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ORIGINAL ARTICLE

Acute Response of Acylated Ghrelin to one Moderate Exercise in Obese men

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ABSTRACT

Ghrelin has been proposed to be a regulator of energy balance, but its response to short time exercise has not drawn much attention. In this study, plasma response of acylated ghrelin to one moderate exercise was investigated. To investigate plasma ghrelin response to short term exercise in obese males. Fifteen adult sedentary healthy obese men aged 35 - 45 years and weight 85 - 101 kg was completed an exercise test included 45 min moderate intensity running test with no slope. Plasma ghrelin levels were measured before and immediately after exercise test. Pre- and post exercise levels of plasma ghrelin were compared using a paired-samples t-test. A p value of less than 0.05 was considered as statistically significant. No significant differences was found in plasma ghrelin values between pre and post exercise test (from 61.2 - 15.5 in pre versus 56.5 - 11.6 in post test, p = 0.062). We conclude that there is no meaningful acute effect of short time exercise on plasma ghrelin in obese men. Future studies should examine the potential role of exercise on ghrelin or other peptides involved in energy balance. **Keywords:** Ghrelin, Exercise, Obesity, Homeostasis

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INTRODUCTION

Numerous findings support the claim that obesity or increases in body fat percentages, especially abdominal obesity, is the main factor for developing type 2 diabetes when people are no longer young [1]. In this decade, hormones secreted by fat tissues, and some peptide hormones called cytokines, have been introduced as regulators of skeletal muscle metabolism and of energy homeostasis [2]. Nowadays, numerous studies are continuously being conducted to identify environmental or genetic factors that are involved in the imbalance between energy intake and energy expenditure. Moreover, the role played by hormonal factors is also a focus of attention for health and wellness specialists [3].

Ghrelin is a 28-amino-acid peptide hormone mainly secreted by the stomach but in smaller amounts by other tissues such as the small intestine too [4]. Plasma level of ghrelin, as an appetite-stimulating factor, increasingly rises before eating food and reaches its minimum level up to one hour after eating [5, 6]. Extensive studies have reported ghrelin has a role in the expansion of the metabolic syndrome and type-2 diabetes [7]. However, in obese people, blood ghrelin levels rise again shortly after eating, and this is accompanied by starting to feel hungry again [8].

Therefore, researchers focus on maintaining a balanced level of ghrelin in order to regulate energy homeostasis and body weight. In this relation, the role played by some external intervening factors such as controlled diets and exercise has always been emphasized so that numerous studies are always being conducted with the purpose of employing many diets and short or long-term training programs to improve systemic levels of ghrelin or other peptide intermediaries effective in energy homeostasis. Nevertheless, findings in this area are still contradictory and divergent: some point to reduced ghrelin levels in response to short or long training programs [9, 10], while others show ghrelin levels do not respond to training [11]. Despite the contradictory results obtained in this regard, most studies have continued investigation of the effects that long-term training programs have on ghrelin levels, and few

studies have measured acute ghrelin response to one-session exercise tests. Therefore, this research was conducted with the purpose of determining acute ghrelin response to a one-session exercise test in the form of average-intensity running by obese people on a flat surface.

MATERIALS AND METHODS

Human subjects and inclusion: Participants were fifteen sedentary healthy obese men aged 35 to 45 year and BMI of 30 to 33 kg/m². All subjects of two groups were inactive, non-smoker and non-alcoholics. Informed consent was obtained from each subject after full explanation of the purpose, nature and risk of all procedures used.

None of the subjects used drugs or therapies for obesity, and none had a past history of disease or injury that would prevent daily exercise. Patients with known history of neuromuscular disease, cardiopulmonary disease, respiratory infections, and type II diabetes or other chronic diseases were excluded.

Anthropometry: The weight and height of the participants were measured by the same person when the participant had thin clothes on and was wearing no shoes by using the standard hospital scales. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. Waist circumference (WC) was measured at the superior border of the iliac crest and was taken to the nearest 0.1 cm after a normal expiration. Body composition monitor (BF508-Omron made in Finland) with a precision error of less than 100 g was used to measure weight and body fat percentage of the subjects.

Body Mass index (BMI) was calculated using the formula body weight/height2 in terms of kg/m². The arterial systolic and diastolic blood pressures (BP) were calculated after they rested for 10 minutes with a mercury manometer with appropriate sleeves from the right and left arm in sitting position.

