Original Article

Zahedan Journal of Research in Medical Sciences

Journal homepage: www.zjrms.ir

Effect of a Single Session of Aerobic and Circuit-Resistance Exercise on Plasma Ghrelin and Agouti-Related Peptide (AgRP) Levels in Well-Trained Females

Mahdiyeh Ebrahimnia, *[1] Amir Rashidlamir,[1] Mehdi Seyed-Alhosseini [2]

1. Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, Ferdowsi University, Mashhad, Iran
2. MSc of Exercise Physiology, Faculty of Physical Education and Sport Sciences, Tehran University, Tehran, Iran

Article information

Article history:
Received: 24 Dec 2012
Accepted: 28 Jan 2013
Available online: 20 May 2013
ZJRMS 2014; 16 (6): 25-29

Keywords:
AGRP
Ghrelin
Exercise

*Corresponding author at:
Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, Ferdowsi University, Mashhad, Iran.
E-mail:
spnt.me@gmail.com

Abstract

Background: Ghrelin and agouti-related protein (AgRP) are orexigenic peptides secreted from stomach mucosa and the arcuate nucleus of the hypothalamus, respectively. Both peptides affect feeding behavior and play a role in energy balance, glucose homeostasis, and adiposity. The purpose of the present study was to investigate the effect of two types of exercises, aerobic and single circuit resistance exercise on plasma ghrelin and AgRP levels.

Materials and Methods: The current study is semi-experimental. Twenty two athletes young females randomly selected and assigned into two experimental groups and one control group. Blood samples were collected before and after the enforced protocol. Plasma AgRP and ghrelin levels were measured using the enzyme-linked immunosorbent assay (ELISA) method.

Results: Results showed a significant increase in plasma AgRP (p=0.001) and ghrelin concentration in control and aerobic (p=0.001) and circulate-resistance (p=0.001) groups.

Conclusion: In healthy females a single session of aerobic and circuit-resistance exercise could be associated with the increase of these peptides in response to the negative balance of energy produced by exercising for increasing appetite to compensate energy resources body subjects.

Introduction

Sedentary life style and low physical activity lead to overweight and obesity [1]. Obesity has long been viewed as an imbalance in the energy equation [2] and represents a state of excess storage of body fat [3]. Rising prevalence of obesity is evident worldwide [4], and it is known to be a main cause of many diseases such as hypertension, atherosclerosis, type II diabetes, certain types of cancer and respiratory and gastrointestinal disorders; a strong relationship between obesity and these diseases has been reported [5, 6]. Body weight is regulated by a balance between food intake and energy expenditure, and it is reported that exercise is an effective method of increasing the latter [7]. The arcuate nucleus (ARC) is a key hypothalamic nucleus in appetite regulation and is an important integration site linked to the control of food intake and energy homeostasis. The hypothalamus and its neural circuits play a critical role in the regulation of feeding behavior and body weight [8-10]. Several different neuronal populations exist in the hypothalamic arcuate nucleus and regulate energy homeostasis [8-11]. Among them, agouti-related peptide (AgRP) neurons are involved in feeding and weight gain [8]. AgRP is a 132-amino acid peptide [12], released by fasting, undernutrition [11] and caloric restriction [13], and is a neuropeptide expressed mainly by the arcuate nucleus of the hypothalamus. Probably, the most important role of AgRP is its role in energy balance and homeostasis. Therefore, these researchers have suggested that AgRP neuropeptide may contribute to the control of food intake and obesity [12-14]. AgRP induces food intake and positive energy balance via its orexigenic effects [8-10]. Ghrelin is a 28-amino acid appetite-stimulating peptide that is released primarily by the stomach and the gastrointestinal tract [14]. Ghrelin levels tend to rise before meals and decrease after meals, suggesting that ghrelin prompts meal initiation [15-17]. Furthermore, ghrelin modulates peripheral metabolism, weight gain, and adiposity [18, 19]. The orexigenic effects of ghrelin are exerted through the growth hormone secretagogue receptor 1a (GHS-R1a) [20], which is expressed in agoutirelated peptide/neuropeptide Y (AgRP/NPY) neurons in the arcuate nucleus of the hypothalamus (ARC) [21]. Exercise and reducing energy intake constitute the first line of treatment for inducing weight loss [22, 23]. Energy balance and maintenance of body weight depend on the balance between energy intake and energy expenditure [24]. Food restriction increased AgRP and NPY mRNAs in both running and sedentary rats, which reflected a stronger negative energy balance and also indicated that AgRP and NPY expression was dependent on the availability of energy stores [25]. Single circuit resistance exercise (at 35% 1RM) increased plasma AgRP in male college students [26]. It has been reported that the effect of fasting resulted in elevation of plasma AgRP levels in obese men [27]. Researches on ghrelin responses to a single session acute exercise have contradictory results. Number of studies found no changes in plasma ghrelin [28, 29]; but, other evidences showed both significant depression [30, 31] and elevation [32, 33]. Conflicting findings is obtained on ghrelin response to exercise training, so some research, increases plasma ghrelin on the effects of exercise training, and has decreased in the others [23, 24]. Rashidlamir and et al. showed significant increase in ghrelin and significant reduction in growth hormone after six weeks of training and wrestling circuits [25]. On the other hand, the information about the acute effect of aerobic and circuit-resistance exercises on AgRP and ghrelin plasma concentration in women whose role in the healthiness of society is undeniable and effective, are lacking. Thus, this study was conducted to investigate the acute effect of two types of exercise training (aerobic and circuit-resistance) on AgRP plasma concentration in female students.

