

## **A Preprandial and Postprandial Plasma Levels of Ghrelin Hormone in Lean, Overweight and Obese Saudi Females**

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**Abstract.** Ghrelin is a novel gastrointestinal peptide hormone isolated from human and rat stomach. Ghrelin administration stimulates growth hormone secretion but also causes weight gain by increasing food intake and reducing fat utilization in rodents. This study aims to determine the plasma level of ghrelin under basal condition and in response to a standard meal and to elucidate the relationship between this peptide and anthropometric measures. Body mass index (BMI), anthropometric measurements were calculated and plasma ghrelin concentrations were determined in 122 obese, overweight and lean Saudi females before and an hour after breakfast. Fasting ghrelin was significantly higher in lean than in obese and overweight subjects and fall after eating in the lean group. There was slight insignificant reduction in circulating ghrelin of the obese and overweight groups. Ghrelin levels were negatively correlated with BMI in obese, overweight and lean subjects. Obese subjects do not exhibit the decline in plasma ghrelin seen after a meal in the lean; the lack of suppression following a meal in obese subjects could lead to increased food consumption and suggest that ghrelin may be involved in the pathophysiology of obesity.

### **Introduction**

Obesity is a major global epidemic problem (Bray, G.A., 2005). It concerns about 35.5% of Saudi adult population (Al-Nozha, M.M., *et al.*, 2005; Al-Othaimen, A.I., *et al.*, 2007). Obesity is a multifactorial disease with genetic, endocrinal and environmental origins (Bouchard, C., 1994; Daghestani, M. H., *et al.*, 2007), resulting from an imbalance between energy intake and expenditure.

Ghrelin, the endogenous ligand for the growth hormone secretagogue receptor (GHSR) (Sun, Y., *et al.*, 2007), is a 28-amino-acid peptide that is secreted primarily by cells in the oxyntic glands of the stomach as well as in the intestine (Date, Y., *et al.*, 2000). It has been reported to have endocrine and nonendocrine actions (Abiko, Y., *et al.*, 2005; Allison, K.C., *et al.*, 2005; Leite-Moreira, A. F., 2008; Taub, D.D., *et al.*, 2008). Ghrelin is a circulating orexigen (Pazos, Y., *et al.*, 2008), and food intake increases after administration of exogenous ghrelin in both rodents and humans (Tschöp, M., *et al.*, 2000; Wren, A. M., *et al.*, 2001). Consistent with

a physiological role for ghrelin in feeding behavior, the administration of antighrelin antibodies or GH secretagogue receptor antagonists reduces food intake (Nakazato, M., *et al.*, 2001). Changes in plasma ghrelin levels might therefore produce important differences in food intake and energy balance and play a significant role in the pathogenesis of obesity (Soriano-Guillén, L., *et al.*, 2004). Ghrelin levels are reported to be increased in Anorexia nervosa (Otto, B., *et al.*, 2001) and after fasting, (Muller, A. F., *et al.*, 2002) whereas in obese patients (Tschöp, M., *et al.*, 2001a) and after feeding they are decreased. (Tschöp, M., *et al.*, 2001b; English, P.J., *et al.*, 2002). These data suggest that ghrelin plays an important endocrine role linking the peripheral mechanisms regulating caloric intake with hypothalamic centers that control energy balance (Muccioli, G., *et al.*, 2002).

This study aims to determine the plasma level of ghrelin under basal condition and in response to a standard meal and to elucidate the relationship between this peptide and anthropometric measures in Saudi females.

## Material and methods

### Subjects

A total of 122 Saudi females volunteers were recruited, aged 20-30 years (mean  $\pm$  SEM). The subjects were divided into 3 groups according to their body mass index (BMI); lean ( $n = 60$ , BMI 18.5-24 kg/m<sup>2</sup>), overweight ( $n = 17$ , BMI 25-29 kg/m<sup>2</sup>) and obese ( $n = 45$ , BMI  $\geq 30$  kg/m<sup>2</sup>). BMI for different groups was determined according to the criteria of the World Health Organization (WHO, 2000). The general characteristics of the subjects are summarized in table 1. All subjects were healthy, free of any medication with regular menstrual cycle, and no history of gastrointestinal or endocrine disorders. Ethical approval was obtained from KFSH&RC Research Ethical committees, and written informed consent was obtained from all subjects.

