Loss of Control Over Eating Predicts Outcomes in Bariatric Surgery Patients: A Prospective, 24-Month Follow-Up Study

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Objective: This study examined the clinical significance of loss of control (LOC) over eating in bariatric surgery patients over 24 months of prospective, multiwave follow-ups.

Method: Three hundred sixty-one gastric bypass surgery patients completed a battery of assessments before surgery and at 6, 12, and 24 months following surgery. In addition to weight loss and LOC over eating, the assessments targeted eating disorder psychopathology, depression levels, and quality of life. The study was conducted between January 2002 and February 2008.

Results: Prior to surgery, 61% of patients reported general LOC; postsurgery, 31% reported LOC at 6-month follow-up, 36% reported LOC at 12-month follow-up, and 39% reported LOC at 24-month follow-up. Preoperative LOC did not predict postoperative outcomes. In contrast, mixed models analyses revealed that postsurgery LOC was predictive of weight loss outcomes: patients with LOC postsurgery lost significantly less weight at 12-month (34.6% vs 37.2% BMI loss) and 24-month (35.8% vs 39.1% BMI loss) postsurgery follow-ups. Postsurgery LOC also significantly predicted eating disorder psychopathology, depression, and quality of life at 12- and 24-month postsurgery follow-ups.

Conclusions: Preoperative LOC does not appear to be a negative prognostic indicator for postsurgical outcomes. Postoperative LOC, however, significantly predicts poorer postsurgical weight loss and psychosocial outcomes at 12 and 24 months following surgery. Since LOC following bariatric surgery significantly predicts attenuated postsurgical improvements, it may signal a need for clinical attention.

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Bariatric surgery is the most effective treatment for severe obesity, yielding average losses of approximately 35% of initial body weight.[1] Despite the rapid and dramatic weight loss that is achieved in the initial months postsurgery, the loss begins to plateau and is frequently followed by weight regain in the 2–10 years following surgery.[2] Similarly, research has documented substantial improvements in psychosocial functioning following bariatric surgery although the longer-term durability of these improvements is less certain.[3] Since a substantial proportion of patients begin to regain weight and some patients fail to benefit psychosocially after bariatric surgery,[4] several lines of research have attempted to identify patient variables that may predict long-term treatment outcome.[5] To date, few psychosocial factors have emerged as reliable predictors of either weight loss or psychosocial functioning following bariatric surgery.[5] For example, research has found that clinically hypothesized variables such as preoperative depression[6] and history of prior sexual abuse or childhood maltreatment[7] do not prospectively predict short-term (eg, 12-month) bariatric surgery outcomes.

As broad psychosocial factors have not emerged as reliable predictors, interest in eating-specific behaviors has grown.[5] In fact, considerable research attention has been devoted to determining whether binge eating confers a poor prognosis of bariatric surgery outcomes,[8] as binge-eating behaviors[9–11] and binge-eating disorder (BED)[12,13] are common in bariatric surgery candidates. Binge eating is defined in the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision (*DSM-IV-TR*[14]) as eating an unusually large amount of food while experiencing a subjective sense of loss of control over the eating. Presurgery, patients who binge eat do not differ in weight status[15] from non–binge-eating patients, but are generally characterized by greater psychosocial problems[10,16–18] and significantly greater psychiatric comorbidity.[13]

Research on the prognostic significance of preoperative binge eating on bariatric surgery outcomes has focused mostly on changes in weight. To date, findings regarding the prognostic significance of preoperative binge eating are mixed, with some reports of baseline binge eating predicting less weight loss,[19,20] while others report the opposite pattern[21] or no relationship.[17,22] In terms of psychosocial functioning outcomes, Malone and Alger-Mayer[23] reported that patients with more severe binge-eating problems before surgery benefited the most in terms of improved quality of life postsurgery. In contrast, Green and colleagues[24] reported that although binge eaters reported heightened depression and deflated self-esteem presurgically, they did not differ from those who did not report preoperative binge eating at 6 months postsurgery. Most recently, White et al[11] reported that patients with preoperative binge eating improved substantially at 12 months postsurgery in all domains measured, including weight loss and global psychosocial functioning. At 12 months postsurgery, none of the patients reported binge eating at the diagnostic threshold frequency specified by the *DSM-IV-TR* for BED; 8.8% reported infrequent binge eating (ie, less than once per week); and only 1 patient (0.7%) reported binge eating weekly. These substantial improvements in binge eating support previous reports of a near remission of binge-eating symptoms following surgery.[17,25,26]

It is important to emphasize that following bariatric surgery, binge eating—traditionally defined as eating unusually large amounts of food—may not be physically possible. Indeed, consumption of either too-large portions or rich or high-fat foods following bariatric surgery typically results in vomiting and/or dumping syndrome. These unpleasant events would likely occur before and effectively prevent (or greatly reduce frequency of) the consumption of an objectively large amount of food. Although binge eating may be physically impossible following surgery, a different facet of binge-eating pathology may remain or emerge postoperatively. Specifically, the subjective sense of loss of control (LOC) over eating may be an important indicator of eating problems after surgery. Indeed, recent research with diverse clinical and community groups across all weight categories has suggested that the presence of LOC—regardless of the amount of food consumed—is clinically meaningful. Eating with LOC (over small or subjectively large amounts of food) generally involves more calories and a higher percentage of fat and carbohydrate intake[27,28] and predicts eating disorder psychopathology[29,30] and psychological disturbance[31] in a manner similar to LOC over objectively large amounts of food. Preliminary research with bariatric surgery patients suggests that LOC may be especially relevant. Kalarchian et al,[32] in a cross-sectional study of 99 bariatric surgery patients, found that patients reporting postoperative LOC had less successful weight outcomes than those reporting no LOC. Unfortunately, conclusions from this study are limited by the cross-sectional design. A recent longitudinal report of 129 bariatric patients followed for 12 months after surgery found that LOC over eating was common following surgery, and it was associated with poorer weight and psychosocial outcomes.[33]

The present study examined the clinical significance of LOC in a large series of bariatric surgery patients over 24 months of prospective, multiwave follow-ups. Patients were assessed preoperatively and then reassessed postsurgery at 6-month, 12-month, and 24-month follow-ups. This design allowed for consideration of both presurgery and postsurgery LOC as concurrent and prospective predictors of weight loss and psychosocial functioning. Specific hypotheses were as follows: (1) Preoperative LOC would predict postoperative LOC, (2) Patients with and without preoperative LOC would differ in BMI and psychosocial domains, (3) Postoperative LOC would be a function of preoperative LOC and length of time since surgery, (4) Weight loss would be a function of preoperative and postoperative LOC and time, and (5) LOC would predict psychosocial outcomes.

