Original Article

Acute Effect of Concurrent Exercise on Serum Leptin and Resistance Insulin Response in Sedentary Men

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Abstract

Aim: This study aimed to determine the delayed effect of one session of concurrent exercise on serum leptin and resistance insulin levels of sedentary men. **Materials and Methods:** Fifteen healthy young men with a mean age of 22.9 ± 1.7 years and a body mass index of 23.3 ± 1 kg/m^2 voluntarily participated in this experimental research. They participated in one session of concurrent exercise including 20 min of aerobic running on a treadmill at 60%–70% maximum oxygen uptake plus a resistance exercise at the intensity of 70% of 1-repetition maximum with ten repetitions per set to second round. Blood serum leptin, glucose, insulin, and insulin resistance index were assessed before, 24, 48, and 72 h after the exercise program. **Results:** The results showed that leptin level did not change statistically significantly immediately and 72 h after the exercise termination ($P > 0.05$) but did decrease statistically significantly 24 and 48 h after the exercise program ($P \leq 0.05$). The result also showed that insulin and insulin resistance levels decreased statistically significantly immediately after the exercise and lasted for up to 48 h postexercise ($P \leq 0.05$); however, it returned to its initial value after 72 h of recovery ($P > 0.05$). **Conclusions:** It was concluded that energy use in one session of concurrent exercise might reduce leptin levels up to 48 h of recovery. In addition, a decrease in insulin, a known inducer of leptin expression in adipocytes, occurs before the decrease in leptin. This change may mediate the decrease of leptin concentration.

Keywords: Exercise, insulin resistance, leptin

Introduction

Leptin is one of the hormones that is synthesized and released by adipose and plays a significant role in regulating the food intake and energy balance.[1] The high accumulation of fat mass results in increased level of leptin that in turn causes the feeling of starvation in addition to increases of basal metabolism rate. Conversely, decrease in fat stores by means of exercise and/or diet results in decrease of leptin levels.[2]

The production of leptin depends on several factors including gender, metabolic hormones, and drug factors.[3] According to reports, insulin secretion induced by drug stimulation, obese individuals, hyperleptinemia cases, and resistant to insulin result in leptin synthesis. On the contrary, it was found that the relationship between hyperleptinemia and insulin resistance is independent of body mass index (BMI) in Dutchmen.[4] These studies demonstrate that leptin and insulin are involved in a complex regulatory chain where leptin plays a significant role in glucose homeostasis. On the other hand, it has been shown that when serum leptin level is in its lowest normal range, it acts as an insulin sensitizer and may be associated with insulin resistance when it is elevated.[4]

Exercise is a potential stimulant for the secretion of several hormones.[5] Although exercise training reduces leptin levels, the effect of short-term exercises with no change on weight is unclear. While the result of some studies shows that short-term exercises have no effect on serum leptin concentrations, some others have shown that long-term exercises reduce leptin levels. These findings may indicate that energy consumption

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thresholds may be needed to reduce leptin levels in short-term exercises.

Essig *et al*. reported that leptin concentrations did not change immediately or 24 h after activity in 70% of the maximum oxygen consumption (VO_2); however, it decreased by 30% after 48 h of activity.[6] Olive and Miller also showed that leptin concentration did not change immediately following the activity (60 min in 70% VO_{2max}), but decreased 24 h later by 18% and 40% after 48 h.[7] In a research including obese women, walking 60%–80% of maximum heart rate (MHR) for 45 min did not change the concentration of leptin.[8] On the other hand, in a study by Abedi (2016), reduction of leptin concentration was observed in sedentary men immediately after 30 min at 60%–70% of heart rate reserve and resistance exercise with 70% of 1-repetition maximum (RM) of ten repetitions for three rounds.[9] In another research, Nindl *et al*. found a decrease in leptin concentration 9, 12, and 13 h following a resistance exercise.[10]

Hickey *et al*. proposed that stability of leptin concentration immediately after short-term exercises is due to delayed exercise effect;[11] in this regard, Weltman *et al*. also did not find any decrease in leptin concentration during 3.5 h of recovery following 30 min of high-intensity exercise in seven healthy young men.[12] Another investigation conducted by Torjman *et al*. also did not observe any decrease in leptin concentration after 60 min of treadmill activity performed at 50% of maximal oxygen consumption by six healthy men.[13]

The delayed effect of exercise may explain the reason of no change in leptin concentration immediately after a short-term exercise. However, the concentration of leptin would disrupt the homeostasis of fuel during recovery by energy consumption. Therefore, the purpose of this study was to determine if an acute effect of exercise response (aerobic resistive) on serum leptin of inactive men occurs immediately, 24, 48, and 72 h after concurrent exercise.

