Hedonic hunger is increased in severely obese patients and is reduced after gastric bypass surgery[1–3]

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ABSTRACT

Background: Overeating as a pathogenetic hallmark of obesity may be promoted by an increase in hedonic hunger, ie, the drive to eat palatable foods in the absence of energy need. Gastric bypass surgery, which effectively reduces severe obesity, might also affect hedonic hunger.

Objective: The objective was to assess hedonic hunger in severely obese patients with and without a history of gastric bypass surgery.

Design: Severely obese patients who had not undergone gastric bypass surgery (n = 123), gastric bypass patients (n = 136), and nonobese control subjects (n = 110) were examined with the Power of Food Scale (PFS)—a questionnaire that reliably measures an individual's motivation to consume highly palatable foods.

Results: Compared with nonobese control subjects, severely obese patients achieved markedly higher aggregated PFS scores and subdomain scores related to generally available as well as physically present foods (all $P < 0.001$). On the aggregated score as well in those 2 subdomains, gastric bypass patients scored significantly lower than did non–gastric bypass obese patients (all $P < 0.001$) and did not differ significantly from the nonobese control group ($P > 0.2$). In contrast, in the PFS domain concerning food tasted, gastric bypass patients had significantly lower scores than did the nonobese control subjects ($P = 0.04$) and the severely obese patients ($P = 0.008$), both of whom did not differ significantly in this measure ($P = 0.90$).

Conclusion: In comparison with nonobese control subjects, severely obese patients display a marked increase in hedonic hunger that is not observed in patients who have undergone gastric bypass surgery, suggesting that the operation normalizes excessive appetite for palatable foods, which may be an important pathophysiologic feature of severe obesity. *Am J Clin Nutr* 2010;92:277–83.

INTRODUCTION

The regulation of hunger and food intake relies on a neuroendocrine network integrating central nervous pathways with signals from the periphery (1). In particular, intestinal hormones such as glucagon-like peptide 1 (GLP-1), peptide YY (PYY), and ghrelin act on hypothalamic pathways regulating energy homeostasis and eating behavior either via direct effects on central nervous structures or via neuronal afferences of the vagal nerve (2, 3). Because of the complex and highly integrated nature of this network, disturbances in the control of food intake and body weight are difficult to improve. Currently, an ever-growing number of severely obese patients (4) are effectively treated with bariatric surgery, particularly gastric bypass surgery (5), which induces malabsorptive effects and reductions in actual food intake (6). However, in addition to the restrictive component, the procedure may also curb food intake by dampening feelings of hunger (7, 8). This is suggested by observations of enhanced release of the satiating hormones GLP-1 and PYY after the operation (9). Also, circulating concentrations of the hunger-promoting hormone ghrelin have been found to be reduced after the operation in some (10–12) but not in all (13, 14) studies.

Overeating as a crucial factor in the pathogenesis of obesity may not only result from increased hunger per se. The experience of pleasure that is associated with the consumption of highly palatable foods as well as imaginative craving for such foods in the absence of current energy needs and of food stimuli, ie, a motivational factor that has recently been referred to as "hedonic hunger" (15), may be an even more important trigger of overeating. By way of conditioning, the reward-related dopamine release in mesolimbic brain structures caused by the ingestion of highly palatable foods can also be elicited by cues that predict food availability in the absence of actual food stimuli (16). Remarkably, the density of dopamine type 2 (D2) receptors in mesolimbic brain areas has been found to be markedly reduced in severely obese patients (17)—a pathophysiologic process that is characteristic of addictive behavior (18). Empirical data on hedonic hunger in the obese are scarce (15), which raises the question of whether these neurobiological alterations are paralleled by changes in hedonic eating motives. How gastric bypass surgery in obese patients affects the hedonic drive to eat has also not yet been explored.

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Reliable assessments of hedonic hunger can be achieved by means of the Power of Food Scale (PFS), a recently developed and validated questionnaire that measures an individual's hedonic appetite for highly palatable foods but not the actual consumption of such foods (19, 20). Using this measure, we tested the hypothesis that severely obese patients, in comparison with nonobese subjects, have an increased level of hedonic hunger. We also hypothesized that hedonic hunger is reduced in severely obese patients who have undergone gastric bypass surgery.

