UNDERSTANDING BULIMIA NERVOSA FROM A NEUROPSYCHOLOGICAL PERSPECTIVE: IMPULSIVITY AND BINGE-PURGE BEHAVIOR IN ADOLESCENT AND YOUNG ADULT WOMEN

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ABSTRACT
Title: Understanding bulimia nervosa from a neuropsychological perspective: Impulsivity and binge-purge behavior in adolescent and young adult women
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According to the biopsychosocial model of bulimia, neurobiological mechanisms called endophenotypes cause eating disordered behavior. Impulsivity has been identified as a possible endophenotype for bulimia nervosa, and individuals with bulimia who present with multiple forms of impulsive behavior are known to have worse prognoses. Executive dysfunction in impulse control purportedly manifests as behavioral under-regulation in binge-purge episodes. Neuropsychological assessments were used to analyze the relationship between impulsivity and symptoms of bulimia. Twenty-eight inpatient adolescent and young adult women with bulimia completed the D-KEFS Color Word Task, which is a version of the Stroop that contains four trials including the classic Stroop and a switching Stroop, as well as the age appropriate versions of the BRIEF rating scale and a Type-T Survey of thrill-seeking. Performance on these measures was correlated with measures of bulimia symptoms, including the EDI-3, EDE-Q, and variables of illness severity. Delay of gratification was assessed by offering subjects a choice of compensation that was either immediate and smaller or delayed and larger. Mixed results were found. The sample did not differ from the D-KEFS normative sample on total number of errors or on speed of task completion for the switching Stroop, and the sample demonstrated faster performance than the normative sample on the classic Stroop. However, a tendency to favor speed over accuracy of performance was identified. On the BRIEF rating scales, the sample self-

reported significantly higher rates of executive dysfunction compared to the normative data. Additionally, some variables of impulsivity, including greater frequency of errors on cognitive tasks and self-reported deficits of executive functioning, were significantly correlated with variables of bulimia symptom severity, including self-reported bulimia symptomatology on the EDI-3 and frequency of bingeing and purgeing. Risk-taking was also found to be correlated with symptoms of bulimia. Differences were found between subjects who chose the immediate prize versus those who chose the delayed prize, including differences in cognitive task performance and symptom severity. Differences were also found for subjects with a comorbid disorder of impulse control, including bipolar disorders and substance abuse. In conclusion, a unilateral deficit of impulse control was not found to be characteristic of this sample; however, a multi-impulsive cohort was identified as having deficits of cognitive impulse control.

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DEDICATIONS

This work is dedicated to my grandmother, Ruth Baird Thompson, who has been a role model for all that women can achieve.

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CHAPTER 1

INTRODUCTION

The study of eating disorders has a long and rich history, including the investigation of risk factors (Strober & Humphrey, 1987), clinical characteristics (Dubois, 1949; Russell, 1979), and treatment methods and outcomes (Bruch, 1982). Increasingly, the involvement of neurobiological mechanisms in the pathophysiology of eating disorders is being recognized (Collier & Treasure, 2004; Chavez & Insel, 2007; Kaye et al., 2004; Ward, Tiller, Treasure, & Russell, 2000). The neuropsychological framework emphasizing brain-behavior relationships has promulgated a growing trend in the field to link eating disorder phenotypes, including behaviors and traits, with putative biological mechanisms.

The relationship between bulimia and impulsivity is well described in the literature (e.g., Grilo, 2001; Kane, Loxton, Staiger, & Dawe, 2004). According to the neuropsychological framework guiding the present study, it is hypothesized that the behavioral dysregulation seen in people with eating disorders can be attributed, in part, to a disruption of the executive functions of the frontal lobes that regulate impulse control. The present research study explores the relationship between eating disordered behavior and impulsivity in adolescent and young adult females with bulimia nervosa. Understanding the cognitive processing differences in individuals with eating disorders will provide insight into their precipitating and perpetuating factors, and can also inform treatment.

Much is known about bulimia; however, most conceptualizations have focused on interpersonal dynamics (e.g., Vandereycken, Kog, & Vanderlinden, 1989), body image dissatisfaction (e.g., Cash & Pruzinsky, 2002; Thompson, Heinberg, Altabe, & Tantleff-Dunn,

1999), or the influence of socio-cultural context (e.g., Smolak & Striegel-Moore, 2001; Stice, 2001). Current etiological models of eating disorders stress the interaction of biological and environmental risk factors (Striegel-Moore & Bulik, 2007). In an age of increasingly sophisticated research methodology and diagnostic technology, the potential to identify biological mechanisms as causal factors is clearly evident (Kaye, Bailer, Frank, Wagner, & Henry, 2005).

The urgency to facilitate research in the area of eating disordered behavior is paramount given the current obesity epidemic affecting the global society (Thompson, 2004). Treatments for eating disorders and obesity are costly, lengthy, and involved. In 2007, the National Institute of Mental Health stressed the need for research to take advantage of advances in genetic mapping, biochemical analyses, and neuroimaging in order to advance explanatory models of eating disorders beyond behavioral descriptions so that treatment and prevention efforts might be more effectively delivered. According to NIMH, variables should be investigated that are amenable to change and that predate the onset of the illness in order to foster prevention and intervention methods to circumvent the onset of a clinical disorder.

The advent of endophenotype research in the study of complex psychiatric diseases has further enabled exploration of gene-environment interactions in the onset of eating disorders. As described by Gottesman and Gould (2003), endophenotypes are measurable components of a disease that are connected to a distal genotype, and can include neurophysiological, biochemical, endocrinological, neuroanatomical, cognitive, or neuropsychological factors. Possible endophenotypic markers for bulimia have been proposed, including abnormalities of serotonin (Steiger & Bruce, 2007) and deficits of attention and executive functioning (Treasure, 2006), though further research and clarification is needed (Bulik et al., 2007).

Eating disorders have the highest mortality rate of all psychiatric disorders and are associated with extensive physiological damage (Sullivan, 1995). The age of onset of these deadly disorders is typically during the adolescent and young adult years. Research in developmental psychopathology emphasizes that adolescence is a critical period for the development of eating disorders because this is a time of immense transition, particularly involving physical and social changes (Smolak, Levine, & Striegel-Moore, 1996). Research on eating disorder prevention has shown that targeted interventions aimed at the adolescent population are most effective (e.g., Levine & Smolak, 2001; Stice & Hoffman, 2004). Thus, further research with the adolescent population is critically necessary to support these targeted treatment and prevention efforts.

There is considerable overlap of symptoms and comorbid psychopathology across eating disorder subtypes (Claes et al., 2006; Finzi-Dottan & Zubery, 2009), and some have called for a reconsideration of diagnostic nosology according to endophenotypes (Treasure, 2006). For example, the symptoms of individuals with eating disorders can be conceptualized as either an over- or under-control of behavior (Steiger, 2004). Anorexia nervosa is characterized by an extreme restriction of food intake involving deliberate control over the biological impulse to eat, consistent with an over-regulation of behavior. Steinglass, Walsh, and Sterm (2006) developed a model of anorexia akin to the neurobiology of obsessive-compulsive disorder, in which perseverative eating behaviors are mediated by structural and metabolic abnormalities in corticostriatal brain circuits. The conceptualization of anorexia as a disorder of behavioral over-control implies that perseverative non-eating is similar to the anxious-avoidant behaviors seen in obsessive-compulsive disorder (Lavender, Shubert, de Silva, & Treasure, 2006; Murphy, Nutzinger, Paul, & Leplow, 2002, 2004).

Conversely, the binge-purge behavior exhibited by individuals with bulimia can be understood as an under-regulation of behavior. These individuals act on the impulse to consume and cannot resist the subsequent impulse to purge. Reports of comorbid impulse control disorders and acts of behavioral impulsivity, including self-harm, substance abuse, suicide attempts, and shoplifting, are frequently seen in samples of patients with bulimia (Grilo, 2001; Treasure, 2006), leading to the description of a multi-impulsive syndrome (Lacey & Evans, 1986). Individuals with bulimia who present with multi-impulsivity appear to reflect a distinct sub-sample of bulimia, characterized by more severe clinical histories and worse prognoses (Sohlberg et al., 1989). Impulsive traits have also been identified as characteristic of individuals with eating disorders who initially present with restricting anorexia and eventually develop binge-purge symptoms of bulimia (Krug et al., 2009). Accordingly, impulse control, presently defined as an aspect of executive functioning that is controlled by prefrontal brain circuitry, is highlighted as a critical variable for investigation of the causes of bulimia.

Like eating disorders, the prevalence of impulsive risk-taking behavior also peaks in prevalence during the adolescent and young adult years. According to the Substance Abuse and Mental Health Services Administration (2007), binge drinking has been found to occur in approximately 30% of adolescents, aged 16 to 20 and 46% of young adults ages 21 to 25. The Center for Disease Control and Prevention (2007) has reported that the leading causes of morbidity and mortality among adolescents are largely related to high risk behaviors, such as motor vehicle death due to not wearing a seatbelt or driving under the influence. Other notably prevalent high risk behaviors occurring in adolescence include unprotected sexual intercourse and drug use, which are associated with poor academic outcomes (CDC, n.d.).

A potential reason for such high rates of impulsive behavior during adolescence is that the executive functions, including impulse control and decision-making skills, are still developing. These higher-order cognitive skills develop in conjunction with the physical maturation of the frontal brain regions (Dawson & Guare, 2004), particularly the prefrontal cortex, which does not complete development until approximately the age range of the mid-20s (Keverne, 2004). While some risky behavior is considered normative, extreme forms of impulsive behavior are considered symptomatic of a dysfunctional executive system. Ingestion of drugs during adolescence can further disrupt normal development of the executive functions by causing neuroadaptations that promote addictive behaviors, including risk-taking and poor impulse control (Volkow & Wise, 2005).

Neuropsychological research studies have investigated the role of executive functioning in children and adults with various psychiatric disorders, such as obsessive-compulsive disorder (Duran, Ricardo-Garcell, Zamorano, & Mendoza, 2007), depression and panic disorder (Kaplan et al., 2006), attention deficit-hyperactivity disorder (Brown, 2006), schizophrenia (Elvevag & Goldberg, 2000), and borderline personality disorder (Bourke et al., 2006). A beginning line of research exists in the investigation of executive skills in eating disorders; however, these studies have largely been conducted using adult samples of women with anorexia (Southgate, Tchanturia, & Treasure, 2006). Further investigation of executive functioning in young women and adolescents with eating disorders is clearly warranted.

Southgate, Tchanturia, and Treasure (2005) proposed a biopsychosocial model of eating disorders that describes a developmental framework and guides the hypotheses of the present study. Specific neurobiological mechanisms are identified as contributing to pre-morbid cognitive deficits in adolescence, which serve as vulnerability factors in the onset and

maintenance of eating-disordered behavior. This model is supported by the convergent evidence specifying adolescence as the critical time period for (1) the age of onset for eating disorders, (2) the onset of peak rates of risky and impulsive behavior, and (3) the development of executive functioning skills in association with brain maturation.

The purpose of this dissertation research is to use neuropsychological measures of executive functioning as the empirical lens through which to investigate the relationship between two problems: eating disorders and impulsive behavior. These problems are common in adolescence and young adulthood, and coincide with the development of frontal brain systems. These issues are relevant to both research and practice in psychology. Research on the development of eating disorders might explore cognitive factors in defining models of risk. Clinicians might be able to monitor youngsters with executive dysfunction or ADHD for eating disorder symptoms (Biederman et al., 2007). Conversely, adolescents with obesity or bulimic symptoms might be screened for cognitive problems associated with poor impulse control (Cortese et al., 2007). Therapeutic strategies might be developed to address the cognitive styles of women with bulimia (Meyer et al., 2005), similar to the treatment of anorexia using the Maudsley method (Treasure, Tchanturia, & Schmidt, 2005).

Finally, while many have pointed to the connection between poor impulse control and conduct problems in males, the involvement of impulsivity in females is less well understood (Chapple & Johnson, 2007; Mikami, Patterson, Hinshaw, & Lee, 2008). Research on ADHD in females suggests that impulsivity may manifest as eating disordered behavior among young women (Quinn, 2008). Therefore, the findings of this research may be applicable to understanding outcomes for impulsive females.

CHAPTER 2

LITERATURE REVIEW

Overview

The following literature review summarizes the findings from three distinct fields of inquiry in the psychological literature: the clinical disorder of bulimia nervosa, the personality trait of impulsivity, and the executive functions of the brain. Although each of these topics has a vast body of literature, certain neurobiological commonalities germane to this study were identified, including specific brain regions, functional circuits, and neurotransmitter systems. For clarity, these common elements, including key terms and concepts, are presently introduced. While it is acknowledged that the following descriptions reflect great oversimplification, only the information relevant to this research study is presented.

First, the prefrontal cortex and limbic system were consistently identified as contributing to symptoms of bulimia, impulsivity, and executive dysfunction. The prefrontal cortex of the frontal lobes is the most anterior region of the brain. As the most recent part of the brain to evolve, the prefrontal cortex is recognized for contributing to higher order thought, including executive functioning and impulse control (Fuster, 2002). The limbic system is composed of cortical and sub-cortical structures that activate emotional and motivational functioning. Some of the key limbic structures include: amygdala, hippocampus, anterior cingulate, nucleus acumbens, and hypothalamus (Haines, 2008).

Second, abnormalities of specific frontal brain circuits were identified as common to bulimia, impulse control, and executive dysfunction. Functional circuits exist to connect different regions of the brain for complex processing. Neuroimaging research indicates that at

least five frontal circuits exist, each involving a particular frontal lobe area, specific projections to basal ganglia, continuation to the thalamus, and back to the frontal region of origin (Alexander & Stuss, 2000; Tekin & Cummings, 2002). Two of these circuits are primarily involved in the motor functions of the supplementary motor cortex and frontal eye fields. The other three frontal circuits are involved in executive functions: (1) the dorsolateral circuit; (2) the orbitofrontal circuit; and (3) the ventromedial circuit, which includes the anterior cingulate. Functionally, the dorsolateral circuits regulate specific cognitive executive functions. The orbitofrontal, ventromedial, and anterior cingulate circuits regulate affective and motivational executive functions through connections with the limbic system. Some authors do not distinguish between the orbitofrontal and ventromedial circuits (Happaney, Zelazo, & Stuss, 2004).

Finally, disturbances to the neurotransmitter systems including serotonin and dopamine were consistently implicated as contributing to bulimia, impulsivity, and executive dysfunction. Although other neurochemicals have been identified, serotonin and dopamine are the most widely researched. Essentially, serotonin is associated with inhibition of behavior (Zuckerman, 2005), including satiety (Heisler et al., 2003); therefore, deficient serotonin activity can lead to deficient inhibition of behavior, or poor impulse control, which may manifest as binge eating, risk-taking, or disinhibited responding on neuropsychological tasks. Conversely, dopamine is associated with facilitation of behavior. Individuals with chronically deficient dopamine activity, including individuals with bulimia and individuals with impulse control disorders, functionally counteract this deficiency by engaging in activities that episodically increase dopamine to very high levels, including binge eating, substance abuse, and risk-taking (Rada, Avena, & Hoebel, 2005).

In summary, the prefrontal cortex, limbic system, ventromedial-orbitofrontal-anterior cingulate circuits, and deficient serotonin and dopamine were identified in the literature as contributing to behaviors seen in bulimia, impulsivity, and executive dysfunction. These commonalities were identified through a thorough review of the literature, including animal studies, neuroimaging, psychopharmacology, comorbid psychopathology, addictions neuroscience, psychological theories, and neuropsychological testing. The common emergence of these neurobiological factors supports the hypothesis that bulimia, impulsivity, and executive dysfunction are inherently connected.

Bulimia Nervosa

Definitions and Characteristics

According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition Text Revision (2000), eating disorders are characterized by severe disturbances in eating behavior. Bulimia nervosa is diagnosed when an individual consumes abnormally large quantities of food during discrete episodes, known as binges, and employs inappropriate compensatory mechanisms to prevent weight gain, called purges. The average caloric consumption during a binge episode among inpatient women with bulimia has been reported to be 3798 calories, with an average of thirteen binges occurring per week (Guertin, 1999).

The DSM-IV-TR states that the most common compensatory technique is the induction of vomiting after a binge episode, which is reported by 80-90% of individuals with bulimia (2000). The immediate effects of vomiting are relief from physical discomfort associated with the binge, as well as the reduction of psychological discomfort associated with fear of gaining

weight and guilt over bingeing. As such, purging is associated with reward for many, which thereby reinforces the behavior (Farmer, Nash, & Field, 2001). Other compensatory techniques include use of laxatives or diuretics, ingestion of syrup of ipecac to induce vomiting, and misuse of enemas. Compulsive exercise can also be characteristic of bulimia. Some individuals with diabetes mellitus may omit or reduce insulin doses in order to reduce the metabolism of food, a syndrome termed *diabulemia* in the literature (Yan, 2007).

The age of onset for bulimia is typically late adolescence or early adulthood (DSM-IV-TR, 2000). Approximately 90% of individuals with bulimia are female. The disorder is primarily reported among Caucasian females in Western cultures; however, the disorder has also been found to affect other ethnic groups, though potentially different causal and contributory factors have been suggested (White & Grilo, 2005). The lifetime prevalence of bulimia is approximately 1-3%; however, studies have found the presence of sub-clinical bulimia in as many as an additional 3-5% (Rosenvinge, Borgen, & Borresen, 1999). Additionally, dieting is known to be a risk factor for the onset of bulimia (Polivy & Herman, 1985; Stice, 2001), and dieting behaviors are highly prevalent in the United States. For example, 70% of a community sample of adult women reported to be engaging in dieting behaviors (French, Jefferey, & Murray, 1999). Thus, low prevalence rates of clinical disorders do not accurately represent the scope of the problem.

Comorbid psychopathology is commonly seen among women with bulimia, further complicating treatment and understanding of etiology. Common comorbid Axis I disorders include substance abuse, anxiety, and mood disorders (Brewerton, Lydiard, Herzog, & Brotman, 1995). Bipolar disorder is noted to be particularly common among this population (McElroy, Kotwal, & Keck, 2006). Personality disorders have also been found to be comorbid with

bulimia, especially borderline personality disorder (Godt, 2008; van Hanswicjk de Jonge, van Furth, Lacey, & Waller, 2003). Additionally, suicidal behavior and self-harm have been reported as commonly co-occurring among individuals with bulimia (Fischer & le Grange, 2007).

Treatment options for bulimia include pharmacotherapy and psychotherapy (Klein & Walsh, 2004). Selective-serotonin reuptake inhibitors (SSRIs) have proven effective in treating bulimia in adults and some have received FDA approval for such usage (Powers & Bruty, 2009). Although little research has been conducted with adolescents, fluoxetine, an SSRI, remains the first-line medication treatment for adolescents with bulimia (Couturier & Lock, 2007). Other pharmacological treatments, such as anti-epileptics or the opioid antagonist naltrexone, may be promising, but lack clinical trials (Ramoz, Versini, & Gorwood, 2008). Cognitive behavioral therapy (CBT) has demonstrated efficacy in treating this population as well (Thompson, 2004). Other treatments such as repetitive transcranial magnetic stimulation are less widely used and lack a sufficient evidence-base (Tsai, 2005).

Bulimia nervosa is associated with many negative outcomes and physical consequences (Chowdhury & Lask, 2000; Thompson, 2004). Recurrent vomiting can lead to dental erosion. Menstrual irregularity or amenorrhea sometimes occurs as a result of weight fluctuation, nutritional deficiencies, or emotional stress. Laxative abuse can lead to bowel movement irregularities and rectal prolapse. Fluid and electrolyte disturbances resulting from purging can sometimes cause medically serious problems, including orthostatic hypotension. The loss of stomach acid through vomiting may produce metabolic alkalosis, and the frequent use of laxatives to induce diarrhea can cause metabolic acidosis. Other medical complications include esophageal tears, gastric ruptures, and cardiac arrhythmias. Additionally, bulimia has been

found to predict obesity (Stice, Cameron, Killen, Hayward, & Taylor, 1999), which in turn increases risk for morbidity and mortality (Hu et al., 2004).

Physical disturbances are not the only poor outcomes associated with bulimia. Epidemiological studies show that eating pathology predicts depression (Stice, Hayward, Cameron, Killen, & Taylor, 2000), which in turn increases risk for many negative life events, such as school dropout, unemployment, delinquency, and legal problems (Gotlib, Lewinsohn, & Seeley, 1998). Among adolescent and college-aged women, academic difficulties have been found consequent to the onset of bulimia for various reasons (Yanover & Thompson, 2008), including increased absenteeism due to psychological and/or medical complications and poor focus and attention due to pre-occupied thoughts.