Materials and Methods

Before administrating this study, the researchers obtained approval from the ethical committee of Ferdowsi University of Mashhad, Iran.

Participants: Twenty-four well-trained females were randomly assigned into three groups of eight participants: (1) Control (no exercise); (2) Circuit resistance group [resistance training at 60% of a one-repetition maximum (1-RM)]; (3) Aerobic group (1.5 miles run at 70% of VO_2max). Subjects were asked to complete a medical examination and a medical questionnaire to ascertain that they did not take any medication, were free of cardiac, respiratory, renal, metabolic diseases. According to research findings on the impact of estrogen on ghrelin [34], all participants were all in luteal phase of their menstrual cycle. All subjects were accustomed to the training protocol.

Dietary control: Participants were asked to complete the dietary questionnaire for details of the energy and macronutrient intakes during 3 days before the protocol. Their daily food intakes (dietary habit) were 55-65% carbohydrate, 15-20% fat and 20-25% protein. Then, the diet for the night before the blood sampling proposed to all participants.

Training protocol: Before the main trial, participants were taken to the weight training room three times. On the first two visits, all of the participants performed strength tests to determine their 1-RM for each of the nine free-weight resistance exercises (arm curl, triceps extension, back extension, squat, leg curl, bench press, overhead press, dead lift, seated row). On the third visit, the subjects completed a practice session to make sure that each participant was able to complete the entire exercise session. Subjects were asked to perform 3 sets of 9 non-stop circuits (9 exercises with 60% of one-repetition maximum for each exercise at their maximum speed) with a 5-minute rest between the sets. The aerobic group was asked to run for 1.5 miles at 70% of their VO_2max. The employed procedure was the same as the previously reported one [28-36]. The training session for both the aerobic and anaerobic groups consisted of a 10-minute warm-up, followed by the main exercises, and a-10 minute cool-down period in the end. The duration of the entire session was 35-45 minutes, and the control group remained sedentary during this time.

The heart rate of the subjects was constantly checked (70-85% of the maximum heart rate) using the polar device (F1tm, Finland). The exercise sessions were held in the morning between 08:30 and 11:00, to avoid the effects of circadian rhythms. Subjects were instructed to follow normal lifestyle habits, avoid medications, refrain from exercise three days before the study, 12 hours of fasting and visit the lab after a 12-hour fast (only allowed to drink pure water) for the blood sampling. All the subjects were randomly divided into two experimental and one control groups.