SUBJECTS AND METHODS

Three groups of subjects participated in the study: 110 nonobese control subjects [body mass index (BMI; in kg/m^2) of 18–27] recruited from the local community, 123 severely obese subjects (BMI $>$ 35) who attended our Interdisciplinary Obesity Center (IOC) for evaluation for bariatric surgery, and 136 patients who had undergone gastric bypass surgery (for details, *see* below) $\geq$1 y before participation and were taking part in a structured IOC follow-up program. Subjects taking drugs known to strongly affect eating behavior, such as psychotropic drugs, were excluded from the study. Written informed consent was obtained from all subjects, and the study protocol was approved by the cantonal ethic committee of St Gallen (Switzerland). Data were collected between September 2007 and September 2009.

Gastric bypass procedures

Of the 136 gastric bypass patients, 33 had undergone a standard proximal gastric bypass operation, whereas the remaining 103 patients had undergone a distal gastric bypass operation. In both the proximal and distal gastric bypass procedure, the largest part of the stomach was transected, thereby creating a small gastric pouch of 20 mL, which was anastomized to the proximal jejunum. The diameter of this pouch-jejunal anastomosis was standardized to 10 mm. In the proximal gastric bypass procedure, the biliopancreatic limb (duodenum and upper part of the proximal jejunum) was side-to-side anatomized to the jejunum 150 cm distal from the pouch-jejunal anastomosis, thereby creating a Roux-en-Y or alimentary limb. In the distal gastric bypass procedure, the biliopancreatic limb was side-to-side anatomized to the ileum 60–100 cm proximal from the Bauhin's valve, thereby establishing a rather short common channel. The length to the biliopancreatic limb, as measured from the ligament of Treitz, was ≈60 cm in the proximal and 60–100 cm in the distal gastric bypass procedure. Thus, our distal gastric bypass, in contrast with traditional biliopancreatic diversion procedures, holds a very long alimentary limb. The main difference between the 2 gastric bypass procedures is that the distal version induces stronger malabsorption, particularly of nutritional fats, whereas the restrictive component of both procedures is comparable.

Body weight

Body height and weight were measured in all subjects while they were wearing light clothes but no shoes. Percentage weight loss was calculated as {[preoperative weight (kg) – current weight]/[preoperative weight]} × 100, percentage excess weight loss (%EWL) as {[preoperative weight – current weight]/[preoperative weight – height (cm) + 100]} × 100, and percentage excess BMI loss (%EBL) was calculated as [(preoperative BMI – current BMI)/(preoperative BMI – 25)] × 100 (21).

Power of Food Scale

All subjects completed the German version of the PFS generously provided to us by the developers of the questionnaire (Michael Lowe, Department of Psychology, Drexel University, Philadelphia, PA) during a visit at our ICO. The PFS was designed to measure appetite for rather than consumption of palatable foods and thus does not include any items describing actual food consumption. It comprises 15 items reflecting the responsiveness to the food environment grouped into 3 domains according to food proximity: *1*) food readily available in the environment but not physically present ("food available"), *2*) food present but not tasted ("food present"), and *3*) food when first tasted but not consumed ("food tasted"). Examples of the 3 domains are, respectively, as follows: "I find myself thinking about food even when I'm not physically hungry"; "If I see or smell a food I like, I get a powerful urge to have some"; and "When I eat delicious food I focus a lot on how good it tastes." For each item, subjects had to score their reactions on a 5-level scale: 1 = I don't agree at all, 2 = I agree a little, 3 = I agree somewhat, 4 = I agree, and 5 = I strongly agree. Thus, the scores on each subdomain indicate hedonic hunger motivation at different levels of food availability, ie, ranging from implicitly knowing that food is generally available to the first tasting of food, but excluding actual food consumption. The mean of the items comprising each of the 3 domain scores was calculated to obtain an aggregated score. Although correlations between the 3 domains have been found to be generally high (all $r > 0.77$), thus supporting the use of an aggregated domain score, the 3-domain model has been found to be superior to the one domain model (19). The PFS displays high reliability, with corrected item-total correlation coefficients ranging from 0.50 to 0.73. Chronbach's α is 0.91, and the 4-mo test-retest reliability coefficient is $r = 0.77$. Translation of the PFS into German was performed by native speakers of German fluent in English (PharmaQuest Ltd, Banbury, Oxon, United Kingdom) and included linguistic validation by 2 forward and 2 back translations, review by the creators, and pilot testing in 5 lay people.

