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ASSOCIATIONS BETWEEN BINGE EATING AND EXECUTIVE FUNCTIONING AMONG
YOUNG WOMEN

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University

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Abstract

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Binge eating is a pervasive disordered eating behavior associated with numerous psychological and physical comorbidities. Preliminary research indicates that emotion regulation difficulties, behavioral impulsivity, and executive dysfunction may contribute to the onset and/or maintenance of these behaviors. However, few studies have utilized neuropsychological measures to examine this link, and the assessment of behavioral and cognitive emotion regulation strategies are limited in scope. The purpose of the current study was to gain a deeper understanding of the emotional, behavioral and cognitive processes associated with binge eating behavior. Greater clarity regarding how these factors relate to binge eating is critical to the development of effective treatment and prevention efforts. To address these aims, the current study examined the executive functioning, depression, behavioral impulsivity, distress tolerance,

and emotion regulation strategies of 50 women engaging in weekly binge eating in the absence of compensatory behaviors; their outcomes were compared to 66 women with no history of binge eating. Hierarchical regression analyses revealed that groups did not significantly differ in executive functioning after controlling for depression, state anxiety, body mass, psychopharmaceutical use, and general intelligence; nonetheless, correlation analyses suggest that, among the binge eating group, individuals endorsing more frequent binge eating might have greater difficulties thinking flexibly or shifting attention. Secondary analyses indicated that individuals who binge eat are more depressed, are more likely to engage in impulsive behavior (but only when distressed), have more difficulties tolerating distress, are more likely to engage in rumination, self-blame, and catastrophizing, and less likely to focus on the positive. Although the current study is unable to determine whether these cognitive, emotional, and behavioral factors precede or follow binge eating episodes, outcomes have clinical implications. Specifically, programs focused on the prevention and treatment of binge eating should help individuals learn to better identify and tolerate difficult affective states and to utilize more adaptive means of coping. Outcomes also provide important directions for future research, including longitudinal designs to better understand the temporal associations of the current study's variables, as well as suggestions to broaden and standardize neuropsychological assessment and scoring procedures to facilitate comparisons across studies.

Associations between Binge Eating and Executive Functioning among Young Women

In the last decade, research examining the neuropsychology of eating disorders (EDs) has increased exponentially. A deeper understanding of the neuropsychological functioning of those with EDs is important as it provides direction for the psychological and pharmacological treatment of these typically chronic disorders (see Williamson, Martin, & Stewart, 2004 for a review). In addition, the identification of neuropsychological impairments associated with EDs has the potential to provide an objective means of evaluating symptom severity and prognosis (Duchesne et al., 2004). Despite the clear advantages of neuropsychological research among EDs, most of this work has focused on individuals with anorexia nervosa (AN) and bulimia nervosa (BN; Duchesne et al.). Very little research has investigated neuropsychological functioning among individuals who engage in binge eating in the absence of regular compensatory behaviors.

What is Binge Eating?

According to the DSM-IV, a binge episode involves the consumption of an amount of food that is larger than most people would eat during a similar period of time and under similar circumstances, followed by a sense of loss of control (American Psychiatric Association, 2000). Binge eating is the hallmark symptom of binge eating disorder (BED). However, the diagnostic criteria for BED, particularly the size and frequency of the binge episodes, are hotly debated (American Psychiatric Association; Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2007; Mathes, Brownley, Mo, & Bulik, 2009). Nonetheless, there is consensus that binge eating is associated with numerous psychosocial and physical comorbidities (Hudson, Hiripi, Pope, & Kessler, 2007; W. G. Johnson, Rohan, & Kirk, 2002; Ross & Ivis, 1999; Saules et al., 2009;

Spoor et al., 2006; Striegel-Moore et al., 2001) and thus represents a significant public health concern (Mathes et al., 2009).

Prevalence of Binge Eating

Binge eating behaviors are both highly prevalent and are found among a wide range of individuals. In a population-based sample of 10 to 20 year old individuals, 46% of females and 30% of males reported engaging in binge eating behaviors at some point during their lifetime (Ross & Ivis, 1999). A similar prevalence estimate (37.2%) was obtained in a web-based survey of college students (Saules et al., 2009). Further, unlike other disordered eating patterns, which have disproportionately affected White/Caucasian women (Striegel-Moore et al., 2003), binge eating symptoms and their psychosocial correlates appear to impact individuals of an array of racial and ethnic groups at similar rates (J. G. Johnson, Cohen, Kasen, & Brook, 2002). For example, J. G. Johnson and colleagues found that 26% and 16.7% of Black/African American adolescent males and females engaged in binge eating, compared to 18.5% and 17.8% of White/Caucasian males and females, respectively. Despite the pervasiveness of binge eating behaviors, particularly in diverse community samples, research has only recently started to investigate their psychosocial and neuropsychological comorbidities.

Psychosocial Comorbidity

Affective functioning. Young adults who engage in binge eating behaviors are more likely to endorse the presence of comorbid psychiatric symptoms. For example, young adults who engage in binge eating behaviors are more likely than their non-bingeing peers to report depression, low self-esteem, and body dissatisfaction (Ross & Ivis, 1999). The relation between depression or general negative affect and binge eating has been duplicated in other studies (e.g., Barker, Williams, & Galambos, 2006; W. G. Johnson et al., 2002). Spoor and colleagues (2006)

noted that depressive symptoms preceded binge eating behaviors among a group of young women. Moreover, compared to a group of young women without disordered eating symptoms, those who engaged in binge eating participated in more mental health treatment and demonstrated greater social impairment and emotional distress from the ages of 12 to 20. Additionally, the degree of impairment did not differ significantly between women who endorsed subthreshold levels of binge eating and women who endorsed full BED criteria. In addition to the psychosocial comorbidities, binge eating in girls has been implicated in the onset of diagnosable EDs (Stice, Marti, Shaw, & Jaconis, 2009; Striegel-Moore, 1995).

Further, although most adults who binge eat are obese, (Hudson et al., 2007; Striegel-Moore et al., 2001), the negative psychological impact of binge eating does not appear to be attributable to obesity (Ivezaj et al., 2010). Obese individuals who binge eat report substantially poorer psychological functioning than do obese individuals who do not binge eat (Bulik, Sullivan, & Kendler, 2002; Grucza, Przybeck, & Cloninger, 2007). Similarly, a study of a community-based sample of women (French, Jeffery, Sherwood, & Neumark-Sztainer, 1999) found no significant interactions between binge eating and overweight status on measures of depression, self-esteem, or binge episodes. Thus, the link between binge eating behaviors and poor mental health outcomes does not appear to be attributable to weight status.

Emotion regulation. Given the relation between negative affect and binge eating, some researchers have proposed that binge eating serves as one type of emotion regulation strategy. Kenardy, Arnow, and Agras (1996) posit that binge eating represents an emotional “trade off” (p.842). More specifically, feelings of guilt, which are common after a binge episode, are reportedly easier to tolerate than anger, anxiety and depression, the affective symptoms typically antecedent to the binge. Depression (Spoor et al., 2006; Stice, Presnell, & Spangler, 2002) and

stress (Corwin & Buda-Levin, 2004; Hagan et al., 2002; Mathes et al., 2009) also predict the onset of binge eating in both humans and animals, suggesting a biological basis to the use of binge eating as a means of affect regulation. Indeed, footshocking, tail-pinching, and food deprivation, techniques used to induce stress, lead to binge eating among mice (Avena & Bocarsly, 2011; Boggiano et al., 2005; Corwin & Buda-Levin, 2004; Hagan et al., 2002). In turn, the consumption of highly palatable foods (those typically consumed during binge episodes) is associated with neurochemical changes that lead to psychological relief (Avena, 2007; Kelley, Baldo, Pratt, & Will, 2005; Mathes et al., 2009), whereas food restriction following bingeing induces chemical responses that mimic anxiety and depression among mice (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008; Galic & Persinger, 2002).

Additional support for the link between emotion dysregulation and binge eating is provided by research with young adults (see Wolfe, Baker, Smith, & Kelly-Weeder, 2009 for a review). College women who reported engaging in binge eating behavior were more likely to do so on days they reported experiencing higher stress (Freeman & Gil, 2004). Similarly, Ross and Ivis (1999) found that marijuana users who engaged in binge eating were more likely than non-binge eating users of marijuana to report using this drug to relax or manage negative emotions. In another undergraduate sample, women with subthreshold BED were more likely to report using alcohol as a coping mechanism (Luce, Engler, & Crowther, 2007). Similarly, studies conducted with undergraduates (Anestis, Selby, Fink, & Joiner, 2007) found that distress tolerance and emotion regulation difficulties (U. Whiteside et al.) were inversely associated with binge eating behaviors even after scores on measures of other related constructs (e.g., anxiety, depression, weight and shape concern, and body dissatisfaction) were controlled. Thus, binge eating may represent maladaptive attempts to self-medicate distress (Peveler & Fairburn, 1990).

Heatherton and Baumeister (1991) propose that binge eating represents one type of emotion regulation technique used to divert attention from negative affect to a more concrete object, in this case food.

Despite evidence for a link between binge eating and emotion dysregulation, the nature of this relation is not well understood. Although several studies (Aldao, Nolen-Hoeksema, & Schweizer, 2010) have examined emotional coping strategies in AN and BN, less have evaluated these variables among individuals who binge eat in the absence of regular compensatory behaviors. One longitudinal study found that, among adolescent girls, rumination predicted the onset of binge eating (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). A second study found a link between binge eating and avoidance coping among college women (Engler, Crowther, Dalton, & Sanftner, 2006). Finally, a third study found that emotion-focused coping mediated the link between stress and binge eating (Sulkowski, Dempsey, & Dempsey, 2011). No additional forms of coping were evaluated in any of these studies. Further, Engler et al. (2006) assessed binge eating with a single item that did not include loss of control, a major component of this pathological eating behavior (Goossens, Braet, & Decaluwe, 2007; Keel, Mayer, & Harnden-Fischer, 2001; Latner & Clyne, 2008; Latner et al., 2007; Wolfe et al., 2009). In sum, these studies paint a limited picture of the association between binge eating and specific emotional coping or regulation strategies.

Impulsivity. Findings from studies using both animals and humans have also identified a link between binge eating and emotional reactivity or impulsivity (e.g., Anestis et al., 2007; Teegarden & Bale, 2008). Specifically, Anestis and colleagues found that individuals are more likely to engage in binge eating behaviors when they exhibit higher levels of emotional impulsivity. Similarly, mice with a genetic predisposition to stress reactivity were more likely to

binge eat highly palatable foods when exposed to various environmental stressors than wild mice (Teegarden & Bale, 2008). A relation between impulsivity and binge eating was also found in undergraduate women (Fischer, Anderson, & Smith, 2004). Specifically, individuals who reported greater tendencies to act impulsively in the face of distress were also more likely to believe that eating would alleviate their negative affect and to binge eat.

In their review of 222 studies, Fischer, Smith and Cyders (2008) also found that negative urgency, compared to other domains of impulsivity (i.e., lack of planning, lack of perseverance, and sensation seeking), demonstrated the strongest relationship with binge eating. These authors recommended the use of a multidimensional assessment of impulsivity. The UPPS Impulsive Behavior Scale captures the varying domains of impulsivity more comprehensively than other measures (which tend to treat impulsivity as a unidimensional construct; S. P. Whiteside & Lynam, 2001). The accurate assessment of impulsivity is important as it this personality characteristic is one of the most powerful predictors of treatment drop out among individuals with EDs (Fassino, Piero, Tomba, & Abbate-Daga, 2009), and thus represents a particularly concerning correlate of binge eating behavior.

In sum, binge eating behavior might reflect difficulties in affect regulation, as well as elevated levels of emotional impulsivity. The consumption of highly palatable foods in particular might be indicative of the use of food as a means of alleviating psychological pain. Food as a means of emotion regulation appears to have biological underpinnings, as binge eating is associated with stress in both humans and animals. Still, little is known about the specific emotion regulation and distress tolerance processes associated with binge eating. Recently, impairments in executive functioning, particularly poor planning abilities and cognitive

impulsivity, have also been linked to binge eating behaviors, which may further compound some individuals' difficulties dealing with negative affect.

What is Executive Functioning?

According to Lezak, Howieson and Loring (2004), executive functioning “consists of those capacities that enable a person to engage successfully in independent, purposive, self-serving behavior” (p.35). The neurocognitive processes that comprise executive dysfunction include decrements in planning, difficulties demonstrating inhibition, heightened impulsivity, cognitive rigidity, and problems with shifting attention (Lezak et al.), among others. Thus, impairments in executive functioning impede one's ability to plan effectively, to adjust to changes in our environment, and to carry out goal-directed behaviors.

Williams, Suchy, and Rau (2009) further note that executive functioning is an integral component of stress management. Evidence for their theory can be found in research exploring the neuropsychological and cognitive functioning of individuals with traumatic brain injuries (TBIs). Compared to their pre-injury functioning, individuals with frontal lobe TBIs (the area of the brain in which many executive functions are housed) demonstrated significant difficulties coping with daily stressors (Marschark, Richtsmeier, Richardson, Crovitz, & Henry, 2000; Williams et al., 2009). Similarly, compared to a group of healthy controls, individuals with frontal lobe TBIs were less able to identify basic emotional states (Henry, Phillips, Crawford, Ietswaart, & Summers, 2006; Hoaken, Allaby, & Earle, 2007). Thus, a relation appears to exist between impaired executive functioning and emotion management, including affect identification and regulation.