Laboratory and exercise test: Venous blood samples were collected before and after exercise test. Exercise test lasted 45 min running in moderate intensity (70 % of maximal heart rate) with no slope. Target heart rate was monitored by polar telemetry. All participants refrained from any severe physical activity 48 h before measurements. Plasma ghrelin was measured by ELIZA (Human Acylated Ghrelin Kit, Biovendor, Austria)

Statistical analysis: All data were tested for normal distribution by the Kolmogorov-Smirnov test. Statistical analysis was performed with the SPSS software version 15.0. Paired t test was used to determine the mean differences between baseline and post-exercise values of plasma ghrelin of studied subjects. P value of less than 0.05 was regarded as indicative of a significant difference.

RESULTS

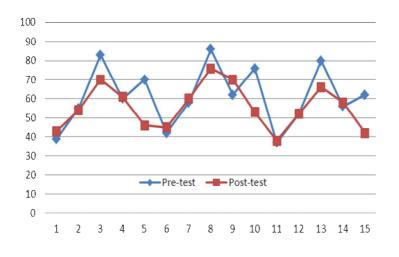
As mentioned previous, this study aimed to assess acute response of acylated ghrelin to on moderate exercise test included 45 min running with no slope in adults healthy obese men. Table 1 show the descriptive anthropometric and biochemical features of the studied subjects. All values are represented as mean \pm SD. All subjects were obese. There are inconsistent finding about serum or plasma ghrelin in response to short or chronic exercise in healthy or diseases population. Data by our study also sowed no significant change in plasma ghrelin between pre and post exercise in studied obese subjects (from 61.2 – 15.5 in pre versus 56.5 – 11.6 in post test, p = 0.062, Fig 1). In other word, 45 min running at 70 % of maximal heart rate did not affect plasma ghrelin in adult obese men.

Table 1: Body weight and blood chemistry parameters of studied subjects			
Variable	Mean	Standard deviation	Range
Age (years)	39.73	2.34	35 - 45
Weight (kg)	94.3	5.18	85 - 101
Height (cm)	173	4.8	163 - 178
Waist circumference (cm)	103	3.7	98 - 109
Hip circumference (cm)	102	4.5	95 - 111
Body mass index (kg/m2)	31.59	1.1	30 - 33
Body fat (%)	31.9	1.1	30 - 34
Cholesterol (mg / dl)	213	24	165 - 240
Triglyceride (mg / dl)	205	49	130 – 253
Low density lipoprotein (mg / dl)	125	16	104 - 152
high density lipoprotein (mg / dl)	47.7	2.79	42 - 51

DISCUSSION

Findings of the research showed that blood ghrelin levels did not significantly change in response to one session of relatively long-duration exercise test. In other words, average-intensity running on a flat

surface without any slope for 45 minutes did not lead to significant changes in the ghrelin level immediately after the test was stopped. However, the question that remains is this: can we emphasize, based on this conclusion, that short-term exercise have no effect on hunger levels or on appetite for food? Concerning this question, recent research has shown that some specific peptide hormones at different physiological concentrations influence the extent of appetite in animals and humans (12, 13, 14) and, hence, their physiological role in satiety after eating points to their role and effect as pharmacokinetic factors for anti-obesity purposes. In humans, ghrelin injections lead to increased appetite, but it has not been completely clarified yet whether intravenous injections of ghrelin can by themselves influence nutritional behavior in humans (15).



Subjects

Fig 1: Acylated ghrelin of 15 obese subjects at pre and post of exercise test. Each number of vertical columns represents

In most people, body weight remains constant for years under constant conditions or under conditions of energy equilibrium. The necessary condition for constant weight is having constant energy equilibrium so that energy intake and expenditure are equal during the 24-hour period of the day. Disruptions in energy equilibrium are mainly accompanied by the problem of weight gain or weight loss in people (3). Scientific sources have referred to the role played by ghrelin in regulating short-term food consumption, and to its role in regulating long-term energy equilibrium [16]. Researchers have also revealed that daily intake of ghrelin leads to increased levels of body fat through preventing the participation of fats in energy metabolism [16]. The importance of this 28-amino-acid peptide in glucose homeostasis and carbohydrates and fats metabolism has been reported in some studies [17, 18]. Moreover, ghrelin intake in humans resulted in reduced blood insulin level and to increased blood glucose concentration [19, 20]. Laboratory experiments have indicated that ghrelin intake by rats disrupts metabolism of fats in fat tissues, in the liver and in skeletal muscles, and is accompanied by body weight increase [21].

