>4.77</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>TBW (kg)</td>
<td>71.62 ± 11.85</td>
<td>69.61 ± 11.83</td>
<td>4.86</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.55 ± 0.08</td>
<td>1.55 ± 0.08</td>
<td>3.97</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BMI (kg m(^{-2}))</td>
<td>29.64 ± 4.27</td>
<td>28.80 ± 4.29</td>
<td>4.86</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BFP (%)</td>
<td>35.73 ± 7.42</td>
<td>33.62 ± 8.38</td>
<td>3.97</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>FW (kg)</td>
<td>25.59 ± 6.85</td>
<td>23.78 ± 7.00</td>
<td>3.02</td>
<td>0.01*</td>
</tr>
<tr>
<td>WL (kg)</td>
<td>12.19 ± 6.27</td>
<td>9.61 ± 6.17</td>
<td>6.37</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BE</td>
<td>15.97 ± 9.19</td>
<td>11.56 ± 6.49</td>
<td>4.68</td>
<td>&lt;0.01*</td>
</tr>
</tbody>
</table>

TBW=Total body weight; BMI = Body mass index; BFP = Body fat percentage; FW=Fat Weight; WL=weight to lose; BE= binge eating; *Significant difference \( p \leq 0.05 \) between the situations before X after.

In the present study, before the training, it was verified the presence of (BE) at different levels. Such results corroborate the findings of Melo and OdORIZZI (2009) and Papelbaum et al. (2005). However in the present study, after the training program there was a significant reduction in this variable (\( p < 0.01 \)), evidencing that the long term RT can positively influence the reduction of BMI and BE in individuals with type II DM. The reductions in these variables may have occurred due to the variations in the levels of leptin and adiposity after the period of training, which in turn may have influenced adjustments in the mechanisms involving hunger, because according to Kershaw and Flier (2004) in human the circulation of blood leptin is reflection of the acute changes in the energy balance resulting from the increase or decrease in caloric intake. For Negrão and Licínio (2000), the body weight oscillations indirectly regulate the concentrations of plasma leptin. The loss of body weight leads to the reduction of plasma leptin and
the increase, on the other hand, causes higher concentration of the same.

Regarding the body composition, Cambri and Santos (2006) applied a program with 12 weeks of RT on type II diabetics, composed of 3 series of 12-15 repetitions with three sessions per week, for the main muscle groups and concluded that the RT is effective for the increase in body mass and lean mass, reduction in the percentage of body fat and levels of capillary blood glucose. Castaneda et al. (2002), by studying the effects of 16 weeks of progressive RT on Hispanic men and women aged over 55 years, verified a decrease of 2.2% in the measurement of waist circumference (WC), even with an increase of 0.3% in body weight. In both studies, the reduction of body fat was followed by an increase in the lean mass, which are beneficial adaptations and desirable to improve the health status, considering that obesity is always present in type II DM.

In the present study, the training was able to reduce significantly the total body weight (p < 0.01); the index of body mass (p < 0.01); fat percentage (p < 0.01); fat weight (p = 0.01); the weight to lose(p < 0.01). These results corroborate the findings of Cambri and Santos (2006) and Castaneda et al. (2002) with regards to slimming. These effects are important for the health of diabetics by reducing cardiovascular complications, because according to Jurca et al. (2004), the mechanisms by which the muscular strength contributes to decrease the obesity and its risk factors include the reduction in the abdominal fat, improvement of triglycerides concentration in the plasma, increase of HDL-C and glucose control. For Tevisan and Burini (2007) the strength training is effective to control the body composition, whether by promoting increase of the lean body mass or increasing the resting energy expenditure (REE), allowing the reduction of the body fat.