Williams and colleagues (2009) further explain that the relation between these two variables is mutually reinforcing. When faced with stressors, individuals with limited executive

functioning are more prone to experience maladaptive stress responses, which lead to additional impairments in executive functioning. Specifically, when exposed to stressful stimuli, individuals with impaired executive functioning release more cortisol than those who respond to stress in other, more healthy ways. Cortisol is a hormone which, when released, is associated with impairments in several domains of cognitive functioning, including executive functioning. This is particularly true among individuals with depression, a common correlate of binge eating (Hinkelmann et al., 2009; W. G. Johnson et al., 2002; Ross & Ivis, 1999). Over time, individuals with decrements in their executive functioning skills become more susceptible to increased stress exposure (e.g., interpersonal conflict, academic and work difficulties). In sum, individuals with impaired executive functioning “may be at risk for a trajectory of compounding and escalating stress regulation difficulty and associated adverse mental health outcomes” (Williams et al.; p. 134).

Given the noted relation between emotion management and executive functioning, binge eating behaviors might also be associated with executive dysfunction. Furthermore, the negative emotions that encircle binge eating episodes (Kenardy et al., 1996) might lead to additional decrements in executive functioning, thereby perpetuating a cycle of poor emotion management and maladaptive eating patterns. Still, few published studies have examined the executive functioning of individuals with binge eating behaviors. Given the dearth of such research, the current study’s aims and hypotheses were also informed by the outcomes of studies investigating emotional and behavioral factors associated with binge eating behavior, as well as the executive functioning of participants with BN and subthreshold BN symptoms. Taken together, studies suggest that binge eating is associated with impairments in several facets of executive functioning, particularly cognitive impulsivity and planning.

Executive functioning and binge eating. To examine this concept further, Svaldi, Brand and Tuschen-Caffier (2009) assessed the executive functioning of individuals who engage in binge eating behaviors. In their study, 17 women with BED were compared to 18 “healthy” controls on two measures of executive functioning. As hypothesized, women with BED demonstrated less cognitive flexibility and greater impairments in decision-making skills compared to those in the control group. The authors concluded that, when continuously presented with an array of highly palatable, calorically dense, food options, it takes a good deal of conscious decision making, behavior inhibition, and delay of gratification (i.e., domains of executive functioning) to engage in healthy food choices. Subsequently, individuals with demonstrated decrements in their executive functioning may find it more difficult than their peers to prevent the onset of binge eating episodes.

A similar link between binge eating and executive dysfunction was identified in a study conducted by Duchesne and colleagues (2010). In this investigation, 30 obese participants with BED and 38 obese participants without BED were matched with respect to their estimated intelligence, age, BMI and education. Although individuals were excluded if they met criteria for major depressive disorder, other comorbid psychiatric conditions were relatively common (e.g., 12% met criteria for a specific phobia, 10% for generalized anxiety). Participants completed a number of neuropsychological tests representing a range of executive functions, including the Wisconsin Card Sorting Task (WCST; Heaton, Chelune, Talley, Kay, & Curtis, 1993). Results from this study indicated that, relative to the non-binge eating group, participants with BED had significantly more perseverative errors and greater difficulties maintaining set, suggesting greater cognitive rigidity and difficulties shifting attention. Planning and problem-solving abilities were also lower for participants engaging in binge eating (Duchesne et al.).

In contrast, outcomes from two recent studies did not support the executive dysfunction-binge eating link (Davis, Patte, Curtis, & Reid, 2010; Galioto et al., 2011). Davis et al. used several neuropsychological measures to examine the decision-making abilities of three groups of women: 1) obese with BED, 2) obese without BED, and 3) normal weight. Results indicated that obese women with and without BED had poorer decision-making skills than the normal weight group, but were not significantly different from one another. However, these statistical differences disappeared when the authors controlled for education level; normal weight individuals had significantly more education. The authors attributed this confound to the inverse association between obesity and socioeconomic status (SES; McLaren, 2007), although SES was not evaluated. Similarly, Galioto et al. examined the memory, attention, language and executive functions of morbidly obese individuals with and without BED ($N = 131$). Analyses revealed no significant differences in any of these outcomes. Interestingly, clinical levels of cognitive impairment (i.e., scores 1.5 standard deviations [SD] below normative values) were evident in both groups.

Although neuropsychological studies present mixed outcomes, investigations examining structural and functional brain abnormalities among individuals engaging in binge eating are more consistent. Schafer et al (2010) found greater grey matter volume in the anterior cingulate cortex and medial orbitofrontal cortex of individuals with BED relative to a normal sample. The orbitofrontal cortex has been implicated in a number of emotional, cognitive, and behavioral processes commonly engaged before, during, and after eating, including decision-making, inhibition, hunger, satiety, and pleasure (Malloy, Bihrlé, Duffy, & Cimino, 1993; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). Although it is unclear whether the presence of localized abnormalities in the brain represent risk factors for or artifacts of binge eating, Schafer et al.

propose that they may be linked to processes identified via functional neuroimaging. Specifically, the orbitofrontal cortex is more reactive when exposed to food, which has been attributed to alterations in reward processing associated with the anticipation of eating something pleasurable (Karhunen et al., 2000; Kringelbach, O'Doherty, Rolls, & Andrews, 2003; Kringelbach & Rolls, 2004; O'Doherty, Deichmann, Critchley, & Dolan, 2002; Schienle, Schäfer, Hermann, & Vaitl, 2009; Small et al., 2001).

Similar outcomes have been ascertained via the Frontal Systems Behavior Scale (FrSBe; Stout, Ready, Grace, Malloy, & Paulsen, 2003), a self-report scale that measures apathy, disinhibition, and executive dysfunction (Boeka & Lokken, 2011; Spinella & Lyke, 2004). Spinell and Lyke found an association between disinhibition or overeating and all three subscales of the FrSBe in a community sample (although correlations were modest; $r = 0.23-0.33$). Similarly, researchers have found that some individuals with frontotemporal dementia exhibit significant disinhibition despite reporting satiety, as well as a heightened preference for sweet foods (Bathgate, Snowden, Varma, Blackshaw, & Neary, 2001; Woolley et al., 2007). Relative to non-overeating individuals with dementia and healthy controls, individuals in Woolley et al.'s study also showed significantly more atrophy in the right ventral insula and right rostral orbitofrontal cortex, regions of the brain associated with responding to food cravings and emotional awareness; the more disinhibition, the greater the atrophy.

Executive functioning and weight status. There is also recent evidence that executive functioning may be impaired in overweight and obese individuals (Cserjési, Luminet, Poncelet, & Lénárd, 2009; Galioto et al., 2011; Gunstad et al., 2007). For example, in a large ($N = 408$) cross-sectional study, BMI was positively correlated with executive dysfunction among adults 21 to 82 years of age (Gunstad et al.). Moreover, when compared to normal weight adults,

overweight and obese participants scored lower on three different domains of executive functioning, including cognitive flexibility and task switching. All analyses controlled for variables previously associated with executive functioning, including depression and estimated intelligence. In a second study, obese women performed significantly worse on three measures of executive functioning (i.e., cognitive flexibility, verbal inhibition, and task-shifting) compared to a group of non-obese women, although half of these significant associations were mediated by depression (Cserjési et al.). The relation between weight status and executive functioning is relevant to the current study's aims as there is a linear association between binge eating and BMI (Yanovski, 2003). Thus, the current study controlled for BMI when exploring differences in executive functioning between those who binge eat and those who do not.

Executive functioning and bulimia nervosa. In a review of the neuropsychology of EDs, Duchesne and colleagues (2004) assert that impairments in executive functioning are one of the most notable neuropsychological findings among individuals with BN. For example, individuals with BN performed significantly worse than a control group on the Computerized Performance Task (Laessle, Bossert, Hank, Hahlweg, & Pirke, 1990; Laessle, Fischer, Fichter, Pirke, & Krieg, 1992; Laessle, Krieg, Fichter, & Pirke, 1989), suggesting impairments in cognitive flexibility. When asked to complete tasks requiring impulse inhibition, participants with BN performed worse than both control groups and those with AN (Jones, Duncan, Brouwers, & Mirsky, 1991; Kaye, Bastiani, & Moss, 1995; Lauer, Gorzewski, Gerlinghoff, Backmund, & Zihl, 1999). Another study found that performance on the WCST (Heaton et al., 1993) was significantly poorer among 15 women with BN compared to 15 healthy controls (Alvarez-Moya et al., 2009). Moreover, high levels of cognitive impulsivity are also implicated in the onset and maintenance of binge eating episodes (Steiger, Lehoux, & Gauvin, 1999).

Neurophysiological disturbances among BN patients, namely alterations in the orbitofrontal cortex, the area of the brain associated with decision-making, are also indicative of difficulties in cognitive impulse control (Frank & Kaye, 2005; Lowe, van Steenburgh, Ochner, & Coletta, 2009).

It is important to note that most of the aforementioned studies involved clinical samples of individuals meeting full diagnostic criteria for an ED. Thus, these findings are not directly generalizable to individuals with subthreshold symptoms or non-treatment-seeking individuals. Individuals who seek treatment for an ED typically manifest more severe symptomatology than individuals with EDs recruited from the community (Shaw & Garfinkel, 1990), including comorbid Axis I and Axis II disorders (Telch & Stice, 1998). Individuals in clinical treatment for EDs are also more commonly White/Caucasian (Pike, Dohm, Striegel-Moore, Wilfley, & Fairburn, 2001; R. H. Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000), and therefore are not representative of non-treatment seeking individuals who binge eat (Mitchell & Mazzeo, 2004). Thus, although extant data provide strong evidence for the presence of comorbid neuropsychological impairments among those who engage in binge eating behaviors, these findings should be replicated in community-based samples to enhance their generalizability (J. G. Johnson et al., 2002; Ross & Ivis, 1999; Saules et al., 2009).

Executive functioning and depression. As noted previously, depression is one of the most common comorbid symptoms of binge eating (J. G. Johnson et al., 2002; Ross & Ivis, 1999; Spoor et al., 2006; Stice et al., 2002). Research suggests that individuals with depression also present with impairments in executive functioning, particularly cognitive inhibition (Moritz et al., 2002) and rigidity (Gualtieri, Johnson, & Benedict, 2006). A review of the relation between executive functioning and depression (Fossati, Ergris, & Allilaire, 2002) indicates that

impairments in cognitive inhibition, problem-solving, and planning lead to the inappropriate allocation of cognitive resources. For example, someone with depression and impairments in their executive functioning may spend less time planning and implementing the steps needed to perform well on an upcoming exam and spend more time worrying about what might happen if they perform poorly in school. Thus, individuals with depression often find it difficult to practice mood controlling techniques or to effectively cope with their symptoms. Given these findings, in the current study depression scores were controlled for in analyses investigating the links between binge eating and executive functioning.

Diagnostic Considerations

To meet DSM-IV diagnostic criteria for a “binge episode,” individuals must consume an amount of food that is larger than most people would eat during a similar period of time and under similar circumstances, followed by a sense of loss of control (American Psychiatric Association, 2000). These episodes are referred to as objective binge episodes (OBEs). Subjective binge episodes (SBEs), however, do not meet this *DSM-IV* standard, as they involve “normal” caloric consumption accompanied by feelings of loss of control. Loss of control is considered by many (Goossens, Braet & Decaluwe, 2007; Keel et al., 2001; Latner et al., 2007; Wolfe et al., 2009) to be the core pathological component of binge eating as it is most strongly associated with negative psychiatric outcomes. Latner et al. found no significant differences in ED-related symptoms, depression, anxiety or stress between women who engaged in OBEs versus SBEs. Goossens et al. reported similar psychosocial outcomes; no significant differences emerged in ED pathology, depression, or self-esteem between adolescents with OBEs or SBEs. Taken together, these findings suggest that feelings of loss of control, rather than amount of food

consumed, are more strongly associated with impaired psychosocial functioning. Thus, the current study recruited participants engaging in SBEs and/or OBEs.

Conclusions and Clinical Implications

Considered together, research suggests that binge eating is not a natural response to one's hunger or metabolic needs, but might instead represent a type of learned or innate maladaptive emotion regulation technique (Mathes et al., 2009). Indeed, there appear to be various biological and environmental systems driving binge eating behaviors. Investigations of the neuropsychology of binge eating are needed to elucidate further the underlying neurobiology of disordered eating. Specifically, cognitive deficiencies, such as difficulties considering alternative options to regulate one's emotions (i.e., poor planning, difficulties shifting set, cognitive rigidity), might modify and/or compound the effects of stress on disordered eating. Also, research suggests that neuropsychological deficits are linked to treatment outcomes among those with EDs; the greater the number of cognitive deficits, the worse the long-term prognosis (Hamsher, Halmi, & Benton, 1981). It is therefore important to gain a better understanding of the neuropsychological dysfunctions associated with binge eating as they might prove to be useful intervention targets.

Thus, the primary purpose of this study was to assess the executive functioning (i.e., cognitive impulsivity and flexibility, set shifting abilities) of young women (18 to 25 years old) with binge eating symptomatology in the absence of regular compensatory behavior and compare their outcomes to a control sample of young women with no history of binge eating. Variables previously associated with executive functioning (i.e., depression, state anxiety, BMI, and estimated intelligence) served as covariates (Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007; Strauss, Sherman, & Spreen, 2006). It was hypothesized that, after controlling for

depression, state anxiety, BMI and estimated intelligence, young adults who reported engaging in binge eating would demonstrate poorer executive functioning (i.e., a more impulsive cognitive style, greater difficulties with shifting and/or maintaining set, greater cognitive rigidity) compared with those who do not engage in binge eating behaviors.

