


Effect of Meal Acceptability on Postprandial Appetite Scores and Hormones of Male Participants with Varied Adiposity

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Objective: This study portrays the effect of hedonic manipulation (high acceptability [HA] vs. low acceptability [LA]) on postprandial hormones and appetite scores in healthy males.

Methods: Thirty participants (15 with normal weight and 15 with obesity) were recruited for a randomized, crossover design. They were randomly assigned to the HA or LA (with acesulfame-K) custard. Blood samples were drawn before the meals and for 4 hours after the meals and were analyzed for glucose, insulin, ghrelin, and glucagonlike peptide 1 (GLP-1). Appetite scores and subsequent energy intake were recorded.

Results: Postprandial glucose, insulin, and ghrelin were different according to adiposity, whereas meal acceptability did not correspond to any significant difference in postprandial glucose, insulin, ghrelin, and GLP-1 concentrations. Appetite scores showed lower hunger, higher satiety, and fullness after the HA meal without a significant difference between the meals. Subsequent energy intake, expressed as a percentage of the resting energy expenditure, was higher in participants with obesity but did not reflect postprandial hormones and appetite scores; there was no significant difference between meals.

Conclusions: Hedonic properties and palatability do not affect gut hormones, mainly ghrelin and GLP-1. Moreover, their postprandial concentrations were not paralleled by similar changes in appetite scores, and both were not found to affect subsequent intake.

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Introduction

A food's sensory characteristics play a very important role in controlling energy intake within and across meals, and for the most part, palatability has been at the center of this (1). Several studies have pointed to the primary role of taste in directing food selection or preference (2). For instance, sweet foods are preferred, whereas bitter foods are avoided and considered indicative of toxicity or spoilage (3,4). Small changes in food taste, such as adding sweeteners, salts, herbs, and spices, can affect appetite and food intake (1). Before the food arrives in the gut, sensory and cognitive processes induced by sight, smell, and the experience of having food in the oral cavity affect not only the quantity eaten at the same meal but also the period after consumption (3). Many postingestive factors such as stomach distention, gastric emptying, and gut hormones (especially ghrelin and glucagonlike peptide 1 [GLP-1]) were reported to have a significant impact on appetite control (4). Ghrelin is the only orexigenic gut hormone and is described as the "hunger hormone" because its circulating levels increase during fasting to stimulate food consumption and then fall after a meal (5). GLP-1 is an intestinal hormone that is released into the circulation

after eating in a magnitude related to the amount of food consumed (6) and that decreases with fasting (7). The postabsorptive status or long-term satiety is known to be controlled by insulin, glucose, and amino acids (8). On the other hand, sweet and bitter receptors, which are expressed in the enteroendocrine cells of the small intestine, were reported to secrete GLP-1 in a dose- and time-dependent manner. Bitter tastes were reported to induce a strong GLP-1 release within 15 minutes because they are associated with harmful toxins (9).

The association between gut peptides and appetite scores remains unclear and weak. Some studies have shown no relation between appetite scores and glucose, insulin, or ghrelin concentrations (10), while others have found progressive significance between visual analogue scale scores and hormones and glucose levels (11,12). Another study showed similar results with GLP-1 concentrations mirroring satiety scores and ghrelin levels paralleling hunger ratings without affecting the energy intake at the subsequent meal (13).

On the other hand, the relationship between weight status and sweet, salty, sour, or bitter tastes is still unclear (14), though participants with

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obesity seem to be less sensitive to sweet and bitter tastes than participants with normal weight (15). In obesity, ghrelin levels are downregulated as an adaptation to positive energy balance (16,17), while GLP-1 response to food consumption is often blunted in participants with obesity compared with their lean counterparts (18,19). In lean participants, food acceptability was reported to alter ghrelin and insulin levels (20). The fact that responsiveness to different types of foods varies between lean participants and those with obesity (21) motivated us to compare the effect of food palatability on gut hormones, including ghrelin and GLP-1. The principal objectives of the proposed work were to investigate whether the hedonic properties of a meal affect postprandial insulin, ghrelin, and GLP-1 levels as well as appetite scores and energy intake at the subsequent meal. Moreover, the purpose is to have a better understanding of whether the trends observed in these variables differ between male lean participants and their counterparts with obesity.

Methods

Participants

Male participants (with normal weight, BMI 18.5–24.9 kg/m², and obesity, BMI 30–39.9 kg/m²) between 18 and 50 years old were enrolled in the study after signing a written informed consent. Exclusion criteria included smoking, alcohol or drug abuse, medical or psychological illness, previous gastrointestinal surgery, dietary restraint, and history of weight fluctuation (>5% in the last 3 months before the study). This study was approved by the Institutional Research Board of the American University of Beirut.