Materials and Methods

Participants

In this quasi-experimental study, 15 healthy men without a history of regular exercise, no body weight change of more than 2 kg, and nonsmoker between the ages of 18 and 25 years were selected from a population of 100 to participate in a concurrent exercise program. Inclusion criteria were age range of 18–25 years, BMI between 18.5 and 24.9 kg/m^2, systolic blood pressure below 140 mmHg or diastolic pressure below 90 mmHg, low-density lipoprotein-cholesterol <130 mg/dL, high-density lipoprotein-cholesterol at 40–60 mg/dL, and total cholesterol <200 mg/L. Blood glucose lower than 100 mg/dL and waist-to-hip ratio (H/W) <0.95, no history of regular exercise, no body weight change of more than 2 kg, no specific illness, and no smoking for at least the past 6 months. Exclusion criteria were BMI of more than 25 kg/m^2, acute illnesses that were inconsistent with exercise, any drug use during the last month, and elimination of any input conditions during the intervention. The study participants were informed of the purpose, benefits, and risks of the study and completed the consent form before the work commenced. The demographic and biochemical characteristics of the participants are presented in Table 1.

Test design

The participants attended a physiology laboratory between 8:00 and 10:00 a.m. on the testing day and fasting blood sample and other measures were collected. VO_{2max} assessment by treadmill and one-RM by weight were described and then practiced by the participants. Three days later, they returned to the laboratory (biochemistry laboratory), and blood sample was drawn.

Following the blood collection, they had breakfast containing approximately 550 calories (100 g of milk, 100 g of egg, 100 g of soup bread, and 100 g of cheese = 550 g). One hour later, they participated in the exercise program. More blood samples were collected immediately, 24, 48, and 72 h following the exercise session. All blood samples were collected in the same time interval.

Physiological assays

Body weight was measured by using a digital scale accurate to +-0.1 kg (PS6600, Befour, Saukville, WI). Height was measured using a stadiometer (Model PE-WM-60-84, Perspective Enterprises, Portage, MI). Waist circumference was measured at the belly point at the end of a normal exhalation and hip size was measured at the most prominent part.

The waist and hip circumference were measured by a flexible tape. BMI was calculated by dividing the body weight (kg) and height (m) squared. The percentage of body fat and body mass was measured by using harpenden model calipers in the three areas of chest, abdomen and thigh calculated by Polak formula[14] and Siri equation.[15]

All the measurements were taken by one person to avoid interrater error. To determine One Repetition Maximum (1RM), subjects first performed 4 to 5 repetitions with light weights (40 to 60% 1RM). After 1 min of rest, along with stretching exercises, they repeated 3–5 repetitions with 60%–

Table 1: Subject physical characteristics

Variable	Concurrent group
Age (years)	22.9±1.66
Height (cm)	173.8±10.32
Weight (kg)	70.68±8.57
BMI (kg.m^{-2})	23.32±1
VO_{2max} (ml/kg/min)	36.27±3.81
Fat (%)	18.11±1.30
Waist-to-hip ratio (cm)	0.88±0.16
1-session energy cost (kcal)	858.1±87.02
1RM bench press (kg)	68.9±11.95
1RM leg press (kg)	126.05±17.64

BMI: Body mass index, 1RM: One repetition maximum, VO_{2max}: Maximum oxygen consumption

80% of the maximum pressure. A small amount of weight was added to the weight to maintain the pressure. If the move was successfully completed, a rest interval of 3–5 min was given. The goal was to find a maximum repeat in 3–5 attempts at maximum effect.