Regardless of the benefits of the muscular strength training on the body composition in terms of reduction of body fat, the resistance exercises play a key role in the maintenance of lean mass and in contributing to the resting metabolic rate, particularly when included the dietary restrictions for treating obesity (MATSUDO; MATSUDO, 2006). Ropelle et al. (2005) affirm that the skeletal muscle represents approximately 40% of the total body mass, with important role in the glucose metabolism, since this tissue is responsible for 30% of the energy expenditure, being one of the main tissues responsible for the capture, release and storage of glucose.

The Table 2 describes the behavior of the blood variables before and after the 12 weeks of training.

Table 2. Mean values, standard deviation and Student t-test and p value for the variables Glucose, Total Cholesterol, Triglycerides, HDL, LDL, Insulin and Leptin, before and after the training.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Mean± DP</th>
<th>After Mean± DP</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg dL⁻¹)</td>
<td>164.03 ± 69.63</td>
<td>147.26 ± 68.92</td>
<td>1.61</td>
<td>0.12</td>
</tr>
<tr>
<td>TC (mg dL⁻¹)</td>
<td>230.32 ± 71.94</td>
<td>226.09 ± 87.51</td>
<td>0.35</td>
<td>0.73</td>
</tr>
<tr>
<td>TG (mg dL⁻¹)</td>
<td>188.71 ± 97.13</td>
<td>174.65 ± 59.58</td>
<td>1.00</td>
<td>0.32</td>
</tr>
<tr>
<td>HDL (mg dL⁻¹)</td>
<td>38.12 ± 9.15</td>
<td>48.65 ± 7.94</td>
<td>-5.87</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>LDL (mg dL⁻¹)</td>
<td>153.26 ± 66.17</td>
<td>145.41 ± 67.72</td>
<td>0.81</td>
<td>0.43</td>
</tr>
<tr>
<td>INS (mg dL⁻¹)</td>
<td>10.60 ± 9.95</td>
<td>11.58 ± 9.88</td>
<td>-0.98</td>
<td>0.34</td>
</tr>
<tr>
<td>LEP (mg dL⁻¹)</td>
<td>15.72 ± 11.89</td>
<td>13.27 ± 9.73</td>
<td>-3.35</td>
<td>&lt;0.01*</td>
</tr>
</tbody>
</table>

Regarding the blood fat, Cauza et al. (2005) compared in two studies the benefits of weight training and the endurance training (ET) in the treatment of DM and verified a significant decrease in the arterial blood pressure and in the blood cholesterol levels (Total, HDL and LDL) for the group that performed the strength training. As for the body composition, the group which performed weight training had 3.2 times more gain of muscle mass and lost 2 times more fat than the group which performed aerobic exercises. Silva et al. (2010) after conducting a Meta-analysis of many studies on the effects of RT on lipoprotein of low density, suggested that RT may be promising in the reduction of LDL-C levels mainly for adult men and women, patients with type I and type II diabetes mellitus, and women in pre-menopause, but not for elderly population. Fahlman et al. (2002) in a study involving 45 healthy women, aged between 70 and 87 years, without dietary restriction, who performed 10 weeks of RT, consisting of eight repetitions for eight different exercises, verified a significant reduction in the LDL cholesterol and total cholesterol, without changes in the body weight.

In our sample it was possible to confirm that the RT caused a significant increase in the HDL level (p ≤ 0.01); as well as non-significant reductions from the statistical point of view in the glucose level (p = 0.12); total cholesterol (p = 0.73) and in triglycerides (p = 0.32) and LDL (p = 0.43), but important to improve the health of individuals. These results are similar to those found by Silva et al. (2010) and Fahlman et al. (2002). Such evidences support the capacity of RT to reduce the LDL-C, although its effects on lipids are still modest and variable, promoting important reductions in morbidity and mortality in the population with heart diseases, being particularly important to patients with atherogenic dyslipidemia (SZAPARY et al., 2006).
2003). Individuals with predisposition to coronary artery disease (CAD) can achieve benefits from the RT, decreasing the risks of atherosclerosis, by the reduction in its factors, being the LDL-C levels the main among them. In general, the changes in the lipid profile induced by exercises occur through the reduction of mass and body fat and through changes in fat distribution and in the activity of enzymes that regulate the metabolism of lipoproteins (CAMBRI; SANTOS, 2006). Thus, the benefits of physical exercise in the treatment of DM, can be identified not only by the improvement of glucose control, but also by reduction in the risk factors for cardiovascular diseases, because the exercise improves the lipid profile, as well as reduces the arterial blood pressure (TASKINEN, 2002).