METHOD

Participants

Participants were 361 (50 male and 311 female) extremely obese patients who underwent gastric bypass surgery at 2 general medical centers in the northeastern United States. Mean age was 43.7 years (SD = 10.0) and mean body mass index (BMI) was 51.1 (SD = 8.3). Of the participants, 81.4% (n = 294) were white, 9.1% (n = 33) were African American, 7.2% (n = 26) were Hispanic American, 0.3% (n = 1) was Asian, and 2.0% (n = 7) were of another ethnicity or unknown. Educationally, 67.9% (n = 245) attended at least some college, and an additional 26.3% (n = 95) completed high school. The 2 sites did not differ in BMI, distribution of gender, or educational attainment.

Informed Consent and Study Procedures

Institutional review board approval was granted at each site, and written informed consent was obtained from all participants. Patients were informed that they were participating in research studies to learn about the effects of bariatric surgery over time on weight, eating behaviors, psychological functioning, and general quality of life. Patients were informed that their participation would not influence the type of care provided by the surgical team. Patients were told there would be no direct medical benefit to them, although it was hoped that the knowledge gained might ultimately benefit other bariatric patients in the future. Patients were also informed that the findings would only be shared with the treatment team if they so desired and provided consent. No compensation was provided.

Patients completed a battery of assessments prior to surgery and at 6-month, 12-month, and 24-month follow-up points. Of the patients who completed the baseline (preoperative) assessment, 86.1% (n = 311) completed the 6-month follow-up, 81.4% (n = 294) completed the 12-month follow-up, and 47.4% (n = 171) completed the 24-month follow-up. In order to be included in the current study, participants had to have completed at least 1 follow-up assessment. Participants who completed the follow-up assessments did not differ from those who did not in terms of preoperative BMI, LOC, or psychological functioning (Beck Depression Inventory [BDI], Short-Form 36 [SF-36], or Eating Disorder

Examination Questionnaire [EDE-Q] scores) with 1 exception. Participants who did not complete the 24-month assessment had reported significantly higher levels of dietary restraint prior to surgery than those who completed the follow-up; the groups did not differ on restraint measured at 6 months or 12 months. The group that participated in the 24-month assessment did not differ from the group that did not on weight loss, LOC, BDI, SF-36, or EDE-Q scores measured at 12 months.

Measures

Weight self-report. Percent weight loss from baseline was the primary outcome variable. BMI (weight in kilograms/height in meters^2) was calculated from self-reported weight and height as part of a larger questionnaire battery and completed at the same time as the psychosocial measures. Concurrently measured (ie, by clinic staff) weight data were available for a subsample (n = 187). In this subsample, the measured (mean = 51.9, SD = 7.9) and self-reported (mean = 51.7, SD = 8.4) BMIs did not differ ($t_{186} = 0.75$, $P = .46$). Further, the degree of misreport was unrelated to increasing BMI ($r_{185} = 0.12$, $P = .11$).

Assessment of features of eating disorders and loss of control. The Eating Disorder Examination Questionnaire (EDE-Q),[34] the self-report version of the Eating Disorder Examination Interview,[35] assesses eating disorder psychopathology. The EDE-Q assesses the frequency of various forms of overeating, including binge eating. The EDE-Q comprises 4 subscales (dietary restraint, eating concerns, weight concerns, and shape concerns) and yields a global score. Items are rated on 7-point scales (0–6), with higher scores reflecting greater severity or frequency. Most items are specific to current symptoms, encompassing the previous 28-day period. LOC was defined as the presence or absence of any LOC episodes in the previous 28-day period. This assessment method for LOC follows the method previously used in studies with diverse clinical groups.[29,30,32] Both objective bulimic episodes (defined as eating unusually large amounts of food while experiencing a subjective sense of LOC) and subjective bulimic episodes (defined as experiencing LOC when eating small or normal amounts of food) were classified as LOC episodes. The EDE-Q has received psychometric support, including adequate test-retest reliability[36] and good convergence with the Eating Disorder Examination Interview.[34,37–41] Further, the EDE-Q has been shown to adequately identify binge eating in bariatric surgery candidates.[10,42]

Assessment of psychological functioning. The Beck Depression Inventory (BDI)[43] 21-item version assesses current depression level and symptoms of depression. It is a widely used and established measure with demonstrated reliability and validity.[44] Higher scores on the BDI reflect higher levels of depression and, more broadly, negative affect[45,46]; higher scores are also an efficient marker for heightened psychopathological and psychiatric disturbances.[46]

The Medical Outcomes Study Short-Form-36 Health Survey (SF-36)[47] is a 36-item, widely used self-report instrument that assesses health-related quality of life (HRQL). The SF-36 has well-established reliability[48] and validity.[49] The instrument comprises 8 subscales: Physical Functioning, Role-Physical, Bodily Pain, General Health, Vitality, Social Functioning, Role-Emotional, and Mental Health. Each subscale is composed of frequency (eg, 6-point scale from 1 [all of the time] to 6 [none of the time]), severity (eg, 3-point scale from 1 [yes, limited a lot] to 3 [no, not limited at all]), or forced choice (eg, 2-point scale for yes or no) items. The SF-36 raw scale scores are transformed to scores ranging from 0 (lowest level of HRQL) to 100 (highest level of HRQL) with a standard deviation of 15.[50] The SF-36 also generates 2 summary scores: the Physical Component Summary (PCS) and the Mental Component Summary (MCS).[51] The PCS and MCS scores are such that the means are 50 and standard deviations are 10 for the general US population.

Statistical analyses. Data were analyzed using SAS v9.1 (SAS Institute Inc, Cary, North Carolina) and SPSS v14.0 (SPSS Inc, Chicago, Illinois). One-way analyses of variance (ANOVAs) were used to test the hypothesis that presurgery LOC groups would differ on BMI and psychosocial outcomes at baseline. A series of binary logistic regression analyses was used to test the hypothesis that time point–specific postoperative LOC would be predicted by LOC at baseline. To test the hypothesis that postoperative LOC would be a function of preoperative LOC and the length of time since surgery, data were analyzed using nonlinear mixed models for binary outcomes. Finally, mixed effects regression analyses were used to test the hypotheses that weight and psychosocial outcomes would be predicted by baseline LOC, postoperative LOC, length of time since surgery, and their interactions.

RESULTS

Baseline Characteristics

Prior to surgery, 42.4% (n = 153) of patients reported LOC for objectively large eating episodes, 40.2% (n = 145) reported LOC for small episodes, and 61.2% (n = 221) reported general LOC (ie, loss of control for *either* large or small amounts of food). Odds ratios (ORs) were generated to determine whether LOC for small and large eating episodes at baseline were related. Analyses revealed that individuals reporting LOC over small episodes were twice as likely to report LOC over large eating episodes (OR = 2.1, Wald = 11.15, $P = .001$; 95% CI = 1.4–3.2).