### Protocol

After an overnight fasting (12 hrs) 5ml venous blood samples were obtained from all subjects in the morning between 08.00h and 09.00h by venipuncture. Following fast blood sample collection, subjects consumed a standard mixed breakfast of about 527 kcal during 15 min. The meal consisted of 50 g white bread, 33g black bread, 18g margarine, 30g cheese, 9g jam and 200 ml of 0.5% fat milk (24.1% fat, 54.4% carbohydrate, 21.5% protein). Blood samples were collected 60 min after the meal ingestion. The samples were collected into chilled tubes containing 1.2 mg EDTA and aprotinin (500 KIU/ml; Trasylol; Bayer Corp., Leverkusen, Germany) for hormone analyses. All samples were kept in an ice bath until centrifugation at 3000 rpm for 15 min at 4°C. Plasma was isolated and stored at -80°C until analysis.

### Anthropometric measurements

Measurements were performed after an overnight fast. Body mass was measured on calibrated balances

or electronic scales to the nearest 0.1 kg. Body height was measured to the nearest centimetre. BMI was calculated as body mass (kilograms) divided by body height (meters) squared. Using a tape measure, with the subject standing, the waist was measured as the narrowest circumference between the lower costal margin and the iliac crest. The hip was the maximum circumference at the level of the femoral trochanters.

### Analytical method

Serum ghrelin levels were measured in duplicate using a commercial ghrelin (human) enzyme immunoassay kit (EIA) from (Phoenix Pharmaceuticals, INC (Belmont,CA) with a lower limit of detection of 0.06 ng/ml.

### Statistical analysis

The descriptive characteristics of the group variables were expressed as mean  $\pm$  SEM. The comparisons among overweight, obese and their lean matched control were done using the independent T-test with respect to all variables. Pearson Correlation Coefficient was used to find the correlation between ghrelin and other studied variables. Significance was declared when P-values are less than 0.05. All statistical analyses were performed using the StatView program for Windows (version 8.0; SAS Institute, Inc., Cary, NC).

## Results

The mean age, BMI, anthropometric, mean ghrelin concentrations of the groups are shown in Table (1).

As presented in Table (1) Student's t-test was applied and significant differences were found in the waist, hip and waist/hip ratio among over weight and obese subjects compared with lean control group.

The fasting ghrelin level was generally decreasing with increasing body weight, it was evident that the

**Table 1. Baseline characteristics of participants**

Variables	Control Lean (n = 60)	Overweight (n = 17)	P value	Obese (n = 45)	P value
Age (yr)	23.95 $\pm$ 0.60	21.59 $\pm$ 0.94	NS	26.49 $\pm$ 0.96	0.03
BMI (kg/m <sup>2</sup> )	20.85 $\pm$ 0.25	27.38 $\pm$ 0.37	<.0001	35.90 $\pm$ 0.92	<.0001
Waist (cm)	66.85 $\pm$ 0.70	81.59 $\pm$ 1.82	<.0001	100.29 $\pm$ 2.14	<.0001
Hip (cm)	94.59 $\pm$ 0.91	105.29 $\pm$ 1.78	<.0001	121.96 $\pm$ 2.14	<.0001
WH Ratio	0.71 $\pm$ 0.01	0.78 $\pm$ 0.01	<.0001	0.82 $\pm$ 0.01	<.0001
Fasting ghrelin (ng/ml)	0.57 $\pm$ 0.02	0.44 $\pm$ 0.02	<.0001	0.28 $\pm$ 0.01	<.0001
Postprandial ghrelin (ng/ml)	0.30 $\pm$ 0.01	0.41 $\pm$ 0.02	<.001	0.27 $\pm$ 0.01	0.02

Note: values are expressed as mean  $\pm$  SE, BMI (body mass index), WH ratio (waist hip ratio), NS (non significant). P level by student's t-test.

levels were remarkably higher among lean females if they were compared with the overweight and obese groups, these differences were statistically significant  $P < 0.001$  (Table. 1). The mean fasting serum ghrelin concentration in these three groups was negatively correlated with BMI (Table. 2). In lean group ghrelin levels negatively correlated with waist and hip (Table. 2). In overweight group the negative correlation was found only between ghrelin levels and both BMI and hip (Table. 2). In obese group ghrelin levels negatively correlated with waist, hip (Table. 2). Ghrelin concentration was declined after the meal in lean control. Meanwhile slight reduction in overweight and obese groups was observed. These differences were statistically significant ( $P < 0.001$  and  $P < 0.05$  respectively).

