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Association between Binge Eating Disorder and Changes in Cognitive Functioning Following Bariatric Surgery

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Abstract

Evidence suggests that both obesity and binge eating disorder (BED) may be associated with deficits in cognitive functioning. The purpose of this study was to examine whether a lifetime history of BED would be associated with changes in several domains of cognitive functioning (attention, executive function, language, and memory) following bariatric surgery. Participants were 68 bariatric surgery patients who completed a computerized battery of cognitive tests within 30 days prior to undergoing surgery and again at a 12-month postoperative follow-up. Results revealed that on the whole, participants displayed improvements from baseline to follow-up in attention, executive function, and memory, even after controlling for diagnostic history of depression; no changes were observed for language. However, individuals with and without a history of BED did not differ in changes in body mass index or in the degree of improvement in cognitive functioning from baseline to follow-up. Such results suggest that a history of BED does

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Conflict of interest

The authors of this manuscript do not have any conflicts of interest.

Contributors

Dr. Lavender, Mr. Alosco, and Dr. Gunstad led the manuscript development. Drs. Spitznagel, Strain, Devlin, Cohen, Paul, Crosby, Mitchell, Wonderlich, and Gunstad designed and/or implemented the study. All authors contributed to and approved the final manuscript.

not influence changes in cognitive functioning following bariatric surgery. Future research will be needed to further clarify the role of BED in predicting cognitive function over time.

Keywords

cognition; obesity; bariatric surgery; binge eating disorder

1. Introduction

Binge eating disorder (BED) is characterized by the recurrence of binge eating episodes involving the consumption of an objectively large amount of food with a simultaneous subjective sense of loss of control over eating (American Psychiatric Association, 2013). BED is distinguished from other eating disorders that involve binge eating (e.g., bulimia nervosa) by the absence of regularly occurring inappropriate compensatory behaviors such as purging, excessive exercise, or laxative/diuretic misuse. BED also tends to differ from the other eating disorders in terms of its prevalence, demographics, and certain clinical correlates. For instance, compared to anorexia nervosa and bulimia nervosa, BED is more prevalent in community samples, is more gender balanced in terms of prevalence, tends to have a later age of onset, and is associated with overweight or obese status (Grucza et al., 2007; Hudson et al., 2007; Kessler et al., 2013; Striegel-Moore et al., 2001). Similar to the other eating disorders, however, BED is associated with elevated rates of psychiatric comorbidity, greater role impairments, and lower quality of life (Grilo et al., 2009; Grucza et al., 2007; Kessler et al., 2013; Wonderlich et al., 2009). Although the diagnosis of BED, specifically the criteria for a binge eating episode, requires the consumption of an objectively large amount of food, evidence suggests that it is the loss of control feature of binge eating that may be the more important characteristic (Latner and Clyne, 2008).

Given that loss of control appears to be a central feature of BED (Colles et al., 2008), it is not surprising that individuals with the disorder often display elevated rates of impulsivity and related constructs including novelty seeking, negative urgency, and difficulty maintaining goal-directed behavior in the context of distress, (Grucza et al., 2007; Manwaring et al., 2011; Svaldi et al., 2012; Wu et al., 2013). Additionally, the co-occurrence of impulsive behaviors and certain forms of psychopathology characterized by elevated impulsivity (e.g., substance use disorders) is common in BED (e.g., Dohm et al., 2002; Wilfley et al., 2000; Yip et al., 2011). This pattern of findings suggests that individuals with BED may display a pattern of neurocognitive deficits underlying the disorder, particularly with regard to executive functions and related processes. However, existing findings regarding neurocognitive test performance in BED have been mixed. For instance, some studies have found evidence for deficits in executive function and decision making among obese individuals with BED compared to overweight or obese non-BED controls (Duchesne et al., 2010; Svaldi et al., 2010). Similarly, Müller et al. (2014) found that obese patients with regular binge eating exhibited greater overall food-independent decision making deficits compared to an obese group without regular binge eating, although no differences were found for working memory deficits. One study also found evidence for impaired ability to delay rewards among those with BED compared to non-obese controls (Manwaring et al.,

2011), although findings were more mixed when comparing those with BED to obese non-BED controls. Studies have also examined potential executive deficits using tasks involving food-specific stimuli. For example, Svaldi et al. (2014) compared individuals with BED to weight-matched controls, finding that the BED group displayed an increased stop signal reaction time (an index of response inhibition), as well as greater inhibitory deficits related to exposure to food stimuli. Additionally, Mobbs et al. (2011) found that, compared to obese participants without BED, obese participants with BED made significantly more errors and omissions on a food/body-based inhibitory control task, although the groups did not differ on mental flexibility or attention to body/food stimuli.