Experimental design

The study was composed of two main phases performed at the Department of Nutrition and Food Sciences of the American University of Beirut by the research team.

Phase 1. A total of 54 participants (24 with normal weight and 30 with obesity) were recruited to determine meal acceptability. Two equicaloric vanilla custard meals were prepared (Supporting Information Table S1) and were rated on a 9-point hedonic scale (22). The regular custard had high acceptability (HA), as confirmed in preliminary tests, while low acceptability (LA) was induced by the addition of 3.5 g of acesulfame-K (HYET Sweet, Gravelines, France), which causes excessive sweetness and bitterness without altering the nutrient composition. Acceptability tests were done using sensory analysis software (Compusense Cloud, Guelph, Ontario, Canada).

Participants with a reported acceptability score ≤ 5 for LA and ≥ 7 for HA ($n = 33$) were asked to fill out the Three Factor Eating Questionnaire R-18 (19). Those with high restraint, uncontrolled, and emotional eating scores (three participants) were excluded from the second stage of the study. Eligible participants were first scheduled for anthropometric and resting energy expenditure (REE) measurements using indirect calorimetry (Quark qCPET; COSMED, Rome, Italy) to calculate their caloric needs.

Phase 2. A random, double-blinded, crossover study was conducted in which baseline and postprandial measures of hormones and appetite scores were monitored for 240 minutes following the ingestion of one of the custard meals (HA or LA), after which another acceptability test was conducted, and the subsequent energy intake (ad libitum access to cheese pizza) was determined. Eligible participants (15 with

normal weight and 15 with obesity) were asked to avoid alcohol and maintain normal physical activity for 3 days before the experiment. Caloric content of the custard meal was designed to provide 30% of each of the participants' REE. Subjects consumed the full meal within 10 to 15 minutes and abstained from any other food item for the 4 hours post meal ingestion; only water consumption was allowed with a maximum amount of 500 mL.

On each experimental session, 12-hour fasted participants were seated comfortably, an intravenous catheter was inserted into an antecubital vein, and the needle was kept in their arm until all samples were collected. A registered nurse withdrew blood samples before the meal (time 0) and at time 15, 30, 60, 120, 180, and 240 minutes after meal ingestion. Participants were asked to complete the appetite score visual analog scale (23) within 5 minutes after each blood withdrawal.

Blood was aliquoted into the following three separate tubes: one EDTA tube with dipeptidyl peptidase-4 (Merck KGaA, Darmstadt, Germany) for GLP-1, one EDTA tube with Pefabloc SC (4-(2-aminoethyl)-benzoylsulfonylfluoride-hydrochloride) (Merck KGaA) for ghrelin, and one serum separator tube with clot activator for insulin and glucose.

Blood analysis for GLP-1, ghrelin, and insulin was performed using commercial enzyme-linked immunosorbent assay (ELISA) kits (EMD Millipore Corp., St. Charles, Missouri). Serum glucose levels were also determined using commercial enzymatic colorimetric tests on a Vitros analyzer (Ektachem DT60 II System; Johnson & Johnson Clinical Diagnostics, Rochester, New York). At the end of each session (240 minutes), participants had ad libitum access to a standard recipe of cheese pizza (3.01 kcal/g) and were asked to eat until they felt "comfortably full." The weight of consumed food was recorded.

Statistical analyses

For the phase 2 experiment, the sample size ($n = 15$) was calculated utilizing our previous ghrelin data (24), using a SD of 8 for the population and a minimal detectable difference in means of 10 with a statistical power of 80% and a significance level of 5%. Unpaired *t* test analysis was used for comparing between baseline characteristics of participants. Paired *t* test analysis was used to analyze the differences within groups following the ingestion of HA or LA meals and for the subsequent energy intakes. A repeated measures ANOVA (general linear model), using time as a random factor, was performed to assess the effect of different predictor variables on the levels of appetite scores, gut hormones (ghrelin, GLP-1, insulin), and glucose. Each participant underwent all the treatments and served as his own control. A separate ANOVA was performed for each hormone. The area under the curve (AUC) for glucose and the hormones was calculated from 0 to 120 minutes using the trapezoid method (GraphPad Prism 7.04; GraphPad Software, San Diego, California). Statistical analyses were performed using SAS statistical software (version 9.02; SAS Institute Inc., Cary, North Carolina). In the statistical model, the response variable was the hormonal level. Each of the main effects and all two-way interactions were tested.