**Table 2. Correlation between fasting ghrelin and different parameters in lean, overweight and obese groups**

Variables	Lean control group		Overweight group		Obese group	
	r	p	r	p	r	p
Age (yr)	-0.310	0.018	-0.032	0.904	-0.0159	0.9175
BMI (kg/m <sup>2</sup> )	-0.616	<0.0001	-0.583	0.014	-0.8102	<0.0001
Waist (cm)	-0.490	<0.0001	-0.353	0.165	-0.692	<0.0001
Hip (cm)	-0.492	<0.0001	-0.549	0.022	-0.718	<0.0001
WH Ratio	-0.041	0.758	0.0204	0.938	-0.212	0.162

## Discussion

Several observations from rodent studies support the hypothesis that ghrelin is a physiological meal initiator. First, ghrelin is synthesized primarily by the stomach (Kojima, M., *et al.*, 1999), an organ that is well positioned to sense short-term fluxes in energy balance. Second, despite being produced peripherally, ghrelin acts centrally to stimulate food intake (Wren, A. M., *et al.*, 2000). Third, ghrelin affects feeding rapidly, increasing both food intake (Asakawa, A., *et al.*, 2001) and gastric acid secretion within 20 min of intraperitoneal injection (Masuda, Y., *et al.*, 2000). Fourth, exogenous ghrelin triggers eating in rodents during the day (Wren, A. M., *et al.*, 2000; Asakawa, A., *et al.*, 2001; Nakazato, M., *et al.*, 2001), a time when food intake is usually nominal. Finally, ghrelin activates hypothalamic neuropeptide Y/Agouti-related peptide neurons (NPY/AGRP) and increases *AGRP* gene expression. *AGRP* has been implicated as a central mediator of meal initiation because mRNA levels in the hypothalamus rise shortly before the onset of maximal daily food intake in *ad libitum*-fed rats, whereas levels of other neuropeptides involved

in energy balance are stable throughout the day (Watson, S. J., *et al.*, 1999). In this study, we measured the plasma ghrelin levels in 122 Saudi females and investigated the associated factors. Our data show that, in lean subjects fasting plasma ghrelin levels were shown to rise nearly twofold before meal and fall to trough levels within 1 h after eating. The finding that preprandial rise and postprandial fall in plasma ghrelin levels has been previously demonstrated (Shiyya, T., *et al.*, 2002; Vicennati, V., *et al.*, 2007), and supports the hypothesis that ghrelin plays a physiological role in meal initiation in humans.

In this study, BMI were inversely correlated with fasting plasma ghrelin levels. This confirmed the view that ghrelin is closely associated with obesity. Ghrelin has been considered as a cause of obesity in some studies, on account of the fact that ghrelin has orexigenic effects in both rats and humans (Haqq, A. M., *et al.*, 2003; Tang-Christensen, M., *et al.*, 2004). However, the results of most studies in humans, similar to ours, have demonstrated that ghrelin levels are negatively correlated with BMI (Tritos, N. A., *et al.*, 2003; Baldelli, R., *et al.*, 2006; Zou, C. C., *et al.*, 2008).

Several studies have reported that fasting plasma ghrelin is reduced in obese subjects as compared to lean controls (Hansen, T. K., *et al.*, 2002; Broglio, F., *et al.*, 2004; Suematsu, M., *et al.*, 2005). Our data show that serum ghrelin levels are significantly decreased in obese and overweight subjects and remain decreased after meal as compared with BMI-matched controls. The absence of this fall in obese and overweight subjects could demonstrate impaired suppression of the drive to eat following a meal in obese subjects leading to increased food consumption and weight gain.