A secondary aim of the current study was to explore differences in distress tolerance, behavioral impulsivity, emotion regulation, and depression between participants who binge eat and those with no history of binge eating. Individual differences in participants' BMI were controlled in these analyses. It was hypothesized that, after controlling for BMI, individuals who engage in regular binge eating would report higher depression, less distress tolerance, higher behavioral impulsivity (particularly negative urgency) and poorer emotion regulation capabilities (particularly rumination), compared with individuals who did not binge eat.

Finally, this study explored the associations between indicators of executive functioning and distress tolerance, behavioral impulsivity, and/or emotion regulation. If statistically indicated, additional analyses were planned to examine whether these associations differed between those who reported engaging in binge eating and those who did not. Specific hypotheses for this aim were not presented here as such analyses have not previously been conducted and are considered exploratory.

Methods

Procedure

This study is a between-groups design comprised of two parts:

Part I included a series of self-report questionnaires completed in-person, in a large computer laboratory, with an online database. Most participants completed this portion of the study in less than 30 minutes. The order in which the questionnaires were completed online was

randomized to avoid response habituation. In an effort to maintain their right to privacy, each participant's height and weight was collected by research staff in a separate room.

Part II entailed the completion of a brief, one hour neuropsychological battery (the order of which was also randomized). Women who reported (in Part I) that they engaged in weekly SBEs or OBEs in the absence of regular (i.e., three or more times per week) compensatory behaviors in the last 28 days were contacted and asked to participate in Part II. Women who reported no history of binge eating (in Part I) were randomly selected to participate in Part II.

The neuropsychological battery was completed individually with doctoral-level psychology graduate students with prior training in psychological and/or neuropsychological assessment. These students were required to attend a two-hour training session in which details of the consent procedures and debriefing process were reviewed, as was the administration and scoring of all neuropsychological measures. All assessment procedures were supervised by a licensed neuropsychologist.

Participants

Sample size. Participants were female undergraduate students recruited from the Psychology Department Subject Pool. Sample size was calculated a priori based on the primary aim. A power analysis was conducted with five predictor variables, including group assignment and four covariates (estimated intelligence, state anxiety, BMI, and depression). The sample size required to detect a medium effect size ($f^2 = .15$; Cohen, 1988; Cohen, Cohen, West, & Aiken, 2003) with a power level of .80 and alpha set at .05 was 91 participants. This effect size was selected based on the pilot nature of the current study. One study using one of the same neuropsychological outcomes and a similar (albeit clinical) sample as the current study has been

published since the current study was conducted (Duchesne et al., 2010), although data provided for this paper were not adequate to calculate effect sizes.

Recruitment. Participants were recruited from the Psychology 101 Participant Pool. Participation in this pool is one way in which students can fulfill their research course requirement. Students receive information from their instructors regarding alternative ways of fulfilling this course requirement. Interested participants register through SONA Systems and sign up for psychological experiments through its website (<http://vcu.sona-systems.com/>). Participants received one research credit for completing Part I of the current study, and two additional research credits for completing Part II. The Psychology 101 Participant Pool is an efficient recruiting system and allows researchers access to large samples (e.g., typically, the subject pool has up to 1200 participants each semester) with diverse racial/ethnic representation (i.e., approximately 25% Black/African American, 10% Asian/Asian American, 3% Hispanic/Latina).

Inclusion criteria. To be included in this study, participants had to be an undergraduate female between the ages of 18 and 25. The selected age restrictions minimize potential variations in the study's outcomes based on age and reflect the predicted age range based on previous studies utilizing similar samples (e.g., Mitchell, Mazzeo, Rausch, & Cooke, 2007; Mitchell & Mazzeo, 2004). To be eligible for Part II of the current study, participants had to meet one of two criteria: (a) engage in weekly SBEs and/or OBEs in the absence of regular (i.e., three or more times per week) compensatory behaviors in the last 28 days (as measured by the Eating Disorder Examination Questionnaire [EDE-Q; Fairburn & Beglin, 1994]; see Part I Measures section); or (b) report no history of or current binge eating behaviors.

Exclusion criteria. Participants were excluded from Part II of the current study if they reported significant or recent brain injuries (i.e., greater than 30 minutes of loss of consciousness, any memory loss or hospitalization, or greater ≥ 2 concussions within last 12 months), or the presence of underlying genetic, neurological, endocrine, or metabolic conditions that could greatly influence their eating behaviors (e.g., Prader-Willi, Cushing's syndrome). As employed in previous studies, if participants reported engaging in regular compensatory behaviors (Mond et al., 2006; Mond, Chen, & Kumar, 2009), met *DSM-IV* diagnostic criteria for AN (American Psychiatric Association, 2000), or had a BMI below 18.5 (i.e., are underweight), they were excluded from the study. Psychotropic medication use was assessed in order to determine its influence on neuropsychological outcomes.

Sample characteristics. A total of 639 women completed Part I of the current study. From this sample, eight participants' data were removed from the dataset due to significant missing data (greater than 75%) and/or the appearance of random responding (e.g., selecting "5" for every question), resulting in a final total sample size of 631; *M* age = 19.2, *SD* = 1.5; 44.4% (*n* = 279) identified as White/Caucasian, 26.6% (*n* = 167) as Black/African American, 14.6% (*n* = 92) as Asian/Asian American, 6.5% (*n* = 41) as Hispanic/Latina, and 7.8% (*n* = 49) as Other. Three people (0.5%) failed to report their race/ethnicity. Based on the EDE-Q (Fairburn & Beglin, 1994), a total of 59.5% of women denied engaging in any current or previous binge eating; 17.3% reported engaging in at least one (but less than four) binge episodes in the last 28 days, and 23.2% reported at least four binge episodes in the last 28 days. Thus, over 40% of women endorsed recent binge eating behavior, consistent with previous research (Ross & Ivis, 1999; Saules et al., 2009).

Of these 631 women, 38 participants (5.9%) failed to provide consent for Part II of this study and were not re-contacted. Others were excluded from participation in Part II for the following reasons: 109 women endorsed binge eating less than weekly, 35 women had a BMI below 18.5, 110 women reported engaging in regular compensatory behaviors (90% of which were exercising excessively), seven women reported an underlying condition that may significantly influence their eating habits (e.g., Ulcerative Colitis), and 14 women indicated a history of significant brain injury or more than three concussions within the last three months. Several women were excluded for more than one of these reasons.

A total of 116 women completed Part II of the current study; 66 denied engaging in current or previous binge eating (no binge group) and 50 endorsed regular binge eating behavior (binge group; see Table 1 for demographic information for each group). Women in the binge eating group endorsed an average of 9.2 binges ($SD = 6.63$; range = 4 - 40) in the previous 28 days. As Table 1 indicates, 12 participants endorsed current psychopharmaceutical use. Chi-square analyses indicated that the percentage of participants using psychopharmaceuticals significantly differed by binge eating group, $\chi^2(1, N = 116) = 5.55, p < .02$; participants engaging in regular binge eating were more likely to report current psychopharmaceutical use relative to their non-bingeing peers. This finding is not surprising considering the comorbid psychopathology reported frequently among women engaging in binge eating (e.g., W. G. Johnson et al., 2002; Ross & Ivis, 1999; Spoor et al., 2006; Stice et al., 2009; R. Striegel-Moore, 1995), including Attention Deficit/Hyperactivity Disorder (ADHD; Cortese, Bernardina, & Mouren, 2007). Groups also differed significantly in regard to racial/ethnic composition, $\chi^2(4, N = 116) = 14.05, p < .007$; relative to the non-binge eating group, a greater percentage of women in the binge eating group identified as White. In contrast, a greater percentage of women

in the non-binge eating group identified as Black/African American, Hispanic/Latina, or Asian/Asian American (see Table 1).

Several 2x2 (binge group x psychopharmaceutical group) and 2x5 (binge group x race/ethnicity group) between-groups analyses of variance (ANOVA) were conducted to determine whether executive functioning outcomes differed significantly between binge eating groups based on race/ethnicity or psychopharmaceutical use, respectively. Interaction terms in ANOVAs examining the influence of race/ethnicity and binge eating group on executive functioning outcomes were not significant, WCST Total Errors, $F(4, 113) = 1.42, p = .233$; WCST Perseverative Responses, $F(4, 113) = 0.90, p = .464$; CPT-II Errors of Commission, $F(4, 113) = 1.11, p = .358$; CPT-II Reaction Time, $F(4, 113) = 1.52, p = .202$, suggesting that between-groups differences in executive functioning were not significantly influenced by race/ethnicity.

With respect to psychopharmaceutical use, the interaction terms for some ANOVAs were non-significant, WCST Total Errors, $F(1, 113) = 1.13, p = .291$; WCST Perseverative Responses, $F(1, 113) = 1.80, p = .183$, while others were significant, CPT-II Errors of Commission, $F(1, 113) = 8.50, p < .004$; CPT-II Reaction Time, $F(1, 113) = 4.59, p < .04$. Significant interaction terms suggest that, among individuals in the binge eating group, psychopharmaceutical use was associated with significantly higher Perseverative Responses T scores and significantly lower Reaction Time T scores, suggesting less difficulties thinking flexibly or shifting cognitive sets, and speedier reaction times. In contrast, among those in the non-binge eating group, psychopharmaceutical use was associated with worse Perseverative Response scores and slower reaction times. As a result of these significant interaction terms, psychopharmaceutical use was added to the primary aims analyses as a covariate.

Table 1.

Participant Demographic Information

Demographic	Binge Group (<i>n</i> = 50)		No Binge Group (<i>n</i> = 66)	
Age	<i>M</i> = 19.32	<i>SD</i> = 1.65	<i>M</i> = 19.03	<i>SD</i> = 1.27
Body Mass Index	<i>M</i> = 24.46	<i>SD</i> = 5.10	<i>M</i> = 23.41	<i>SD</i> = 5.18
Year in School				
Freshman	56.0%	<i>n</i> = 28	57.6%	<i>n</i> = 38
Sophomore	16.0%	<i>n</i> = 8	21.1%	<i>n</i> = 14
Junior	18.0%	<i>n</i> = 9	16.7%	<i>n</i> = 11
Senior	10.0%	<i>n</i> = 5	4.5%	<i>n</i> = 3
Race/Ethnicity				
White/Caucasian	56.0%	<i>n</i> = 28	25.8%	<i>n</i> = 17
Black/African American	24.0%	<i>n</i> = 12	43.9%	<i>n</i> = 29
Asian/Asian American	8.0%	<i>n</i> = 4	15.2%	<i>n</i> = 10
Hispanic/Latina	2.0%	<i>n</i> = 1	9.1%	<i>n</i> = 6
Other	10.0%	<i>n</i> = 5	6.1%	<i>n</i> = 4
Current Medication Use				
Yes	18.0%	<i>n</i> = 9	4.5%	<i>n</i> = 3
No	82.0%	<i>n</i> = 41	95.5%	<i>n</i> = 63

The total sample size of 116 is higher than suggested based on the aforementioned power analysis. It also parallels Tabachnick and Fidell's (2007) rule of thumb for adequate sample sizes when using multiple regression. Specifically, N should be $\geq 50 + 8m$ (where m is the number of predictor variables). Including the addition of the psychopharmaceutical use covariate, the current study includes six predictor variables suggesting that at least 98 individuals should participate in the current study. Thus, the sample size is sufficient for analysis of the primary aim.

Informed consent. Prior to completing any component of the current study, participants provided consent in person. Informed consent procedures were conducted in accordance with the Institutional Review Board (IRB). Specifically, participants were asked to provide informed consent for this protocol acknowledging that they were informed about the purpose, description, potential risks or discomforts, benefits, cost of participation, payment for participation, confidentiality, and withdrawal procedures associated with the current study.

Part I Measures

Demographic questionnaire. Participants were asked to provide information about their age, year in school, race/ethnicity, psychotropic medication use, previous brain injuries, and presence of pre-existing brain lesions and/or underlying genetic, neurological, endocrine, or metabolic conditions that could greatly influence their eating behaviors (e.g., Prader-Willi, Cushing's syndrome).

Body mass index (BMI). Height and weight data were ascertained in-person, in a private area, and BMI was calculated. This method of assessment was selected over self-report as research suggests that women have a tendency to under-report their weight, particularly if they

are overweight or obese (Elgar & Stewart, 2008; Ezzati, Martin, Skjold, Vander Hoorn, & Murray, 2006; Shields, Gorber, & Tremblay, 2008; Stommel & Schoenborn, 2009).

State-Trait Anxiety Inventory for Adults (STAI). The STAI is a self-report measure of anxiety, consisting of two subscales, state and trait anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The complete measure includes 40 items with response options ranging from 1 (*not at all or almost never*) to 4 (*very much so or almost always*). The state subscale, the only one administered in this study, consists of 20 statements that measure how an individual is feeling in the moment. The range of total scores is 20-80; the higher the score indicating greater anxiety. This measure was given to participants in Part II immediately before completing the neuropsychological battery.