Hypothesis 1: Preoperative LOC would predict postoperative LOC. The hypothesis that postoperative LOC would be predicted by preoperative LOC was tested using simple binary logistic regression analyses for each follow-up time point. In this series of analyses, baseline LOC for both objectively large episodes as well as general LOC

Table 1. Point Prevalence of Postsurgical LOC Over Eating Episodes as a Function of Preoperative LOC[a]

	6 Months LOC, % (n)			12 Months LOC, % (n)			24 Months LOC, % (n)		
Baseline Status	Present	Absent	Statistics	Present	Absent	Statistics	Present	Absent	Statistics
Objective			Wald = 12.4, $P < .001$; Exp(B) = 2.47			Wald = 13.1, $P < .001$; Exp(B) = 2.51			Wald = 2.68, $P = .102$; Exp(B) = 1.69
No objective LOC (large episodes)	22.4 (38)	77.6 (132)		28.1 (47)	71.9 (120)		33.7 (30)	66.3 (59)	
Objective LOC (large episodes)	41.5 (54)	58.5 (76)		49.6 (57)	50.4 (58)		46.2 (36)	53.8 (42)	
Total	30.7 (92)	69.3 (208)		36.9 (104)	63.1 (178)		39.5 (66)	60.5 (101)	
Subjective			Wald = 9.7, $P = .002$; Exp(B) = 2.20			Wald = 9.34, $P = .002$; Exp(B) = 2.16			Wald = 6.70, $P = .010$; Exp(B) = 2.32
No subjective LOC (small episodes)	23.7 (41)	76.3 (132)		29.4 (50)	70.6 (120)		31.4 (32)	68.6 (70)	
Subjective LOC (small episodes)	40.6 (52)	59.4 (76)		47.4 (54)	52.6 (60)		52.5 (34)	48.5 (32)	
Total	30.9 (93)	69.1 (208)		36.6 (104)	63.4 (180)		39.3 (66)	60.7 (102)	
General			Wald = 14.0, $P < .001$; Exp(B) = 2.99			Wald = 14.1, $P < .001$; Exp(B) = 2.77			Wald = 9.94, $P = .002$; Exp(B) = 3.00
No LOC	17.3 (19)	82.7 (91)		23.0 (36)	77.0 (87)		24.2 (16)	75.8 (50)	
General LOC[b]	38.4 (73)	61.6 (117)		45.3 (77)	54.7 (93)		49.0 (50)	51.0 (52)	
Total	30.7 (92)	69.3 (208)		36.4 (103)	63.6 (180)		39.3 (66)	60.7 (102)	

[a]Percentages reflect number reporting LOC at follow-up as a proportion of baseline subgroup (LOC vs no LOC) and are based on the number of patients who provided data for each item. Missing data are due to patients skipping items on the assessment.
[b]General LOC refers to objective (large) or subjective (small) bulimic episodes.
Abbreviation: LOC = loss of control.

(ie, for both large and small amounts of food) was considered. Point prevalence and test statistics are presented in Table 1. As shown in the table, either type of baseline LOC is highly predictive of LOC postsurgery. Therefore, general LOC (for *either* small or large episodes) at baseline was considered the primary variable for the remainder of the analyses.

Hypothesis 2: Patients with and without preoperative LOC would differ in BMI and psychosocial domains. Baseline values for BMI and psychosocial outcomes appear in Table 2; patients with and without LOC at baseline were compared on BMI and psychosocial outcomes with one-way ANOVAs. The groups did not differ in terms of presurgical BMI. Significant differences were observed such that the group experiencing LOC reported significantly higher levels of depressive symptoms, diminished quality of life as measured by the SF-36, and greater eating, shape, and weight concerns.

Hypothesis 3: Postoperative LOC would be a function of preoperative LOC and length of time since surgery. The point prevalence of LOC at each assessment point is presented graphically in Figure 1. Although the proportion of the sample reporting general LOC decreased immediately following surgery ($\chi^2_1 = 129.5$, N = 307, $P < .001$), the prevalence of postoperative LOC increased with each follow-up point. A χ^2 goodness of fit test found that compared to the 6-month point, the prevalence of LOC was significantly greater at 12 months ($\chi^2_1 = 4.52$, N = 289, $P = .03$) and at 24 months ($\chi^2_1 = 5.67$, N = 170, $P = .02$) following surgery. The difference in prevalence between the 12-month to 24-month assessment was not significant, ($\chi^2_1 = 0.546$, N = 170, $P = .46$). The hypothesis that postoperative LOC would be a function of preoperative LOC and the assessment point (ie, time interval) postsurgery was tested using a nonlinear mixed model analysis (PROC NLMIXED; SAS, Cary, North Carolina) with a random intercept. Models were tested in which session was left as a continuous variable and included along with logarithmic, cubic, exponential, and quadratic transformation of time. Of these, the logarithmic transformation yielded the best model fit statistics. Models were tested including preoperative LOC status, session, and the interactions. Through model-building, nonsignificant ($P > .10$) interaction terms were dropped to yield a final model in which only the main effects significantly predicted postoperative LOC. The final model indicated that preoperative LOC significantly predicted postoperative LOC ($\beta = 1.43$, $t = 5.09$, $P = .0001$). Further, the coefficient for the time effect was significant ($\beta = 0.36$, $t = 2.03$, $P = .04$), indicating that as the length of time between surgery and assessment increased, the likelihood of reporting LOC also increased. The intraclass correlation coefficient (ICC) was highly significant (ICC = 0.40, $P < .0001$), indicating a substantial amount of variance attributable to the subject effect.

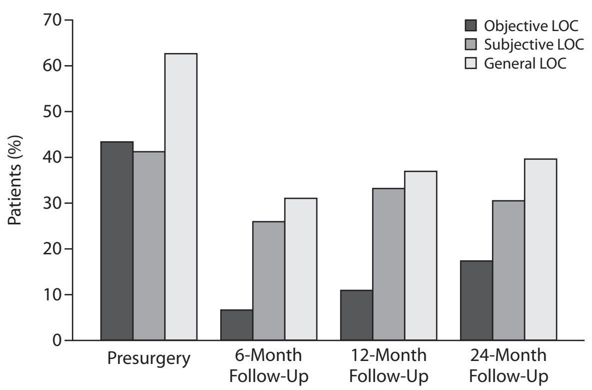
Hypothesis 4: Weight loss would be a function of preoperative LOC, postoperative LOC, and time. The hypothesis that weight loss (percent weight loss from presurgery weight) would be a function of preoperative and postoperative LOC was tested with mixed models analysis. Variables entered into the initial analysis were baseline LOC, time point-specific LOC (ie, LOC status at each follow-up

Table 2. Baseline BMI and Psychosocial Outcome Values of Sample as a Function of Preoperative LOC