Psychological Theories

Bulimic-type syndromes have been described throughout modern history, although it was not until 1979 that Gerald Russell defined bulimia nervosa as distinct from other recognized eating disorders (Cooper, 2003). Many causative models have been proposed in the psychological literature to explain bulimia. Psychodynamic theorists (e.g., Chassler, 1998; Farrell, 1995) have discussed the relationships between the self, food, and others. Social psychology and feminist theories (e.g., Bendfeldt-Zachrisson, 1992) have explored the role of women in society and the effects of gender and class inequalities. Family systems theories have been developed to explain the contribution of the family environment in causing and maintaining bulimic syndromes (e.g., Humphrey, 1989).

Body image theories have played an especially prominent role in the eating disorders literature (Cash & Pruzinsky, 2002). The well-documented “normative discontent” (Thompson,

Heinberg, Altabe, & Tantleff-Dunn, 1999), describing the largely universal body image dissatisfaction found in Western societies, has prompted population-based studies of risk and resiliency. Various risk factors for bulimia have been identified, including individual, familial, and socio-cultural influences (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Some of the most consistently identified risk factors include obesity or high levels of body mass index, body image dissatisfaction, history of abuse or trauma, and family history significant for eating disorders or psychopathology among close relatives.

Cognitive-behavioral theories have been well explicated to describe how an individual's schemata, attributions, and behavioral coping patterns contribute to the development and maintenance of eating disorders (e.g., Cooper, Wells, & Todd, 2004), particularly as a means to escape heightened awareness in which the individual focuses his or her attention on the present and immediate environment in order to avoid any unpleasant or threatening thoughts or feelings (Ainsworth, Waller, & Kennedy, 2002; Heatherton & Baumeister, 1991; Quinton, 1998). Vitousek and Hollon (1990) describe a cognitive framework for understanding eating disorders that involves assessing the content and processing of cognitive schemata. The authors emphasize that automatic processing of salient information, particularly related to self- and weight-schemata, serves a function for the individual by simplifying, organizing, and stabilizing their self- and world-purview, but also contributes to the intractability of symptoms.

Theories of normal and restrained eating (e.g., dieting) have been extended to explain the onset of symptoms of bulimia within the context of physical and psychological hunger (Herman & Polivy, 1975; Laessle, Platte, Schweiger, & Pirke, 1996). Research has shown that individuals with bulimia can be categorized as either dietary or dietary-depressive subtypes and the addition of negative affect is associated with greater functional impairment and worse prognosis (Stice &

Fairburn, 2003). The set-point theory of normal eating states that hunger is triggered by the decline of the body's energy reserves below their set-point. Restrained eating occurs when an individual attempts to maintain her body weight below its natural level. Ignoring the physiological drive to eat leaves the individual in a constant state of hunger and susceptible to over-eating in order to compensate for below set-point energy levels. Indeed, neuroscience research has shown that food deprivation potentiates the activation of the brain's neural reward system in response to food (Volkow & Wise, 2005).

The Neurobiology of Eating Behavior

While the physiological consequences of bulimia are well understood, the role of biology in the onset of the disorder remains less clear. Family and twin studies indicate that a genetic contribution exists (Bulik et al., 2000; Kaye et al., 2004), with heritability estimates reported to be as high as 83% in twin studies (Bulik, Sullivan, & Kendler, 1998). Studies of molecular genetics have identified genes associated with neurochemical variants that contribute to the expression of specific symptoms of bulimia (Bulik, 2004) as well as associated psychopathology, including drive for thinness (Nisoli et al., 2007) and impulsivity (Steiger et al., 2005).

Research on the neurobiology of bulimia is complicated by the trait-versus-state distinction (Klein & Walsh, 2004). Neuroendocrine changes have been found among individuals with bulimia, including abnormalities of feeding-related chemicals, such as neuropeptide-Y and CCK (Lydiard et al., 1993); however, such findings are often considered to be state-related consequences of the disorder (Bailer & Kaye, 2003), associated with short-term changes in weight (Jimerson, Wolfe, & Naab, 2006) and disruption of the digestive system (Monteleone et al., 2005). Reduced brain metabolism (Delvenne, Goldman, Simon, De Maertelaer, & Lotstra,

1997; Uehara et al., 2007) and gross structural changes, including brain atrophy and enlarged ventricles (Lauer, Lässle, Fichter, & Pirke, 1990), have also been cited as common state-related consequences that are typically reversible following recovery.

Although trait-related variables cannot be definitively identified without prospective research, structural and functional neurobiological irregularities have been suggested as possibly pre-morbid. Structurally, the right frontal and temporal lobes are most consistently implicated in the pathophysiology of bulimia (Eviatar, Latzer, & Vicksman, 2008; Frank, Bailere, Henry, Wagner, & Kaye, 2004; Uher & Treasure, 2005). Patients with bulimia are found to have deficient activation of prefrontal areas in response to symptom-specific stimulation (Nozoe et al., 1995; Peñas-Lledó, Koeb, Martin, & Fan, 2007; Uher et al., 2005), and the absence of activity in the prefrontal cortex is associated with lack of control when eating (Uher et al., 2004). Case studies have demonstrated that lesions to the prefrontal cortex are associated with impulsive behaviors and bulimic symptoms (Erb et al., 1989). Patients with dementia due to frontotemporal lobar degeneration often demonstrate binge eating behavior (Grossman, 2007), particularly when atrophy is found in orbitofrontal-insular-striatal regions (Woolley et al., 2007). There is some evidence to suggest that body image distortions are associated with dysfunctional perceptual-evaluative functions of the parietal lobe (Goethals et al., 2007).

Functionally, two complimentary neurobiological systems interact in contributing to eating disordered behavior (Lutter & Nestler, 2009; Saper, Chou, & Elmquist, 2002): (1) the serotonin-mediated homeostatic mechanisms of the hypothalamus (Klein & Walsh, 2004), and (2) the dopamine-regulated hedonic reward circuitry involving the prefrontal cortex and limbic system structures, such as the amygdala and nucleus acumbens (Mercer, 2007). Both systems have substantial neural connections with the frontal lobes (Erb, Gwirtsman, Fuster, & Richeimer,

1989; Uher & Treasure, 2005), indicating that the executive functions of the frontal lobes can control the feeding behavior operated by sub-cortical structures.

It is well-known that the hypothalamus plays a critical role in regulating homeostatic energy balance via food consumption. Neuropeptides such as leptin and ghrelin are active in the hypothalamus to control feeding behavior in response to peripheral body signals of energy abundance or insufficiency (Zigman & Elmquist, 2003). Serotonin acts to signal satiation in the hypothalamus, and disruption of serotonin interferes with proper neuropeptide functioning, leading to disturbances of eating behavior (Heisler et al., 2003). The literature indicates that individuals with bulimia show deficient serotonin activity (Stamatakis & Hetherington, 2003; Steiger, Israël, Gauvin, Ng Ying Kin, & Young, 2003; Tiihonen et al., 2004) particularly in the hypothalamus (Brewerton, 1995; Jimerson, Wolfe, & Naab, 2006; Tauscher et al., 2001), which causes impaired signaling of post-ingestive satiety (Jimerson, Wolfe, Metzger, & Finkelstein, 1997).

The evidence suggests that dysfunctional serotonin systems are trait-related in individuals with bulimia. Several studies have shown that alterations in serotonin functioning persist among recovered individuals with bulimia (Bailer et al., 2007; Kaye et al., 2005; Kaye et al., 1998). Recent studies of genetic markers of bulimia have demonstrated an association between extent of associated psychopathology and specific serotonin polymorphism genotypes (Ribasés et al., 2008; Steiger et al., 2005). Successful treatment of mood disturbances using SSRIs has been shown to concurrently improve bulimic symptoms, which suggests that serotonin dysregulation in bulimia does not only affect feeding behavior, but the regulation of emotion as well (Kaye et al., 2005). Some have speculated that recovery from bulimia results in return to normal

serotonin functioning within the hypothalamus, but abnormal serotonin functioning may persist elsewhere in the central nervous system (Wolfe et al., 2000).

The hedonic reward system involved in feeding behavior is characterized by dopamine-regulated pathways between primary taste centers in the insular cortex, areas of the prefrontal cortex and anterior cingulate that operate cognitive processing, and the emotional/motivational limbic system, which assigns value to the eating experience as pleasurable or rewarding (Morton et al., 2006). Using fMRI, Frank and colleagues (2006) found that individuals who had recovered from bulimia showed hypo-activation of hedonic reward circuitry when ingesting a nutritive substance compared to controls, suggesting that individuals with bulimia have a diminished reward response to food and are thus vulnerable to overeating, or bingeing, in order to activate the reward circuits.

The Neurobiology of Addictive Behavior

Bulimia and substance abuse share many behavioral characteristics, including loss of control and excessive intake. Research from the field of addictions indicates that considerable brain circuitry is shared in the development of addictions to both illicit substances and highly palatable food (Avena, 2007; Volkow & Wise, 2005). The neurological circuits shown to be activated by addictive consumption of both food and drugs include (1) the dopamine- and opioid-regulated reward circuits of the limbic system, (2) the neuroendocrine-regulated stress and affect circuits of the HPA axis, and (3) the serotonin-regulated behavior inhibition circuits of the prefrontal cortex (Goodman, 2008).

Within the reward circuitry, the nucleus accumbens plays a critical role by assigning reward value to stimuli. Both binge eating and substance abuse cause increased dopamine

activity in the nucleus accumbens, indicating that the behavior is experienced as rewarding. Opioid receptors in the nucleus accumbens have also been implicated as critical for both feeding and drug addictive behavior (Saper, Chou, & Elmquist, 2002). Binge eating causes the release of endogenous opioids, which are associated with an experience of pleasure. Aberrant opioid activity is associated with hyperphagia and preferential intake of highly palatable foods, particularly sweets (Mercer, 2007). Blocking opioid activity has been one avenue of treatment for both substance abuse and bulimia, and the opiate antagonist naltrexone has been used to successfully treat binge eating (Jonas & Gold, 1988). Animal research has shown that bingeing induced by opioid stimulation in the nucleus accumbens can be counteracted through inactivation of the amygdala, suggesting that the reward circuits offer a potential vector for pharmacotherapy (Will, Franzblau, & Kelley, 2004).

Affect regulation circuits are often discussed in terms of stress reactivity (Goodman, 2008). Stress is a precipitating factor for the onset of eating disorders; however, the mechanism of the neuroendocrine stress response in mediating eating disordered behavior is not well understood (Lo Sauro, Ravaldi, Cabras, Faravelli, & Ricca, 2008). As described in the Reward Based Stress Eating model (Adam & Epel, 2005), the hypothalamic-pituitary-adrenal (HPA) system is chronically hypo-aroused in individuals with a history of bulimia, and subsequently hyper-reactive when exposed to agents that stimulate it (Majewska, 2002). The HPA axis is directly aroused by the stress-hormone cortisol when the individual encounters psychological stress and indirectly aroused by highly palatable foods via dopaminergic reward-circuitry. When activated, the HPA axis subsequently triggers the release of endogenous opioids, which is experienced pleasurably. When engaged in a binge episode, individuals with bulimia are confronted with both highly palatable food and psychological stress. Frequent bingeing and

repeated stimulation of this system over time is proposed to cause neuroadaptive changes in dopamine and opioid receptor binding, ultimately resulting in the development of compulsive eating patterns or binges. Similar neuroadaptations have been found in substance abuse addicts (Kalivas & Volkow, 2005).

There are multiple behavioral inhibition networks in the brain, many of which are mediated by serotonergic prefrontal brain activity (Goodman, 2008). Within the prefrontal cortex, ventromedial and dorsolateral regions must collaborate to regulate behavioral inhibition. A pattern of imbalanced activity has been demonstrated on fMRI in individuals with substance addiction (Goldstein & Volkow, 2002), as well as individuals with bulimia in response to food images (Uher et al., 2004). The behavioral-monitoring functions of the dorsolateral cortex are found to be disproportionately hypo-active in relation to the behavioral-activation functions of the ventromedial cortex. This is consistent with poor impulse control seen in these conditions.

A Biopsychosocial Model of Bulimia

Theoretical and empirical advances have linked neurobiological and psychological mechanisms with the expression of eating disordered behavior. Steiger and Bruce (2007) argue that the phenotypic presentation of an eating disorder in an individual depends upon genotypic variations affecting the expression of neuropsychological traits, such as executive functioning and impulsivity. Such variations lead to distinct endophenotypes that within bulimia are viewed as a group whose members are highly impulsive versus a group whose members do not have other problems associated with impulse control.

Southgate, Tchanturia, and Treasure (2005) proposed a biopsychosocial model describing the putative mechanisms through which dysfunctional endophenotypes develop, in which early

neurodevelopmental risk factors compromise cognitive and emotional processing abilities in adolescence that subsequently manifest as eating disordered behavior. Predisposing factors occurring during gestation and early childhood disrupt the early development of the central nervous system, thereby hindering later brain maturation into adolescence. Immaturity of brain function, specifically in the prefrontal cortex and the limbic system, leads to poor cognitive control of behavior and maladaptive coping skills that are expressed as specific eating disorder phenotypes depending upon latent personality traits, such as rigidity or impulsivity.

The biopsychosocial model of eating disorders contends that early predisposing factors directly contribute to later precipitating and perpetuating factors (Treasure, Tchanturia, & Schmidt, 2005). Early neurodevelopmental risk factors, including genes, perinatal complications, early life experiences such as trauma or feeding difficulties, and dysregulation of the neuroendocrine functions of the hypothalamus (e.g., dysfunction of serotonin or the HPA axis) interact to cause an underlying abnormality of the central nervous system (Connan et al., 2003; Favaro, Tenconi, & Santonastaso, 2006). The full extent of this disruption to the central nervous system (CNS) is not realized until adolescence, when these early neurodevelopmental abnormalities interfere with typical adolescent brain maturation and the development of social information processing abilities, which involves the limbic system for affective processing and the prefrontal cortex for cognitive regulation (Nelson, Liebenluft, McClure, & Pine, 2005).

Typical brain maturation during adolescence involves structural changes, including synaptic pruning, dendritic arborization, and increased myelination, that maximize the efficiency of processing among neural networks shaped by learning and experience (Luna & Sweeney, 2004). Enhanced connections between the prefrontal cortex and sub-cortical structures such as the basal ganglia and hypothalamus make inhibitory processes more efficient and consistent.

Disruption to the development of these connections can lead to dysfunction of higher-order cognitive skills, resulting in immature behavioral coping and impaired ability to self-regulate, organize, and inhibit behavior. During pubertal onset, changes in hormones impact the emotional saliency of stimuli and lead to heightened emotional responsiveness. A compromised limbic system contributes to addictive consumption cycles of eating (Volkow & Wise, 2005), and without well-developed coping skills, the individual is left with poorly regulated emotionality that affects psychosocial functioning (Troop & Bifulco, 2002).

Finally, Collier and Treasure (2004) indicate that latent personality traits affect the expression of eating disorder phenotypes. The trait of rigidity is hypothesized to manifest as food restriction, and conversely, the trait of impulsivity is hypothesized to manifest as binge-purge behavior (Lowe & Eldridge, 1993). Neurobiological research shows that personality-trait variations among individuals with eating disorders coincide with variations of the serotonin system (Steiger, 2004). The trait of impulsivity has consistently differentiated samples of bulimia from other eating disorders (Cassin & von Ranson, 2005; Fink, Smith, Gordon, Holm-Denoma, & Joiner, 2009), and within samples of bulimia, subtypes correspond to latent personality traits, with impulse control dysregulation most robustly identified (Claes et al., 2006; Duncan et al., 2005; Steiger & Bruce, 2007; Wonderlich et al., 2005; Wonderlich et al., 2007).

Impulsivity

Definitions and Characteristics

Impulsivity is a broad term that is used to describe behaviors, cognitive events, and personality traits. Although the literature lacks consensus regarding a single definition, research suggests that impulsivity exists as a multi-factored personality trait (Laos et al., 2001; Smith et al., 2007; Zuckerman, 2005) that manifests as different behaviors via several interacting neural mechanisms (Evenden, 1999; Talpos, Wilkinson, & Robbins, 2006). Many synonyms for impulsivity exist and are used interchangeably in the literature (Lowe & Eldredge, 1993), including disinhibition, risk-taking, sensation-seeking, and inability to delay gratification. Although these terms capture different aspects of the larger construct of impulsivity, they are all related to action without forethought in regard to consequence.

Along with challenges in defining impulsivity, issues of measurement have also contributed to contradictory findings in the literature. Additionally, assessment of personality traits such as impulsivity among the eating disordered population is further complicated by the young age of onset, denial and distortion in self-report, instability of eating disorder subtype, and potential state-effects of dieting or starvation. Evaluation conducted outside acute onset of illness or initial treatment is likely more valid (Vitousek & Stumpf, 2005).

The construct of impulsivity can show low correlations across measures (Olson, 1989). This suggests that impulsivity can be situation or task specific, and results may vary depending on how impulsivity is defined. For example, Wonderlich, Connolly, and Stice (2004) found in their prospective study of impulsivity as a predictor of eating disorders that self-reported trait impulsivity failed to predict onset of eating disorders, however, when more objective behavioral

criteria were used, such as the occurrence of delinquency or substance abuse, the onset of eating disorders was significantly predicted by the construct of behavioral impulsivity.

Discrepant findings may also be due to the multi-componential nature of impulsivity (Fischer, Smith, & Cyders, 2008; Loas et al., 2001). For example, Swann, Bjork, Moeller, and Doughtery (2002) differentiated rapid-response impulsivity from reward-delay impulsivity using different measures. Dawe and Loxton (2004) discussed reward sensitivity as a motivational force that drives women with bulimia to procure food to use for binges, whereas rash impulsivity is what causes the behavioral act of bingeing. Peñas-Lledó, Vaz, Ramos, and Waller (2002) found that their sample of outpatients with bulimia were more likely to demonstrate external impulsivity, such as theft, than internal impulsivity, such as self-harm. Other variables, such as gender, have also been suggested to mediate the demonstration of aspects of impulsivity (Reynolds et al., 2007).

The role of impulsivity has been well defined among many psychiatric disorders, including borderline personality disorder (Links, Heslegrave, & van Reekum, 1999), antisocial personality disorder (Barratt, Stanford, Kent, & Felthous, 1997), bipolar disorder (Swann et al., 2001), substance abuse (Brady, Myrick, & McElroy, 1998), and suicidality (Mann, Waternaux, Haas, & Malone, 1999). One of the most well researched disorders associated with impulsivity is attention deficit-hyperactivity disorder (ADHD). Impulsivity is included in the DSM-IV-TR as a symptom of sub-categorization for diagnosis (2000). Barkley's well-developed model of ADHD (1997) posits that a deficit in behavioral inhibition is the centrally defining characteristic of the disorder and leads to secondary impairments in other aspects of cognition.

Aside from the qualitative evidence of shared impulsive clinical presentation, there is also empirical evidence linking ADHD and bulimia (Biederman et al., 2007; Cortese,

Bernardina, & Mouren, 2007; Nazar et al., 2008; Quinn, 2008). Prevalence rates reported by Surman, Randall, and Biederman (2006) indicate that bulimia is significantly more common among the ADHD population (11-12% of females with ADHD) than the general population (1-3%). Prospective research by Mikami and colleagues (2008) found that ADHD-Combined Type in childhood predicted the onset of symptoms of bulimia in adolescence, with childhood symptoms of impulsivity noted as the best predictors of later eating pathology.

The clinical utility of understanding the comorbidity between ADHD and bulimia is most apparent for pharmacological treatment options (Cortese et al., 2007). Dukarm (2005) and Drimmer (2003) independently reported that patients with either bulimia alone or bulimia with comorbid ADHD showed decreased frequency or complete elimination of bulimic symptoms when treated with psychostimulants, lending credence to the notion of a shared underlying deficit in impulse control that can develop into phenotypically distinct disorders.

Personality Theories

As with bulimia, theories of impulsivity are prevalent in the psychological literature. In the pioneering work of Eysenck, impulsivity was defined primarily as a component of the psychoticism trait, which was derived via factor analysis and subsequently described in relation to low cortical arousal (Eysenck, 1967; Eysenck, Eysenck, & Barratt, 1985). Other personality theorists have addressed impulsivity as either a unique trait (e.g., Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993) or a subcomponent of a larger personality construct, such as neuroticism (Costa & McCrae, 1992) or reward dependence (Cloninger, 1987). The construct of disinhibition has also been discussed as occurring on both trait and state levels (Leeman, Grant,

& Proenza, 2009), although there is some suggestion that trait-level disinhibition is a risk factor for later state-dependent disinhibition (e.g., substance abuse, binge eating, etc.).