Structured interview in gastric bypass patients

In a subsample of 58 gastric bypass patients, we performed a structured clinical interview focusing on eating-related symptoms during a regular follow-up outpatient visit. In particular, patients rated their hunger/appetite as a trait on a 5-point scale (from 0 to 4). Postprandial pain and feelings of early fullness after starting to eat were assessed and categorized as "never," "sometimes," or "always." Vomiting, symptoms of "early dumping," such as crampy abdominal pain, bloating, and diarrhea as well as postprandial hypoglycemia-related symptoms (eg, sweating, shivering, and hunger) as indicators of "late dumping" were also assessed and categorized as "never," "seldom," "sometimes," "frequently," or "always occurring."

Statistical analyses

Data were analyzed by using SPSS 12.0 for Windows (SPSS Inc, Chicago, IL). Unless otherwise indicated, data are reported

TABLE 1
Characteristics of the subjects

	Nonobese subjects (n = 110)	Obese patients (n = 123)	Gastric bypass patients (n = 136)
Sex (female/male)	89/21	96/27	101/35
Age (y)	42.1 ± 10.6 (21–69)[1]	39.9 ± 12.0 (19–67)	41.4 ± 10.6 (20–64)
Current BMI (kg/m^2)	22.4 ± 2.1 (18.4–26.9)	45.1 ± 6.3[2] (35.4–66.6)	29.5 ± 4.7[2,3] (19.7–42.1)
Preoperative BMI (kg/m^2)	—	—	47.0 ± 5.7 (37.0–67.0)
Time since operation (mo)	—	—	22.5 ± 17.2 (12–132)

[1] Mean ± SD; range in parentheses (all such values).
[2] Significantly different from nonobese, $P < 0.001$ (Tukey's test).
[3] Significantly different from obese, $P < 0.001$ (Tukey's test).

as means ± SDs. Differences across groups were assessed with univariate analysis of variance for continuous variables or with chi-square tests for discrete variables. For pairwise comparisons post hoc Tukey tests and chi-square tests were used as appropriate. Associations between PFS scores and the characteristics of subjects were evaluated with Pearson's correlation coefficients and partial correlation analyses. A P value <0.05 was considered significant.

RESULTS

Characteristics of subjects

Characteristics of the subjects are presented in **Table 1**. By selection, the obese subjects had a higher BMI than did the nonobese participants and the gastric bypass patients ($P < 0.001$ for both comparisons). As a result of surgery, the gastric bypass patients had lost an average of 48.2 ± 14.1 kg (ie, 37.1 ± 7.8% of their initial body weight), corresponding to an average BMI reduction of 17.5 ± 4.7, but still had a higher BMI at the time of participation than did the nonobese control subjects (29.5 ± 4.7 compared with 22.4 ± 2.1; $P < 0.001$). Their mean preoperative BMI had been higher than that of the current obese group (47.0 ± 5.7 compared with 45.1 ± 6.3; $P = 0.013$). Sex and age did not differ significantly between groups ($P = 0.48$ and $P = 0.47$, respectively).

Group differences in PFS scores

Marked differences between the 3 study groups in all domains as well as in the aggregated domain score were indicated by analysis of variance models comprising all groups (all $P <$ 0.006). Obese subjects had a markedly higher aggregated PFS domain score than did the nonobese patients (2.8 ± 0.9 compared with 2.3 ± 0.7; $P < 0.001$, **Figure 1**). The gastric bypass patients had a significantly lower aggregated score (2.2 ± 0.7) than did the non–gastric bypass obese patients ($P < 0.001$) and did not differ from the nonobese control subjects ($P = 0.40$).

Separate analyses of the domain scores "food available" and "food present" showed a similar pattern, ie, obese patients had significantly higher scores than did the nonobese group (2.6 ± 1.0 compared with 1.9 ± 0.8 and 3.0 ± 1.0 compared with 2.3 ± 0.9, respectively; both $P < 0.001$) and the gastric bypass group (2.0 ± 0.7 and 2.1 ± 0.9; both $P < 0.001$). Compared with the nonobese control group, gastric bypass patients had comparable values on the "food available" ($P = 0.80$) and the

