

ASSESSMENT OF EXECUTIVE FUNCTIONING IN BINGE EATING
DISORDER INDEPENDENT OF WEIGHT STATUS

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ABSTRACT

Executive functioning (EF) problems may serve as vulnerability or maintenance factors for Binge-Eating Disorder (BED). However, it is unclear if EF problems observed in BED are related to overweight status or BED status. The current study extends this literature by examining EF in overweight-BED ($n=32$), normal-weight BED ($n=23$), overweight healthy controls ($n=48$), and normal-weight healthy controls ($n=48$). Participants were administered an EF battery which utilized tests from the National Institutes of Health (NIH) toolkit and Delis-Kaplan Executive Function System (D-KEFS). After controlling for years of education and minority status, overweight individuals with and without BED performed more poorly than normal-weight individuals with and without BED on a task of cognitive flexibility ($p < 0.01$) requiring generativity and speed and on psychomotor performance tasks ($p < 0.01$). Normal-weight and overweight BED performed worse on working memory tasks compared to normal-weight healthy controls ($p = 0.04$). Unexpectedly, normal-weight BED individuals outperformed all other groups on an inhibitory control task ($ps < 0.01$). No significant differences were found between the four groups on tasks of planning. Our findings support a link between poorer working memory performance and BED status. Additionally, overweight status is associated with poorer psychomotor performance and cognitive inflexibility. Replication of the finding that normal-weight BED is associated with enhanced inhibitory control is needed.

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CHAPTER 1: ASSESSMENT OF EXECUTIVE FUNCTIONING IN BINGE EATING DISORDER INDEPENDENT OF WEIGHT STATUS

Introduction

Binge-Eating Disorder (BED) is the most prevalent eating disorder, affecting approximately 3.5% of the population, and is characterized by recurrent episodes of binge eating accompanied by a loss of control over eating (Hudson, Hiripi, Pope, & Kessler, 2007). BED is highly comorbid with obesity, as over 40% of individuals with BED are obese (Hudson et al., 2007). Moreover, BED appears to confer greater medical, psychological and psychosocial problems than obesity alone, including depression and serious cardiovascular events (Bulik, Sullivan, & Kendler, 2002; Javaras et al., 2008; Succurro et al., 2015).

Preliminary evidence suggests that executive functions (EFs), broadly defined as overlapping frontal lobe processes enabling one to engage in self-initiated and adaptive behaviors, may be compromised in both individuals with BED and obesity (Fitzpatrick, Gilbert, & Serpell, 2013; Voon et al., 2015). EFs are often referred to as “higher level” abilities, due to their role in modulating “lower level” abilities, such as feeding behaviors (Alvarez & Emory, 2006; Gilbert & Burgess, 2008). It has been proposed that difficulties in EF may contribute to both the development and maintenance of binge eating and obesity (Boeka & Lokken, 2008; Cohen, 2008). Specific EF processes that may be associated with binge eating and obesity include cognitive flexibility or the ability to shift mental set; inhibitory control or stopping a pre-potent response towards a stimulus; planning; and working memory, the ability to retain and manipulate mental content and goal-relevant information (Fitzpatrick et al., 2013; Voon et al., 2015).

Preliminary evidence suggests that a spectrum of eating and weight disorders is associated with deficits in EF; however, at this time, the neuropsychology of BED is poorly understood (Manasse et al., 2015). The studies that have investigated EF in a BED sample have produced mixed results. Existing research suggests that individuals who are both obese and have BED perform significantly worse on EF measures of cognitive flexibility, inhibitory control, and planning than obese individuals without BED (Voon et al., 2015). However, these studies differ in what covariates have been controlled and the criteria used for BED status. Additionally, our understanding of the neurocognitive correlates of this disorder that may exist independently of body weight has been limited in part by a lack of studies including full-threshold, normal-weight individuals with BED.

Cognitive flexibility

Impaired cognitive flexibility may lead to an over-focus on eating as a coping strategy. Obese individuals without BED, when compared to normal-weight controls, have shown significant differences in set-shifting, characterized by rigid approaches to changing rules and perseverative thinking and behavior (Boeka & Lokken, 2008; Fagundo et al., 2012; Lokken, Boeka, Yellumahanthi, Wesley, & Clements, 2010; Solís-Ortiz, Gutiérrez-Muñoz, Morado-Crespo, Trejo-Bahena, & Kala, 2016). In studies examining BED, overweight individuals with BED exhibit poorer cognitive flexibility than controls in two of the six studies available (Aloi et al., 2015; Svaldi, Brand, & Tuschen-Caffier, 2010). Other studies showing no significant differences were limited by including either sub-threshold BED group or those without a current diagnosis of BED (Duchesne et al., 2010; Galioto et al., 2012; Lavender et al., 2014; Manasse et al., 2014). To our knowledge, there has been one study that included normal-weight women who

engaged in either subjective or objective binge episodes compared to normal-weight women with no eating disordered behavior (Kelly, Bulik, & Mazzeo, 2013). This study found no group differences on a measure of cognitive flexibility, but did find a significant negative correlation between BMI and performance.

Inhibitory control

Inhibitory control deficits may contribute to eating in response to a trigger as well as the drive to continue eating despite being uncomfortably full. On tasks of inhibition, obese individuals have demonstrated poorer performance when compared to normal-weight individuals (Ariza et al., 2012; Catoira et al., 2016; Fagundo et al., 2012; Gunstad et al., 2007; Smith et al., 2010; Solís-Ortiz, Gutiérrez-Muñoz, Morado-Crespo, Trejo-Bahena, & Kala, 2016; Volkow et al., 2009). While obese individuals without BED show inhibitory control deficits compared to normal-weight individuals, it is possible that these deficits exist on a continuum, with overweight individuals with BED having a more severe inhibitory control problem than overweight individuals without BED. Of the seven studies that have examined this question in BED, three found significant differences, with individuals with BED demonstrating poorer performance on measures of inhibitory control (Manasse et al., 2015; Manasse et al., 2014; Mobbs, Iglesias, Golay, & Van der Linden, 2011). Three studies (Duchesne et al., 2010; Voon et al., 2014; Wu et al., 2013) did not find differences in inhibitory control and one study found that overweight individuals with BED performed better than their weight-matched controls (Mole et al., 2015).

Planning

Difficulties with planning could explain an inability to develop and engage in adaptive behaviors to prevent overeating or bingeing. Poorer performance on measures of planning have been demonstrated in overweight BED when compared to overweight non-BED individuals in two studies utilizing different assessment measures (Duchesne et al., 2010; Manasse et al., 2015). Additionally, significant differences have been found between obese and overweight individuals and matched healthy controls on tasks of organization and planning (Boeka & Lokken, 2008; Lokken et al., 2010; Roberts, Demetriou, Treasure, & Tchanturia, 2007).

Working memory

To date, studies examining working memory deficits in BED samples have been inconclusive, with two studies showing that overweight individuals with BED display poorer working memory capacity than overweight individuals without BED (Duchesne et al., 2010; Manasse et al., 2014) and two studies finding no group differences (Galioto et al., 2012; Muller et al., 2014). Of the seven studies which compared the performances between individuals with obesity and normal-weight controls on working memory tasks, only Volkow and colleagues (2009) found significant differences between groups (Cserjési, Luminet, Poncelet, & Lénárd, 2009; Gonzales et al., 2010; Gunstad et al., 2007; Smith et al., 2010; Spitznagel et al., 2013; Volkow et al., 2009), though a relationship between performance and BMI was found in two of these studies when combined with age (Cserjési et al., 2009; Gunstad et al., 2007).

Psychomotor processing speed

Although not defined as a measure of EF, psychomotor performance, the coordination of a cognitive activity and motor performance is thought to be affected by

subtle disturbances in the EF network (Reijmer et al., 2013). There have been no studies examining psychomotor processing speed in individuals with BED. Of the three studies (Ariza et al., 2012; Cournot et al., 2006; Etou et al., 1989) examining psychomotor processing speed in obesity, two studies showed that obese individuals have slower psychomotor processing speed compared to normal-weight controls (Cournot et al., 2006; Etou et al., 1989).

In summary, the strongest data currently available suggest that overweight BED is associated with greater cognitive inflexibility, poorer inhibitory control, and poorer planning than non-BED overweight control groups (Voon et al., 2015). Similarly, obese individuals without BED show difficulties in those three domains, as well as in psychomotor performance when compared to normal-weight individuals without BED (Cournot et al., 2006; Fitzpatrick, et al., 2013). There is limited data available concerning working memory problems in obesity and BED, and no available data to assess psychomotor processing in BED. Previous studies examining the neurocognitive processes in BED been limited by their use of participant groups with sub-threshold (Kelly et al., 2013; Manasse et al., 2015; Manasse et al., 2014) or past BED. Although comparisons between obese individuals with BED and obese individuals without BED have been made, as well as between obese (non-BED) compared to normal-weight (non-BED) individuals, no studies have included a normal-weight BED group. The current study will be the first to examine the neurocognitive processes of normal-weight BED participants in order to better differentiate the effects of weight from BED status.

The primary aim of the current study is to assess the neurocognitive processes associated with weight status and BED by comparing normal-weight BED, overweight BED, overweight non-BED and healthy weight-non-BED groups. Overweight individuals with BED compared to overweight individuals without BED have in previous studies demonstrated lower performance on measures of cognitive flexibility, inhibitory control, and planning (Duchesne et al., 2010; Manasse et al., 2015; Voon et al., 2015). Therefore, we predict that the overweight BED group will perform significantly worse than the overweight non-BED group on these EF measures. Similarly, overweight individuals without BED have demonstrated poorer performance on measures of cognitive flexibility, inhibitory control, planning, as well as psychomotor processing speed relative to normal-weight individuals (Cournot et al., 2006; Fitzpatrick, et al., 2013). Therefore we predict that the overweight non-BED group will perform significantly worse than the normal-weight non-BED group on those measures. As there are few data available that examine the difference between normal-weight BED and the other three groups of interest, we have not described directional hypotheses for this group. Planned comparisons between diagnostic and weight groups will be conducted and we predict that overweight individuals will perform more poorly than normal-weight individuals and that individuals with BED will perform more poorly than individuals without BED on these EF measures.

Method

Participants

Participants ($N=132$) were part of an ongoing NIH-funded fMRI research study aimed at investigating the relationship among Binge Eating Disorder and cognitive

processes in adult women aged 18 – 65 years. Four different groups were recruited: overweight women with BED, normal-weight women with BED, overweight women without BED, and normal-weight women without BED. Participants were recruited using flyers and local newspaper advertisements and received monetary compensation for their participation; no additional incentive or treatment was provided.

Inclusion/exclusion criteria. Individuals were excluded during an initial phone screening if they reported medical conditions including diabetes, seizure disorder, or history of head injury with a loss of consciousness for more than 10 minutes. Individuals in the healthy control groups were excluded if they had any lifetime psychiatric conditions. Individuals with hypertension, high blood pressure, and hypothyroidism were excluded unless their conditions were managed with medication and had been stable for at least 6 months. BED was diagnosed based on DSM-5 (American Psychiatric Association, 2013) criteria: at least one episode per week for at least three months with loss of control, eating large amount of food, distress regarding binge episodes, lack of compensatory behavior, and three (or more of the following): eating much more rapidly than normal; eating until feeling uncomfortably full; eating large amounts of food when not feeling physically hungry; eating alone because of being embarrassed by how much one is eating; feeling disgusted with oneself, depressed, or very guilty after overeating. Participants in the non-BED group could not have any eating binges in the past three months or a previous eating disorder diagnosis. Normal-weight individuals had a BMI between 18.5 and 24.9 kg/m² and overweight individuals had a BMI \geq 25 kg/m². Four groups were recruited: overweight women with BED (OW-BED) $n = 32$, normal-weight

women with BED (NW-BED) $n = 23$, overweight women without BED (OW-HC) $n = 48$, and normal-weight women without BED (NW-HC) $n = 29$. See Table 1 for details.