Based on this evidence, Researchers in nutrition and wellness sciences have focused on developing suitable strategies to improve the level of blood circulation and to regulate the secretion and release of this hormone. Findings of a recent research showed that a 12-week aerobic training led to significant reduction in the serum ghrelin level of obese adult men [3].In another study also, plasma ghrelin level significantly declined following average- intensity aerobic activities [9]. Findings of this research indicated that one session of bicycling significantly reduced ghrelin concentration (9). Moreover, in another study, four weeks of exercise training were followed by significant reductions in blood ghrelin level (10). However, contrary to the findings mentioned above, a recent study showed that long-term exercise programs for obese people were followed by reduced body weight but led to significant increases in plasma ghrelin concentration [22]. Nevertheless, in another study, a 60-minute session of walking on a treadmill was not accompanied by changes in ghrelin and obestatin levels [11].

In another study, the combination of long-term diet and exercise training was accompanied by increased obestatin and significantly reduced leptin with no changes in ghrelin concentration in obese people [23]. Scientific sources have revealed that if exercise training are not accompanied by long-term diet together with significant reductions in body weight, they will not lead to changes in insulin resistance, insulin, ghrelin and other peptide hormones [23]. Researchers have pointed out that a reduction in ghrelin levels in the hypothalamus due to long-term exercise training leads to reduced appetite and body weight (11). However, molecular mechanisms that are responsible for the effects of short-term exercise, such as those studied in this research, and for the effects of other peptides influencing appetite regulation and body weight, have not been completely found yet. It seems that exercise test types, based on the intensity and duration of engaging in exercise, on the measurement tools, and on the studied population, are among the main intervening factors in ghrelin's response to exercise tests. For example, some studies have reported that a single session of a exercise test can only lead to a change or improvement in peptide levels secreted from fat tissues if the test leads to negative energy equilibrium with the expenditure of more than 800 kilocalories, or if the test lasts more than 60 minutes (24, 25).

