- education for health, in parallel with a raise of their interest for physical activities.
- The final test proves the statement above, through the increase of the number of people who know their blood group, from 66 to 240 students, which took us very close to the research hypothesis, according to which many individuals belonging to the young population do not know their blood group because they have no information on the importance of this piece of information, and on the implications it may bring (accidents, etc.);
- The subject discussed in this article is a challenge for specialists, but may be perfected and completed, especially in the section concerning indicators that measure health problems, Physical Education, and life.

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DECEREMENT OF SERUM LEPTIN LEVELS INDUCED BY RESISTANCE TRAINING IN SEDENTARY OVERWEIGHT WOMEN

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Abstract

Purpose. Leptin is a regulator of energy intake and exercise training is an increase factor of energy expenditure. Association of these factors can be new view in prevention and control of obesity.

Methods. The purpose of this quasi-experimental study was too determined of the effect of incremental resistance training on serum Leptin levels, %BF, BMI, and WHR in sedentary, overweight females (n=25). Training program (%60-95 of 1-RM) was performed for 12 weeks, 3 days and 90 min. Within groups comparisons of means were done by

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used a two-tailed paired samples t test (P≤0.05).
Results. Incremental resistance training was reduced body fat percent and serum Leptin levels in sedentary, overweight females.
Conclusions. Therefore, Leptin changes by resistance training are related to body fat reduction.
Key words: Leptin, resistance training, body fat, obesity, exercises.

Introduction

The World Health Organization, at the beginning of the third millennium, was warned the obesity epidemic in the world. Prevention, control and treatment of obesity are designed based on two principles; reduce energy intake and increase energy expenditure (World Health Organization, 2000).

Leptin is a protein hormone that produced and secreted by adipose tissue. The effects of Leptin on the brain are regulating appetite, weight control and some metabolic processes. Leptin dysfunction caused disturbance in energy intake, increased fat mass and obesity.

Serum Leptin levels have a strong positive correlation with fat mass and body mass index. Serum Leptin levels in women are higher than men (Bouassida, Zalleg, Bouassida, 2006; Meier, Gressner, 2004); But when the ratio of body fat measured, the Leptin levels in women and men are equal (Bouassida, Zalleg, Bouassida, 2006; Farah, Cyndy, 2000). A significant increase in energy expenditure (>800 kcal) by exercise training can cause a significant decrease in Leptin concentrations. Leptin is a regulator of energy intake and exercise training is an increaser factor of energy expenditure. Understanding of correlation of these factors can be new view in prevention and control of obesity (Bouassida, Zalleg, 2006).

Weltman, 2000, found that 30 minutes training at different severity and energy costs (from 150 ± 11 to 529 ± 45 kcal) in 7 healthy young men, could regulate Leptin levels during exercise and recovery. Torjman (1999) found that serum Leptin concentrations after 60 minutes treadmill exercise at 50% maximum oxygen consumption in 6 healthy men did not changed and exercise training has no effect on Leptin concentrations.

Landt, 1997, found that non-significant decrease (%8) in fasting Leptin concentrations after two hours exercise at stationary bike in 12 men.

The researchers suggested that moderate exercise training is associated with reduced Leptin. The researchers reported no significant changes in Leptin concentrations were observed (Zoladz, et al. 2005).

Kramer in 2002 stated that short-term exercise (60 minutes <) and training with less than 800 kcal of energy expenditure, cannot change Leptin concentrations.

Reductions recorded in these studies, possibly due to circadian rhythm of Leptin. Essig (2002) reported Leptin concentrations decrease after two hours running on treadmill in each exercise test (800 and 1500 kcal). Miller (2001) stated that the running for 60 minutes at 70% maximum oxygen consumption significantly decreased Leptin concentration;

immediately after exercise, 24 and 48 hours of recovery period. Kramer (1999a) stated that 30 minutes of exercise at 80% maximum oxygen consumption was associated with decreased Leptin concentrations in postmenopausal women (with and without hormone therapy).

Leptin concentrations were measured after 50 sets of resistance training (114.38 ± 855.42 kcal of energy expenditure). Serum Leptin concentrations were lower than control groups at 9, 12 and 13 hours after exercise (Meier, Gressner, 2004). Serum Leptin concentrations significantly decreased in the 30-minute recovery period after exercise compared with baseline values (Zafeiridis, Smilios, Considine, 2003).

The effects of exercise training duration on Leptin concentrations were studied. These studies represent a short duration of exercise (12 weeks >) has no effect on Leptin concentrations, but prolonged exercise (Week 12 ≤) reduced levels of Leptin (Bouassida, Zalleg, Bouassida, 2006).