Procedure

In the first session, participants were screened for eating disorder pathology and for psychological disorders with interview measures described below by Masters-level PhD candidates (KE, AY, and JMA). Diagnoses were confirmed at a weekly best-estimate meeting with a licensed clinical psychologist (EYC) (Klein, Ouimette, Kelly, Ferro, & Riso, 1994; Kosten & Rounsaville, 1992). Weight and height were taken using a scale with a stadiometer to calculate body mass index (BMI). Neuropsychological measures were administered in the second session in accordance with published manuals.

Measures

Eating Disorders Examination (EDE) Version 16.0. This is a standardized semi-structured interview, measuring the severity and frequency of psychopathology and key behaviors of eating disorders (Cooper & Fairburn, 1987). Participants reported when binge eating became a regular occurrence, which was used to calculate illness duration. The EDE has good internal consistency, high test-retest reliability and inter-rater reliability (Rizvi, Peterson, Crow, & Agras, 2000). The BED module of the EDE reliably assesses objective and subjective binge eating episode frequency (Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Wilfley, Schwartz, Spurrell, & Fairburn, 1997).

The Structured Clinical Interview for DSM–IV (SCID) (First, 2002). This is a semi-structured clinical interview used to assign diagnoses in accordance with the DSM-IV-Text Revision (American Psychiatric Association, 2000) Axis-I disorders. The SCID has adequate interrater reliability (First, 2002).

Raven's Progressive Matrices (RPM). This test assesses non-verbal abstract reasoning and is regarded as an estimate of fluid intelligence (Raven, 1998). Percentile scores based on age were used for the analysis.

Delis-Kaplan Executive Function System (D-KEFS). This is a comprehensive set of tests that assess higher-level cognitive functions (Delis, Kramer, Kaplan, & Holdnack, 2004). The following subtests were administered: Trail Making Test, Verbal Fluency, Design Fluency, Color-Word Interference Test, and the Tower Test. Age-stratified scaled scores were used for the analysis, with scaled scores having a mean of 10 and a standard deviation of 3. See Table 2 for description of tests and associated EF constructs.

NIH Toolbox Cognition Battery. This battery comprises standardized, computer-administered tests assessing cognitive flexibility, inhibitory control, psychomotor processing speed, and working memory (Figley, Asem, Levenbaum, & Courtney, 2016). The NIH Toolbox uses age-stratified standardized scores that have a mean of 100 and a standard deviation of 15. See Table 2 for details and the list of subtests used.

Data Analytic Plan

Age-scaled scores for all the EF measures were used for analyses, thus age was not included as a covariate. Minority status was coded where 0=Caucasian and 1=non-Caucasian (African-American, Hispanic/Latina, Asian or multiracial). A Chi-square test assessed minority status differences (Caucasian vs. non-Caucasian) between the four study groups. If minority composition differed between study groups, minority status was included as a covariate. Likewise, years of education were compared between groups via

one-way ANOVA and if significant groups differences existed, education would also be included as a covariate.

Primary analyses compared the four study groups (i.e., NW-HC, OW-HC, NW-BED, OW-BED) on each of the five EF facets (i.e., cognitive flexibility, inhibitory control, planning, psychomotor performance, working memory) using five multivariate analysis of co-variance (MANCOVA). If the multivariate F was significant, follow-up ANCOVAs on individual EF facets were examined. Where significant univariate mean effects were found, post-hoc Bonferroni ($\alpha = .0083$) corrected comparisons examined group differences, with SPSS-Bonferroni adjusted p -values reported for post-hoc group comparisons. Two-sided planned contrasts, one for weight class (NW versus OW) and one for diagnosis (BED versus HC) were also conducted within each construct. All tests were two-sided where $\alpha = 0.05$.

Results

Preliminary Analyses

Demographic Variables. Groups differed significantly on age ($F(3, 124) = 9.13$, $p < 0.01$), such that OW-BED and OW-HC individuals were older than NW-BED and NW-HC individuals. Age was not included as a covariate because age-stratified standard scores were used for all measures and previous neuropsychological research suggests that statistically controlling for age is unnecessary when using age-stratified standard scores (Salthouse, 2013). Groups differed significantly on BMI ($F(3, 124) = 96.82$, $p < 0.01$), such that OW-BED and OW-HC individuals had greater BMIs than NW-BED and NW-HC individuals. Groups differed significantly on years of education ($F(3, 124) = 8.86$, p

< 0.01), such that OW-BED and OW-HC individuals had fewer years of education than NW-BED and NW-HC individuals. Groups also differed significantly on race ($\chi^2[3] = 38.08, p < 0.01$), with a higher proportion of individuals of minority status in the OW-BED and OW-HC groups than the NW-BED and NW-HC groups. Groups differed significantly on their performance on the Raven's fluid intelligence measure ($F(3, 124) = 8.59, p = 0.02$), such that OW-BED and OW-HC individuals performed more poorly than NW-BED and NW-HC individuals. Performance on the Raven's was significantly correlated with years of education ($r = .41, p < 0.01$). Therefore, years of education and minority status were controlled for in all subsequent analyses. See Table 1 for demographic information.

Psychiatric and medical characteristics of BED participants. All BED participants met DSM-5(American Psychiatric Association, 2013) BED criteria for the last three months. Because, by study definition, non-BED participants could not have any eating binges in the past three months, we only compared the two BED groups on number of eating binges and duration on BED illness. Participants with BED reported an average of 2.98 ($SD = 1.29$) episodes of binge eating per week, with no significant differences in binge eating frequencies between NW-BED and OW-BED ($t(52) = 1.23, p = 0.23$; see table 1). There was a significant difference in illness duration ($t(52) = 2.76, p < 0.01$, see table 1), with NW-BED having a shorter illness duration than OW-BED. Duration of illness and the age of BED participants were significantly correlated ($r = .68, p < 0.01$), as was duration of illness and BMI in BED ($r = .40, p < 0.01$).

Because the non-BED group by definition could not have any psychiatric disorders, we examined the psychiatric comorbidity of the two BED groups. Of the participants with BED, 25 had co-occurring psychiatric disorders, though there were no significant differences between prevalence in the two groups for lifetime history of mood disorders ($\chi^2[1] = .92, p = 0.34$), current mood disorders ($\chi^2[1] = 1.80, p = 0.18$), lifetime history of anxiety disorder ($\chi^2[1] = 2.90, p = 0.09$), current anxiety disorder ($\chi^2[1] = 1.66, p = 0.20$), and lifetime history of substance abuse ($\chi^2[1] = 1.45, p = 0.22$). Number of psychiatric disorders did not significantly correlate with performance on any executive functioning measures ($r_s = -.08$ to $.16, p_s > 0.05$). Prevalence of hypertension ($\chi^2[3] = 3.32, p = 0.35$) and thyroid conditions ($\chi^2[3] = 0.95, p = 0.81$) did not significantly differ between groups, nor did medication use ($\chi^2[3] = 1.12, p = 0.18$). Number of comorbid medical conditions did not significantly correlate with performance on any executive functioning measure ($r_s = -.06$ to $.11, p_s > 0.05$), nor did number of medications ($r_s = -.09$ to $.13, p_s > 0.05$). Therefore, only years of education and minority status were controlled for in all subsequent analyses. See Table 1 for a summary of demographic information.

Correlations between measures. Within each construct, the correlations between measures were significant (see Table 3). Within the cognitive flexibility construct, the DKEFS Trail-Making Number-Letter Switching, DKEFS Verbal Fluency, DKEFS Design Fluency, and NIH Toolbox Dimensional Change Card Sort tasks were all significantly correlated with one another. Of note, almost all utilized measures were correlated with each other, with the exception of the DKEFS Tower Test (which only

correlated with DKEFS Verbal Fluency, NIH TB Picture Sequence, RCFT Copy) and the RCFT Copy (which did not correlate with any of the NIH Toolbox tasks with the exception of the NIH TB Picture Sequence Task).

Executive Functioning analyses

Cognitive Flexibility: A one-way MANCOVA for cognitive flexibility (i.e., DKEFS Trail-Making Number-Letter Switching, DKEFS Verbal Fluency, DKEFS Design Fluency, and NIH Toolbox Dimensional Change Card Sort) revealed a significant multivariate group effect, $F(12, 320) = 1.89, p = .03$; Wilk's $\Lambda = 0.83$, partial $\eta^2 = .06$. Subsequent ANCOVA's showed a significant overall group difference for the DKEFS Design Fluency subtest (see Table 4, Figure 1), but not for the DKEFS Trail-Making Number-Letter Switching, Verbal Fluency, or the NIH Toolbox Dimensional Change Card Sort. On the DKEFS Design Fluency subtest, NW-BED performed better than OW-BED ($p < 0.01$) and OW-HC ($p < 0.01$). The NW-HC group performed better on the DKEFS Design Fluency subtest than OW-BED ($p < 0.01$) and OW-HC ($p = 0.03$). OW-BED and OW-HC did not differ significantly on their performance, nor did NW-HC and NW-BED.

Inhibitory Control: A one-way MANCOVA on inhibitory control (i.e., DKEFS Color Word Interference and NIH Flanker Inhibitory Control tasks) showed a significant multivariate effect of group, $F(6, 246) = 2.34, p = .03$; Wilk's $\Lambda = 0.90$, partial $\eta^2 = .05$. Follow-up ANCOVAS revealed group differences for the NIH Toolbox Flanker Inhibitory Control test (see Table 4, Figure 2). Post-hoc comparisons revealed that NW-BED performed better than each of the other three groups ($ps < 0.01$). There were no

significant group differences for the DKEFS Color Word Interference subtest (see Table 4).

Planning: The one-way MANCOVA for planning using the DKEFS Tower test and RCFT Copy scores as dependent variables revealed no significant multivariate effect of group, $F(6, 246) = 1.06, p = .39$; Wilk's $\Lambda = 0.95$, partial $\eta^2 = .03$.

Working Memory: A one-way MANCOVA on working memory (i.e., NIH Toolbox List Sorting Working Memory and the NIH Toolbox Picture Sequence Memory task) showed a significant multivariate effect of group, $F(6, 246) = 2.98, p < .01$; Wilk's $\Lambda = 0.87$, partial $\eta^2 = .07$. Follow-up ANCOVAS revealed group differences for both tasks (see Table 4). Post-hoc comparisons revealed that NW-HC performed better than the remaining groups on both the NIH Toolbox List Sorting Working Memory and the Picture Sequence Memory task ($ps < 0.01$) (see Figure 2).

Psychomotor Performance: A one-way MANCOVA on psychomotor performance (i.e., DKEFS Trail-making Motor Speed task and the NIH Toolbox Pattern Comparison test) showed a significant multivariate effect of group, $F(6, 246) = 2.67, p = .02$; Wilk's $\Lambda = 0.88$, partial $\eta^2 = .06$. Follow-up ANCOVAS revealed group differences for both tasks (see Table 4). Post-hoc comparisons on the DKEFS Motor Speed (see Figure 1) revealed that NW-BED performed better than OW-BED ($p < 0.01$) and OW-HC ($p < 0.01$). NW-HC also performed better than OW-BED ($p = 0.03$) and OW-HC ($p = 0.02$). On the NIH Toolbox Pattern Comparison task, NW-BED performed better than OW-BED (see Figure 2, $p < 0.01$) and there was a trend for better performance in NW-BED compared to OW-HC ($p = 0.07$) and for NW-HC compared to OW-HC ($p = 0.07$).