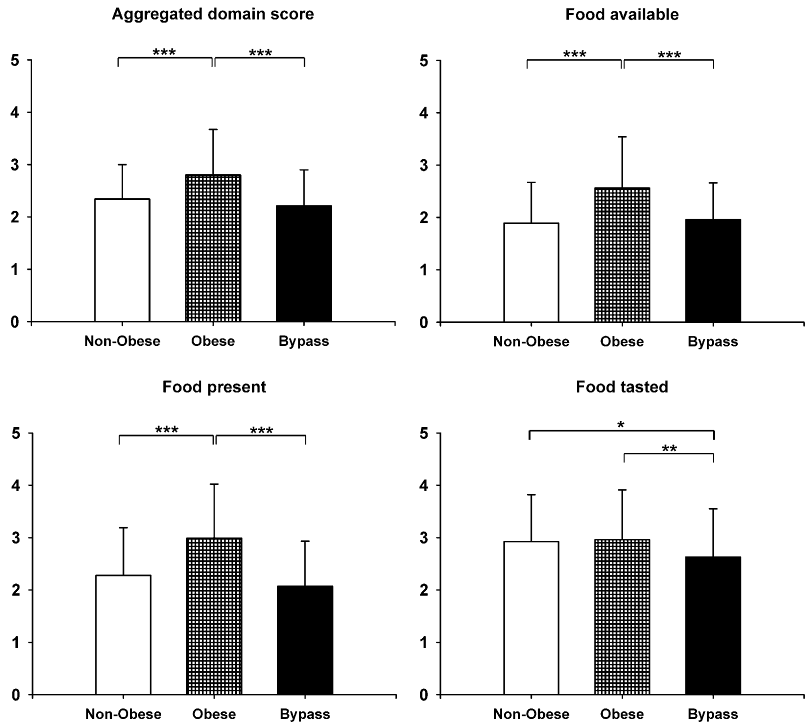

FIGURE 1. Mean (±SD) aggregated Power of Food Scale (PFS) score and mean (±SD) scores of the PFS subdomains "food available" (regarding food readily available in the environment but not physically present), "food present" (regarding food present but not tasted), and "food tasted" (regarding food when first tasted but not consumed) in 110 nonobese control subjects, 123 severely obese patients, and 136 gastric bypass patients. ******Univariate ANOVA: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

"food present" domain ($P = 0.20$). The "food tasted" score did not differ between obese and nonobese subjects (3.0 ± 1.0 and 2.9 ± 0.9; $P = 0.90$), but was lower in the gastric bypass patients than in the non–gastric bypass obese patients (2.6 ± 0.9; $P = 0.008$) and the nonobese control subjects ($P = 0.04$). None of the PFS scores differed significantly between men and women in any of the 3 groups (all $P > 0.09$). In the gastric bypass patients, there were no differences between patients who had undergone proximal as compared with distal gastric bypass operations (all $P > 0.41$).

Correlational analyses

Results of correlational analyses between PFS scores and subject characteristics are summarized in **Table 2**. Correlations between the 3 PFS domain scores and current BMI, as well as age, did not reach significance in any of the 3 subject groups (all $P > 0.06$). In the gastric bypass patients, the domain scores "food available" and "food present" were inversely correlated with %EBL (both $P = 0.044$). PFS scores (domains and aggregated) were not significantly related to the time elapsed since the gastric bypass operation, which was also the case after adjustment for %weight loss, %EWL, or %EBL (all $r < 0.07$, $P > 0.48$).

Postoperational eating-related symptoms in a subgroup of gastric bypass patients

The subgroup of gastric bypass patients who participated in the structured interview did not differ significantly in age (42.9 ± 10.9 compared with 40.2 ± 10.4 y; $P = 0.15$), sex (71.9% compared with 75.6% women; $P = 0.63$), initial BMI (47.2 ± 5.9 compared with 46.8 ± 5.7; $P = 0.67$), and current BMI (29.9 ± 4.6 compared with 29.2 ± 4.8; $P = 0.45$) from the remaining gastric bypass patients. Average time elapsed since the operation tended to be slightly shorter in the interviewed than in the noninterviewed patients (19.4 ± 12.7 compared with 24.9 ± 19.8 mo; $P = 0.07$). The "food available" (2.0 ± 0.7 compared with 2.0 ± 0.7; $P = 0.96$), "food present" (2.1 ± 0.9 compared with 2.1 ± 0.9; $P = 0.80$), and "food tasted" (2.5 ± 1.0 compared with 2.2 ± 0.7; $P = 0.14$) scores as well as the aggregated domain score (2.2 ± 0.7 compared with 2.2 ± 0.7; $P = 0.56$) did not differ between the interviewed and noninterviewed patients.

