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Effects of a single session of concurrent exercise on leptin levels and insulin resistance index in sedentary males

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Abstract

Physical activity is known to influence and regulate leptin hormone secretion, which assists in the regulation of body weight and energy homeostasis. The purpose of this study was to investigate the effects of a single session of concurrent exercise on leptin levels and insulin resistance, until 24 h post-exercise. In semi-experimental study, ten male subjects (22.9±1.7 years) healthy volunteer performed a single session of concurrent aerobic and resistance exercise (Aerobic exercise: 60%-70% of their maximal oxygen up take for 20 min and resistance exercise: 2 sets of 10 repetitions at 70% of 1 repetition maximum). Leptin, glucose, insulin and insulin resistance index were measured before and 24 h after exercise. Significant difference was observed between serum leptin levels (5.75±1.06 ng.ml-1 vs, 5.06±0.62), insulin resistance index (1.21±0.18, vs. 1.03±0.13) and insulin concentration (6.02±0.54 UIμ.ml^{-1} vs, 5.30±0.44). However, serum glucose levels (4.52±0.27 mmol.l-1 vs., 4.37±0.36) remained unchanged before and 24 h post-exercise. Significant correlations was observed between insulin resistance index to insulin levels and glucose and also between serum leptin to insulin levels and insulin resistance index, Obtained results of this study showed that a bout concurrent exercise could improve glycemic indexes in sedentary males; therefore, it is suggested to use this exercise as a preventive method for insulin resistance disorders.

Keywords: Glycemic, Concurrent, Insulin Resistance, Leptin, Sedentary

Introduction

In recent decades, the post industrialized world has experienced a sharp increase in energy intake and a proportional decrease in daily physical activity. A great concern is the evidence that excessive body weight and an absence of regular physical activity or physical unfitness are associated with the risks of type 2 diabetes, hypertension, and other chronic diseases in progressively younger individuals [1]. Human adipose tissue secretes multiple cytokine proteins that mediate several biological effects. These proteins are in the adipocytokine family and include tumor necrosis factor-alpha (TNF-α), adiponectin, resistin, interleukin-6 (IL-6), adipsin and leptin [2]. The targets of TNF-α are included immediate inhibition of insulin-induced insulin receptor and insulin

receptor substrate 1 (IRS-1) phosphorylation. Adiponectin, adipsin and resistin are direct involvement in insulin sensitivity changes which secrets from pancreatic β-cell. Leptin and insulin are the master hormones, which are controlling body weight. Leptin, in particular, signals the brain to store or burn fat and used by the body to regulate energy use through metabolism and appetite. Leptin, the product of the adipocyte ob gene is a secreted hormone that communicates to the brain the amount of energy content and also affected by gender [3]. Insulin hormone regulates how fat is used or stored in the body. However, cells in the body can become resistant to leptin and insulin. When this happens, the body requires higher levels of these hormones to get across the proper signals. This often leads to a distorted appetite, poor energy levels, and unwanted fat gain. In the long term, it can also increase risk for developing metabolic conditions like diabetes and heart disease. Although serum leptin levels are most strongly related to the amount of fat in the adipose tissue in the body [4], several other factors have also been shown to influence serum leptin concentrations [3,4]. For example, weight loss results in a reduction and weight gain in an increase in leptin levels [5,6]. Therefore, it is clear that leptin and insulin play a vital role in controlling food intake and metabolism in the form of energy expenditure which is affected by life such as food habits.

It has been reported that insulin as a recreational drug (except the obesity) has no effect on leptin synthesis in hyperlipedemia and insulin resistant resistance individuals. While there has been report which indicating the relation between hyperlipidemia and insulin resistance independent to the BMI in the Dutch males and females [7]. In contrast, the leptin was found to be increased independently in obesity and showed a negative correlation with physical activity level [9,10]. When the leptin level is low and normal, it acts as insulin inducer and its elevation may related to the insulin resistant [7]. There is a similar relationship between the leptin level and bodily inactivity. The mice with leptin deficiency have low activity, while leptin injection in such animals induces their physical activity [8]. However, other cross sectional studies did not agree with these findings [11,12]. Indeed a positive relation was found between the leptin level and physical activity in the Dutch natives children [12]. As it is known, exercise is a potent stimulus for secretion of many hormones [13]. Although exercise training has reduced serum leptin levels [14,15,16], the effects of exercise is independent of loss of percent fat reduction. In the limited number of studies where acute exercise was examined [17, 18], short-term exercise had no effect on serum leptin levels. In contrast, recent data suggest that prolonged exercise [marathon run, ultra marathon, 3-h cycling] may result in a fall in serum leptin levels [19].