Variable	No Preoperative LOC (n = 131) Mean	SD	Preoperative LOC (n = 221) Mean	SD	Total Mean	SD	F	P
BMI	52.0	(8.5)	50.5	(8.3)	51.1	(8.4)	2.39	.123
BDI	11.1	(8.0)	17.1	(9.7)	14.9	(9.5)	35.82	.000
SF-36, MCS	49.5	(11.2)	44.6	(10.8)	46.4	(11.2)	16.19	.000
SF-36, PCS	33.9	(11.0)	30.8	(9.3)	31.9	(10.1)	8.02	.005
EDE-Q, restraint	2.4	(1.4)	2.4	(1.4)	2.4	(1.4)	0.13	.714
EDE-Q, eating concerns	1.2	(1.0)	2.5	(1.2)	2.0	(1.3)	106.43	.000
EDE-Q, shape concerns	3.8	(1.2)	4.6	(1.0)	4.3	(1.2)	44.99	.000
EDE-Q, weight concerns	3.1	(1.1)	3.9	(1.1)	3.6	(1.1)	46.40	.000
EDE-Q, total	2.6	(0.9)	3.4	(0.9)	3.1	(1.0)	59.38	.000

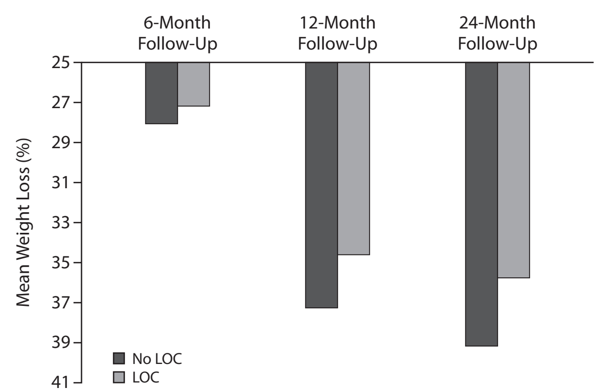
Abbreviations: BDI = Beck Depression Inventory, BMI = body mass index, EDE-Q = Eating Disorder Examination Questionnaire, LOC = loss of control, MCS = Mental Component Summary, PCS = Physical Component Summary, SF-36 = Short-Form 36.

Figure 1. Point Prevalence of LOC



Abbreviation: LOC = loss of control.

Figure 2. Weight Loss (% loss from baseline) as a Function of Postoperative, Point-Specific LOC



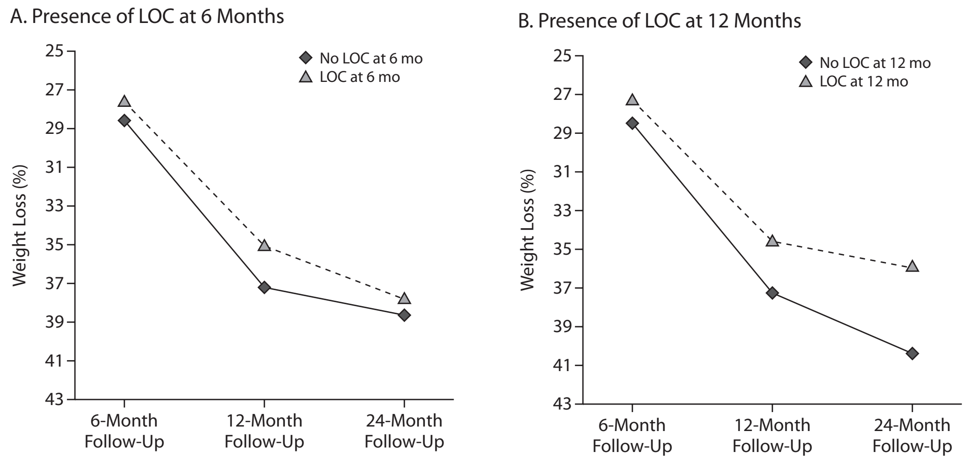
Abbreviation: LOC = loss of control.

point), assessment time point, and all possible 2-way interactions. Nonsignificant interaction terms (at $P > .10$) were removed from analysis, and tests of various covariance structures (ie, unstructured, simple, Toeplitz, compound symmetry, autoregressive) revealed that the autoregressive covariance structure was most appropriate for the data. Analyses revealed that preoperative LOC did not influence degree of weight loss, $F_{1,381} = 0.025$, $P = .87$. Time point of assessment was a highly significant predictor of weight loss ($F_{1,338} = 381.9$, $P < .001$), as was postoperative LOC ($F_{1,605} = 4.61$, $P = .032$). None of the interaction terms were significant. Figure 2 shows the pattern of percent weight loss as a function of postoperative LOC, measured at the concurrent assessment.

To investigate further the pattern of weight loss over time as a function of LOC, analyses were repeated within time points (ie, restricted to time point–specific outcomes). At the 6-month follow-up point, LOC did not predict weight loss ($F_{1,287} = 2.262$, $P = .134$). LOC was a significant predictor of weight loss at 12 months ($F_{1,272} = 7.595$, $P = .006$) and at 24 months postsurgery ($F_{1,156} = 4.298$, $P = .040$). At 12 months, the group reporting LOC lost a mean of 17.7 (SD = 5.4) BMI units, whereas the group reporting no LOC lost 19.1 (SD = 5.4) BMI units; at 24 months, the mean BMI units lost were 18.3 (SD = 6.0) and 20.5 (SD = 6.9), respectively. Preoperative LOC did not significantly predict weight outcomes at any follow-up assessment point.

