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Effects of acute exercise on serum Leptin and some of its related hormones in active and non-active (men)

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ABSTRACT

Leptin, is a hormone that synthesized by fat tissue, has an important role to regulate energy balance and metabolism and thus body weight. The influence of acute exercise and chronic exercise training on circulating leptin and its relationship with hormonal and metabolic changes that induce energy balance had been noted by researchers. The purpose of this study was comparison of serum leptin(L) , cortisol(C) and testosterone(T) responses to one bout acute aerobic exercise in active and non-active men. In this semi-empirical study, 18 men in two groups: nine active (21 /88 ±1/36yr with 6/11± 1/45yr active training for the preceding) and nine non- active (22/2±1/64yr with non-specific training for the preceding) were not taking any medication, and were free of cardiac, respiratory, renal, or metabolic diseases, volunteered. Blood samples were collected before and immediately after 12 minutes intensive running exercise (cooper aerobic test).The concentrations of L, C and T serum was determined by Chemiluminescence laboratory method by Elisa kits. For statistical analysis of data we used from T-test, ANOVA and pearson correlation methods by spss software version 16. The results were showed that immediately after exercise, leptin levels (active group) and testosterone (in both groups) significantly increased, however, not founded significant differences inleptin, cortisol and testosterone between two groups before and after exercise. Also at baseline and after immediately exercise the relationship between serum L with PBF, Vo2max, BMI, WHR, serum concentrations of C and T in each groups were not significant (P<0/05). One bout intensive aerobic exercise such as12 minutes running not create real changes in concentration of leptin serum in active and non-active men.

Key words: leptin, cortisol, testosterone, active and non- active men

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INTRODUCTION

Leptin was discovered in 1994 following the isolation of the *ob* gene [1]. Leptin, a protein with a helical structure similar to cytokines and a relative mass of 16 kDa with 146 amino acids [2], assists in the regulation of body weight and energy homeostasis [3,4]. Adipose tissue is the major source of leptin expression; however, other sites have been identified, including skeletal muscle, mammary epithelium, the placenta, and the brain [2]. It is thought that a major role of leptin is to relay information to signal transducing receptors in the hypothalamus concerning the status of energy stores [5,6] and thus aid in reduced feeding [3,4]. The leptin receptor (with long and short isoforms) is a member of the cytokine family of receptors and is expressed in a variety of tissues including the hypothalamic nuclei [7,8]. Neurons in the arcuate, ventromedial, and dorsomedial hypothalamic nuclei that are sensitive to leptin express neuropeptides/neurotransmitters that are associated with central regulation of energy balance [5, 2].

Leptin is regulated by the status of fat storage, with larger adipocytes containing more leptin than smaller ones in the same individual [9]. Nutrition-related control of leptin has been suggested to be partially regulated by insulin. It has been demonstrated that leptin expression occurs after elevation of insulin in response to feeding [10], and a decline in leptin levels follows reduction in insulin during fasting [11]. Some early studies reported no acute effect of eating

on leptin concentrations, but later studies that controlled for fasting demonstrated that meals and insulin acutely affect leptin concentrations [12]. Other hormones associated with the regulation of leptin that are altered by exercise include glucocorticoids, growth hormone, and catecholamines [13, 14].

Growth hormone and cortisol have been shown to promote leptin production [15, 16], whereas catecholamines and testosterone inhibit leptin production [17].

Exercise is a potent stimulus for secretion of many hormones [18, 19] and exercise mediated negative energy balance may contribute to the regulation of plasma leptin concentrations [18,20]. long-term (≥60 min) exercise, a reduction in leptin concentrations reported from 1 to 3 hr of running or cycling has been attributed to diurnal reduction in circulating leptin, independent of exercise [21, 22,23]. Kraemer RR (2003) suggested that the decline may be associated with elevated production of non-esterfied fatty acids during exercise that to be inversely correlated with leptin levels [24]. On the other hand, recent studies have shown that endurance exercise with marked negative energy balance – for example, marathon swimming, marathon running may cause an immediate or delayed decrease in leptin concentration [18, 25]. In contrast, short term (<30 minutes) exercise studies suggest that leptin concentration is not acutely affected by exercise regardless of intensity [18, 26], as the exercise mediated energy expenditure may not reach the critical level needed for plasma leptin concentration to be reduced by acute exercise [20,27]. Most reports of reductions in serum leptin may be attributed to circadian rhythms or hemoconcentration or elevation of production non-esterfied fatty acids during exercise [28].

On the other hand a few studies has been reported significant increases in leptin responses to a graded exercise test to exhaustion and moderate exercise after immediately of exercise [12, 29]. The increases in these studies may have been due to hemoconcentration, which would have presented higher concentrations of leptin to the leptin receptors [28].

Thus, due to insufficient studies and controversial results of previous studies regarding the effects of acute exercise on leptin, in this study, the researcher and colleagues are seeking to answer the question that what's leptin and cortisol and testosterone responses to a single bout of exhaustive aerobic exercise in active and non-active, and dose fitness level has an effect on the possible changes of leptin concentration?