REFERENCES

- 1. Kouidhi, S. Jarboui, S. Marrakchi, R. Clerget Froidevaux, M.S. Seugnet, I. Abid, H et al. (2010). Adiponectin expression and metabolic markers in obesity and type 2 diabetes. J Endocrinol Invest. [Epub ahead of print].
- 2. Ariga, H. Imai, K. Chen, C. Mantyh, C. Pappas, T.N. Takahashi, T. (2008). Does ghrelin explain accelerated gastric emptying in the early stages of diabetes mellitus? Am J Physiol Regul Integr Comp Physiol. 294(6):1807-12.
- 3. Eizadi, M. Bakhshi, S. Abripham, P. Khorshidi, D .(2011). Chronic aerobic exercise associated with reduced body weight decreases serum ghrelin in adult obese individuals. Advances in Bioresearch. 2(2): 156-162.
- 4. Korbonits, M. Grossman, A.B. (2004). Ghrelin: update on a novel hormonal system. Eur J Endocrinol. 151(Suppl 1): 67–70.
- 5. Tschop, M. Wawarta, R. Riepl, R.L. Friedrich, S. Bidlingmaier, M. Landgraf, R. Folwaczny, C. (2001). Post-prandial decrease of circulating human ghrelin levels. J Endocrinol Invest. 24:19–21.
- 6. Cummings, D.E. Purnell, J.Q. Frayo, R.S. Schmidova, K. Wisse, B.E. Weigle, D.S. (2001). A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. Diabetes. 50:1714–1719.
- 7. Ukkola, O. Kunnari, A. Jokela, M. Päivänsalo, M. Kesäniemi, Y.A. (2009). Ghrelin and metabolic disorders. Current Protein and Peptide Science. 10 (1): 2–7.
- 8. Colombo, M. Gregersen, S. Xiao, J. Hermansen, K. (2003). Effects of ghrelin and other neuropeptides (CART, MCH, orexin A and B, and GLP-1) on the release of insulin from isolated rat islets. Pancreas. 27(2): 161-6.
- 9. Malkova, D. McLaughlin, R. Manthou, E. Wallace, A.M. Nimmo, M.A. (2008). Effect of Moderate-intensity Exercise Session on Preprandial and Postprandial Responses of Circulating Ghrelin and Appetite. Horm Metab Res. [Epub ahead of print].
- 10. Vestergaard, E.T. Dall, R. Lange, K.H. Kjaer, M. Christiansen, J.S. Jorgensen, J.O. (2007). The ghrelin response to exercise before and after growth hormone administration. J Clin Endocrinol Metab. 92(1): 297-303.
- 11. Wang, J. Chen, C. Wang, R.Y. (2008). Influence of short- and long-term treadmill exercises on levels of ghrelin, obestatin and NPY in plasma and brain extraction of obese rats. Endocrine. [Epub ahead of print].
- 12. Wren, A.M. Small, C.J.Ward, H.L. Murphy, K.G. Dakin, C.L. Taheri, S. et al. (2000). The novel hypothalamic peptide ghrelin stimulates food intake and growth hormone secretion. Endocrinology. 141: 4325–4328.
- 13. Wren, A.M. Seal, L.J. Cohen, M.A. Brynes, A.E. Frost, G.S. Murphy, K.G. et al. (2001). Ghrelin enhances appetite and increases food intake in humans. J Clin EndocrinolMetab. 86: 5992.
- 14. Cohen, M.A. Ellis, S.M. le Roux, C.W. Batterham, R.L. Park, A. Patterson, M. et al. (2003). Oxyntomodulin suppresses appetite and reduces food intake in humans. J Clin Endocrinol Metab. 88: 4696–4701.
- 15. Schmid, D.A. Held, K. Ising, M. Uhr, M. Weikel, J.C. Steiger, A. (2005). Ghrelin stimulates appetite, imagination of food, GH, ACTH, and cortisol, but does not affect leptin in normal controls. Neuropsychopharmacology. 30(6):1187-92.
- 16. Tschop, M. Smiley, D.L. Heiman, M.L. (2000). Ghrelin induces adiposity in rodents. Nature. 407: 908–913.
- 17. Zigman, J.M. Nakano, Y. Coppari, R. Balthasar, N. Marcus, J.N. Lee, C.E. et al. (2005). Mice lacking ghrelin receptors resist the development of diet-induced obesity. J Clin Invest. 115: 3564-3572.
- 18. Sun, Y. Butte, N.F. Garcia, J.M. Smith, R.G. (2008). Characterization of adult ghrelin and ghrelin receptor knockout mice under positive and negative energy balance. Endocrinology. 149: 843-850.
- 19. Broglio, F. Arvat, E. Benso, A. Gottero, C. Muccioli, G. Papotti, M. van der Lely, A.J. (2007). Ghrelin, a natural GH secretagogue produced by the stomach, induces hyperglycemia and reduces insulin secretion in humans. J Clin Endocrinol Metab. 86: 5083–5086.
- 20. Broglio, F. Gianotti, L. Destefanis, S. Fassino, S. Abbate Daga, G. Mondelli, V. (2004). The endocrine response to acute ghrelin administration is blunted in patients with anorexia nervosa, a ghrelin hypersecretory state. Clin Endocrinol (Oxf). 60:592-599.
- 21. Barazzoni, R. Bosutti, A. Stebel, M. Cattin, M.R. Roder, E. Visintin, L. (2005). Ghrelin regulates mitochondriallipid metabolism gene expression and tissue fat distribution in liver and skeletal muscle. Am J Physiol Endocrinol Metab. 288: 228-235.

- 22. Kelishadi, R. Hashemipour, M. Mohammadifard, N. Alikhassy, H. Adeli, K. (2008). Short- and long-term relationships of serum ghrelin with changes in body composition and the metabolic syndrome in prepubescent obese children following two different weight loss programs. Clin Endocrinol (Oxf). [Epub ahead of print].
- 23. Reinehr, T. de Sousa, G. Roth, C.L. (2008). Obestatin and ghrelin levels in obese children and adolescents before and after reduction of overweight. : Clin Endocrinol (Oxf). 68(2):304-10.
- 24. Hqjbjerre, L. Rozenzweig, M. Deala, F. Bruun, J.M. Stallknecht, B. (2007). Acute exercise increases adipose tissue interstitial adiponectin concentration in healthy overweight and lean subjects. European Journal of Endocrinology. 157(5): 613–623.
- 25. Kraemer, R.R. Chu, H. Castracane, D.V. (2000). Leptin and exercise. Experimental Biology and Medicine 227: 701–708.