Furthermore, the same analysis was conducted for the appetite scores, whereby each appetite score was the dependent variable. The discrete variables were acceptability level (LA and HA), adiposity (lean participants and those with obesity), and their interactions. Significance was preestablished at $\alpha < 0.05$.

Results

A total of 54 participants were recruited for the acceptability test; 94.5% ($n=51$) responded with a score ≥ 7 for the HA custard, while 67% ($n=36$) responded with a score ≤ 5 for the LA custard. When both scores were combined ($HA \geq 7$ and $LA \leq 5$), 33 participants (61%) were found to conform to the first acceptability test, but 3 participants were excluded for having high restraint, uncontrolled, and emotional eating scores. Thus, a total of 30 participants (15 lean and 15 with obesity) enrolled in the second stage of the study (Figure 1).

The baseline characteristics of the participants enrolled in the postprandial measure experiments are summarized in Table 1. Participants were of similar age and height. Baseline glucose and GLP-1 levels were similar between the groups, while participants with obesity had lower baseline ghrelin concentrations and higher baseline insulin and homeostatic model assessment levels compared with lean participants. The second acceptability test, during each session, showed similar results when compared with the first test. There were no significant differences between the acceptability ratings of lean participants and those with obesity. Following meal ingestion, plasma glucose levels were found to peak at 30 minutes and decrease after that to below baseline level from time 120 minutes. Participants with obesity had a delayed response compared with the normal weight participants, while plasma glucose was not affected by meal acceptability. Consequently, glucose levels were found to be significantly different according to adiposity ($P=0.001$) but not meal acceptability or the interaction of these two variables (Figure 2).

Insulin levels after meal ingestion followed a similar pattern to that of glucose but with a higher magnitude. Participants with obesity were found to have higher levels starting from time 15 until 120 minutes, and thus insulin levels were highly significant according to adiposity ($P=0.000$) (Figure 2). Conversely, meal acceptability failed to affect postprandial insulin significantly. Additionally, insulin AUC_{0-120} was positively correlated with that of glucose AUC_{0-120} ($r=0.267$, $P=0.039$) (Table 2). Absolute AUC values of glucose, insulin, ghrelin, and GLP-1 are summarized in Supporting Information Table S2.

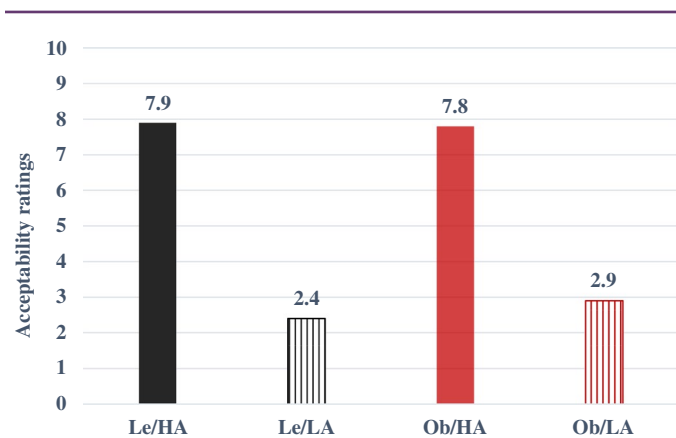


Figure 1 Ratings of the inclusion acceptability test for those who completed the study. All values are presented as averages of the ratings on the 9-point hedonic scale. Le, lean participants; Ob, participants with obesity; HA, high acceptability meal; LA, low acceptability meal. [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 1 Participant characteristics

	Lean participants (n = 15)	Participants with obesity (n = 15)	Unpaired t test, P value
Age (y)	20.1 ± 0.4	21.7 ± 0.9	NS
Height (cm)	179.1 ± 1.7	179.1 ± 1.7	NS
Weight (kg)	70.9 ± 2.8	113.2 ± 4.3	0.000
BMI (kg/m ²)	22.0 ± 0.5	35.1 ± 1.0	0.000
Fasting ghrelin (pg/mL)	243.4 ± 36.3	161.3 ± 9.4	0.050
Fasting GLP-1 (pmol)	11.5 ± 5.2	13.2 ± 6.1	NS
Fasting insulin (μU/mL)	24.8 ± 6.0	53.0 ± 12.2	0.050
Fasting glucose (mg/dL)	81.6 ± 1.3	82.8 ± 2.2	NS
HOMA	5.53 ± 0.90	10.70 ± 1.80	0.018

Results are the mean of both visits and are reported as mean ± SE. Level of statistical significance set at $P < 0.05$. HOMA, homeostatic model assessment; GLP-1, glucagonlike peptide 1; NS, not significant.

