THE URGE TO PURGE: AN ECOLOGICAL MOMENTARY ASSESSMENT OF PURGING DISORDER AND BULIMIA NERVOSA

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by

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CHAPTER I

INTRODUCTION

Though there is general consensus among psychologists that Anorexia Nervosa (AN) and Bulimia Nervosa (BN) are clinically significant and distinct categories of eating disorders, the majority of individuals who experience clinically significant symptoms of eating psychopathology do not meet the diagnostic criteria for either diagnosis (Keel, Haedt, & Edler, 2005; Machado, Machado, Goncalves, & Hoek, 2007; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006). Thus, such individuals most often receive a diagnosis of an Eating Disorder Not Otherwise Specified (EDNOS). According to the criteria set forth by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*; American Psychological Association [APA], 2000), 40-60% of patients at eating disorder treatment centers receive EDNOS diagnoses (e.g., Fairburn, Cooper, Bohn, Conner, Doll, & Palmer, 2007; Martin, Williamson, & Thaw, 2000; Ricca et al., 2001; Rockert, Kaplan, & Olmsted, 2007; Turner & Bryant-Waugh, 2004), and it has been estimated that EDNOS accounts for over 75% of eating disorder diagnoses in community settings (Machado et al., 2007). Moreover, even with the publication of more relaxed criteria for eating disorder diagnoses in the fifth edition of the *DSM* (*DSM-5*; APA, 2013), evidence suggests that EDNOS still accounts for 44 to 51% of community eating disorder diagnoses after applying the new *DSM*-5 criteria (Machado, Goncalves, & Hoek, 2013).

The diagnostic status of EDNOS is problematic for a variety of reasons. In general, the "not otherwise specified" (NOS) or Other Specified Feeding or Eating Disorder (OSFED) diagnoses in the *DSM* (APA, 2000, 2013) are reserved for residual, atypical disorders that are not

consistent with common presentations of recognized disorders. That is, these disorders meet some, but not all of the necessary criteria for the mental disorders specified in the current *DSM-5*. Thus, the "catch-all" category of NOS usually represents uncommon, relatively anomalous forms of established disorders. While the NOS diagnosis may fill in the gap between existing diagnostic categories, it conveys little information to clinicians regarding the etiology, presentation, course, and treatment of a particular individual's psychopathology, which results in minimal clinical utility of such diagnoses (Widiger & Samuel, 2009). Moreover, EDNOS has been the most commonly diagnosed eating disorder in treatment settings, which contradicts the "atypical" nature of NOS diagnoses. Experts have recognized the problematic nature of such broad categorization of subthreshold eating disorders, as this is a group of highly heterogeneous patterns of behaviors that are believed to have clinical significance (Crow, 2007; Fairburn & Cooper, 2007; Fairburn et al., 2007; Machado et al., 2007).

Purging Disorder (PD), which is characterized by recurrent purging behavior in the absence of binge episodes (e.g., Keel & Striegel-Moore, 2009), is one EDNOS subtype that has been the focus of increasing study. Purging behavior (i.e., self-induced vomiting, laxative, and/or diuretic abuse) is of particular clinical concern, as this behavior is associated with significant medical problems, including metabolic disturbances, electrolyte imbalances, edema, dental problems, esophageal tears, swollen salivary glands, and gastrointestinal reflux (Fairburn, 1985; Keel, 2005). Currently PD is classified under the EDNOS category in the *DSM-5*, yet recent literature suggests it may be a distinct and clinically significant disorder.

Research indicates that the prevalence rate of PD is comparable to other eating disorders. Lifetime prevalence rates of PD have ranged from 1.1% (Favaro, Ferrara, & Santonastaso, 2003) to 5.3% (Wade et al., 2006). In addition, Crowther, Armey, Luce, Dalton, and Leahey (2008)

examined the point prevalence of BN, EDNOS, and related eating disorder symptomatology among 6,844 female undergraduates across five 3-year time periods from 1990 to 2004; during this 15-year period, the percentage of PD ranged from 0.3 to 1.0%. More recently, Haedt and Keel (2010) reported a point prevalence of PD among a sample of 1736 women that ranged from 0.6% (using a PD definition requiring purging at least twice per week) to 5.5% (using a definition requiring purging and/or nonpurging behavior at least once per week).