Humard (2000) stated that short-term aerobic exercise (60 minutes at 75% maximum oxygen consumption for 7 days) not changed Leptin concentrations in healthy middle-aged and young men.

Kramer in 2001 showed that despite the significant reduction of subcutaneous fat after 7 weeks training, Leptin levels were not changed. Kramer (1999b) showed that the 9 week training in obese middle-aged women increased maximum oxygen consumption after the training period, but no significant changes in fat mass or Leptin concentration was observed. Gomez (2002) reported reduction in blood Leptin after 3 weeks of military training.

Channel (2005a, 2005b) stated that regular exercise reduces body fat and serum Leptin levels in young male athletes (in various sports) and healthy sedentary subjects. Plasma Leptin concentrations decreased after resistance training (6 months, 3 days a week, 10 sets per exercise) were reported in 50 sedentary men and this reduction was associated with decreased of total loss of subcutaneous fat mass and body mass index (Fatouros, et al. 2005). Ishii (2001) showed reduction of blood Leptin levels after 6 weeks of aerobic training in type 2 diabetic subjects. Hickey (1997) reported Leptin concentrations decreased after 12 weeks of aerobic training in young women.

This decrease of Leptin concentrations was associated with significant changes in fat mass (Hickey, et al. 1997). Differences in Leptin circadian rhythm after a marathon running and a slight reduction in blood Leptin concentrations associated with the energy costs of marathon running (Leal-Cerro, Garcia-Luna, Astorga, Casanueva, 1998).

Energy imbalance (estimated energy expenditure, 7000 kcal) was associated with reduced blood Leptin levels (Karamouzis, et al. 2002). The results of the close relationship between blood Leptin and energy expenditure, there will be significant. These results suggest that delayed Leptin response to exercise training can be expected to reduce energy equal or greater than 800 kcal (Bouassida, Zalleg, Bouassida, 2006).

It seems that Leptin changes by physical activity and exercise training is related to several factors. These factors include the severity and duration of exercise training, nutritional status of subjects, Leptin circadian rhythm, and the amount of caloric imbalance is caused by exercise.

Therefore, what are the effects of resistance training on serum Leptin levels in sedentary overweight females? Whether, 12 weeks incremental resistance training has the effect on serum Leptin levels in sedentary overweight females?

Methods

The purpose of this quasi-experimental study was too determined and compared of the effect of 12 weeks incremental resistance training on serum Leptin levels in sedentary, overweight females.

Twenty five subjects randomly selected from the 40 sedentary (Physical Activity Rating ≤ 2), overweight (30 > BMI (kg.m^{-2}) ≥ 25), volunteers (Age: 19-25 yrs.) based on a health and disease risk questionnaire and level of physical activity [based on American College of Sports Medicine (ACSM) and Physical Activity Rating (PA-R) Questionnaires].

Physical activities determined by a self-reported exercise habits questionnaire (LeMura, Duvillard, 2004; Nieman, 2003).

All volunteers underwent a medical history, physical examination, and oral-glucose-tolerance test. Medical screening excluded individuals with heart, kidney, liver, thyroid, intestinal, and pulmonary disorders or those taking medications known to affect our outcome variables.

Participants' physicians were consulted and approved the withdrawal of antihypertensive and glucose-lowering therapy for the duration of the study. Pre-intervention washout periods were determined from drug half-lives. In order to participate in the study 25 the subjects signed an informed consent form. At the onset of the study, the subjects were informed about the purpose of the study.

They were told that the results would help researchers to develop better strategies for improving methods of obesity treatments. All the subjects were informed of their rights to anonymity and confidentiality. The Institutional Review Board for Human Subjects at the university approved this study.

Previously physical activity levels were recorded with the use of the Physical Activity Rating (PA-R) Questionnaire; volunteers were deemed sedentary if their PA-R was ≤ 2. Subjects were required to be weight stable for ≥ 6 mount before study participation. This subjects randomly divided in two groups such as, Exercise (BMI: 28.02 ± 3.65; %BF: 41.36 ± 3.64; n= 15) and Control groups (BMI: 27.43 ± 1.25; %BF: 40.23 ± 3.53; n= 10).

All females in the exercise and control groups had no symptoms of cardiovascular diseases, diabetes, or hypertension; based on health/risk factors questionnaire. They had not received any special medications, hormone replacement, or supplements and did not follow any specific diet, based on health/risk factors questionnaire.

Participants were instructed to fast, consume no alcohol, or engage in physical activity for 48 h prior to blood sampling. The research study was conducted at a local indoor aerobic & weight training club in the university. The independent variable was 12 weeks incremental resistance training based on progressive overload training principal.