Regarding the leptinemia, it does not seem to be altered after a session of low to moderate intensity exercise, unless the exercise is extended (MCMURRAY; HACKNEY, 2005). Besides, the reduction in the leptin levels seems to occur after few hours and/or days from the end of a prolonged physical exercise, or even immediately after a prolonged exercise session, but not short (PRADO et al., 2008).

Kraemer and Ratamess (2005) in a review on the hormonal responses to exercise also concluded that there were no changes in leptin levels in response to a strength exercise session. However, Fantouros et al. (2005) verified significant reductions (3-19%; p < 0.01) after six months of a strength training program in elderly people.

Fatouros et al. (2005) investigating different intensities of strength training in elderly people with overweight, employing (45-50% of 1 RM); (60-65% of 1 RM) and (80-85% of 1 RM) respectively, in a 24 weeks-program, with three weekly sessions, verified chronic reductions in the leptin levels, in which for the group performed training of greater intensity (80-85% of 1 RM) the effects were of greater magnitude. After 24 weeks of detraining, the leptin values have increased again.

In our sample after 12 weeks of RT it was possible to confirm a significant reduction of leptin from 15.72 ± 11.89 to 13.27 ± 9.73 (p < 0.01), corroborating the results of Kraemer and Ratamess (2005), and Fantouros et al. (2005). Such evidences signalize to RT as an exercise modality capable of regulating the leptin level sat long term, assisting in the weight loss. The reductions in the leptin plasma levels after physical training are associated mainly with the reduction in the amount of adipose tissue (EGUCHI et al., 2008). The reference values for women with normal weight are up to 15.1 ng mL⁻¹; for men with normal weight range between 2 and 5.3 ng mL⁻¹ and finally for obese (BMI > 27) with values around 7.02 and 55.04 ng mL⁻¹ (OLIVEIRA, 2009).

As for the blood glucose, Fachineto et al. (2011) analyzed the effects of six months of RT in hypertensive and diabetic people and verified that the blood glucose suffered an average reduction from 181 to 95 mg dL⁻¹, (90.53%). Medeiros (2010) observed a 23% increase in the glucose metabolism after four months of strength training, thereby improving the sensitivity of cells to insulin, thus promoting the improvement in the glucose pattern. Santa Rosa and Domingues (2006) after 2 weeks of RT with 3 weekly sessions in a group of adult men and women with type II diabetes verified an average decrease of plasma glucose from 175.6 to 131.9 mg dL⁻¹ (p = 0.0024).

In the present study the values verified in the blood glucose reduction, even not reaching statistical significance, corroborate the findings of Fachineto et al. (2011), Medeiros (2010) and Santa Rosa and Domingues (2006), becoming a desirable effect to this population. Katzer (2007) explained that the increase in the glucose uptake as a result of physical exercise may be due to the increase in the insulin action caused by the enhanced metabolism, the specific action of exercise on the glucose transporter GLUT4 and the consequent increase of sensitivity to insulin. Besides, in the period post-exercise, the increase in the glucose uptake may be responsible for hypoglycemia within 48 hours after physical activity, being explained by replacement of glycogen by cells and energy expenditure caused by the body recovery. These effects have duration around 2-3 days; for that reason there are the prescriptions of routines of at least three days a week interspersed, being inadvisable for these and other reasons the exclusive activities of weekends.