However, other studies have not found support for neurocognitive deficits in BED. Wu et al. (2013) found no difference in inhibitory control and decision making between overweight/obese participants with BED and body mass index (BMI)-matched controls, and Davis et al. (2010) similarly found no differences between obese individuals with and without BED in terms of performance on a decision making task, although both groups displayed greater deficits than normal-weight controls. There are numerous factors that may contribute to the mixed findings that have emerged from research examining neurocognitive deficits in BED versus non-BED samples. For instance, many studies utilize different measures that assess similar but not identical constructs (e.g., tasks assessing inhibitory control using a go/no-go paradigm versus a stop signal reaction time paradigm), which may produce conflicting findings due differences in the specific domains of a broader construct that are assessed. Additionally, as noted above, studies may utilize BED-specific (e.g., food- or body-related) or nonspecific stimuli that could produce differing findings if certain aspects of neurocognitive functioning are more heavily implicated in the context of disorder-specific processes (e.g., disinhibition of eating) versus more general processes (e.g., disinhibited behaviors in multiple contexts). Finally, evidence suggests that more generally, elevated BMI and obesity are associated with neurocognitive deficits (Gunstad et al., 2007; Kerwin et al., 2010; Wolf et al., 2007). Thus, given the mixed findings noted above and the independent contribution of overweight/obesity to neurocognitive deficits, the extent to which a BED diagnosis among obese individuals may confer greater risk for impairments in neurocognitive functioning beyond that associated with an elevated BMI remains unclear.

A growing literature has also examined cognitive functioning among individuals seeking bariatric surgery, a group in which a history of BED is fairly common (Kalarchian et al., 2007; Mühlhans et al., 2009). This population is at risk for neurocognitive deficits due to their obese status, as well as due to the presence of certain conditions that are often comorbid with obesity (e.g., depression, diabetes; Austin et al., 2001; van den Berg et al., 2010). Consistent with the broader literature on the association between obesity and cognitive functioning, bariatric surgery patients have been found to display neurocognitive deficits (e.g., Miller et al., 2013). Evidence also suggests that these deficits tend to improve following bariatric surgery (Alosco, Galioto, et al., 2014; Alosco, Spitznagel, et al., 2014; Miller et al., 2013). Given evidence that obesity is associated with neurocognitive deficits, such that higher BMIs are associated with greater deficits, the improvement in neurocognitive functioning following bariatric surgery may be due, at least in part, to weight loss associated with the surgical intervention. However, given the effects of bariatric surgery on a number of systems and functions that have been linked with cognitive functioning (e.g.,

insulin resistance, leptin levels; Ballantyne et al., 2006; Beckman et al., 2010; Park, 2001; Zupancic & Mahajan, 2011), the specific mechanisms underlying these changes remains unclear.

1.1. Current Study

More than one-third of adult men and women in the U.S. are obese (Flegal et al., 2012), a condition which is associated with a variety of negative health consequences (e.g., hypertension, diabetes, cardiovascular disease; Must et al., 2006) and psychosocial impairments (e.g., depressive symptoms, social stigma, reduced quality of life; Kolotkin et al., 2001; Luppino et al., 2010; Puhl & Heuer, 2010). Additionally, evidence suggests that obesity is associated with deficits in neurocognitive functioning and increased risk Alzheimer's disease, vascular dementia, and other neurological changes (Fitzpatrick et al., 2009; Gunstad et al., 2007; Kivipelto et al., 2005; Wolf et al., 2007). Further, as discussed above, there is some evidence that BED may confer additional risk for neurocognitive deficits beyond that associated with obesity alone. However, it is unknown to what extent BED diagnostic status may be associated with differential improvements in neurocognitive functioning following bariatric surgery.