Planned Contrasts: Planned contrasts for weight class (NW versus OW) and BED diagnostic status (BED versus non-BED) were also conducted for each tasks within each construct (see Table 5). On the Design Fluency Task, contrasts revealed that normal-weight individuals performed better than overweight individuals and that there were no differences between BED and non-BED groups. On the Flanker Inhibitory Control Task, planned contrasts indicated that NW individuals performed better compared to OW individuals and BED performed better than non-BED. On both working memory tasks, HCs performed better than BED for both subtests. On both psychomotor speed tasks, contrasts suggest that NW individuals performed better compared to OW individuals.

Discussion

The aim of the current study was three-fold: to replicate previous findings demonstrating unique associations between BMI and BED status and EF, to explore how EF profiles may differ with membership to one or both BED and BMI groups, and to characterize the EF profile of the previously unexamined NW-BED group. We confirmed previous findings showing that NW-HC individuals significantly outperformed OW-HC individuals on tasks requiring psychomotor speed (DKEFS Trail-making Motor Speed, NIH Pattern Comparison) or cognitive flexibility (DKEFS Design Fluency). Participants in both BED groups showed significantly poorer performance on tasks of working memory relative to NW-HCs (NIH Toolbox List Sorting Working Memory and Picture Sequence Memory), though OW-HCs also performed significantly poorer than NW-HCs. NW-BED individuals demonstrated significantly greater inhibitory control as measured by the Flanker Inhibitory Control task than all other groups. While younger age and

greater education in NW compared to OW groups may have partially accounted for the performance on a task of inhibitory control for the NW-BED group, these factors were controlled for in the analysis. The co-occurrence of other psychological and medical conditions was not associated with EF findings. Finally, there were no significant differences between groups on planning.

Our findings support a link between poorer psychomotor performance and overweight status (Cournot et al., 2006; Etou et al., 1989). Planned contrasts revealed that weight group was significantly related to performance on three tasks where psychomotor speed and reaction time were central (DKEFS Trail-making Motor Speed, DKEFS Design Fluency, and the NIH Pattern Comparison tasks). Thus, our findings suggest that the EF profiles of those who are NW, even with a diagnosis of BED, are generally characterized by better psychomotor performance than OW, as captured by multiple EF tasks. Higher BMI has been associated with a reduction in white matter integrity (Bolzenius et al., 2015; Figley et al., 2016; Verstynen et al., 2012) and poorer performance on tasks of speed (Bolzenius et al., 2015), suggesting a possible explanation for lower performance of the OW group on psychomotor tasks.

Moreover, NW groups outperformed OW groups on DKEFS Design Fluency, a task of cognitive flexibility which is distinguished from other tasks of cognitive flexibility, such as the Wisconsin Card Sort, by a component of generativity. Weight was a significant factor in performance, with NW individuals generating an average of four more designs in the same time frame as OW individuals. This may be confounded partially by the aforementioned psychomotor abilities required by the task. However, this

task also has attention and set-shifting demands (Suchy, Kraybill, & Larson, 2010); as designs cannot be repeated, an individual must “update” what designs are available to generate with each new design drawn. Similar tasks, such as the Wisconsin Card Sort task, which target set-shifting, have shown that OW groups perform poorer when compared to normal-weight groups (Fitzpatrick et al., 2013; Kelly et al., 2013). The current study is the first to use the DKEFS Design Fluency task in BED and OW groups, further research is needed to clarify whether difficulties with set-shifting are driving group differences and to control for the potential confound of motor speed.

Individuals with BED performed poorly on tasks assessing working memory (i.e., the NIH Toolbox List Sorting Working Memory and Picture Sequence Memory). The performances of both BED groups were below the normative sample on the Toolbox and significantly lower than NW-HCs; additionally, OW-HCs also performed significantly poorer than NW-HCs. Working memory performance is dependent on one’s ability to retain and manipulate target information while ignoring distractors. Past studies have found poorer working memory in OW and obese groups with and without BED (Duchesne et al., 2010; Manasse et al., 2014; Volkow et al., 2009); however, this study is the first to observe such differences in a NW-BED group. This may have important clinical applications, as compromised working memory may lead to the maintenance of binge-eating by allowing distractors to overwhelm self-regulation goals. Further, it may also be that binge consumption of a high-energy, highly palatable foods leads to working memory deficits, as recent research suggests a link between poor diet quality and memory deficits on a task thought to recruit the hippocampus, even among normal weight

participants (Francis & Stevenson, 2011). Rodent models also show that the consumption of a high-energy diet can have significant adverse effects on hippocampal-dependent memory processes, deficits that can be observed even prior to weight gain (Beilharz, Maniam, & Morris, 2015). The foods consumed during a binge, typically characterized by high fat and sugar, may contribute to the difficulties in working memory observed in this study. The role of working memory in eating behavior merits further investigation, especially in light of a recent finding (Houben, Dassen, & Jansen, 2016) in an overweight sample suggesting that working memory training may help to reduce emotional eating.

An additional aim of this study was to characterize the EF processes of the previously unexamined NW-BED group. While the NW-BED group relative to both OW groups exhibited enhanced psychomotor performance and cognitive flexibility, BED status in the NW group had a negative impact on working memory performance. In contrast, the NW-BED group performed significantly better on the Flanker Inhibitory Control task relative to all other groups, even when age and education status was controlled for. The superior performance of NW-BED on the Flanker Inhibitory Control task, which involves a large speed component, may be partially accounted by the overall speed of the NW-BED group, although this was similar to NW-HC. In contrast to a past study that showed that obese individuals with BED did better on motor inhibitory tasks than obese HCs (Mole et al., 2015), we did not observe better performance in our OW-BED group, only in our NW-BED group. It is possible that the superior inhibitory control observed in NW-BED serves as a protective factor, preventing weight gain. Given that

this is the first study to examine this group, our findings with NW-BED require replication.

We found no differences in neurocognitive performance on tasks of planning across all four groups. A previous study found lower performance in overweight individuals with loss-of-control eating in planning on the D-KEFS Tower Test compared to overweight individuals without loss-of-control eating (Manasse et al., 2014). That study did not use a full-threshold BED sample, and included individuals who reported loss of control eating without meeting full criteria for BED, making it difficult to fully compare findings. Moreover, there was a large amount of variability within groups on the Rey Complex Figure Task (RCFT)-Copy, which may have masked differences between groups. The three studies that found differences between obese and normal-weight groups on the RCFT included individuals with diabetes (Boeka et al., 2008; Lokken et al., 2010; Roberts et al., 2007), which may account for the discrepancy in our results, as diabetes has been associated with an increased risk of cognitive dysfunction (Van den Berg, Kloppenborg, Kessels, Kappelle, & Biessels, 2009). Future studies may examine planning in these groups using larger samples and inclusion of comorbid medical disorders.

The present study was limited by lack of a comprehensive, examiner-paced battery of IQ assessment (including verbal intelligence measures). Another limitation of the study was the cross-sectional nature of the design. For example, our data cannot address the question of whether inhibitory control is an antecedent or consequence or covariate of binge-eating. Understanding the longitudinal trajectory of NW-BED and

OW-BED may add to our understanding of the risk factors for obesity and the protective factors against the development of obesity. Despite these limitations, the current study had a number of strengths. Our study is novel in utilizing the NIH Toolbox Cognition module in a sample with eating disorders. Additionally, we used well-validated D-KEFS tasks to examine a number of executive functioning constructs. Additionally, we included the previously unexamined NW-BED group, and controlled for several potential alternative explanatory variables, allowing us to be more confident that detected differences can be attributed to the presence of binge eating.

This study replicated findings in the existing literature showing that OW status is associated with reduced psychomotor performance and reduced cognitive flexibility. Working memory performance was poorer among overweight participants and both normal- and overweight participants with BED compared to normal weight healthy controls. These findings have implications for treatment additions that may increase the efficacy of existing treatments for binge eating, as shown by existing research in overweight samples demonstrating the effects of inhibition and working memory training to reduce food intake. The success of these trainings in OW samples suggests that adding neurocognitive targets to intervention has the potential to improve treatment for BED.

Table 1: Demographic Information

Variable	OW-BED (<i>n</i> = 32)	NW-BED (<i>n</i> = 23)	OW-HC (<i>n</i> = 48)	NW-HC (<i>n</i> = 29)
Age (<i>M, SD</i>)*	36.34 (2.03) ^a	23.34 (0.67) ^b	38.04 (1.78) ^a	24.52 (1.23) ^b
BMI (<i>M, SD</i>)*	34.20 (0.83) ^a	22.93 (0.40) ^b	31.30 (0.56) ^a	21.56 (0.29) ^b
Education Level (<i>M, SD</i>)*	13.84 (2.37) ^a	15.39 (2.04) ^b	15.10 (2.23) ^a	15.21 (1.59) ^b
Race (% , <i>n</i>)*				
White	31.25 (10) ^a	70.00 (16) ^b	33.33 (16) ^a	76.00 (22) ^b
Black	59.38 (19) ^a	8.70 (2) ^b	62.50 (30) ^a	6.90 (2) ^b
Hispanic/Latino	3.13 (1)	0 (0)	2.08 (1)	3.45 (1)
Asian	3.13 (1) ^a	21.74 (5) ^b	2.08 (1) ^a	13.79 (4) ^b
Multiracial	3.13 (1)	0 (0)	0 (0)	0 (0)
Objective Binge Episodes (<i>M, SD</i>)	3.27 (1.33) ^a	2.40 (1.24) ^b	0 (0)	0 (0)
Duration of illness (<i>M, SD</i>)*	14.46 (12.02) ^a	6.87 (5.84) ^b	0 (0)	0 (0)
Psychiatric Comorbidity (% , <i>n</i>)				
Current Mood	3.1 (1)	17.4 (4)	0 (0)	0 (0)
Lifetime Mood	15.6 (5)	26.1 (6)	0 (0)	0 (0)
Current Anxiety	12.5 (4)	26.1 (6)	0 (0)	0 (0)
Lifetime Anxiety	0 (0)	8.7 (2)	0 (0)	0 (0)
Current Substance	0 (0)	0 (0)	0 (0)	0 (0)
Lifetime Substance	6.3 (2)	0 (0)	0 (0)	0 (0)
Medical Comorbidity (% , <i>n</i>)				
Type II Diabetes	0 (0)	0 (0)	0 (0)	0 (0)
Hypertension	3.13 (1)	0 (0)	6.25 (3)	0 (0)
Thyroid Condition	3.13 (1)	0 (0)	4.16 (2)	3.45 (1)
Rate of Medications (% , <i>n</i>)	12.50 (4)	13.04 (3)	10.42 (5)	13.79 (4)
Raven's Progressive Matrices (<i>M, SD</i>)*	27.26 (26.86) ^a	54.70 (26.26) ^b	29.21(20.46) ^a	50.55 (25.68) ^b

Note: Asterisks (*) indicate significant group differences; different superscript letters denote significant group differences. Objective Binge Episodes reported per week; Duration of illness is reported in years.

Table 2: Neuropsychological Tests administered, grouped by construct.