Neither hunger nor appetite ratings assessed in the structured interview correlated with any of the PFS scores (all $r < 0.11$, $P > 0.4$). As shown in **Table 3**, "early dumping" was the only symptom significantly related to PFS scores. Patients who never experienced early dumping symptoms had the highest scores on the "food present" ($P = 0.043$) and the "food tasted" ($P = 0.017$) domains.

DISCUSSION

Compared with nonobese subjects, severely obese patients have a greater drive to consume hedonically salient and palatable foods, as evidenced by means of the PFS that comprises 15 items reflecting the responsiveness to the food environment. This increase in hedonic hunger particularly pertains to PFS domains that concern generally available and acutely present foods, whereas the taste component is not affected. Patients after gastric bypass surgery have distinctly lower values in all PFS scores as compared with the non–gastric bypass obese patients and have values that are comparable to or even lower than those obtained in nonobese controls, even though most of these patients at the time of the study were still overweight or obese.

The PFS, which measures the hedonic appetitive drive to eat, does not permit any conclusion on actual food intake. PFS validation studies (19, 20) showed modest correlations (approximately −0.16) between PFS subdomains and cognitive restraint assessed with the Three Factor Eating Questionnaire (TFEQ) (22) and with self-reported restraint ($r = 0.30$) measured with the Restraint Scale (23). Subdomain scores as well as the aggregated domain score of the PFS significantly correlated with uncontrolled eating and emotional eating in the revised TFEQ, but with correlation coefficients not exceeding 0.70 (20). Multivariate regression analyses (19) showed the aggregated PFS score to be a significant predictor of

TABLE 2
Relation between characteristics of the subjects and Power of Food Scale scores[1]

	Food available	Food present	Food tasted	Aggregated domain score
Nonobese control group				
Age	0.03	0.03	0.08	0.05
BMI	0.13	0.17	0.14	0.16
Obese control group				
Age	−0.14	−0.12	−0.06	−0.14
BMI	0.05	−0.02	−0.04	−0.00
Gastric bypass group				
Age	−0.02	−0.05	−0.08	−0.06
Preoperative BMI	0.11	0.03	−0.00	0.05
Current BMI	0.17	0.13	−0.00	0.11
Weight loss (%)	−0.11	−0.15	0.01	−0.09
Excess weight loss (%)	−0.17	−0.17	0.01	−0.12
Excess BMI loss (%)	−0.18[2]	−0.17[2]	0.01	−0.12
Time since gastric bypass operation (mo)	0.06	0.04	0.03	0.05

[1] Pearson's correlation coefficients between the 3 Power of Food Scale domain scores (food available, food present, and food tasted) as well as the aggregated domain score and age and current and preoperative BMI in 110 nonobese control subjects, 123 severely obese subjects, and 136 gastric bypass patients.

[2] $P < 0.05$.

TABLE 3
Power of Food Scale scores according to postoperative symptoms in gastric bypass patients[1]

Symptom[2]	Food available	Food present	Food tasted	Aggregated domain score
Postprandial pain				
None (82.5%)	1.90 ± 0.67[3]	2.13 ± 0.87	2.50 ± 0.98	2.16 ± 0.72
Sometimes (17.5%)	2.18 ± 1.02	1.93 ± 1.03	2.50 ± 0.82	2.22 ± 0.77
P value	0.281	0.507	0.995	0.823
Early fullness				
Never (23.2%)	2.09 ± 0.65	2.42 ± 1.02	2.80 ± 0.90	2.42 ± 0.71
Sometimes (41.1%)	1.87 ± 0.74	1.96 ± 0.85	2.47 ± 0.95	2.09 ± 0.69
Always (35.7%)	1.93 ± 0.81	1.96 ± 0.77	2.35 ± 1.00	2.08 ± 0.76
P value	0.694	0.245	0.415	0.357
Vomiting				
Never (81.5%)	2.03 ± 0.67	2.22 ± 0.86	2.63 ± 0.94	2.28 ± 0.67
Seldom (18.5%)	1.77 ± 1.02	1.78 ± 1.02	2.08 ± 0.99	1.87 ± 0.91
P value	0.310	0.164	0.103	0.108
Early dumping symptoms				
Never (58.9%)	2.10 ± 0.74	2.36 ± 0.94	2.82 ± 0.88	2.41 ± 0.68
Seldom (28.6%)	1.80 ± 0.83	1.72 ± 0.77	2.06 ± 0.90	1.87 ± 0.73
Sometimes (7.1%)	1.54 ± 0.16	1.63 ± 0.32	1.65 ± 0.30	1.60 ± 0.16
Frequently (5.4%)	1.61 ± 0.35	1.58 ± 0.38	2.20 ± 1.25	1.80 ± 0.52
P value	0.305	0.043	0.011	0.017
Late dumping symptoms				
Never (36.8%)	1.87 ± 0.63	1.96 ± 0.78	2.62 ± 1.02	2.14 ± 0.69
Seldom (40.4%)	2.07 ± 0.84	2.24 ± 1.04	2.38 ± 0.94	2.22 ± 0.80
Sometimes (15.8%)	1.76 ± 0.65	1.86 ± 0.70	2.16 ± 0.75	1.92 ± 0.57
Frequently (7.0%)	2.21 ± 0.91	2.50 ± 0.94	3.30 ± 0.62	2.65 ± 0.68
P value	0.314	0.352	0.323	0.369