Thus, the goal of the present study was to examine the extent to which a history of BED was associated with differential changes in neurocognitive test performance among bariatric surgery patients from baseline to 12-months post-surgery. The use of a bariatric surgery sample is beneficial for several reasons, including evidence suggesting the presence of neurocognitive deficits among those seeking bariatric surgery, the common occurrence of lifetime BED among those seeking bariatric surgery, and the ability to examine changes in neurocognitive functioning following substantial weight-loss over a relatively brief period of time.

Consistent with existing evidence suggesting that bariatric surgery is associated with improvements across several domains of neurocognitive functioning, it was first hypothesized that participants as a whole would display significant improvements from baseline to 12-month postoperative follow-up in four domains of neurocognitive functioning that have been the focus of prior research using bariatric surgery samples (i.e., attention, executive function, memory, and language). Additionally, given findings suggesting that BED may be associated with neurocognitive deficits independent of obesity (which commonly co-occurs with BED), it was further hypothesized that a history of BED would be associated with less improvement in neurocognitive functioning from baseline to follow-up.

2. Methods

2.1. Participants

A total of 131 bariatric surgery candidates were recruited into a multi-site prospective study examining the neurocognitive effects of bariatric surgery. All bariatric surgery patients were part of the Longitudinal Assessment of Bariatric Surgery (LABS) parent project and were recruited from existing LABS sites (Columbia, Cornell, and Neuropsychiatric Research Institute; Belle, 2007). For study inclusion, bariatric surgery patients were required to be

enrolled in LABS, between 20–70 years of age, and English-speaking. Exclusion criteria included history of neurological disorder or injury (e.g., dementia, stroke, seizures), moderate or severe head injury (defined as >10 minutes loss of consciousness), past or current history of severe psychiatric illness (e.g., schizophrenia, bipolar disorder), past or current history of alcohol or drug abuse (defined by DSM-IV criteria), history of a learning disorder or developmental disability (defined by DSM-IV criteria), or impaired sensory function. Within the sample, just one bariatric surgery patient underwent a gastric banding procedure and thus no comparisons for type of surgery were conducted.

The present sample represents individuals that completed baseline and 12-month postoperative cognitive testing and had complete baseline BED history data. Thus, due to missing data at the time points and/or attrition the sample size for the current study was reduced from 131 to a final sample size of 68. The sample averaged 42.93 ($SD = 10.74$) years of age and was 89.7% female. See Table 1 for baseline demographic and clinical characteristics. Participants excluded were not significantly different from the remaining sample in terms of age ($t(129) = 0.511, p = 0.61$) or baseline BMI ($t(120.22) = 7.52, p = 0.50$). However, participants excluded were more likely to be males ($\chi^2(1, N = 131) = 7.40, p = 0.03$).

2.2. Procedure

All procedures were approved by Institutional Review Boards at each site and all participants provided written informed consent for their involvement in the study. Bariatric surgery patients completed a series of self-report measures and a computerized cognitive test battery within 30 days prior to and 12 months post-surgery. Each participant's height and weight was measured at both time points. Medical and demographic characteristics were ascertained through participant self-report and medical record review conducted by research staff to corroborate and supplement participant self-report.

2.3. Measures

2.3.1 History of BED—Lifetime diagnoses of BED were established using the Structured Clinical Interview for the *DSM-IV* Axis I disorders (SCID; First et al., 1996). Participants were categorized into one of two groups: those with ($n = 20$) or without a lifetime history of BED ($n = 48$). Lifetime history of BED was examined in order to include all individuals that exhibit possible cognitive vulnerabilities due to the effects of any history of BED. We did not investigate current BED status, as current BED status would exclude asymptomatic individuals at the time of presurgical assessment that may have had a previous history of BED that has indeed led to heightened vulnerability to cognitive dysfunction. Nevertheless, of the sample, only 6 participants met past-month criteria for BED on the SCID. This small sample precludes performance of formal analyses examining current BED status.