Although PD is currently categorized as an EDNOS subtype, research has indicated that BN, PD, and subclinical BN all can be conceptualized as bulimic spectrum disorders (Darcy et al., 2012; Steiger & Bruce, 2007). Therefore, there is literature to support conceptualizing both BN and PD as falling within the same superordinate category of eating psychopathology. Thus, the present research will seek to further understand PD by examining this disorder within a range of bulimic symptomatology.

Moreover, although there is a substantial theoretical and empirical literature on BN, there is a dearth of literature that has investigated the theoretical conceptualization and topography of PD symptoms. Therefore a diagnosis of PD as an EDNOS subtype conveys little information to clinicians regarding the typical presentation of the disorder. Moreover, the existing research on PD has relied on participants' retrospective reports, which are prone to memory and recall bias. However, Ecological Momentary Assessment (EMA) methods reduce this concern by collecting repeated momentary assessments from participants in their natural environments, which enhances the ecological validity of data (Shiffman & Stone, 1998; Stone & Shiffman, 2002). Thus, the present study proposes to use EMA methodology to examine the cognitive, affective, and behavioral antecedents and consequences of eating and compensatory behavior among individuals with bulimic spectrum disorders.

The remainder of the introduction will first review the existing literature on PD, including evidence of how PD compares to BN, as well as the current discrepancies and deficiencies in the literature. Next, theoretical models of bulimic symptomatology will be reviewed, as these models inform the research questions of the present study. Finally, the aims and research questions of the present study will be discussed, as well as the rationale for using EMA methodology.

Purging Disorder

Although there is a wealth of literature examining BN, less research has examined other bulimic spectrum disorders, namely PD. In a review of the published literature on PD, Keel and Striegel-Moore (2009) proposed five diagnostic criteria for PD: (1) recurrent purging in order to influence weight or shape (e.g., self-induced vomiting, laxative abuse, enemas, diuretics); (2) purging occurs, on average, at least once a week for three months; (3) self-evaluation is unduly influenced by body shape or weight or there is an intense fear of gaining weight or becoming fat; (4) the purging does not occur exclusively during the course of anorexia nervosa or bulimia nervosa; and (5) the purging is not associated with objectively large binge episodes. Additionally, in an investigation of the syndrome validity of PD, Haedt and Keel (2010) varied the diagnostic criteria by type of compensatory behavior (purging vs. non-purging) and frequency (once vs. twice per week) and found that distinguishing between purging and non-purging behavior maximized the difference between PD and normality (i.e., excluding non-purging behavior from the PD definition). However, reducing the minimum frequency of purging behavior to once per week did not significantly impact the levels of external validators associated with PD. This research supports the diagnostic criteria proposed above (Keel & Striegel-Moore, 2009).

Thus far research has supported the clinical significance of PD. Compared to individuals without PD, individuals with PD show increased levels of impairment, including higher levels of general psychopathology, distress, eating pathology, and personality disorders (Keel, Wolfe, Gravener, & Jimerson, 2008; Keel et al., 2005). However, research has yielded mixed findings regarding how individuals with PD compare to individuals with other eating disorder diagnoses, particularly BN. For example, some studies have demonstrated that women with PD do not significantly differ from those with BN on measures of symptom severity, impairment, body dissatisfaction, or dietary restraint (Binford & le Grange, 2005; Keel, Mayer, & Harnden-Fischer, 2001; Keel et al., 2005). In contrast, other literature suggests that, compared to individuals with PD, individuals with BN generally report greater shape/weight/eating concerns (Binford & le Grange, 2005), lower levels of self-esteem (Binford & le Grange, 2005), and greater levels of psychopathology, including current mood disorders (Keel, et al., 2008; Keel et al., 2005), anxiety levels (Fink, Smith, Gordon, Holm-Denoma, & Joiner, 2009), and impulsive behaviors (Fink et al., 2009; Keel et al., 2001), although other studies have failed to replicate the findings regarding impulsivity (Keel et al., 2008; Keel et al., 2005).