MATERIALS AND METHODS

In this semi-empirical study, 18 men in two groups: nine active (21 /88 ±1/36yr with 6/11± 1/45yr active training for the preceding) and nine non- active (22/2±1/64yr with non-specific training for the preceding) were not taking any medication, and were free of cardiac, respiratory, renal, or metabolic diseases, volunteered. Blood samples were collected before and immediately after 12 minutes intensive running exercise (cooper aerobic test). Body weight was measured with a digital scale (sensitivity of 0.1 kg) and height and WHR was measured to the nearest millimeter using JENIX (DS-102, Korea). BMI was calculated as weight in kilograms divided by the square of height in meters. Body fat percentage was calculated by Lipid Caliper with sensitivity of 0.2mm (YAGAMI, Japan) using Jackson and Pollock 3-site skinfold equations (30). Also VO_2 max was estimated by one mile Rockport Fitness Walking Test (31).

For determine concentrations of leptin, cortisol and testosterone serum, 5 cc blood samples was obtained from an elbow vein of subjects, before and immediately after performance of 12 minute intensive aerobic exercise. Blood samples were collected in test tubes and anticoagulated with EDTA. Plasma was separated by centrifuging for 15 minutes at 1000 ×g at 2-8°C within 30 minutes of collection and divided into three aliquots. The aliquots were frozen and stored at −80 °C for subsequent analyses (within 2–3 weeks). leptin and cortisol was determined by chemiluminescence method in laboratory by using each special kits (leptin by Diagnostic Biochem kit manufactured by Hungary and cortisol and testosterone also Immonotech kits (IM1119 and IM1841) manufactured by Czech Republic) were measured.

Exercise program

Exercise program was include12 minute running (cooper aerobic test) with 85-80% of HR_{max} in active and 75-70% of HR_{max} in non-active subjects. Heart rate was controlled by polar stethoscope manufactured by Sweden. For elimination of unpredicted factors such as quantity and composition of nutrient, all of subjects after a fasting night, had eaten a meal with similar calorie about 1 hr before exercise (13, 16).

Statistical analysis

After confirming normal distribution of data with the Kolmogorov-Smirnov (k-s) test, to compare the difference of variables in pre and post-exercise, we used from T-test and ANOVA statistical methods, and to determine of relation

between leptin with other variables, pearson correlation method were used. $p<0.05$ was used as the criterion of statistical significance. Data analyzed by $spss_{16}$ software.

RESULTS

The non-parried T-test analyzes of data at baseline were showed that there are no significant differences between two groups in all variables (except Vo_{2max}) (Table 1).

Table1: Physical, physiological and hormonal variables of subjects at baseline

Variables	group		p-value
	Non-active	Active	
Age(yr)	22.66±1.65	23.11±1.26	0.532
Height(cm)	177.8±2.31	177.66±6.68	0.969
Weight(kg)	72.33±8.52	68.43±7.57	0.32
BF(%)	17.41±4.109	13.91±5.15	0.131
BMI (kg/m^2)	22.186±2.01	21.63±2.53	0.615
VO_2 max(ml/kg/min)	40.17±1.22	49.01±1.05	0.01*
WHR(cm)	97.5±0.07	0.93±0.06	0.203
Leptin(ng/ml)	4.833±1.94	3.478±0.48	0.059
Cortisol(ng/ml)	4.968±2.03	4.962±3.42	0.996
Testosterone(ng/ml)	3.74±1.31	4.7±0.96	0.097

Values expressed by mean and standard deviation (X±SD); p < 0.05

Also, the results of parried T-test were showed, in active group after exercise, the mean concentration of leptin, cortisol and testosterone were increased, that this increase was significant in relation to leptin and testosterone (p=0/01 and p=0/044). In non- active group, after exercise, mean concentration of leptin not significantly decreased and serum cortisol and testosterone levels had a non-significant increase. (Table 2)

Table 2: The paired T-Test results of subjects in two groups

Group	Variables	Pre- Exercise	Post- Exercise	p-value
Non-active	Leptin(ng/ml)	4.833±1.94	4.32±1.53	0.551
	Cortisol(ng/ml)	4.968±2.03	5.84.2±2.41	0.291
	Testosterone(ng/ml)	3.74±1.31	4.4±1.48	0.081
Active	Leptin(ng/ml)	3.478±0.48	3.84±0/5	0.01*
	Cortisol(ng/ml)	4.962±3.42	6.43±2.92	0.384
	Testosterone(ng/ml)	4.7±0.96	5.16±0.73	0.044*

Values expressed by mean and standard deviation (X±SD); p < 0.05

The results of analysis of variance (ANOVA) showed a mean difference of leptin, cortisol and testosterone levels after exercise in both active and inactive groups was not significant. (Table 3)

Table 3: Results of analysis of variance (ANOVA) after exercise in both groups

Variables	Group	Pre- Exercise	Post- Exercise	F	p-value
Leptin(ng/ml)	Active	4.833±1.94	4.32±1.53	0.753	0.398
	Non-active	4.968±2.03	3.84±0/5		
Cortisol(ng/ml)	Active	3.74±1.31	6.43±2.92	0.217	0.604
	Non-active	3.478±0.48	5.84.2±2.41		
Testosterone(ng/ml)	Active	4.962±3.42	5.16±0.73	3.187	0.093
	Non-active	4.7±0.96	4.4±1.48		

Values expressed by mean and standard deviation (X±SD); p < 0.05

Also the results were showed that except of pre-exercise body fat (%) of non- active subjects (p=0.035), there aren't significant relations between body weight, BF%, BMI, WHR, Vo2max, cortisol and testosterone serum with leptin serum levels in before and after exercise in two groups (Table 4).