2.3.2 Cognitive Function—The IntegNeuro cognitive test battery was used to assess cognitive functioning. This cognitive test battery assesses performance in multiple cognitive domains and can be completed in 45–60 minutes. Following completion of the IntegNeuro, individual scores on each measure are compared to a normative sample to automatically derive standardized scores that take into account age, gender, and premorbid intelligence

(Brain Resource Company, Ltd). This instrument demonstrates excellent psychometric properties such as convergent validity with standardized neuropsychological measures ($r = 0.53$ to $r = 0.77$) and test-retest reliability across the tasks ranging from 0.52 to 0.89 (Paul et al., 2005; Williams et al., 2005). It has also been used in past studies examining cognitive functioning in obesity (Gunstad et al., 2007). The cognitive domains included attention/executive function, memory, and language.

2.3.2.1 Attention/Executive Function

Digit Span Total: This test taps into aspects of executive function (e.g., working memory) and is well known to be a sensitive clinical measure of basic auditory attentional processes. Participants are presented with a series of digits on the touch-screen, separated by a one-second interval. The subject is then immediately asked to enter the digits on a numeric keypad on the touch-screen. The number of digits in each sequence is gradually increased from 3 to 9, with two sequences at each level. Participants are then asked to enter the digits in a backward sequence. The total number of correct forward and backward trials was used in the current analyses.

Switching of Attention: This test is a computerized adaptation of the Trail Making Test A and B (Reitan, 1958). Participants are first asked to touch a series of 25 numbers in ascending order as quickly as possible. Then, an array of 13 numbers (1–13) and 12 letters (A–L) is presented. Participants are asked to touch numbers and letters alternately in ascending order. The first part of this test assesses attention and psychomotor speed and the second part assesses executive function.

Verbal Interference: This task taps into the ability to inhibit automatic and irrelevant responses and demonstrates similarities to the Stroop Color Word Test (Golden, 1978). Participants are first presented with colored words one at a time that include “red,” “yellow,” “green,” and “blue.” Below each colored word is a response pad with the four possible words displayed in black. The subject is required to name the color of each word as quickly as possible, assessing executive functioning. Total number of words correctly identified served as the dependent variable in this study. For a full description of this task, see Paul et al., 2005.

Maze Task: This task is a computerized adaptation of the Austin Maze (Walsh, 1985) and assesses executive function. Participants are presented with a grid (8×8 matrix) of circles on the computer screen. They are asked to identify the hidden path through the grid, starting from the beginning point at the bottom of grid to the end at the top of the grid. A total of 24 correct moves are required to complete the entire maze and distinct auditory and visual cues are presented for correct and incorrect responses. For example, the participant was presented with one tone for an incorrect move and a different tone for a correct move. The purpose was to assess the how quickly the participant can learn and complete the correct maze route. The trial ends when the subject completed the maze twice without error or after 10 minutes has elapsed. The dependent variable is the number of total errors on the task. For a full description of this task (including a graphical depiction), see Paul et al., 2005.

2.3.2.2 Memory

Verbal list-learning: Participants are read a list of 12 words a total of 4 times and asked to recall as many words as possible following each trial. Following presentation and recall of a distraction list, participants are asked to recall words from the original list. After a 20-minute filled delay during which participants completed intervening tasks from the IntegNeuro, participants are again asked to recall target words. Finally, a recognition trial comprised of target words and foils is completed. Total learning, Long Delay Free Recall, and Recognition of these verbal list items were used to assess memory. Specifically, total learning included the sum of the number of words correctly recalled across the four learning trials. Long Delay Free Recall included the total number of words recalled after the 20-minute delay and Recognition consisted of the total number of correctly identified words on the recognition trial (Paul et al., 2005).

2.3.2.3 Language

Letter fluency: This test asks individuals to generate words beginning with a given letter of the alphabet for 60 seconds. A different letter is used for each of the three trials. Total number of correct words generated across the three trials served as the dependent variable.

Animal fluency: Participants are asked to generate as many animal names as possible in 60 seconds. Total correct served as the dependent variable.

2.4. Statistical Analyses

All raw scores of neuropsychological measures assessing cognitive functioning were transformed to T-scores (a distribution with a mean of 50, and a standard deviation of 10) using existing normative data correcting for age, gender, and premorbid intelligence. Consistent with clinical interpretation, a T-score < 35 was reflective of impairment. A composite score was computed for each of the domains (e.g., attention, executive function, memory, and language) that consisted of the mean of T-scores of the respective neuropsychological measures that comprise the domains.