Temperament research has supported the concept of impulsivity as an inborn and enduring characteristic of the individual (e.g., Buss & Plomin, 1975; Caspi et al., 2003; Zawadzki, Strelau, Oniszcenko, Riemann, & Angleitner, 2001). Impulsivity is often discussed in the temperament literature in terms of its converse, behavioral inhibition. Kagan (1989) and Reznick (1989) have described behavioral inhibition as an early-established tendency to either approach or withdraw in response to the unfamiliar, with longitudinal evidence showing that the inhibited child is more likely to demonstrate shyness or anxiety later in life, while the uninhibited child is more likely to demonstrate affective and behavioral spontaneity (Kagan, Reznick, & Gibbons, 1989).

Impulsivity can also be conceptualized as an inability to delay gratification. Similar to the behavioral inhibition research, longitudinal studies have found that the ability to delay gratification in early childhood correlates negatively with impulsive traits in adolescence (Eigste et al., 2006; Mischel, Shoda, & Rodriguez, 1988). The ability to delay gratification involves a complex web of previous experiences, attention, and anticipation. An individual who can delay gratification can withhold his or her immediate reaction to gratify some need or desire long enough to appraise the situation of possible consequences, weigh judgment of options, and make a conscious and deliberate choice to postpone action until a later time (Mischel, Shoda, & Rodriguez, 1989).

Impulsivity is also discussed in relation to sensation-seeking or risk-taking. The construct of sensation-seeking is defined as seeking intense, complex, and novel experiences and the willingness to take risks for the sake of having such experiences (Zuckerman, 1993).

Cognitive mechanisms such as risk appraisal are identified as affecting the expression of the trait of impulsivity and risk-taking behaviors. Impaired impulse control is likely to interfere with one's ability to accurately and sufficiently appraise potential risks before acting. The ability to 'pause and think' is generally absent among individuals who are highly impulsive. Thus, the act of engaging in risky behavior may, under some conditions, reflect a lack of premeditated thought and poor impulse control.

The Type-T theory of risk-taking defines the sensation-seeking trait as potentially both positive and negative (Farley, 1991; 2001). Individuals with a risk-taking personality might be daring enough to venture into new and unknown places or to stand up against injustice regardless of consequences. Conversely, risk-takers might make risky decisions that are likely to cause harm or trouble, such as the high risk behaviors seen in teenagers who engage in unprotected sex or drug use. If the trait of risk-taking can lead to either positive or negative outcomes, other mediating variables might affect the expression of the trait (Kirkpatrick et al., 2007).

The Neurobiology of Impulsivity

Empirical studies of genetics, biophysiology, and functional brain imaging have provided extensive evidence for the biological basis of impulsivity (Leeman et al., 2009). Heritability estimates ranging from 30-50% have been reported (Zuckerman, 2007). As with the research on neurobiological correlates of eating dysfunction, both structural and functional neurobiological irregularities have been associated with impulsivity.

The evidence suggests that hypo-activity of limbic structures leads to impulsivity. Greater limbic arousal has been found among timid infants who showed a fear response or withdrawal behavior, than among those with a tendency to demonstrate uninhibited behavior

(Kagan, 1989). This is consistent with research indicating that adults who had been characterized as inhibited children showed increased activation in limbic areas on fMRI (Schwartz, Wright, Shin, Kagan, & Rauch, 2003). Conversely, impulsive adults have been found to show hypo-activation in the amygdala as compared to non-impulsive adults (Glahn, Lovallo, & Fox, 2007).

Research also suggests there is an important relationship between the integrity of the frontal lobes and impulse control. Acquired frontal lobe damage is often associated with increased impulsivity as well as reduced anxiety and its inhibiting effects (Jentsch & Taylor, 1999). Raine and colleagues (2004) found that impulsive prisoners diagnosed with antisocial personality disorder showed 11-14% reduction in prefrontal gray matter compared with normal controls and a substance abuse group. Some researchers have suggested that congenital or early-onset damage to prefrontal areas, particularly the ventromedial prefrontal cortex, disrupts the normal development of social and moral reasoning, resulting in a poorly self-regulated syndrome similar to psychopathy in adults (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Damasio, Tranel, & Damasio, 1990). Neuroimaging and lesion studies suggest that different regions of the prefrontal cortex are associated with different aspects of impulsivity, although these regions functionally operate in concert (Bechara & van der Linden, 2005).

Early research suggested that low cortical arousal and sensitivity of the reticular activating system were the primary biological causes of impulsivity (Eysenck, 1967). Refinement in understanding of functional brain systems led to the development of more sophisticated models. Gray (1987) proposed two interdependent dopaminergic systems that lead to the expression of personality characteristics: the Behavioral Inhibition System (BIS), underlying the expression of anxiety, and the Behavioral Activation System (BAS), underlying

the expression of impulsivity. Gray indicated that the BIS receives cortical input from orbitofrontal and ventromedial prefrontal cortex, lesions to which are known to disinhibit behavior. The BAS is described as sensitive to signals of reward and insensitive to signals of punishment, which would be reflected in behaviors such as risk-taking and gambling.

Barrett (1991) emphasized the temporal learning context of impulsivity and described interrelated circuitry involving the orbitofrontal cortex, amygdala, basal gangalia, and cerebellum. Zuckerman (2005) proposed three interactive neurobehavioral systems that contribute to impulsive behavior: (1) approach, associated with dopamine; (2) inhibition, associated with serotonin; and (3) arousal, associated with norepinephrine. Zuckerman argued that high sensation-seeking is caused by high reactivity of the approach system and low reactivity of the inhibition and arousal systems.

The role of dopamine in effecting approach behaviors via motor activity has been attributed to activity in the prefrontal cortex and basal ganglia (Nieoullon, 2002). Animal studies have shown that highly impulsive animals had high levels of dopamine (Dellu, Piazza, Le Moal, & Simon, 1996; Siegel, Sisson, & Driscoll, 1993). Low levels of the enzyme monoamine oxidase (MAO), particularly MAO type B, which oxidizes dopamine in humans, are also associated with impulsivity (Evenden, 1999). Dopaminergic brain pathways involved in the reward circuitry of the limbic system running from the ventrotegmental area to the nucleus accumbens, lateral hypothalamus, and prefrontal cortex, are thought to contribute to impulsive behavior by causing the euphoria experienced by sensation-seekers (Zuckerman, 1993).

Serotonin activity causes inhibition of behavior, and deficient serotonin has been linked to disinhibition (Soubrié, 1987). Many studies (e.g., Croft, Klugman, Baldeweg, & Gruzelier, 2001; Lucki, 1998; Manuck et al., 1998; Steiger, 2004) have demonstrated a negative correlation

between serotonin activity and impulsivity across populations, such that abnormally low levels of serotonin are related to increased impulsivity, and vice versa. The ascending serotonergic system originating in the raphe nuclei and innervating the amygdala, hippocampus, hypothalamus, and orbitofrontal cortex has been associated with self-control, and a weakness in this system would result in impulsive behavior. Similarly, the arousal circuitry moderated by the ascending noradrenergic system running from the locus coeruleus through the entire limbic system and cortex is connected with fear arousal. A weakness of this system may also result in high sensation seeking.

Multi-Impulsivity

As described in the DSM-IV-TR (2000), an essential characteristic of bulimia is feeling a lack of control when eating that is experienced by some as being in a frenzied state while bingeing, or feeling a dissociative quality during the binge-purge episodes. Similar to the experiences of substance abusers described in the addictions literature (Avena, Rada, & Hoebel, 2008), indicators of impaired self-control are also reported among women and adolescents with bulimia (Tanofsky-Kraff et al., 2007), including feeling out of control when eating, difficulty resisting the urge to binge, or difficulty stopping a binge once it has begun (Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2007). Other behaviors signifying a lack of control are commonly found in individuals with bulimia, such as self-injurious behaviors (Favaro & Santonastaso, 1999; Wonderlich, Myers, Norton, & Crosby, 2002), aggressive behavior (Miotto et al., 2003), alcohol abuse (Gadalla & Piran, 2007) and substance abuse (Engel et al., 1995; Herzog, Keller, Sacks, & Yeh, 1992; Thompson-Brenner et al., 2008), sexual promiscuity

(Culbert & Klump, 2005), stealing (Rowston & Lacey, 1992), and other disorders of impulse control such as trichotillomania (Christenson & Mitchell, 1991; Lowe & Eldredge, 1993).

Poor self-control when eating, along with the constellation of impulsive behaviors that are commonly comorbid with bulimia, has been described in the literature as a *multi-impulsive syndrome*, characterized by a trend of impulsive behaviors, a general failure to consider risks and consequences with deliberation, and an accompanied sense of being out of control and a desire to block distressing feelings from awareness (Bell & Newns, 2002; Lacey & Evans, 1986). Although a single accepted definition for multi-impulsive bulimia could not be found in the literature, research has shown that individuals with bulimia who present with at least three additional impulsive behaviors, including alcohol or drug use, self-harm, suicide attempts, stealing, or sexual promiscuity, are distinct from and have a different course of illness from other individuals with bulimia (Fichter, Quadfleig, & Rief, 1994). A similar concept has emerged from the mood disorders literature termed the *soft bipolar spectrum* (Katzow, Hsu, & Ghaemi), which is characterized by a cyclothymic-anxious-sensitive temperament that causes mood reactivity and behavioral impulsivity (Perugi & Akiskal, 2002), and has been used to explain the high rates of comorbidity between bulimia and bipolar disorders (Lunde, Fasmer, Akiskal, Akiskal, & Oedegaard, in press; McElroy, Kotwal, Keck, & Akiskal, 2005).

The literature suggests that multi-impulsive patients with bulimia represent a distinct sub-sample of the larger population of individuals with bulimia (Duncan et al., 2005; Myers et al., 2006; Steiger & Bruce, 2007; Wonderlich et al., 2002). Serotonergic abnormalities appear to be more pervasive among women with bulimia who have multi-impulsivity than those without (Steiger et al., 2001; Steiger, Lehoux, & Gauvin, 1999). Patients with bulimia who present with multi-impulsivity also tend to have more extensive clinical histories, including past trauma,

longer duration of illness, earlier onset of symptoms, more pervasive psychopathology (Corstorphine, Waller, Lawson & Ganis, 2007; Favaro et al., 2005), including greater difficulties with negative affect and anger (Fassino, Daga, Pierò, Leombruni, & Rovera, 2001), and worse prognoses than do patients without multi-impulsivity (Sohlberg et al., 1989; Steiger et al., 2008). Among the compensatory behaviors that are seen among individuals with bulimia, laxative abuse was found to be most highly correlated with impulsivity (Tozzi et al., 2006).

Research supports the relationship between multi-impulsivity and bulimia (Claes, Vandereycken, & Vertommen, 2005; Fernandez-Aranda et al., 2006; Lejoyeux, Arbaretaz, McLoughlin, & Ades, 2002; Newton, Freeman, & Munro, 1993; Yeomans, Leitch, & Mobini, 2008). Lacey (1993) identified 80% of inpatient women with bulimia as having three or more types of impulsive behaviors. Favaro and colleagues (2005) found that specific predictors of multi-impulsive bulimia included purging behavior, novelty seeking, and low persistence. Survey research of community samples has likewise found self-report of bulimic symptoms to be significantly correlated with impulsivity in adults (Lyke & Spinella, 2004) and adolescents (Kaltiala-Heino, Rissanen, Rimpelä, & Rantanen, 2003). Cross-cultural research conducted by Matsunaga and colleagues (2000) found that multi-impulsivity among Japanese women with bulimia existed at equal rates as has been found in Western cultures, supporting the external validity of this construct. Research also suggests that multi-impulsivity may predict worse prognosis by contributing to the refractory nature of bulimia, as symptoms of impulsivity often predate the onset of eating pathology (Nagata, Kawarada, Kiriike, & Iketani, 2000) and problems of impulse control are not directly treated in most eating disorder treatment programs (Evans & Lacey, 1992).

It is argued that an underlying deficit in cognitive processing contributes to behavioral impulsivity, which manifests as bulimic symptoms as well as the associated "multi-impulsive" behaviors. Vitousek and Holland (1990) described the essential connection between bulimia and impulse control according to cognitive processing parameters, specifically automatic processing. The literature suggests that individuals with bulimia often display a behavioral repertoire that is highly reward-dependent and that is not well-mediated through cognitive appraisal mechanisms, including executive functions such as planning and response inhibition (Hinson, Jameson, & Whitney, 2003). Individuals with bulimia were compared to pathological gamblers in a study of clinical samples known to demonstrate impulsivity (Alvarez-Moya et al., 2007), and individuals with bulimia were identified as having more pronounced disorganization, immaturity, and lack of goal-directed behavior than the pathological gamblers. This suggests that executive dysfunction is not circumscribed to impulse control among the bulimic population.

Executive Functions

Definitions and Characteristics

Executive functioning is an umbrella term denoting those processes primarily localized in the prefrontal areas of the brain that enable higher order thinking, regulate behaviors and emotions, and coordinate the cognitive activities that take place elsewhere in the brain, such as memory, learning, and reasoning, in order to facilitate complex human endeavors (Baddeley, Della Sala, Gray, Papagno, & Spinnler, 1997). Although many definitions have been offered, most include the concepts of working memory, set maintenance or shifting, and inhibitory

control, to varying degrees (Fuster, 2002). Fundamentally, the executive functions are processes of control (Denckla, 1996) for various domain-specific cognitive functions, including motor activity, attention, language, learning, etc.

The executive functions are notoriously difficult to define (Lyon & Krasnegor, 1996; Stuss & Alexander, 2000). As indicated by Denckla (1996), the executive functions have historically been understood from a neuroantomical context rather than by theory or model. Some have argued that the executive functions are too heterogenous to be considered under a single construct (Parkin, 1998). Others have argued that they are better understood as general intellectual ability, akin to Spearman's concept of g, rather than as distinct processes (Duncan, Burgess, & Emslie, 1995). However, most contend that the executive functions are structurally and functionally unique aspects of cognition (Anderson et al., 2001) that exist not as a unitary construct, but as sub-functions localized in different areas of the frontal lobes (Heyder, Suchan, & Daum, 2004; Stuss & Alexander, 2000) and that are highly consistent with psychometric fluid intelligence (Horn & Cattell, 1967; Kane & Engle, 2002).

Attempts to operationally define executive functions have met with similar difficulties found in defining intelligence, namely that no single task or subset of tasks provides an acceptably comprehensive or exemplary definition (Rabbit, 1997). A major problem has been the inconsistent and interchangeable use of terms, such as "executive" and "frontal" (Stuss & Alexander, 2000). Poorly understood construct validity and confusion about what constitutes an executive process versus other cognitive processes is exacerbated by the use of psychological assessment tools that are complex and multi-factorial, performance on which likely reflects several co-occurring cognitive processes including executive skills. Neuropsychological research has shown that different regions of the frontal lobes contribute to performance on some

tasks but not others and that typical assessment batteries are not uniformly sensitive to all varieties of frontal lobe damage (Bigler, 1988). Despite these definitional difficulties, several executive functioning instruments exist that show good psychometric qualities and that can assist in evaluating an individual's cognitive profile (Goldberg & Bougakov, 2005).

Developmentally, the executive functions emerge in early childhood and continue maturation into adolescence, paralleling the growth and myelination of the frontal lobes (Happaney, Zelazo, & Stuss, 2004; Jurado & Roselli, 2007). Research indicates that the executive functions emerge during pre-school years (Hongwanishkul, Happaney, Lee, & Zelazo, 2005) and are generally mature by approximately age 15 (Anderson et al., 2001), although different aspects of executive functioning have been found to develop at different rates lasting into the 20s (Anderson, 2002; Brocki & Bohlin, 2002). Early development of response inhibition is demonstrated by success on the Piagetian A-not-B task by approximately age one, followed by reversal learning and distress regulation by age two to three, and delay of gratification and conflict resolution for incongruent stimuli by age five (Evans, Lewis, & Iobst, 2004). Inhibitory controls as demonstrated on neuropsychological measures are generally fully developed by age 12 (Jurado & Roselli, 2007).

Frontal lobe dysfunction in children can have varying outcomes (Powll & Voeller, 2004) that may be quite dissimilar from the profiles of adults (Anderson, Jacobs, & Harvey, 2008). Congenital disorders can offset the development of executive functioning early and have lasting effects. Longitudinal evidence confirms that children with executive skill deficits early in childhood maintain those deficits later in life (Biederman et al., 2008). Acquired frontal injuries can have little, immediate, or delayed impact. Basic brain functions, such as motor output, are generally quite resilient to early insult, while complex functions, such as social behavior and

decision making, often show chronic impairment (Anderson, Damasio, Tranel, & Damasio, 2000). Some frontal injuries show delayed onset of impairment following a period of normal developmental progression. The multi-stage maturational process of the frontal lobes extends into adolescence, and early neurological compromise can disrupt the acquisition of basic competencies that are necessary for later development of more complex processing abilities (Eslinger, Grattan, Damasio, & Damasio, 1992).

Theories of Executive Functions

The executive functions were initially understood according to the clinical impairment demonstrated after focal prefrontal brain injury (Luria, 1966; Walsh, 1978); theory formulation and empirical research in the area have developed secondarily. Current conceptualizations have drawn substantially from the cognitive psychology literature. Derived from classic information processing models (e.g., Atkinson & Shiffrin, 1968), executive functions became distinguished for their role in novel versus routine processes and their ability to control or coordinate routine cognitive skills (Norman & Shallice, 1986).

Several models of executive functioning have been proposed. Baddeley's influential model of working memory initially described the central executive system as a manager for visual and auditory memory and long-term information storage (Baddeley & Hitch, 1974). Contemporary conceptualizations (e.g., Baddeley, 1998; Kane & Engle, 2002) have generally maintained the role of management, but extend into other realms of cognitive processing in addition to memory systems, including the self-regulation of affect as well as the regulation of behavior via task initiation, goal-directed persistence, cognitive flexibility, response inhibition,

planning and organizing problem-solving strategies, self-monitoring and metacognition, and time management (Dawson & Guare, 2004).

The supervisory attentional system (SAS) was first described by Norman and Shallice (1986) and later expanded by others (e.g., Shallice & Burgess, 1996; Stuss, Shallice, Alexander, & Picton, 1995). This model describes two complementary processes: contention scheduling, which conducts automatic processing of well-formed schemata, and the SAS. The SAS becomes involved in situations where schemata do not yet exist, such as the novel or complex tasks that compose typical executive functioning assessments.

Developmental models of executive function have also been proposed. Barkley's (1997) model of self-regulatory functions describes executive dysfunction as the essential characteristic of ADHD. Further, behavioral inhibition is identified in Barkley's model as a prerequisite for developing self-regulatory processes, including working memory, self-regulation of affect/motivation, internalization of speech, and reconstitution. The executive control system described by Anderson (2002) was derived from factor analytic studies of executive functioning measures and longitudinal studies of executive functioning development. It discusses four independent domains that are consistently found in factor analyses and that maintain different developmental trajectories, including cognitive flexibility, goal setting, attentional control, and information processing.

Others have rejected the hierarchical models of executive functioning posited by authors such as Baddeley and Shallice. For example, Zelazo and colleagues discuss the integrative nature of executive functions within a macro-construct framework, in which the overall goal of problem solving is achieved by interaction among component sub-functions (Zelazo, Carter, Reznick, & Frye, 1997). Rather than focus on localization within the frontal lobes, the

interconnected circuitry of the entire brain is identified as necessary for successful problem solving.

The Neurobiology of Executive Functions

Historically, the executive functions have been associated with the frontal lobes of the brain (Bianchi, 1895; Luria, 1966). An abundance of contemporary neuroimaging research has converged to substantiate clinical lesion studies indicating that executive skills are associated with the frontal regions of the brain, notably the prefrontal cortex (Rabbitt, 1997). The prefrontal cortex is described as meta-modal because it contains connections to virtually every other region of the brain and can implement top-down control processes by integrating information that has already been processed at a lower level (Royall et al., 2002). As the only location of the brain to integrate connections from limbic structures and the basal ganglia, the prefrontal cortex is able to modulate input of motivational and emotional information into goal-directed behavior.

However, research also indicates that the frontal lobes are not the exclusive domain of the executive functions and that damage to the frontal lobes can have varying outcomes (Andres, 2003). This has led to the re-conceptualization of the frontal lobes as necessary but not sufficient for proper executive functioning (Anderson, 2008). As demonstrated by animal studies and human imaging studies, a network of brain regions, including prefrontal cortex, posterior cortical regions, and sub-cortical structures, are involved in successful executive functioning (Goethals, Audenaert, Van de Wiele, & Diercks, 2004; Heyder, Suchan, & Daum, 2004).