Experimental group I (N=8) followed researchers'recommended running 1.5 miles with a fixed heart rate (VO_2max 70%) (AE group); experimental group II (N=8) was asked to completed a circuit-resistance exercise protocol (RE group). The participants were instructed to follow a normal lifestyle, maintain daily habits, avoid all medications, and refrain from exercising 3 days before the experiment session.

Blood samples: Blood samples (10 ml) were taken before and immediately after the protocol, while the subjects were overnight fast (at least 12 h). Plasma was obtained for AgRP and ghrelin determination. Plasma AGgRP was measured by using a special kit, which was performed with commercially available enzyme-linked immuno sorbent assay (ELISA) kits (Pharmaceuticals, Belmont, USA, Sensitivity 0.07 ng/ml).

The total level of plasma ghrelin was measured by using a special kit, which was performed with commercially available enzyme-linked immuno sorbent assay (ELISA) kits (Pharmaceuticals, Belmont, USA, Sensitivity 6.25 pg/ml)

Body fat percentage measurements: Percentage of body fat from skin fold thickness of subjects was measured by using the 3-point skin fold thickness method [30]. The skin fold thickness at three sites was obtained using a caliper. The skin fold sites were triceps, sub scapular and abdomen.

Anthropometric measurement: The subjects were weighted by a digital scale of 0.01 kg accuracy; all subjects were without any clothes and shoes. Subjects' height was measured using a meter on a wall; all subjects were without clothes and shoes. Body mass index (BMI) was also calculated as weight (kg) divided by height (m) squared. Fat percentage of the subjects was measured using caliper device by the method of three skin fold thickness (subscapular, abdominal, and triceps) [37].

Statistical analysis: All calculations were made using SPSS-16.0. All data are presented as mean±standard error of mean (SE). We used Levene's test to assess homogeneity of variances and used Kolmogorov-Smirnov test to examine normal distribution of data. One-way analysis of variance (one-way ANOVA) and Tukey post hoc test were used to examine the difference between the groups. Statistical significance was accepted at the 5% level.

Results

Individual characteristics of the subjects determined by are highlighted in table 1. According to the result of Levene's test and the Kolmogorov-Smirnov test, all dependent variables were normally distributed and homogeneous. Significant increases were noted in circulating AgRP of control group (t=-8.775; p=0.001), cicuit resistance (t=-24.77; p=0.001) and aerobic group (t=-36.940; p=0.001). Also, there was significant increases in circulating Ghrelin of control (t=-12.320; p=0.0001), circuit resistance (t=-92.884; p=0.001) and aerobic subjects (t=-135.227; p=0.00).

At the end of the protocols, one-way ANOVA indicated a statistically significant difference between the groups in plasma AgRP levels (F=385.715; p=0.001), plasma Ghrelin levels (F=4011.9; p=0.001). At the end of the protocols, the results of the Tukey post hoc test showed that plasma AgRP levels (ng/ml) in circuit resistance group were significantly higher when compared to the aerobic group (p=0.011) (Fig. 1, Table 2) (0.0575 ng/mL to 1.235 ng/ml). According to the results of the Tukey post hoc test, there was also an increase in the plasma Ghrelin levels of circuit resistance subjects when compared to the aerobic subjects, but the increase was not significant (p=0.545).

Discussion

As the results show, a single bout of circuit resistance exercise and aerobic training can significantly increase AgRP and ghrelin concentration in plasma. Andersson et al. [38]. Reported that a single session of treadmill running (22 m/min, 10% grade) resulted in a significant increase in plasma total ghrelin levels. In a study, regardless of the increase in GH, ghrelin was significantly depressed following the single bout of circuit resistance exercise [32]. Currently two studies exist concerning plasma ghrelin response to an aerobic exercise [39, 40].

Acute effects of a single bout of exercise on appetite and energy intake have been investigated by several researchers [35, 41]. According to previous studies findings, negative energy balance to stimulate appetite and increase food intake depends on intensity, duration, mode of exercise, initial levels of fuel sources, and nutritional status [41, 42].