A “critical” level of exercise may mediate energy expenditure may needed in order for serum leptin levels to be lowered by acute exercise. In contrast to continuous running of moderate intensity, heavy resistance exercise is a potent non-oxidative stimulus that produces differential neural, metabolic and neuroendocrine responses.

Information regarding the response of serum leptin to a single bout of resistance exercise is limited. One study reports reduced 24-hr serum leptin in diabetic not in healthy individuals [20] and another study reports reduction of serum leptin levels 9-13 hrs post-exercise in healthy lean men [21]. It has been stated that leptin plays role in control of insulin resistance and improvement of physical activity but little is known about the acute effect of concurrent exercise on these parameters in sedentary males. Hence, it is important to know the effect of acute exercise on leptin level, which may help in understanding the mechanism of leptin action on obesity prevention.

Therefore, this study was conducted to determine the effect of one session of combinative exercise (aerobic- resistance) on leptin, glucose, insulin concentration, insulin resistance and also their relation among the sedentary young males.

Method

This is a semis experimental study that performed for investigation of acute leptin concentration and insulin resistance indexes responses in (young sedentary males. Ten healthy [young sedentary male university student at 2013) with no history of having participation in any regular exercise program, Smoking, drugs, endocrinological or psychological disordered were selected randomly by using pre participation physical and history evaluation forms. participant characteristics are shown in Table 1. All participant were informed the nature and possible risks of the experimental procedures before their informed consent was obtained. To obtain a meaningful result, sample size was calculated by taking leptin and insulin levels seropositivity amongst sedentary males as 60% and alpha 0.05 as observations in a statistical sample.

All subjects reported to the laboratory in the morning after an overnight fast, for measurement of body composition, and to become familiar with exercise plan and sports equipments. Body weight was measured by Digital Glass Scale (GES-07, USA with accuracy of ±0.1kg) without shoe and minimum clothing cover. Their height was measured by wall height meter (model 44440, made by Kaveh Co., Iran, ± 0.1cm) in standing mode next to the wall without shoes while their shoulders were in normal conditions. Waist size was measured in the thinnest part when subjects were ending their expiration. For measuring the hip size, the most projected part was selected. Waist and hip size were measured by non-tactile tape meter without forcing pressure. Body mass index (BMI) was calculated by dividing body weight (kg) by height [meters squared]. Body density was estimated using the three-skinfold site method of Jackson and Pollock [22]. Percentage body fat was estimated with the formula of Siri [23]. To remove the individual errors, all measurements were conducted by the same person. Techniques for proper use of treadmill and weightlifting were taught to the subjects. Subjects began their activity to know and determine a one maximum repeat (1RM) to estimate the maximum oxygen uptake (VO_{2max}) by Bruce maximal exercise testing after 10 min special warming up. Three days later determining the one repeat maximum test (1RM) and test for estimating the VO_{2max}, subjects came to the laboratory at 8:00 a.m. in fasting conditions for measuring leptin and insulin resistance. After sampling their blood, they ate the same breakfast containing 550 kcal and one hour later began their activity. After 24-hour activity, they came to the lab for the second time to be measured for blood factors. The specialist collected 2 ml their blood samples in the same temporal and spatial conditions.

