Full Length Research Paper

Effects of Resistance Exercises on Serum Leptin and Some Inflammatory Markers in Obese Males

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Abstract. The main purpose of the present study was to assess the effects of resistance exercise (RE) regimens on leptin concentrations and on risk factors for coronary heart disease in obese sedentary males. Thirty subjects were recruited into this study, 20 control subjects and 20 individuals for resistance exercise. Subjects ranged in age from 20 to 40 years. The subjects reported in the laboratory on the morning after a 12-h fast. Blood (10 ml) was obtained from an antecubital vein, with the subject in an upright position, before exercise and at 10 h after six weeks of resistance training. In conclusion, after resistance exercise, leptin and CRP showed significant differences when compared to resting values. In addition, these values were related to each other after exercise. Other cytokines and different types of subjects should be included in further studies.

Key words: Leptin, IL-6, CRP, Resistance Exercise

1. INTRODUCTION

Obesity is a complex disorder characterized by the accumulation of excess adipose tissue. The prevalence of obesity and its secondary health risks have dramatically increased over the last decades (Mokdad et al., 2003). Obesity is an important risk factor for the development of vascular disorders (Cooke and Oka, 2002; Krauss et al., 1998). Adipose tissue, in addition to the storage of lipids, secretes bioactive peptides termed “adipokines” which act locally and distally through autocrine, paracrine, and endocrine pathways. In obesity, increased production of most adipokines impacts on multiple functions such as appetite, energy balance, immunity, insulin sensitivity, angiogenesis, blood pressure, lipid metabolism and homeostasis, all of which are linked to cardiovascular disease (Ronti et al., 2006).

Discovery of the hormone leptin in 1994 catalyzed the field of obesity research by demonstrating the existence of an afferent hormonal signal from adipose tissue to the central nervous system (Kraemer et al., 2002; Zhang et al., 1994). Leptin is a 16 kDa peptidic hormone produced mainly by adipose tissue, which acts as a signalling mechanism to regulate body-fat content through binding to leptin receptors located in hypothalamic nuclei (Kraemer et al., 2002; Thong et al., 2000). Increases in percent body fat are associated with enhanced adipose tissue synthesis and secretion of leptin in humans (Barbato et al., 2006; Hilton and Loucks 2000). Leptin, commonly termed the obese protein, has been implicated in regulating an array of physiological processes such as appetite, metabolic rate, reproduction, immunity (Kraemer et al., 2002).

Leptin has been demonstrated to induce insulin resistance (Houmard et al., 2000). Leptin is involved in the pathogenesis of vascular disease and may represent a link between obesity, diabetes, inflammation and atherosclerosis (Krauss et al., 1998; Livshits et al., 2005). Leptin increases energy expenditure by enhancing sympathetic nervous activity and lipolysis (Kraemer et al., 2002; Fenkci et al., 2006; Konstantinides et al., 2001). It also suppresses appetite through acting on the hypothalamus (Mokdad et al., 2003; Ronti et al., 2006; Mendosa-Nunez et al., 2002). In recent years, leptin has been reported to increase arterial pressure and heart rate by peripherally or centrally mediated mechanisms (Livshits et al., 2005; Franklin 2005; Rahmouni and Haynes 2004). The finding that leptin is linked to heart-disease risk independently from C-reactive protein (CRP), an inflammation marker, strongly suggests that fat may be important in heart- disease risk (Altman 2003; Canavan et al., 2005). Leptin deficiency and resistance against the effects of leptin are each associated with weight gain. Leptin resistance is much more common than leptin deficiency in human obesity (Mokdad et al., 2003; Krauss et al., 1998; Mendosa-Nunez et al., 2002). There are receptors for leptin on the endothelium and on vascular smooth muscle cells. Accordingly, leptin can exert receptor-mediated influences on vessel tone.
and growth. In cell culture, leptin stimulates vascular smooth muscle proliferation (Rahmouni et al., 2004; Singhal et al., 2002). Vascular calcification is also accelerated by leptin in experimental models (Cooke and Oka, 2002; Franklin, 2005). Additionally, leptin induces oxidative stress in endothelial cells (Altman, 2003; Canavan et al., 2005). Accordingly, it is possible that the high level of leptin observed in obesity contributes to its adverse effects on cardiovascular health (Wallace et al., 2001).