This process continues up to the maximum point of effort. The highest load the individual lift is considered as the maximal repetition. Estimation of VO_{2max} is determined based on Bruce's maximum test.[16] Heart rate was recorded by a treadmill for every minute. The prediction equation for VO_{2max} estimating for inactive men was determined based on the following equation: $VO_{2_{max}}$ =14.76 – 1.379 (time) +0.451 (time)2 – 0.12 (time)3.

For defining intensity as a function of VO_{2max}, *Karvonen* formula was applied, and MHR at the moment of reaching the exhaustion stage was used as follows:

Heart rate = MHR – resting heart rate × exercise intensity + resting heart rate.

Necessary guidance was given to the participants to elicit their maximum effort before the test started.[16]

Diet

Data related to the participant's diet was collected by food frequency questionaire (FFQ) (first 2 days and the last day of the week). Dorosty Food Processor for Windows software (DFPW-2.1) was employed to analyze the conversion of food ingredients to grams to determine the macronutrients consumed. On the exercise days, all the participants consumed a standard diet. Basal metabolism rate was calculated based on age, gender, and weight by applying Harris Benedict equation following the adjustment made to the total energy need per day.[17]

A session of concurrent exercise

A session of concurrent exercise included general warm-up (10 min), special warm-up (3–5 min), training, and stretching and cooling (5 min). Concurrent exercise included aerobic exercise running on treadmill for 20 min at 60%–70% of VO_2, and resistance exercise was performed with 70% intensity RM with ten repetitions per exercise for two consecutive sets of 30-s rest interval between stations and 2 min rest between every round. Resistance exercise included ten cyclic station movements. The stations included: (1) leg flexion, (2) leg extension, (3) foot press, (4) scout, (5) armpit pull, (6) chest press, (7) cross-dumbbell movement, (8) the front of the arm, (9) behind the arm, (10) and sit-ups.

Estimated energy cost of aerobic exercise per session

To calculate the energy cost of every aerobic exercise session, the heart rate equation was used; therefore, immediately after the completion of training, the Polar RS800 heart rate monitor for heart rate, and caloric per minute was estimated by the equation. Multiplying this formula, the total final activity time per minute is necessary to calculate the calorie intake during the activity.

([−55.0969+ [0.6309 × heart rate per minute] + [0.483 × weight to pound] + [0.2017 × age to year])/4.184.

Estimation of energy cost per session of resistance exercise

Estimation of energy consumption in one session of resistance exercise to calculate the energy consumption of one session of resistance training is calculated using the following equation.[18] Energy consumption resistance training (kcal) = 0.086 × body weight (kg) × activity (min).

Blood collection and analysis

Immediately after the blood samples were collected, serum was isolated and kept at −70°C temperature for the analysis. The participants were instructed to avoid taking part in any exercise program for 2 days prior to the blood sampling day. Fasting blood sugar was measured by using enzymatic glucose oxidize (Pars azmoon Co., Tehran, Iran) and Hitachi 912 Autoanalyzer (Roche Diagnostics GmbH, Germany). Serum leptin level was measured via ELIZA using commerical DRG Leptin sandwich ELIZA EIA-2395 Kits (DRG Diagnostics, Marburg, Germany). Serum insulin level was measured by an insulin kit (DRG-Diagnostica, GmbH, Germany) with a sensitivity of 1.76 μUI/ml and intracontrol and extraversion coefficients of 2.19% and 4.43%, respectively, by sandwich-type ELISA Competitive. The Homeostasis Model Assessment index was calculated based on the product of fasting blood glucose concentration (mmol/L) in the fasting insulin concentration (μg/ml) divided by the factor constant of 22.5.

All the samples were tested at a specific time for every specific variable to minimize variability.[8] Hormonal densities after the activity were adjusted for plasma volume changes.

Statistical analysis

Shapiro–Wilk method confirmed the normality of the variables. Therefore, analysis of variance-repeated measures and Tukey's *post hoc* test were used to test the data with the alpha level set to 0.05 for test of significance. All statistical analyses were performed using SPSS software version 21 (IBM Corp., Armonk, NY, USA).