Descriptive analyses were first performed to characterize the demographic, medical, and cognitive status of the current sample. Given the high rates of depression in bariatric surgery patients and individuals with BED, diagnostic history of depression was included as a covariate in all analyses that examined cognitive function. To determine possible medical covariate inclusion, independent samples *t*-tests and chi-square analyses were performed to examine differences between those with and without a history of BED on BMI, diagnostic history of hypertension, type 2 diabetes mellitus (T2DM), sleep apnea, chronic obstructive pulmonary disease (COPD), and hyperlipidemia.

Analysis of covariance (ANCOVA) investigated differences between those with and without a history of BED on baseline cognitive test performance for each domain (i.e., attention, executive function, memory, and language). Repeated measures analysis of variance (ANOVA) was used to examine changes in BMI from baseline to 12-months postoperative for the overall sample, as well as for those with and without a history of BED. Of note, due to missing height and weight data at the 12-month follow-up, the sample size for analyses

examining BMI over time was reduced to 64. Repeated measures were again conducted to determine changes in cognitive functioning in each domain from baseline to 12-month follow-up. For each cognitive domain, history of BED was also entered as a grouping variable in the repeated measures ANOVA models in order to examine group differences in changes in neuropsychological test performance between those with and without a history of BED.

3. Results

3.1. BED History and Demographic/Clinical Characteristics

Of the sample, 29.4% met subthreshold or full criteria for a lifetime history of BED. Independent samples *t*-tests and chi-square analyses showed no significant group differences between those with and without a history of BED in terms of age ($t(66) = 0.14, p = 0.89$), gender ($\chi^2(1, N = 68) = 3.25, p = 0.07$), education ($t(66) = -1.09, p = 0.28$), hypertension ($\chi^2(1, N = 68) = 0.17, p = 0.68$), T2DM ($\chi^2(1, N = 68) = 0.14, p = 0.71$), sleep apnea ($\chi^2(1, N = 68) = 2.14, p = 0.14$), COPD ($\chi^2(1, N = 68) = 0.86, p = 0.35$), or hyperlipidemia ($\chi^2(1, N = 68) = 1.49, p = 0.22$). However, participants with a history of BED were more likely to also have a diagnostic history of depression ($\chi^2(1, N = 68) = 7.22, p = 0.01$). Indeed, only diagnostic history of depression was included as a covariate. See Table 1 for a full summary of medical and demographic characteristics of the sample.

In the overall sample, the average baseline BMI was 46.53 ($SD = 6.10$) kg/m², placing the sample in the severely obese category. The 12-month follow-up mean BMI for the sample was 30.10 ($SD = 5.13$) kg/m², placing the sample in the obese category and representing a significant decrease in BMI from baseline to 12-months postoperative follow-up ($F(1,63) = 936.10, p < 0.001$). BMI at baseline did not significantly differ between those participants with and without a history of BED ($F(1, 66) = 0.65, p = 0.42$, partial eta-squared = 0.01). Likewise, when BED was entered as a grouping variable, there were no between BED group differences on pre- to postoperative changes in BMI ($F(1, 62) = 1.33, p = 0.25$, partial eta-squared = 0.02). See Table 2.

3.2. Cognitive Functioning at Baseline and 12-Months Post-Surgery

See Table 3 for cognitive test performance at baseline and at the 12-month postoperative follow-up. In the overall sample, when using a T-score cutoff of < 35 , baseline impairments were common in memory and language abilities. Specifically, 16.2% of the sample exhibited impairments on total learning, 11.8% on long delayed free recall, and 16.2% on the recognition task. For language, 13.2% of the sample demonstrated impairments on the letter fluency task. Deficits in baseline attention/executive function were not as common. At 12-months postoperatively, impairments on cognitive testing were less prevalent on most neuropsychological measures. Results from the repeated measures ANOVA that controlled for diagnostic history of depression showed that cognitive function improved from baseline to 12-month follow-up in the following domains: Attention ($F(1,66) = 19.48, p < 0.001$, partial eta-squared = 0.23), executive function ($F(1,66) = 23.43, p < .001$, partial eta-squared = 0.26), and memory ($F(1,66) = 20.60, p < .001$, partial eta-squared = 0.24). This pattern did not emerge for language ($F(1,66) = 0.10, p = 0.75$, partial eta-squared = 0.001). Of note,

BMI did not correlate with cognitive function in any domain at either time points ($p > 0.05$ for all).