In a recent meta-analysis (Smith, unpublished manuscript), three EDNOS subtypes (i.e., subclinical AN, subclinical BN, and PD) were systematically compared to healthy controls, AN, and BN, as well as to each other on levels of general and eating psychopathology. Upon examination of nine studies that compared BN and PD groups (yielding 68 effect sizes) and five studies comparing PD and control groups (yielding 35 effect sizes), results indicated that PD groups reported significantly higher levels of general psychopathology, anxiety, depression, eating psychopathology, body image disturbances, and impulsivity compared to control groups. When BN was compared to PD, BN groups exhibited significantly higher levels of anxiety,

depression, body image disturbance, and eating psychopathology. However, BN and PD did not differ significantly on levels of general psychopathology, impulsivity, obsessive compulsiveness, perfectionism, or self-esteem. Although these comparisons were based on a small number of studies, it appears that there are both similarities and differences between BN and PD; thus, it is yet unclear whether similar psychological processes drive these disorders.

Moreover, it is unclear what cognitions, affect, and/or behaviors precipitate purging behavior in PD. In the purging subtype of BN, there is a consensus that purging behavior is preceded by objective binge episodes (OBEs), in which individuals (1) consume an amount of food that is larger than most people would eat during a similar period of time and under similar circumstances, and (2) experience a sense of lack of control overeating (APA, 2013; Stice, 2001). Orleans and Barnett (1984) proposed a model of the antecedents of the binge-purge cycle. According to this model, dietary restriction results in feelings of hunger, frustration, fatigue, and physiological and psychological deprivation, which in turns increases preoccupation with food. Because these individual also have dichotomous cognitions about "good" and "bad" foods, which only allow for dieting or disinhibition, when their attempts to control their diet fail due to environmental and psychological stress, they often engage in binge eating. These binge episodes result in increased negative affect, which these individuals attempt to alleviate via compensatory behaviors. Thus, purging behaviors are generally studied in conjunction with binge episodes. However, consistent with PD diagnostic criteria (Keel & Striegel-Moore, 2009), individuals with PD do not engage in OBEs, which potentially challenges the existing theories that conceptualize purging behavior as a response to OBEs.

However, a recent study by Jones and Crowther (2012) examined the onset of compensatory behaviors independent of binge eating among a sample of 237 undergraduate

women, who were assessed at two time points (one year apart). Results indicated that body dissatisfaction and perfectionism at time one significantly predicted the onset of compensatory behaviors at time two. Additionally, women who began compensatory behaviors reported more body dissatisfaction at time one than women who engaged in compensatory behaviors at both time points. Such findings suggest that body dissatisfaction and perfectionism are predisposing factors in the onset of compensatory behaviors. However, the immediate affective and behavioral antecedents of purging behavior have yet to be elucidated.

That is, it is possible that subjective binge episodes (SBEs) directly precede purging in PD. Individuals with PD may experience a loss of control when they consume an objectively moderate amount of food (Fairburn & Wilson, 1993). For example, Smith and Crowther (2013) found that individuals with PD reported significantly more SBEs than healthy controls, but fewer than those of individuals with BN. However, thus far, few studies have examined the presence of SBEs in eating disorders (e.g., Keel et al., 2001). Therefore additional research is needed to investigate the frequency and phenomenology of such episodes in PD.

Furthermore, there is some evidence that the distinction between SBEs and OBEs may not be clinically significant, as participants report similar levels of distress regardless of whether they experience an SBE or OBE (Niego, Pratt, & Agras, 1997; Pratt, Niego, & Agras, 1998). For instance, Mond et al. (2006) found that individuals who reported SBEs (in absence of any OBEs) had similar levels of functional impairment as individuals who reported OBEs. Such evidence suggests that the psychological experience of loss of control, regardless of objective caloric intake, is what causes distress for individuals with eating pathology. The distress associated with SBEs may be sufficient to precipitate subsequent purging behavior. Therefore, the similar

phenomenology of SBEs and OBEs may have important treatment implications for both PD and BN.