Currently factors thought to inhibit ghrelin secretion include leptin, interleukin-1 $\beta$ , GH and high fat diet (Shintani, M., *et al.*, 2001; Lee, H. M., *et al.*, 2002). Stimulatory factors appear to be fasting and a low protein diet. These factors may explain why ghrelin is suppressed in obese subjects but do not explain the dynamic response of ghrelin to eating, why concentration of peptide that stimulates gastric emptying (Vicennati, V., *et al.*, 2007) fall after food ingestion or why these responses are altered in obese individuals. Zou *et al.*, 2008 (Zou, C. C., *et al.*, 2008) speculate that the lower ghrelin levels in obesity are part of negative feedback to inhibit appetite and body weight, but not the primary cause of obesity (McLaughlin, T., *et al.*, 2004). This is also supported by the fact that circulating ghrelin levels increase in anorexia and cachexia (Misra, M., *et al.*, 2005; Janas-

Kozik, M., *et al.*, 2007). However, animals without ghrelin do not have significantly altered body weight or food intake when compared with their wild-type littermates (Sun, Y., *et al.*, 2003; Wortley, K. E., *et al.*, 2004). This suggests that it is a part of a reversible feedback mechanism, but not a determinant factor (Zou, C. C., *et al.*, 2008).

**In conclusion**, there are profound differences between ghrelin baseline concentration and dynamic responses to food intake in lean and obese subjects. The fall in plasma ghrelin concentration in lean subjects may represent suppression of a hunger signal. The lack of similar fall in obese subjects may indicate that ghrelin secretion is already maximally suppressed in this group, or a persistent orexigenic drive, failing to respond to food intake, that predisposes to obesity. Further studies are required to investigate the effect and the mechanism of ghrelin deficiency in individuals with obesity.

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## مستوى هرمون الغريلين في النساء السعوديات ذوات الوزن الطبيعي والوزن الزائد والبدينات قبل وبعد تناول وجبة الإفطار

مها حسن داغستاني

قسم علم الحيوان، كلية العلوم، جامعة الملك سعود، أقسام العلوم والدراسات الطبية

الرياض ص.ب. ٢٢٤٥٢، ١١٤٩٥، المملكة العربية السعودية

(قدم للنشر في ٢٤/٢/١٤٢٩هـ، وقبل للنشر في ٢٠/٥/١٤٢٩هـ)

الكلمات المفتاحية: هرمون الغريلين، البدانة، مؤشر كتلة الجسم، القياسات الجسدية.

ملخص البحث. يعتبر هرمون الغريلين ببتيد معدي معوي تم عزله من معدة الإنسان ومعدة الجرذ. يؤدي حقن هذا الهرمون في القوارض إلى تخفيز إفراز هرمون النمو وأيضاً إلى زيادة الوزن عن طريق زيادة الشهية وتراكم الدهون. تهدف الدراسة إلى تحديد مستوى هرمون الغريلين في حالاتي الصيام وبعد وجبة إفطار متوازنة، أيضاً إلى توضيح العلاقة بين مستوى هرمون الغريلين والقياسات الجسدية. شملت الدراسة ١٢٢ سيدة سعودية، تم تقسيمهن حسب مؤشر كتلة الجسم إلى ثلاث مجموعات كالتالي: ذوات الوزن الطبيعي، ذوات الوزن الزائد والبدينات. تم حساب مؤشر كتلة الجسم والقياسات الجسدية وتقدير تركيز الغريلين في الدم قبل وبعد تناول وجبة الإفطار بساعة واحدة لجميع عينات البحث. أوضحت النتائج ارتفاع مستوى هرمون الغريلين ارتفاعاً ذي دلالة إحصائية في النساء ذوات الوزن الطبيعي في حالة الصيام عند مقارنتهن بالنساء البدينات حيث كان مستوى الهرمون منخفضاً لديهن. كما أنخفض مستوى هرمون الغريلين انخفاضاً ذا دلالة إحصائية في النساء ذوات الوزن الطبيعي وانخفاضاً بسيطاً غير معنوي في النساء البدينات وذوات الوزن الزائد بعد تناول وجبة الإفطار، كما ارتبط هرمون الغريلين عكسياً مع مؤشر كتلة الجسم في المجموعات الثلاث. خلصت الدراسة إلى أن انخفاض مستوى الهرمون في الدم وإنعدام التثبيط الغذائي لمستواه قد يؤدي إلى زيادة الشهية لدى البدينات مما قد يرجح دوره في المسار الفسيولوجي المرضي للسمنة.