[1] Power of Food Scale (PFS) domain scores (food available, food present, and food tasted) and aggregated PFS domain scores according to eating-related symptoms assessed in 58 gastric bypass patients during a standardized clinical interview. *P* values were derived by univariate ANOVA.
[2] Percentages in parentheses indicate the relative proportion of patients experiencing the respective symptom in the respective frequency.
[3] Mean ± SD (all such values).

disinhibition and hunger on the TFEQ and of emotional eating and external eating on the Dutch Eating Behavior Questionnaire (24) after control for self-reported restraint. These results suggest that the subscales of the PFS, TFEQ, and DEBQ, although associated with each other, measure distinct aspects of eating behavior. Against this background, it will be most interesting to see how PFS scores relate to other measures of eating behavior in severely obese and in postgastric bypass patients.

Our findings of enhanced aggregated and subdomain PFS scores in severely obese patients as compared with healthy control subjects corroborate and extend previous observations of a positive relation between BMI and PFS scores (20), which indicates that hedonic eating motives are markedly enhanced in these patients and may be a critical factor in the pathogenesis of severe obesity. Interestingly, the increase in hedonic hunger motivation concerns thoughts of consuming available and present foods but not the respective taste component, although our obese patients had BMIs twice as high as those of the control subjects. This pattern supports the notion that obesity is associated with an increased drive to eat rather than with enhanced feelings of pleasure while eating (25). According to Berridge (26), food reward can be dissociated into 2 distinct components: "liking," which reflects the experience of pleasure while eating, and "wanting," which reflects appetite/incentive motivation. At a neurobiological level, the 2 processes rely on different neurotransmitter systems. Processes of "liking" are assumed to be mediated by opioidergic and GABAergic pathways, whereas "wanting" depends on mesolimbic dopaminergic transmission. Although the concept of hedonic hunger as assessed by means of the PFS incorporates both processes (15), the observed pattern of changes in PFS subdomain scores speaks for enhanced "wanting" but unaltered "liking" in severe obesity. This interpretation is in line with the assumption that food wanting but not food liking differentiates obese and normal-weight individuals (25) and also fits well with the low D2 receptor density in reward circuits of obese subjects (17).

Given the cross-sectional nature of our study, it remains to be seen whether gastric bypass surgery in fact normalizes hedonic hunger in individual obese subjects. However, because our non–gastric bypass obese patients were being evaluated for bariatric surgery at our department, it is reasonable to assume that their hedonic drive for palatable foods was generally comparable with that displayed preoperatively by our gastric bypass patients. It also cannot be ruled out that the motivated and help-seeking patients in our study, who were recruited at a highly specialized medical center (IOC), may deviate from severely obese subjects in the general population. Nonetheless, the major finding of a lower hedonic hunger motivation in the gastric bypass patients than in the non–gastric bypass obese patients was clearly not affected by this potential selection bias.

The mechanism behind the reduction in hedonic hunger associated with gastric bypass surgery could not be derived from our data. Gastric bypass surgery has repeatedly been shown to enhance the secretion of gastrointestinal satiety hormones (9), which directly or indirectly act on hypothalamic structures crucially involved in the homeostatic regulation of eating behavior (27). Increasing evidence suggests that neuronal circuits of homeostatic hunger regulation are highly interlinked with circuits that control nonhomeostatic aspects of eating behavior, such as food reward (28, 29). Gastrointestinal hormones influencing hypothalamic and higher brain structures to reduce hedonic appetite could thus account for our findings. In line with this assumption, intravenous infusion of the gastrointestinal hormone PYY modulates the response of corticolimbic and higher cortical brain areas to visual stimulation with food pictures (30), and a recent study has provided evidence that D2 receptor density in reward-processing brain areas rapidly increases after gastric bypass surgery (31).

