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The Response of Plasma Leptin and Some Selected Hormones to One Session of Progressive Running in Non-athlete Males

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Abstract

Introduction: Leptin, a protein with a cytokine-like helical structure and molecular weight of 16 KDa, regulates the body weight and homeostasis of the energy in the body. The aim of this study was to investigate the response of plasma leptin and some of selected hormones to one session of progressive running in non-athlete males.

Material and Methods: Twenty three male (age 19.58 ± 2.12 yr, BMI 21.63 ± 2.7 kg/m^2 and weight 64.86 ± 6.46 kg) performed a session of exhausting session running at 60-85% maximal heart rate. Blood samples were taken before and immediately after exhausting exercise to measure of plasma leptin, insulin, cortisol, lactate and glucose.

Results: results of this study indicated that one progressive running session decreased plasma leptin but this decrease was not significant (P=0/42). Also there was a significant decrease in plasma insulin (P=0/009) and glucose (P=0/03) but no significant decrease was observed in cortisol levels. But significant increase was observed in lactate concentration (P=0/0001).

Discussion and Conclusion: We have concluded that, lack of a significant decrease in leptin concentrations were due to low energy expenditure during of acute exercise. The result of this study and other research suggest that for the leptin concentration to decrease, exercise must be at least as long as than 60 minutes with an energy expenditure of about 800 kcal in non- athletes.

Key Word: Progressive running, Leptin, insulin, Cortisol, Non-athletes

Introduction

Adipose tissue is not only a passive energy store but also an active endocrine organ that produces biologically active substances termed ‘‘adipocytokines’’. These adipocytokines include several novel and highly active molecules abundantly released by adipocytes, as leptin, adiponectin, resistin, apelin or visfatin, as well as some more classical molecules, like tumour necrosis factor-alpha (TNF-a) and interleukin 6 (IL-6) [1].

Leptin is one of the first known adipocytokins [2, 3]. This hormone is a 167 amino acid protein [4] and is involved in the regulation of metabolic processes furthermore this hormone indicates levels of stored body fat. Leptin system is very complex and effective in a wide range of physiological processes, where it is a determining factor in balancing basis fat content [2,5]. Leptin concentration had a high positive correlation with BMI and body fat percentage and parallel increases to adipose stores [6, 7].

Leptin is a hormone that participates in cell metabolism and its dysfunction can be compensated by regular and light physical activity that increases hypothalamic cell receptor sensitivity [8]. Physiological stress resulting from physical activity is one of the potential regulators of leptin secretion from adipose tissue [9]. Possible mechanism that decreases the amount of leptin is the sympathetic nervous system activity under physical exercise or the circulation epinephrine, concurrent with reduction in cell adipose content and increased receptor sensitivity to leptin [10, 11]. Exercise

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training affects carbohydrate and fat metabolism. On the other hand, leptin is also associated with health status, glucose and free fatty acids metabolism, so survey the effects of exercise on leptin levels has been considered [12].

The effects of exercise on leptin concentrations have recently been controversial. Some researchers have reported that exercise may reduce leptin [13, 16], which depends on caloric consumption and the duration of activity. But some other researchers found no change in leptin [17-20] and a new study, has reported increase in leptin by physical activity in rats [21]. Vatansever (2011) investigated the effect 120 minutes of running at 50-70% of maximum oxygen consumption in 10 healthy males. Results showed that exercise had no significant effect on serum leptin levels [20]. Sari et al (2007) showed that serum leptin concentrations did not change in 23 obese women after 45 minutes walk at 60-80% of the maximum heart rate [17]. In contrast, Zaccaria et al (2002), studied the effect of three race endurance (Half-marathon run, ski-alpinism race and Ultramarathon race, with the energy cost of 1400, 5000 and 7000 kcal) respectively on serum leptin concentrations and found that only long activity of the ski-alpinism race and Ultramarathon race, decreased leptin concentrations [14].

Recent studies have shown that leptin productions in vivo and in vitro are controlled by several hormones and chemical substances. These studies have showed that leptin production is controled by up regulation of insulin, glucocorticoids and NPY and down regulation of cAMP or derivatives thiazolidine [22, 23]. Insulin is a key regulator ob gene and leptin secretion [24]. The rapid decrease in serum leptin on fasting and no change in insulin and glucose levels, indicates the important role of insulin in regulating leptin secretion [25]. On the other hand, cortisol also stimulates leptin gene expression and enhances its secretion from fat cells [26-28]. Previous study has shown the stimulation of leptin production by cortisol in vivo and in vitro. Also the stimulatory effect of glucocorticoids on the synthesis and secretion of leptin in separate adipocyte is well observed [29]. Infusion of glucocorticoids increases leptin secretion in human, however, the acute stimulation of corticotropic tract dos not always significantly change leptin levels [30]. Accordingly, it is suggested that chronical increase in cortisol secretion not only may cuase hyprleptinemia implantation, but also may lead to insulin resistance in obese individuals [31].