Results

Descriptive statistics of the concurrent training group is presented in Table 1. The result of the analysis of dietary data showed no significant difference in micronutrient dietary intake between the three times of the preactivity measurements [Table 2]. The results showed that leptin level did not change statistically significantly immediately and 72 h after the exercise termination ($P > 0.05$) but did decrease statistically significantly 24 and 48 h after the exercise program ($P \leq 0.05$). The result also showed that insulin levels decreased statistically significantly immediately after the exercise and lasted for up to 48 h postexercise ($P \leq 0.05$); however, it returned to its initial value after 72 h of recovery ($P > 0.05$) [Table 3].

Discussion

Leptin circulating levels increase after feeding for a short period and decrease during starvation. These variations indicate that leptin may be regulated by sudden rate of changes in energy intake or use. It is, therefore, possible that other mechanisms capable of altering energy use (such as exercise) will regulate the synthesis and release of leptin. The research hypothesis was based on the assumption that energy use due to a concurrent exercise session (approximately 1 h) may reduce serum leptin levels designed to test the effect of one session of the concurrent exercise on serum leptin levels in sedentary individuals up to 72 h of recovery.

Several studies examined the effects of acute exercise on leptin metabolism and reported opposite result. The majority of researches did not report any acute changes of leptin after a moderate intensity and time duration,[11,19] whereas other investigators found decrease of leptin level immediately[9,20] or delayed decrease in recovery.[6,21]

The present study showed no change of leptin levels immediately or 72 h after the concurrent exercises but found a significant decrease after 24–48 h of recovery. In general, most studies that examined the acute effect of exercise on leptin concentrations used the duration and intensity of the exercise that required considerable amount of energy (e.g., 1000 kcal). Leal-Cerro *et al*. reported a 32% lower level of leptin immediately after the activities and 16% decrease after 14 h after a marathon run.[22] Similary, the results showed a decrease in leptin after 3 hours of ergometer and marathon activity.[23]

The result of these studies shows that a severe decrease of energy is associated with an acute decrease in leptin levels of blood serum of sedentary individuals. On the other hand, pedaling cycle with an intensity of 50% of maximum oxygen uptake did not reduce the concentration of leptin or subcutaneous adipose tissue in sedentary individuals[24] or leptin levels did not change after a submaximal training.[19]

These contradictory results may be attributed to factors such as study population and intensity or duration of exercise because they influence energy cost and subsequently change serum leptin levels.

Considering the energy cost of more than 800 Kcal for the concurrent exercise in this study, serum leptin levels did not change immediately after exercise, but the delayed effect on serum leptin levels occurred 24–48 h after the exercise. These results were consistent with the results of some other studies.[6,7,25] Diet analysis suggests that the changes in leptin level during the recovery were not due to the changes in energy intake. The energy intake of the participants calculated by applying 24-h dietary recall did not show any significant change of leptin before, 24, 48, and 72 h after the activity.

Another variable that may have caused a decrease in leptin level is the decrease of adipocyte mass. Although body fat percentage is a factor that determines the primary levels of leptin, it is difficult to imagine that changes in body fat mass may reduce leptin levels over only 2 days. Therefore, in the absence of changes in energy intake and body fat percent, it is assumed that energy use of activity was the main cause of decrease in leptin concentration during the recovery.[26]

The decrease in serum leptin in this study did not occur immediately after the activity. It has been proposed that the time for leptin drop is related to the onset of energy decrease. The result of analysis of dietary reference intake showed no change in energy intake during the activity. Therefore, it is likely that energy decrease has occurred during the activity. The second reason may be related to the nature of the exercise session or the degree of fasting of the participants.

In a study conducted by Koistinen *et al*., a decrease in leptin concentrations was observed immediately after 3 h of cycling, a result contradictory with the results of the present study and some other studies.[23] However, it seems clear that the participants showed hypoglycemic response to exercise in the study of Koistinen *et al*. while the participants had hyperglycemic response to the activity in the study. The result of one study showed that when glucose was injected during fasting, serum leptin decrease was inhibited. Therefore, hyperglycemic effect in this study may have an inhibitory effect on leptin levels. On the other hand, the time to decrease leptin may be related to the integrity of regulatory factors in both energy absorption and the amount of fuel used during the energy use.[27]