Moreover, postprandial levels of ghrelin were found to decrease following meal ingestion, and this seems to be affected by adiposity but not by meal acceptability. The magnitude of drop was higher among lean participants compared with those with obesity ($P=0.000$) (Figure 2).

The magnitude of GLP-1 response to meal ingestion was minimal, and thus no significant differences were detected according to meal acceptability or adiposity. The effect of meal acceptability showed a significant effect on the AUC_{1-120} of insulin ($P=0.000$) and ghrelin ($P=0.009$) (Table 3).

On the other hand, the varied measures of appetite scores (hunger, satiety, fullness, prospective consumption, and desire for sweet, salty, savory, and fatty food consumption) over the whole experiment (Supporting Information Table S3) showed no differences between meals and adiposity (Table 4). Although hunger ratings were slightly different, according to adiposity, the difference did not reach significance. The LA meal caused a slight decrease in hunger ratings of participants with obesity in contrast to that in lean participants.

Satiety and fullness ratings mirrored each other; they were similar between meals and groups, and thus no significant differences were detected according to meal acceptability and adiposity. Both fullness and satiety increased after meal ingestion, peaked between times 15 and 30 minutes in all cases, and gradually decreased after that. Moreover, prospective consumption was found to be lower in participants with obesity compared with lean participants over the experimental period with no significant difference. The change in the desire for something sweet, salty, savory, and fatty was slightly lower in subjects with obesity compared with lean participants without reaching statistical significance, and this was not affected by meal acceptability. This result implies that participants with obesity had less desire to eat salty, savory, or fatty food following meal ingestion.

On the other hand, the energy intake at the *ad libitum* test meal, which consisted of cheese pizza, was expressed as a percentage of the REE of each participant. Subsequent intake was not affected by meal acceptability. Paired *t* test showed no significant difference between meals and adiposity (Figure 3). The correlation between the AUC of hormones and