However, more research is needed that how to determine the impact of hormone and metabolic materials on leptin, and to clarify the reason that leptin concentrations are decreased in some cases but not in other conditions [32]. Therefore the purpose of this study was to investigate the response of plasma leptin and some selected hormones to one progressive running session in non-athlete males.

Material and Methods

Participants

23 male students (age 19.58 ± 2.12 yr, BMI 21.63 ± 2.7 kg/m^2 and weight 64.86 ± 6.46 kg) volunteered to participate in this study (Table 1). All the participants assigned in experimental group and completed an informed consent form before the study. The experimental protocol was approved by the local university ethics committee and all participants were informed of the risks and purposes of the study before obtaining their written consents. They were not taking any medication at the time of the study. The participants were non-smokers, had a sedentary life style and were not on a diet to lose weight. At least 3 months prior to the study.

Table 1: Anthropometric and physiological characteristics of study participants (mean ± SD)

Variables	mean ± SD
Body weight (kg)	64.86 ± 6.46
BMI (kg/m^2)	21.63 ± 2.7
VO_{2max} (ml.kg^{-1}.min^{-1})	39.94 ± 6.68

Experimental design

According to research plan schedule, one week before the exhaustion exercise, anthropometric and physiological factors including weight, body mass index and maximal oxygen consumption were measured. To determine maximal oxygen consumption (VO_2 max), all participants performed the 12-min Cooper's running and walk test. Participants were also asked to refrain from vigorous exercise 72 hours prior to the main trials. The diet of subjects (breakfast and lunch) was similar(controlled)prior the exercise.

Main trial (Exhaustion exercise session)

Exhaustion running sessions was done in three minute periods and 30 to 60 s active rests between periods. Participants started to run with 60% of maximum heart rate. The intensity of activity was increased by 5% in each period to reach 85% of maximum heart rate. In this intensity, the participants continued the activity until exhaustion. Perceived exertion was recorded using Borg scale. Heart rate was measured continuously using a HR monitor (Polar F1 – Polar Electro Oy, Finland).

Blood sampling

Blood samples (8 ml) were collected at 2.5 hours after lunch, before and immediately after exercise. 2 ml of blood were collected in micropipettes to measure hemoglobin concentration, and determine hematocrit. Hemoglobin and hematocrit values were used to assess plasma volume changes. Blood samples were centrifuged for 10 minutes at 3500 rpm. Blood samples were stored at -80 °C before being analysed for leptin, lactate, insulin, glucose and cortisol .

Blood biochemistry

Plasma leptin concentrations were determined by ELISA (Mercoda, Uppsala Sweden). The within-batch Intraassay CV was 4.7%. Plasma cortisol concentrations were determined by ELISA (Diagnostics Biochem Canada Inc., Ontario, Canada). The within-batch CV was 5.9%. Plasma lactate concentrations were determined by enzymatic, colorimetric methods (Randox, Antrim, United Kingdom). The within-batch CV was 2.2%. Plasma insulin concentrations were determined by ELISA (Mercoda, Uppsala Sweden). The within-batch Intraassay CV was 6.1%. Plasma glucose concentrations were determined by enzymatic, colorimetric methods (Pars Azmon, tehran). The within-batch Intraassay CV was 3%.

Statistical analysis

Data were analysed using the Statistical Package for Social Sciences (SPSS) for Windows software ver. 18.0 (SPSS, Chicago, IL, USA). For statistical analysis, paired – samples T test was used in two stages, before and after exhaustion exercise. Statistical significance was accepted at the 5% level. Results are given as means ± SD.

Results

Results showed that progressive running led to a reduction in plasma leptin concentrations, but this reduction was not significant (P =0/42) (Figure 1). It also resulted in a significant decrease in insulin (P =0/009), plasma glucose (P =0/03) and a significant increase in plasma lactate (P =0/0001). But it did not change plasma cortisol levels (P =0/8) (Table 2).