One session concurrent exercise was including warming up (10 minutes), special warming up (3-5 minutes), aerobic training, resistance training, stretching training and cooling (5 minutes). Aerobic training included 20 minutes running on treadmill and 60%-70% of Vo2 max and resistant training at intensity of 70%, of one maximal repeat with 10 repeats in each activity, in two sets, with 30 seconds rest between stations and 2 minutes rest between each round was considered. The resistant training included 10 circulatory stations movement. The stations included: (1) leg flexion, (2) leg extension, (3) leg press, (4) squat, (5) lat pull down, (6) bench press, (7) cross movement by dumbbell, (8) biceps curl, (9) triceps push-down, (10) sit-up, respectively. Blood samples were obtained from all subjects at 8am after an overnight fasting before and after the exercise program. Post-exercise blood samples from subjects in the exercise groups were obtained 24 h after their exercise session. Subjects were asked to avoid strenuous exercise for 2 days prior to the blood collection.

Serum glucose concentration was measured by the enzymatic glucose oxidative method (kit from Pars Azmon Co. Iran) by Hitachi Auto Analyzer 902 Germany. Serum leptin level was measured using ELISA technique by leptin kit (DRG-Diagnostica, GmbH, Germany). With sensitivity of 1 ng/ml and intra- and inter- assay coefficients for leptin

were 4.5% and 6.6% respectively. The serum insulin level was determined using ELISA technique by insulin kit (DRG-Diagnostic, GmbH, Germany) with sensitivity of 1.76 µUI/ml and intra- and inter- assay coefficients for insulin were 2.19% and 4.43% respectively. Insulin resistant index was assessed utilizing the Homeostasis Model Assessment (HOMA-IR) method and calculated using the following formula: fasting plasma glucose (mg.dl-1) * fasting plasma insulin (µUI.ml-1) * 405-1 [24]. Subjects were prescreened prior to entry into the exercise study to ensure compliance with the typical American Heart Dietary intake recommendations [i.e., 50–60% carbohydrate, <30% fat, 10–15% protein] [25]. Three-day [two-week day and one weekend day] dietary recalls were used for this analysis. Subjects were also given standard dietary instructions for nutrient intake during the 3 days prior to the exercise trial. Total mega joule (MJ) (total kilocalories/238.95) intake range recommendations were based on age, gender and body weight and from estimated resting metabolic rate Harris & Benedict [26]. Information from physical activity questionnaires (high, low, moderate activity) was also used to aid in the calculation of total MJ intake to ensure that subjects were in energy balance prior to the exercise trial [27]. Food exchange lists with serving sizes were used for nutrient recommendations (Health Management Resources, Boston, Mass., USA). Subjects were asked to complete dietary records for all 3 days prior to the exercise trial. Nutrient intake and distribution (total MJ intake, % fat, % carbohydrate, and % fat) was completed using Software Dorosty Food Processor (NIII, FP2). All data sets were normally distributed according to the Shapiro-Wilk procedure. Data are presented as means ± standard deviations (SD). Results after rest and after exercise were compared with Student's paired, two-tailed t test. Relationships between variables were examined with Pearson correlation coefficient. Differences were considered significant at $P<0.05$. SPSS-18 (SPSS, Chicago, IL, USA) was used for all statistical analysis.

Results

Physical: Body compartment and demographic characteristic of the participants are shown in Table 1. Ten adult healthy volunteer male with average age of 22.9±1.7 years have participated in this study. Data have presented as mean and standard deviation for the impedance measurements. Measurement was performed 3times to omit any error and the average values entered was the final measurement. In addition, glucose, insulin, leptin serums concentration and insulin resistance before exercise were in normal ranges for healthy individual adults.

Table 1 *Physical characteristic of the participants*

Variable	Mean ± SD
Age [years]	22.9±1.7
Height [m]	173.8±10.3
Weight [kg]	70.67±8.57
BMIa [kg.m-2]	23.32±1
Body Fat [%]	18.11±1.3
Vo_{2max} [ml.kg-1.min-1]	36.27±3.81
W/Hb	0.88±0.016

a- Body Mass Index (BMI)
b- Waist Hip Ratio (WHR)

Energy and macronutrient intake: Analysis of diet regimen data in the subjects showed there is no significant difference in food absorption at three times of measuring before exercise, energy (P=0.32), Carbohydrates (P=0.66), Fat (P=0.79), and Proteins (P=0.84). (Table 2).