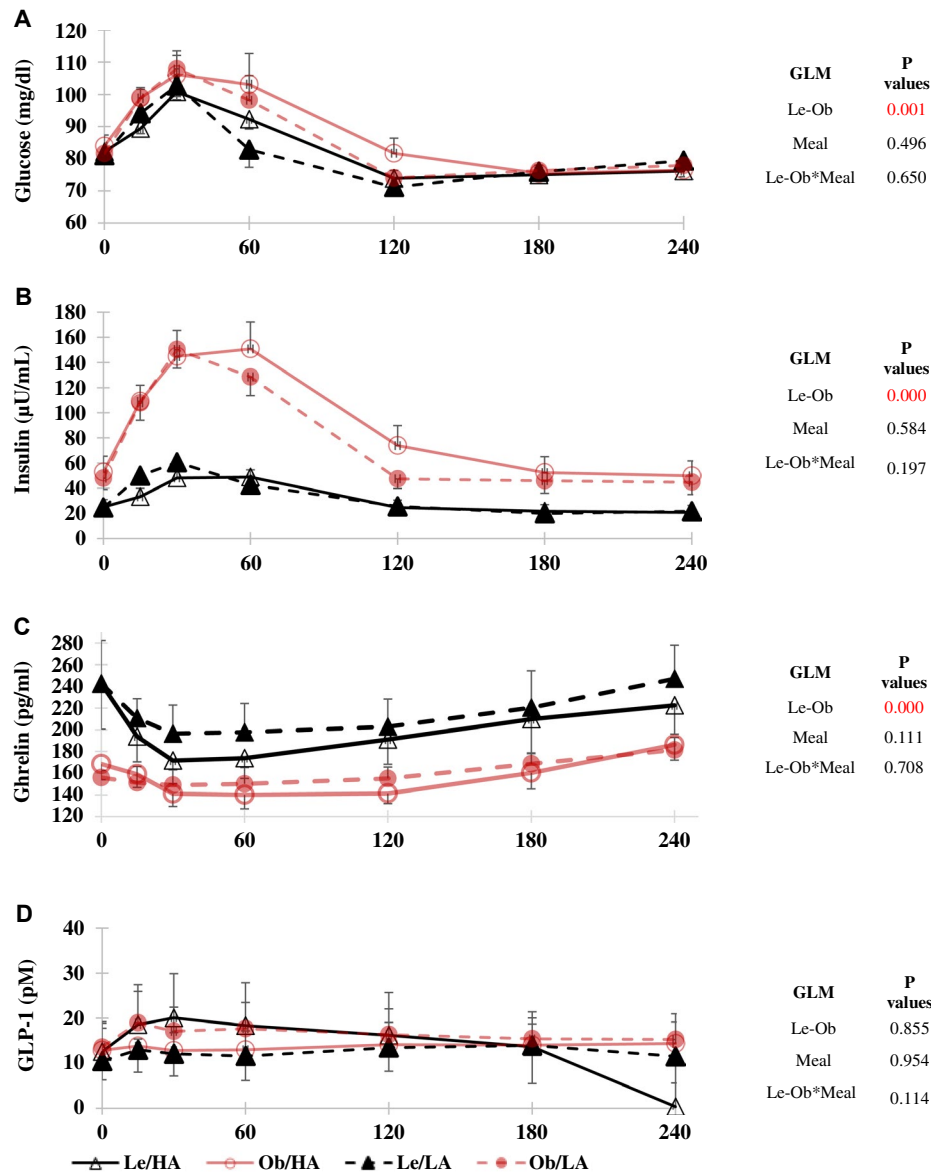


Figure 2 Glucose, insulin, ghrelin, and GLP-1 concentrations after the HA and LA meals in lean participants and those with obesity at different time points. Level of statistical significance was set at $P < 0.05$. GLP-1, glucagonlike peptide 1; Le, lean participants; Ob, participants with obesity; HA, high acceptability meal; LA, low acceptability meal. [Color figure can be viewed at wileyonlinelibrary.com]

subsequent energy intake was used to investigate the relationship between hormonal changes and subsequent energy intake. Results showed that glucose AUC_{0-120} was positively correlated with the percentage of subsequent energy intake ($r=0.0.272, P=0.036$), while AUC of other hormones failed to reach significance (Table 2).

Discussion

The objective of the study was to determine the effect of meal acceptability on postprandial measures of hormones and appetite scores, as well as subsequent intake. The acceptability of the meal was altered

by the addition of acesulfame-K to a custard meal, as confirmed by the significant reduction in acceptability ratings, and the magnitude of the reduction was found to be similar between lean participants and those with obesity. This is in line with previous observations, whereby the acceptability ratings, in terms of hedonic and sensory responses, decreased in a similar manner between lean participants and participants with obesity during a meal (25). It is worth noting that the inclination for lower appetite measures (prospective food consumption and desire for salty, savory, and fatty consumption) in participants with obesity was not paralleled by low subsequent intake, and this is probably due to the participants' tendency to underestimate the quantity they can eat (26). In fact, appetite scores are not a good predictor of energy intake (27,28).

TABLE 2 Pearson correlation between AUC of hormones and subsequent intake expressed as percentage of REE

	Ghrelin AUC ₀₋₁₂₀	GLP-1 AUC ₀₋₁₂₀	Insulin AUC ₀₋₁₂₀	Glucose AUC ₀₋₁₂₀
GLP-1: AUC₀₋₁₂₀				
<i>r</i>	0.235			
<i>P</i>	0.081			
Insulin: AUC₀₋₁₂₀				
<i>r</i>	-0.178	-0.178		
<i>P</i>	0.173	0.173		
Glucose: AUC₀₋₁₂₀				
<i>r</i>	0.004	0.004	0.004	
<i>P</i>	0.976	0.976	0.976	
Subsequent intake				
<i>r</i>	0.030	0.030	0.030	0.030
<i>P</i>	0.822	0.822	0.822	0.822

Level of statistical significance set at $P < 0.05$.

AUC, area under the curve; REE, resting energy expenditure; GLP-1, glucagonlike peptide 1.

TABLE 3 General linear model analysis of effect of meal acceptability and adiposity on AUC₀₋₁₂₀ of glucose, insulin, ghrelin, and GLP-1

	Le/Ob, <i>P</i> value	HA/LA, <i>P</i> value	Le/Ob × HA/LA, <i>P</i> value
Glucose	0.054	0.409	0.986
Insulin	0.000	0.682	0.528
Ghrelin	0.009	0.303	0.918
GLP-1	0.995	0.875	0.513

Level of statistical significance set at $P < 0.05$.

Le, lean participants; Ob, participants with obesity; HA, high acceptability meal; LA, low acceptability meal; AUC, area under the curve; GLP-1, glucagonlike peptide 1. Bold values are statistically significant.