Table 1. Anthropometric characteristics of participants

Variables	Control group (N=8)	Circuit resistance group (N=8)	Aerobic group (N=8)
Age (years)	21.37±1.76	21.87±3.04	21.12±2.35
Height (cm)	161.69±1.85	160.5±3.99	163.12±5.20
Weight (kg)	59.68±7.65	56.35±9.31	58.25±7.80
BMI(kg/m^2)	22.88±3.49	21.8±3.01	21.82±1.92
Body fat (%)	19.67±1.65	18.29±3.14	19.67±5.51

Values are means±SE

Table 2. Pre and post training values of plasma AgRP and ghrelin concentrations in the three groups

Variable	Control group (N= 8)		Circuit resistance group (N=8)		Aerobic group (N=8)		*p*-Value
	Before	After	Before	After	Before	After	
Ghrelin (ng/ml)	0.712 ± 0.0324	1.300 ± 0.150	0.752 ± 0.0296	8.302 ± 0.2014	0.700 ± 0.0385	8.155 ± 0.177	0.001*
AgRP (ng/ml)	0.0375 ± 0.0158	0.0650 ± 0.0220	0.0575 ± 0.019	1.235 ± 0.1421	0.0575 ± 0.0265	1.090 ± 0.0767	0.000 *

Values are means±SD, *: p<0.05

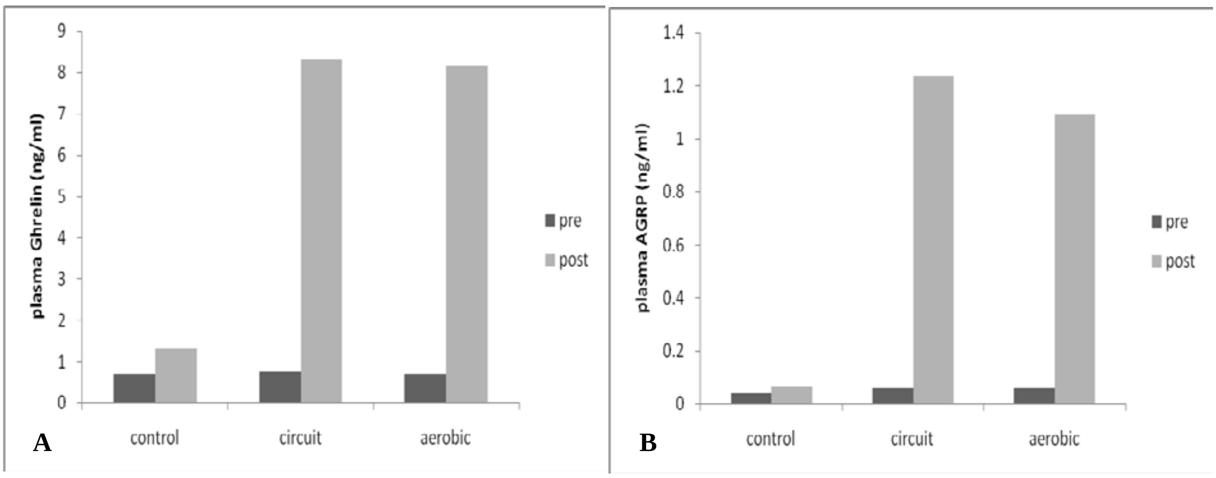

Figure 1. Plasma ghrelin (A) and AgRP (B) levels in the three groups before and after training protocol

The main findings of this research are as follows: plasma AgRP and ghrelin levels were significantly increased in the three groups, whereas the increase in experimental groups was significant higher when compared to the control group. There are few researches about the effect of exercising on plasma AgRP, especially in humans. In these few studies, the effects of fasting and satiety have been investigated on plasma AgRP [39]. It has been shown that AgRP had both an acute and long-term effect on food intake [43].