The STAI yields internally consistent scores (Cronbach's alphas $> .90$; Spielberger et al., 1983). Additionally, concurrent, convergent, divergent, and construct validity of the STAI were demonstrated by its developers (Spielberger et al., 1983). Novy et al. (1993) investigated the utility of the STAI among White/Caucasian, Black/African American, and Hispanic/Latino/a men and women (the sample included college students, high school students, military recruits, and working adults; $N = 285$). These researchers found that the STAI demonstrated acceptable internal consistency in all gender and ethnic groups (Cronbach's alphas $> .90$). Furthermore, Novy and colleagues (1993) found that the STAI demonstrated convergent and divergent validity among all ethnic groups with various subscales of the Minnesota Multiphasic Personality Inventory. Cronbach's alpha in the current study was .94.

Eating Disorder Examination-Self-report Questionnaire (EDE-Q). Binge eating behavior (both subjective and objective) was assessed with the EDE-Q (Fairburn & Beglin, 1994), a 41-item instrument adapted from a structured clinical interview (i.e., EDE). The EDE-Q parallels the EDE (Fairburn & Beglin), the "gold standard" of ED pathology assessment (Garner,

1995), and items were adapted only to make them suitable for administration as a self-report measure. Good two week test-retest reliability for this measure was ascertained in an undergraduate sample of women (.81; Luce & Crowther, 1999). In other studies, EDE and EDE-Q subscales were significantly correlated ($r = 0.60$ to 0.77), suggestive of adequate convergent validity (Kalarchian, Wilson, Brolin, & Bradley, 2000; Wilfley, Schwartz, Spurrell, & Fairburn, 1997). For the current study, an additional item was added to this measure to assess for the presence of lifetime binge eating behaviors.

Center for Epidemiological Studies Depression Scale (CES-D). The CES-D is a 20-item self-report measure of depressive symptomatology (Radloff, 1977) that yields internally consistent scores (Cronbach's alpha = 0.85). The measure also discriminates between depressed and non-depressed individuals and exhibits convergent validity with other measures of depression (Radloff, 1977). Depressive symptoms for the previous week are rated on a four point scale from 0 (*Rarely, None of the Time, or Less than One Day*) to 3 (*Most or All of the Time, or 5-7 Days*). Positive items are reverse scored; higher scores indicate more severe depressive symptomatology (on a scale of zero to 60). The CES-D has a cutoff of 16, indicating risk of clinical depression (Myers & Weissman, 1980; Roberts & Vernon, 1983). However, this suggested cutoff score has been shown to overestimate the prevalence of depression among undergraduate students (Santor, Zuroff, Ramsay, & Cervantes, 1995), and a clinical cutoff score of 24 has been recommended for use with women in college (Price, McLeod, Gleich, & Hand, 2006). Cronbach's alpha in the current study was .92.

UPPS Impulsive Behaviour Scale (UPPS). The UPPS Scale is a 45-item questionnaire which assesses four domains of impulsivity: lack of planning, lack of perseverance, negative urgency, and sensation seeking (S. P. Whiteside & Lynam, 2001). Each item is rated on a scale

ranging from 1 (*Not true of me*) to 5 (*Very true of me*). Total scores for each subscale range from one to four, with higher scores indicating impulsive behavior. Subscale scores manifest discriminant validity from one another (Smith et al., 2007). In addition, the UPPS effectively discriminated between a healthy control group and a group of individuals with significant pathological symptoms, including substance abuse, pathological gamblers, and borderline personality traits (S. P. Whiteside, Lynam, Miller, & Reynolds, 2005). In a sample of undergraduate men and women, internal consistencies were as follows: lack of premeditation 0.83, negative urgency 0.87, sensation seeking 0.84, and lack of perseverance 0.79 (Cyders, Flory, Rainer, & Smith, 2009). Similar internal consistency scores have been ascertained with other undergraduate samples (Anestis et al., 2007; Magid & Colder, 2007). In the current study, Cronbach's alphas were: .83 (lack of premeditation), .87 (lack of perseverance), .91 (negative urgency), and .85 (sensation seeking).

Cognitive Emotion Regulation Questionnaire (CERQ). Emotion regulation was assessed using the CERQ (Garnefski, Kraaij, & Spinhoven, 2002). The CERQ consists of 36 items and nine conceptually different subscales (i.e., self-blame, acceptance, rumination, positive refocusing, refocus on planning, positive reappraisal, putting into perspective, catastrophizing, and other-blame). Each subscale consists of four items and each item is rated on a five-point scale ranging from 1 (*Almost Never*) to 5 (*Almost Always*). Subscale scores are obtained by adding up the four items (giving a sub-scale range from 4 to 20), indicating the extent to which a certain cognitive coping strategy is used. The measure's subscales are internally consistent with Cronbach's alphas that range from .75 to .87 (Garnefski & Kraaij, 2007; Garnefski et al., 2002); alphas ranged from .73 to .86 in the current study.

Distress Tolerance Scale (DTS). The DTS examined participants' capacity to experience and withstand negative psychological states (Simons & Gaher, 2005). The measure includes 15 self-report items with a response format ranging from 1 (*Strongly Agree*) to 5 (*Strongly Disagree*). It is comprised of four subscales: tolerance, appraisal, absorption and regulation. Lower scores indicate greater difficulties tolerating distress. One study utilizing a college sample ($N = 642$) provided evidence for the convergent, discriminant, and criterion validity of this scale (Simons & Gaher). The DTS has also demonstrated good internal consistency in a sample of undergraduate women (Cronbach's $\alpha = .91$; Anestis et al., 2007). In the current study, Cronbach's alphas were .82 (tolerance), .90 (appraisal), .86 (absorption), and .75 (regulation).

Part II Measures: Neuropsychological Tests

Conner's Continuous Performance Task (CPT-II). The CPT-II is a computerized measure of sustained attention, behavioral disinhibition, and cognitive impulsivity (Conners & MHS Staff, 2000). Instructions for this measure are presented on the computer screen. Participants are first administered a brief set of practice items, after which the test begins. For each item, participants are asked to strike a key when the letter "X" is presented. Test administration time is always 14 minutes. The measure has good internal consistency (Cronbach's $\alpha = .66$ to $.95$; Conners, 1994) and is able to discriminate effectively between normal controls and individuals with ADHD (Conners & MHS Staff). The errors of commission (i.e., when responses are given to non-targets) T-Score was used to assess behavioral disinhibition or cognitive impulsivity; higher scores are indicative of greater cognitive impulsivity and behavioral disinhibition. Participants' Hit Reaction Time T-scores were also evaluated; higher scores are indicative of a slower reaction speed.

Wisconsin Card Sorting Test (WCST). The WCST is an assessment of strategic planning and the ability to utilize environmental feedback to shift cognitive sets (Heaton et al., 1993). This measure of executive functioning is completed on a computer (Heaton & PAR Staff, 2005). Participants are asked to place the top card from a single deck on to the top of one of four stimulus cards. The computer then informs the participant as to whether or not his or her card placement was correct based on a pre-established set of patterns. Participants are then asked to use this information to obtain as many correct cards as possible. Total administration time varies between 15 and 30 minutes. Performance outcomes extracted for analyses included T-score values for participants' Total number of Errors, as well as their Perseverative Responses; higher T-scores are indicative of better performance. Together, these outcomes indicate individuals' ability to think abstractly, shift and maintain cognitive set, and demonstrate cognitive flexibility (Heaton et al.).

Factor-analytic studies and structural equation modeling provide evidence for the construct validity of the WCST and its ability to assess executive functions, namely the ability to shift set (or think flexibly) and problem-solve (Fisk & Sharp, 2004; Greve, Stickler, Love, Bianchini, & Stanford, 2005; Miyake et al., 2000). Additional validity for this measure is demonstrated by previous research in which individuals with disorders and injuries characteristic of executive dysfunction, such as TBI, schizophrenia, Parkinson's disease and autism, demonstrate impaired performance on the WCST; moreover, in many cases, symptom severity is significantly correlated with WCST outcomes (see Lezak et al., 2004 for a review). Finally, according to 747 randomly selected members of American Psychological Association's Division 40, National Academy of Neuropsychology, and the International Neuropsychological Society, the WCST was the most favored tool for assessing executive functioning (Rabin, Barr, & Burton,

2005), with over 75% of respondents ranking it their first choice assessment instrument for executive functioning.

Wide Range Achievement Test 4 (WRAT4). The WRAT4 is a measure of academic achievement in the domains of reading, spelling and arithmetic (Wilkinson & Robertson, 2006). Studies suggest that executive functioning is correlated with general cognitive functioning (i.e., intelligence (Strauss et al., 2006). In an effort to minimize participant burden, general cognitive functioning was not assessed in the current study. Instead, the Reading subtest of the WRAT-4 was administered as an estimate of general cognitive functioning it has been correlated with IQ ($r = .69-.70$). Thus, the WRAT-4 Reading subtest was referred to as *estimated intelligence*, individual differences in which were controlled for when examining performance on measures of executive functioning. Items from the Reading subscale are internally consistent with Cronbach's alphas that range from .88 to .93 for 17 to 24 year olds; coefficients for split-half reliabilities were similar (Wilkinson & Robertson). WRAT4 subtests also correlate significantly with other tests of achievement and cognitive abilities providing evidence of concurrent validity (Wilkinson & Robertson).

Statistical Procedures

Preliminary analyses. IBM SPSS Statistics 19.0 (Chicago, IL) was used for data entry and analyses. Data that were not collected online were entered into SPSS and verified twice by undergraduate Research Assistants. Cronbach's alphas were computed to assess internal consistency reliability for all of the current study's measures. Descriptive statistics were calculated to ensure all data were in range and to determine what percentage of values were missing for each variable. Unless otherwise noted, all statistical tests were interpreted at the 5% significance level.

Descriptive statistics conducted with those that completed Part II of this study ($N = 116$) indicated that missing data were minimal. Specifically, two items from the UPPS were missing three values (2.6%), one item from the CESD was missing two values (1.7%), and two items from the CERQ were missing one value (0.9%). Scoring instructions for the CERQ (Garnefski et al., 2002) and UPPS (S. P. Whiteside & Lynam, 2001) describe how to calculate subscale mean scores in the presence of missing data; as such, missing values were not imputed for these measures. For the missing CESD values, individual mean imputation (i.e., mean of a given participant's complete responses to other scale items) was used. This approach produces accurate results with the CESD (Bono, Ried, Kimberlin, & Vogel, 2007) and with other single construct measures in which missing data are minimal (Shrive, Stuart, Quan, & Ghali, 2006) and their estimated internal consistency is high ($\alpha > .70$; Osborne, 2008).

Four participants also had incomplete neuropsychological data (i.e., completed two of three tests) due to computer malfunction. As such, these data were assumed to be missing completely at random (MCAR), or not related to other measured variables. Little's MCAR analyses conducted separately for each group resulted in non-significant chi-square tests (binge group $\chi^2 = 62.71$, $df = 58$; $p = .313$; non-binge group $\chi^2 = 20.67$, $df = 88$; $p > .100$), confirming that the missing neuropsychological data were not related to any demographic, predictor or criterion variable. In studies in which missing data are both minimal (i.e., less than 5%; Schlomer, Bauman, & Card, 2010) and MCAR, any method of handling missing data is considered appropriate and unlikely to lead to biased statistical analyses (Buhi, Goodson, & Neilands, 2008; Duffy, 2006). Thus, pairwise deletion was employed in the current study.

As suggested, assumptions of parametric data were evaluated within groups (Tabachnick & Fidell, 2007). Skewness and kurtosis values, as well as probability plots, were examined to

identify non-normal variables and univariate outliers, respectively. Several variables were skewed and/or kurtotic. To determine whether data entry errors were contributing to this non-normality, univariate outliers were identified and evaluated. None of these values were outside the expected score range and were thus retained. To produce a distribution more robust to the effects of outliers, BMI scores were Winsorized (Erceg-Hurn & Mirosevich, 2008; Keselman, Algina, Lix, Wilcox, & Deering, 2008; Wilcox, 2005). To compute Winsorized scores, the highest and lowest 10% of scores were replaced with the smallest and highest untrimmed scores, respectively. For the remaining nonnormal variables, Winsorized scores could either not be computed (e.g., 25% of participants endorsed the lowest possible total score) or would significantly change the distribution (i.e., variables were highly skewed). In these cases, transformations were applied.

If a variable was significantly non-normal in one group, the variable was transformed in both groups to facilitate between-group comparisons. Prior to conducting transformations, all negatively skewed variables were reflected, or made positive by subtracting each value from a constant number one value about the highest value for this variable. Then, square root, log and inverse transformations were conducted sequentially with all skewed and/or kurtotic variables until the normality assumption was adequately met. After transformations were applied, the Appraisal subscale of the DTS remained significantly skewed in one group. When significantly skewed variables are incapable of being normalized via transformations, it is recommended that the variable be dichotomized (MacCallum, Zhang, Preacher, & Rucker, 2002; Streiner, 2002). Thus, the DTS Appraisal subscale was dichotomized based on its median score (0 = below the median, 1 = above the median).

After all variables were either normalized via transformations or dichotomized, the following assumptions were examined and determined to be sufficiently met: multivariate outliers (via the Mahalanobis distance cutoff as determined by number of predictor variables) and normality, multivariate linearity and homoscedasticity (via a plot of the residuals), and multicollinearity (via a correlation matrix, as well as Tolerance and Variance Inflation Factor [VIF] totals; Tolerance values were not less than $< .1$ and VIF values were not >10 .; Tabachnick & Fidell, 2007).