Bulimia Nervosa

In order to further understand bulimic spectrum disorders, it is necessary to first review empirically supported theories of BN, as this disorder is also characterized by the regular use of compensatory behavior. Understanding the etiological and maintaining mechanisms underlying BN will help determine whether the cognitions, affect, and behaviors evidenced by individuals with PD are consistent with what would be predicted by theories of BN. If so, then it is arguable that BN and PD have similar underlying psychological mechanisms. However, some behavioral manifestations may differ between these disorders, such as the presence and/or severity of binge eating behavior, as well as the frequency of compensatory behavior. Elucidating the psychological and behavioral characteristics of PD will further support the conceptualization of PD as existing along a spectrum of bulimic symptomatology. Alternatively, if the cognitions, affect, and behaviors evidenced by individuals with PD are inconsistent with existing theories of BN, then PD may be a distinct disorder.

Restraint Theory

It is well-established that restrained eating precedes OBEs and subsequent purging behavior in BN (Fairburn & Garner, 1986), and it is known that individuals with PD engage in clinically significant purging behavior (Keel et al., 2005; Keel & Striegel-Moore, 2009). Given that restraint theory offers an explanation of the mechanisms underlying binge and purge behaviors in BN, it also may provide an understanding of the antecedents to purging in PD (Engelberg, Gauvin, & Steiger, 2005; Polivy & Herman, 1985; Wilson, 2002). Furthermore, studies have

documented significant dietary restraint among individuals with PD (e.g., Smith & Crowther, 2013).

According to restraint theory, both the physiological and psychological aspects of restrained eating increase the likelihood of subsequent binge eating (Herman & Polivy, 1980; Herman & Polivy, 1975; Herman & Polivy, 1983; Polivy & Herman, 1985). In an effort to lose weight, restrained eaters attempt to adhere to unrealistic, rigid dietary rules in order to restrict their food intake (Polivy & Herman, 1985). Such individuals dichotomously discriminate between "good" foods and "bad" foods, and restrained eaters rely heavily on these inhibitory cognitive controls to suppress normal hunger and satiety signals. Rigid dietary restraint, when coupled with other biological, cognitive, and affective factors related to dieting, may lead dieters to feel a loss of control after any lapse in their diet. Such feelings, combined with "all-or-nothing" dietary rules, increase the likelihood that the person will temporarily abandon all dietary rules and engage in binge episodes after a perceived transgression in their diet (Wilson, 2002). In response to such transgressions, it is possible that individuals engage in compensatory behaviors (i.e., self-induced vomiting, laxative abuse, diet pill use, excessive exercise, and/or fasting).

In support of restraint theory, empirical evidence suggests that dietary restraint may potentiate binge eating and contribute to the development of eating disorders (Lowe et al., 1996; Stice, 2002; Wilson, 2002). Two findings are particularly relevant. First, using ecological momentary assessment, Engelberg, Gauvin, and Steiger (2005) found that restraint was elevated before binge cravings, but not binge episodes. Such findings support restraint theory, in that dietary restraint potentiates, but does not directly cause, binge eating. Rather, other factors, such as emotional distress, may trigger binge episodes. Thus, in PD, it is possible that restraint is associated with binge cravings, but not necessarily objectively large binge episodes unless other

factors are present. Second, Kerzhnerman and Lowe (2002) examined dieting intensity and the frequency of objective and subjective binge episodes; results indicated higher levels of dieting intensity were related to more frequent subjective, yet not objective, binge episodes. This finding suggests that the relationship between dietary restraint and subsequent perceived disinhibited eating (i.e., SBEs) may be stronger and more significant than the relationship between dietary restraint and objective caloric consumption (i.e., OBEs).

In sum, restraint theory offers a potential theoretical framework from which to conceptualize the development of PD. Restraint theory would suggest that because individuals with PD appear to evidence significant dietary restraint, they are likely to experience a subsequent lapse of control over eating behaviors (i.e., SBEs) prior to purging. Although studies have retrospectively assessed the presence of dietary restraint and SBEs in PD (e.g., Keel et al., 2001; Smith & Crowther, 2013), to date, no study has assessed the daily eating habits and subjective experiences of individuals with PD using EMA methodology. Doing so may provide clarification as to whether PD is associated with significant dietary restraint and a subsequent loss of control over eating prior to purging.