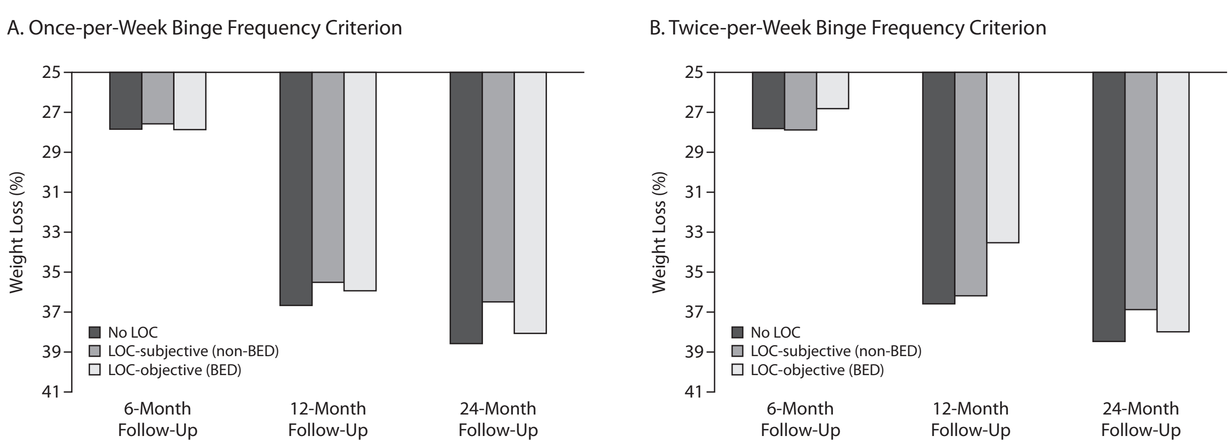
A series of analyses was conducted to test the prospective effects of LOC. Mixed models analysis revealed that LOC at 6 months significantly predicted weight loss at the latter follow-up points, $F_{1,252} = 4.748$, $P = .03$. Similarly, LOC at 12 months significantly predicted percent weight loss at 24 months, $F_{1,130} = 8.788$, $P = .004$. In terms of BMI units, the group reporting LOC at 12 months lost an average of 18.3 (SD = 5.6) BMI units at the 24-month assessment, compared to an average 21.2 (SD = 7.2) BMI unit loss for the group that denied LOC at 12 months. The pattern of results in terms of percent weight loss is presented graphically in Figure 3. Finally, the influence of postsurgical LOC on weight regain from 12 to 24 months was examined. From 12 to 24 months, 32.6% (n = 45) of the patients had regained ≥ 2 kg. Analysis by LOC category found that LOC at 12 months

Figure 3. Prospective Relations of LOC at 6 and 12 Months With Percent Weight Loss



Abbreviation: LOC = loss of control.

Figure 4. Weight Outcomes as a Function of Presurgery Binge Eating



Abbreviations: BED = binge-eating disorder, LOC = loss of control.

predicted weight regain; $\chi^2 = 3.855$, $P = .05$, OR = 2.16 (95% CI = 0.995–4.687). Of those patients reporting LOC at 12 months, 44.2% went on to regain ≥ 2 kg between 12 and 24 months, whereas of those who did not report LOC, 26.8% went on to regain ≥ 2 kg.

As a conservative test, the influence of presurgery binge eating (ie, LOC over objectively large episodes of food) on postoperative weight loss was tested. Utilizing *DSM-IV-TR* criterion for BED of binge eating twice weekly, patients were classified as those having BED (ie, loss of control over eating large amounts of food at least twice weekly), those having non-BED LOC (reporting LOC but no overeating, or objective LOC but at subthreshold frequency), and those denying LOC. Analyses revealed no group differences at any of the follow-up points, suggesting that the presence of BED prior to surgery did not affect weight outcomes. These analyses were repeated using a less stringent frequency criterion for binge-eating episodes of 1 time per week. Again, the presence of binge eating at or approaching the diagnostic level for BED did not affect postsurgery weight outcomes. Figure 4 summarizes these results.

Hypothesis 5: LOC would predict poorer psychosocial outcomes. Mixed models analyses were conducted to examine the influence of postoperative LOC on psychosocial outcomes. For each analysis, baseline scores on respective psychosocial outcomes were entered as covariates. Results paralleled the weight loss outcomes. For all analyses, time of assessment was highly significant, indicating substantial improvements in all measured psychosocial domains following surgery. Mixed models restricted to only postsurgical values

Table 3. Psychosocial Outcomes at 12 and 24 Months Post-Surgery as a Function of Concurrent LOC

	12-Month Follow-Up						24-Month Follow-Up						Statistics		
	No LOC		LOC		Total		No LOC		LOC		Total				
Psychosocial Outcome	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	F^a	*df*	*P*
BDI	6.4	(9.8)	12.5	(20.6)	8.7	(15.0)	7.2	(19.8)	10.5	(10.6)	8.5	(16.8)	8.80	1,751	.003
SF-36, MCS	53.2	(10.5)	48.0	(12.8)	51.3	(11.7)	52.3	(10.6)	45.9	(14.3)	49.8	(12.5)	9.95	1,696	.002
SF-36, PCS	50.6	(9.2)	47.6	(10.8)	49.5	(9.9)	49.2	(10.7)	46.5	(11.6)	48.1	(11.1)	1.99	1,640	.159
EDE-Q, restraint	1.8	(1.4)	2.3	(1.4)	2.0	(1.5)	1.4	(1.3)	2.4	(1.4)	1.8	(1.4)	33.21	1,751	< .001
EDE-Q, eating concerns	0.5	(0.7)	1.5	(1.2)	0.9	(1.0)	0.5	(0.7)	1.8	(1.3)	1.0	(1.2)	104.50	1,734	< .001
EDE-Q, shape concerns	2.0	(1.3)	2.8	(1.3)	2.3	(1.4)	2.1	(1.4)	3.3	(1.3)	2.6	(1.5)	57.13	1,726	< .001
EDE-Q, weight concerns	1.4	(1.1)	2.2	(1.1)	1.7	(1.1)	1.4	(1.1)	2.5	(1.2)	1.8	(1.3)	59.99	1,743	< .001
EDE-Q, total	1.5	(0.9)	2.2	(1.0)	1.7	(1.0)	1.3	(0.9)	2.5	(1.1)	1.8	(1.2)	88.42	1,718	< .001

[a]*F* statistics reflect results of mixed models analysis; *F* effect reported is for postsurgical LOC, after controlling for baseline values of specific psychosocial outcomes. Effects for time of assessment are not reported; all psychosocial domains improved following surgery.
Abbreviations: BDI = Beck Depression Inventory, EDE-Q = Eating Disorder Examination Questionnaire, LOC = loss of control, MCS = Mental Component Summary, PCS = Physical Component Summary, SF-36 = Short-Form 36.

Table 4. Correlations and Frequency of LOC Episodes With Percent Weight Loss and Psychosocial Outcomes at Each Follow-Up Assessment

	LOC Episodes (sum of objective and subjective)		
Outcome	6 Months	12 Months	24 Months
Percent weight loss	−0.10	−0.07	−0.03
BDI	0.28	0.20	0.31
SF-36, MCS	−0.18	−0.31*	−0.42*
SF-36, PCS	−0.17	−0.17	0.06
EDE-Q, restraint	0.17	0.17	0.32*
EDE-Q, eating concerns	0.40*	0.49*	0.63*
EDE-Q, shape concerns	0.27*	0.44*	0.44*
EDE-Q, weight concerns	0.24*	0.40*	0.52*
EDE-Q, total	0.31*	0.45*	0.54*

*Significant after correction for multiple (n = 27) analyses ($P < .001$).
Abbreviations: BDI = Beck Depression Inventory, EDE-Q = Eating Disorder Examination Questionnaire, LOC = loss of control, MCS = Mental Component Summary, PCS = Physical Component Summary, SF-36 = Short-Form 36.

also revealed significant time effects—indicating continued trajectories of improvement—for SF-36 mental and physical health, as well as for the EDE-Q restraint, eating concerns, and weight concerns subscales. After controlling for baseline psychosocial variables, the effect of postoperative LOC on psychosocial outcomes was significant. The only exception was the SF-36 PCS score; postoperative LOC did not influence perceptions of postoperative physical functioning. As with weight loss outcomes, the pattern of differences between LOC groups was most prominent at 12-month outcomes and 24-month outcomes; Table 3 summarizes the uncorrected means at these time points.