During positive energy balance, reduced AgRP gene expression inhibits feeding and suppresses appetite. In contrast, during fasting, when energy balance is negative and also energy stores are low, increased AgRP gene expression stimulates feeding or promotes appetite [8, 11, 13, 25]. This change in the resting plasma AgRP and ghrelin levels may be due to decreased energy stores and/or cellular charge during the protocol. A changed ATP/ADP ratio stimulates or suppresses several mechanisms, which increase/decrease orexigenic gene expression such as AgRP and ghrelin [11]. These positive changes in energy balance could reintegrate homeostasis of energy in the body. In study by Ghanbari-Niaki et al. [26]. Plasma AgRP levels increased immediately after exercising and returned to the pre-exercise level in the recovery period. It has been determined that food restricting would significantly increase plasma AgRP levels both on rats in humans [25, 44]. On the other hand, exercising and physical activity disorders energy balance and homeostasis between muscle cells and increases the demands of energy for cells. This probable decrease of energy resources of cells with overnight fasting could be an important factor in increasing plasma AgRP and ghrelin concentration in the subjects. It could be concluded from the findings that hypothalamus increases plasma AgRP after exercising. Findings of this study are in compliance with this concept that: inner and outer signal channels of the cells are responsible for increasing appetite due to increase of energy expenditure after exercising and AgRP appetite increasing signal channel could be an important channel toward response to exercising [45].

The present study has investigated the effect of two types of different exercising in young athlete females. The results obtained from this study are in accordance with these few researches. Since ghrelin and AgRP collaborate in increasing appetite, and in previous studies increasing of these two peptides have been reported to be concurrent [45], has agreed with the present study. Nonetheless, it seems that further investigation is necessary to understand possible mechanisms in this field. It seems that the increase in plasma AgRP can be in line with the increase of this peptide in hypothalamus and in response to the negative energy balance due to the exercising which increases the appetite in order to recover energy resources of the subjects' body. In summary, the data indicate that a single circuitresistance exercise (at 60% 1RM) and aerobic exercise increased plasma AgRP and ghrelin. A post-exercise elevated plasma AGRP and ghrelin level might thus be considered as a possible stimulus for a post-exercise induced hyperphagia state.

Acknowledgements

We wish to thank Dr. Fatemeh Rahbarizade for her helpful comments and guides.

Authors' Contributions

All authors had equal role in design, work, statistical analysis and manuscript writing.

Conflict of Interest

The authors declare no conflict of interest.

Funding/Support

Ferdowsi University of Mashhad.

References

1. Oszanecka-Glinianowicz M, Zahorska-Markiewicz B, Plewa M and Janowska J. The effect of short-term exercise on nitric oxide (no) serum concentrations in overweight and obese women. Biol Sport 2008; 25(2): 125-134.
2. Walley AJ, Asher JE, Froguel P. The genetic contribution to nonsyndromic human obesity. Nat Rev Genet 2009; 10(7): 431-442.
3. Arora S, Anubhuti. Role of neuropeptides in appetite regulation and obesity: A review. Neuropeptides 2006; 40(6): 375-401.
4. Rossner S, Sjostrom L, Noack R, et al. Weight loss, weight maintenance, and improved cardiovascular risk factors after 2 years treatment with orlistat for obesity. Obes Res 2000; 8(1): 49-61.
5. Castaneda TR, Tong J, Datta R, et al. Ghrelin in the regulation of body weight and metabolism. Front Neuroendocrinol 2010; 31(1): 44-60.
6. Matsuoa T, Murotakeb Y, Kima MJ, et al. High general self-efficacy is associated with less weight loss under a supervised dietary modification program. Obes Res Clin Pract 2010; 4(2): 135-144.
7. Bilski J, Teleglow A, Zahradnik-Bilska J, et al. Effects of exercise on appetite and food intake regulation. Med. Sportowa 2009; 13: 82-94.
8. Krashes MJ, Koda S, Ye C, et al. Rapid, reversible activation of AgRP neurons drives feeding behavior in mice. J Clin Invest 2011; 121(4): 1424-1428.
9. Remmers F, Delemarre-van de Waal HA. Developmental programming of energy balance and its hypothalamic regulation. Endocrine Rev 2011; 32(2): 272-231.
10. Sousa-Ferreira L, Alvaro AR, Aveleira C, et al. Proliferative hypothalamic neurospheres express NPY, AGRP, POMC, CART and OREXIN-A and differentiate to functional neurons. PLoS One 2011; 6(5): e19745.
11. Benite-Ribeiro SA, Santos JM, Soares-Filho MC and Duarte JAR. Influence of regular physical exercise on increased caloric intake triggered by stressors. Annu Rev Biomed Sci 2010; 12: 30-45.
12. Ghanbari-Niaki A, Nabatchian S, Hedayati M. Plasma agouti-related protein (AgRP), growth hormone, insulin responses to a single circuit-resistance exercise in male college students. Peptides 2007; 28(5): 1035-1039.