The frontal lobes are considered functionally heterogenous (Alexander & Stuss, 2000). The left prefrontal cortex is associated with convergent processing, including language, and internally mediated self-regulatory processes, such as the use of working memory. The right

prefrontal cortex is associated with divergent processing of novel information and externally mediated self-regulatory processes, such as the use of feedback to alter responding (Goldberg, Podell, & Lovell, 1994). Hypo-activity of the left prefrontal region has been associated with depressive symptoms, and hypo-activity of the right prefrontal region has been associated with poor impulse control, as seen in samples of bulimia (Marsh et al., 2009) as well as ADHD and substance abuse (Majewska, 2002).

Various neurotransmitter systems operate within the prefrontal cortex with different effects on executive functions (Robbins & Roberts, 2007). Dopamine is particularly noted for its role in modulating attention, including selection and allocation of attention to relevant stimuli and inhibition from irrelevant stimuli. Serotonin has been found to regulate reversal learning, such that deficits in this system result in perseverative responding. Norepinephrine and acetylcholine have also been implicated in contributing to set-switching and reversal learning, respectively. Treatment of executive dysfunction, as seen among individuals with ADHD, often involves pharmacological intervention to increase dopamine and norepinephrine activity in the prefrontal cortex (Arnsten & Li, 2005). There is also evidence for lateralization of neurotransmitter activity associated with mood in the prefrontal cortex: increased levels of dopamine in the right prefrontal cortex are associated with positive mood, and conversely, lower levels of serotonin in the left prefrontal cortex are associated with negative mood (Mitchell & Phillips, 2007).

"Hot" and "Cool" Executive Functions

Within the literature, a general distinction exists between meta-cognitive ("cool") and emotional/motivational ("hot") executive functions (Ardila, 2008; Zelazo, Qu, & Muller, 2004).

The “hot” and “cool” executive functions operate via separate, yet interacting, frontal circuits. Although often discussed as distinct, imaging research shows that the frontal circuits associated with “hot” and “cool” functions work together concurrently with divergent activation, rather than in isolation (Moghaddam & Homayoun, 2008).

The “cool” executive functions pertain to meta-cognitive abilities and cognitive control, such as planning, working memory, and problem solving. These “cool” executive functions are localized to the dorsolateral prefrontal cortex, which maintains substantial connections to motor cortex, thereby effecting motor planning, volitional behavior, selection of behavioral goals, and generation of conceptual action sequences (Tanji & Hoshi, 2008; Tanji, Shima, & Mushiake, 2007). The left dorsolateral prefrontal cortex has been identified as involved with task-setting and verbal regulation of behavior, while the right dorsolateral prefrontal cortex is associated with monitoring and error-detection (Stuss & Alexander, 2007). Dorsolateral lesions produce deficits in verbal and nonverbal fluency, set shifting, and learning and retrieval.

The “hot” executive functions pertain to emotional/motivational executive functions and affective control, including impulse control, decision-making, and coordination of emotional processing with behavioral problem solving. These “hot” executive functions are localized to the orbitofrontal, ventromedial, and anterior cingulate regions, all of which maintain substantial interconnections with limbic structures. Damage to these regions has been associated with personality change similar to the well-known case of Phineas Gage (Harlow, 1848). More specifically, orbitofrontal lesions tend to cause disinhibition and irritability, and ventromedial-anterior cingulate lesions often result in apathy and decreased motivation (Alvarez & Emory, 2006; Tekin & Cummings, 2002). The right orbitofrontal region is particularly involved in inhibitory control (Collette, Hogge, Salmon, & van der Linden, 2006; Robbins, 2007). Lesions

to this area, as opposed to left or other prefrontal regions, result in pronounced social disinhibition (Tekin & Cummings, 2002), poor impulse control on neuropsychological tasks (Aron, Robbins, & Poldrack, 2004; Marsh, 2009), and riskier decision-making deficits (Clark, Mnes, Antoun, Sahakian, & Robbins, 2003).

As the locus for "hot" executive functions, the orbitofrontal, ventromedial, and anterior cingulate regions have been identified as critical for decision-making in response to emotion (Bechara, Damasio, & Damasio, 2000; Fellows, 2007). Using experimental gambling tasks, Damasio and colleagues developed the somatic marker hypothesis to explain how persons with damage to these circuits show poor decision-making skills in relation to affective signals (Clark et al, 2008) and make decisions contingent on immediate prospects while future consequences tend to be ignored (Bechara, Tranal, & Damasio, 2000). Imaging research has found that damage to the ventromedial and orbitofrontal regions is associated with the onset of impulsive behaviors, including substance abuse (Bechara & Damasio, 2002; London, Ernst, Grant, Bonson, & Weinstein, 2000), bulimia (Marsh et al., 2009), and increased high-risk behavior (Bechara, Tranel, Damasio, & Damasio, 1996).

The anterior cingulate cortex has been identified as critical for motivated attentional allocation, error-detection, and conflict resolution during interference tasks such as the Stroop task (Carter & van Veen, 2007; Royall et al., 2002), in which the subject must inhibit the automatic process (pre-potent response) of reading a word and instead name the color of the word's ink (Stroop, 1935). It has been suggested that the anterior cingulate functions by detecting incongruent stimuli (e.g., subject is confronted with Stroop interference task), assigning the affective value of the outcome of a particular action (e.g., "reading the word = wrong"), and signaling to dorsolateral prefrontal cortex to activate cognitive control (e.g., "do

not read the word, say the color") in responding (Kane & Engle, 2002; Rushworth, Walton, Kennerley, & Bannerman, 2004). Subsequently, damage to the anterior cingulate is associated with disinhibition of responding in Stroop interference tasks (Tekin & Cummings, 2002).

Neuropsychological Studies of Bulimia Nervosa

Deficits of Executive Functioning

Research shows that general intellectual ability among patients with eating disorders does not differ from the distribution of the normal population (Blanz et al., 1997; Ranseen & Humphries, 2002; Touyz, Beumont, & Johnstone, 1986); however, there have been findings of impaired cognition in these patients (Steinglass et al., 2006). Neuropsychological assessment tools have enabled researchers to look more closely at different aspects of cognition that are functional versus deficient. There is some evidence indicating that cognitive processing deficits persist after patients are no longer in treatment, which suggests they are likely pre-morbid deficits that are trait-related (Gillberg, Rastam, Wentz, & Gillberg, 2007).

As compared to anorexia, the investigation of the neuropsychology of bulimia has been less thoroughly addressed in the literature. Southgate, Tchanturia, and Treasure (2006) reviewed 52 studies of the neuropsychology of eating disorders, and while all 52 included a sample of patients with anorexia, only 18 included a sample with bulimia. Those few studies that have included a sample with bulimia have generally found impairments in attention (Bosanac et al., 2007; Jones, Duncan, Brouwers, & Mirsky, 1991; Lauer, Gorzewski, Gerlinghoff, Backmund, & Zihl, 1999), including poor vigilance during continuous performance tasks (Laessle, Bossert,

Hank, & Hahlweg, 1990; Laessle, Fischer, Fichter, & Pirke, 1992; Laessle, Krieg, Fichter, & Pirke, 1989), attentional biases for disorder-relevant stimuli (Shafran, Lee, Cooper, Palmer, & Fairburn, 2007), and higher susceptibility to distraction (Alvarez-Moya et al., 2009). Other notable deficits include poor mental flexibility (Tchanturia et al., 2004), difficulty maintaining cognitive set (Ferraro, Wonderlich, & Jocic, 1997), and decision-making deficits during gambling tasks, in which they chose the more risky options that had the potential for higher payout but with far less likely odds (Boeka & Lokken, 2006; Brand, Franke-Sievert, Jacoby, Markowitsch, & Tuschen-Caffier, 2007; Liao et al., 2008; Task, Boeka, & Lokken, 2006).

These findings suggest that neuropsychological deficits among individuals with bulimia are most likely found among the domains of executive functioning. An alternative hypothesis of poor central coherence has been described (Lopez, Tchanturia, Stahl, & Treasure, 2008), although similar neural substrates are implicated. Namely, the right prefrontal cortex is discussed for its role in processing global information through environmentally-bound regulatory processes, which is found to be deficient among bulimic samples, while left hemisphere local processing for details remains adept (Lopez, Tchanturia, Stahl, & Treasure, 2009).

Studies investigating executive functioning skills in samples with bulimia have produced mixed findings (Galderisi et al., 2003; Murphy et al., 2002, 2004; Touyz et al., 1986), although this is partly due to the multi-faceted nature of the executive functions and their measures. For example, Bowers (1994) found that his sample with bulimia showed less impairment on a task of verbal fluency than the sample with anorexia. This can be explained by the rigid cognitive style seen in anorexia, which may have restricted patients from exhibiting generativity in responding. Conversely, there appears to be a trend among neuropsychological research indicating that samples of bulimia demonstrate an impulsive cognitive style (Heilbrun & Bloomfield, 1986) that

results in fluctuating rather than rigid response patterns (Tchanturia et al., 2004; Tchanturia, Serpell, Troop, & Treasure, 2001). Thus, neuropsychological research with the bulimia population may better be described as elucidating a particular pattern of impulsive responding, rather than demonstrating an absolute deficit in executive functioning abilities.

The notion of impulsive cognitive style among individuals with bulimia is further supported by studies showing that these patients favor speed over accuracy of performance. Ferraro and colleagues (1997) found that women with bulimia performed faster and made more errors than controls on the Symbol-Digit Modalities Task. Kaye, Bastiani, and Moss (1995) likewise found that their sample with bulimia performed faster and made more mistakes on the Matching Familiar Figures Test than did their sample with anorexia.

The Go/No-Go paradigm is a commonly used neuropsychological task that has demonstrated impulse control deficits among people with bulimia. Bruce, Koerner, Steiger, and Young (2003) found that women with bulimia who abused laxatives made more commission errors under cues for punishment, indicating that they were more disinhibited despite possible negative outcomes than were non-laxative abusing women with bulimia or controls. Mobbs, Van der Linden, d'Acremont, and Perroud (2008) found that compared to controls, their sample with bulimia showed problems with inhibition, faster reaction times, and worse discrimination ability, especially when the No-Go target stimuli were food-related.

Other studies have also produced findings that are consistent with poor impulse control. Beatty, Wonderlich, Staton, and Ternes (1990) found that their sample with bulimia made significantly more rule violations on a design fluency task than did their sample with depression, suggesting poor self-control in maintaining cognitive set. In a study of bulimia comorbid with borderline personality disorder, an additive effect occurred when both conditions were present,

resulting in worse performance on the Stroop and Trails B than if either condition was present in isolation (Bourke et al., 2006). Brand and colleagues (2007) found that frequency of disadvantageous decisions made by women with bulimia on their gambling task was significantly correlated with poor performance on the Stroop and Trails B.

Because neuropsychological task performance sometimes fails to correspond with everyday functioning (Burgess, 2000), several studies have investigated the relationship between executive functioning and eating disorder symptoms using self-report questionnaire measures. Using the Frontal Systems Behavior Scale and Eating Inventory questionnaires, Spinella and Lyke (2004) found significant correlations between self-reported dysexecutive traits, self-reported disinhibited eating, and self-reported food cravings. Fisher, Smith, and Anderson (2003) found that self-reported aspects of impulsivity were correlated with symptoms of bulimia especially when under conditions of negative affect.

Evaluating Executive Functioning in Bulimia: The Stroop Task

One of the most commonly used paradigms to investigate executive/attentional dysfunction in bulimia nervosa is the Stroop task (Stroop, 1935). Some have argued that the Stroop effect is evidence of an attentional bias, defined as a discrete change in the direction in which a person's attention is focused so that he or she becomes aware of a particular aspect of the stimulus environment (Williams, Watts, MacLeod, & Matthews, 1988). When a person cannot resist the pull of their attention towards that particular aspect of the stimulus and a response is made, this is considered an act of disinhibition or impulsive responding. Consequently, the Stroop is one of the most commonly used measures of inhibitory control (Ainsworth et al., 2002; Jurado & Boselli, 2007). While attentional bias among eating disorders

has been explored using other experimental tasks (e.g., Johansson, Ghaderi, Hallgren, & Andersson, 2008; Maner et al., 2006), they generally have not measured disinhibited responding.

The emotional Stroop is a modified Stroop task in which emotionally-salient words are intermingled with neutral words and patient reaction times are measured and compared. The hypothesis underlying this task is that individuals with bulimia will demonstrate an attentional bias towards emotionally salient words (i.e., demonstrate longer reaction times or latencies) and will be disinhibited in their responses (i.e., make more errors of reading the word rather than saying the color).

Although mixed findings have been reported for the emotional Stroop (Black, Wilson, Labouvie, & Heffernan, 1997; Carter, Bulik, McIntosh, & Joyce, 2000; Davidson & Wright, 2002; Huan, 1995; Lokken, Marx, & Ferraro, 2006; Quinton, 1998), a recent meta-analysis of emotional Stroop performance among eating disorders by Johansson, Ghaderi, and Andersson (2005) indicated that eating disordered patients had greater color-naming delays for negatively-valenced food/weight/shape related words than for neutral words compared to normal controls, with a medium effect size reported. In a similar meta-analysis, Dobson and Dozois (2004) indicated that significant differences were found when comparing samples of bulimia with controls and that medium effect sizes were found across studies that used either the classic or the emotional Stroop.

A number of conceptual and methodological issues have been raised regarding the emotional Stroop task (Ainsworth et al., 2002; Dobson & Dozois, 2004; Huon, 1995; Lee & Shafran, 2004). First, it is not a standardized task and there has been substantial inconsistency in content and presentation of the items (Faunce, 2002; Johansson, Carlbring, Ghaderi, & Andersson, 2008; Walker, Ben-Tovim, Paddick, & McNamara, 1995). Some studies used

disorder-non-specific terms with general negative emotional valence such as “failure” (Seddon & Waller, 2000; McManus, Waller, & Chadwick, 1996) while others used only food-specific words such as “cheese,” body-specific words such as “thighs,” shape-related words such as “fat,” or any combination thereof (Ben-Tovim & Walker, 1991; Cooper, Anastasiades, & Fairburn, 1992; Cooper & Todd, 1997; Fairburn, Cooper, Cooper, & Frank, 1991; Fornea & Burns, 1996; Jones-Chesters, Monsell, & Cooper, 1998; Lovell, Williams, & Hill, 1997; Perpiña, Leonard, Treasure, Bond, & Baños, 1998).

Secondly, the emotional Stroop does not control for the contribution of personal salience for hypothesized emotional words, and words with a positive emotional valence (e.g., “thin”) have generally been excluded. The control stimuli, or neutral words, have generally not been described in the literature, so it cannot be discerned whether emotional versus non-emotional words are appropriately matched for emotional valence or usage frequency (Huon, 1995). Those studies in which the words are reported have not been systematically replicated.

Additionally, latency has almost exclusively been the dependent variable of investigation, without the incorporation of baseline measures for word reading or color naming, and errors have not been systematically studied (Dobson & Dozois, 2004). Finally, the finding of longer reaction times for weight/shape/food related words on the emotional Stroop has not been limited to clinical samples of eating disorders (Cooper & Fairburn, 1992; Huon, 1995; Perpiñá, Hemsley, Treasure, & de Silva, 1993; Rofey, Corcoran, & Tran, 2004; Waller, Watkins, Shuck, & McManus, 1996), thereby limiting the interpretability of findings.

The classic Stroop procedure overcomes many of these limitations. The validity and reliability of the Stroop is well documented through decades of psychological research, and meta-analytic studies have concluded that the Stroop is the measure of choice for identifying

response disinhibition (e.g., Royall et al., 2002). It is a standardized task with only neutral words (colors). Because it does not attempt to measure selective attentional bias for disorder-relevant stimuli, the results of classic Stroop administration can be considered an indication of general ability to inhibit automatic processing. Although the classic Stroop has been used with samples of bulimia (Alvarez-Moya et al., 2009; Heilbrun & Bloomfield, 1986), it has been used far less often than the emotional Stroop paradigm.

Methodological Limitations

Southgate, Tchanturia, and Treasure (2006) conclude that the extant literature suggests there are neurobiological anomalies and cognitive processing deficits in eating disorders; however, the data can be unclear. Although the evidence from neuropsychological and neuroimaging studies suggest a pattern consistent with irregular executive functioning, the deficits are often subtle in comparison to preserved general intellectual functioning. Published studies are typically methodologically flawed, containing small samples or failing to control for potential mediating variables. Additionally, the preponderance of studies has focused exclusively on anorexia, and virtually none have included an adolescent sample.

One issue of concern is the confounding of results due to comorbid psychiatric conditions and unreported histories of head trauma. A thorough screening can prevent the inclusion of individuals with head trauma. The issue of comorbidity is not easily addressed. Many patients in treatment have comorbid mood or anxiety disorders. The literature suggests that cognitive function is unrelated to current mood status in both anorexia and bulimia, although very high levels of anxiety may be related to impaired performance on cognitive measures (Southgate, Tchanturia, & Treasure, 2006).

The effects of medication have not been consistently controlled for in the literature. Modification of serotonin can affect cognition, so patients taking SSRIs could exhibit decreased performance due to medication. Southgate and colleagues (2006) identified two studies specifically addressing this issue by running analyses both with and without the inclusion of patients who were taking medications. The results for both studies did not change when participants on medications were excluded, suggesting that medication effects may be minor on neuropsychological tasks.

Research Questions

In accordance with the biopsychosocial model outlined by Southgate, Tchanturia, and Treasure (2005) supported by the literature on impulsivity, executive functions, and the neurobiology of bulimia, it is hypothesized that problems of disordered eating seen in bulimia arise from problems of impulse control, which are caused, in part, by dysfunction of the executive skills of the frontal lobes. Neuropsychological measures of executive functioning provide the tools to test this hypothesis, and the literature supports the prediction that young women with bulimia nervosa will exhibit poor impulse control on neuropsychological measures of response inhibition (Sohlberg, 1991). This research study will extend previous research findings by studying an adolescent and young adult sample and by focusing exclusively on bulimia. Specific research questions to be addressed are as follows:

1. Does this sample of young women with bulimia differ significantly from the normative sample reflecting the general population on standardized measures of impulsivity?
2. Do levels of impulsivity correlate with symptom severity?
3. Does this sample show a preference for immediate versus delayed gratification?

CHAPTER 3

METHODOLOGY

Subjects

A between-groups research design was used to investigate the relationship between bulimia and impulsivity. The control group was accessed through normative samples for each of the standardized assessments, in which the normal curve distribution of performance including the mean and standard deviation of scores is described in each test's manual. The normative samples for the cognitive assessment tools (e.g., WASI, D-KEFS, BRIEF rating scales) reflect national samples of the general population stratified according to United States census data for demographic characteristics including age, ethnicity, and level of education. The normative sample for one of the eating disorder rating scales (EDE-Q) reflects a community based sample of non-eating disordered adult females; the other eating disorder rating scale's normative sample (EDI-3) is a clinical sample of adult and adolescent eating disorder patients with bulimia.

The treatment group was composed of patients with bulimia who were recruited from The Renfrew Center, a local inpatient treatment facility for eating disorders. Criteria for inclusion were that the individual be female, between the ages of 14 and 22, currently diagnosed with bulimia nervosa, and an inpatient at the treatment facility. Exclusion criteria included male gender, younger than 14 or over the age of 22, diagnosed with anorexia nervosa or eating disorder – not otherwise specified, history of neurological impairment or head injury, or performing below the cutoff score of 70 on the 2-Subtest Full-Scale IQ of the cognitive screening measure. The age range for inclusion was limited to ages 14 to 22 because this age range generally encompasses the developmental period of adolescence, extending into the

college years, and 14 is the youngest age that patients are admitted to The Renfrew Center. The sample was limited to females because eating disorders are known to largely affect women and because The Renfrew Center does not admit male patients.

The results of the power analysis indicated that 64 subjects would be required for sufficient statistical power. Given the time restraints and lack of resources affecting this research study, only 31 patients were initially recruited; however, three did not complete data collection because they decided they no longer wanted to participate. The final sample was composed of 28 women ages 15 to 22 years (see Appendix F for demographic characteristics). The modal age was 20. The ethnic composition of the sample was predominantly educated Caucasian females who were either employed or full-time students (see Table 3). All subjects reported being single or unmarried in committed relationships.