Construct	Test	Description of Test	Scoring Criteria
Cognitive Flexibility	D-KEFS Trail-Making Number-Letter Switching	Participants switch back and forth between connecting numbers and letters in sequential order.	Time to completion
	D-KEFS Verbal Fluency Category Switching	Participants are asked to generate words, switching between two difference semantic categories (fruits and pieces of furniture).	Number of responses in 60 seconds
	D-KEFS Design Fluency Switching	Participants are asked to connect dots, switching between empty and filled dots to generate novel designs.	Number of designs in 60 seconds
	NIH Toolbox Dimensional Change Cart Sort	Participants match test pictures to target pictures, first on one dimension (e.g. shape) and then on the other dimension (e.g. color).	Accuracy and reaction time
Inhibitory Control	D-KEFS Color Word Interference Inhibition	Participants must inhibit proponent response (reading) in order to say color ink as quickly as possible.	Time to completion
	NIH Toolbox Flanker Inhibitory Control	Participants focus on middle arrow while inhibiting attention to surrounding arrows flanking it and respond by selecting direction of middle arrow.	Accuracy and reaction time
Planning	D-KEFS Tower	Participants must move circles on pegs to obtain a target order while following specific rules.	Number of moves to complete trial
	Rey-Ostereith Complex Figure Test - Copy	The subject must copy a complex geometric figure using organizational strategies	Scored on accuracy and location
Psychomotor Performance	D-KEFS Trail-Making Motor Speed	Participant trace over a dotted line as quickly as possible	Time to completion
	NIH Toolbox Pattern Comparison	Participants discern whether two side-by-side pictures are the same or not. The items are designed to be simple to measure processing speed.	Number of responses in 90 seconds
Working Memory	NIH Toolbox List Sorting Working Memory	Recall and sequencing of different visually and orally presented stimuli. Pictures are displayed with accompanying audio recording and written text, and the participant is asked to say the items back in size order	Number of items recalled and sequenced correctly
	NIH Toolbox Picture Sequence Memory	Participants recall increasingly lengthy series of illustrated objects. Sequence length varies increases in difficulty.	Number of items recalled and sequenced correctly

Table 3: Bivariate correlations between Neuropsychological Tests administered.

Subtest	DKEFS Trails: Switching	DKEFS Trails: Motor	DKEFS Verbal: Switching	DKEFS Design: Switching	DKEFS Color Word Inhibition	DKEFS Tower Test	NIH TB Dimensional Sort	NIH TB Flanker Inhibition	NIH TB List Sorting	NIH TB Pattern Comparison	NIH TB Picture Sequence	RCFT Copy
DKEFS Trails: Switching	1	-	-	-	-	-	-	-	-	-	-	-
DKEFS Trails: Motor Speed	.49**	1	-	-	-	-	-	-	-	-	-	-
DKEFS Verbal: Switching	.32**	.17*	1	-	-	-	-	-	-	-	-	-
DKEFS Design: Switching	.43**	.40**	.35**	1	-	-	-	-	-	-	-	-
DKEFS Color Word Inhibition	.48**	.27**	.40**	.44**	1	-	-	-	-	-	-	-
DKEFS Tower Test	0.15	0.09	.18*	0.11	0.06	1	-	-	-	-	-	-
NIH TB Dimensional Sort	.49**	.30**	.21*	.47**	.44**	0.10	1	-	-	-	-	-
NIH TB Flanker Inhibition	.38**	.34**	.23**	.33**	.36**	0.04	.53**	1	-	-	-	-
NIH TB List Sorting	.44**	.23**	.24**	.31**	.41**	0.11	.29**	.18*	1	-	-	-
NIH TB Pattern Comparison	.36**	.28**	.28**	.43**	.42**	0.10	.44**	.41**	.26**	1	-	-
NIH TB Picture Sequence	.32**	.36**	.32**	.35**	.39**	.21*	.39**	.29**	.41**	.26**	1	-
RCFT Copy	.34**	.19*	.21*	.26**	.22*	.21*	0.17	0.16	0.10	0.08	.36**	1

Note: ** Correlation is significant at the 0.01 level (2-tailed). *Correlation is significant at the 0.05 level (2-tailed).

Table 4: Scaled scores on individual subtests, grouped by constructs and accompanying MANCOVA analysis, controlling for Minority Status and Education.

	OW-BED	NW-BED	OW-HC	NW-HC	F	Sig.	η_p^2
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>df =</i> <i>3, 124</i>		
Cognitive Flexibility							
DKEFS Trail-Making Number-Letter Switching	9.39 (3.35)	10.61 (2.37)	10.35 (2.63)	10.38 (1.95)	0.37	0.77	0.01
DKEFS Verbal Fluency Category Switching	11.23 (3.52)	12.30 (3.36)	12.21 (3.56)	13.10 (4.00)	0.50	0.69	0.01
DKEFS Design Fluency Switching	9.74 (3.07) ^a	13.26 (3.45) ^b	10.65 (2.99) ^{a,c}	13.00 (3.12) ^{b,d}	4.03	0.01	0.09
NIH Toolbox Dimensional Change Cart Sort	89.58 (8.56)	95.24 (7.27)	89.64 (10.02)	92.47 (8.11)	1.38	0.25	0.03
Inhibitory Control							
DKEFS Color Word Interference Inhibition	9.19 (3.47)	11.83 (1.99)	9.65 (3.00)	10.69 (2.92)	1.56	0.20	0.04
NIH Toolbox Flanker Inhibitory Control	88.9 (10.46) ^a	99.35 (10.47) ^b	88.67 (11.31) ^{a,c}	91.64 (10.22) ^{a,d}	4.05	0.01	0.09
Planning							
DKEFS Tower Achievement Score	9.52 (2.90)	10.52 (2.71)	9.69 (2.71)	12.97 (2.12)	1.11	0.35	0.03
Rey Complex Figure Copy T-score	35.50 (34.8)	37.80 (35.0)	37.85 (35.67)	38.45 (34.68)	0.54	0.66	0.02
Psychomotor Performance							
DKEFS Trail-Making Motor Speed	10.10 (2.47) ^a	11.78 (2.04) ^b	10.21 (2.35) ^{a,c}	11.45 (1.88) ^{b,d}	4.16	0.01	0.09
NIH Toolbox Pattern Comparison	94.19 (20.8) ^a	108.95 (18.28) ^b	99.63 (20.35) ^{a,b}	102.67 (21.36) ^{a, b}	2.85	0.04	0.06
Working Memory							
NIH Toolbox List Sorting Working Memory	93.46 (13.02) ^a	96.24 (11.16) ^{a,b}	96.24 (12.39) ^{a,c}	106.32 (13.98) ^d	3.14	0.02	0.08
NIH Toolbox Picture Sequence Memory	87.73 (19.11) ^a	99.56 (19.61) ^{a,b}	94.14 (17.02) ^{a,c}	110.79 (18.99) ^d	3.67	0.02	0.08

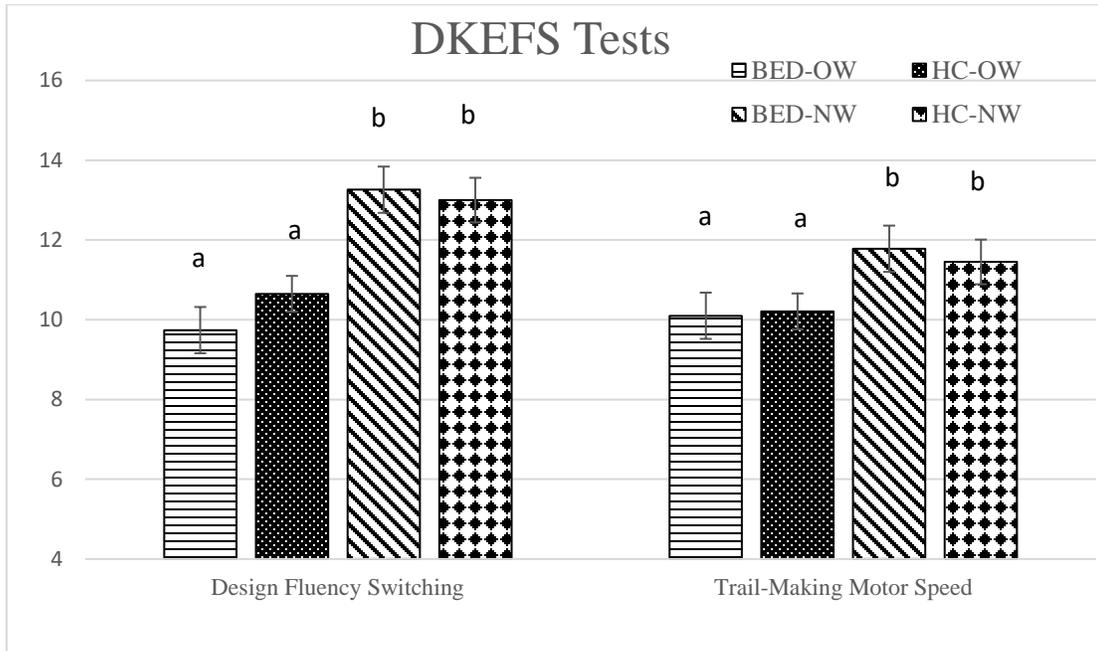
Note: F statistics are of overall group comparisons for univariate ANCOVAs within subtest; Different superscript letters denote significant group differences.

Table 5: Scaled scores on planned contrasts (weight group; diagnostic group) grouped by EF constructs.

	OW	NW	F			BED	HC	F		
	(<i>n</i> = 79)	(<i>n</i> = 51)	<i>df</i> =	<i>Sig.</i>	η_p^2	(<i>n</i> = 53)	(<i>n</i> = 77)	<i>df</i> =	<i>Sig.</i>	η_p^2
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	1, 124			<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	1, 124		
Cognitive Flexibility										
DKEFS Trail-Making Switching	10.03 (2.90)	10.49 (2.15)	0.24	.63	.01	10.00 (2.96)	10.36 (2.38)	.03	.86	<.01
DKEFS Verbal Fluency Switching	11.77 (3.34)	12.92 (3.42)	0.01	.91	<.01	11.77 (3.39)	12.53 (3.40)	.91	.34	.01
DKEFS Design Fluency Switching	10.33 (3.03)	13.13 (3.26)	13.17	.01	.10	11.28 (3.67)	11.53 (3.23)	.06	.80	.00
NIH Toolbox Dimensional Sort	90.04 (9.34)	93.52 (7.69)	1.76	.19	.01	92.36 (8.20)	90.75 (9.29)	1.97	.16	.02
Inhibitory Control										
DKEFS Color Word Interference	9.49 (3.16)	11.23 (2.59)	2.77	.10	.02	10.37 (3.19)	10.04 (2.99)	1.39	.24	.01
Inhibition										
NIH Toolbox Flanker Inhibitory	88.4 (10.69)	94.55 (11.05)	5.82	.02	.05	92.64 (11.52)	89.57 (10.88)	4.92	.03	.04
Planning										
DKEFS Tower Achievement Score	9.71 (2.73)	11.92 (9.42)	0.71	.40	.01	10.06 (2.79)	10.93 (7.88)	1.07	.30	.01
Rey Complex Figure Copy	36.62 (35.25)	38.13 (34.75)	2.79	.10	.02	36.62 (35.02)	38.23 (35.18)	0.49	.48	<.01
Psychomotor Performance										
DKEFS Trail-Making Motor Speed	10.16 (2.38)	11.69 (1.84)	7.34	.01	.06	10.89 (2.39)	10.67 (2.26)	0.26	.61	<.01
NIH Toolbox Pattern Comparison	97.79 (21.22)	105.29 (19.67)	5.56	.02	.05	100.29 (21.68)	101.05 (20.45)	0.01	.95	<.01
Working Memory										
NIH Toolbox List Sorting Working	96.70 (12.67)	102.55 (13.41)	0.28	.60	<.01	93.04 (20.03)	100.86 (19.49)	5.08	.03	.04
Memory										
NIH Toolbox Picture Sequence	92.53 (18.35)	105.85 (19.88)	4.22	.06	.03	93.04 (18.34)	100.85 (19.49)	6.22	.01	.05