Table 2: Dietary intake by participants using a 24-h dietary recall questionnaire

Variable	Saturday	Sunday	Friday	*P*
Energy intake (kcal)	2850±155.9	2540±355.9	2750±245.7	0.34
Carbohydrate (%)	50.3±5.3	49.5±3.5	48.1±5.2	0.63
Fat (%)	37.4±3.5	35.2±3.3	36.2±3.6	0.72
Protein (%)	13.5±2.1	15.4±2.48	15.8±2.4	0.73

Table 3: Insulin, glucose, insulin resistance, and leptin level following one session of concurrent training in different time intervals (mean±standard deviation)

Variable	Concurrent training (before)	Immediately	24 h	48 h	72 h	*P*
Insulin (μIU/ml)	6.02±0.54	5.3±0.44*	5.04±0.24*	5.5±0.6+4*	5.9±0.44	0.03
Glucose (mmol/l)	4.53±0.27	4.43±0.77	4.6±0.47	4.33±0.57	4.73±0.47	0.28
Insulin resistance	1.21±0.18	1.04±0.08*	1.03±0.24*	1.06±0.068*	1.24±0.14	0.002
Leptin (ng/ml)	5.75±1.06	5.26±0.82	4.76±0.62*	5.06±0.42*	5.55±0.96	0.001

*Significance level relative to baseline

In the present study, insulin and insulin resistance levels decreased immediately after the exercise; this decrease continued up to 48 h after the exercise, but 72 h after the recovery, the level of insulin returned to its original state.

The effective factors of this decrease can be mentioned include muscle lipid content in relation to physical inactivity, adenosine monophosphate kinase activity, reduced release and increased clearance of free fatty acids, increased mRNA expression of glucose transporter protein (Glut-4).[28] Few studies that have investigated the acute effects on insulin resistance have had conflicting results.[29,30] For example, Fluckey *et al*. reported no change in glucose concentration after glucose ingestion 18 h after resistance exercise, but low insulin response was observed.[31] In the study by Fenicchia *et al*., low glucose response was reported 12–24 h after resistance training in women with Type 2 diabetes without any difference in insulin levels.[32] In a study by Chapman *et al*. and Little *et al*., no change in insulin and glucose response after a session of resistance exercise was reported in healthy nonobese individuals with normal glycemic control.[33,34] In this study, a decrease in insulin response was reported 48 h after an exercise session. The difference in results may be due to the use of the insulin resistance measurement method and the delayed insulin response to return to baseline from an activity session. The mechanism of delayed insulin response to a physical activity session has not been well characterized. Changes in insulin resistance through exercise have been linked to skeletal muscle glycogen depletion or triglyceride stores. Muscle glycogen depletion results in increased glucose uptake after exercise to facilitate glycogen re-establishment. Most cellular events under the circumstances of this phenomenon led to an increase in the translocation of the 4-Glut isoform to the glucose transporter from its intracellular storage areas. Other factors that may explain the antagonism of insulin resistance include the duration of the training session, energy absorption, daily physical activity, as well as the population under study. The endurance training program used in the studies by Fluckey *et al*. and Chapman *et al*. may not be sufficiently effective to improve overall insulin resistance.[31,33] In addition, some studies did not standardize feed intake or physical activity. In the present study, both food intake and physical activity were standardized to reduce the effects of these two factors on insulin resistance.

This temporary decrease in insulin levels occurred earlier than the significant decrease in leptin concentration. This may be a symptom for a temporary decrease in the synthesis or release of leptin from fatty cells, which ultimately leads to lower levels of leptin.[6] However, the subject of acute effects of insulin on leptin is contradictory. Some studies have shown that insulin effects on leptin only occur after 6–24 h of hyperinsulinemia that may occur with changes in fat mass.[27] Therefore, further research to examine the relationship between insulin and leptin levels after exercise seems necessary.

Conclusions

Therefore, it seems that energy consumption can reduce leptin up to 48 h after a session of combined exercise. In addition, the decrease in insulin, the known stimulant of the expression of leptin in adipose tissue, occurs prior to the reduction of leptin, which may be considered as a mediator of leptin concentrations.

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Conflicts of interest

There are no conflicts of interest.

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