These patient characteristics are consistent with the total bulimia patient population at The Renfrew Center during the time of data collection (O'Planick, 2009). According to quarterly reports, 165 patients were discharged from The Renfrew Center from April through December 2008. Individuals with bulimia comprised 27% to 43% of the total patient population during this time, and the percentage of patients who were adolescents ranged from 16% to 30%. The majority of patients were Caucasian, ranging from 82% to 92% of the total population. Other common ethnic groups included Hispanic which was 10% of the total population from July through September, Multi-racial which was 4.7% of the population from October to December, Asian or Pacific Islander which was 2.3% of the total population from October to December, and African-American which was 2.3% of the total population from October to December. Less commonly represented ethnic groups included Native American and Other, which never exceeded 2% of the population during this time frame.

Table 3.1: Subject Demographics

	N	Percent of Sample
Ethnicity		
Caucasian	25	89
Asian/Pacific Islander	2	7
African-American	1	4
Religious Affiliation		
Catholic	12	43
Christian	6	21
None	3	11
Jewish	3	11
Other Spirituality	2	8
Hindu	1	4
Educational Attainment		
Completed Some College	16	57
Completed High School	5	18
Currently In High School	5	18
Completed 2-year Associate's Degree	1	4
Completed 4-year Bachelor's Degree	1	4
Employment Status		
Student Only	14	50

Table 3.1 (continued)

	N	Percent of Sample
Employed Part-Time	6	21
Employed Full-Time	1	4
Unemployed	6	21

* Percentages rounded to nearest whole number.

Procedures

Subject recruitment lasted for nine months. Patients at The Renfrew Center volunteered to participate in response to a brochure that publicized the study. Each week, the patient census was reviewed and a brochure was placed in the mailboxes of the newly admitted patients. The brochure listed the purpose of the study, the inclusion criteria, and directions for how to volunteer. The brochure specified that those patients who met inclusion criteria and were interested should come to an informational meeting, which was held every Saturday morning during a time that did not interfere with treatment activities.

Patients who attended the Saturday meetings were given a detailed explanation of the study and were told that their participation was voluntary and not required by The Renfrew Center. Those patients who were interested in volunteering were administered an informed consent procedure that involved verbal and written explanation of the purpose of the study, procedures, compensation, risks and benefits, confidentiality, and the subject's right to withdraw from the study at any time. Permission to use the patient's responses to The Renfrew Center's

routinely administered admissions survey was also included in the study's informed consent. An additional IRB-required HIPAA document was also explained at this time. The HIPAA document stated that by signing, the individual was allowing the investigator to review her medical chart in order to verify specific study-related information, including age, diagnosis, and any medications that the patient was taking.

Those subjects who were ages 18 or older signed the consent form and the HIPAA document following the informed consent procedure. Those subjects who were under the age of 18 were told that parental consent was required and were asked to sign assent forms. With permission from the subject, the investigator called the parents, explained the study, and obtained verbal consent on the phone. Written consent was subsequently obtained from parents when they came to visit the facility.

Following the acquisition of consent or assent/parental consent, the subject was assigned a study identification number and was scheduled to return for data collection during a time that did not interfere with treatment activities. The medical chart was then reviewed to record medications and to verify that the patient was eligible for the study. Demographic data and subject responses to the Eating Disorders Inventory and Eating Disorders Examination were obtained from The Renfrew Center's admissions survey, which all newly admitted patients complete during their first three days at the facility.

Each subject was seen individually for data collection, in which she met with the investigator in a private room at The Renfrew Center. Each data collection session lasted approximately one hour. Subjects were administered three psychological tests and completed two self-report questionnaires. Measures were administered in counter-balanced order.

Upon completion of the tasks, each subject was offered a choice of compensation: either an immediately available Best Buy gift card for $5 or a Best Buy gift card for $10 that the subject was told would be sent to her in the mail approximately one month following the date of her participation. Subjects' choice of prize was used as a measure of the ability to delay gratification (see Appendix E). It was hypothesized that those subjects who could wait for their prize were engaging in behavioral inhibition, forethought, and planning, which is contrary to the hypothesized impulsivity underlying bulimia.

Measures

The Wechsler Abbreviated Scale of Intelligence (WASI) was used for cognitive screening. The WASI is an individually administered, standardized psychological test of cognitive ability that is designed for use with individuals between the ages 6 and 89 years (Wechsler, 1999). The normative data are composed of a nationally representative sample of 2,245 individuals, stratified according to the 1997 U.S. census data for variables of age, sex, ethnicity, level of education, and geographic region. External reviewers have indicated that the WASI is a reliable and valid brief assessment of cognitive functioning (Hays, Reas, & Shaw, 2002; Homack & Reynolds, 2007) that can be used as a cognitive screening instrument for research (Stano, 2004). The WASI provides age-based normative data to convert raw scores into T-scores for individual subtests and standard scores for composite indexes, which allows for comparisons of abilities to be made across ages. The WASI consists of four subtests; however, two subtests, Vocabulary and Matrix Reasoning, were used for the purposes of screening (see

Appendix C). All standardized administration and scoring procedures were followed as indicated in the test manual (Wechsler, 1999).

The Vocabulary subtest of the WASI is composed of 42 questions requiring oral definitions of words of increasing difficulty. The four easiest items contain pictures, and items 5-42 are words. The subjects' responses were recorded verbatim and scored on a scale of zero, one, or two points according to predetermined criteria outlined for each item, zero being completely wrong, one being a partially correct definition, and two being a correct and fully complete definition. Each subject continued to answer questions until she reached a ceiling of five consecutive scores of zero.

The Matrix Reasoning subtest of the WASI is composed of visual matrixes from which a section is missing and that requires a response be chosen from five options in order to complete a pattern, classification, analogy, or serial reasoning item. There are 35 items of increasing difficulty which are scored either as correct or incorrect. Items involve showing the subject a picture of a matrix with an empty box and asking the subject to point to the response option that completes the matrix.

Raw scores from the WASI Vocabulary and Matrix Reasoning subtests are converted to T-scores, which is a standardized score with a mean of 50 and standard deviation of 10. These T-scores are then combined and converted into the 2-Subtest Full-Scale IQ composite score, which is an age-based standard score with a mean of 100 and a standard deviation of 15. Participants with a composite score below 70 on the WASI 2-Subtest Full-Scale IQ screening were to be excluded from the study in order to ensure that any conclusions about executive function deficits were not confounded by the presence of general cognitive impairment;

however, none of the participants scored below 70 on the WASI 2-Subtest Full-Scale IQ and therefore none were excluded from the study on the basis of this criterion.

The Delis-Kaplan Executive Function System (D-KEFS) is a standardized psychological instrument that is composed of nine different subtests that assess higher-level cognitive functions in people ages 8 to 89 (Delis, Kaplan, & Kramer, 2001). The normative data are composed of a nationally representative sample of 1,750 non-clinical individuals, stratified according to the 2000 U.S. census data for variables of age, sex, ethnicity, level of education, and geographic region. External reviewers have confirmed the validity and reliability of the D-KEFS subtests (Homack, Lee, & Riccio, 2005; Shunk, Davis, & Dean, 2006). The D-KEFS Color-Word Interference task has been shown to be a significant predictor for daily functioning (Jefferson, Paul, Ozonoff, & Cohen, 2006) and to differentiate between control samples and disorders of impulse control such as ADHD (Wodka et al., 2008). All standardized administration and scoring procedures were used as indicated in the test manual.

The Color-Word Interference subtest of the D-KEFS was used to measure response disinhibition. The D-KEFS Color Word Interference task is based on the classic Stroop procedure (Stroop, 1935). The primary executive function measured by this task is the ability to inhibit the pre-potent (automatic) verbal response of reading a printed word in order to generate a conflicting response of naming the dissonant ink color in which the word is printed. The D-KEFS Color-Word Interference Test consists of four trials. The first two are baseline measures of simple word reading and color naming. The third trial is the traditional Stroop task in which words of colors (e.g., red, blue, yellow) are presented in dissonant colored ink and the participant must name the color of the ink. For the fourth trial, the subject has to switch between naming the

color of the word and reading the word, adding cognitive flexibility and working memory demands to the task.

Various scores can be obtained from the D-KEFS Color-Word Interference task. For this study, the subjects' speed of task completion was timed and recorded for each of the four trials and was also compared against age-corrected normative data, thereby generating two variables for analyses: a raw score (e.g., time in seconds) and a standard score (see Appendix C). The number of self-corrected and uncorrected errors was also recorded for each trial, and these raw frequency data were used in analyses. Errors on the third and fourth trials (e.g., classic Stroop and switching Stroop) were also compared against age-corrected normative data to generate standard scores. Because errors on the first and second trials of the Color-Word Interference task (e.g., color naming and word reading) are uncommon, normally-distributed standardization data for errors on the first and second trials are not available and standard scores could not be generated.

The Behavior Rating Inventory of Executive Function – Self Report for adolescents aged 11 to 17 (BRIEF-SR) and the Behavior Rating Inventory of Executive Function – Adult version for adults aged 18 to 89 (BRIEF-A) were administered as a dependent measure (Guy, Isquith, & Gioia, 2004a; 2004b) (see Appendix D). The BRIEF rating scales were designed to be ecologically valid measures of executive functioning in everyday life (Gioia & Isquith, 2004), which many agree is a necessary supplement to standard neuropsychological assessment (Manchester, Priestley, & Jackson, 2004). The normative data are composed of nationally representative samples of non-clinical individuals, stratified according to the 2000 U.S. census data for variables of age, sex, ethnicity, level of education, and geographic region. Acceptable psychometric qualities for reliability and validity have been reported (Guy, Isquith, & Gioia,

2004a; 2004b). External reviewers have also indicated that the BRIEF rating scales demonstrate good reliability, (Malloy & Grace, 2005), construct validity with other executive functioning measures (Sullivan & Riccio, 2006), convergent and discriminant validity as a measure of frontal lobe functioning (Mahone, Martin, Kates, Hay, & Horska, 2009), and utility in clinical work and in research (Walker & D'Amato, 2006).

The BRIEF-SR is composed of 80 items within eight subscales: Inhibit, Shift, Emotional Control, Monitor, Working Memory, Plan/Organize, Organization of Materials, and Task Completion. The BRIEF-A is composed of 75 items within nine subscales: Inhibit, Shift, Emotional Control, Self-Monitor, Initiate, Working Memory, Plan/Organize, Task Monitor, and Organization of Materials. Both versions of the BRIEF contain composite scores representing a Behavioral Regulation Index, Metacognition Index, and the Global Executive Composite. Validity indexes that are generated include Inconsistency and Negativity.

The primary variable from the BRIEF under investigation is the Inhibit scale. This is a self-report measure of inhibitory control, or the ability to inhibit, resist, or not act on an impulse, as well as the ability to stop one's own behavior when appropriate. The BRIEF-SR Inhibit scale is composed of 13 questions. The BRIEF-A Inhibit scale is composed of 8 questions. The age-appropriate versions were administered and compared against normative data to determine age-corrected standard scores.

In order to assess self-perceptions of risk-taking behavior, the three-item Type-T Survey was administered (see Appendix E). This questionnaire asks the participants to rate themselves on a Likert-type scale from 1 to 10 on their tendency to engage in risky behavior. Item 1 asks "Are you an excitement-seeker/thrill-seeker?" Item 2 asks "Do you get most of your excitement internally (in your head, from fantasy, thoughts, etc.) or externally (from the things you do, your

behavior)?" Completely internally was represented as 1 on the Likert scale, and completely externally was represented as 10. Item 3 asks "Are you a risk-taker?" Risky behavior is highly correlated with trait impulsivity, and therefore scores from this survey were considered a third dependent measure of impulsivity.

The Eating Disorders Inventory, Third Edition (EDI-3; Garner, 2004) and Eating Disorders Examination – Questionnaire (EDE-Q; Fairburn & Beglin, 1994) are self-report questionnaires that measure the typology and severity of eating disorder symptoms (see Appendix B). Both scales are commonly used in research with eating disordered populations and both have been shown to have acceptable psychometric qualities of reliability and validity (Binford, le Grange, & Jellar, 2005; Cumella, 2006; Peterson et al., 2007; Sysko, Walsh, & Fairburn, 2005)

The EDI-3 is a standardized norm-referenced measure with both clinical and non-clinical normative samples. The normative samples that were used for this study were composed of clinical adult (ages 18 to 53) and adolescent (ages 13 to 17) eating disordered patients diagnosed with bulimia that were in inpatient or outpatient treatment. The EDI-3 consists of 91 items organized onto 12 subscales, including three eating-disorder-specific scales (Drive for Thinness, Bulimia, and Body Dissatisfaction) and nine psychological scales (Low Self-Esteem, Personal Alienation, Interpersonal Insecurity, Interpersonal Alienation, Interoceptive Deficits, Emotional Dysregulation, Perfectionism, Asceticism, and Maturity Fears). The three validity indexes that are generated are the Inconsistency, Infrequency, and Negative Impression indexes. The primary variable of interest for the present study is the Bulimia scale, which is composed of eight items that assess concerns about bingeing and eating in response to emotions. The test's manual states that the Bulimia scale reliably differentiates patients with bulimia from those with anorexia, is

stable over time, and predicts the onset of eating problems. The items on the bulimia scale include the following, for which the respondent must answer if the item is true about herself always, usually, often, sometimes, or never: I eat when I am upset, I stuff myself with food, I have gone on eating binges where I felt that I could not stop, I think about bingeing (overeating), I eat moderately in front of others and stuff myself when they're gone, I have thought of trying to vomit in order to lose weight, I eat or drink in secrecy, and When I am upset, I worry that I will start eating. Higher scores reflect worse symptomatology.

The EDE-Q provides frequency data on key behavioral features of eating disorders. It also provides index scores for the severity of the disorder according to Restraint, Eating Concern, Shape Concern, and Weight Concern. Community based normative data are available, based on a sample of 285 non-eating disordered women between ages 16 and 35 in rural and suburban regions of England (Fairburn & Beglin, 1994). For this study, the items that generate frequencies of bulimia-specific behaviors were used. Items 13 - 18 are all questions about the frequency of symptoms over the past 28 days. Item 13 asks the total number of binges that occurred, Item 14 asks the total number of times feeling out of control during binges, Item 15 asks the number of days out of the last 28 days on which bingeing occurred, Item 16 asks the total number of times that vomiting purge episodes occurred, Item 17 asks the total number of times laxatives were used, and Item 18 asks the total number of compulsive exercise episodes that occurred.

Additional data were obtained for comorbid diagnoses, medication usage, and severity of illness variables (see Appendix A). Data accessed from the medical charts included comorbid diagnoses and the number and type of medications being taken. Data accessed from The Renfrew Center's admissions database included frequency and type of self-harm, age of eating

disorder onset, number of previous hospitalizations, and length of stay in treatment at The Renfrew Center, which was calculated by research personnel after the patients had discharged. Additionally, percentage of ideal body weight (IBW) at admissions was calculated by nursing staff at The Renfrew Center and entered in the admissions survey database. Percent IBW is a measure of the extent an individual is over- or under-weight based on their body mass index. A person with 100% IBW means her weight is exactly average for a healthy person of similar height and age.

CHAPTER 4

RESULTS

In order to analyze the results, the following statistical analyses were used: descriptive statistics, including frequency, range, median, standard deviation, and mode; inferential statistics, including the z-test and the t-test; and correlational analyses. When the assumptions for parametric testing were not met, such as when the Levene test for the homogeneity of variance or the Shapiro-Wilk test of normality were significant, a nonparametric test was used. The Mann-Whitney U-Test was used as a nonparametric equivalent to the t-test, and the Spearman Rank Order correlation was used as a nonparametric equivalent to the Pearson correlation. Where indicated, effect sizes were calculated for Cohen's *d*, in which the mean of the control group was subtracted from the mean of the treatment group, and this difference was divided either by the standard deviation of the control group for t-tests and Mann-Whitney U-tests, or by the pooled standard deviation of both groups for independent samples t-tests.

The Shapiro-Wilk test of normality revealed that the following variables were not normally distributed: previous hospitalizations, frequency of self-harm, EDE-Q items 13-18, number of self-corrections on the WASI Matrix Reasoning subtest, D-KEFS Trial 4 raw score completion time, and D-KEFS raw score self-corrected and uncorrected errors on Trials 1-4. As might be expected from a clinical sample, the responses to the EDE-Q questionnaire were negatively skewed, indicating that as a whole, the sample reported high frequencies of bulimia symptoms. The kurtosis of the D-KEFS variables of raw score completion times and errors revealed very pointy distributions, with much of the variance explained by a few scores that were drastically higher than the others.

Descriptive statistics were used to characterize the sample according to comorbid diagnoses, self-harm, medication usage, and other severity of illness variables. Rates of self-harm and comorbidity for this sample were generally consistent with the rates for the total bulimia patient population at The Renfrew Center from April through December of 2008 (O'Planick, 2009) as well as reports in the literature of the inpatient bulimia population (Fichter, Quadfleig, & Hedlund, 2008) (see Table 4.1 and Table 4.2).

Table 4.1: Self-Harm Behaviors

	N	Percent of Sample
Frequency of Self-Harm Behavior		
Several times per year	9	32
Several times per month	4	14
Several times per week	1	4
Type of Self-Harm Behavior		
Cutting	13	46
Scratching	5	18
Bruising	2	7
Burning	2	7
Overdose on pills	1	3
Pull out hair	2	7

* Percentages rounded to nearest whole number.

Table 4.2: Comorbid Diagnoses

	N	Percent of Sample
Mood Disorders	23	82
Major Depression	12	43
Bipolar I	1	4
Bipolar II	2	7
Bipolar – NOS	7	25
Depressive Disorder – NOS	1	4
Anxiety Disorders	5	18
Generalized Anxiety Disorder	3	11
PTSD	2	7
Substance Abuse Disorders	8	29
Alcohol Abuse	4	14
Substance Abuse	1	4
Polysubstance Abuse	3	11
Personality Disorder – NOS with borderline features	1	4
Sexual Arousal Disorder	1	4

* Percentages rounded to nearest whole number.

Medical charts were reviewed to determine the number and types of medications that each subject was taking (see Table 4.3). Fifty-seven percent of the sample was taking at least one psychotropic medication subsequent to admitting to The Renfrew Center. Hence, 43% of the sample was not taking any medications. Of those subjects who were taking medications, 28% were taking only one medication, 21% were taking two, 4% were taking three, and 4% were taking four. With the exception of one individual, all medicated subjects were taking some form of an anti-depressant.

SSRIs were the most commonly encountered class of medication, including 21% taking fluoxetine (Prozac), 11% taking sertraline hydrochloride (Zoloft), 11% taking escitalopram (Lexapro), and 7% taking venlafaxine (Effexor), which is also a norepinephrine reuptake inhibitor. Anti-psychotic medications were also common, including 11% taking quetiapine (Seroquel) and 4% taking ariprapazole (Abilify), as were anti-epileptic medications with known efficacy in treating bipolar disorder, which included 11% taking lamotrigine (Lamictal) and 7% taking topiramate (Topamax). Benzodiazepine anti-anxiety medications were also found, including 7% taking clonazepam (Klonopin) and 4% taking alprazolam (Xanax). Buspirone (Buspar), which is a non-sedative anti-anxiety medication, was found among 4%. Zolpidem (Ambien), which is used to treat insomnia, was also found among 4%.

Table 4.3: Medications

	N	Percent of Sample
Alprazolam (Xanax)	1	4
Ariprapazole (Abilify)	1	4
Buspirone (Buspar)	1	4
Clonazepam (Klonopin)	2	7
Escitalopram (Lexapro)	3	11
Fluoxetine (Prozac)	6	21
Lamotrigine (Lamictal)	3	11
Quetiapine (Seroquel)	3	11
Sertraline (Zoloft)	3	11
Topiramate (Topamax)	2	7
Venlafaxine (Effexor)	2	7
Zolpidem (Ambien)	1	4

* Percentages rounded to nearest whole number

Additional severity of illness variables were calculated, including age of onset, number of previous hospitalizations, length of inpatient stay at The Renfrew Center, and percent ideal body weight (IBW) at the time of admissions (see Table 4.4). The self-reported age of onset for eating disorder symptoms ranged from 10 to 19 years and the modal age of onset was 13 years old. Self-reported number of previous hospitalizations ranged from 0 to 15 and the modal number of previous hospitalizations was zero. The average length of stay for inpatient treatment at The Renfrew Center was 26 days. The average percent ideal body weight (IBW) for the sample was 101% and ranged from 77% to 141%. These findings are consistent with the severity of illness variables found among the total bulimia patient population at The Renfrew Center during the time of data collection (O'Planick, 2009).