Analyses to Determine Clinically Significant Loss of Control

Finally, a series of analyses was conducted to determine a clinically significant cut-point for LOC episodes. That is, we attempted to determine whether a severity or frequency threshold for LOC exists at which LOC must occur in order to affect outcomes. As reported above, the presence of LOC over unusually large eating episodes and frequency at which they occurred prior to surgery did not predict weight outcomes. The rates of LOC over unusually large episodes following surgery were too low to permit analysis with group contrasts (ie, ANOVA designs), as were the rates of patients reporting LOC at least twice weekly. Therefore, patient groups were generated based on frequency of LOC episodes utilizing a once-per-week cut-point (ie, once weekly vs less than once weekly vs none). Categorical analyses at each follow-up point revealed that frequency of LOC episodes was unrelated to weight loss at the concurrent time point.

As an additional test, LOC episodes were tallied and left as a continuous frequency variable. Table 4 summarizes correlation analyses testing whether the frequency of LOC episodes over the previous 28 days was related to psychosocial functioning following surgery. Moderate correlations were observed between LOC frequency and psychosocial and eating-specific impairment, and these correlations were stronger at the later time points (farther out from surgery). Overall, the most informative clinical cut-point for predicting postsurgical weight outcomes appears to be the simple presence or absence of LOC following surgery. In terms of predicting postsurgical psychosocial outcomes, a graded effect exists, with increased frequency of LOC episodes generally associated with worsened clinical outcomes across both eating-specific and broad psychosocial outcomes.

DISCUSSION

This study investigated the prognostic significance of preoperative and postoperative LOC over eating in extremely obese bariatric surgery patients over 24 months of prospective, multiwave follow-ups. Prior to surgery, 61% of surgical patients reported LOC over eating, which is comparable to reports from other research groups utilizing similar assessment procedures (see Niego et al[52] for a review). Preoperative LOC was associated concurrently with significantly elevated eating disorder psychopathology and psychosocial difficulties, and it predicted prospectively postoperative LOC.

Preoperative LOC, however, was unrelated to postoperative weight loss or psychosocial functioning. In contrast, LOC following surgery was a negative prognostic indicator for weight loss, with postoperative LOC predicting less weight loss at 12-month and 24-month follow-up points. The influence of postoperative LOC became more pronounced as the time postsurgery increased, and may correspond with the weight loss plateau that occurs some years following surgery.[2,53] Further, the rates of LOC increased over the course of the study: at 6 month follow-up, approximately 31% of the sample reported LOC, and by the 24 month follow-up, this percentage had increased to 39% overall and to nearly 50% among those experiencing LOC prior to surgery. The group reporting postoperative LOC reported elevated depressive symptoms and eating disturbances, as well as lower levels of quality of life as measured by the MCS scale of the SF-36. Of note, LOC was not predictive of the PCS scale, suggesting that the significant physical improvements attained through bariatric surgery are not easily influenced by a subjective sense of LOC. On the other hand, LOC does predict important bariatric outcomes, such as eating-specific and broad psychosocial functioning in addition to weight loss.

The current findings suggest that gastric bypass surgery results in considerable improvements, both in terms of weight loss and psychosocial outcomes, through 24 months postsurgery. A substantial percentage of patients, however, begins to plateau by 12–24 months postsurgery and may experience weight regain.[54] In the current study, through 24-months of postsurgical follow-up, LOC following surgery was significantly associated with weight regain at subsequent assessment points. Collectively, research investigating presurgical psychosocial, historical, and even eating-specific factors has reported little impact on postsurgical outcomes.[3,5,55] This is unfortunate since identification of such characteristics would suggest possible targets for supplemental interventions that might enhance outcomes even further. Therefore, our inclusion of both presurgical and postsurgical problematic eating marks one of the first studies to prospectively identify patient-specific factors predicting a worsened clinical profile and identifies a potential area for clinical intervention.

Our results support and extend findings from previous research reporting that preoperative binge eating and/or LOC over eating do not impede weight loss.[11,17,21–24] Collectively, this research indicates that preoperative binge eating, although common,[52] may not require specific additional clinical intervention before treatment. Our results, however, do suggest that the emergence of postoperative eating problems has negative prognostic influence on weight loss outcomes, as well as some of the psychosocial benefits associated with surgery. The current study parallels previous research with other patient groups[29–31] identifying the clinical significance of LOC over eating as a correlate of eating-specific and more global psychopathology. Our postsurgical findings indicate that LOC may be a marker of poorer outcomes and suggest a need for intervention. Since nearly 40% of the patients in this study reported LOC over eating in the 24 months following surgery, our findings suggest that this is not an uncommon problem and clinicians should routinely inquire about this rather than traditional foci on types and amounts of food consumed. Fortunately, clinicians can readily assess whether a patient experiences subjective LOC over eating episodes based on verbal report. Further, clinicians should be aware that while the mere presence or absence of LOC following surgery predicts weight outcomes, a graded effect exists with increasing frequency of LOC predicting commensurate worsening of psychosocial outcomes. An apt focus of clinical attention would be on developing coping strategies or on cognitive restructuring adapted from the best-established treatments for eating disorders.[56] More broadly, recent findings suggest that behavioral weight loss treatments have efficacy for reducing both binge eating (and LOC) and weight.[57] Future research will be required to identify the best treatments to ensure weight maintenance or continued losses in the years following surgery.

This study has some potential limitations that should be considered when interpreting the findings. The findings pertain to extremely obese patients who seek bariatric surgery at an urban general medical center and undergo gastric bypass procedures. The findings may not generalize to less obese patients or to obese patients who seek different (nonsurgical) forms of treatment or different forms of bariatric surgeries. Although the questionnaire we used to assess LOC elicits specific estimates in terms of the number of eating episodes in which LOC was experienced, self-report measures are potentially limited by retrospective recall and response biases. Previous psychometric evaluations have found that the EDE-Q may overestimate certain aspects of eating disorder pathology relative to clinician interview,[42,58] so it is possible that the rates of LOC were overreported. Alternatively, some research suggests that patients are more candid when reporting symptoms in questionnaire format than in clinician interview.[59] Our reliance on self-reported weights is an additional limitation, however, research has found that self-reported weight is an adequate proxy for measured weights.[60,61] Since we were primarily interested in time-varying outcomes, however, we opted to employ self-reported data corresponding with the time of assessment rather than measured weights taken at a different time point. The findings are further limited by the amount of missing data, particularly for the 24-month follow-up point, although the use of mixed models analyses permitted use of all available data for all participants, and these models offer important advantages over other methods of imputation for missing data in longitudinal research.[62] However, it should be noted that our analyses on the rate of dropouts found few differences between participants who provided data at the follow-up points compared to those who did not; participants providing follow-up data at 24 months did not differ from those who did not on any outcome variable measured at 12 months.