Rest among HOMA-IR, Leptin, Insulin and Glucose:

The results of Pearson correlation coefficients showed that due to one session exercise there was significant positive relation between insulin resistance index with insulin level (r=0.97, P=0.000) and glucose (r=0.91, P=0.000) and also between serum leptin with insulin level (r=0.73, P=0.02) and insulin resistant index (r=0.68, P=0.038), ($P<0.05$) (Table 3).

Table 2 *Energy and macronutrient intake of the study participants (mean ± SD)*

Variable	Friday	Sunday	Saturday	P-value*
Energy (kcal)	2024±212.7	1940±363.9	2039±200.9	0.32
Carbohydrates (%)	47.2±6.2	49.5±4.8	50.6±4.3	0.66
Fat (%)	37.4±3.4	37.9±2.3	36.9±2.9	0.79
Proteins (%)	15.4±1.8	14.6±2.4	14.5±1.3	0.84

*P<0.05

Table 3 *Pearson's correlation coefficients at rest among HOMA-IR, Leptin, Insulin and Glucose*

	HOMA-IR	Leptin	Insulin	Glucose
HOMA-IR				
Leptin	r=0.68 P=0.038			
Insulin	r=0.97 P=0.000	r=0.73 P=0.02		
Glucose	r=0.91 P=0.000	r=0.57 P=0.08	r=0.76 P=0.01	

Serum leptin levels: Serum leptin levels before and 24 h after exercise is showed in Figure 1. One session concurrent exercise led to significant reduction of serum leptin concentration (5.74±1.06 ng.ml-1 vs 5.07±0.62), (t=4.39, P=0.002).

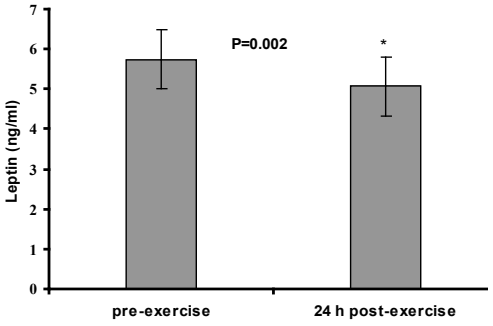

Figure 1 *Serum leptin levels before and 24 h after exercise. *P<0.05 VS. Respective baseline.*

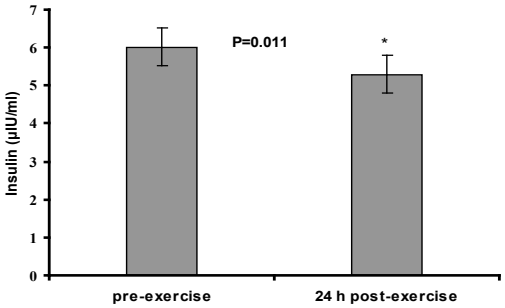

Figure 2 *Serum insulin levels before and 24 h after exercise. *P<0.05 VS. respective baseline.*

Serum insulin levels

Serum insulin levels before and 24 h after exercises are presented in Figure 2. Insulin level significantly decreased after concurrent exercise (6.02±0.54 µIu.ml-1 vs 5.30±0.43), (t=3.21, P=0.01).

HOMA-[IR] index levels

HOMA index levels before and 24 h after exercise is showed in Figure3. Insulin resistance index was decreased significantly after concurrent exercise (1.21±0.18 vs 1.03±0.13), (t=3.37, P=0.008;).