Primary aim analyses. Multiple linear regression is an extension of simple linear regression in which the relation between one predictor variable and one outcome variable is explored. Multiple linear regression models attempt to predict individuals' performances on one variable (i.e., criterion variable) on the basis of several other variables (i.e., predictor variables). Having more than one predictor variable is useful when predicting human behavior, as individuals' actions, thoughts and emotions are all likely to be influenced by a combination of factors. In this way, multiple linear regression models represent a more accurate model or theory of human behavior than simple linear regression models as they explore how multiple variables, or variable sets, influence outcomes.

For the current study's primary aim analyses, there are four criterion variables, including Errors of Commissions T-Score, Reaction Time T-Score, Total Errors T-Score, and Perseverative Responses T-Score, collectively referred to as executive functioning. There are six predictor variables (i.e., depression, state anxiety, BMI, estimated intelligence, psychopharmaceutical use group, and binge eating group). The primary hypothesis posits that, after controlling for depression, state anxiety, BMI, psychopharmaceutical use, and estimated intelligence, young adults who engage in regular binge eating will demonstrate poorer executive

functioning compared to those with no history of engaging in binge eating. To test this hypothesis, hierarchical regression analyses (HRA) were employed. Separate HRAs were conducted for each criterion variable or indicator of executive functioning.

HRA is one type of multiple linear regression model in which the predictive power of certain variables is evaluated simultaneously in stages or blocks. The order in which the predictor variables are entered into the regression model is specified by the researcher and should reflect some theoretical consideration or previous findings. For the primary hypothesis, each HRA had two blocks/steps. The predictor variables entered in to the first block of each regression model were depression, state anxiety, BMI, psychopharmaceutical use (dummy coded 0 = no use, 1 = current use) and estimated intelligence (Model 1). Most of these variables were pre-selected based on previous research which showed that they are associated with executive functioning (Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007; Strauss et al., 2006); the addition of psychopharmaceutical use as a covariate was dictated by the current study's preliminary analyses described previously. Then, participants' binge eating status was dummy coded (0 = those with no history of binge eating; 1 = those currently engaging in binge eating behaviors) and entered in to the second block of each regression model (Model 2). Outcomes from these models indicated whether binge eating behaviors were uniquely associated with measures of executive functioning above and beyond the variance due to depression, state anxiety, BMI, psychopharmaceutical use, and estimated intelligence.

The following output for each HRA was used to determine whether the primary hypothesis was supported. In the first output box (titled Model Summary), R-square indicated the amount of variance in the criterion variable that was accounted for by each block of predictor variables. R-square for Model 2 indicated whether additional variance was added to the model by

including the grouping variable (i.e., those who binge eat versus those who do not). The Sig. F Change value indicates whether such a change is significant, and thus directly addresses the primary hypotheses.

Hierarchical regression analyses were selected to address the current study's aims due to limitations in using Analyses of Covariance (ANCOVA) with pre-existing groups. ANCOVA is a common statistical method employed to control for between-group differences in various variables, or covariates, which are associated with the criterion variables. More specifically, the ANCOVA adjusts the mean of each group's criterion variables as if all participants scored equally on the covariates (Tabachnick & Fidell, 2007). In the current study, an ANCOVA would assume that participants in each group are not significantly different when it comes to their depression scores or BMI. However, as has been noted previously, those who binge eat are more likely to endorse higher depressive symptomatology and body mass compared to individuals who do not binge eat (Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007; Strauss et al., 2006). Thus, performing analyses in which it is assumed that the two groups do not differ on these variables is likely to lead to biased outcomes. For this reason, and as others have noted (e.g., Jamieson, 2004; G. A. Miller & Chapman, 2001), ANCOVAs are frequently misused. HRA, on the other hand, is not limited by this assumption and can be utilized with naturally occurring or experimentally manipulated predictor variables (Grimm & Yarnold, 1995).

Moreover, statistical assumptions for the ANCOVA indicate that covariates must be unrelated to or independent from the study's predictor variable (Tabachnick & Fidell, 2007). In the current study, it is predicted that BMI and depression will be related to the predictor variable (i.e., group assignment; Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007). Another notable advantage of multiple linear regression techniques is their flexibility in handling

correlated predictor variables (Grimm & Yarnold, 1995; Tabachnick & Fidell). Assumptions for multiple regressions permit such associations as long as they are not correlated above .8, thereby violating the assumption of multicollinearity (Tabachnick & Fidell).

Secondary aim analyses. HRAs were also used to examine the study's secondary hypotheses, that, after controlling for BMI, individuals who engage in binge eating will report higher depressive symptoms, less distress tolerance, higher behavioral impulsivity (especially negative urgency), and poorer emotion regulation capabilities (particularly rumination), compared with individuals who do not binge eat. One HRA was completed for each criterion variable. Once again, the selection of BMI as a covariate was based on previous research; those with higher BMIs are often more likely to endorse poorer psychosocial functioning across multiple domains (Herman, Hopman, & Craig, 2010). Thus, to statistically control for variations in the outcome variables due to BMI, participants' body mass was entered in to the first block (Model 1). Group assignment was dummy coded and added to the second block (Model 2).

To examine whether groups differed on the DTS Appraisal subscale, the only criterion variable dichotomized due to significant normality violations, a logistic regression analysis was conducted. Logistic regression can be used to predict a dichotomous categorical variable, such as group membership, from a set of predictor variables (which can be categorical or continuous). In this analysis, BMI was entered in to the first block (Model 1) and the DTS Appraisal subscale (dummy coded as 0 and 1 based on its median) was entered into the second block (Model 2). Group membership (binge eating or not) served as the criterion variable. Results from these analyses indicated whether, after controlling for variance due to participants' body mass, DTS Appraisal subscale scores account for significantly more variance in the model, thereby predicting the likelihood that participants are binge eaters or not. Thus, these analyses essentially

indicated whether the groups differed significantly with respect to their DTS Appraisal scores after controlling for the predictive power of BMI.

Exploratory aim analyses. The final aim of the study was to explore whether indicators of executive functioning were related to distress tolerance, behavioral impulsivity, and/or emotion regulation. This aim was addressed with a single Pearson's correlation matrix table. To correct for multiple comparisons and avoid Type I error (i.e., false positives), a Bonferroni correction was applied. This correction resulted in a new alpha level of .0025 to determine significance (.05/20 comparisons).

Results

Results Related to the Primary Aim

HRAs suggest that, after controlling for variance due to depression, state anxiety, BMI, psychopharmaceutical use, and estimated intelligence, the binge eating grouping variable did not account for any additional significant variance in the model associated with executive functioning outcomes. In other words, individuals who engage in binge eating did not differ significantly from those who do not with respect to their cognitive impulsivity or rigidity, or their ability to shift or maintain set, after accounting for differences in depression, state anxiety, BMI, psychopharmaceutical use, and intelligence (see Table 2). Moreover, both groups' adjusted mean scores (back transformed, as appropriate) were within one standard deviation of the mean T-score (i.e., $M = 50$, $SD = 10$) for each outcome, indicating "normal" executive functioning (CPT-II Errors of Commission, $M_{\text{binge}}=55.99\pm 11.74$, $M_{\text{non-binge}}=53.65\pm 11.53$; WCST Total Errors, $M_{\text{binge}}=48.90\pm 12.69$, $M_{\text{non-binge}}=54.72\pm 12.41$; WCST Perseverate Responses, $M_{\text{binge}}=52.82\pm 14.92$, $M_{\text{non-binge}}=58.15\pm 14.59$; and CPT-II Reaction Time, $M_{\text{binge}}=45.77\pm 9.74$, $M_{\text{non-binge}}=45.76\pm 9.57$). Contrary to previous research, most covariates did not have a significant effect on any indicators

of executive functioning. The only significant effects were found for general intelligence ($M_{\text{binge}}=99.35\pm 9.75$, $M_{\text{non-binge}}=101.12\pm 9.42$) on WCST Total Errors and BMI on WCST Perseverative Responses (refer to Table 3 for correlation coefficients for continuous predictor and criterion variables).

Table 2.

Results of Hierarchical Regression Analyses Examining Between Group Differences in Indicators of Executive Functioning

Outcome	Predictor	Model 1					Model 2					
		B	SEB	β	R ²	F Δ	B	SEB	β	R ²	F Δ	f ²
CPT-II Commission												
	BMI	-0.26	0.31	-0.08	0.04	0.82	-0.33	0.32	-0.10	0.04	0.74	0.01
	CESD	0.22	0.87	0.03			-0.02	0.92	0.00			
	SAI	7.86	9.55	0.10			7.06	9.61	0.09			
	WRAT4	-0.09	0.12	-0.08			-0.09	0.11	-0.08			
	Psychopharmaceutical Group	3.45	3.40	0.10			3.05	3.44	0.09			
	Binge Eating Group						2.06	2.39	0.10			
CPT-II Reaction Time												
	BMI	0.03	0.02	0.14	0.04	0.81	0.03	0.02	0.14	0.04	0.020	0.00
	CESD	-0.02	0.05	-0.03			-0.02	0.06	-0.04			
	SAI	0.40	0.58	0.08			0.39	0.58	0.08			
	WRAT4	0.00	0.01	0.00			0.00	0.01	0.00			
	Psychopharmaceutical Group	-0.27	0.21	-0.13			-0.28	0.21	-0.13			
	Binge Eating Group						0.02	0.15	0.02			
WCST Total Errors												
	BMI	-0.02	0.01	-0.15	0.06	1.36	-0.02	0.01	-0.16	0.06	0.06	0.00
	CESD	0.01	0.03	0.02			0.00	0.03	0.01			
	SAI	-0.01	0.32	0.00			-0.01	0.33	-0.01			
	WRAT4	-0.01	0.00	-0.19			-0.01	0.00	-0.20			
	Psychopharmaceutical Group	0.01	0.12	0.01			0.00	0.12	0.00			
	Binge Eating Group						0.02	0.08	0.03			

WCST Perseverative Responses

BMI	0.67	0.40	0.16	0.05	1.05	0.85	0.41	0.20	0.07	2.95	0.03
CESD	-0.41	1.13	-0.04			0.21	1.17	0.02			
SAI	9.68	12.30	0.09			11.72	12.24	0.11			
WRAT4	0.18	0.14	0.13			0.20	0.14	0.14			
Psychopharmaceutical Group	-1.94	4.38	-0.04			-0.94	4.38	-0.02			
Binge Eating Group						-5.24	3.05	-0.19			

Note. * $p \leq .05$; BMI = Body Mass Index; CESD = Center for Epidemiological Studies Depression Scale; SAI = State Anxiety Inventory; WRAT4 (Wide Range Achievement Test - 4th edition)

Table 3.

Associations between Predictor and Criterion Variables for Primary Aim

	1	2	3	4	5	6	7
1 CPT-II Commissions	—						
2 CPT-II Reaction Time	** <i>-0.57</i>	—					
3 WCST Total Errors	0.12	-0.10	—				
4 WCST Perseverative	0.02	0.08	** <i>-0.81</i>	—			
5 WRAT4 Reading	-0.06	0.00	* <i>-0.19</i>	0.13	—		
6 CESD	0.09	0.00	0.00	0.02	0.07	—	
7 SAI	0.13	0.03	-0.01	0.07	0.10	** <i>0.57</i>	—
8 BMI	-0.07	0.13	-0.15	0.15	-0.01	0.07	-0.02

Note. ** $p < .01$, * $p < .05$; CPT-II = Conner's Continuous Performance Task (2nd edition); WCST = Wisconsin Card Sorting Task; WRAT4 = Wide Range Achievement Test (4th Edition); CESD = Center for Epidemiological Studies Depression Scale; SAI = State Anxiety Inventory; BMI = Body Mass Index

Interestingly, despite the lack of significant between-group differences in indicators of executive functioning, among the binge eating group, total number of binge episodes (log transformed to meet the normality assumption), was significantly, albeit moderately, correlated with Perseverative Responses T scores ($r = -.33$); those engaging in more binge episodes demonstrated more difficulties thinking flexibly or shifting cognitive set. Total binge episodes was not significantly correlated with any other indicator of executive functioning (CPT-II Errors of Commission, $r = .20$; CPT-II Reaction Time, $r = -.16$; WCST Total Errors, $r = .26$).

Results Related to the Secondary Aims

HRAs suggest that, after controlling for variations in BMI, the binge eating grouping variable accounted for significantly more variance in the models associated with several secondary outcomes (see Table 4). Specifically, compared to those in the non-binge eating group, individuals in the binge eating group reported significantly greater depressive symptoms (CESD; $M_{\text{binge}} = 23.91 \pm 10.58$, $M_{\text{non-binge}} = 13.19 \pm 10.53$), as well as less perseverance (UPPS Perseverance; $M_{\text{binge}} = 2.04 \pm 0.54$, $M_{\text{non-binge}} = 1.73 \pm 0.54$) and greater negative urgency (UPPS Negative Urgency; $M_{\text{binge}} = 2.98 \pm 0.62$, $M_{\text{non-binge}} = 2.28 \pm 0.61$). As hypothesized, the link between binge eating and negative urgency produced the greatest effect size relative to any other variable in these analyses (see Table 4; by convention, f^2 effect sizes of 0.02, 0.15, and 0.35 are considered small, moderate, and large, respectively; Cohen, 1988); approximately 27% of the variability in negative urgency was predicted by binge eating group. The second largest effect size, in the moderate-to-large range, was found for the link between depression and binge eating; approximately 20% of the variability in depression was predicted by membership in the binge eating group.

Table 4.