13. Murphy M, Ebling FJP. The role of hypothalamic tri-iodothyronine availability in seasonal regulation of energy balance and body weight. J Thyroid Res 2011; 2011: 1-7.
14. Kojima M, Hosoda H, Date Y, et al. Ghrelin is a growth-hormone-releasing acylated peptide from stomach. Nature 1999; 402(6762): 656-660.
15. Hosoda H, Kojima M, Matsuo H and Kengawa K. Ghrelin and des-acyl ghrelin: two major forms of rat ghrelin peptide in gastrointestinal tissue. Biochem Biophys Res Commun 2000; 279(3): 909-913.
16. Wren AM, Seal LJ, Cohen MA, et al. Ghrelin enhances appetite and increases food intake in humans. J Clin Endocrinol Metab 2001; 86(12): 5992-5995.
17. Kirchner H, Gutierrez JA, Solenberg PJ, et al. GOAT links dietary lipids with the endocrine control of energy balance. Nat Med 2009; 15(7): 741-745.
18. Tschop M, Wawarta R, Riepl R, et al. Post-prandial decrease of circulating human ghrelin levels. J Endocrinol Invest 2001; 24(6): 19-21.
19. Sangiao-Alvarellos S, Vazquez MJ, Varela L, et al. Central ghrelin regulates peripheral lipid metabolism in a growth hormone-independent fashion. Endocrinology 2009; 150(10): 4562- 4574.
20. Sun Y, Wang P, Zheng H and Smith RG. Ghrelin stimulation of growth hormone release and appetite is mediated through the growth hormone secretagogue receptor. Proc Natl Acad Sci USA 2004; 101(13): 4679-4684.
21. Zigman JM, Jones JE, Lee CE, et al. Expression of ghrelin receptor mRNA in the rat and the mouse brain. J Comp Neurol 2006; 494(3): 528-548.
22. Sarika AA. Role of neuropeptides in appetite regulation and obesity: A review. Neuropeptides 2006; 40(6): 375-401.
23. Wit B, Lerczak K, PanczenkoKresowska B, et al. Effects of “fat-burning” exercise and low-energy diet on lipid peroxidation products (TBARS) in plasma of subjects with overweight or obesity. Biol Sport 2003; 20(4): 321-330.
24. Yuksel B. Neuroendocrinological control of obesity. J Clin Res Ped Endocrinol 2009; (Suppl 1): 58-64.
25. Rijke CE, Hillebrand JJG, Verhagen LA, et al. Hypothalamic neuropeptide expression following chronic food restriction in sedentary and wheel-running rats. J Mol Endocrinol 2005; 35(2): 381390.
26. Ghanbari-Niaki A, Nabatchian S, Hedayati M. Plasma agouti-related protein (AgRP), growth hormone, insulin responses to a single circuit-resistance exercise in male college students. Peptides 2007; 28(5): 1035-1039.
27. Katsuki A, Sumida Y, Gabazza EC, et al. Plasam levels of agouti-related protein are increased in obese men. J Clin Endocrinol Metab 2001; 86(5): 1921-4.
28. Burns SF, Broom DR, Myaishita M, et al. A single session of treadmill running has no effect on plasma total ghrelin concentrations. J Sports Sci 2007; 25(6): 635-642.
29. King JA, Wasse LK, Ewens J, et al. Differential acylated ghrelin, peptide YY3-36, appetite, and food intake responses to equivalent energy deficits created by exercise and food restriction. J Clin Endocrinol Metab 2011; 96(4): 1114-1121.