3.3. BED History and Cognitive Functioning

Table 3 also presents baseline and 12-month cognitive test performance in bariatric surgery patients with and without a history of BED. Baseline rates of cognitive impairment were also prevalent in both the BED and no BED subgroups; however, for both groups impairments on many of the measures across the domains became less common after surgery. Analysis of covariance controlling for diagnostic history of depression showed no significant differences between those with and without a history of BED on baseline cognitive test performance in attention ($F(1, 65) = 0.07, p = 0.80$, partial eta-squared = 0.001), executive function ($F(1, 65) = 0.05, p = 0.83$, partial eta-squared = 0.001), memory ($F(1, 65) = 0.23, p = 0.64$, partial eta-squared = 0.003), or language ($F(1, 65) = 0.00, p = 0.99$, partial eta-squared = 0.000). Further, in examining potential differences in changes in cognitive functioning based on the presence of a lifetime BED diagnosis, the BED history X time interaction was similarly found to be nonsignificant for all of the cognitive domains after controlling for diagnostic history of depression: Attention ($F(1, 65) = 1.11, p = 0.30$, partial eta-squared = 0.02), executive function ($F(1, 65) = 1.13, p = 0.29$, partial eta-squared = 0.02), memory ($F(1, 65) = 0.98, p = 0.33$, partial eta-squared = 0.02), and language ($F(1, 65) = 2.73, p = 0.10$, partial eta-squared = 0.04). Follow-up analyses also showed that current BED status also did not influence pre- and post-surgery cognitive function in any domain ($p > 0.10$ for all).

4. Discussion

The primary objective of the current study was to examine the degree to which a history of BED would be associated with changes in cognitive functioning in bariatric surgery patients from baseline to 12-month postoperative follow-up. Evidence supports the presence of deficits in neurocognitive performance across multiple domains in both obesity (Kerwin et al., 2010; Wolf et al., 2007) and eating disorders (Lena et al., 2004), including some mixed evidence for BED (Davis et al., 2010; Duchesne et al., 2010; Manwaring et al., 2011; Svaldi et al., 2010; Wu et al., 2013). As hypothesized, participants as a whole displayed significant improvements in test performance from pre-surgery baseline to post-surgery follow-up across three of four cognitive domains: attention, executive function, and memory; improvements were not seen in the domain of language. However, in contrast to expectations, the BED and non-BED groups did not significantly differ in their respective degree of improvements over time. Specifically, while both groups displayed significant improvements from baseline to follow-up, there were no significant group differences in the degree of those changes.

Although some studies have found evidence of neurocognitive deficits in BED, the results of this study suggest that, among bariatric surgery patients, those with and without a history of BED do not differ in the extent to which they exhibit postoperative improvements in cognitive functioning. The existing literature on the impact of BED on neurocognitive functioning is mixed, likely due in part to the confound of obesity, which commonly co-

occurs with BED. The current findings are consistent with previous research suggesting no differences among obese individuals with and without BED (Davis et al., 2010; Wu et al., 2013). Importantly, these findings build upon the existing literature by providing evidence that a history of BED is also unrelated to the changes in neurocognitive performance that accompany the extensive weight-loss and other physical adaptations that follow bariatric surgery.

There are several possible explanations for the unexpected findings in the current study. Of particular relevance, and in contrast to much of the previous research examining the relation of BED status to cognitive performance, the current sample was comprised of bariatric surgery patients. As such, the mean baseline BMI of participants was notably higher than in several prior studies. Given the negative association that has been identified between BMI and neurocognitive performance, it may be that the improvements in cognitive functioning related to postoperative weight-loss may have outweighed any subtle group differences that may exist among those with and without a history of BED. This would be suggestive of a smaller independent contribution of BED to neurocognitive deficits versus a potentially larger contribution of obesity. Additional studies examining neurocognitive deficits in BED using patient and control samples displaying a range of BMIs (reflecting normal weight, overweight, and obese categories) may be useful in better clarifying this issue, as it is possible that BED may play a more significant role in the cognitive function of those individuals with lower BMIs.