Differences in Behavioral Impulsivity, Emotion Regulation, Distress Tolerance and Depression between Binge Eating and Non-Binge Eating Group

Outcome	Predictor	Model 1					Model 2					
		B	SEB	β	R ²	F Δ	B	SEB	β	R ²	F Δ	f ²
UPPS Premeditation	BMI Group	0.01	0.01	0.05	0.00	0.24	0.00	0.01	0.00	0.03	3.62	0.03
							0.17	0.09	0.18			
UPPS Perseverance	BMI Group	0.00	0.00	-0.04	0.00	0.19	0.00	0.00	-0.12	0.08	9.17**	0.09
							0.07	0.02	0.28			
UPPS Negative Urgency	BMI Group	0.04	0.02	0.18	0.03	3.99*	0.01	0.02	0.05	0.27	36.78**	0.33
							0.71	0.12	0.51			
UPPS Sensation Seeking	BMI Group	0.02	0.02	0.13	0.02	2.05	0.02	0.02	0.10	0.03	1.35	0.01
							0.13	0.12	0.11			
CERQ Self-Blame	BMI Group	0.02	0.02	0.15	0.02	2.67	0.01	0.01	0.05	0.16	18.91**	0.17
							0.40	0.09	0.39			
CERQ Acceptance	BMI Group	0.12	0.10	0.11	0.01	1.49	0.08	0.10	0.08	0.03	1.95	0.02
							0.93	0.67	0.13			
CERQ Rumination	BMI Group	0.05	0.10	0.05	0.00	0.27	-0.04	0.10	-0.04	0.11	12.97**	0.12
							2.32	0.64	0.33			
CERQ Positive Refocusing	BMI Group	0.19	0.10	0.18	0.03	3.98*	0.26	0.10	0.25	0.10	7.82**	0.08
							-1.79	0.64	-0.26			
CERQ Refocus on Planning	BMI Group	0.17	0.10	0.15	0.02	2.68	0.24	0.10	0.22	0.08	6.86**	0.07
							-1.77	0.68	-0.25			

CERQ Positive Reappraisal	BMI Group	0.17	0.12	0.14	0.02	2.14	0.25 -2.09	0.12 0.78	0.20 -0.25	0.08	7.19**	0.07
CERQ Perspective	BMI Group	0.11	0.11	0.09	0.01	0.92	0.16 -1.37	0.11 0.74	0.14 -0.18	0.04	3.44	0.03
CERQ Catastrophizing	BMI Group	0.01	0.01	0.13	0.02	1.86	0.00 0.14	0.01 0.03	0.02 0.42	0.18	22.18**	0.20
CERQ Other Blame	BMI Group	0.01	0.00	0.13	0.02	2.03	0.00 0.04	0.00 0.03	0.10 0.14	0.03	1.99	0.01
CESD	BMI Group	0.03	0.04	0.07	0.01	0.63	-0.02 1.30	0.04 0.25	-0.05 0.45	0.20	27.07**	0.24
DTS Tolerance	BMI Group	0.02	0.10	0.00	0.00	0.03	0.11 -2.38	0.10 0.64	0.11 -0.35	0.11	14.07**	0.12
DTS Absorption	BMI Group	-0.07	0.12	-0.06	0.00	0.42	0.05 -3.10	0.10 0.67	0.05 -0.41	0.16	21.47**	0.19
DTS Regulation	BMI Group	-0.08	0.09	-0.90	0.01	0.81	0.01 -2.20	0.08 0.56	0.01 -0.36	0.13	15.49**	0.14

Note. $n = 116$; ** $p \leq .01$, * $p \leq .05$; UPPS = UPPS Impulsive Behavior Scale; CERQ = Cognitive Emotion Regulation Questionnaire; CESD = Center for Epidemiological Studies Depression Scale; DTS = Distress Tolerance Scale; estimated marginal means were transformed to reflect their original score range

Participants in the binge eating group were also more likely to use self-blame (CERQ Self-Blame; $M_{\text{binge}} = 11.81 \pm 3.24$, $M_{\text{non-binge}} = 9.18 \pm 3.22$), rumination (CERQ Rumination; $M_{\text{binge}} = 12.63 \pm 3.38$, $M_{\text{non-binge}} = 10.32 \pm 3.36$), and catastrophizing (CERQ Catastrophizing; $M_{\text{binge}} = 9.67 \pm 3.29$, $M_{\text{non-binge}} = 6.99 \pm 3.28$) to regulate their emotions. Effect sizes for these associations were moderate; the binge eating grouping variable accounted for 16%, 11% and 18% of variability in each outcome, respectively. Relative to their non-bingeing peers, those engaging in regular binge eating were also less likely to refocus on the positive (CERQ Positive Refocusing; $M_{\text{binge}} = 9.22 \pm 3.37$, $M_{\text{non-binge}} = 11.01 \pm 3.35$), refocus on planning (CERQ Refocus on Planning; $M_{\text{binge}} = 11.22 \pm 3.56$, $M_{\text{non-binge}} = 12.99 \pm 3.49$), and utilize positive reappraisal (CERQ Positive Reappraisal; $M_{\text{binge}} = 11.81 \pm 4.09$, $M_{\text{non-binge}} = 13.90 \pm 4.08$). Effect sizes for these associations were small-to-moderate; the binge eating grouping variable accounted for 8%, 7% and 7% of variability in each outcome, respectively.

With respect to distress tolerance, the current study's groups differed on each DTS subscale. Participants in the binge eating group reported greater difficulties tolerating their distressing feelings (DTS Tolerance; $M_{\text{binge}} = 7.61 \pm 3.34$, $M_{\text{non-binge}} = 9.99 \pm 3.32$) relative to their non-bingeing peers. They also reported feeling as though they put more effort towards regulating their emotions (DTS Regulation; $M_{\text{binge}} = 6.99 \pm 2.93$, $M_{\text{non-binge}} = 9.19 \pm 2.92$) and that it absorbs more of their attention (DTS Absorption; $M_{\text{binge}} = 7.17 \pm 3.51$, $M_{\text{non-binge}} = 10.27 \pm 3.50$). Effect sizes for these associations were moderate; the binge eating grouping variable accounted for 11%, 16% and 13% of variability in each outcome, respectively.

Results from logistic regression also indicated a between-groups difference in participants' appraisal of distress (DTS Appraisal). A test of the full model with all predictors (BMI and DTS Appraisal dichotomized into high and low scoring groups) against a constant-

only model was statistically significant, $\chi^2(2, N = 116) = 7.72, p < .02$, indicating that the predictors, as a set, reliably distinguished between those who engage in binge eating and those who do not. According to the Wald criterion, the DTS Appraisal grouping variable was the only single significant predictor of binge eating group ($B = -1.11, SE = 0.41, Wald = 7.35, p < .007$, odds ratio = 0.331). Logistic regression coefficients indicated that, as DTS Appraisal scores increased, binge eating group decreased. In other words, those with DTS Appraisal scores above the median (dummy coded “1”) were more likely to be in the non-binge eating group (dummy coded “0”) and those with scores below the median (dummy coded as “0”) were more likely to be in the binge eating group (dummy coded as “1”). Thus, participants in the binge eating group subjectively evaluated their distress as greater than those in the non-binge group. However, *Nagelkerke’s R²* value of .086 suggests that the strength of this relationship is small (8.6%).

There were several outcomes on which the binge eating groups did not differ significantly, including their lack of premeditation (UPPS Premeditation; $M_{\text{binge}} = 2.02 \pm 0.05$, $M_{\text{non-binge}} = 1.85 \pm 0.05$) or sensation seeking behavior (UPPS Sensation Seeking; $M_{\text{binge}} = 2.86 \pm 0.60$, $M_{\text{non-binge}} = 2.73 \pm 0.60$), or their use of acceptance (CERQ Acceptance; $M_{\text{binge}} = 12.72 \pm 3.50$, $M_{\text{non-binge}} = 11.79 \pm 3.48$), other blame (CERQ Other-Blame; $M_{\text{binge}} = 8.22 \pm 2.93$, $M_{\text{non-binge}} = 7.50 \pm 2.92$), or putting into perspective (CERQ Perspective; $M_{\text{binge}} = 12.12 \pm 3.89$, $M_{\text{non-binge}} = 13.50 \pm 3.90$) as means of emotion regulation. Finally, contrary to previous research, BMI only demonstrated a significant effect for negative urgency (UPPS) and Positive Refocusing (CERQ).

Exploratory Analyses Results

A Pearson’s correlation matrix revealed no significant associations between indicators of executive functioning, and behavioral impulsivity, emotion regulation, and/or distress tolerance (see Table 5).

Table 5.

Correlations among Indicators of Executive Functioning, and Emotion Regulation, Behavioral Impulsivity and Distress Tolerance

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1 CPT-II Commissions																			
2 CPT-II Reaction	*-0.57																		
3 WCST Total Errors	0.12	-0.10																	
4 WCST Perseverative	-0.19	0.08	*-0.81																
5 CERQ Self-Blame	0.01	0.03	0.07	-0.08															
6 CERQ Acceptance	0.10	-0.12	0.07	-0.05	*0.40														
7 CERQ Rumination	0.10	-0.06	0.09	-0.11	*0.66	*0.48													
8 CERQ PosRefocus	0.04	-0.09	-0.04	0.19	-0.01	0.26	-0.09												
9 CERQ Planning	-0.04	-0.07	0.00	0.11	-0.02	*0.33	0.00	*0.67											
10 CERQ Reappraisal	-0.04	-0.01	-0.03	-0.01	-0.12	*0.34	-0.06	*0.61	*0.81										
11 CERQ Perspective	-0.05	0.02	0.02	0.02	0.00	0.23	-0.13	*0.50	*0.66	*0.69									
12 CERQ Catastrophize	0.20	-0.13	0.06	-0.13	*0.44	0.19	*0.54	-0.22	0.28	*-0.34	*-0.43								
13 CERQ OtherBlame	0.18	-0.20	-0.01	-0.20	0.14	0.23	*0.29	0.01	0.02	0.01	-0.04	*0.44							
14 UPPS Premeditation	0.04	0.00	-0.15	0.14	0.02	-0.10	-0.11	-0.15	*-0.30	-0.21	0.25	-0.01	-0.15						

15	UPPS Urgency	0.24	-0.08	0.00	-0.02	*0.45	0.07	*0.43	-0.22	*-0.38	*-0.36	*-0.28	*0.53	0.17	0.17					
16	UPPS SenSeeking	-0.15	0.05	-0.13	0.17	0.01	0.08	-0.16	0.16	0.18	0.27	0.25	-0.11	-0.06	0.17	-0.09				
17	UPPS Perseverance	0.17	-0.10	-0.04	0.06	*0.292	-0.03	0.26	-0.28	*-0.44	*-0.49	*-0.31	0.26	0.04	*0.47	*0.29	0.17			
18	DTS Tolerance	-0.11	0.04	0.02	-0.02	*-0.45	-0.09	*-0.57	0.16	0.29	*0.34	*0.29	*-0.53	-0.15	-0.02	*-0.55	0.22	*-0.36		
19	DTS Absorption	-0.13	0.05	-0.05	0.03	*-0.54	-0.14	*-0.67	0.24	*0.32	*0.40	0.27	*-0.58	-0.20	0.00	*-0.65	0.23	*-0.48	*0.83	
20	DTS Regulation	-0.05	0.13	-0.01	0.00	*-0.31	-0.13	*-0.38	0.02	0.00	0.09	0.09	*-0.42	-0.22	0.00	*-0.39	-0.10	-0.18	*0.55	*0.53

Note. * $p < .0025$; CPT-II = Conner's Continuous Performance Task (2nd edition); WCST = Wisconsin Card Sorting Task; CERQ = Cognitive Emotion Regulation Questionnaire ; UPPS = UPPS Impulsive Behaviour Scale; DTS = Distress Tolerance Scale

Discussion

The current study extends previous research in a number of ways. In particular, it is one of few to investigate the neuropsychology of binge eating (Davis et al., 2010; Duchesne et al., 2010; Galioto et al., 2011; Svaldi et al., 2009). Although some preliminary findings suggest that impairments in executive functioning play a role in binge eating (Duchesne et al.; Svaldi et al.), outcomes are mixed (Davis et al.; Galioto et al.) and are only generalizable to clinical samples meeting diagnostic criteria for BED. Rates of BED are extremely low (American Psychiatric Association, 2000), while percentages of individuals experiencing subclinical forms of these disturbances are much higher (J. G. Johnson et al., 2002; Saules et al., 2009). Thus, utilizing a sample with subthreshold symptoms greatly enhances the generalizability of outcomes. In addition, previous research (Davis et al.; Duchesne et al.; Galioto et al.; Svaldi et al.) investigating executive functioning among individuals who binge eat has not controlled for the influence of depression or BMI (Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007). Controlling for these variables in the current study provides a clearer picture of the association between executive functioning and binge eating.

The current study is also the first to examine an array of behavioral, cognitive and emotional processes associated with binge eating. In response to increased awareness of the negative physical and psychosocial correlates of this disordered eating behavior, research exploring the efficacy of treatment options has increased substantially in recent years. Nonetheless, little is known about how affected individuals attempt to deal with the emotions related to these binge episodes. The current study provides important insight regarding these processes, thereby making new contributions to the development of relevant interventions. The following sections briefly review the current study's findings in the context of previous research.

The clinical implications of these outcomes, as well as the study's strengths, limitations and suggestions for future research follow.

