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Ghrelin response to protein and carbohydrate meals in relation to food intake and glycerol levels in obese subjects

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Abstract

Obese subjects have lower basal and an attenuated decrease of postprandial plasma ghrelin following carbohydrate-rich meals, while the response to protein is unknown. Therefore, plasma ghrelin levels were examined after ingestion of satiating amounts of a protein- or carbohydrate-rich meal in relation to food and energy intake and hunger/satiety ratings in 30 obese subjects followed 240 min later by ad lib sandwiches. Food intake and hunger/satiety ratings were identical while energy intake was significantly greater after bread (861 ± 62.7 vs. 441 ± 50.4 kcal, $p < 0.001$). Second meal food and energy intake were not different. Ghrelin decreased after bread, but increased by 50 pg/ml ($p < 0.001$) after meat. The corresponding increase of insulin was 55 vs. 9 μU/ml ($p < 0.001$). Glycerol levels decreased significantly less after the protein meal compared to carbohydrates. After protein glycerol was significantly correlated to the rise of ghrelin but not insulin. These data demonstrate that, in obese subjects, protein has no different satiating effect than carbohydrate despite divergent ghrelin levels. Energy intake corresponds to energy density of the respective food items. Ghrelin response to both meals is qualitatively similar but quantitatively attenuated compared to normal weight subjects. The relationship between ghrelin and glycerol would support recent observations of a possible role of ghrelin in fat metabolism.
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1. Introduction

Satiety signals are activated by gastric filling with subsequent distension of the gastric wall and they are conveyed to the hypothalamic areas of feeding regulation mainly by vagal afferents [1–7], which can be modified by cognitive and sensory mechanisms (for review, see [8–10]). As the macronutrient content of a meal is able to decrease the threshold for distension-induced satiety signals [5] and total gastric denervation did not completely abolish the regulation of feeding behaviour, it has long been assumed that there must be an additional hormonal pathway [11–13]. The recently discovered gastric hormone ghrelin [14,15] is a good candidate, since it has been shown to stimulate food intake in rodents and humans [15–19]. Application of a ghrelin receptor blocker attenuates food intake and weight gain in mice [20] indicating that endogenous ghrelin contributes to feeding regulation.

Following the ingestion of carbohydrate-rich meals, plasma ghrelin levels decrease to a nadir at 60–90 min returning to baseline thereafter [21–23]. While it remains rather uncertain that the initial decrease of the endocrine orexigenic tone supports neurally mediated satiety signals [23], ghrelin's subsequent increase could initiate the recurrence of appetite and feeding [21]. Such a concept is supported by several studies, which show a good correlation between plasma ghrelin and corresponding food intake [23,24] or hunger scores, respectively [25].

Since termination of feeding is largely determined by meal volume, energy dense food items facilitate a greater energy intake [26–31]. Accordingly, therapeutic modifications of eating behaviour of obese subjects attempt to reduce the intake of fat, which has the greatest energy density of the three macronutrients and low fat diets have been shown to be successful [32]. Both

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carbohydrate and protein have an identical lower energy density. For weight reduction regimens, several aspects favour a moderate restriction of carbohydrates in conjunction with an increase of the protein content of a low-fat hypocaloric diet. About one-third of obese subjects compensate fat reduction by carbohydrate overconsumption in long-term weight reduction programs [33,34]. Moreover, a greater carbohydrate content of a hypocaloric diet dramatically impairs weight reduction in grossly obese type 2 diabetic subjects [35]. The satiating effect of protein is a matter of debate. Some studies show a greater effect in comparison to the other macronutrients [29,36–39], although it must be kept in mind that meal size was higher in favour of protein in some studies [40], while others do not support this concept [23,29,41–43] including a small group of overweight subjects [44]. Despite this uncertain role of protein in feeding regulation, there is no evidence that excess protein consumption is responsible for overfeeding and weight gain. In normal weight subjects carbohydrate and protein-rich meals affect ghrelin release in opposite directions [21–23]. In obese subjects, basal ghrelin is lower and the postprandial response to a carbohydrate-rich meal is attenuated indicating a substantial difference of ghrelin regulation [45–47]. Therefore, it was of interest to examine in obese subjects (1) the as yet unknown effect of a protein-rich meal on ghrelin release and (2) the relationship between the ghrelin response and feeding parameters following the ingestion of carbohydrate and protein meals.