Regarding the insulin, Ibanez et al. (2005) verified an improvement of 46.3% for sensitivity to insulin in type II diabetics, after 16 weeks of strength training performed twice a week. The improvement in resistance to insulin was accompanied by decrease of 10.3% in visceral fat; 11.2% in subcutaneous fat; and decrease of 7.1% in fasting blood glucose. Ciolac and Guimarães (2004) emphasized an improvement of 22% in sensitivity to insulin after the first exercise session, and in 42% after six weeks, demonstrating acute and chronic effects on the sensitivity to insulin.

In the present study, it was verified an increase of the mean values of insulin levels, increasing from 10.60 ng dL⁻¹ during the pre-training to 11.58 ng dL⁻¹ after 12 weeks of resistance training. In the sample there was also a prevalence of individuals with overweight (50%)
and obese (35.5%). These results are similar to Ibanez et al. (2005). However, many studies aiming to clarify the mechanisms involved in this relationship have demonstrated that the alterations in sensitivity to insulin associated with obesity are the result of abnormalities in this hormone signaling in target tissues. However, the biochemical basis involved in these abnormalities are not yet completely defined (Marreiro, 2004). For that reason the physical activity is decisive for the musculoskeletal sensitivity to insulin. Trained, obese or normoponderal individuals have less insulin than sedentary ones owing two reasons: because regular physical activity increases the muscle capacity in the circulating glucose uptake; because decreases the intramuscular fat, which is associated with the resistance to insulin. In reality, active individuals have lower levels of triglycerides and other intramuscular lipids (Themudo-Barata; Mendes, 2009). Besides, insulin and exercise use different paths that lead to the activation of glucose in the transport to the skeletal muscle, explaining perhaps why patients with resistance to insulin can usually trigger the transport of glucose in the muscle with exercise, but not with insulin (Cardoso et al., 2007).

The adjustments occurred for blood variables in the present study are benefits evidenced as capable to improve the health of type II diabetic people, even considering that some were not statistically significant. In this sense, it is suggested that physical activities, particularly the RT should be part of the treatment for patients with type II DM, not only as a metabolic factor that work independently from the lipid metabolism. Ryan (2000), by studying obese women after menopause found a reduction of 36% in the leptin plasma level after 16 weeks of training with weights, result associated with the reduction in the body weight. Jiménez (2005) verified that the serum levels of leptin and insulin in fasting were significantly associated with the BMI, being higher in obese than in non-obese (p ≤ 0.0001), suggesting a resistance to insulin in obese individuals.

The present study, after 12 weeks of resistance training, the decrease in the leptin values was accompanied by the reduction of the BMI, fat percentage, fat weight and binge eating. Some correlations were significant, suggesting that the long term resistance training may influence the reduction in leptin levels, also imposing modifications in binge eating and in some variables of the body composition.

Feitosa et al. (2007) found that when leptin was corrected per kilo of fat, it did not influence the lipid profile and blood glucose in women with moderate to severe obesity with similar fat percentage, pointing out that leptin should not be considered a factor that work independently from the lipid metabolism. Besides, insulin and exercise use different paths that lead to the activation of glucose in the transport to the skeletal muscle, explaining perhaps why patients with resistance to insulin can usually trigger the transport of glucose in the muscle with exercise, but not with insulin (Cardoso et al., 2007).

Table 3. Pearson’s linear correlation between the variables of the study before and after the training.

<table>
<thead>
<tr>
<th>Correlation</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEPX BMI</td>
<td>0.55</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>LEPX INS</td>
<td>0.29</td>
<td>0.09</td>
</tr>
<tr>
<td>LEPX GLU</td>
<td>0.07</td>
<td>0.64</td>
</tr>
<tr>
<td>LEPX F%</td>
<td>0.75</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>LEPX FW</td>
<td>0.74</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>LEPX BE</td>
<td>0.37</td>
<td>0.03*</td>
</tr>
<tr>
<td>BEX BMI</td>
<td>0.54</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BEX BW</td>
<td>0.55</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BEX TC</td>
<td>0.41</td>
<td>0.02*</td>
</tr>
<tr>
<td>BEX LDL</td>
<td>0.35</td>
<td>0.04*</td>
</tr>
<tr>
<td>BEX FW</td>
<td>0.45</td>
<td>0.01*</td>
</tr>
</tbody>
</table>