Training program was based on Association of Sport Sciences Guidelines and it was adjusted by subject's physical condition, gender and age. Training program was performed for 12 weeks, 3 days/week and 90 min/ days. Total time of training program divided as warming up (15 min), main training program (70 min) and cooling down (5 min) at the morning of days (10 – 11.30 am). Training program was started at 60% of One Repetition Maximum (1-RM) at the beginning week and 95% of 1-RM at last week.

Subjects eating habits and other daily physical activity in groups didn't change for 12 weeks; but eating habits and daily physical activity was controlled for two days before tests (table 1).

Dependent variables included fasting serum concentration of Leptin, Body Fat percent (%BF), Body Mass Index (BMI), and Waist to Hip Ratio (WHR) measured at beginning and the end of training program in two groups.

Fasting whole blood samples were collected from the left antecubital vein (seated position) at 7–8 AM after 9–12 hours of fasting by a certified phlebotomist and aspirated into a 5 ml evacuated tube containing sodium citrate in exercise physiology laboratory. Tube were mixed to avoid coagulation, chilled, and centrifuged at 3000 g for 20 min within one hour after sampling. The plasma fraction from tube was each transferred to two plastic vials and frozen at -70° C.

Leptin measured by ELISA from sandwich competitive method type using DRG-Diagnostica, GmbH, Germany kits. Body Fat percent estimated by three-site (Triceps, Suprailiac and/or Suprailium, and Mid-thigh) skin fold method (Jackson et al. Formula); by used a Harpenden caliper. Body mass index (BMI) calculated by used Quetelet Index (W/H^2) [body mass in kilograms (kg), divided by height in meters squared (m2)].

Subject's height and weight measured by used a calibrated medical Seca Bella-840 scale (Germany). Waist to Hip Ratio (WHR) was calculated by dividing the waist circumference in centimeters (cm) by hip circumference in centimeters (cm); by used an inelastic Tape Technique.

The results are presented as the Mean ± Standard Deviation (M ± SD). The normality of the distribution and homogeneity of variances tested with Shapiro-Wilk and Levene's tests respectively. Baseline values for each variable were compared between groups with the use of two-tailed independent samples t tests.

Within group's comparison were done with two tailed paired sample t-test. Between groups comparison were done with two-tailed independent sample t-test. The data were analyzed using SPSS-18 software. Significant levels in all tests were set at P≤0.05.

Table 1. Research Plan.

<table>
<tr><th colspan="18">Table 1. Research Plan.</th></tr>
<tr><td colspan="3">Days</td><td colspan="12">12 Weeks Resistance Training * 3 days/ Week * 90 Min / Day</td><td colspan="3">Days</td></tr>
<tr><td>-3</td><td>-2</td><td>-1</td><td>1</td><td>2</td><td>3</td><td>4</td><td>5</td><td>6</td><td>7</td><td>8</td><td>9</td><td>10</td><td>11</td><td>12</td><td>1</td><td>2</td><td>3</td></tr>
<tr><td colspan="2">Diet & Exercise Controlled
●Cho:%55
●Fat:%30
●Pro:%15</td><td>Pre Test
● Weight
● Height
● Blood Sampling
● 3 Site Skin fold (Triceps, Suprailiac, and Mid-thigh)
● Waist & Hip circumferences
● 1-RM Test</td><td colspan="4">● 4 Sets
● 12 - 15Rep
● 60 -70 %@ 1-RM
● 1 Min Rest between Sets</td><td colspan="4">←1-RM retest
● 3 Sets
● 8 - 12 Rep
● 70 -85 % @ 1-RM
● 2 Min Rest between Sets</td><td colspan="4">←1-RM retest
● 2 Sets
● 3 - 5 Rep
● 90 -95 % @ 1-RM
● 3 Min Rest between Sets</td><td colspan="2">Diet & Exercise Controlled
●Cho:%55
●Fat:%30
●Pro:%15</td><td>Post Test
● Weight
● Blood Sampling
● 3 Site Skin fold (Triceps, Suprailiac, and Mid-thigh)
● Waist & Hip circumferences</td></tr>
<tr><td colspan="3">● Diet & other Physical Activity Controlled</td><td colspan="12">● Diet & other Physical Activity Not Controlled
● General Warm Up: 10 Min (Slow Running and Stretched Training)
● Specific Warm Up: 5 Min (ONE Set Resistance Training @ 50 % of 1-RM @ 15 Rep.
● Main Program of Resistance Training: 70 Min.
1. Leg Extension (Quadriceps Muscles)
2. Chest Press (Pectorals Major Muscles)
3. Elbow Flexion (Biceps Braches Muscles)
4. Elbow Extension (Triceps Braches Muscles)
5. Hip Abduction (Hip Abductors & Gluteus Medius Muscles)
6. Weighted Alternating Sit-up (Rectus Abdominis and Internal & External Oblique's)
● Cool Down: 5 Min (Slow Running and Stretched Training)</td><td colspan="3">● Diet & other Physical Activity Controlled</td></tr>
</table>

Results

IN table 2, Means ± Standard Deviation (M ± SD) of the variables and the results of statistical tests of groups are presented. Means differences of Leptin (Exercise: 15.46 ± 2.36 vs. Control: 16.90 ± 1.78 $ng.ml^{-1}$) were significant between groups in posttest.