Exercise has been shown to reduce leptin levels regardless of weight loss (Bouassida et al., 2006). Young obese but otherwise healthy subjects are characterized by reduced coronary vasoreactivity (Mcgill et al., 2002). It has become of interest to examine whether physical activity, through its disruptive effects on energy balance, sympatoadrenal drive, and hormonal and metabolic homeostasis, affects serum leptin concentration (Zafeiridis et al., 2003). If leptin is reflective of energy balance, it is conceivably that an increase in energy expenditure, i.e. physical activity, may also modulate plasma leptin (Houmard et al., 2000). The effect of exercise on leptin is also potentially important in relation to insulin action (Hickey et al., 1996; Perusse et al., 1997; Pasman et al., 1998; Ryan et al., 2000). Most studies examined the effects of resistant exercise on serum leptin by the utilization of continuous running regimens (Kraemer et al., 2002; Perusse et al., 1997; Racette et al., 1997; Weltman et al., 2000). Most of these studies have reported a reduction or no change in leptin concentrations. Information regarding the response of serum leptin to a single bout of resistance exercise is limited. Zafeiridis et al., 2003 reported that leptin concentration had significantly decreased 30 min after resistance exercise protocols compared with the respective baseline value, but the decline in serum leptin compared with that observed during the control session. Some authors reported that exercise training reduced fasting leptin disproportionately more in a subgroup of women with high adiposity than in leaner women (Hickey et al., 1996; Houmard et al., 2000). There is thus the potential that individuals with larger fat masses respond differently to exercise than leaner subjects in terms of leptin (Houmard et al., 2000). Evidence suggests that, in trained individuals, acute exercise has no effects on circulating leptin levels (Weltman et al., 2000). Exercise can improve metabolic risk variables such as insulin and leptin in overweight and obese post-menopausal women (Barbato et al., 2006). Leptin concentrations exhibited a delayed reduction in the systemic circulation after a resistance exercise protocol (Zafeiridis et al., 2003). Leptin response patterns are a direct result from the intensity and duration of high-energy expenditure and the subsequent excess post-exercise oxygen consumption of the acute resistance exercise protocol (Nindl et al., 2002). In addition, energy expenditure, in exercise is the most important factor that affects leptin concentrations (Kraemer et al., 2002; Zafeiridis et al., 2003).

Although many studies have been published about the effects of exercise on leptin, numerous questions remain to be answered. It is necessary to compare the effects of different exercise protocols on leptin levels in males and females. There is clear evidence for the importance of exact control of energy balance in leptin and exercise studies (Hilton and Loucks, 2000). The evidence that leptin levels decline 9 h after resistance exercise suggests that there is a delayed reduction in leptin that may be due to energy imbalance (Nindl et al., 2002). Thus, in order to determine the true dynamics of exercise-induced leptin responses, studies should determine changes in leptin concentrations for much longer periods of time following exercise. The studies should involve strict controls of energy balance. The main purpose of the present study was to assess the effects of resistance exercise (RE) regimens on leptin concentrations and on risk factors for coronary heart disease in obese sedentary males.

2. MATERIALS AND METHODS

2.1. Subjects

Thirty subjects were recruited into this study, 20 control subjects and 20 individuals for resistance exercise. Subjects ranged in age from 20 to 40 y (Table 1). The control subjects had a mean body mass index of 32.5±1.1 kg/m2. All subjects signed informed consent. Individuals taking insulin or with symptomatic coronary artery disease, peripheral vascular disease, uncontrolled hypertension or any other metabolic diseases were excluded from the study. Both the control and exercise subjects had participated in regular exercise for the preceding six weeks, and all subjects had the stable body weight. All subjects were non-smokers.