At the metabolite level, the observed differences in postprandial glucose and insulin responses between lean participants and those with obesity indicate the presence of reduced insulin sensitivity in the latter, and this was in line with other studies (29). The failure of meal acceptability to affect glucose and insulin responses of both lean participants and those with obesity implies that hedonic factors do not control these parameters; it is mainly the taste. Therefore, the previously reported elevated insulin (30) and glucose (31) responses with higher palatability are likely to be related to the use of meals with varied texture or composition rather than palatability per se. In line with other studies (32), fasting ghrelin levels of participants with obesity were lower than those of lean participants, and the magnitude of the postprandial suppression was found to be higher among lean participants (17). Ghrelin levels of all groups went back to their fasting levels by the end of the experimental period (240 minutes). In line with other studies (33), participants with obesity had a blunted response after the LA meal. These results contradict those of Karhunen et al. (34) in which ghrelin concentrations were higher after the LA meal because they altered the palatability by

TABLE 4 General liner model analysis of appetite scores over experimental time (hunger, satiety, fullness, prospective consumption, and desire for sweet, salty, savory, and fatty food consumption)

	Le/Ob, <i>P</i> value	HA/LA, <i>P</i> value	Le/Ob × HA/ LA, <i>P</i> value
Hunger	0.298	0.262	0.235
Satiety	0.615	0.229	0.114
Fullness	0.540	0.789	0.142
PC	0.789	0.194	0.056
Sweet	0.545	0.161	0.542
Salty	0.169	0.448	0.832
Savory	0.052	0.537	0.956
Fatty	0.338	0.928	0.159

Level of statistical significance set at $P < 0.05$.

PC, prospective consumption; Le, lean participants; Ob, participants with obesity; HA, high acceptability meal; LA, low acceptability meal.

adding fiber. It is worth noting that the pattern of postprandial ghrelin and GLP-1 statuses was not interrelated. Although postprandial GLP-1 status was not statistically significant between the groups, the influence of meal acceptability tended to differ between lean participants and those with obesity. Under HA, GLP-1 changes in lean participants were higher than those with obesity, and this is in line with others (16,37), whereby the GLP-1 response to normal meal acceptability was reported to be reduced in participants with obesity compared with their lean counterparts (19). However, in lean participants, GLP-1 response to LA was reduced in contrast to that of participants with obesity.

Therefore, in participants with obesity, the similarity in postprandial insulin response following the varied meal acceptability implies that the changes in GLP-1 response were not related to insulin sensitivity, though humans are known to have a range of GLP-1 sensitivity and number of receptors (35) that are influenced by insulin sensitivity. Taste receptors, namely TAS1R and TAS2R for sweet and bitter stimuli, respectively, release GLP-1, which has a wide effect on glucose metabolism, including insulin secretion (36). In our study, the only difference between the meals was the taste. The HA meal was sweet, and the LA was excessively sweet and bitter. Thus, the blunted GLP-1 response after the HA meal in participants with obesity may be related to a decrease in TAS1R or low sensitivity to sweet taste, which is common in participants with obesity (37), although this failed to reach a significant difference.

On the other hand, the impact of meal acceptability on appetite measures remains unclear. Participants have previously reported that they were hungrier after a preferred meal (38), whereas hunger was found to be reduced after a palatable meal (39). This controversy is thought to be related to the use of meals with varied energy content, density, composition, etc. (40). Nonetheless, when we corrected for these variables by using meals of the same composition, volume, quantity, and energy density to avoid any effect, other than the taste on appetite scores, meal acceptability failed to affect appetite scores. Our findings are in line with other studies in which the addition of oregano or citric acid to modify the acceptability of meals by lean participants failed to affect appetite measures (41,42). A similar pattern of appetite scores did not parallel ghrelin and GLP-1 levels, and thus gastrointestinal hormones

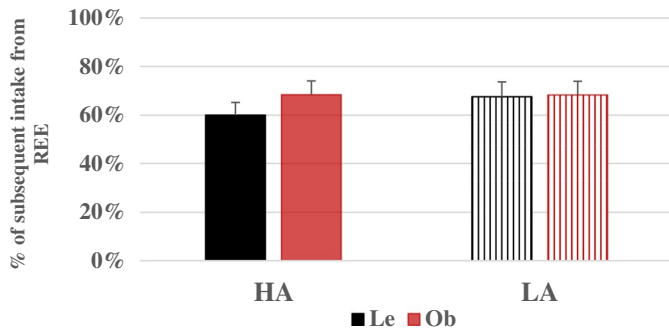


Figure 3 Subsequent intake of cheese pizza expressed as a percentage of the REE after the HA and LA preloads in both groups. All values are presented as percentages. Unpaired *t* test. Level of statistical significance was set at $P < 0.05$. REE, resting energy expenditure; Le, lean participants; Ob, participants with obesity; HA, high acceptability meal; LA, low acceptability meal. [Color figure can be viewed at wileyonlinelibrary.com]

involved in hunger and satiety regulation are not related to clinically assessed appetite scores (10).

Furthermore, subsequent energy intake was not influenced either by the changes in appetite scores and gut hormones (ghrelin and GLP-1) or by the preload's meal acceptability. Subsequent energy intake was highest among participants with obesity, and this is relative to the energy requirement of each. Subsequent energy intake was positively correlated to the magnitude of glucose levels.