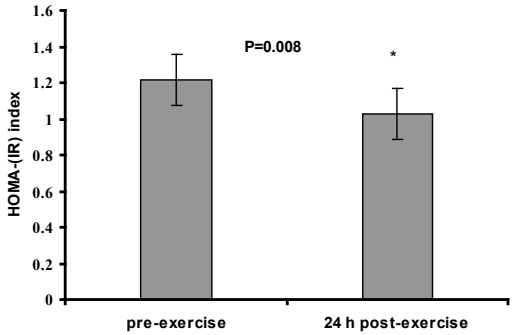

Figure 3 *HOMA-[IR] index levels before and 24 h after exercise. *P<0.05 VS. respective baseline.*

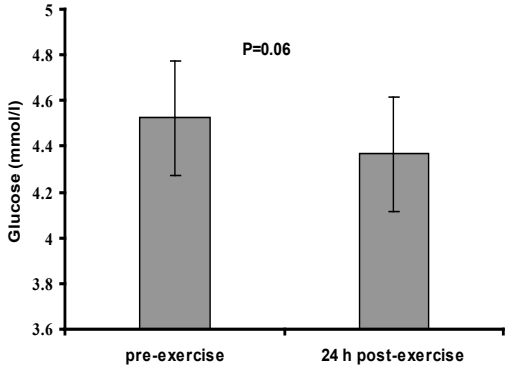

Figure 4 *Serum glucose levels before and 24 h after exercise. *P<0.05 VS. respective baseline.*

Serum glucose levels:
Serum glucose levels before and 24 h after exercises are presented in Figure4. There is no significant change after concurrent exercise (4.53±0.27 mmol.l-1 vs 4.37±0.36), (t=2.13, P=0.06).

Discussion

Our findings indicate that serum leptin, insulin concentration and insulin resistance index decreased under effect of concurrent exercise whereas there is no significant change in serum glucose levels. Significant positive relations have observed between insulin resistance index with insulin and glucose level, serum leptin, insulin level and insulin resistant index. Three times evaluating of diet regimen included energy, carbohydrates, fat, and protein indicate that there is no significant difference in food absorption.

Obesity has been known as an insulin resistant condition, and strongly is related to the hyperinsulinemia and hyperlipidemia. In humans, altering body energy requirements through dietary manipulation also affects serum leptin. Serum leptin falls dramatically with short-term fasting and increases with overfeeding [5]. Thus, it was hypothesized that an increase in energy expenditure, as a result of acute exercise, would also alter serum leptin. Results of the present study showed that, one session physical activities [aerobic-resistance] reduced serum leptin effectively. This change was associated with the reduction of insulin resistance until 24 hours after exercise in the sedentary healthy young men. The mechanism in expressing the nature of leptin secretion stimulation by insulin could be explained that, the insulin through glucose transfer protein causes transfer of glucose in to the adiposities. Therefore, glucose acts as an intracellular signal and causes stimulation of leptin secretion from adiposities [28]. On the other hand, Havel et al (1998) stated that except insulin, any other factor that causes uptake of glucose by lipid cells might stimulate leptin secretion. They found that stimulation of leptin secretion from the lipid cells in mouse has close relation with the effects of insulin in increasing the glucose uptake by lipid cells [29].

Endurance exercise training has been reported to improve whole-body insulin sensitivity in young, elderly and insulin resistant subjects. The latter is attributed to the concomitant induction of weight loss and the up regulation of skeletal muscle GLUT-4 expression. [30]. In addition to prolonged endurance training, resistance exercise interventions have also been reported to improve glucose tolerance and/or whole-body insulin sensitivity [31,32]. The latter is generally attributed to a concomitant gain in skeletal muscle tissue, which improves whole-body glucose disposal capacity [33]. However, in the development of most exercise intervention programs, the focus generally lies on the implementation of endurance exercise, because of its acute stimulating effect on whole-body insulin sensitivity. So far, the few studies that investigated the acute effects of resistance exercise on insulin sensitivity have provided contradictory findings [33, 34, 35]. On the effect of physical activity and serum leptin it should be stated that several studies analyzed the effect of one session exercise on the leptin concentration. In obese females, walking at 60-80% of heart rate maximum (HRM) for 45 minutes did not alter leptin concentrations [16] although it decreased insulin resistance. There was a decline in leptin concentrations in trained rowers immediately after and 30 min after maximal rowing exercise (30 min). [36] Thus, leptin was sensitive to short-term intense exercise when all major muscles were involved. Similarly, significantly lowered leptin concentrations were shown in sedentary middle-aged male and females after 20 min vigorous running [37]. Nevertheless, after 1 h supine recovery, leptin levels returned to basal values. These authors indicated the existence of a relationship between stressful physical exercise and plasma leptin levels in middle-aged subjects [37, 38]. Serum leptin concentrations were analyzed in healthy males in four conditions: control and three acute resistance exercise protocols [18].

