Association between binge eating disorder and changes in cognitive functioning following bariatric surgery

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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 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.

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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; Wille 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; Mühlhaas 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 et al., 2014a; Alosco et al., 2014b; Miller et al., 2013). Given findings suggesting 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., 1999) and psychosocial impairments (e.g., depressive symptoms, social stigma, reduced quality of life; Kolotkin et al., 2001; Luppino et al., 2010; Puhl and Heuer, 2010). Additionally, evidence suggests that obesity is associated with deficits in neurocognitive functioning and increased risk of 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
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