Conclusion

Added acesulfame-K was found to reduce meal (custard) acceptability for both lean participants and those with obesity. Reduced meal acceptability did not alter the postprandial status of glucose, insulin, ghrelin, and GLP-1. Additionally, altered meal acceptability failed to affect appetite scores (hunger, satiety, fullness, prospective consumption, and desire for sweet, salty, savory, and fatty food consumption). Similar changes in appetite scores did not mimic gut hormones. Furthermore, subsequent energy intake was not dependent on the changes in appetite scores and gut hormones except for that of glucose. These findings imply that an alteration in meal acceptability does not seem to be translated into an alteration in subsequent energy intake. It is the composition, texture, and energy content of a certain food that can affect gut hormones, appetite scores, and subsequent energy intake.

The major limitation of the present study is that it assessed the response of acceptability manipulation in males only, and thus any gender differences may have been missed. Secondly, a larger number of participants would have provided more strength to the results, as some trends probably did not reach significance because of the number of participants. **O**

Acknowledgments

Data will be shared to an institutional repository (AUB Data Bank) and will be accessible for the public in 2 years.

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References

- McCrickerd K, Forde C. Sensory influences on food intake control: moving beyond palatability. *Obes Rev* 2016;17:18-29.
- Anguah KO-B, Lovejoy JC, Craig BA, et al. Can the palatability of healthy, satiety-promoting foods increase with repeated exposure during weight loss? *Foods* 2017;6:16. doi:10.3390/foods6020016
- Chambers L, McCrickerd K, Yeomans MR. Optimising foods for satiety. *Trends Food Sci Technol* 2015;41:149-160.
- Chambers L. Food texture and the satiety cascade. *Nutr Bull* 2016;41:277-282.
- Chen Y. Regulation of food intake and the development of anti-obesity drugs. *Drug Discov Ther* 2016;10:62-73.
- Dailey MJ, Moran TH. Glucagon-like peptide 1 and appetite. *Trends Endocrinol Metab* 2013;24:85-91.
- Cappadona SR. The Effects of Nighttime Protein Intake on Morning Appetite, Insulin and Ghrelin Levels in Overweight and Obese Men [master's thesis]. Tallahassee: Florida State University; 2016. http://purl.flvc.org/fsu/fd/FSU_2016SU_Cappadona_fsu_0071N_13083
- Amin T, Mercer JG. Hunger and satiety mechanisms and their potential exploitation in the regulation of food intake. *Curr Obes Rep* 2016;5:106-112.
- Geraedts MC, Troost FJ, Saris WH. Different tastants and low-calorie sweeteners induce differential effects on the release of satiety hormones. *Food Chem* 2011;129:731-738.
- Maffei C, Surano MG, Cordioli S, et al. A high-fat vs. a moderate-fat meal in obese boys: nutrient balance, appetite, and gastrointestinal hormone changes. *Obesity (Silver Spring)* 2010;18:449-455.
- Erdmann J, Hebeisen Y, Lippel F, Wagenpfeil S, Schusdziarra V. Food intake and plasma ghrelin response during potato-, rice- and pasta-rich test meals. *Eur J Nutr* 2007;46:196-203.
- Lemmens SG, Martens EA, Kester AD, Westerterp-Plantenga MS. Changes in gut hormone and glucose concentrations in relation to hunger and fullness. *Am J Clin Nutr* 2011;94:717-725.
- Lemmens SG, Martens EA, Born JM, Martens MJ, Westerterp-Plantenga MS. Staggered meal consumption facilitates appetite control without affecting postprandial energy intake. *J Nutr* 2011;141:482-488.
- Cox DN, Hendrie GA, Carty D. Sensitivity, hedonics and preferences for basic tastes and fat amongst adults and children of differing weight status: a comprehensive review. *Food Qual Prefer* 2016;48:359-367.
- Proserpio C, Laureati M, Bertoli S, Battezzati A, Pagliarini E. Determinants of obesity in Italian adults: the role of taste sensitivity, food liking, and food neophobia. *Chem Senses* 2015;41:169-176.
- Müller TD, Nogueiras R, Andermann ML, et al. Ghrelin. *Mol Metab* 2015;4:437-460.
- Tschöp M, Weyer C, Tataranni PA, et al. Circulating ghrelin levels are decreased in human obesity. *Diabetes* 2001;50:707-709.
- Meyer-Gerspach AC, Wölnerhanssen B, Beglinger B, et al. Gastric and intestinal satiation in obese and normal weight healthy people. *Physiol Behav* 2014;129:265-271.
- Anglé S, Engblom J, Eriksson T, et al. Three factor eating questionnaire-R18 as a measure of cognitive restraint, uncontrolled eating and emotional eating in a sample of young Finnish females. *Int J Behav Nutr Phys Act* 2009;6:41. doi:10.1186/1479-5868-6-41
- Olabi A, Hwalla N, Daroub H, Obeid O, Cordahi C. If it does not taste good, don't eat it! Food acceptability affects ghrelin and insulin levels in healthy male subjects. *A pilot study. Nutr Res* 2018;49:48-55.
- Stice E, Borczyk A, Menke K. Heritability of hyperresponsivity of brain reward regions to high-calorie food. *Am J Clin Nutr* 2016;103:299-300.
- Peryam DR, Pilgrim FJ. Hedonic scale method of measuring food preferences. *Food Technol* 1957;11:9-14.
- Flint A, Raben A, Blundell J, Astrup A. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord* 2000;24:38-48.
- Tannous dit El Khoury D, Obeid O, Azar ST, Hwalla N. Variations in postprandial ghrelin status following ingestion of high-carbohydrate, high-fat, and high-protein meals in males. *Ann Nutr Metab* 2006;50:260-269.
- Zhu Y, Hollis JH. Increasing the number of chews before swallowing reduces meal size in normal-weight, overweight, and obese adults. *J Acad Nutr Diet* 2014;114:926-931.
- Chandon P, Wansink B. Is obesity caused by calorie underestimation? A psychophysical model of meal size estimation. *J Mark Res* 2007;44:84-99.
- Harper A, James A, Flint A, Astrup A. Increased satiety after intake of a chocolate milk drink compared with a carbonated beverage, but no difference in subsequent ad libitum lunch intake. *Br J Nutr* 2007;97:579-583.
- Miller S, Petocz P. Interrelationships among postprandial satiety, glucose and insulin responses and changes in subsequent food intake. *Eur J Clin Nutr* 1996;50:788-797.
- Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature* 2006;444:840-846.
- Bellisle F, Drewnowski A, Anderson GH, Westerterp-Plantenga M, Martin CK. Sweetness, satiation, and satiety. *J Nutr* 2012;142:1149S-1154S.
- Sawaya AL, Fuss PJ, Dallal GE, et al. Meal palatability, substrate oxidation and blood glucose in young and older men. *Physiol Behav* 2001;72:5-12.
- Monteleone P, Bencivenga R, Longobardi N, Serritella C, Maj M. Differential responses of circulating ghrelin to high-fat or high-carbohydrate meal in healthy women. *J Clin Endocrinol Metab* 2003;88:5510-5514.
- Lindqvist A, de la Cour CD, Stegmark A, Håkanson R, Erlanson-Albertsson C. Overeating of palatable food is associated with blunted leptin and ghrelin responses. *Regul Pept* 2005;130:123-132.
- Karhunen LJ, Juvonen KR, Flander SM, et al. A psyllium fiber-enriched meal strongly attenuates postprandial gastrointestinal peptide release in healthy young adults. *J Nutr* 2010;140:737-744.