Means differences of Leptin in pretest (16.03 ± 2.58 $ng.ml^{-1}$) and posttest (15.46 ± 2.36 $ng.ml^{-1}$) of exercise group were significant (p =0.012*).

Means differences of %BF (Exercise: 40.94 ± 3.33 vs. Control: 40.45 ± 3.83) were not significant between groups in posttest. Means differences of %BF in pretest (41.36 ± 3.64) and posttest (40.94 ± 3.33) of exercise group were significant (p = 0.036*).

Means differences of BMI (Exercise: 27.89 ± 3.54 vs. Control: 27.48 ± 1.32 $kg.m^{-2}$) were not significant between groups in posttest.

Means differences of BMI in pretest (28.02 ± 3.65 kg.m^{-2}) and posttest (27.89 ± 3.54 kg.m^{-2}) of exercise group were not significant (p = 0.090).

Means differences of WHR (Exercise: 0.78 ± 0.052 vs. Control: 0.79 ± 0.030) were not significant between groups in posttest.

Means differences of WHR in pretest (0.78 ± 0.055) and posttest (0.78 ± 0.052) of exercise group were not significant (p = 0.141).

Table 2. Means ± Standard Deviation of the variables and the results of statistical tests in groups (P≤0.05).

Variables	Groups	Pre test	Post test	sig
Leptin (ng.ml^{-1})	Exercise	16.03 ± 2.58	15.46 ± 2.36	0.012*
	Control	16.89 ± 1.96	16.90 ± 1.78	0.900
%BF	Exercise	41.36 ± 3.64	40.94 ± 3.33	0.036*
	Control	40.23 ± 3.53	40.45 ± 3.83	0.190
BMI (kg.m^{-2})	Exercise	28.02 ± 3.65	27.89 ± 3.54	0.090
	Control	27.43 ± 1.25	27.48 ± 1.32	0.450
WHR	Exercise	0.78 ± 0.055	0.78 ± 0.052	0.141
	Control	0.79 ± 0.029	0.79 ± 0.030	0.170

Discussions

Physical activity and exercise training associated with Leptin reduction and energy imbalance, increased insulin sensitivity, changes in lipid metabolism and lipid concentrations and related factors (Bouassida, Zalleg, Bouassida, 2006).

Due to these reasons, it seems that Leptin changes by physical activity and exercise training is related to several factors. These factors include the intensity of exercise (moderate to severe), duration of exercise per session (≥ 60 min), duration of training period (≥ 12 week), the type of exercise (aerobic and / or resistance), nutritional status of subjects, Leptin circadian rhythm, time and amount of caloric imbalance (≥ 800 kcal) is induced by exercise.

In this study, incremental resistance training was reduced body fat percent and serum Leptin levels in exercise group and mean difference of WHR and BMI were not significant.

The results of Hickey (1997), Leal-Cerro (1998) Okazaki (1999), Kramer (1999a), Olive (2001), Ishii (2001), Gomez (2002), Zaccaria (2002), Karamouzis (2002), Essig (2002), Nindl (2002), Zafeiridis (2003), Unal (2005a and 2005b) and Fatouros (2005), indicated that the relationship between exercise training and physical activity and reducing serum concentrations of Leptin, % Fat and BMI in males and females with different age, severity and duration of exercise training and physical activity; But the results of Landt (1997), Torjman (1999), Gippini (1999), Kramer (1999b), Weltman (2000), Houmard (2000), Kramer (2002 and 2001), Bouassida (2004) and Zoladz (2005) showed Leptin responses during exercise and recovery periods is still not completely clear.

There are several reasons that can explain the behavior of Leptin to physical activity and / or exercise training; such as reduction of fat mass and the concentration of hormones (insulin, cortisol, growth hormone, catecholamine, testosterone, etc.) and metabolites (free fatty acids, lactic acid, high triglyceride, etc.) have a decisive role in energy consumption (Bouassida, Zalleg, Bouassida, 2006).

Conclusions

Therefore, incremental resistance training was reduced body fat percent and serum Leptin levels in sedentary, overweight females. Due to these reasons, it seems that Leptin changes by incremental resistance training are related to body fat percent reduction.

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