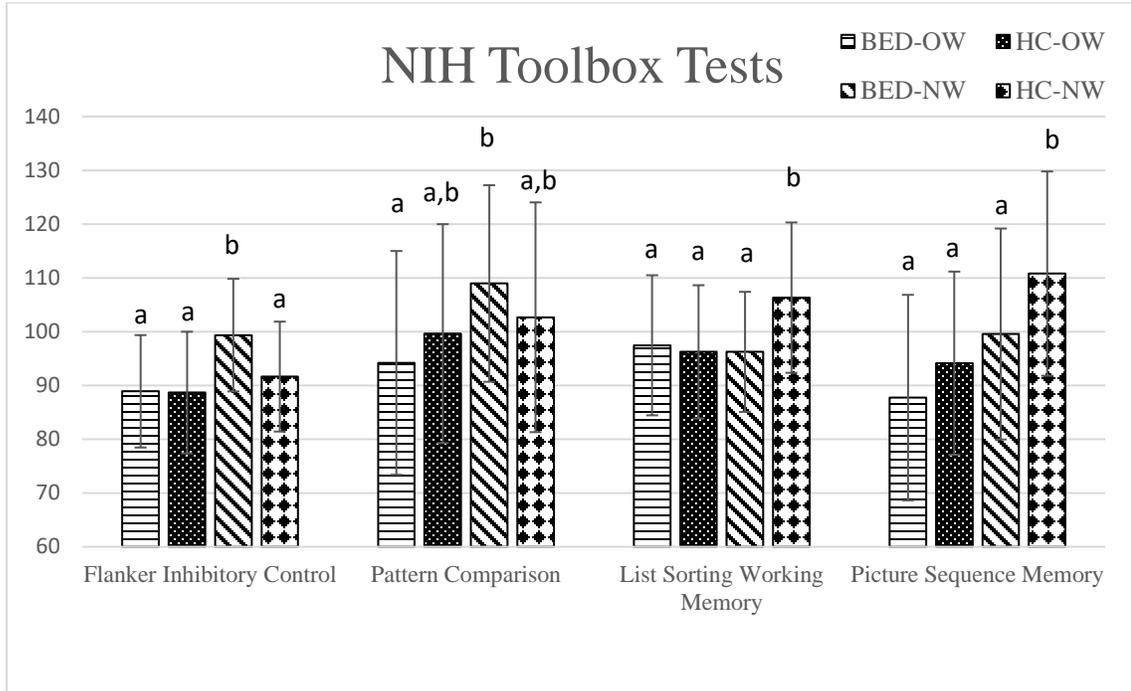
Note: F statistics are of contrast comparisons for univariate ANCOVAs (controlling for minority status and education level) within subtest.

Figure 1: Delis-Kaplan Executive Function System: Design Fluency Switching (Cognitive Flexibility) and Trail-Making Motor Speed (Psychomotor Speed) Scaled Scores



Note: Groups with different subscripts were significantly different, $p < .05$.

Figure 2: NIH Toolbox Scaled Scores: Flanker Inhibitory Control (Inhibition), Pattern Comparison (Psychomotor Speed), List Sorting Working Memory (Working Memory), Picture Sequence Memory (Working Memory)



Note: Groups with different subscripts were significantly different, $p < .05$.

CHAPTER 2: EXTENDED LITERATURE REVIEW - NEUROPSYCHOLOGICAL CORRELATES OF OBESITY AND BINGE EATING DISORDER

Abstract

Binge eating disorder (BED) is the most commonly occurring eating disorder in the United States and individuals with BED are twice as likely to become overweight or obese. While binge eating and obesity are often co-morbid, the majority of obese individuals do not have BED, suggesting that the development of binge eating is at least partially independent of that of obesity. The impact of overeating and high body mass index (BMI) on physical health is well known and widely studied, yet little is known about the consequences of BED or being overweight or obese on cognitive function. A growing body of literature suggests that implicit neuropsychological processes, such as executive functioning, may underlie the regulation of eating behavior and food intake. Neuropsychology provides researchers with normed, widely-used, psychometric indices that can examine the underlying cognitive constructs that may contribute to overeating in these populations. In this review, existing research on the neuropsychology of obesity and BED will be discussed separately in order to elucidate differences and overlaps between BED and obesity. Individual neuropsychological constructs and their potential contributions to overeating and bingeing will be described. Current gaps in the literature are discussed and implications and future directions for research are presented.

Introduction

The prevalence of obesity has increased dramatically over the last several decades with corresponding increases in obesity-related morbidities and associated health costs. Although there are many pathways to obesity, there is substantial evidence that disordered eating can be a significant factor in its development and maintenance (Marcus & Wildes, 2014). While binge eating disorder (BED) affects approximately 3% of the population, it affects approximately 12% of individuals who are obese, and individuals who have BED are twice as likely to be obese (Field et al., 2012; Johnson, Spitzer, & Williams, 2001; Wilfley, Wilson, & Agras, 2003). Given these comorbidities, it is important to elucidate differences and overlaps between BED and obesity. Neuropsychology provides researchers with normed, widely-used, psychometric indices that can examine the underlying cognitive constructs that may contribute to overeating in these populations. In order to gain understanding of necessary targets of interventions in these populations, it is important to see which constructs are more significantly expressed in one group over the other and which are common to both. The following review summarizes the existing research of neuropsychological studies in obese and BED populations, respectively.

Neuropsychological Studies of Obesity

Obesity and Cognitive Functioning: An Overview

Obesity is a significant and increasing challenge to public health and obese individuals often have a reduced life expectancy and increased healthcare needs, mostly due to the elevated risk of co-morbidities which are responsible for 2.5 million deaths a

year (Buchwald et al., 2004). The physical consequences associated with obesity, including elevated risk of developing Type 2 Diabetes, hypertension and other cardiovascular diseases, have been well documented (Avenell et al., 2004) and have influenced public policy relating to the treatment of obesity (NICE, 2014). Obesity is defined by accumulation of a Body Mass Index (BMI) over 30 kg/m², while those who are overweight have a BMI between 25-30 kg/m². While genetics and hormones play a role, obesity results mainly from overeating, although a regular meal time structure is still preserved (O'Rahilly & Farooqi, 2008; Raman, Smith, & Hay, 2013; Wang et al., 2001). Overeating has been assumed to result from an interaction between exaggerated appetitive motivation and weak inhibitory control towards food, two domains of cognitive functioning that may serve to maintain obesity (Appelhans, 2009). The impact of a high BMI on the body is well known and widely studied, yet little is known about the consequences of being overweight or obese on cognitive function (Yesavage et al., 2014). The interaction between high BMI and cognition has been experimentally demonstrated in rodent models by Kanoski and Davidson (2011) to be a “vicious cycle” with changes in cognition arising from excess adipose tissue, which serve to maintain suboptimal dietary choices (p. 65).

Cognitive function refers to the processing, integration, storage and retrieval of information, and includes perception, attention, memory and executive function. Cognitive and executive functions encompass a diverse, overlapping group of higher-level neuropsychological processes that enable an individual to perform autonomous, self-organized, and goal-directed behavior. In essence a new concept, executive function was first described as a “central executive” by Baddeley and Hitch (1974) and later

defined by Lezak (1995) as the dimension of human behavior that deals with “how” behavior is expressed. Executive functioning can be thought of as an umbrella term encompassing a variety of different cognitive domains involved in regulating behavior and adapting to novelty, and includes planning, organizing, problem-solving, attention, set-shifting and inhibitory control (Lezak, 1995; Gilbert & Burgess, 2008). Executive functions are often referred to as “higher level” cognitive abilities due to their role in modulating “lower level” abilities, such as feeding behaviors (Alvarez & Emory, 2006; Gilbert & Burgess, 2008). Lezak added that these behaviors are all necessary for appropriate, socially responsible and effectively self-serving adult conduct (Lezak, 2004). Executive dysfunction includes deficits in planning, heightened impulsivity, cognitive rigidity, and problems with shifting attention (Grafman & Litvan, 1999). Thus, deficits in executive functioning have significant implications for an individual’s ability to carry out self-initiated, goal-directed behavior, such as healthy, regulated eating patterns.

Initial studies examining the impact of obesity on cognitive functioning found that those individuals with a high BMI scored significantly lower on tests of general cognitive ability and had a steeper longitudinal decline in cognitive ability in comparison to their normal weight peers (Dahl et al., 2010; Elias, Elias, Sullivan, Wolf, & D’Agostino, 2005; Smith, Hay, Campbell, & Trollor, 2011). In a review by Smith et al. (2011), the authors found that 14 out of 15 cross-sectional studies in human adult participants reported a negative association between obesity and cognition. Interestingly, executive functioning was the cognitive domain most often affected (11 out of 15 studies reported an association). In particular, they discuss the top-down regulatory role of executive functioning and report that less efficient response inhibition and poorer decision-making

has been found in individuals with an elevated BMI. Indeed, fMRI research on individuals who are overweight and obese has shown reduced brain activation in the lateral and medial prefrontal cortex and orbitofrontal cortex, regions associated with executive control, as well as increased activation in food reward regions (e.g. insula and temporal operculum) during response inhibition towards visual food cues (Cornier et al., 2015; Hendrikse et al., 2015; Puzziferri et al., 2016). These findings have been consistent amongst groups of overweight children and adolescents as well as older obese individuals (Smith et al., 2011).

Of those studies which did report an association between obesity and impaired executive functioning, the direction of the relationship remains unclear and most studies stress that causality may occur in either direction, with either obesity impacting executive functioning or impaired executive functioning increasing the risk of obesity (Boeka & Lokken, 2008). Evidence for the idea that impaired executive functioning increases the risk of obesity includes findings that individuals with executive functioning weaknesses have greater difficulty maintaining weight loss and adhering to post-bariatric lifestyle changes, providing evidence to support the idea that these individuals have a propensity to become obese (Boeka & Lokken, 2008). Additional findings have suggested that making good food choices in modern life requires forethought, planning and good self-regulation in order to avoid overeating (Davis, Patte, Curtis, & Reid, 2010; Duchesne, Paulo, et al., 2010). These are skills which require good executive functioning abilities and so there is a suggestion that impaired executive functioning leads to obesity via poor food choices and lack of planning.

In contrast to this potential causal relationship, additional research suggests that being obese may, even in the absence of other co-morbidities that might impact cognitive abilities, lead to impaired executive functioning (Gunstad et al., 2008; Volkow, Wang, Fowler, & Telang, 2008). For example, individuals with a larger body mass may experience reduced blood flow to certain areas of the brain, therefore reducing the metabolic activity of the brain and negatively affecting cognitive functioning (Gonzales et al., 2010). Obesity may impair glucose and insulin regulation in prefrontal regions (including those associated with dopaminergic systems) and consequently executive functioning (Volkow et al., 2008). The increase in adipocytes seen in obese individuals may also lead to reduced cognitive functioning through increased inflammatory processes (Boeka & Lokken, 2008). Following weight loss and normal weight status, the cognitive deficits associated with obesity improve (Hendricks, Greenway, Westman, & Gupta, 2011; Smith et al., 2011), but do not fully remit (Spitznagel et al., 2013). This suggests that even when body mass is reduced, individuals still show a cognitive deficit, maintaining the “vicious cycle” in those individuals who were previously obese.