Executive Functioning Outcomes

Results from the current study suggest that, compared to their non-bingeing peers, individuals who engage in regular binge eating in the absence of compensatory behaviors do not differ with respect to their executive functioning, specifically in their cognitive impulsivity, shift and maintain cognitive set, or think flexibly. Moreover, both groups' mean scores were within the normal range. These findings are inconsistent with some research investigating the executive functioning of clinical populations with BED (Duchesne et al., 2010; Svaldi et al., 2009), although consistent with others (Davis et al., 2010; Galioto et al., 2011). A number of factors, most notably significant variations in sample characteristics, render it difficult to compare the results of the current study to that of previous investigations examining similar constructs. These differences might, at least partially, account for discrepant findings across studies.

Previous studies (Davis et al., 2010; Duchesne et al., 2010; Galioto et al., 2011; Svaldi et al., 2009) only included obese individuals with BED in their evaluation of binge eating and executive functioning. Indeed, Svaldi et al. excluded individuals with a BMI below 25. Considering the inverse association between obesity and executive functioning (Cserjési et al., 2009; Gunstad et al., 2007; Yanovski, 2003), individuals in these studies may have had poorer executive functioning outcomes than those engaging in binge eating in the current study, whose average BMI was below 25 (Davis et al.). Moreover, some studies retained individuals with concurrent mood disorders (Duchesne et al.; Svaldi et al.), although the impact of depression or anxiety on participants' neuropsychological outcomes was not controlled. As depressive symptoms are typically higher among those who binge eat (W. G. Johnson et al., 2002; Ross &

Ivis, 1999), as was found in the current study, the variance in outcomes associated with depression or other comorbid disorders might account for other studies' significant outcomes.

Moreover, the current study evaluated a young, college-enrolled sample. The average participant in this study was 19 years old. In contrast, previous studies evaluated older adults with an average educational attainment of less than 12 years (Davis et al., 2010; Duchesne et al., 2010; Galioto et al., 2011; Svaldi et al., 2009). Research indicates that executive functions develop steadily across childhood and adolescence and peak in young adulthood (Romine & Reynolds, 2005). Executive functioning is also correlated with general intellectual functioning (Strauss et al., 2006). This might explain why outcomes differed in the current study. Similarly, although scoring procedures for measures used in this study yield age- and/or education-normed T-scores, other studies utilized raw scores as outcomes (Duchesne et al.; Svaldi et al.). Consequently, comparisons regarding mean scores were not possible, nor would they necessarily be meaningful considering the samples' variations in age and education.

Another interesting age-related consideration is the potential influence of binge eating on structural or functional brain abnormalities associated with binge eating (Schafer et al., 2010). If these abnormalities are the consequences of regularly engaging in binge eating, it might not be until mid-to-late adulthood that such structural and functional changes translate to significant differences in one's neuropsychological functioning. In any case, it is important to note that, despite finding significant between-group differences in outcomes, both groups' mean scores in Svaldi et al.'s (2009) study were in the normal range, as was the case in the current study. It is thus unclear whether these group differences have practical implications.

An alternative explanation for the lack of significant between-group findings in the current study involves the nature of the binge eating episodes themselves. Previous research

distinguishes between two subtypes of binge eating, one that occurs solely in the context of dietary restriction and another that occurs in the context of both dietary restriction and negative affect (Carrard, Crepin, Ceschi, Golay, & Van der Linden, 2011; Masheb & Grilo, 2008). Individuals who endorse the dietary-negative affect subtype of binge eating also report more severe disordered eating and general psychopathology, as well as greater negative urgency and sensitivity to punishment (Carrard et al.; Stice et al., 2001). Based on this research, neuropsychological differences may be more evident among a binge eating group comprised exclusively of the dietary-negative affect subtype. Similarly, the current study identified a moderate and positive correlation between binge eating and Perseverate Responses, suggesting that more frequent binge eating is associated with greater difficulties thinking flexibly or shifting cognitive sets. Thus, executive dysfunction might only be evident among a subset of individuals endorsing particularly high amounts of binge eating behavior (although no other indicator of executive functioning was significantly correlated with total binge episodes). Indeed, binge eating behavior within the previous 28 days in the current sample ranged from weekly to more than once per day.

Further, although this study's sample was not extremely large, it is unlikely that the absence of significant differences in neuropsychological functioning is attributable to Type II error. Between-group differences in executive functioning were identified in two previous studies (Duchesne et al., 2010; Svaldi et al., 2009) with sample sizes of 78 and 35, respectively. Moreover, although these previous studies examined clinical samples with BED, self-reported binge eating frequency was comparable to that found in the current study. Nonetheless, it is important to consider how the current study's sample may have influenced results. Some research has suggested that the WCST is only sensitive to executive dysfunction among

individuals with severe neurological impairments, while others have found that the frontal lobe is activated when healthy individuals complete the WCST (see Nyhus & Barcelo, 2009 for a review). Nonetheless, tests of executive functioning require individuals to integrate and mobilize a number of cognitive skills, such as attention and memory, to name a few (Lezak et al., 2004). Thus, it is possible that the current sample's restricted range in general intellectual functioning impeded the detection of variations in executive dysfunction.

A final consideration is that differences in executive functions may only be evident in situations in which these functions are taxed, such as when experiencing negative affect or when confronted with food- or body-related stimuli. In support of this idea, participants in the binge eating group only reported higher behavioral impulsivity when experiencing negative affect. Moreover, attentional biases for emotionally-laden or food- or body-related stimuli may detract from one's overall cognitive resource pool. Thus, assessments of executive functions when individuals are distressed or placed in front of palatable foods, for example, might look very different than in the current, more sterile research setting. Indeed, individuals with BED demonstrate attentional biases for high-calorie food and negative weight- and shaped-related stimuli (Mobbs, Iglesias, Golay, & Van der Linden, 2011; Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010). Similarly, several studies evaluating structural and functional brain processes associated with binge eating were conducted in the context of eating or in the presence of food (Bathgate et al., 2001; Schienle et al., 2009).

Generally speaking, executive functioning is a notoriously complex and nebulous neuropsychological construct (Alvarez & Emory, 2006; Jurado & Rosselli, 2007). As Jurado and Rosselli note, there is consistent disagreement regarding the structure, definition, and assessment of these higher order cognitive functions, and different tests often evaluate diverse aspects of

executive functions. Indeed, correlations among neuropsychological measures of executive functioning are moderate at best (Lezak et al., 2004), and non-significant in the current study. Thus, findings from the current study may simply highlight a lack of significant group differences in executive functioning as captured by the WCST (Heaton & PAR Staff, 2005) and the CPT-II (Conners & MHS Staff, 2000) in this sample. However, as reviewed here, outcomes from animal models, neuroimaging studies, cross-sectional self-report and neuropsychological data, and research with individuals with brain injury support the notion that individuals who binge eat also manifest difficulties with higher order executive functions. In one example (Woolley et al., 2007), neuroimaging identified a link between atrophy in the right rostral orbito frontal cortex and binge eating. In the same study, executive functioning, measured via neuropsychological tests, did not differ between those engaging in binge eating and a normal, healthy sample. Thus, findings from Woolley et al. and the current study support the notion that neuroimaging and neuropsychological assessments lack convergence regarding their assessment and identification of cognitive and/or functional disabilities and should be considered different albeit complimentary sources of objective data (Nyhus & Barcelo, 2009). A similar factor to consider when interpreting the current study's outcomes is the questionable ecological validity of executive function tests (Jurado & Rosselli, 2007). It is unclear to what extent performance on measures of executive functioning predict an individual's day-to-day functioning (Chan, Shum, Touloupoulou, & Chen, 2008). As will be reviewed shortly, the current study's self-report data support the notion that significant depression, as well as difficulties tolerating and coping with distress, exist in the absence of neuropsychological impairments.

Depression Outcomes

Consistent with previous research (W. G. Johnson et al., 2002; Ross & Ivis, 1999; Spoor et al., 2006), individuals who reported engaging in regular binge eating behavior reported significantly higher depressive symptoms than their non-bingeing peers after controlling for BMI. In fact, participants in the binge eating group endorsed an average depression score equivalent to the clinical cutoff level recommended for undergraduate students (Price et al., 2006; Santor et al., 1995). Thus, the average undergraduate woman engaging in regular binge eating in the absence of regular compensatory behaviors is also at risk for clinical levels of depression.

Behavioral Impulsivity Outcomes

Also consistent with previous research (Fischer et al., 2004, 2008), the strongest statistical association in the current study was found between binge eating and negative urgency, or “the tendency to engage in impulsive behaviors under conditions of negative affect...despite the potentially harmful longer-term consequences” (p.561; S. P. Whiteside et al., 2005). This finding lends additional support to the theory that binge eating serves as a maladaptive emotion regulation technique. Findings from both self-report and neuroimaging studies suggest that individuals who engage in regular binge eating also demonstrate heightened sensitivity to reward and punishment (Avena & Bocarsly, 2011; Carrard et al., 2011; Schafer et al., 2010; Schienle et al., 2009), which is hypothesized to translate to greater reactivity to distressing emotions. Individuals who experience difficult emotions and negative urgency more intensely may be more prone to binge eating as a means of coping with their feelings despite the potential physical (e.g., stomach discomfort, weight gain) and emotional (e.g., guilt, shame) consequences. In these instances, binge eating serves as an emotional avoidance tool of sorts, and reinforces both the

beliefs that these individuals are incapable of tolerating difficult affective states and the binge eating behavior itself.

In addition to negative urgency, participants in the binge eating group also reported greater lack of perseverance, indicating difficulties avoiding distracting stimuli in order to stay focused on tasks. This finding may be due in part to the clinically significant levels of depression in the current study's binge eating sample. Some depressive symptoms, including low energy, difficulties concentrating, and diminished interest (American Psychiatric Association, 2000) certainly contribute to difficulties staying on task. Similarly, as will be seen shortly, individuals who binge eat, and individuals with depressive symptoms (e.g., Nolen-Hoeksema, Wisco, & Lyubomirsk, 2008), are more likely to engage in emotion regulation strategies such as rumination and catastrophizing. These effortful cognitive coping processes inevitably detract from one's general cognitive resource pool, making it more difficult to attend to other stimuli. Although not evaluated in the current study, this finding may also be due in part to the frequent co-occurrence of binge eating and ADHD (Cortese et al., 2007).

Contrary to the current study's hypotheses, groups did not differ with respect to their lack of premeditation or sensation seeking behavior. Lack of premeditation refers to difficulties considering the consequences of one's behavior before acting. Sensation seeking includes a desire to engage in activities that are exciting and potentially dangerous. These subscales differ from negative urgency in that they do not refer to behavioral tendencies in response to difficult emotions. Thus, findings indicate that individuals who binge eat are not necessarily more impulsive in general, but are more likely to engage in impulsive behaviors when attempting to alleviate intense and uncomfortable feelings.

Distress Tolerance Outcomes

Findings regarding the DTS further support the belief that individuals who binge eat have difficulties tolerating their distressing feelings. These individuals are more likely to describe their distress as unbearable and to report feeling shameful about its presence, as well as their inability to manage it. Those engaging in regular binge eating may also have a tendency to avoid negative affect and, when it ensues, prefer methods to quickly alleviate their distress over more thoughtful approaches. They also reported feeling as though they put more effort and attention towards regulating their emotions. Individuals in the binge eating group thus have some insight regarding their difficulties managing their emotions and indicated that their efforts to do so are burdensome. These outcomes are similar to other studies in which undergraduate students who binge ate more frequently also reported greater difficulties knowing how to manage negative affect (U. Whiteside et al., 2007) and identified feeling “helpless” as the most distressing emotion (Kenardy et al., 1996). As a possible extension of feeling unsure about how to deal with their emotions, findings from the current study suggest that binge eating was positively associated with the use of maladaptive emotion regulation strategies.

Emotion Regulation Outcomes

Specifically, when faced with distress, the binge eating group was more likely to report using self-blame, rumination, and catastrophizing to mitigate their emotions. Relative to other emotion regulation efforts, these coping strategies demonstrated the strongest association with binge eating. This is consistent with a meta-analysis examining the link between emotion regulation and psychopathology (Aldao et al., 2010). One previous longitudinal study also identified a link between binge eating and rumination (Nolen-Hoeksema et al., 2007). Specifically, Nolen-Hoeksema et al. found that rumination, or the tendency to dwell on the potential causes and consequences of one’s distressing symptoms, led to the onset of binge

eating behavior among adolescent girls. These authors also found that rumination predicted the onset of clinically significant depressive symptoms, which in turn, predicted increases in ruminative coping. It was proposed that children who did not learn adaptive coping skills were more likely to focus on their symptoms and develop a sense of helplessness in managing them. These tendencies, in turn, reinforce the distress and may lead to the use of maladaptive behaviors to help avoid this distressing introspection. Although self-blame and catastrophizing have not previously been examined in the context of binge eating behaviors, they could serve the same functions as rumination in regards to promoting and maintaining binge eating (e.g., “What’s wrong with me? Why can’t I stop binge eating?” or “I’ll never be able to stop bingeing”).

In contrast, positive reappraisal, or assigning meaning to a negative event for the purposes of self-growth, has demonstrated the strongest inverse association with general psychopathology (Aldao et al., 2010; Garnefski & Kraaij, 2007). Findings from the current study extend this link to binge eating behavior. Participants were also less likely to focus on pleasant thoughts rather than the stressful experience, or to attempt to develop a plan to deal with the distressing experience. According to a recent review, engaging in problem-solving in the face of distress has demonstrated one of the strongest inverse associations with psychopathology (Aldao et al.). Thus, it could prevent the onset of binge eating if individuals learned to think ahead of time about how to manage difficult stimuli (e.g., palatable foods, negative affect). However, considering the non-experimental nature of this study, it remains unclear whether these coping strategies directly influence binge eating or represent general emotion dysregulation.