Another potential limitation is the possibility that extremely obese patients seeking bariatric surgery may minimize the existence of certain problems (eg, distress level, binge eating) in order to appear psychologically healthy and appropriate for the surgery. Indeed, research has shown that patients undergoing psychological evaluation prior to surgery have elevated scores on social desirability and commonly deny active problems.[63] Although this possibility must be considered, the research study procedures and informed consent methods should have served to minimize this likelihood. Specifically, participants completed the assessments as part of a research study and were informed that the results would not be shared with the clinical treatment team unless the patients specifically requested it. It was stressed that the assessments would have no medical benefit to patients and were intended solely to advance knowledge regarding psychosocial needs and outcomes of bariatric surgery patients. Further, LOC predicted emotional and psychological outcomes, but not physical domains, which suggests that response sets were not responsible for the pattern of results. Finally, since baseline prevalence of LOC was much higher than prevalence of LOC postsurgery, the possibility that patients minimized problems prior to surgery is unlikely.

In summary, this study examined preoperative and postoperative LOC over eating in relation to 6-month, 12-month, and 24-month postoperative outcomes in gastric bypass patients. The findings suggest that preoperative LOC does not appear to be a negative prognostic indicator for gastric bypass surgery. However, postoperative LOC predicts poorer weight loss, particularly as the time since surgery increases. Similarly, postoperative LOC predicts psychosocial outcomes, including depressive symptoms, additional eating disturbances, and some aspects of quality of life. Our postsurgical findings indicate that LOC may be a marker of poorer outcomes and suggest a need for intervention during postsurgical care. Longer-term follow-up is needed to determine the durability of these bariatric surgery outcomes and LOC's predictive utility.

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REFERENCES

1. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA.* 2004;292(14):1724–1737.
2. Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med.* 2004;351(26):2683–2693.
3. Sarwer DB, Wadden TA, Fabricatore AN. Psychosocial and behavioral aspects of bariatric surgery. *Obes Res.* 2005;13(4):639–648.
4. van Hout GC, Boekestein P, Fortuin FA, et al. Psychosocial functioning following bariatric surgery. *Obes Surg.* 2006;16(6):787–794.
5. Herpertz S, Kielmann R, Wolf AM, et al. Do psychosocial variables predict weight loss or mental health after obesity surgery? a systematic review. *Obes Res.* 2004;12(10):1554–1569.
6. Masheb RM, White MA, Toth CM, et al. The prognostic significance of depressive symptoms for predicting quality of life 12 months after gastric bypass. *Compr Psychiatry.* 2007;48(3):231–236.
7. Grilo CM, White MA, Masheb RM, et al. Relation of childhood sexual abuse and other forms of maltreatment to 12-month postoperative outcomes in extremely obese gastric bypass patients. *Obes Surg.* 2006;16(4):454–460.
8. Rusch MD, Andris D. Maladaptive eating patterns after weight-loss surgery. *Nutr Clin Pract.* 2007;22(1):41–49.
9. Kalarchian MA, Wilson GT, Brolin RE, et al. Binge eating in bariatric surgery patients. *Int J Eat Disord.* 1998;23(1):89–92.
10. Elder KA, Grilo CM, Masheb RM, et al. Comparison of two self-report instruments for assessing binge eating in bariatric surgery candidates. *Behav Res Ther.* 2006;44(4):545–560.
11. White MA, Masheb RM, Rothschild BS, et al. The prognostic significance of regular binge eating in extremely obese gastric bypass patients: 12-month postoperative outcomes. *J Clin Psychiatry.* 2006;67(12):1928–1935.
12. Kalarchian MA, Marcus MD, Levine MD, et al. Psychiatric disorders among bariatric surgery candidates: relationship to obesity and functional health status. *Am J Psychiatry.* 2007;164(2):328–334 [quiz 374].
13. Rosenberger PH, Henderson KE, Grilo CM. Psychiatric disorder comorbidity and association with eating disorders in bariatric surgery patients: a cross-sectional study using structured interview-based diagnosis. *J Clin Psychiatry.* 2006;67(7):1080–1085.
14. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition-Text Revision. Washington, DC: American Psychiatric Association; 2000.
15. de Zwaan M, Mitchell JE, Howell LM, et al. Characteristics of morbidly obese patients before gastric bypass surgery. *Compr Psychiatry.* 2003;44(5):428–434.
16. Mazzeo SE, Saunders R, Mitchell KS. Binge eating among African American and Caucasian bariatric surgery candidates. *Eat Behav.* 2005;6(3):189–196.
17. Powers PS, Perez A, Boyd F, et al. Eating pathology before and after bariatric surgery: a prospective study. *Int J Eat Disord.* 1999;25(3):293–300.
18. Wadden TA, Sarwer DB, Womble LG, et al. Psychosocial aspects of obesity and obesity surgery. *Surg Clin North Am.* 2001;81(5):1001–1024.
19. de Zwaan M, Lancaster KL, Mitchell JE, et al. Health-related quality of life in morbidly obese patients: effect of gastric bypass surgery. *Obes Surg.* 2002;12(6):773–780.
20. Pekkarinen T, Koskela K, Huikuri K, et al. Long-term results of gastroplasty for morbid obesity: binge-eating as a predictor of poor outcome. *Obes Surg.* 1994;4(3):248–255.
21. Latner JD, Wetzler S, Goodman ER, et al. Gastric bypass in a low-income, inner-city population: eating disturbances and weight loss. *Obes Res.* 2004;12(6):956–961.
22. Burgmer R, Grigutsch K, Zipfel S, et al. The influence of eating behavior and eating pathology on weight loss after gastric restriction operations. *Obes Surg.* 2005;15(5):684–691.
23. Malone M, Alger-Mayer S. Binge status and quality of life after gastric bypass surgery: a one-year study. *Obes Res.* 2004;12(3):473–481.
24. Green AE, Dymek-Valentine M, Pytluk S, et al. Psychosocial outcome of gastric bypass surgery for patients with and without binge eating. *Obes Surg.* 2004;14(7):975–985.
25. Kalarchian MA, Wilson GT, Brolin RE, et al. Effects of bariatric surgery on binge eating and related psychopathology. *Eat Weight Disord.* 1999;4(1):1–5.
26. Hsu LK, Sullivan SP, Benotti PN. Eating disturbances and outcome of