Table 4.4: Severity of Illness Variables

	Range	Mean (SD)	Mode
Age of Eating Disorder Onset	10-19	14 (2)	13
Number of Previous Hospitalizations	0-15	1 (3)	0
Length of Inpatient Stay	8-50	26 (9)	28
Percent IBW	77-141	101 (15)	88

Analyses were conducted to determine if the study sample was representative of the clinical eating disorder population (see Table 4.5). One-sample z-tests were conducted because the population means and standard deviations are known (e.g., normative data for the EDE-Q and EDI-3 are available). The sample was expected to differ from the non-eating disordered, community-based normative data for eating disorder symptoms on the EDE-Q. As expected, the results indicate that the sample differed significantly from the non-eating disordered, community-based normative sample for eating disorder symptoms on the EDE-Q. The sample reported significantly greater eating disorder symptomatology, with large effect sizes found for all indexes compared to the EDE-Q normative data: Global Scale (d = 2.22), Restraint (d = 2.23), Eating Concerns (d = 6.14), Shape Concerns (d = 2.21), and Weight Concerns (d = 2.57). The scores of this sample on the EDE-Q were also found to be consistent with the total bulimia patient population at Renfrew during the time of this study.

The sample was not expected to differ from the clinical bulimia normative sample for eating disorder symptoms on the EDI-3. Unexpectedly, adult subjects age 18 and older reported significantly greater drive for thinness and body dissatisfaction as compared to the adult patient normative data, with a medium effect size found for drive for thinness (d = .57) and a small effect size found for body dissatisfaction (d = .33). Adolescent subjects younger than age 18 did not report significant differences compared to the adolescent patient normative sample. It is probable that these results were found because the normative sample of the EDI-3 is derived from the outpatient population, while the sample in this study is currently inpatient. Worse symptomatology would be expected among an inpatient sample compared to an outpatient sample. Indeed, the scores of this sample were consistent with the admissions EDI-3 scores for the total bulimia patient population during the time of this study.

Table 4.5: z-tests for Eating Disorder Measures

	Mean (SD)		
	Test Norms	Sample	*p*
EDE-Q (compared to general population norms)			
Global Scale	1.554 (1.21)	4.813 (.795)	.000***
Restraint Scale	1.251 (1.32)	4.200 (1.76)	.000***
Eating Concerns	0.624 (0.62)	4.453 (.613)	.000***
Shape Concerns	2.149 (1.60)	5.514 (.672)	.000***
Weight concerns	1.587 (1.36)	5.076 (.896)	.000***
EDI-3 (compared to clinical bulimia adolescent and adult patient populations)			
Drive for Thinness Subscale			
Adolescents	20.72 (6.80)	25.00 (4.35)	.231
Adults	21.49 (5.68)	24.58 (3.32)	.000***
Bulimia Subscale			
Adolescents	16.59 (8.93)	8.33 (4.50)	.087
Adults	20.40 (7.27)	18.70 (8.03)	.313
Body Dissatisfaction Subscale			
Adolescents	28.82 (10.0)	32.67 (4.16)	.251
Adults	29.92 (9.10)	33.00 (6.60)	.032*

EDI-3: Adolescents n = 3; Adults n = 25

*Sig. at .05 level

***Sig. at .001 level

For general cognitive ability, the sample was not expected to differ from the general population on the WASI. Individual subtests (e.g., Vocabulary and Matrix Reasoning) were compared against the normal population using T-scores (mean of 50, standard deviation of 10) and the 2-Subtest Full-Scale IQ was compared against the normal population using standard scores (mean of 100, standard deviation of 15). As expected, the sample's performance on the WASI Matrix Reasoning subtest did not differ from the general population. However, the sample's 2-Subtest Full-Scale IQ and Vocabulary performance did differ significantly from that of the general population. A medium effect size was found for 2-Subtest Full-Scale IQ (d = .53) as well as for Vocabulary (d = .67) (see Table 4.6).

The sample performed better on the Vocabulary subtest than was expected, which subsequently impacted their overall performance on the 2-Subtest Full-Scale IQ. The Vocabulary score is generally an index of crystallized ability, or factual knowledge based on exposure, learning, and culturally relevant experience. The demographic characteristics of this sample are not identical to the demographics of the WASI normative sample, which is a stratified sample of the national population according to U.S. census data. It is possible that this sample, composed of primarily Caucasian, well-educated, employed, treatment-seeking females, is functioning at a higher cognitive level than the general population.

Table 4.6: z-tests for WASI Cognitive Screening Measure

	Mean (SD)			
WASI Scale	Test Norms	Sample	*p*	*d*
2-Subtest Full-Scale IQ	100 (15)	108 (12.62)	.002**	.53
Vocabulary Subtest	50 (10)	56.57 (9.48)	.001***	.67
Matrix Reasoning Subtest	50 (10)	51.57 (6.87)	.237	-

**Sig at .01 level

***Sig. at .001 level

A series of one-sample z-tests was performed to answer the first research question (Does this sample of young women with bulimia differ significantly from the normative sample reflecting the general population on measures of impulsivity?). The D-KEFS and BRIEF raw data were transformed into standard scores so these variables were able to meet the assumptions for parametric testing. This was confirmed using the Shapiro-Wilk test of normality. The normative sample for the D-KEFS and BRIEF are assumed proxies for the population means and standard deviations, indicating that a z-test is an appropriate analysis.

The sample was not expected to differ from the general population on the first two trials of the D-KEFS Color-Word Interference test because these trials do not require any executive functioning skills and merely reflect speed of color naming and reading. The sample was expected to differ from the general population on the second two trials of the D-KEFS because these trials do require executive functioning skills, specifically inhibition of automatic processing on Trial 3 (classic Stroop) and cognitive flexibility as well as working memory on Trial 4 (switching Stroop).

As expected, the treatment group did not differ significantly from the normative sample on speed of color naming (Trial 1 Completion Time). Unexpectedly, the treatment group was found to differ significantly from the normative sample for speed of reading (Trial 2 Completion Time), with faster reading speed demonstrated by the treatment group; however, the effect size was small (d = .27). The treatment group was also found to perform significantly faster than the normative sample on the classic Stroop (Trial 3 Completion Time), which was opposite from the expected result. Again, the effect size for this difference was found to be small (d = .41). The treatment group did not differ from the normative sample for speed of task completion on the

switching Stroop (Trial 4 Completion Time) or for total number of errors on Trials 3 or 4 (see Table 4.7).

The results of the D-KEFS Color Word test are inconsistent with the hypothesis that individuals with bulimia will demonstrate poor response inhibition. It was expected that this sample would demonstrate longer task completion times on the Stroop task due to interference from automatic processing and would make a greater number of disinhibited errors. Rather, this sample performed faster on the classic Stroop than the general population and demonstrated a similar number of errors.

Table 4.7: z-tests for D-KEFS Color-Word Interference Task

	Mean (SD)			
	Test Norms	Sample	*p*	*d*
Completion Time				
Trial 1	10 (3)	9.964 (2.42)	.938	-
Trial 2	10 (3)	10.82 (2.00)	.039*	.27
Trial 3	10 (3)	11.25 (2.53)	.015*	.41
Trial 4	10 (3)	10.57 (2.39)	.218	-
Total Errors				
Trial 3	10 (3)	9.714 (2.41)	.537	-
Trial 4	10 (3)	10.25 (1.83)	.468	-

*Sig. at .05 level

For the BRIEF rating scales, a total of 25 subjects age 18 and older completed the BRIEF-A, and a total of 3 subjects younger than age 18 completed the BRIEF-SR. The treatment group was expected to differ from the normative sample across all subscales. The Inhibit scale, in particular, was expected to be elevated in comparison to the normative sample.

The results of one-sample z-tests show that adults and adolescents self-reported considerable executive functioning difficulties across behavioral and metacognitive domains. Specifically, adults age 18 and older had significantly higher scores compared to the BRIEF-A normative sample with large effect sizes found for the following scales: Inhibit (d = 1.62), Shift (d = 2.06), Emotional Control (d = 1.50), Initiation (d = 1.38), Working Memory (d = 1.44), Plan/Organize (d = 1.03), and Task Monitor skills (d = 1.08). The Self-Monitor scale was found to have a medium effect size (d = .75). The only domain not found to have significantly higher scores than the BRIEF-A normative sample was Organization of Materials (see Table 4.8).

Adolescents younger than age 18 reported significantly greater problems than the BRIEF-SR normative sample for Task Completion index. While it appears that the adolescents reported less executive function difficulties than the adults, the base rate for such difficulties is lower in adulthood, so even if the adolescent and adult groups were reporting similar absolute levels of difficulties, the adult group might be expected to look “worse” compared to the adolescent group. However, the interpretability of these results is limited because of the small adolescent sample size. It is unlikely that these data are reliable or have sufficient variance for the results to be meaningful. Normality of the distribution cannot be assumed for such a small sample, thereby limiting the applicability of the z-test. Interpretation of these results is further complicated by measurement differences between the BRIEF-A and BRIEF-SR.

Table 4.8: z-tests for BRIEF-A Rating Scale

	Mean (SD)			
Subscale	Test Norms	Sample	*p*	*d*
Inhibit	50 (10)	66.24 (11.7)	.000***	1.62
Shift	50 (10)	70.60 (11.8)	.000***	2.06
Emotional Control	50 (10)	65.08 (9.49)	.000***	1.50
Self-Monitor	50 (10)	57.48 (11.5)	.004**	.75
Initiate	50 (10)	63.84 (12.3)	.000***	1.38
Working Memory	50 (10)	64.44 (13.4)	.000***	1.44
Plan/Organize	50 (10)	60.36 (10.4)	.000***	1.03
Task Monitor	50 (10)	60.88 (13.1)	.000***	1.08
Organization of Materials	50 (10)	53.60 (11.7)	.138	-
Behavioral Rating Index	50 (10)	68.12 (11.2)	.000***	1.81
Metacognition Index	50 (10)	62.24 (11.6)	.000***	1.22
Global Executive Composite	50 (10)	65.9 (11.3)	.000***	1.59

**Sig. at .01 level

***Sig. at .001 level

Table 4.9: z-tests for BRIEF-SR Rating Scale

	Mean (SD)			
Subscale	Test Norms	Sample	*p*	*d*
Inhibit	50 (10)	66.33 (12.7)	.156	-
Shift	50 (10)	67.33 (15.5)	.192	-
Emotional Control	50 (10)	61.66 (11.0)	.208	-
Monitor	50 (10)	53.66 (7.63)	.493	-
Working Memory	50 (10)	75.00 (12.1)	.070	-
Plan/Organize	50 (10)	67.33 (15.5)	.192	-
Organization of Materials	50 (10)	81.00 (†)	†	-
Task Completion	50 (10)	71.33 (8.50)	.049*	1.66
Behavioral Rating Index	50 (10)	66.6 (11.37)	.126	-
Metacognition Index	50 (10)	77.3 (11.8)	.057	-
Global Executive Composite	50 (10)	76.3 (12.2)	.065	-

† = could not be computed because the standard deviation was 0

*Sig. at .05 level

To answer the second research question (Do levels of impulsivity correlate with symptom severity?), Spearman nonparametric correlations were performed using measures of eating disorder symptom severity and measures of impulsivity. First, inter-correlations were analyzed for variables of illness severity and bulimia, as well as for variables of impulsivity, in order to characterize patterns of responding.

Some aspects of severity of illness and bulimia symptoms showed significant correlations. Significant positive correlations were found for the frequency of binge episodes (EDE-Q 13), frequency of feeling out of control during binges (EDE-Q 14), and number of days per month on which binges had occurred (EDE-Q 15). Significant positive correlations were found for frequency of vomiting purge episodes (EDE-Q 16), frequency of binge episodes (EDE-Q 13), and frequency of feeling out of control during binges (EDE-Q 14). Frequency of compulsive exercise (EDE-Q 18) was not correlated with other bulimia symptoms, although a significant negative correlation did exist for compulsive exercise and number of previous hospitalizations. This suggests that among this sample, compulsive exercise is not a characteristic of bulimia or severity of illness. Significant positive correlations were found for the Bulimia scale of the EDI-3 and frequency of binge episodes (EDI-Q 13) and days per month on which binges had occurred (EDE-Q 15). Frequency of laxative use (EDE-Q 17), length of stay at The Renfrew Center, and frequency of self-harm episodes were not significantly correlated with any other bulimia symptoms or severity of illness variables.

Table 4.10: Nonparametric Correlations between Severity of Illness and Bulimia Symptoms

	1	2	3	4	5	6	7	8	9	10
1. EDE-Q 13	-	.87**	.91**	.42*	.24	-.13	.46*	-.35	.34	.13
2. EDE-Q 14		-	.80**	.38*	.12	-.16	.36	-.29	.23	-.18
3. EDE-Q 15			-	.30	.21	-.14	.62**	-.28	.16	.09
4. EDE-Q 16				-	-.11	-.01	.16	-.23	.24	-.04
5. EDE-Q 17					-	.08	.15	-.23	.16	-.06
6. EDE-Q 18						-	-.06	.36	-.46*	.14
7. EDI-3 BN							-	-.12	.027	.13
8. LOS†								-	-.31	-.01
9. PH†									-	.32
10. FSH†										-

†LOS = Length of Stay, †PH = Previous Hospitalizations, †FSH = Frequency of Self-Harm

*Sig. at .05 level

**Sig. at .01 level

Some aspects of impulsivity were found to be correlated. Completion times for the D-KEFS Color Word Task Trials 1, 2, 3, and 4 were significantly correlated. Number of self-corrected errors on Trials 3 and 4 were significantly correlated, as were number of uncorrected errors on Trials 3 and 4. Number of self-corrected errors on Trial 3 (classic Stroop) was also correlated with number of self-corrections on the Matrix-Reasoning subtest of the WASI (r = .41, $p < .05$). Faster completion time on Trial 4 (switching Stroop) was significantly correlated with more uncorrected errors on Trial 4, consistent with the speed-over-accuracy trade-off that has been found in other studies to be characteristic of individuals with bulimia.

No significant correlations were found between the BRIEF index scores and the D-KEFS Color Word Trials 1-4 completion times or number of self-corrected errors. A significant correlation was found between frequency of uncorrected errors on D-KEFS Trial 1 and the Working Memory Index of the BRIEF-A (r = 3.90, $p <$. 05). The Type-T Survey did not correlate with any other variables of impulsivity. Within the Type-T Survey, Item 3 (Are you a risk-taker?) was significantly correlated with Item 1 (Are you a thrill-seeker?) and Item 2 (Do you get excitement internally vs. externally?), but Items 1 and 2 were not correlated.

Table 4.11: Nonparametric Correlations between Variables of Impulsivity: D-KEFS and Type-T

	1	2	3	4	5	6	7	8	9	10	11
1. D-K1 Time	-	.70**	.55**	.41*	-.33	-.31	.08	.09	.06	.02	.09
2. D-K2 Time		-	.43*	.14	-.26	-.37	-.08	.18	.21	.00	.22
3. D-K3 Time			-	.19	.20	-.07	-.01	-.21	.28	-.07	.30
4. D-K4 Time				-	-.05	.14	.19	.43*	-.18	-.15	-.16
5. D-K3 SE					-	.49**	-.02	-.17	-.07	-.21	-.02
6. D-K4 SE						-	.11	-.08	-.07	-.02	.10
7. D-K3 UE							-	.51**	-.18	.28	.05
8. D-K4 UE								-	-.27	-.10	-.12
9. T-T 1									-	.17	.67**
10. T-T 2										-	.42*
11. T-T 3											-

D-K1 Time = D-KEFS Trial 1 Completion Time, D-K2 Time = D-KEFS Trial 2 Completion Time, D-K3 Time = D-KEFS Trial 3 Completion Time, D-K4 Time = D-KEFS Trial 4 Completion Time, D-K3 SE = D-KEFS Trial 3 Self-Corrected Errors, D-K4 SE = D-KEFS Trial 4 Self-Corrected Errors, D-K3 UE = D-KEFS Trial 3 Uncorrected Errors, D-K4 UE = D-KEFS Trial 4 Uncorrected Errors, T-T 1 = Type T Survey Item 1, T-T 2 = Type T Survey Item 2, T-T 3 = Type T Survey Item 3

*Sig. at .05 level

**Sig. at .01 level

Spearman nonparametric correlations between bulimia symptoms and D-KEFS performance revealed a significant negative correlation for self-corrected errors on Trial 3 (classic Stroop) and frequency of vomiting purge episodes (EDE-Q 16). Scores on the Bulimia scale of the EDI-3 were also found to have a significant positive correlation with number of self-corrected errors on Trial 2 (word reading) as well as speed of completion time on Trial 4 (switching Stroop).

Spearman nonparametric correlations between bulimia symptoms and self-ratings on the BRIEF-A were conducted; an insufficient number of subjects (n = 3) completed the BRIEF-SR for these analyses. The findings revealed that the Bulimia scale of the EDI-3 was significantly and positively correlated with the Emotion Control, Self-Monitor, Plan/Organize, and Behavior Rating Indexes of the BRIEF-A. The Organization of Materials index was found to be negatively correlated with frequency of compulsive exercise (EDE-Q 18), suggesting that individuals with greater difficulty organizing their environments tend to engage in less frequent compulsive exercise, which is consistent with the notion of compulsive behavior associated with rigidity and over-regulation rather than disorganization and under-regulation.

Table 4.12: Nonparametric Correlations for D-KEFS Color-Word Task and Bulimia Symptoms

	EDE-Q 13	EDE-Q 14	EDE-Q 15	EDE-Q 16	EDE-Q 17	EDE-Q 18	EDI-3 BN
D-KEFS Trial 1							
Time	.045	-.134	.025	.095	.224	.108	-.083
SC Errors	-.111	-.115	-.126	-.312	.008	.056	-.288
UC Errors	.088	.058	.065	.181	.199	.019	-.007
D-KEFS Trial 2							
Time	-.017	-.095	.058	.297	.041	.170	-.176
SC Errors	.019	-.100	.158	.166	.292	.145	.419*
UC Errors	-.012	.012	.253	-.036	-.110	-.220	.265
D-KEFS Trial 3							
Time	-.240	-.323	-.206	-.099	.138	-.338	-.136
SC Errors	-.282	-.250	-.184	-.513**	-.235	-.255	-.187
UC Errors	.255	.262	.114	.242	.249	.076	.031
D-KEFS Trial 4							
Time	-.211	-.160	-.337	-.165	.180	-.107	-.501**
SC Errors	-.088	-.198	-.077	.051	-.090	-.118	.074
UC Errors	.020	.112	-.052	.262	.061	.240	-.147

*Sig. at .05 level

**Sig. at .01 level

Table 4.13: Nonparametric Correlations between BRIEF-A and Bulimia Symptoms

	EDE-Q 13	EDE-Q 14	EDE-Q 15	EDE-Q 16	EDE-Q 17	EDE-Q 18	EDI-3 BN
Inhibition	-.049	.016	.107	-.050	.090	-.127	.352
Shift	.322	.236	.373	-.054	-.118	-.012	.271
Emotion Control	-.143	-.117	-.011	-.175	-.002	-.023	.409*
Self-Monitor	-.122	-.083	.070	-.105	.007	-.136	.417*
Initiate	.133	.106	.196	.025	.032	-.089	.295
Working Memory	.120	.092	.212	-.344	.252	-.053	.252
Plan/Organize	.224	.166	.356	-.059	.039	-.310	.470*
Task Monitor	.063	.043	.149	-.066	.181	-.098	.181
Org. of Materials†	.141	.183	.175	.135	-.086	-.432*	.141
Behavior Rating Index	-.065	-.054	.116	-.070	.024	-.014	.451*
Metacognition Index	.144	.123	.255	-.109	.078	-.253	.317
GEC †	.135	.127	.285	-.114	.062	-.147	.385

*Sig. at .05 level

† Org. of Materials = Organization of Materials, GEC = Global Executive Composite.

Spearman nonparametric correlations between the Type-T Survey items and symptoms of bulimia revealed a significant positive correlation between Type-T Item 2 (Do you get excitement internally vs. externally?) and frequency of binge episodes (EDE-Q 13), frequency of feeling out of control during binges (EDE-Q 14), and number of days per month on which binges had occurred (EDE-Q 15). Type-T Item 3 (Are you a risk taker?) was significantly correlated with frequency of vomiting purge episodes (EDE-Q 16).

Spearman nonparametric correlations were conducted for all variables of impulsivity and severity of illness variables, including length of stay at The Renfrew Center, number of previous hospitalizations, and frequency of self-harm. The only significant correlation found was number of previous hospitalizations and completion time on the D-KEFS Trial 3 ($r = .39$, $p < .05$).