Gastric bypass surgery may also reduce hedonic hunger via learning processes. After the operation, the consumption of highly rewarding foods such as chocolate can provoke dumping syndrome–like adverse reactions such as abdominal pain, dizziness, or nausea that may strongly reduce the rewarding value of such foods and even result in avoidance behavior. Supporting this view, we found a significant association between reduced scores on the "food present" and "food tasted" domains and early dumping symptoms in a subsample of gastric bypass patients. Fittingly, intake of palatable foods such as chocolate, cakes, biscuits, and cookies was previously shown to be markedly lower in gastric bypass patients than in non–gastric bypass severely obese patients (32). Alternatively, such changes in food preferences may derive from alterations in taste perception after gastric bypass surgery (33)—a notion supported by the lower hedonic drive for food actually tasted that was observed in our gastric bypass patients as compared with the obese and nonobese subjects. In principle, reduced hedonic hunger could also be a consequence of generally decreased postoperation food consumption associated with a diminished reinforcing effect of food. Importantly, however, hedonic hunger was not related to general hunger motivation in a subgroup of our gastric bypass patients, which indicated that changes in PFS scores do not merely mirror alterations in global hunger motivation. Thus, our results put an intriguing new complexion on previous findings of reduced hunger feelings (7, 8) and decreased food intake (34) after gastric bypass surgery that is also associated with reduced disinhibition and increased dietary restraint (7, 8, 35).

The "food available" and "food present" domain scores of the gastric bypass patients correlated inversely and significantly, albeit mildly, with the relative %EBL, which indicate that those patients with the lowest scores had experienced the greatest reduction in body weight. This result of our cross-sectional study clearly calls for longitudinal follow-up experiments to explore whether changes in hedonic hunger depend on or even determine the loss of body weight due to gastric bypass surgery. Also, future studies should test whether changes in binge-eating behavior that were not examined in our study contribute to the reduction in hedonic hunger associated with gastric bypass surgery.

In conclusion, our results indicate that the hedonic appetite for palatable foods is markedly enhanced in severely obese subjects as compared with nonobese subjects, but that this enhancement is not found in patients who have undergone gastric bypass surgery. Investigating the gut-brain axis mechanisms that mediate the relation between successful weight loss due to gastric bypass surgery and the reduction in experienced food reward may lead to new perspectives concerning more effective and nonsurgical therapies for severe obesity.

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The authors' responsibilities were as follows—BS, BE, BW, MT, and MH: study concept and design and critical revision of the manuscript for important intellectual content; BS, BE, and MT: data acquisition; BS, BE, BW, and MH: data analysis and interpretation; BS and BE: draft of manuscript; and BS: study supervision. All authors had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. None of the authors reported conflicts of interest.