The etiology of obesity is unknown and the possibility that there is a dysfunction of the central nervous system in individuals with obesity has been explored in several ways, including studies of neuropsychological test performance. The abilities that are required to make good food and lifestyle choices include planning, problem-solving and decision-making, all of which have been extensively studied through neuropsychological tests. Furthermore, an increasing number of studies assessing the relationship between mechanisms of cognitive processing and certain eating behaviors have been conducted, aiming to achieve a better understanding of the pathophysiology of obesity. The

following review examines the available literature conducted on neuropsychological evaluations comparing overweight or obese adults to normal-weight control groups, without the inclusion of studies examining comorbid conditions. The studies are organized by the construct investigated, beginning with global intellectual functioning, then the components of executive functioning, and finally examining memory.

Intellectual functioning

Intellectual function is a construct hypothesized to measure global cognitive ability. Early research suggested that obese individuals may have diminished cognitive functioning (Chelune, Ortega, Linton, & Boustany, 1986; Gonzales et al., 2010; Gunstad et al., 2008; Lokken, Boeka, Yellumahanthi, Wesley, & Clements, 2010; TIA Sørensen & Sonne-Holm, 1985). Of significant importance, none of these studies found differences in general intelligence between obese individuals and normative or control comparisons when participants were compared within the same education levels (Chelune et al., 1986; Gunstad et al., 2008; Lokken et al., 2010; TIA Sørensen & Sonne-Holm, 1985). Significant methodological issues have been found in additional studies reporting significant differences in cognitive abilities between obese and normal-weight individuals, therefore it cannot be concluded that this effect is fully accounted for by obesity (Halkjær, Holst, & Sørensen, 2003; TIA Sørensen & Sonne-Holm, 1985; Thorkild Sørensen, Sonne-Holm, Christensen, & Krøner-Møller, 1982).

Psychomotor performance and speed

Psychomotor performance, considered the coordination of a cognitive activity and motor performance, is hypothesized to be prominently affected by subtle cognitive disturbances (Reijmer, Leemans, Brundel, Kappelle, & Biessels, 2013). This construct

has been assessed in obese individuals in three studies (Ariza et al., 2012; Cournot et al., 2006; Etou et al., 1989). While there is some evidence that obese individuals do not differ from normal weight control on very simple psychomotor tasks (i.e., symbol digit modalities; Ariza et al., 2012), for most psychomotor tasks obese individuals performed worse than normal weight individuals (Cournot et al., 2006; Etou et al., 1989).

Inhibitory Control

Eating behaviors of obese individuals have been described as compulsive, suggesting a difficulty with inhibition, the ability to refrain from engaging in a proponent and/or rewarding behavior (Van Hout, Verschure, & Van Heck, 2005). A majority of neuropsychological studies on obese individuals have examined this construct and have utilized either the verbal or motor inhibition to target this construct.

Verbal inhibition: Seven studies used the Stroop task (Ariza et al., 2012; Catoira et al., 2016; Fagundo et al., 2012; Gunstad et al., 2007; Smith et al., 2010; Solís-Ortiz, Gutiérrez-Muñoz, Morado-Crespo, Trejo-Bahena, & Kala, 2016; Volkow et al., 2008). In this task, individuals are asked to say the ink color of written words while inhibiting reading the word, e.g. “red” written in blue ink. Four study found that performance on the Stroop task was lower for individuals who were overweight or obese when compared to normal-weight controls (Catoira et al., 2016; Fagundo et al., 2012; Gunstad et al., 2007), and one study found that performance on this task improved following weight loss (Smith et al., 2010). Three studies found no relationship between Stroop performance and BMI (Ariza et al., 2012; Solís-Ortiz et al., 2016; Volkow et al., 2008). Thus, there was no consistent evidence that Stroop performance is impaired for obese individuals. On another verbal inhibition test, the Hayling task, individuals have to inhibit a logical

response to specific sentences. Cserjési, Luminet, Poncelet, and Lénárd (2009) found that obese individuals performed worse than normal weight controls and demonstrated difficulty inhibiting their responses.

Motor inhibition: Four studies used the Go/No-Go task, a variant of the stop signal task (Calvo, Galioto, Gunstad, & Spitznagel, 2014; Hendricks et al., 2011; Mobbs, Iglesias, Golay, & Van der Linden, 2011; Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006). In the Go/No-Go task, participants are asked to respond as quickly as possible on a choice reaction time task, unless a stop signal is presented in which case they must inhibit their response. The Calvo et al. (2014) study found that obese individuals had a significantly slower reaction time than normal weight individuals. One study comparing healthy controls, obese participants, and obese participants with BED used a slightly different version of the task using food-related and body-related stimuli (Mobbs et al., 2011). In this study, both obese groups made more errors than healthy weight controls. The other two studies found that there were no significant differences in reaction times between obese and normal weight groups (Hendricks et al., 2011; Nederkoorn et al., 2006). It would seem that obese individuals show mixed performance on the Go/No-Go task. Overall, studies of inhibitory control including obese individuals indicate that verbal tests of inhibition highlight differences in performance of obese individuals better than motor tests.

Cognitive Flexibility

Cognitive flexibility, or set-shifting, requires the ability to use abstraction, flexibility, and novel problem solving to form concepts and shift back and forth between mental sets in response to the environment (Lezak, 2004). Inefficient set-shifting, which

is characterized by concrete and rigid approaches to changing rules and stereotypic or perseverative thinking and behavior (Friederich & Herzog, 2010), has been associated with obesity. This construct has been examined through the Trailmaking Test, the Wisconsin Card Sorting Task, and the Controlled Oral Word Association Test in overweight and obese individuals.

Trail Making Test (TMT) The TMT is a brief executive task with two parts. Part A asks participants to connect a series of numbers on a page in numerical order; Part B asks participants to draw a line connecting numbers and letters spread across a page in alpha numerical order as quickly and accurately as possible. Part A is used to measure speed of attention and sequencing and part B measures switching, in addition to these factors (Strauss, Sherman, & Spreen, 2006). Nine studies used the TMT, or a variant of it, as an outcome measure (Boeka & Lokken, 2008; Catoira et al., 2016; Cserjési et al., 2009; Fergenbaum et al., 2009; Gonzales et al., 2010; Gunstad et al., 2007; Pierobon, Giardini, Fanfulla, Callegari, & Majani, 2008; Smith et al., 2010; Spitznagel et al., 2013). Four studies demonstrated significantly reduced performance in obese adults compared to comparison groups (Boeka & Lokken, 2008; Cserjési et al., 2009; Fergenbaum et al., 2009; Smith et al., 2010). One study found this effect was only significant for females and that it disappeared when other factors, including education, were controlled for (Boeka & Lokken, 2008). Three others showed no difference between normal, overweight or obese groups (Catoira et al., 2016; Gonzales et al., 2010; Pierobon et al., 2008). Of the two studies using a modified version of the TMT (Gunstad et al., 2007; Spitznagel et al., 2013), both found significant differences between obese and normal weight individuals.

Wisconsin Card Sort Test (WCST) Six studies used the Wisconsin Card Sort Test (WCST) as part of their testing battery (Ariza et al., 2012; Boeka & Lokken, 2008; Fagundo et al., 2012; Lokken et al., 2010; Solís-Ortiz et al., 2016; Volkow et al., 2008). In the original version of this task, participants are given a deck of cards and asked to sort them according to four different key cards (color, shape, number etc.). Participants are given feedback on their performance as they sort through the cards and every few trials the sorting rule changes without the participant's knowledge. Their task is to recognize that a change has happened and start sorting the cards according to the new rule as quickly as possible. Five studies comparing the performance of obese groups to normal weight individuals found that obese individuals made more errors and more perseverations than healthy weight controls (Boeka & Lokken, 2008; Fagundo et al., 2012; Lokken et al., 2010; Solís-Ortiz et al., 2016; Volkow et al., 2008). The Ariza et al. (2012) study found no significant deficits in obese performance on the WCST relative to comparisons, but this study did not control for education levels. Overall, obese individuals do seem to show a tendency to make more perseverative errors on this task.

Controlled Oral Word Association Test (COWAT) In this task, individuals are asked to name as many words as possible beginning with a particular letter within one minute (Benton, 1976). Seven studies utilized a Verbal Fluency task (Ariza et al., 2012; Boeka & Lokken, 2008; Catoira et al., 2016; Cserjési et al., 2009; Gonzales et al., 2010; Smith et al., 2010; Solís-Ortiz et al., 2016). Six studies found no difference in test performance between obese and non-obese individuals (Ariza et al., 2012; Catoira et al., 2016; Cserjési et al., 2009; Gonzales et al., 2010; Smith et al., 2010; Solís-Ortiz et al., 2016), and one study found obese individuals' scores were better than normal weight

individuals (Boeka & Lokken, 2008). Generally, obese individuals show no differences on verbal fluency tasks when compared to control participants and seem to perform equivalently or even better than normed data.

Overall, individuals who are obese show significantly lower performances on the WCST, variable performance on the TMT, and no differences in verbal fluency when compared to normal weight individuals. Taken together, these findings suggest difficulty with updating and maintaining set, an important component of the WCST that is not examining in the TMT or verbal fluency tasks.

Sustained Attention

Attentional bias towards food cues has been consistently demonstrated in obese individuals (Nijs & Franken, 2012) and experimental studies provide relatively consistent evidence that an attention bias for food contributes to subsequent food intake (Werthmann, Jansen, & Roefs, 2015). Further, it has been demonstrated that obese individuals are more likely to have ADHD and difficulties with sustained attention (Nigg et al., 2016). A computerized version of the Continuous Performance Test (CPT) was used to examine the ability to sustain attention in two studies (Calvo et al., 2014; Solís-Ortiz et al., 2016). No significant differences in either reaction time or percentage of correct answers were found between obese and normal weight individuals in either study. A third study (Cournot et al., 2006) used the Sternberg Test, consisting of two subtests where individuals are instructed to quickly scan for a target letter, with increasing attentional loads. Obese individuals performed significantly worse than those in lower BMI quintiles on both subtests of the Sternberg test.

Working memory

Working memory is the ability to retain information over the short term and perform mental operations on these contents. Seven studies used WAIS-III digit span as an outcome measure within their study (Cserjési et al., 2009; Gonzales et al., 2010; Gunstad et al., 2007; Smith et al., 2010; Spitznagel et al., 2013; Volkow et al., 2008). Participants are asked to repeat verbally increasing number strings that they hear until they fail to complete two successive trials. For digits backwards, individuals repeat a reversed version of number strings that they hear until they fail two successive trials at a given number string length. One study found a correlation between increased BMI and poorer performance on the digit span task (Volkow et al., 2008). All other studies found no relationship between BMI and digit span (Gonzales et al., 2010) and performance on digit span tasks did not predict weight loss (Spitznagel et al., 2013) or change as a result of weight loss (Smith et al., 2010). A relationship between digit span and BMI was only found when combined with mood measures (Cserjési et al., 2009) or age (Gunstad et al., 2007). The Gonzales et al. (2010) study also used a verbal *N*-back test and found that accuracy and reaction time performance on the did not differ significantly between the obese, overweight, and normal weight groups. Ariza et al. (2012) utilized a Letter-Number Sequencing task and also failed to find a difference between obese and healthy control groups. Accordingly, there is no evidence for an independent link between obesity and working memory performance.