Table 4.14: Nonparametric correlations between Type-T Survey Items and Bulimia Symptoms

	EDE-Q 13	EDE-Q 14	EDE-Q 15	EDE-Q 16	EDE-Q 17	EDE-Q 18	EDI-3 BN
Type-T Item 1	-.269	-.324	-.148	-.020	-.104	-.145	.243
Type-T Item 2	.409*	.438*	.471*	.251	.123	-.035	.265
Type-T Item 3	.050	.040	.112	.414*	-.024	-.314	.244

*Sig. at .05 level

To answer the third research question (Does this sample show a preference for immediate versus delayed gratification?), each participant's choice of prize was coded as either 1 for choosing the immediate prize or 2 for choosing the delayed prize. The independent samples t-test and the Mann-Whitney U-test were used to analyze if subjects who chose the immediate prize (n = 7) differed significantly from the subjects who chose delayed prize (n = 21) according to variables of impulsivity and bulimia symptoms.

Results indicate that very few differences were found between these two groups. The t-test showed that the immediate prize group reported significantly higher rates of seeking external excitement on the Type-T Item 2 (mean score = 8.85, SD = 1.34) than the delayed prize group (mean score = 6.04, SD = 2.20), with a large effect size found (d = 1.58). The Mann-Whitney U-test showed that the immediate prize group demonstrated slower completion time on Trial 4 (switching Stroop) of the D-KEFS Color Word task (mean completion speed = 74.2 seconds, SD = 46.9) than the delayed prize group (mean completion speed = 52.7 seconds, SD = 7.68), with this difference approaching statistical significance (p = .07).

Additional analyses were conducted to determine if comorbidity affected the results of this study. Spearman nonparametric correlations revealed that greater number of comorbid diagnoses was significantly correlated (r = .378, $p < .05$) with elevated ratings on the Bulimia scale of the EDI-3. The results of Mann-Whitney U-tests revealed that subjects with a comorbid bipolar disorder engaged in fewer episodes of compulsive exercise over the past month (EDE-Q 18 mean score = 3.78, SD = 1.67) than did subjects without a comorbid bipolar disorder (EDE-Q 18 mean score = 11.89, SD = 10.2), with a p-value approaching statistical significance (p = .059). Subjects with a comorbid substance abuse disorder were found to have significantly greater frequency of bingeing over the past 28 days (EDE-Q 15 mean score = 21.38, SD = 10.9,

$p < .05$) than subjects without a comorbid substance abuse disorder (EDE-Q 15 mean score = 11.90, SD = 11.6), with a large effect size found ($d = .81$). Additionally, independent samples t-tests revealed that subjects with a comorbid substance abuse diagnosis rated themselves higher on Item 1 of the Type-T survey (mean score = 7.8, SD = 1.3), which asks "Are you a thrill seeker?" than subjects without a comorbid substance abuse diagnosis (mean score = 6.3, 1.7), with a p-value approaching significance ($p = .054$).

When evaluated according to the defition of multi-impulsivity (Fichter et al., 1994), three subjects met criteria as multi-impulsive by having all three of the following characteristics: comorbid diagnosis of substance abuse, comorbid diagnosis of bipolar disorder, and also engaging in self-harm. Independent samples t-tests revealed that the multi-impulsive group had a significantly shorter average length of stay in treatment (mean LOS = 17 days, SD = 10, $p < .05$) than the non-multi-impulsive group (mean LOS = 27.8 days, SD = 8.4), with a large effect size found ($d = 1.28$). Additionally, when equal variance was not assumed, the multi-impulsive group also engaged in significantly less frequent compulsive exercise over the past month (mean = 1.67, SD = 1.5, $p < .001$) than the non-multi-impulsive group (mean = 10.2, SD = 10.5), with a large effect size found ($d = .81$).

Eight subjects were found to meet two out of three criteria for multi-impulsivity. The Mann-Whitney U-test revealed that this sub-clinical multi-impulsive group engaged in more frequent bingeing over the past month (mean = 44.6, SD = 55.4) than the non sub-clinical multi-impulsive group (mean = 14.7, SD = 19.5), and this difference was trending towards significance ($p = .06$). Taken together, these findings support the construct of a multi-impulsive cohort that is distinct from other individuals with bulimia.

Medication effects were also analyzed. The results show that no differential medication effects were found for cognitive task performance on the WASI or D-KEFS. However, medication effects were found for self-report. According to Spearman nonparametric correlations, the number of medications was inversely related to problems with Inhibition (r = -.407, $p < .05$) and Self-Monitoring (r = -.427, $p < .05$), on the BRIEF-A, suggesting that subjects who were taking more medications were experiencing fewer problems in these domains. Independent samples t-tests revealed that individuals taking at least one medication (n = 16) rated themselves significantly lower on Item 1 of the Type-T survey (mean score = 6.2, SD = 1.8, $p < .05$), which asks "Are you a risk taker?," than those who were not taking any medication (mean score = 7.6, SD = 1.4), with a large effect size found (d = .87). These results indicate that subjects who were being treated with pharmacotherapy perceived themselves as experiencing less difficulty with impulse control. It is possible that pharmacological treatment of eating disorder symptoms resulted in decreased impulsivity. It is also possible that these results are due to placebo effect, in which the act of taking medications caused the individuals to believe they were experiencing less impulsivity.

Mann-Whitney U-tests were conducted to determine if performance was affected by medications with known cognitive side-effects including somnolence. Among this sample, the following drugs were identified as having potential cognitive side-effects: topiramate (Zaccara, Gangemi, & Cincotta, 2008), alprazolam (Verster & Volkerts, 2004), clonazepam (Dowd, Strong, Janicak, & Negrusz, 2002), quetiapine (Dunner, 2005), and zolpidem (Terzano, Rossi, Palomba, Smerieri, & Parrino, 2003). Results revealed that no significant differences were found based on differential effects of medications with known cognitive effects.

CHAPTER 5

DISCUSSION

Summary of Results

In response to the first research question (Does this sample of young women with bulimia differ significantly from the normative sample on standardized measures of impulsivity?), the results from two measures must be considered. First, on the D-KEFS Color Word test, the results showed that this sample did not differ from the normative sample on total number of errors or on speed of task completion for the switching Stroop, and the sample demonstrated faster performance than the normative sample on the classic Stroop. These findings are inconsistent with the hypothesis that individuals with bulimia would demonstrate poor response inhibition on the Stroop task.

Secondly, on the BRIEF rating scales, the results showed that adults and adolescents self-reported considerable executive functioning difficulties. Compared with the normative sample, the adults reported significantly higher scores for the following domains of the BRIEF-A: Inhibit, Shift, Emotional Control, Self-Monitor, Initiation, Working Memory, Plan/Organize, and Task Monitor skills. For adolescents, significantly higher scores were reported for the Task Completion domains of the BRIEF-SR as compared to the normative sample.

The finding of self-reported problems of inhibition by adults on the BRIEF-A, and more generally, problems of executive functioning by both adults and adolescents on their respective BRIEF forms, is contrary to the absence of deficient response inhibition on the D-KEFS Color Word task. This indicates that perhaps the D-KEFS Color Word task and the BRIEF rating scales are tapping into different constructs. In fact, the BRIEF rating scale was not found to

correlate significantly with any scores from the D-KEFS, except for frequency of uncorrected errors on D-KEFS Trial 1 which correlated with the Working Memory Index of the BRIEF-A. The construct of impulsivity is known to be situation or task specific (Wonderlich, Connolly & Stice, 2004), and results are often subject to how impulsivity is operationally defined, which can result in low correlations across measures (Olson, 1989).

In response to the second research question (Do levels of impulsivity correlate with symptom severity?), the results showed that some variables of impulsivity were significantly correlated to variables of bulimia symptom severity. Greater self-reported bulimia symptomatology on the EDI-3 was correlated with more self-corrected errors during word reading on the D-KEFS, slower completion time on the switching Stroop, higher ratings of seeking external excitement on the Type-T Survey, and greater rates of executive functioning problems on several domains of the BRIEF-A, including problems of Emotion Control, Self-Monitoring, and Plan/Organize skills. Greater frequency of purging was correlated with fewer self-corrected errors on the classic Stroop and higher ratings of being a risk taker on the Type-T Survey Item 3. Greater frequency of bingeing was correlated with higher ratings of seeking external excitement on the Type-T Survey. As an indicator of illness severity, greater number of previous hospitalizations was correlated with slower completion time on the classic Stroop of the D-KEFS. Taken together, in response to the second research question, these results confirm that among individuals with bulimia, worse symptoms are generally associated with greater problems of impulsivity, which is consistent with the construct of a multi-impulsive syndrome.

In response to the third research question (Does this sample show a preference for immediate versus delayed gratification?), the results revealed that most subjects preferred the delayed prize. Therefore, it cannot be concluded that this sample of women with bulimia lacked

the ability to delay gratification. However, the immediate prize group was found to have significantly slower completion time on the switching Stroop than the delayed prize group, and they also self-reported significantly higher rates of seeking external excitement on the Type-T Survey. It appears that among individuals with bulimia, a subset can be identified as having difficulty with delay of gratification as well as other characteristics of poor impulse control. Although base rate data for impulse control problems was not available for review, compared to control samples, there does not appear to be a difference between the bulimia population and the general population with regards to the presence or absence of any characteristic of impulsivity; however, among patients with bulimia there is an increased likelihood for having multiple characteristics of impulsivity (Bushnell, Wells, & Oakley-Browne, 1996; Kaltiala-Heino, Rissanen, Rimpelä, & Rantanen, 2003).

Additional analyses revealed that performance on cognitive tasks was not affected by comorbidity. However, comorbidity was associated with severity of bulimia. Increased number of comorbid diagnoses was correlated with increased symptomatology of bulimia on the Bulimia scale of the EDI-3. Findings that trended towards significance included fewer episodes of compulsive exercise among subjects with a comorbid bipolar diagnosis, and higher ratings of being thrill-seekers on the Type-T Survey among subjects with a comorbid substance abuse diagnosis. Subjects with a comorbid substance abuse disorder were found to have significantly more frequent bingeing over the past 28 days.

When analyzed for differences according to the definition of multi-impulsivity (e.g., bulimia plus three additional symptoms of impulse control, including comorbid bipolar disorder, comorbid substance abuse, and self-harm), the multi-impulsive group was found to have a significantly shorter length of stay in treatment and significantly less frequent compulsive

exercise over the past month. A sub-clinical multi-impulsive group who had two out of three additional impulsive symptoms was found to engage in significantly more frequent bingeing over the past month. Taken together, these findings support the construct of a multi-impulsive cohort that is distinct from other individuals with bulimia and who have more severe symptoms of bulimia. These findings are consistent with the literature on multi-impulsivity.

Finally, analyses revealed that performance on cognitive tasks was not affected by medication use. Greater number of medications was correlated with fewer self-reported problems of Inhibition and Self-Monitoring on the BRIEF-A. Subjects taking at least one medication rated themselves lower for being thrill-seekers on the Type-T Survey Item 1. Subjects taking medications with known cognitive side-effects had higher self-reported problems with Organization of Materials on the BRIEF-A.

Interpretation of Findings

The results of this study are interpreted within the developmental framework of the biopsychosocial model of eating disorders proposed by Southgate, Tchanturia, and Treasure (2005). This model proposes that specific neurobiological mechanisms, called endophenotypes, contribute to pre-morbid cognitive deficits in adolescence, which subsequently serves as vulnerability factors in the onset and maintenance of eating disordered behavior. Executive dysfunction has been proposed as a possible endophenotype for bulimia (Treasure, 2006), caused by deficient activity of the prefrontal cortex, limbic system, ventromedial-orbitofrontal-anterior cingulate circuits, and specific serotonin and dopamine pathways, and expressed phenotypically

as poor impulse control. The relationship between bulimia and impulsivity has been well described. Poor self-control when eating, along with the constellation of impulsive behaviors that are commonly comorbid with bulimia, has been described as a multi-impulsive syndrome (Bell & Newns, 2002; Lacey & Evans, 1986). Individuals with bulimia who present with multi-impulsivity reflect a distinct sub-sample of bulimia, characterized by more severe clinical histories and worse prognoses (Duncan et al., 2005; Myers et al., 2006; Sohlberg et al., 1989; Steiger & Bruce, 2007; Wonderlich et al., 2002).

This research study explored the relationship between eating disordered behavior and impulsivity in adolescent and young adult females with bulimia using neuropsychological measures, including the Stroop task and self-report rating scales. It was hypothesized that the under-control of behavior seen in people with bulimia is attributable, in part, to a disruption of the executive functions of the frontal lobes that regulate impulse control. It was therefore predicted that this sample would demonstrate poor performance on an executive functioning task of response inhibition, report higher rates of problems on a rating scale of executive functioning, and show a relationship between symptom severity and impulsivity.

The results of this study were not entirely consistent with predictions based on the biopsychosocial model. Overall, this sample with bulimia was not found to have unilaterally deficient impulse control in comparison to the general population. Although the sample self-reported problems of executive dysfunction and inhibition in everyday life, performance on a neuropsychological task of response inhibition did not reflect impairment. Compared to the normative sample, the sample performed faster on the classic Stroop task, performed equally on the switching Stroop task, and made an equal number of errors overall.

There are several possible reasons to explain these findings. First, the null hypothesis must be considered. It may be that bulimia is unrelated to executive dysfunction, or specifically to response disinhibition as measured by the Stroop. Although typically thought of as measuring response inhibition, Brocki and Bohlin (2004) found that Stroop performance did not load onto a disinhibition factor when neuropsychological testing of individuals with eating disorders was factor analyzed. Perhaps other domains of executive dysfunction more appropriately characterize individuals with bulimia, or perhaps other assessment tools could better identify this impairment.

According to self-report, the individuals in this sample clearly felt they struggle with problems of executive functioning in their everyday lives. Yet, they did not show associated deficits of performance on the neuropsychological tasks of executive functioning. As suggested in the literature, it is possible that these two measures, while presumed to both measure executive functioning, were tapping into different constructs. Perhaps the Stroop task was measuring an aspect of cognitive impulse control for language processing, while the BRIEF was measuring an aspect of behavioral impulse control that is socially- and contextually-mediated. Similar mixed findings have been reported in other studies investigating executive functioning in samples with bulimia due to the multi-faceted nature of the executive functions and their measures (Galderisi et al., 2003; Murphy et al., 2002, 2004; Touyz et al., 1986).

Behavioral disinhibition on cognitive tasks is not found uniformly among all disorders characterized by impulsivity (Lampe et al., 2007), suggesting that impulsivity is indeed multi-componential and subject to differential results based on choice of measurement tool (Biederman et al., 2008). Correlations between rating scales of executive function and neuropsychological tests of executive function can be weak, suggesting that each method of assessment is tapping

into separate factors (Anderson, Anderson, Northam, Jacobs, & Mikiewicz, 2002; Burgess, 2000). While the Stroop task may be tapping more into inhibition of automatic processing of verbal responses, the BRIEF rating scales may be tapping more into behavioral inhibition within an environmental and social context. Ventromedial and anterior cingulate circuits have been specifically implicated in Stroop performance; perhaps more elaborate patterns of frontal circuitry are responsible for the complex patterns of behavior demonstrated by impulsive individuals in daily functioning.

The state-trait distinction must also be considered. It is possible that the executive functioning of the sample as demonstrated on neuropsychological tasks while in a structured, stable treatment setting, may truly differ from their executive functioning in their home environments immediately prior to seeking treatment, as demonstrated by their self-reported ratings of executive dysfunction in their everyday lives. Such dysfunction may have prompted these individuals to seek treatment, and subsequently become stabilized once in the treatment setting. As forewarned by Stuss and Alexander(2000), it is relatively easy to "become the frontal lobes" of the patient by imposing external structure in the environment while they are completing an executive function task. It is interesting that medication usage was unrelated to performance on neuropsychological tasks, but it was related to self-perceived executive functioning in everyday life. It is possible that medication actually facilitated performance on neuropsychological testing. Deficient impulse control is perhaps trait-like and more typical of everyday functioning, but was effectively controlled for with medication and minimized during the patient's present state during testing.

Another important factor to consider is that the sample was not entirely characteristic of a pure sample of bulimia, as indicated by z-test analyses of the EDI-3 showing that the sample

reported significantly greater drive for thinness and body image dissatisfaction than the normative sample for the clinical bulimia patient population. The validity of interpretations of these results based on an assumed bulimic population is therefore questionable. As suggested by Vitousek and Stumpf (2005), assessment of individuals with eating disorders is especially difficult because diagnoses are unstable and within-group differences are often substantial with regard to previous eating disorder histories and current presentations. A debate exists as to the relative utility of reclassifying eating disorders according to over- versus under-control, rather than the current diagnostic schema (Dawe & Loxton, 2004). Indeed, behavioral disinhibition is seen not exclusively among eating-disordered individuals with bulimia (Rosval et al., 2006).

Additionally, the general cognitive ability of this sample was not entirely consistent with that of the general population, as indicated by z-test analyses of the WASI showing that the sample had a significantly higher verbal reasoning ability. Therefore, the validity of interpretations of these results based on an assumed normal distribution for cognitive ability is also questionable. If the sample was found to have a significantly enhanced ability in one cognitive domain, it is quite possible that other aspects of cognition, including executive functioning skills, were also enhanced. The sample demonstrated significantly faster reading speed than the normative sample on the D-KEFS Trial 2, lending further support to the hypothesis that this sample was functioning at an overall higher baseline cognitive level, thereby confounding interpretation of executive functioning performance.

Although the results of this study do not support a unilateral conclusion linking poor impulse control with bulimia, the results do indicate that within this sample of young women with bulimia, impulsive characteristics clustered together to characterize a sub-sample, consistent with a multi-impulsive syndrome. Subjects with a preference for immediate gratification

performed slower on the switching Stroop and also reported higher rates of seeking excitement externally. Subjects with comorbid diagnoses of poor impulse control, including bipolar disorders and substance abuse, had worse bulimic symptom severity and reported higher rates of thrill-seeking. Taken together, these findings confirm that among individuals with bulimia, a subset can be identified as having multiple characteristics of poor impulse control that coincide with worse bulimia symptomatology.

Additionally, the results of this study indicate that the subset of the sample characterized by multi-impulsivity also demonstrated an impulsive cognitive style. Most notably, faster completion time on the switching Stroop was correlated with more uncorrected errors. This is consistent with the tendency among this population to favor speed over accuracy that has been reported by others (Ferraro et al., 1997; Kaye, Bastiani, & Moss, 1995). Interestingly, this pattern of favoring speed over accuracy was found only on the switching Stroop and not the classic Stroop. The switching Stroop trial of the D-KEFS Color Word task is more challenging than the classic Stroop trial. It requires not only inhibition of automatic processing, but also working memory to keep the switching rule in mind, as well as cognitive flexibility to efficiently manage the constant shift between mental sets. This suggests that the impulsive cognitive style found in individuals with bulimia may be more pronounced when the brain is taxed or experiencing stress, such as when under greater cognitive load.

This impulsive cognitive style was found to be more pronounced among subjects with more severe bulimia. Subjects with greater reported bulimia symptomatology not only reported higher rates of seeking external excitement, but also made more errors during a simple task of word reading, which suggests poor response inhibition on an over-learned activity that perhaps was approached with less focused attention than the novel tasks of the switching and classic

Stroop. Indeed, these subjects were slower to complete the switching Stroop, suggesting they were using additional cognitive resources to purposefully attend to the stimuli and inhibit incorrect responding. Subjects with greater frequency of purging not only reported higher rates of being risk-takers, but also made fewer self-corrected errors on the classic Stroop, which perhaps indicates a lack of awareness or, more likely, an unwillingness to correct an error in order to achieve faster speed of completion, consistent with the speed-over-accuracy trade-off. Greater rates of self-reported bulimia symptomatology on the EDI-3 Bulimia scale was correlated with greater rates of executive dysfunction on several domains of the BRIEF-A, including problems of emotional control, self-monitoring, and planning and organization skills. As an indicator of illness severity, greater number of previous hospitalizations was correlated with slower completion time on the classic Stroop.

In conclusion, the results of this study indicate that some aspects of impulsivity are characteristic of individuals with bulimia. Problems of disinhibition and general executive dysfunction in everyday life were highly prevalent in this sample, whereas response disinhibition on cognitive tasks was not. The results of this study confirm that among individuals with bulimia, a multi-impulsive subset exists that is characterized by comorbid disorders of impulse control, inability to delay gratification, and an impulsive cognitive style. Additionally, increased severity of bulimia symptoms was associated with more severe impulsivity as well as overall executive dysfunction. Thus, the hypothesis that unilateral deficits of impulse control exist in individuals with bulimia was not substantiated. The hypothesis that a subset of individuals with bulimia present with multi-impulsivity, including response disinhibition on measures of executive functioning, was substantiated.