REFERENCES

1. Morton GJ, Cummings DE, Baskin DG, Barsh GS, Schwartz MW. Central nervous system control of food intake and body weight. Nature 2006;443:289–95.
2. Chaudhri OB, Field BC, Bloom SR. Gastrointestinal satiety signals. Int J Obes (Lond) 2008;32(suppl 7):S28–31.
3. Cummings DE, Overduin J. Gastrointestinal regulation of food intake. J Clin Invest 2007;117:13–23.
4. Sturm R. Increases in morbid obesity in the USA: 2000-2005. Public Health 2007;121:492–6.
5. Buchwald H, Williams SE. Bariatric surgery worldwide 2003. Obes Surg 2004;14:1157–64.
6. DeMaria EJ. Bariatric surgery for morbid obesity. N Engl J Med 2007; 356:2176–83.
7. Thirlby RC, Bahiraei F, Randall J, Drewnoski A. Effect of Roux-en-Y gastric bypass on satiety and food likes: the role of genetics. J Gastrointest Surg 2006;10:270–7.
8. Bocchieri-Ricciardi LE, Chen EY, Munoz D, et al. Pre-surgery binge eating status: effect on eating behavior and weight outcome after gastric bypass. Obes Surg 2006;16:1198–204.
9. Ashrafian H, le Roux CW. Metabolic surgery and gut hormones—a review of bariatric entero-humoral modulation. Physiol Behav 2009;97: 620–31.
10. Cummings DE, Weigle DS, Frayo RS, et al. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. N Engl J Med 2002; 346:1623–30.
11. Tritos NA, Mun E, Bertkau A, Grayson R, Maratos-Flier E, Goldfine A. Serum ghrelin levels in response to glucose load in obese subjects post-gastric bypass surgery. Obes Res 2003;11:919–24.
12. Fruhbeck G, Diez CA, Gil MJ. Fundus functionality and ghrelin concentrations after bariatric surgery. N Engl J Med 2004;350: 308–9.
13. Stoeckli R, Chanda R, Langer I, Keller U. Changes of body weight and plasma ghrelin levels after gastric banding and gastric bypass. Obes Res 2004;12:346–50.
14. Stratis C, Alexandrides T, Vagenas K, Kalfarentzos F. Ghrelin and peptide YY levels after a variant of biliopancreatic diversion with Roux-en-Y gastric bypass versus after colectomy: a prospective comparative study. Obes Surg 2006;16:752–8.
15. Lowe MR, Butryn ML. Hedonic hunger: a new dimension of appetite? Physiol Behav 2007;91:432–9.
16. Epstein LH, Leddy JJ. Food reinforcement. Appetite 2006;46:22–5.
17. Wang GJ, Volkow ND, Logan J, et al. Brain dopamine and obesity. Lancet 2001;357:354–7.
18. Volkow ND, Wise RA. How can drug addiction help us understand obesity? Nat Neurosci 2005;8:555–60.
19. Lowe MR, Butryn ML, Didie ER, et al. The Power of Food Scale. A new measure of the psychological influence of the food environment. Appetite 2009;53:114–8.
20. Cappelleri JC, Bushmakin AG, Gerber RA, et al. Evaluating the Power of Food Scale in obese subjects and a general sample of individuals: development and measurement properties. Int J Obes (Lond) 2009;33: 913–22.
21. Deitel M, Gawdat K, Melissas J. Reporting weight loss 2007. Obes Surg 2007;17:565–8.
22. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res 1985; 29:71–83.

23. Herman CP, Mack D. Restrained and unrestrained eating. J Pers 1975;43:647–60.
24. Van Strien T, Rookus MA, Bergers GP, Frijters JE, Defares PB. Life events, emotional eating and change in body mass index. Int J Obes 1986;10:29–35.
25. Mela DJ. Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. Appetite 2006;47:10–7.
26. Berridge KC. 'Liking' and 'wanting' food rewards: brain substrates and roles in eating disorders. Physiol Behav 2009;97:537–50.
27. Kral JG, Naslund E. Surgical treatment of obesity. Nat Clin Pract Endocrinol Metab 2007;3:574–83.
28. Shin AC, Zheng H, Berthoud HR. An expanded view of energy homeostasis: neural integration of metabolic, cognitive, and emotional drives to eat. Physiol Behav 2009;97:572–80.
29. Konner AC, Klockener T, Bruning JC. Control of energy homeostasis by insulin and leptin: targeting the arcuate nucleus and beyond. Physiol Behav 2009;97:632–8.
30. Batterham RL, Ffytche DH, Rosenthal JM, et al. PYY modulation of cortical and hypothalamic brain areas predicts feeding behaviour in humans. Nature 2007;450:106–9.
31. Steele KE, Prokopowicz GP, Schweitzer MA, et al. Alterations of central dopamine receptors before and after gastric bypass surgery. Obes Surg 2010;20:369–74.
32. Ernst B, Thurnheer M, Wilms B, Schultes B. Differential changes in dietary habits after gastric bypass versus gastric banding operations. Obes Surg 2009;19:274–80.
33. Miras AD, le Roux CW. Bariatric surgery and taste: novel mechanisms of weight loss. Curr Opin Gastroenterol 2010;26:140–5.
34. Kenler HA, Brolin RE, Cody RP. Changes in eating behavior after horizontal gastroplasty and Roux-en-Y gastric bypass. Am J Clin Nutr 1990;52:87–92.
35. Kalarchian MA, Wilson GT, Brolin RE, Bradley L. Effects of bariatric surgery on binge eating and related psychopathology. Eat Weight Disord 1999;4:1–5.