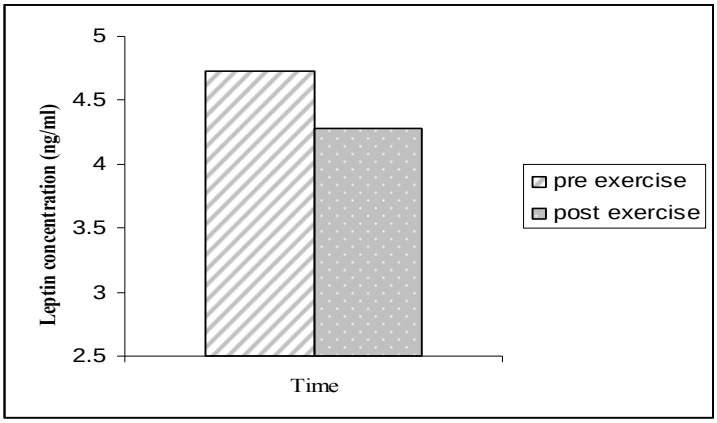

Figure1. Plasma leptin concentration (ng/ml) at the beginning and end of the progressive running ($p<0.05$**)**

Table 2: Mean and SD of plasma Leptin, lactate, Insulin, glucose and cortisol on pre and post exercise

Variables	Pre exercise	Post exercise
leptin (ng/ml)	4.73± 3.92	4.28 ± 3.09
insulin (mu/L)	11.47 ± 6.21	8.13 ± 3.96*
glucose (mg/dl)	91.15 ± 11.83	85± 8.89*
cortisol (µg/dl)	16.44 ± 4.47	16.12 ± 5.51
lactate (mg/dl)	20.45 ± 5.97	51.30± 11.56*

(*)Significant at $p< 0.05$

Discussion and Conclusion

Investigation of leptin response to exercise has been investigated in many studies. Leptin is a hormone that plays a role in maintaining hemostasis and regulating energy balance. Exercise creates a negative energy balance through increasing energy expenditure [18]. Previous studies on different reports have shown that, leptin response to acute exercise depends on the exercise duration. Many studies have demonstrated that short term exercise decreases or does not change leptin concentrations [12].

Our results showed that progressive running led to a reduction in plasma leptin concentrations, but this reduction was not significant. In consistent with our findings, Sari (2007), Zolandz (2005), Hilton (2000), Vatansever (2011) have also reported no significant change in plasma leptin levels after exercise [17-20]. Bouassida et al (2004) showed that acute exercise (45 s) in 17 trained participants did not change plasma leptin after exercise [33]. As Bouassida et al (2004) have investigated acute exercise, it is difficult to compare their results with our findings.. Sari et al (2007) showed that serum leptin concentrations did not change in 23 obese women after 45 minutes walking at 60-80% maximum heart rate [17]. Vatansever (2011) investigated the effect of 120 minutes of running at 50-70% of VO_{2max} in 10 healthy men. The results showed that exercise training had no significant effect on serum leptin levels [20]. Unlike the present results, Jurimae (2005), Zaccaria (2002), Keller (2005) and Pop (2010), observed significant decrease in leptin levels after exercise [13-16]. Zaccaria et al (2002) investigated the effects of three long term exercise on serum leptin concentrations and found that leptin concentrations decreased only after long term exercise [14]. One probable reason for the decrease in the leptin concentrations in the present study could be a lower energy cost in this study. . As Bouassida (2010) stated exercise does not affect leptin concentrations when energy expenditure is less than 800 kcal [12].

Insulin concentrations were also measured in this study. The results showed that progressive running, led to significant decrease in insulin levels. Insulin is a hormone that increases as the blood glucose levels raise after meals [34]. It has been shown that insulin levels decrease after exercise [19]. Increase in circulatory catecholamine inhibits the release of insulin [35]. In consistent with the results of this study, Martins (2007) has also reported a decrease in insulin levels, after acute exercise [36].

The results of the present study showed a significant decrease in glucose levels. Progressive exercise leading to exhaustion, with its high energy costs, has probably decreased glucose concentrations, and resulted in the consumption of the body's energy sources as the consequence.

Plasma cortisol concentrations were also measured and did not significantly change as a response to the progressive running. The observed relationship between leptin and cortisol in previous studies [37, 38], was not observed in this study. It is likely that the decrease in leptin levels was not enough to inhibit the secretion of cortisol.

Furthermore, exercise caused a significant increase in plasma lactate, which is consistent with the findings of many studies that have investigated high intensity exercise leading to exhaustion [39,40].

The design of the present study was quasi-experimental and there was no control group. All the participants performed progressive running. Given this, if we are able to control the progressive effects of running, we will be able to compare their results with ours in order to reach better conclusions. It is possible that other factors, besides progressive running, have also affected plasma leptin concentrations, but as the present study lacks

a control group, we cannot discuss this issue clearly.

Conclusion

Rising the energy costs of the exercise through increasing the exercise duration, can be one of the factors affecting negative energy balance, leading to positive changes in leptin concentrations. One of the reasons of observing no significant changes in leptin concentration could be low energy costs due to short-term exercise. According to the findings of this study and other similar research in this area [12], exercise longer than 60 minutes with energy expenditure higher than 800 kcal can be recommended for the reduction of leptin concentration in non-athletes.

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