A second explanation for this finding may be that the sample was recruited with comparatively limited exclusion criteria. Whereas certain previous studies (Davis et al., 2010; Duchesne et al., 2010; Wu et al., 2013) have excluded participants based on the presence of numerous conditions that may also affect neurocognitive functioning (e.g., history of major depression, borderline personality disorder, diabetes), the current study discounted participation based on a fewer number of conditions, most of which were strongly relevant to neurocognitive functioning (e.g., history of neurological disorders or a head injury). Thus, although the current sample may better reflect the broader population of bariatric surgery patients, findings regarding changes in cognitive functioning related to BED status may have been influenced by these comorbid conditions at baseline or by changes in these conditions during the follow-up period.

Although there were several strengths to the current study, particularly the longitudinal follow-up and the examination of multiple domains of cognitive functioning, there were also several limitations that should be noted. An important limitation was that participants in the BED group had a lifetime diagnosis of BED, but were not required to have a current diagnosis of the disorder. It is possible that the current presence of the disorder could impact cognitive functioning in ways that a history of the disorder would not, such as repeated episodes of loss of control over eating having negative impacts on executive function or other cognitive processes. Alternatively, recovery from BED may be an indication of improved functioning in certain areas, such as inhibitory or attentional processes. Moreover, while the SCID is a reliable assessment of psychopathology, categorization of participants into groups (e.g., those with and without a history of BED) precludes examination of the spectrum of symptoms that comprise BED and their differential effects on cognitive

function. For example, assessment of current symptomatology using a dimensional approach would permit investigation of individuals that may not meet full threshold criteria for a diagnosis of BED, but exhibit subclinical BED symptoms that may play a role in post-surgery cognitive outcomes that was not captured in the current study. Indeed, future studies examining the impact of a current BED diagnosis would be important to address this limitation. Relatedly, accounting for the duration of BED may prove useful in future research, as it is possible that the extent of neurocognitive deficits associated with BED may differ in nature or degree based upon the duration of the illness.

This study is limited in several other ways. Given that psychiatric diagnoses may sometimes be a rule-out for bariatric surgery, it is possible that some participants may have denied or minimized reports of BED symptoms at baseline out of concern for being denied the opportunity to undergo surgery. Such response biases could have impacted the accuracy of group classifications, and thus masked possible group differences. Third, while several important domains of neurocognitive functioning were investigated in this study, other domains that have received attention in prior studies (e.g., inhibitory processes, decision-making, delay discounting) were not examined. Future studies incorporating measures of these other potentially relevant neurocognitive constructs are thus recommended. Relatedly, the current study utilized a cognitive test battery that relied on non-BED specific stimuli and material. As such, findings may have differed if BED-specific stimuli were used (e.g., food or body-related stimuli). Further, the vast majority of the current participants were female, thus the extent to which BED status may affect neurocognitive functioning differentially by gender will need to be addressed in future research. Finally, due to the lack of a control group in the current study, it was not possible to discount that improvements from pre- to post-surgery may reflect training effects resulting from the repeated administration of the cognitive test battery.

In sum, the results of the current study revealed that on the whole, participants displayed a significant improvement following bariatric surgery across most domains of cognitive functioning that were examined, although a lifetime history of BED was not associated with differential changes in neurocognitive test performance over time. Additional research will be needed to replicate these results, particularly controlling for comorbid medical conditions and other psychiatric disorders that may affect cognitive functioning, as well as utilizing experimental tasks to more directly assess domains not investigated in the current study (e.g., delay discounting, decision making, inhibition). Further, research exploring how bariatric surgery results in changes in neurocognitive functioning will be needed to clarify the mechanisms underlying these improvements.

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Highlights

- Obesity and binge eating disorder (BED) may be associated with cognitive deficits
- Bariatric surgery patients completed cognitive tests pre-surgery and 1-year post-surgery
- Overall, participants improved in attention, executive function, and memory, but not in language
- Individuals with and without a history of BED did not differ in BMI change or degree of improvement in cognitive functioning