Planning

Planning is an important component of executive function, in that deficits could lead to an inability to have optimal strategy selection and use (e.g., making a healthy

choice when faced with a trigger food). Thus, deficits in these areas could help maintain unhealthy eating behavior in that the ability to develop, organize, and execute adaptive behaviors could be compromised. Planning has been examined in three studies utilizing the Rey-Osterrieth Complex Figure (RCFT) (Boeka & Lokken, 2008; Lokken et al., 2010; Roberts, Demetriou, Treasure, & Tchanturia, 2007). In the Roberts et al. (2007) study, authors reported that for the RCFT Copy trial, the overweight population was both less accurate and more piecemeal in their drawing than the healthy-weight group, producing a poorer-quality figure on completion. Additionally, the two other studies utilizing the RCFT both found significant differences between the obese and healthy weight individuals (Boeka & Lokken, 2008; Lokken et al., 2010), with obese individuals performing more poorly in the planning and design of their figure.

Memory

Episodic memory is the ability to store, maintain, and retrieve contextually rich representations of events from one's own life (Tulving & Donaldson, 1972). There is increasing evidence to suggest that this type of memory may play a major role in allowing us to regulate consumption. Manipulations of memory for recent meals have considerable impact on the long-term satiating effect of those meals (Brunstrom et al., 2012; Higgs & Donohoe, 2011; Higgs, Williamson, & Attwood, 2008; Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2011), while amnesic patients who are unable to remember recent consumption can sometimes eat several consecutive meals without reporting satiety or discomfort (Hebben, Corkin, Eichenbaum, & Shedlack, 1985; Higgs, Williamson, Rotshtein, & Humphreys, 2008; Rozin, Dow, Moscovitch, & Rajaram, 1998). Further, individuals who were overweight or obese have smaller hippocampal

volume at baseline and experience greater hippocampal atrophy than their normal-weight peers, suggesting structural differences in the neural memory center (Jacka, Cherbuin, Anstey, Sachdev, & Butterworth, 2015). Memory has been assessed in obese individuals using the Treasure-Hunt Task, which assesses memory for object information (“what”), location information (“where”), and temporal order information (“when”) within the same paradigm, as well as testing the ability to integrate these features into a single event recollection. Higher BMI was associated with significantly lower performance on the what–where–when (WWW) memory task and all individual elements: object identification, location memory, and temporal order memory (Cheke, Simons, & Clayton, 2015). In the study by Kuo et al. (2006), there was no statistical difference in memory performance between obese and normal-weight individuals. The authors assessed memory through a composite score created from three test scores, the Hopkins Verbal Learning Test, the Rey Auditory-Verbal Learning Test, and the Rivermead Behavioral Memory Test Paragraph Recall task. Three studies assessed visual memory using the recall administration of the RCF task (Boeka & Lokken, 2008; Gonzales et al., 2010; Roberts et al., 2007). One study found no differences between normal weight, overweight and obese individuals using this measure (Gonzales et al., 2010). While this study was adequately controlled, its sample size was small ($N = 32$), limiting power to detect potential between group differences. Two studies found that obese individuals had significantly poorer recall performance compared to normal weight individuals on the RCF recall tasks (Boeka & Lokken, 2008; Roberts et al., 2007), however, both groups of participants were found to have reduced performance on the copy portion of this task compared to the normative sample, which indicates limited encoding of the figure and

compromises its use as a measure of delayed visual memory. Overall given the conflicting findings, evidence for a relationship between visual memory performance in obese adults remains inconclusive. Furthermore, the available research provides no evidence of an independent relationship between visual memory and obesity.

Obesity Conclusion

The key question which prompted this review was whether obese individuals demonstrate impaired performance on neuropsychological tasks of cognitive and executive functioning when compared to healthy weight controls. Methodological differences and incomplete information in relation to task procedure make this a difficult question to answer. Overall, when considering specific domains, obese individuals seem to demonstrate an impaired performance consistently on tasks measuring set-shifting domain. All other domains of cognitive functioning report mixed findings across studies in relation to obesity. A possible explanation why we might see impairment in only certain areas might be to do with the sensitivity of the tasks used and that obese individuals show subtle impairments over a range of different aspects of cognitive functioning, but that studies which failed to detect this effect may have been underpowered and be using an insufficient sample size to detect what may be quite a small effect. Even studies which do suggest that obese individuals might demonstrate specific executive impairments need to be interpreted with caution. These studies have their limitations as they are based on a small number of subjects and additional factors such as education, gender and mood also impact task performance, influences which are not consistently controlled for. Across the board, there is a need for more study replication and consistency across study design to allow for more adequate comparison in

obese individuals and to gain a greater understanding of the underlying neuropsychological constructs that maintain obesity. Although there are many pathways to obesity, there is substantial evidence that disordered eating can be a significant factor in its development and maintenance (Marcus & Wildes, 2014). Individuals with BED are frequently obese and BED is often observed in obese populations, especially the severely obese (Hudson et al., 2006). However, it remains unclear whether BED represents an etiologically distinct behavioral phenotype of obesity or simply a nonspecific eating pattern, seen in some obese individuals with no particular etiologic significance (Kessler, Hudson, Herman, & Potenza, 2016). Further study of the cognitive constructs underlying BED is necessary in order to elucidate differences and overlaps between BED and obesity.

Neuropsychological Studies of Binge Eating Disorder

Binge Eating Disorder and Cognitive Functioning: An Overview

Binge eating disorder (BED), now a distinct diagnosis in the fifth edition of The Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) is defined as recurrent episodes of objective overeating with a loss of control occurring at least once a week over a period of three months. During these episodes, an individual may experience a lack of control and feelings of guilt and marked distress. BED is the most commonly occurring eating disorder in the United States and affects approximately 3% of adults (Hudson, Hiripi, Pope, & Kessler, 2007). Individuals with BED also have high rates of psychiatric comorbidity, reduced quality of life, and are twice as likely to become overweight or obese (Field et al., 2012; Johnson et al., 2001;

Wilfley et al., 2003). While some psychological treatments, including Cognitive-Behavioral Therapy and Interpersonal Psychotherapy have achieved modest success in achieving binge-abstinence in the short-term, interventions for BED have not proven especially successful at eliminating bingeing or achieving weight control in the long-term (Iacovino, Gredysa, Altman, & Wilfley, 2012). The relative lack of efficacy of available treatments could partially be attributable to a poor understanding of the neuropsychological underpinnings of loss of control eating. Identification of neuropsychological weaknesses in this population has the potential to better classify risk factors, suggest markers for severity and prognosis, and provide direction for developing more effective interventions.

While binge eating and obesity are often co-morbid, the majority of obese individuals do not have BED, suggesting that the development of binge eating is independent of that of obesity. Factors that are specifically associated with the development of binge eating are a history of caloric restriction, differential responses to past stressors, hunger, weight-teasing, and available highly palatable food (Mathes, Brownley, Mo, & Bulik, 2009; Neumark-Sztainer et al., 2007; Svaldi et al., 2014). Differential responses in individuals with BED have also been investigated through neuroimaging, with a recent review by Balodis, Grilo, and Potenza (2015) demonstrating decreased frontostriatal processing to the anticipation and receipt of rewards and losses and inhibitory control, along with increased reactivity to food cues and palatable taste cues in the orbitofrontal cortex, insula, and striatum—regions implicated in reward and motivation processing. In humans, differences in response to stressors, hunger, highly palatable food, and other factors in the environment could be explained by existing

differences in executive function. For example, an inability to balance a desire for immediate comfort with future consequences, and deliberately plan, choose, and execute an adaptive strategy in the context of binge eating cues (e.g. negative emotion, food cues, interpersonal conflict) potentially represent weaknesses in EF, which may contribute to the development and maintenance of binge eating. Binge eating clearly results in negative consequences, including psychological distress, increased negative affect, and weight gain, but is maintained regardless (Hilbert & Tuschen-Caffier, 2007).

Several related processes that comprise cognitive functioning could potentially influence binge eating behavior. Preliminary evidence suggests that disordered eating behavior (such as binge eating), and excess weight are associated with relative deficits in several executive function dimensions (Fagundo et al., 2012; Gunstad et al., 2007; Tchanturia et al., 2004). Cognitive inflexibility (a weakness in shifting mental sets) can lead to an over-focus on eating as a coping strategy in the presence of an uncomfortable internal or external binge cue (e.g., negative mood, food cues). Inhibitory control problems may contribute to the start of a binge in response to a trigger, and the marked drive to continue eating (loss of control) until uncomfortably full. Binge episodes often end in physical discomfort and emotional distress, suggesting a prioritization of immediate (i.e., short-term comfort or numbing out of emotions) versus delayed reward in decision-making. Relatedly, difficulties with planning could explain an inability to develop and engage in adaptive behaviors (e.g., a regular eating schedule) that could prevent many instances of binge eating. However, many executive functioning constructs are multi-dimensional in nature and overlap in their hypothesized influence on eating behavior. Cognitive flexibility, working memory, and inhibitory control are critical

components of successful planning and decision-making. In order to parse out the association of binge eating with specific dimensions of executive function (to the extent that this is possible), use of a comprehensive and validated battery with several tasks measuring well-defined constructs is necessary.

Despite having implications for treatment development and prognosis, few studies have investigated these neuropsychological variables in a BED sample, as most research thus far has focused on anorexia nervosa (AN) and bulimia nervosa (BN). With the recent addition of BED to the DSM-5, more research is necessary to begin to understand the executive functioning underpinnings of this disorder. Seventeen studies investigating the neuropsychological profile of BED exist at this time. These studies have displayed some preliminary evidence for relative neuropsychological weaknesses in adults with BED; however, due to differing methodology (e.g. use of just one measure of executive functioning, unreliable screening of binge eating), results have been mixed. The following review, organized by constructs details findings from studies that have examined neuropsychological underpinnings of BED.

Intellectual functioning

There have been no studies on BED versus non-BED individuals examining intellectual functioning. This is an important construct that has been investigated in obesity and warrants investigation in BED.

Psychomotor performance

Similarly to intellectual functioning, there have been no studies on BED versus non-BED individuals examining psychomotor performance. Given the aforementioned importance

of psychomotor performance in capturing subtle disturbances in the brain network, this construct should be examining in individuals with BED (Reijmer et al., 2013).

Inhibitory control

Both internal (emotional, cognitive) and external (interpersonal, environmental) cues (Vanderlinden, Dalle Grave, Vandereycken, & Noorduin, 2001) trigger urges to binge eat. Fischer, Smith, and Anderson (2003) suggested that high levels of ‘urgency’ impulsivity (lack of inhibitory control and the tendency to act rashly in the context of negative affect) is associated with binge eating. Thus, inhibitory control deficits may serve as a risk factor for BED. Additionally, poor inhibitory control could contribute to the experience of loss of control, in that individuals with BED have extreme difficulty with discontinuing a binge episode once initiated, even when they wish not to be eating. While obese individuals without BED show inhibitory control deficits compared to normal weight individuals, it is possible that these deficits exist on a continuum, with overweight individuals with BED having a more severe inhibitory control problem than overweight individuals without BED. Mood disorders and impulsive behaviors, such as compulsive gambling, are more common in individuals who engage in binge eating than those who do not (Wiederman & Pryor, 1996), which could point to a more severe behavioral impulse control problem in BED.