gastric bypass surgery: a pilot study. *Int J Eat Disord.* 1997;21(4): 385–390.
27. Guss JL, Kissileff HR, Devlin MJ, et al. Binge size increases with body mass index in women with binge-eating disorder. *Obes Res.* 2002;10(10): 1021–1029.
28. Theim KR, Tanofsky-Kraff M, Salaita CG, et al. Children's descriptions of the foods consumed during loss of control eating episodes. *Eat Behav.* 2007;8(2):258–265.
29. Latner JD, Hildebrandt T, Rosewall JK, et al. Loss of control over eating reflects eating disturbances and general psychopathology. *Behav Res Ther*; 2007;45(9):2203–2211
30. Elder KA, Paris M Jr, Anez LM, et al. Loss of control over eating is associated with eating disorder psychopathology in a community sample of Latinas. *Eat Behav.* 2008;9(4):501–503.
31. Colles SL, Dixon JB, O'Brien PE. Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity (Silver Spring).* 2008;16(3):608–614.
32. Kalarchian MA, Marcus MD, Wilson GT, et al. Binge eating among gastric bypass patients at long-term follow-up. *Obes Surg.* 2002;12(2): 270–275.
33. Colles SL, Dixon JB, O'Brien PE. Grazing and loss of control related to eating: two high-risk factors following bariatric surgery. *Obesity (Silver Spring).* 2008;16(3):615–622.
34. Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? *Int J Eat Disord.* 1994;16(4):363–370.
35. Fairburn CG, Cooper Z. The Eating Disorder Examination. In: Fairburn CG, Wilson GT, eds. *Binge Eating: Nature, Assessment, and Treatment*. 12th ed. New York, NY: Guilford Press; 1993:317–360.
36. Reas DL, Grilo CM, Masheb RM. Reliability of the Eating Disorder Examination-Questionnaire in patients with binge eating disorder. *Behav Res Ther.* 2006;44(1):43–51.
37. Black CM, Wilson GT. Assessment of eating disorders: interview versus questionnaire. *Int J Eat Disord.* 1996;20(1):43–50.
38. Grilo CM, Masheb RM, Wilson GT. A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *J Consult Clin Psychol.* 2001;69(2):317–322.
39. Grilo CM, Masheb RM, Wilson GT. Different methods for assessing the features of eating disorders in patients with binge eating disorder: a replication. *Obes Res.* 2001;9(7):418–422.
40. Mond JM, Hay PJ, Rodgers B, et al. Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behav Res Ther.* 2004;42(5):551–567.
41. Wilfley DE, Schwartz MB, Spurrell EB, et al. Assessing the specific psychopathology of binge eating disorder patients: interview or self-report? *Behav Res Ther.* 1997;35(12):1151–1159.
42. Kalarchian MA, Wilson GT, Brolin RE, et al. Assessment of eating disorders in bariatric surgery candidates: self-report questionnaire versus interview. *Int J Eat Disord.* 2000;28(4):465–469.
43. Beck AT, Steer R. *Manual for Revised Beck Depression Inventory*. New York, NY: Psychological Corporation; 1987.
44. Beck AT, Steer RA, Carbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. *Clin Psychol Rev.* 1988;8(1):77–100.
45. Watson D, Clark LA. Negative affectivity: the disposition to experience aversive emotional states. *Psychol Bull.* 1984;96(3):465–490.
46. Grilo CM, Masheb RM, Wilson GT. Subtyping binge eating disorder. *J Consult Clin Psychol.* 2001;69(6):1066–1072.
47. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. *Med Care.* 1992;30(6):473–483.
48. McHorney CA, Ware JE Jr, Lu JF, et al. The MOS 36-item Short-Form Health Survey (SF-36): III. Tests of data quality, scaling assumptions, and reliability across diverse patient groups. *Med Care.* 1994;32(1): 40–66.
49. McHorney CA, Ware JE Jr, Raczek AE. The MOS 36-Item Short-Form Health Survey (SF-36), II: psychometric and clinical tests of validity in measuring physical and mental health constructs. *Med Care.* 1993; 31(3):247–263.
50. Ware JE Jr, Snow KK, Kosinski M, et al. *SF-36 Health Survey: Manual and Interpretation Guide*. Boston, MA: The Health Institute, New England Medical Center; 1993.
51. Ware JE Jr, Kosinski M, Keller SD. *SF-36 Physical and Mental Health Summary Scales–A User's Manual.* Boston, MA: New England Medical Center, The Health Institute; 1994.
52. Niego SH, Kofman MD, Weiss JJ, et al. Binge eating in the bariatric surgery population: a review of the literature. *Int J Eat Disord.* 2007; 40(4):349–359.
53. Pories WJ, MacDonald KG Jr, Morgan EJ, et al. Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr.* 1992;55(suppl 2):582S–585S.
54. Magro DO, Geloneze B, Delfini R, et al. Long-term weight regain after gastric bypass: a 5-year prospective study. *Obes Surg*. 2008;18(6): 648–651.
55. van Hout GC, Verschure SK, van Heck GL. Psychosocial predictors of success following bariatric surgery. *Obes Surg.* 2005;15(4):552–560.
56. Wilson GT, Grilo CM, Vitousek KM. Psychological treatment of eating disorders. *Am Psychol.* 2007;62(3):199–216.
57. Grilo CM, Masheb RM, Brownell K, et al. Randomized comparison of cognitive behavioral therapy and behavioral weight loss treatments for obese patients with binge eating disorder: 12-month outcomes. Presented at: World Congress of Behavioral and Cognitive Therapy, October 25–27, 2007; Barcelona, Spain.
58. de Zwaan M, Mitchell JE, Swan-Kremeier L, et al. A comparison of different methods of assessing the features of eating disorders in post-gastric bypass patients: a pilot study. *Eur Eat Disord Rev.* 2004;12(6): 380–386.
59. Keel PK, Crow S, Davis TL, et al. Assessment of eating disorders: comparison of interview and questionnaire data from a long-term follow-up study of bulimia nervosa. *J Psychosom Res.* 2002;53(5):1043–1047.
60. Kuczmarski MF, Kuczmarski RJ, Najjar M. Effects of age on validity of self-reported height, weight, and body mass index: findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *J Am Diet Assoc.* 2001;101(1):28–34 [quiz 35-6].
61. Stunkard AJ, Albaum JM. The accuracy of self-reported weights. *Am J Clin Nutr.* 1981;34(8):1593–1599.
62. Gueorguieva R, Krystal JH. Move over ANOVA: progress in analyzing repeated-measures data and its reflection in papers published in the *Archives of General Psychiatry*. *Arch Gen Psychiatry.* 2004;61(3):310–317.
63. Glinski J, Wetzler S, Goodman E. The psychology of gastric bypass surgery. *Obes Surg.* 2001;11(5):581–588.