Affect Regulation Model

In addition to dietary restraint, affective changes have been implicated in the onset and maintenance of bulimic symptomatology. The affect regulation model (Stice, 2001) has been the focus of a substantial body of research investigating the antecedents and consequences of binge eating in BN. This model is based on two central tenets: (1) negative affect triggers binge eating episodes, and (2) binge eating functions to reduce this negative affect (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003; Gross, 2007; McManus & Waller, 1995; Stice, 2001). Thus, via the process of negative reinforcement, binge

eating becomes a habitual, learned response to the experience of negative emotions, which serves to maintain this behavior. Given that there is a potential that individuals with PD experience SBEs, it is important to consider the role of negative affect in PD.

Overall, empirical studies have yielded strong support for the first tenet of the affect regulation model, in that negative affect is a proximal antecedent of binge eating (Agras & Telch, 1998; Chua, Touyz, & Hill, 2004; Telch & Agras, 1996). Regarding specific negative emotions, naturalistic studies of women with BN have consistently found that levels of depression (Davis, Freeman, & Garner, 1988; Powell & Thelen, 1996), hostility (Powell & Thelen, 1996; Rebert, Stanton, & Schwarz, 1991), and anger (Rebert et al., 1991) are elevated prior to binge episodes. Furthermore, Abraham and Beaumont (1982) and Elmore and de Castro (1990) reported increases in anxiety before binges among women with BN, although some studies (Powell & Thelen, 1996) have failed to replicate this finding. Moreover, studies using negative mood inductions (Agras & Telch, 1998; Chua et al., 2004; Telch & Agras, 1996) have demonstrated that individuals consume more food when assigned to a negative mood induction as compared to a neutral mood induction. Davis and Jamieson (2005) examined the function and phenomenology of binge eating. These authors administered the Binge Eating Adjective Checklist (BEAC) to a sample of women at an eating disorders clinic and found that two-thirds of the participants retrospectively reported feeling anxious, depressed, frustrated, helpless, lonely, bored, having no will power or control, and craving food prior to binge episodes.

However, evidence supporting the second tenet of the affect regulation model has been mixed, with some studies reporting a decrease in participants' negative affect following binges (e.g., Abraham & Beaumont, 1982; Hawkins & Clement, 1984; Hsu, 1990), and others reporting an increase in negative affect following binges (e.g., Arnow et al., 1992; Lingswiler, Crowther,

& Stephens, 1989; Mitchell et al., 1985 1999; Mizes & Arbitell, 1991; Pyle et al., 1981; Tachi, Murakami, Murotsu, & Washizuka, 2001). For instance, Cooper et al (1988) examined mood changes over the binge-purge cycle among women with eating disorders. Results indicated that feelings of panic, helplessness, guilt, disgust, and anger were highest during the time immediately following binge episodes but prior to purging behavior. Additionally, positive emotions, including energy, excitement, security, and relief, decreased significantly during this time period. The negative emotional consequences of binge episodes also appear to persist over time, as demonstrated by Lingswiler et al. (1989). These authors examined emotions immediately following and one hour after daily eating episodes among women who engaged in binge eating. Results indicated that binge episodes were followed by significantly greater immediate and delayed negative emotions (i.e., one hour after eating) compared to non-binge episodes. Furthermore, evidence has suggested that negative affect is not a unidimensional construct, as different changes have been observed in types of negative affect after binge episodes. For example, in a diary study of women with BN, Elmore and de Castro (1990) found that individuals reported reduced anxiety but increased depression following binge episodes. This suggests that affective changes during the binge-purge cycle are more complex than what has been postulated by the affect regulation model.