35. Aulinger BA, Vahl TP, Wilson-Pérez HE, Prigeon RL, D'aleccio DA. β -Cell sensitivity to GLP-1 in healthy humans is variable and proportional to insulin sensitivity. *J Clin Endocrinol Metab* 2015;100:2489-2496.
36. Dotson CD, Zhang L, Xu H, et al. Bitter taste receptors influence glucose homeostasis. *PLoS One* 2008;3:e3974. doi:10.1371/journal.pone.0003974
37. Janssen S, Depoortere I. Nutrient sensing in the gut: new roads to therapeutics? *Trends Endocrinol Metab* 2013;24:92-100.
38. Rogers PJ, Schutz HG. Influence of palatability on subsequent hunger and food intake: a retrospective replication. *Appetite* 1992;19:155-156.
39. Warwick ZS, Hall W, Pappas TN, Schiffman SS. Taste and smell sensations enhance the satiating effect of both a high-carbohydrate and a high-fat meal in humans. *Physiol Behav* 1993;53:553-563.
40. Tey SL, Chia EME, Forde CG. Impact of dose-response calorie reduction or supplementation of a covertly manipulated lunchtime meal on energy compensation. *Physiol Behav* 2016;165:15-21.
41. De Graaf C, De Jong LS, Lambers AC. Palatability affects satiation but not satiety. *Physiol Behav* 1999;66:681-688.
42. Yeomans MR. Palatability and the micro-structure of feeding in humans: the appetizer effect. *Appetite* 1996;27:119-133.