DISCUSSION

The present study was showed that at baseline, there are no significant differences between serum levels of leptin, cortisol and testosterone in two groups.

The results most of previous studies has shown that leptin is regulated by the status of fat storage, with larger adipocytes containing more leptin than smaller ones in the same individual. These studies expressed that, there are a

significant relationship between of body fat with leptin, also a relationship between of body fat with exercise training [28, 32, 33, 34].

Table 4: The relationship between leptin with other variables ($p < 0.05$)

Variables	Group	Active Leptin(ng/ml)		Non-active Leptin(ng/ml)	
		r	P	r	P
Weight(kg)	Pre- Exercise	- 0.1	0.797	- 0.247	0.522
	Post- Exercise	0.38	0.923	0.729	0.026*
BF(%)	Pre- Exercise	0.09	0.981	- 0.246	0.524
	Post- Exercise	0.017	0.965	0.554	0.122
BMI (kg/m²)	Pre- Exercise	0.101	0.797	0.466	0.206
	Post- Exercise	-0.68	0.861	0.484	0.187
WHR(cm)	Pre- Exercise	-0.46	0.97	0.209	0.59
	Post- Exercise	-0.04	0.918	0.492	0.179
VO_2 max(ml/kg/min)	Pre- Exercise	0.206	0.594	-0.68	0.861
	Post- Exercise	0.113	0.772	0.153	0.695
Cortisol(ng/ml)	Pre- Exercise	-0.387	0.304	-0.656	0.055
	Post- Exercise	-0.328	0.388	0.337	0.375
Testosterone(ng/ml)	Pre- Exercise	-0.345	0.346	-0.527	0.145
	Post- Exercise	0.13	0.74	0.375	0.32

In this research the results were showed level of leptin in active subjects before exercise were lower than non-actives and also the results was showed in non-active group, there was a significant relationship between body weight and serum leptin (p=0.026).

On the other hand the effect of long term aerobic exercise on leptin to be proved [36, 35]. Thus accordance of these findings, in this study the lower levels of leptin at baseline in active group, probably be related to preceding physical activity (6.11±1.45 year). By attention this subject that in this study, the relationship between body fat with leptin was waked, thus probably the other factors such as hormonal or metabolically changes were effected on leptin in active group [33, 37, 38].

Also, the results of parried T-test were showed, in active group after exercise, the mean concentration of leptin, cortisol and testosterone were increased, that this increase was significant in relation to leptin and testosterone (p=0/01 and p=0/044).

This results consistent with finding of Fisher JS & et al (2001), Kraemer RR, et al (2001) and contrast with finding of Weltman A & et al (2000), Jurimae J, et al(2005), Essig DA & et al (2000) and Duclos (1999) [18,28,26, 39, 40, 41]. Increasing of leptin in active group may have been due to hemoconcentration, which would have presented higher concentrations of leptin to leptin receptors [28, 42].

On the other hand, the significant deference in leptin probably related to increases of cortisol and particularly testosterone and other important hormones such as insulin, catecholamine's and other sexual hormones (estrogen, …) that in this study was not measured [28]. According to previous studies on animals, insulin and sexual hormones has important role in expression of *ob gene*. Also studies has showed that exercise was increased insulin sensitiveness [33,43]. Insulin stimulate leptin secretion, whereas, β- adrenergic agonists limit the leptin secretion [44]. Exercise caused redaction of insulin secretion and increased of lipid metabolism in muscle, in conclusion, share of glucose in energy production was decreased[45].

In non-active group, results indicated that level of leptin after exercise reduced. This reduction was not significant. This results consistent with finding of Weltman A, et al (2000), Jurimae J, et al(2005), Essig DA & et al (2000) and Duglos (1999) [18,26,39, 40, 41]. This reduction may have been due to circadian rhythms or elevation of non-esterfied fatty acids or decline of insulin during exercise [28, 42].

The results of this study were showed that changes in levels of the leptin, cortisol and testosterone hormones in pre and post exercise, between the two groups was not significant, these findings was in line with the findings of Kraemer & et al (2003) and Bouassida & et al (2004) (46,47). So it seems that acute exercise cannot have a significant effect on the concentration of hormones that investigated in this study, especially leptin and cortisol.

CONCLUSION

One bout intensive aerobic exercise such as 12 minutes running not create real changes in concentration of leptin serum and some leptin affected hormones in active and non-active men. Moreover, leptin levels may be more dependent on body condition. Regular exercise can be caused some adjustment that may be leading to a reduction in leptin secretion.

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