<table>
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<th>Table 1: Subject characteristics</th>
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<td><strong>Age (y)</strong></td>
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<tr>
<td><strong>Height (cm)</strong></td>
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<td><strong>Weight (kg)</strong></td>
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<td><strong>BMI (kg/m²)</strong></td>
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2.2. Blood sampling

The subjects reported in the laboratory on the morning after a 12-h fast. The resistance sessions as well as the control sessions were conducted at the same time of the day in each subject to avoid the effects of fasting and circadian rhythm. The time each exercise session started was adjusted so that the blood samples drawn at 10 h of recovery were obtained at the same time of the day for all sessions. Blood (10 ml) was obtained from an antecubital vein, with the subject in an upright position, before exercise, immediately after exercise and at 10 h of recovery. Blood was collected in glass vacutainers, allowed to clot at room temperature, and centrifuged for 30 min at 800×g at 4°C. After centrifugation, serum was aliquoted into a storage vial in liquid nitrogen, and stored at –80°C for later analysis. Leptin and CRP concentrations were determined using an enzyme-linked immunosorbent assay from Linco Research Inc., (St Charles, MO, USA) and Immulite (Diagnostics Products Corp., Los Angeles, CA, USA), respectively. The sensitivities of these kits were 0.01 ng/ml, and the intra- and interassay coefficients of variation were 7.8% and 5.1%, respectively. The limit of detection and the intra- and interassay coefficients of variation were 0.15 μIU/ml, 5.3% and 9.5%, respectively.

2.3. Body composition

On the first and last testing day, subject height and weight were measured. Sagittal diameter and waist measurements were taken at the umbilicus. Body composition was measured utilizing a Quantum Bioelectrical Impedance Analysis Machine (BIA101Q) by RJL Systems. All subjects were measured between 08:00 and 09:00 h and were in a hydrated state, such that fluids were not restricted and no caffeine had been consumed in the previous 24 h. The premenopausal women were studied in the same phase of their menstrual cycle pre- and post-training.

2.4. Diet

Subjects were asked to consume the same diet prior to each testing period. They were asked to record their diet the day prior to the blood sampling day. After the first sampling day, subjects were reminded what to eat on the days prior to the following sampling days. To ensure compliance, all subjects kept a dietary record on the day prior to the sampling day.

2.5. Resistance exercises training

All subjects performed six weeks of progressive, resistance training. Subjects trained at 80% of 3RM, three times per week. All training sessions were supervised and conducted on alternate days. For each muscle group, the subject was required to complete three sets of 8 – 12 repetitions to failure. Three sets of 15 abdominal crunches were also prescribed. Once subjects were able to complete the 12 repetitions, the weight was increased by 2.3 kg. At the completion of the training, the subjects repeated a 3RM.

2.6. Statistical analysis

The statistical analysis was run using the Statistical Package for the Social Sciences (SPSS, version 8.0). All data are expressed as mean±s.e. An analysis of variance (ANOVA) with repeated measures was used to evaluate the leptin, CRP and IL-6 differences; group (control vs experimental) by time (pre-training vs post training).

3. RESULTS

In Table 2, mean and standard deviation of age, height, weight, body masses were shown. Table 3 shows mean and standard deviation for leptin, CRP and IL-6 before and after training. The results of Tables 3 show that there is significant difference between leptin, CRP and IL-6 concentrations before and after training to a resistance training group.

Table 2: Mean and standard deviation of age, height, weight and body mass index

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>Resistance</th>
<th>control</th>
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<tbody>
<tr>
<td></td>
<td>pre</td>
<td>post</td>
</tr>
<tr>
<td>BMI</td>
<td>30.7±1.1</td>
<td>29.8±0.8</td>
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<tr>
<td>WEIGHT</td>
<td>87.2±5.57</td>
<td>79.2±2.6</td>
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<tr>
<td>HEIGHT</td>
<td>170.2±5.77</td>
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</tr>
<tr>
<td>AGE</td>
<td>32.5±2.1</td>
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</table>
4. DISCUSSION