**LEP = Leptin; INS = Insulin; GLU = Glucose; F% = Fat percentage; FW = Fat Weight; BE=Binge eating; BMI = Body mass index; BW = Body weight; TC = Total cholesterol; LDL = Low density lipoprotein** *Significant difference (p < 0.05) between the situations before X after.*

The verification of high serum levels of leptin corroborates the theory of resistance to leptin in obese people (NCHS, 2007). This resistance to leptin would be explained by a low sensitivity to the action of the hormone; or the high serum levels of leptin would lead to an inappropriate response (relative deficiency of leptin), however, it is not clear the reasons why high serum concentrations of endogenous leptin are found in obese resistant to leptin (Gomes et al., 2010).

In the present study, after 12 weeks of resistance training, the decrease in the leptin values was accompanied by the reduction of the BMI, fat percentage, fat weight and binge eating. Some correlations were significant, suggesting that the long term resistance training may influence the reduction in leptin levels, also imposing modifications in binge eating and in some variables of the body composition.

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According to Ropelle et al. (2010) in regards to obesity, the chronic intake of saturated fatty acids (fats found in meats) interferes with hypothalamic neurons, causing their inflammation and reducing the anorexigenic response. The dietary intake control is performed a priori by two hormones, insulin and leptin. Consequently, when such inflammation is measured by the excessive consumption of fatty acids, insulin and leptin do not work properly. Indeed, fatty acids can lead to a low-grade subclinical inflammation. Therefore, they do not allow the neuron to recognize well these hormones, which reduce the intake.

In the present study, it was possible to show that the reduction in the leptin levels may have influenced the decrease of binge eating in the studied people, which makes this effect highly beneficial, due to the high presence of risks associated with food factors and obesity in this population.

The high insulin can stimulate the adipose tissue to produce leptin, inhibiting the production and secretion of NPY, causing a reduction in the dietary intake and an increase in the energy expenditure. Independent of the adiposity, insulin may be decisive for leptin concentration. The effect of insulin on the leptin production decreases with the resistance to insulin and with increased adiposity (UEHARA et al., 2004).

Broom et al. (2007) evaluated the hunger behavior (through a visual scale) in 9 eutrophic adult men divided into 2 groups (exercise and control) during and after the practice of exercise for a period of 1 hour at 72% of the VO2 max. The hunger was lower during the first 3 hours for the exercise group when compared with the control group (p = 0.013), suggesting that hunger is suppressed during and immediately after the exercise.

Martins et al. (2007) evaluated the influence of 60 minutes of exercise in cycloergometer, employing 60% of the maximum heart rate on the subjective sensation of hunger in a group of healthy men and women with normal weight, after having a standardized diet. They concluded that the acute exercise of moderate intensity is capable of temporarily decrease the hunger sensation and the negative energy balance.

Conclusion

The program of 12 weeks of RT reduced the binge eating in diabetics. This effect becomes important due to decrease in the dietary intake and consequently cardiovascular complications associated with obesity. Besides, the RT proved to be able of causing positive adjustments in body composition, decreasing the fat weight and the fat percentage, as well as causing slight increase in the lean mass, which may have influenced the body weight reduction and the BMI after 12 weeks of training. Regarding the blood variables, it demonstrated to cause positive adjustments, including a significant reduction in the leptin and HDL levels.

The correlations verified between the variables demonstrated that the decrease in the leptin levels through the RT may positively affect the binge eating, body composition and blood variables, emphasizing the importance of leptin as an important regulator in these variables levels. Given these evidences, it is suggested the RT as a healthy and important alternative of exercise, by the fact that its combined effects promote the improvement of health status of type II diabetic patients and contribute to improve the quality of life, decreasing the risks associated with this disease.

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