Serum leptin concentrations at 30 min of recovery exhibited similar reductions from baseline after the resistance protocols that were comparable to fasting-induced reduction in the control session. Indeed, resistance exercise protocols did not result in serum leptin changes when sampled immediately or after 30 min post-exercise. These observations underlined the importance of using a resting session when evaluating the effect of exercise on leptin concentrations to control for fasting- and/or circadian rhythm-induced leptin reductions [18]. Serum leptin concentrations was analyzed after three competitive endurance races performed by 45 males who participated in one of three competitive endurance races: a half-marathon run (estimated energy expenditure "EE" ~1400 kcal), a ski-alpinism race (estimated EE ~5000 kcal), and an ultra-marathon race (estimated EE ~7000 kcal) [38]. Only prolonged endurance exercise involving high-energy expenditure (over 1,400 kcal) (e.g., alpine skiing and ultra-marathon race) induced a marked reduction in circulating serum leptin levels [38]. In the present study one session of concurrent exercise (aerobic-endurance) effectively reduced serum leptin with resistance to insulin, which agrees with some studies [36, 37, 39] and disagree with the other studies [15, 16, 17, 38]. Some of the reasons of the differences could be due to lack of nutrition control in the subjects, difference in designing of the examination like, nature of the physical exercise session, the subjects under the study, lack of partial information about the type of energy absorption during physical exercise. We tried to match the nutrition of the study subjects using the 24-hour Diet Regimen Reminding Questionnaire and with the standard nutrition on the day of exercise, and by a combinative training (aerobic-resistance) control some of these different probable items. Results of this study showed a positive relation between leptin and insulin resistance in physical activity. Leptin helps in energy metabolism regulation, growth and fertility. Hyperleptinemia, which indicates adipose tissue increase and the insulin resistances, may develop mechanism through obesity pathophysiologic relation, and resistance to insulin. In the obese mice (zucher) with leptin receptors disorder, hyperlipidemia and β-cell disorder, it was noticed that leptin injection in the β-cell isolated from this animal significantly reduced in cellular triglyceride contents [28]. In tissue of obese individuals the oxidation of the fatty acid in the right side abdominal muscle as compared with the rate of oxidation in the thin individual's muscle following leptin injection declined [40]. Therefore, it is suggested that muscle cells of obese individual shows leptin resistance. On the other hand, Kondal and associates suggested that the fatty acid oxidation inhibition and its consequence, which is muscle in cellular lipid accumulation, are due to insulin resistance through reduction of insulin signal. Since, leptin plays important role in lipid oxidation, hence, the relation between hyper leptinemia and insulin resistance could be mediated by fatty acid oxidation disorder. In addition, leptin increases the low level inflammation by synthesizing α-TNF and IL-6. Therefore, lipid toxicity and cellular inflammation by leptin resistance are the main mediators for establishing the relation between obesity and insulin resistance [40].

Conclusion

The results obtained in this study showed that, even one session concurrent exercise could have short-term effect on serum leptin concentration and insulin resistance. Therefore, it is suggested that, concurrent exercise could be a proper method to help glucose transference into the muscle cells and leptin secretion regulation, most probably, could reduce insulin resistance hence may delay diabetes type II in the sedentary young men. It is also suggested that since adaptation is the results of permanent acute responses towards a one bouts of exercise, such type of exercise may be applied in case of young sedentary male having over weight status. In this study, we faced some limitations such as controlling the daily physical activity and

more blood sampling, so another study with more controlling daily physical activity and serial blood sampling is warranted to understand the glycemic index pattern in physical exercise better.

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Contributions

Study design: MAA
Data collection, analysis: and manuscript preparation: BA, MJR,FD

Conflict of interest

"The authors declare that they have no competing interests."

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