Previous studies have reported conflicting findings regarding the leptin response to exercise. Acute resistance exercise has been reported to have no impact (Livshits et al., 2005; Fenkci et al., 2006; Konstantinides et al., 2001; Franklin, 2005; Rahmouni et al., 2004; Altman, 2003) or to decrease plasma leptin levels in these studies (Mendosa-Nunez et al., 2002). After 20 weeks of training a decrease in leptin levels was observed in men, but only if decreases in body fat occurred (Franklin, 2005). Recent work has suggested that leptin levels may not only indicate the quantity of adipose tissue but may reflect disturbances, in energy balance, disturbances which may not manifest themselves for a number of hours after exercise (Bouassida et al., 2006). In the present study, we observed a significant decrease in leptin concentrations 24 h after a resistance exercise in experimental subjects, and only a slight decrease in control men. Further this decrease was an acute response because 6 weeks of resistance training resulted in only slight changes in the leptin concentrations compared to pre-training values when resting samples were taken 72 h post-exercise. Twenty-four hours post-exercise, we observed a 30% decrease in leptin concentrations in the experimental subjects, and a 7% decrease in the control subjects. The decrease observed in the experimental subjects is similar to that observed in moderately trained is similar to a decrease observed with weight loss with a diet and exercise program 24h. In addition, an exercise bout causing a 28% increase in 24 h energy expenditure, while maintaining energy balance, significantly decreased peak and average 24 h plasma leptin levels by 20% compared to pre-exercise values. They also noted that this effect could only be detected after 24 h, but not immediately after the exercise bout. It was suggested that the delay in leptin response was due to the time needed for changes in on gene expression in adipose tissue (Singhal et al., 2002; Weltman et al., 2000). This could potentially explain why many earlier studies did not observe a change in leptin levels immediately post-exercise (Livshits et al., 2005; Fenkci et al., 2006; Konstantinides et al., 2001; Franklin, 2005; Rahmouni et al., 2004; Altman, 2003). Recently, Livshits et al., 2005; Bouassida et al., 2006 have noted no change in leptin concentrations with exercise. Hilton and Loucks, 2000 stated that exercise stress per se does not have a suppressive effect on either the 24 h mean or amplitude of the diurnal rhythm of leptin release, other than impacting energy availability. These authors state that low energy availability to the tissues or a negative energy balance has a much greater impact on leptin release than the exercise stress. In addition to a negative energy balance, it is possible that the decreased leptin concentrations in the present study were due to reduced glucose availability in the post-exercise period (Bouassida et al., 2006). Intense aerobic30 and anaerobic exercise 31 have been shown to significantly decrease muscle glycogen. Our resistance training protocol involved whole-body lifting at 80% of 3RM and most likely decreased glycogen stores.

We know that 72 h following our 6 weeks of training no decrease in mean plasma leptin levels was observed, but we are unable to conclusively state that a chronic acute effect at lower leptin levels occurs with six weeks of resistance training.

CRP is an inflammatory index in the human body and a predictor of heart-disease risk at rest. With regard to the effect of exercise on CRP, the results from the current study are similar to those of previous studies, i.e. CRP increased significantly after each acute bout of exercise at the three different intensities. Although post-exercise CRP levels showed an upward trend with higher exercise intensity, a significant difference was not detected among the different exercise intensities. CRP might be related to leptin because CRP binds with the receptor of leptin. This would result in a “leptin resistance” phenomenon as CRP occupies the leptin receptors, and more leptin circulates through the blood. Although the exercise model for this study differed from that of Chen et al. study, this study provided another direction for exploring the relationship between leptin and CRP.

5. CONCLUSION

After resistance exercise, leptin and CRP showed significant differences when compared to resting values. In addition, these values were related to each
other after exercise. Other cytokines and different types of subjects should be included in further studies.

REFERENCES


Alireza zamani is a member of faculty in physical education & sport sciences at Islamic Azad University - Dehaghan branch in Iran. He graduated from Islamic Azad university - Khurasgan branch Malaysia in 1998. He had many publishing about management and methods of research on physical education and sport sciences.

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