In addition, glycerol levels were determined as an index of lipolysis to examine if any correlation exists between lipolysis and ghrelin levels since recent animal and in vitro studies support the notion that ghrelin has an additional anabolic action via inhibition of lipolysis or stimulation of adipogenesis, respectively [48–50].

2. Patients and methods

The experiments were performed in 30 obese subjects (10 male, 20 female, age 37.7±0.48 years, BMI 35.6±1.5 kg/m^2). Patient characteristics are given in Table 1. Subjects who reported to have neither satiety nor hunger sensations were excluded. None of the subjects had signs or symptoms of an acute or chronic disease or was taking any medication. Subjects with diabetes mellitus were excluded. After informed consent was obtained, all examinations were performed according to the guidelines of the ethical committee of the Technical University of Munich and in accordance to the principles of the Declaration of Helsinki.

Table 1
Demographic data of the study population (mean±S.E.M.)

n (m/f)	30 (10/20)
Age (years)	38±0.48
BMI (kg/m^2)	35.6±1.5
Height (m)	1.72±0.02
Weight (kg)	112±4.5
Waist (cm)	111±5.8
Hip (cm)	122±3.5
W/H	0.91±0.2
Systolic BP (mm Hg)	128±2.3
Diastolic BP (mm Hg)	81±2.8
Leptin (ng/ml)	25.5±2.97
HOMA	1.8±0.5
BMR (kcal/24 h)	1606±88.7

2.1. Experimental design

All subjects were instructed to consume a weight-maintaining diet containing 50% carbohydrate, 20% protein and 30% fat (energy percent) at least 2 weeks prior to and throughout the study period. All subjects were asked to refrain from smoking and alcohol consumption.

All experiments started at 8.00 a.m., after a 12-h overnight fast. An indwelling catheter was inserted into a forearm vein for collection of blood samples.

On two separate occasions, each subject received in random order one of two test meals with at least 5 days interval. They were asked to eat as much of the meal until feeling comfortably satiated: (1) a carbohydrate-rich meal consisting of bread (2.5 kcal/g, energy percent: 79.7% carbohydrate, 12.4%protein and 7.9%fat) and (2) a protein-rich meal of lean pork meat (1.2 kcal/g, 0% carbohydrate, 83%protein and 17%fat). Ratings of subjective feelings of hunger and satiety were made on 100-mm visual analogue scales (VAS) before and in 15-min intervals after starting meal consumption as described previously [23]. After 240 min, all subjects were offered standardized sandwiches consisting of bread, butter and ham (2.73 kcal/g, energy percent: 44.4% carbohydrate, 16.2% protein and 39.4% fat), which they had to eat until reaching satiety as before. This second meal was intended to examine subsequent food intake in relation to the preceding degree of hunger and satiety feelings and preprandial plasma ghrelin levels. 2 days prior to the feeding tests, the subjects were asked to rate the pleasantness of the test meals on a score between 1 (totally unpleasant) and 10 (very pleasant). Subjects who assigned a rating of 6 or less to at least one test meal or who had a difference of 3 or more between test meals were excluded from the study.

Blood samples were taken at −15, 0, 15, 30, 45, 60, 90, 120, 150, 180, 210 and 240 min, after the second meal samples were taken at 255, 270, 285 and 300 min. The samples were collected into plastic tubes containing 1.2 mg EDTA and 500 kIU Trasylol for hormone analysis and into NaF-containing tubes for the determination of glucose. All samples were kept chilled in an ice bath until centrifugation at 2000 rpm for 15 min at 4 °C. The separated plasma was stored at −20 °C until the time of assay. All samples of one subject were run in duplicate in the same assay.

Plasma ghrelin levels were determined with a commercial radioimmunoassay which has been employed in previous studies [21,23] (Phoenix Pharmaceuticals, Belmont, CA). The assay uses ^{125}I-labeled bioactive ghrelin as a tracer molecule and a polyclonal antibody raised in rabbits against full-length octanylated human ghrelin, which detects both active and inactive ghrelin. Previous studies have shown a similar pattern of both molecular forms [51,52]. The interassay coefficient of variation was 10%. The intraassay coefficient of variation was 4%. No cross-reactivity was observed with gastrin, somatostatin,

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