Table 1

Baseline Demographic and Clinical Characteristics of Bariatric Surgery Patients

Variables	Overall Sample (N = 68)	BED (n = 20)	No BED (n = 48)	t/X ² statistic ^a	P-value
Age, mean (SD)	42.93 (10.74)	43.04 (11.01)	42.65 (10.34)	.14	.89
Education, mean (SD)	13.59 (1.28)	13.85 (1.31)	13.48 (1.27)	-1.09	.28
Sex (% women)	89.7	85.4	100.0	3.25	.07
Hypertension (%)	41.2	39.6	45.0	.17	.68
Type 2 Diabetes (%)	22.1	20.8	25.0	.14	.71
Sleep Apnea (%)	36.8	31.3	50.0	2.14	.14
Hyperlipidemia (%)	48.5	60.0	43.8	1.49	.22
COPD (%)	3.0	0.0	4.2	.86	.35
Depression (%)	60.3	85.0	50.0%	7.22	.01

^a *Note.* Statistical comparison is between the lifetime history of BED vs. No lifetime history of BED group; BED = Lifetime history of Binge Eating Disorder; COPD = Chronic obstructive pulmonary disease.

Table 2

Baseline and 12-Month Postoperative Body Mass Index (BMI) among Participants with and without a Lifetime History of BED

Group	Baseline BMI	12-Month BMI	F statistic
BED (<i>n</i> = 19)	47.53 (5.52)	30.49 (5.81)	281.36 *
No BED (<i>n</i> = 45)	45.65 (4.84)	29.93 (5.13)	659.68 *

* *Note.* $p < 0.001$; BED = Binge Eating Disorder; analyses examine lifetime history of BED.

Table 3

Baseline and 12-Month Neuropsychological Test Performance (N = 68)

Neuropsychological Domains/Tests	Baseline <i>M (SD)</i>			% T-score < 35			12-months <i>M (SD)</i>			% T-score < 35		
	Total	BED	No BED	Total	BED	No BED	Total	BED	No BED	Total	BED	No BED
Attention/Executive Function												
Digit Span Total	50.77 (8.23)	51.43 (8.34)	50.50 (8.26)	1.5	0.0	2.1	53.74 (11.05)	55.86 (8.07)	52.85 (12.04)	0.0	0.0	0.0
SOA-A	56.45 (12.95)	56.93 (10.79)	56.25 (13.85)	7.4	5.0	8.3	64.44 (12.80)	56.93 (10.79)	64.08 (12.39)	2.9	5.0	2.1
SOA-B	54.35 (13.03)	55.51 (8.53)	53.87 (14.56)	5.9	0.0	8.3	60.01 (12.77)	65.30 (14.06)	58.70 (14.36)	7.4	0.0	10.4
Verbal Interference	55.47 (13.83)	52.72 (9.73)	51.49 (10.76)	7.4	10.0	6.3	64.04 (12.56)	57.01 (7.77)	64.04 (14.08)	1.5	0.0	2.1
Maze Errors	51.85 (10.41)	55.28 (13.19)	55.55 (14.22)	7.4	5.0	8.3	55.44 (8.71)	64.06 (8.10)	54.79 (9.07)	1.5	0.0	2.1
Memory												
Total Learning	44.24 (12.21)	44.57 (16.62)	44.10 (10.04)	16.2	10.0	18.8	50.84 (12.40)	52.14 (13.08)	50.30 (12.21)	7.4	10.0	6.3
LDPR	46.89 (9.45)	48.85 (10.55)	46.07 (8.94)	11.8	10.0	12.5	53.25 (9.01)	55.30 (7.35)	52.39 (9.55)	4.4	0.0	6.3
Recognition	42.56 (10.05)	42.54 (12.84)	42.57 (8.80)	16.2	15.0	16.7	49.24 (9.52)	51.76 (8.79)	48.19 (9.71)	5.9	0.0	8.3
Language												
Verbal Fluency	47.06 (11.67)	48.67 (8.18)	46.23 (11.67)	13.2	10.0	14.6	47.72 (10.60)	50.82 (8.74)	46.43 (11.12)	11.8	0.0	14.6
Animals	50.67 (9.94)	48.67 (8.18)	51.50 (10.55)	2.9	5.0	2.1	50.36 (9.72)	51.24 (10.98)	50.00 (9.24)	5.9	5.0	6.3

Note. Test scores are reported as T-Scores. SOA = Switching of Attention; LDPR = Long Delay Free Recall; Sample size for BED = 48 and No BED = 20; BED = lifetime history of binge eating disorder