30. Shiiya T, Ueno H, Toshinai K, et al. Significant lowering of plasma ghrelin but not des-acyl ghrelin in response to acute exercise in men. Endocr J 2011; 58(5): 335-342.
31. Hsu YW, Pan YJ, Cho YM, et al. Aging effects on exercise-induced alternations in plasma acylated ghrelin and leptin in male rats. Eur J Appl Physiol 2011; 111(5): 809-817.
32. Jurimae J, Jurimae T, Purge P. Plasma ghrelin responses to acute sculling exercises in elite male rowers. Exp Biol Med (Maywood) 2007; 232(7): 904-909.
33. Abaassi-Daluee A, Ghanbari-Niaki A, Fathi R and M. Hedayati. The effect of a single session aerobic exercise on plasma ghrelin, GH, insulin and cortisol in non-athlete university male students. Iran J Endocrinol Metab 2011; 13(2): 197-201.
34. Matsubara M, Sakata I, Wada R, et al. Estrogen modulates ghrelin expression in the female rat stomach. Peptides 2004; 25(2): 289-97.
35. Imbeault P, Saint-Pierre S, Almeras N and Tremblay A. Acute effects of exercise on energy intake and feeding behavior. Br J Nutr 1997; 77(4): 511-21.
36. George JD, Vehrs PR, Allsen PA, et al. Development of a submaximal treadmill jogging test for life college-aged individuals. Med Sci Sports Exerc 1993; 25(5): 643-647.
37. Wagner Dale R. Body composition assessment and minimal weight recommendations for high school wrestlers. J Athl Train 1993; 31(3): 262-265.
38. Andersson U, Treebak JT, Nielsen JN, et al. Exercise in rats does not alter hypothalamic AMP-activated protein kinase activity. Biochem Biophys Res Commun 2006; 329(2): 719-25.
39. Kraemer RR, Durand RJ, et al. Ghrelin and other glucoregulatory hormone responses to eccentric and concentric muscle contractions. Endocrine 2004; 24(1): 93-8.
40. Schmidt A, Maier C, Schaller G, et al. Acute exercise has no effect on ghrelin plasma concentrations. Horm Metab Res 2004; 36(3): 174-7.
41. Maraki M, Tsofliou F, Pitsiladis YP, et al. Acute effects of a single exercise class on appetite, energy intake and mood. Is there a time of day effect? Appetite 2005; 45(3): 272-278.
42. Ferguson MA, Alderson NL, Trost SG, et al. Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. J Appl Physiol 1998; 85(3): 1169-1174.
43. Hillebrand JJG, De WD, Adan RA. Neuropeptides. food intake and body weight regulation: A hypothalamic focus. Peptides 2002; 23(12): 2283-2306.
44. Foster-Schubert KE, McTiernan A, Frayo RS, et al. Human plasma ghrelin levels increase during a one- year exercise program. J Clin Endocrinol Metab 2005; 90(2): 820-5.
45. Ghanbari-Niaki A, Saghebjoo M, Rashid-Lamir A, et al. Acute circuitresistance exercise increases expression of lymphocyte agouti-related protein in young women. Exp Biol Med 2010; 235(3): 326-334.

Please cite this article as: Ebrahimnia M, Rashidlamir A, Seyed-Alhosseini M. Effect of a single session of aerobic and circuit-resistance exercise on plasma ghirelin and agouti-related peptide (agrp) levels in well-trained females. Zahedan J Res Med Sci (ZJRMS) 2014; 16(6): 25-29.