There were several emotion regulation outcomes for which the binge eating groups did not differ significantly. Specifically, groups did not differ in regards to their use of acceptance (i.e., resigning to the occurrence of an experience), blaming others, or putting into perspective.

The lack of significant between-group differences in regards to acceptance is surprising. Acceptance is proposed to be the adaptive alternative to avoidance (Hayes, Strosahl, & Wilson, 1999), which has been associated with disordered eating (Aldao et al., 2010; Engler et al., 2006). However, research on whether acceptance is an adaptive means of coping with distress is mixed. In their meta-analysis, Aldao et al. did not identify a significant link between acceptance and general psychopathology.

Clinical Implications

There is much to glean from the current study's findings regarding suggestions for the prevention and treatment of binge eating, as well as their common comorbidities. To date, studies have examined only a limited number of coping mechanisms in the context of binge eating (Engler et al., 2006; Nolen-Hoeksema et al., 2007), and have been inconsistent in their assessment of this disordered eating behavior (e.g., using single item measures, only including objective binges). Studies examining behavioral impulsivity and binge eating are also limited, both in their frequency and their assessment of impulsivity as a multi-faceted behavioral construct. Thus, despite the lack of significant group differences in executive functioning, the current study provides some direction regarding potential emotional, cognitive, and behavioral processes to target in clinical programs for binge eating among women.

Overall, outcomes support the notion that binge eating behavior may originate from and/or be perpetuated by some combination of difficulties tolerating distress, a desire to mitigate distress quickly, the perception of a lack of skills to manage these feelings adequately, and over-reliance on maladaptive emotion regulation strategies. Thus, therapeutic interventions focused on reducing binge eating behavior should help individuals learn to better identify and tolerate difficult affective states, to increase their self-efficacy for coping, and to utilize more adaptive

ways of coping. It also seems important that such interventions address multiple types of coping, including emotional, cognitive, and behavioral. Dialectical (DBT; Linehan, 1993) and cognitive behavior therapies (CBT), currently recognized as the most effective treatment options for binge eating among adults (American Psychiatric Association, 2006; National Institute for Health and Clinical Excellence, 2004; Wilson, Grilo, & Vitousek, 2007), attend to many of these factors. Results of the current study could also be used to fine tune existing DBT and CBT interventions for binge eating, as well as alternative interventions which include variations of these approaches' strategies, including Acceptance and Commitment Therapy (ACT; Hayes et al., 1999) and Mindfulness-Based Eating Awareness Training, both of which show promise in treating binge eating (Kristeller & Wolever, 2011; Lillis, Hayes, & Levin, 2011).

Findings from the current study also reinforce the importance of addressing negative urgency and distress tolerance in the treatment of binge eating behaviors. Thus, DBT's distress tolerance skills (Linehan, 1993), such as relaxation and positive imagery, should be components of any binge eating treatment. A specific suggestion to address negative urgency was provided by Carrard et al. (2011). To help individuals find new ways of responding to negative emotions or powerful urges, they recommend that current treatment options include "if-then" interventions. Specifically, individuals establish a new link between any emotional cue and an action plan (e.g., "If situation X arises, then I will do Y") that differs from binge eating. As this link is strengthened, this new, more adaptive action plan becomes easier to access and subsequently implement in the face of distress. It is also important for clinicians and clinical researchers alike to be aware of how processes such as difficulties tolerating distress and negative urgency may influence treatment adherence. Indeed, distress tolerance and negative urgency predict treatment dropout among several patient populations, including those with EDs

(Daughters et al., 2005; Fassino et al., 2009). As is suggested in DBT (Linehan), this likelihood should be discussed explicitly with patients, and individualized action plans could be developed should these desires emerge in treatment.

Another important consideration for binge eating treatment is the finding that individuals in the binge eating group perceived themselves as expending significantly more energy to manage their emotions relative to their non-bingeing peers. Simultaneously, it is important to recognize that, despite its potentially negative consequences, binge eating functions as an efficient emotion regulation technique. Motivational interviewing (MI) strategies (W. R. Miller & Rollnick, 2002) could be used to acknowledge the potential ambivalence related to behavior change, and to help patients explore the pros and cons of replacing their binge eating behavior with other, more adaptive behaviors, and to become more aware of the extent to which this behavior influences their lives. It is also a present-focused therapeutic approach (W. R. Miller & Rollnick), and may thus reinforce efforts to avoid cognitive coping that dwells on the past and/or future, such as catastrophizing and rumination. Preliminary research on the efficacy of motivational interviewing to reduce binge eating is promising (Cassin, von Ranson, Heng, Brar, & Wojtowicz, 2008), although additional research is needed.

It is further recommended that individual or group-based interventions targeting binge eating behavior assist patients with identifying their own existing (but perhaps latent) adaptive coping techniques, as well as new ones. Findings from the current study suggest that individuals would benefit specifically from learning to minimize their tendency to ruminate, self-blame and catastrophize, all of which may be addressed via CBT methods, such as thought-stopping, distancing, and distraction (Beck, 1995). These maladaptive cognitive coping strategies should be a priority in treatment as they were most strongly associated with binge eating in the current

study. Learning to problem-solve in the face of distress, and to focus on the positive aspects of difficult events may also be helpful.

To enhance the efficiency and efficacy of clinical options for binge eating, measures from the current study could be administered to treatment-seeking individuals to identify domains of distress tolerance, behavioral impulsivity and emotion regulation to focus on in treatment. However, it is important to note that, individuals' perceptions regarding their ability to tolerate and regulate their emotions could be greatly influenced by the presence of negative affect (Aldao et al., 2010), which is more common among binge eaters (Barker et al., 2006; W. G. Johnson et al., 2002; Ross & Ivis, 1999; Spoor et al., 2006). Similarly, considering the more frequent use of rumination and catastrophizing among the binge eating group, their retrospective recall of their ability to tolerate and cope with distress may be biased, thereby underestimating actual coping abilities. Thus, treatment may also help patients identify the coping behaviors they may have but are either unaware of or unable to access during times of distress.

Outcomes from the current study could also inform programs focused on disordered eating prevention. This study included undergraduate students who are already binge eating and having difficulties tolerating and managing their emotions. Thus, binge eating prevention efforts may want to focus on helping younger children better regulate their emotions and impulsive behaviors. Such interventions would likely have significant public healthy utility as they may also prevent the onset of other disorders characterized by pathological features similar to binge eating (e.g., BN, substance abuse, pathological gambling). Similarly, in their ED outreach efforts, colleges and universities tend to focus almost exclusively on AN and BN. Considering the pervasiveness of binge eating among this undergraduate sample, and the potential presence of

significant comorbid depression, staff should also educate students about available treatments options for binge eating and related symptomatology.

Limitations, Strengths and Future Studies

Although this study addresses a significant gap in the binge eating literature, several limitations must be noted. First, most data were self-report, which can be limited by response biases. However, the EDE-Q, as discussed previously, is highly correlated with the interview format of the EDE (Fairburn & Beglin, 1994) and offers assessment of ED characteristics in a less intrusive format which has the potential to elicit more candid responses. Self-report measures also have the advantage of being cost and time efficient. As noted previously, self-report biases may be more relevant to the CERQ (Garnefski et al., 2002), and this could be an alternative explanation for some of the non-significant results.

A second concern with the current study's design is its use of undergraduate women and the subsequent limitations on the outcomes' generalizability. However, rates of binge eating are high among undergraduate samples (Saules et al., 2009). Thus, this is a particularly appropriate population in which to investigate binge eating behaviors. Moreover, the sample was racially/ethnically diverse. Nonetheless, future research should replicate these findings with more diverse samples, including those with a broader range of age and educational attainment, men, and clinical samples. Changes in behavioral impulsivity, negative urgency and cognitive emotion regulation should also be examined in the context of binge eating treatment. For example, focusing on certain clusters of emotional coping strategies may maximize treatment outcomes. Ecological momentary assessment methods in clinical research would further enhance understanding of which emotion regulation strategies precede and prevent binge eating onset.

Another limitation of the current study is its design. As noted previously, using a cross-sectional design does not provide information about the temporal associations among the study's variables. Longitudinal designs beginning in early childhood would greatly assist with clarifying these relations. Experimental designs could also be used to evaluate individual's cognitive, emotional, and behavioral reactions, including efforts to cope, eating behavior, and behavioral indicators of executive functioning such as decision-making, to the introduction of negative affect or palatable foods. Moreover, negative urgency, distress tolerance, and emotion regulation difficulties are theoretically and statistically similar constructs; indeed, several subscales from the current study's measures were highly correlated with one another. It will be important that future research disentangle these variables to see if and how they uniquely mediate the relationship between negative affect and binge eating.

It is also important to acknowledge the limitations associated with the assessment of executive functioning in the current sample. As noted previously, executive functioning is a complex neuropsychological construct that engages several cognitive processes and is assessed via a number of different tests. Thus, the current study paints a limited picture of the executive functions of those engaging in regular binge eating as it included only two indicators of cognitive impulsivity, rigidity, and shifting and maintaining set. Moreover, it included a relatively high functioning and educated sample, which contributed to a limited range of general intellectual and executive functioning. Nonetheless, it is not suggested that researchers abandon the investigation of neuropsychological functioning in the context of binge eating. Instead, it is recommended that researchers standardize assessment and scoring procedures to enhance the feasibility of comparisons across studies.

Rather than controlling for age and educational differences, for example, it is recommended that researchers calculate T-scores if available. As noted previously, recent research has found that education differed among obese individuals with BED and normal weight controls (Galioto et al., 2011). Other psychological and physiological variables associated with impaired cognitive functioning, such as psychopharmaceutical use, depression, and BMI (Cserjési et al., 2009; Fossati et al., 2002; Gunstad et al., 2007), should also be statistically controlled. Finally, although it can be costly and burdensome to administer a large neuropsychological battery to research participants with some degree of psychopathology, such a process may be necessary. Based on numerous comprehensive evaluations, researchers in Norway (Stedal, Frampton, Landrø, & Lask, 2011) have identified a cognitive profile associated with AN and, based on this profile, recommended a standardized battery of neuropsychological tests called The Ravello Profile. Their aim is to promote consistent examination of the cognitive functioning of individuals with AN to facilitate cross-study comparisons and more conclusive findings regarding this ED. A similar process is recommended to better understand the cognitive processes that contribute to the onset and maintenance of binge eating behavior. Similarly, additional research is needed to clarify if there is a degree of binge eating severity at which point executive dysfunction becomes evident, and how this extreme level of disordered eating behavior and associated neuropsychological difficulties might influence treatment.

Summary

In sum, contrary to hypotheses, individuals engaging in regular binge eating did not differ from their non-binge eating peers in regards to their executive functioning. Although correlation analyses suggest that, among the binge eating group, individuals endorsing more frequent binge eating may have greater difficulties thinking flexibly or shifting attention, additional research is

needed to clarify the link between binge eating severity and neuropsychological dysfunction; indeed, other indicators of executive functioning did not correlate significantly with total binge episodes. Consistent with secondary hypotheses, individuals in the binge eating group endorsed significantly higher levels of depression, reported that they are more likely to engage in impulsive behavior (but only when distressed), have more difficulties tolerating distress, are more likely to engage in rumination, self-blame, and catastrophizing, and less likely to focus on the positive. Although this study has several limitations, as noted previously, it also makes potential contributions. The most notable strength of this study was its exploration of a broad range of behavioral, cognitive, and emotional processes, including adaptive and maladaptive coping strategies, and their association with binge eating. As a result, the current study provides greater understanding of a disordered eating behavior negatively influencing a broad range of individuals. Findings can inform the modification and subsequent improvement of current intervention and prevention programs for binge eating behavior, while also providing direction for the future examination of its neuropsychological contributors.

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Vita

Nichole Rhea Kelly was born October 24, 1981 in Tucson, Arizona. She graduated from Massaponax High School in Fredericksburg, Virginia in June of 2000. She completed her undergraduate studies in May of 2004 at the University of Virginia (UVA), in Charlottesville, Virginia with a Bachelor of Science in Psychology. During her time at UVA, she worked as a research assistant for Dr. Bethany Teachman, examining mental health stigma, anxiety/phobia, and disgust cognitions. After finishing her undergraduate studies, she remained in Charlottesville for one year working as a research assistant for Dr. Melvin Wilson and the Early Steps Project, a multi-site, randomized control-group trial, evaluating a family-centered intervention for reducing the early emergence of risk in young children, including aggressive and withdrawn behavior. In June of 2005, she moved to Boulder, Colorado where she worked with the Women's Wilderness Institute, a non-profit organization. A year later, she spent 10 months in Austin, Texas at the Austin Area Urban League (AAUL). Nichole came to Richmond, Virginia in May of 2007 to begin graduate work in the doctoral program in Counseling Psychology at Virginia Commonwealth University (VCU) under the direction of Dr. Suzanne Mazzeo. She earned her Master of Science Degree in Counseling Psychology from VCU in May of 2009, and continues to work with Dr. Mazzeo and fellow lab members on obesity- and eating disorder-related program and research design. She has specific research and clinical interests in eating disorder prevention and treatment, binge eating, emotion regulation, and distress tolerance, as well as personality and neuropsychological assessment.