Limitations

A primary limitation to this study is the small sample size, which not only limited the degree of statistical power but also limits the ability to generalize these findings to the broader bulimia patient population. Additional limitations to internal and external validity must also be addressed. Limitations to internal validity affect the degree to which the results can be attributed to the research hypotheses in question, rather than to extraneous variables or error confounding the results. First, the issue of sample bias must be considered. The sample was composed of volunteers who self-selected to participate, so it is possible that only those patients who felt they would perform well volunteered. This may also have affected the retention of subjects. The three patients who withdrew from the study may have been fundamentally different from those subjects who completed the study, perhaps due to disinterest or performance anxiety. Among those who self-selected to participate, subject inclusion was dependent upon a psychiatric diagnosis given at the time of admissions; therefore, the sample may have been further biased due to variables unique to the psychiatrist, such as personal judgment and training experience, as well as situational variables unique to the time of admissions, such as the patient's current weight and the presence or absence of a regular menstrual cycle.

Minors who self-selected to participate were also confronted with the additional requirement of parental permission. It is suspected that several minors who initially showed interest in the study declined to participate once it was made evident that their parents would be contacted to obtain parental consent. This is not surprising, given that many individuals with eating disorders have disturbances in family functioning. These issues of sample bias could not be controlled for using this study design.

Issues of measurement must also be considered. Self-report of behavior can be inaccurate. It would have been preferable to obtain an informant's rating of the patient's behavior, but under the constraints of the setting and with limited research support, this was not a feasible option. Performance effects might also have confounded results, meaning that subjects might have felt compelled to perform better than normal in order to appear good to the examiner. Fatigue and performance effects were partly addressed by counter-balancing the order of the assessments.

Variables related to time of testing could also have contributed. Every effort was made to recruit and assess patients on their first weekend in treatment; however, this still allowed for considerable variability with regards to time in treatment prior to testing. For example, one subject may have been admitted for treatment on Monday and spent the whole week in treatment before being tested on Saturday, while another may have admitted on Friday and spent only one night in treatment before being tested on Saturday. Differences in adjusting to new medication, to the treatment setting, and to physiological changes (e.g., bowel irregularities, feeling bloated in response to eating without purging, etc.) may also have confounded subjects' performance. Additionally, all data collection and data entry were conducted by the student investigator; therefore, there was no inter-rater reliability and the absence of human error cannot be assumed.

Other variables aside from those under investigation may also have impacted the subjects' performance, including situational and constitutional variables. Most data collection sessions were held at similar times on weekend afternoons when there were no other treatment activities; however, a few had to be conducted during the week due to scheduling conflicts. Time of day may have differentially impacted subjects' performance, particularly in relation to their experiences on that particular day with meals, therapy sessions, or family issues that came

up during a visit. These experiences could likewise exert influence on the subjects' composure, feelings of anxiety or depression, or willingness to perform optimally. Prior to testing, all subjects were briefly asked how they were doing and how their day was going in order to assess if they were feeling prepared for the testing; however, the veracity of this information was subject to their willingness to self-disclose.

Limitations to external validity pertain to the generalizability of findings. The sample was narrowly defined in order to facilitate testing specific hypotheses regarding the relationship between bulimia and impulse control. While the inclusion of other diagnoses would provide a broader picture to understand the role of executive deficits in eating disorders, it was beyond the scope of the present study. Additionally, the sample was restricted to a convenience sample recruited from The Renfrew Center's inpatient facility because subjects were easily accessible and space for research activities was provided. The inpatient population is problematic because such individuals are, by definition, the most severely ill among this population and can be expected to show greater functional impairment as well as greater rates of comoribidity, medication use, and compromised health status than the general population. These confounding variables complicate interpretation and generalizability of findings; however, it is quite likely that an outpatient sample would have involved similar confounds, perhaps just not as severe as the inpatient sample.

Measurement restriction must also be considered. An initial battery of executive functioning tests was proposed; however, at the suggestion of the Research Committee of The Renfrew Center, it was determined that a lengthy battery would likely interfere with treatment activities and would be prohibitive for completing data collection. There are other assessment tools available for measuring general cognitive ability, executive functioning, and eating

disorders aside from the WASI, D-KEFS, EDI-3, and EDE-Q. However, these assessment instruments were selected because they demonstrate good psychometric qualities of reliability and validity, have normative samples that could be used as proxies for a control group, and are widely accepted and used in the field as measures of their respective constructs.

It is possible that the Stroop paradigm is not sufficient to capture the cognitive disinhibition of individuals with bulimia. As suggested by the findings of this study, problems of executive functioning that are experienced in the everyday environment may not be apparent on neuropsychological testing. This may necessitate researchers to use assessment instruments that are less structured and more reflective of the complex decision-making deficits of this population. As indicated by Stuss and Alexander (2000), a challenge with conducting research on executive functioning is that the structured research setting used to ensure reliability and accuracy of assessment can effectively invalidate the subjects' demonstrated executive functioning by minimizing their need to self-regulate their own behavior. Being tested one-on-one in a quiet space with directions clearly given and expectations for performance externally supplied, the subjects do not have to self-impose structure or use higher level decision-making skills. Thus, external validity of the construct is sacrificed for the sake of internal validity of measurement.

Future Directions

Research has suggested that executive dysfunction is not circumscribed to impulse control among the bulimic population (Alvarez-Moya et al., 2007), which was confirmed by the findings of this study showing that self-reported problems of executive function ranged across multiple domains. Future research may wish to expand on these findings by conducting more broad neuropsychological evaluations of different domains of executive skills in addition to impulse control. Conversely, a more thorough battery assessing multiple aspects of impulse control might provide clarification regarding the mixed findings of this study. The inclusion of additional informants aside from self-report would also enhance future research. Finally, in order to truly appreciate the neuropsychological relationship between brain and behavior, the use of brain imaging technology would be useful.

Although adolescents were targeted for this investigation, it was difficult to obtain a sufficient number of adolescents under the age of 18, thereby limiting their inclusion in certain analyses. The biopsychosocial model specifies the importance of adolescence in understanding the onset of bulimia. Therefore, future research should focus more specifically on this age group.

Additionally, the results of this study indicate that medication effects and comorbidity play a role in understanding the relationship between impulsivity and bulimia. Future research might explore this further by evaluating a group with bulimia compared to a group with bulimia and comorbid diagnoses of poor impulse control. Another avenue for further exploration is to evaluate two groups with bulimia, one taking medication and another that is medication-free. Longitudinal studies would allow for exploration of the temporal onset of symptoms of bulimia and impulse control deficits, thereby allowing for conclusions regarding risk factors.

Additionally, longitudinal studies following individuals with bulimia through treatment and back into their everyday lives would further clarify some inconsistencies in the findings regarding state-versus-trait deficits of impulse control.

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APPENDIX A

RAW DATA: COMORBIDITY, MEDICATIONS, & SEVERITY OF ILLNESS VARIABLES

Subject	Comorbid Diagnoses	Medications	%IBW	LOS	PH	FSM
1	Bipolar NOS	Lamictal, Abilify, Topamax, Zoloft	118	28	0	None
2	None	Lexapro	92	35	0	None
3	MDD	Zoloft, Buspar	88	23	1	Yearly
4	Bipolar II	Prozac, Klonopin, Xanax	100	28	0	Monthly
5	MDD, PTSD	Zoloft	106	36	0	Monthly
6	Bipolar NOS	None	118	28	0	None
7	None	None	91	23	1	None
8	MDD	Lexapro	102	28	0	None
9	Bipolar NOS, Sexual Arousal Disorder	Lamictal, Seroquel	91	28	3	None
10	MDD, Alcohol Abuse	Prozac, Seroquel	132	30	0	None
11	Bipolar NOS	Effexor XR, Ambien	107	32	2	None
12	MDD, Alcohol Abuse	None	77	29	0	None
13	MDD	None	109	28	1	Yearly
14	MDD, Poly. Abuse	None	100	14	0	None
15	Bipolar NOS	None	88	19	0	None

APPENDIX A (continued)

Subject	Comorbid Diagnoses	Medications	%IBW	LOS	PH	FSM
16	MDD, PTSD	None	141	42	0	Yearly
17	Bipolar NOS, Substance Abuse	Prozac, Lexapro	83	15	1	Yearly
18	MDD, GAD, PD NOS	Klonopin, Lamictal, Prozac	111	20	2	Monthly
19	Depressive Disorder NOS	Seroquel	82	28	6	None
20	MDD	Effexor XR	104	28	0	Weekly
21	Bipolar II, Poly. Abuse	Prozac	89	28	0	Yearly
22	None	None	88	27	0	Yearly
23	GAD, Alcohol Abuse	None	112	18	0	None
24	MDD	None	88	50	0	None
25	MDD, Alcoho Abuse	Prozac	90	40	0	None
26	None	None	124	14	0	None
27	Bipolar I, GAD, Poly. Abuse	None	113	8	5	None
28	Bipolar NOS	Topamax	101	21	15	None

NOS = Not Otherwise Specified, MDD = Major Depressive Disorder, GAD = Generalized Anxiety Disorder, Poly. Abuse = Polysubstance Abuse, PD = Personality Disorder, %IBW = Percent of Ideal Body Weight at Admission, LOS = Length of Stay in days, PH = Number of Previous Hospitalizations, FSM = Frequency of Self-Harm

APPENDIX B

RAW DATA: BULIMIA SYMPTOMS

Subject	EDE-Q 13	EDE-Q 14	EDE-Q 15	EDE-Q 16	EDE-Q 17	EDE-Q 18	EDI BN
1	0	0	0	3	0	1	13
2	7	7	7	300	0	20	14
3	35	35	25	50	0	28	22
4	28	28	28	20	28	28	26
5	1	1	1	30	0	28	3
6	0	0	0	3	0	1	13
7	26	26	26	140	8	5	26
8	25	25	25	25	0	25	28
9	20	20	20	64	0	0	20
10	56	50	28	65	0	8	17
11	0	0	0	2	0	0	23
12	28	28	28	28	0	0	26
13	2	1	0	168	0	12	8
14	7	7	7	56	15	15	15
15	9	9	28	28	0	0	28
16	2	2	2	7	25	15	16
17	27	0	27	28	3	3	24

APPENDIX B (continued)

Subject	EDE-Q 13	EDE-Q 14	EDE-Q 15	EDE-Q 16	EDE-Q 17	EDE-Q 18	EDI BN
18	10	10	10	50	5	0	27
19	12	12	12	60	0	0	13
20	20	20	16	30	0	0	10
21	1	1	1	28	0	2	17
22	0	0	0	20	0	22	5
23	70	70	28	75	0	5	29
24	1	2	2	8	0	24	9
25	2	2	24	28	0	18	29
26	0	3	0	15	0	0	4
27	112	112	28	112	0	0	
28	150	150	28	10	10	0	9

EDE-Q = Eating Disorders Examination Questionnaire Item, EDI BN = raw score from Bulimia Scale of Eating Disorders Inventory Third Edition

APPENDIX C

RAW DATA: WASI & D-KEFS SCORES

Subj.	WV	WM	WF	WM SC	D1 CS	D2 CS	D3 CS	D4 CS	D1 SC	D2 SC	D3 SC	D4 SC	D1 UC	D2 UC	D3 UC	D4 UC
1	60	56	117	0	25	18	42	58	0	0	1	1	0	0	0	0
2	61	51	110	3	31	22	42	64	1	1	1	4	0	0	1	3
3	77	53	127	2	27	19	37	53	0	0	0	0	0	0	0	0
4	53	45	98	1	34	23	46	55	1	0	0	0	0	0	1	1
5	73	63	133	0	33	26	41	60	0	0	0	0	0	0	0	2
6	60	56	117	0	25	18	42	58	0	0	1	1	0	0	0	0
7	52	49	100	2	26	20	42	45	0	1	0	1	1	0	1	3
8	67	59	123	2	26	18	36	47	1	1	2	2	0	0	1	1
9	51	49	99	5	22	20	52	50	0	1	4	2	0	0	2	1
10	59	57	114	1	25	20	37	43	3	0	1	3	0	0	0	0
11	46	45	92	2	30	17	55	55	1	0	2	1	0	0	0	0
12	46	55	100	0	22	17	38	39	0	0	1	1	0	0	0	0
13	47	43	91	0	42	29	77	68	0	0	0	1	2	0	5	7
14	64	51	113	4	27	22	44	54	0	1	0	0	0	0	0	0
15	54	57	109	1	30	23	54	54	0	1	3	2	0	1	0	0
16	63	53	114	2	23	16	31	63	1	1	2	3	0	0	4	1
17	54	53	106	2	34	22	52	52	1	1	2	3	0	0	0	0
18	47	51	98	0	29	18	56	56	0	0	0	2	0	0	0	0

APPENDIX C (continued)

Subj.	WV	WM	WF	WM SC	D1 CS	D2 CS	D3 CS	D4 CS	D1 SC	D2 SC	D3 SC	D4 SC	D1 UC	D2 UC	D3 UC	D4 UC
19	70	63	130	2	26	22	43	49	1	0	1	2	0	0	0	0
20	51	55	106	0	30	18	47	56	2	0	1	1	0	0	3	0
21	60	63	123	0	28	24	48	50	2	0	2	1	0	0	0	0
22	49	51	100	6	23	19	49	44	1	0	3	2	0	0	0	0
23	70	39	109	0	19	16	36	45	0	0	1	1	0	0	2	1
24	60	55	113	1	29	22	52	50	3	0	3	1	0	0	0	0
25	52	49	100	0	30	25	44	48	0	1	0	0	1	0	0	1
26	40	48	90	3	23	19	44	66	1	0	3	1	0	0	0	2
27	63	49	110	1	30	23	40	61	0	0	0	0	0	0	0	1
28	43	33	82	1	35	23	75	180	0	0	1	1	1	0	1	1

WV = WASI Vocabulary T-Score, WM = WASI Matrix Reasoning T-Score, WF = WASI 2-Subtest FSIQ Standard Score, WM SC = Number of Self-Corrected Errors on the WASI Matrix Reasoning subtest, D1 CS = D-KEFS Trial 1 Completion Time in seconds, D2 CS = D-KEFS Trial 2 Completion Time in seconds, D3 CS = D-KEFS Trial 3 Completion Time in seconds, D4 CS = D-KEFS Trial 4 Completion Time in seconds, D1 SC = D-KEFS Trial 1 Number of Self-Corrected Errors, D2 SC = D-KEFS Trial 2 Number of Self-Corrected Errors, D3 SC = D-KEFS Trial 3 Number of Self-Corrected Errors, D4 SC = D-KEFS Trial 4 Number of Self-Corrected Errors, D1 UC = D-KEFS Trial 1 Number of Uncorrected Errors, D2 UC = D-KEFS Trial 2 Number of Uncorrected Errors, D3 UC = D-KEFS Trial 3 Number of Uncorrected Errors, D4 UC = D-KEFS Trial 4 Number of Uncorrected Errors

APPENDIX D:

RAW DATA: BRIEF RATING SCALES T-SCORES

Subject	BI	BS	BEc	BSm	BM	BIn	BWm	BPo	BTm	BOm	BTc
1	77	86	80	76	-	82	82	68	81	61	-
2	50	47	49	42	-	50	50	46	54	47	-
3	57	81	58	50	-	53	53	52	50	36	-
4	63	81	78	54	-	63	63	52	59	58	-
5	40	51	43	37	-	37	37	41	45	36	-
6	77	86	80	76	-	82	82	68	81	61	-
7	80	60	60	63	-	66	66	57	50	58	-
8	77	81	72	59	-	79	79	70	59	45	-
9	67	69	65	50	-	69	69	60	59	58	-
10	53	86	60	42	-	76	76	62	68	64	-
11	60	60	67	50	-	47	47	52	50	56	-
12	53	60	54	54	-	50	50	57	50	36	-
13	81	67	69	-	62	-	-	85	-	81	81
14	74	60	67	59	-	69	69	60	72	42	-
15	84	81	67	80	-	69	69	73	63	67	-
16	70	69	60	54	-	66	66	57	54	39	-
17	63	81	63	54	-	69	69	68	63	50	-
18	63	64	69	59	-	69	69	68	68	69	-

APPENDIX D (continued)

	BI	BS	BEc	BSm	BM	BIn	BWm	BPo	BTm	BOm	BTc
19	59	52	49	-	47	-	-	56	-	81	68
20	67	86	60	46	-	79	79	70	68	67	-
21	70	60	65	63	-	53	53	52	50	50	-
22	60	69	73	58	-	54	54	51	53	56	-
23	77	86	78	63	-	85	85	84	86	78	-
24	87	77	67	76	-	69	69	60	68	56	-
25	77	77	80	72	-	73	73	78	90	39	-
26	59	83	67	-	52	-	-	61	-	81	65
27	74	73	76	76	-	50	50	49	40	58	-
28	63	64	58	50	-	66	66	68	77	47	-

BI = BRIEF-A or BRIEF-SR Inhibition Scale T-Score, BS = BRIEF-A or BRIEF-SR Shift Scale T-Score, BEc = BRIEF-A or BRIEF-SR Emotional Control Scale T-Score, BSm = BRIEF-A Self-Monitor Scale T-Score, BM = BRIEF-SR Monitor Scale T-Score, BIn = BRIEF-A Initiation Scale T-Score, BWm = BRIEF-A Working Memory Scale T-Score, BPo = BRIEF-A or BRIEF-SR Planning & Organization Scale T-Score, BTm = BRIEF-A Task Monitor Scale T-Score, BOm = BRIEF-A or BRIEF-SR Organization of Materials Scale T-Score, BTc = BRIEF-SR Task Completion Scale T-Score

APPENDIX E

RAW DATA: TYPE-T SURVEY AND CHOICE OF COMPENSATION

Subject	Type-T Item 1	Type-T Item 2	Type-T Item 3	Choice of Comp.
1	8	5	4	Delay
2	5	7	8	Immediate
3	3	7	3	Delay
4	6	8	5	Delay
5	3	4	2	Delay
6	8	5	4	Delay
7	6	7	7	Delay
8	7	2	6	Delay
9	7	7	9	Delay
10	6	9	6	Delay
11	8	3	7	Delay
12	7	8	8	Delay
13	9	7	8	Delay
14	9	6	9	Delay
15	8	8	8	Delay
16	7	10	7	Immediate
17	7	5	7	Delay
18	7	8	8	Delay
19	7	6	8	Delay

APPENDIX E (continued)

Subject	Type-T Item 1	Type-T Item 2	Type-T Item 3	Choice of Comp.
20	5	10	7	Immediate
21	8	5	7	Delay
22	7	7	6	Delay
23	7	9	7	Delay
24	9	9	9	Immediate
25	9	9	8	Immediate
26	5	1	5	Delay
27	10	10	10	Immediate
28	4	7	5	Immediate

Choice of Comp = Choice of Compensation

APPENDIX F:

DEMOGRAPHIC DATA

Subject	Ethnicity	Religion	Education	Employment
1	caucasian	Catholic	some college	student
2	caucasian	Christian	less than high school	employed part-time
3	caucasian	Spiritual	some college	student
4	caucasian	Other	some college	employed part-time
5	caucasian	Catholic	some college	student
6	caucasian	Catholic	some college	student
7	caucasian	Catholic	Associate's Degree (2-year)	student
8	caucasian	None	Four-year college	unemployed
9	caucasian	Catholic	completed high school	student
10	african american	Christian	some college	employed part-time
11	caucasian	Catholic	some college	student
12	caucasian	Christian	some college	unemployed
13	caucasian	Catholic	less than high school	student
14	caucasian	Jewish	some college	unemployed
15	Asian or Pacific Islander	Christian	some college	employed full-time
16	caucasian	Jewish	completed high school	employed part-time
17	caucasian	Catholic	some college	student
18	caucasian	Catholic	some college	student
19	caucasian	Catholic	less than high school	employed part-time

APPENDIX F (continued)

Subject	Ethnicity	Religion	Education	Employment
20	caucasian	Catholic	completed high school	unemployed
21	caucasian	None	some college	student
22	caucasian	None	completed high school	student
23	caucasian	Catholic	some college	unemployed
24	caucasian	Christian	less than high school	student
25	Asian or Pacific Islander	Hindu	some college	
26	caucasian	Christian	less than high school	unemployed
27	caucasian		completed high school	student
28	caucasian	Jewish	some college	employed part-time