Preliminary evidence from fMRI studies support this notion in that individuals with BED show differential patterns of brain activation in response to food images in the ventromedial prefrontal cortex, inferior frontal gyrus, and insula (Filbey, Myers, & DeWitt, 2012), all of which have been implicated in self-regulation and impulse control. Seven studies (Duchesne, Mattos, et al., 2010; Manasse et al., 2014; Mobbs et al., 2011;

Mole et al., 2015; Svaldi et al., 2014; Voon et al., 2015; Wu et al., 2013) thus far have examined response inhibition or inhibitory control in individuals with BED. Four studies have utilized computerized tasks of inhibitory control, i.e., a Go/No-Go Task and a Stop-signal Task, only one of which reported significantly poorer inhibitory control in BED groups as compared to controls. In Mobbs et al. (2011), obese individuals with BED displayed slower inhibitory response and made more errors when responding to food-related stimuli compared to obese individuals without BED. Three studies observed no differences in obese subjects with or without BED in a computerized premature responding task (Mole et al., 2015; Voon et al., 2015; Wu et al., 2013). Two studies (Duchesne, Mattos, et al., 2010; Manasse et al., 2015) reported significant differences between obese BED and non-BED individuals, as in time to complete the Stroop task. In another study (Manasse et al., 2014), overweight individuals with loss of control eating committed more errors on the Stroop task compared to overweight individuals without loss of control eating. Thus far, research supporting inhibitory control deficits in BED is mixed; it appears that food-specific inhibitory control may be particularly relevant to binge eating. Further investigation of this construct, particularly the use of multiple instruments to tap into multiple facets of impulsivity and using food and on-food stimuli, is warranted.

Cognitive Flexibility

Cognitive inflexibility may lead to habitual behaviors (e.g. binge eating), even when detrimental to a goal (weight control, abstinence from binge eating). For example, individuals with BED may perseverate on the behavior of binge eating as a way to regulate emotion (i.e. to reduce or avoid negative affect) and thus have problems shifting

cognitive set to generate and choose a more adaptive strategy than binge eating. A study examining only individuals with BED indicated that individuals with poorer set-shifting abilities and more changes in negative mood experienced more feelings of loss of control over eating than individuals with BED whose set-shifting abilities were better and whose mood did not change (Dingemans, Visser, Paul, & van Furth, 2015). Problems with set-shifting during a binge episode may also explain why binge eaters are unable to stop eating even once uncomfortably full. Thus, set-shifting deficits may be a key neuropsychological characteristic in the development and maintenance of loss of control eating. Thus far, evidence for cognitive inflexibility in individuals with BED is mixed. Two studies (Duchesne, Mattos, et al., 2010; Svaldi, Brand, & Tuschen-Caffier, 2010) reported that overweight individuals with BED performed worse on set-shifting tasks compared to overweight individuals without BED, as measured by the Trail Making B and Wisconsin Card Sort (WCST), respectively. Another study by Aloï et al. (2015) found that overweight individual with BED performed more poorly than normal weight HCs and individuals with AN when controlling for BMI on Trail Making B and the WCST. Conversely, three studies (Galioto et al., 2012; Kelly, Bulik, & Mazzeo, 2013; Manasse et al., 2014) reported no differences in set-shifting (measured by Trail Making B, Verbal Fluency, WCST, and the Penn Conditional Exclusion Task, a computerized set-shifting task) between a binge eating and non-binge eating sample. The Kelly et al. (2013) study is one of the few studies to utilize a normal weight BED sample, but also utilized a self-report measure and included sub-threshold binge-eaters in the sample, which may have precluded finding differences between the two groups. Of note, perseverative errors on the WCST were significantly associated with number of binge

episodes in this study (Kelly et al., 2013). The Manasse et al. (2014) study that reported no significant differences in cognitive flexibility between binge eating and non-binge-eating groups also used a sub-threshold group, perhaps clouding any differences that may exist. These mixed results may be partly explained by the differences in characteristics of participants in the five studies such as age and BMI and by the different tests that were used to assess set-shifting. Thus, overall, there is mixed evidence to suggest cognitive flexibility may be relatively impaired in individuals with BED and is an area worth further investigation.

Sustained Attention

The high comorbidity of BED with attention-deficit disorder (Cortese, Dalla Bernardina, & Mouren, 2007), which is associated with deficits in attention and inhibitory control (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005), suggests that individuals with BED could have a dysregulated attentional system. Deficits in attention might cause difficulties in adhering to a regular eating pattern, thus favoring abnormal eating behaviors. Further, individuals with attentional difficulties may be relatively inattentive to internal signs of hunger and satiety (Fleming & Levy, 2002). The study by Kelly et al. (2013) examined sustained attention in normal weight individuals with subthreshold BED, as compared to normal weight controls and found no significant differences in attentional performance, as measured by the Conner's Continuous Performance Task.

Working Memory

Working memory refers to an individual's ability to keep goal-relevant information in mind in the face of distractors (e.g., the environment, emotions). Working

memory capacity is strongly associated with self-regulation (Hofmann, Gschwendner, Friese, Wiers, & Schmitt, 2008). The ability to shield goal-relevant information from distraction is particularly important in those who experience binge eating, given the strength of binge eating cues. Weak working memory capacity would predispose an individual to let self-regulative goals (e.g., to not binge eat) be overcome by cues and the desire to start and continue eating. Four studies to date have examined working memory in a BED sample, two of which showing that individuals with BED displayed poorer working memory capacity than overweight individuals without BED utilizing the *N*-back test and Digit Span Backwards (Duchesne, Mattos, et al., 2010; Manasse et al., 2014). Two study found no group differences in task performance (Galioto et al., 2012; Müller et al., 2014).

Planning

Planning is an important aspect of executive function and deficits in this area could help maintain binge eating. For example, a regular pattern of eating (e.g., eating every 3-4 hours with regular meals and snacks) is a known protective factor against binge eating; however, an inability to plan out meals ahead of time (e.g., get what is needed at the grocery store, make meals to-go for work) could lead to dysregulated patterns that can put an individual at risk for binge eating. However, planning ability is difficult to measure discretely, given its multidimensionality. Planning in a BED sample has been examined in two studies, both of which reported relative deficits in planning in the BED group compared to controls. Duchesne, Mattos, et al. (2010) used three subtests from the Behavioral Assessment of Dysexecutive Syndrome (BADS) which predominantly examine planning and problem-solving. The Zoo Map task requires individuals to plan a

journey around a zoo following specific instructions and rules. On this task Duchesne, Mattos, et al. (2010) found that obese individuals with BED made more errors than obese controls but did not differ on planning or completion time or in a second condition when provided with increased task structuring. On the modified six elements task where participants are required to complete some of six different tasks following specific rules, the authors found that obese participants with BED performed worse than obese controls. This finding was repeated again on the Action Program Task, where individuals have to problem-solve a practical task with minimal guidance, with obese controls able to complete more of the stages of the task unaided than those with BED. In a study comparing overweight individuals with and without loss-of-control eating, Manasse et al. (2015) found significant differences on the Delis Kaplan Executive Functioning System (DKEFS) Tower Task to measure planning between obese women with and without BED. Organization, similar to planning, has been assessed by the Rey Complex Figure Test, (RCFT) which assesses visual organization, short-term visual memory and visuospatial abilities. The subject must copy and recall, after an interval of 3 minutes, a complex geometric figure. The accuracy of the reproduction of all the details of the figure was a measure of visuospatial and visual memory abilities. In the study by Aloï et al. (2015) individuals with BED had the lowest score on Rey-Accuracy Index compared to AN and HC in the copy condition of the RCFT, suggesting difficulties with visuospatial planning.

Memory

Memory refers to a range of processes that enable information from the past to be used to guide present and future behavior. With respect to long-term memory or the

continuing storage of information, little research has been conducted with individuals with BED, but research on BN suggests that there are deficits in long-term memory and that this may maintain the disorder. Further, memory for what has been eaten recently has an influence on appetite, where individuals with amnesia demonstrate binge-like behavior (Rozin et al., 1998). In individuals with BED, verbal memory was measured in three studies, where participants were read a list of 12 words, a total of 4 times and asked to recall as many words as possible after each trial. Obese individuals with BED did not differ from obese individuals in their initial recall or their recall after a 20 minute delay in all of the studies (Galioto et al., 2012; Lavender et al., 2014; Müller et al., 2014).

Disorder-related memory was assessed and overweight individuals with BED were found to remember fewer positive body-related words compared to overweight individuals, with no differences for negative body-related words and control stimuli (Svaldi et al., 2014), suggesting a bias for positive body-related associations in BED. In the Aloï et al. (2015), which utilized the RCFT, overweight individuals with BED had a significantly poorer performance on RCFT percentage of recall than HC. The differences between groups remained significant after controlling for BMI and diagnosis, indicating potential memory differences between the groups.

BED Conclusion

Overall, individuals with BED display deficits in executive function compared to individuals without BED, specifically in the areas of planning, delayed discounting, and inhibition/self-regulatory control. One important gap in research of the current studies is that none of the studies used a normal-weight, full threshold BED sample. Future studies should seek to include a normal-weight BED group to examine the interplay of weight

status and binge eating in order to gain a more complete picture of the neurocognitive profile of binge eating pathology. Additional demographic factors, such as education might also play a mediating role. In these domains and also across the board, there is a need for more study replication and consistency across study design to allow for more adequate comparison.

Similarly observed executive functioning deficits are found in both obese individuals and individuals with BED. These differences may help to explain the development and maintenance of unhealthy eating. These findings suggest that executive planning and decision making, specifically with regards to normalization of eating, should be emphasized in treatment. In CBT for BED, an emphasis is placed on regular eating patterns and planning meals and snacks, however, increased emphasis on how to plan and attending to stimuli, including provision of repeated planning exercises, may be necessary. Further, in obese individuals, overvaluation of short-term versus long-term reward may additionally have implications for treatment development. Acceptance-based treatments, such as Acceptance and Commitment Therapy, emphasize using movement towards long-term values (e.g., a healthy lifestyle, serving as a role model for children), rather than short-term avoidance of everyday distress, as the primary motivator for choosing behavior. Inclusion of a values-based intervention component in standard treatment may aid individuals to tolerate the discomfort of giving up short-term comfort in the service of greater goals or life values. A greater understanding of the neuropsychological constructs underlying obesity and BED can provide further treatment guidelines for individuals who are struggling. Further, understanding the effects of

binging and obesity on cognitive modalities may allow providers to fill the gaps and allow for the end to the “vicious cycle.”

Conclusion

Given that BED in the context of obesity confers greater problems than obesity alone, it is important to disentangle the effects of weight status on BED. With the recent addition of BED to the DSM-5, more research is necessary to begin to understand if the neurocognitive profile of BED differs given weight status. Preliminary evidence suggests that executive functions, which are comprised of diverse, overlapping frontal lobe processes that enable an individual to engage in self-initiated, healthy, and adaptive behavior may be weak in individuals with BED. In the existing literature, there is evidence that overweight individuals with BED exhibit greater cognitive inflexibility, poorer inhibitory control, and poorer planning than non-BED overweight control groups. This is also seen in overweight individuals without BED, who show greater cognitive inflexibility, greater disinhibition, poorer planning than normal-weight individuals without BED. These similarities suggest similar cognitive profiles, despite distinctions in the characteristics of individuals with BED versus those who are obese.

Identification of the neurocognitive profile of individuals with BED has the potential to better classify risk factors and suggest markers for severity and prognosis. While some psychological treatments, including Cognitive-Behavioral Therapy and Interpersonal Psychotherapy have achieved modest success in achieving binge-abstinence in the short-term, interventions for BED have not proven especially successful at weight loss long-term and only modestly successful at long term binge abstinence (Iacovino, Gredysa, Altman, & Wilfley, 2012). Adding neurocognitive targets to intervention has

the potential to improve treatment for BED, by adapting interventions to anticipate potential treatment interfering behaviors. In order to gain understanding of necessary targets of interventions in these populations, it is important to see which constructs are more significantly associated with BED over and above the effects of obesity.

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