Taken together, while there appears to be general consensus in the literature that negative affect precedes binge eating, the tenet that binge eating functions to reduce negative affect has been challenged consistently by empirical findings. Furthermore, a recent meta-analysis (Haedt-Matt & Keel, 2011) evaluated the tenets of the affect regulation model using 36 prospective studies that used Ecological Momentary Assessment (EMA) methods. Overall, this review found greater levels of pre-binge negative affect compared to participant's average affect and their

affect before non-binge eating, supporting the first tenet of the affect regulation model. However, the second tenet was not supported in this review, as results indicated negative affect increased following binge episodes. Therefore, the hypothesized reduction in negative affect after binge eating (i.e., the second tenet) may not be the maintenance factor of bulimic symptomatology, as it has been posited by this model.

Nevertheless, this model has important implications for understanding bulimic spectrum disorders. Although there is substantial literature documenting the role of affect in BN (Haedt-Matt & Keel, 2011), to date, no studies have investigated daily affective changes in PD. If individuals with PD experience SBEs, it is possible that such episodes are preceded by negative affect, as suggested by the first tenet of the affect regulation model. Moreover, given the inconclusive evidence supporting the second tenet of the affect regulation model, it is necessary to examine other behaviors that may contribute to the maintenance of bulimic symptomatology, such as purging behavior. Notably, the meta-analysis by Haedt-Matt and Keel (2011) found that negative affect decreased following purging behavior. However, despite the substantial body of literature investigating the affect regulation model of binge eating, few studies have examined the affective influence of compensatory behaviors, even though purging behavior is often coupled with binge episodes in BN.

Anxiety Reduction Model

In contrast to the affect regulation model, the anxiety reduction model (Rosen & Leitenberg, 1982, 1988; Rosen, Leitenberg, Gross, & Willmuth, 1998) offers a framework from which to understand the role of purging behavior in BN, and therefore this model may inform the conceptualization of PD. The anxiety reduction model posits that purging is the maintaining factor in the binge-purge cycle. Consistent with the findings of Haedt-Matt and Keel (2011),

several studies have supported the hypothesis that purging behavior, rather than binge eating, serves to reduce negative affect and maintain the binge-purge cycle (Abraham & Beaumont, 1982; Johnson & Larson, 1982; Kaye et al., 1986; Powell & Thelen, 1996). Such studies have found that individuals report reductions in anxiety from post-binge to post-purging behavior. For instance, in a study of 29 females with BN who retrospectively reported their emotions during the binge-purge cycle, Mizes and Arbitell (1991) found that negative emotions increased after binging and decreased after purging. Additionally, positive emotions decreased after binging and increased after purging. This study also highlights an important conceptual consideration, in that positive emotions as well as negative emotions fluctuate as a function of binging and purging. Thus, positive and negative emotions may be important to consider in the maintenance models of bulimic behavior.

Furthermore, studies have found that individuals with BN will not engage in binge eating if they are aware they subsequently will be prevented from purging (Rosen & Leitenberg, 1982; Rosen et al., 1985), which is consistent with the theory that purging drives binge eating behavior among individuals with BN. That is, individuals with BN will not binge if they know they cannot purge. Additionally, the results of experimental studies indicate that individuals with BN not only refrain from binge eating if the opportunity to purge is prevented, but they also eat minimal amounts of anxiety-provoking foods (e.g., one bite of one candy bar) and significantly less compared to healthy controls during laboratory test meals (Rosen & Leitenberg, 1982; Rosen et al., 1985). Based on such observations, Rosen and Leitenberg (1982, 1988) suggest that BN is analogous to obsessive-compulsive disorder (OCD), in that individuals have a fear of eating (especially foods perceived as fattening) and may consider even normal quantities of many foods to be "unsafe, repulsive, and fattening." Vomiting is similar to the compulsive behaviors in OCD,

such as repetitive hand-washing or compulsive checking, and thus vomiting becomes a "magic ritual" that protects against anxiety and feared consequences (i.e., weight gain) in BN. Furthermore, vomiting "removes inhibitions against binge-eating episodes in the future, thereby maintaining a cycle of bulimic symptomatology" (Rosen & Leitenberg, 1982, p. 166).

The function of purging behavior was further explored in a study by Rebert et al. (1991), who examined a sample of individuals with purging-type BN and binge eaters who self-monitored affect and eating episodes over 20 days. Results indicated the number of binge episodes did not differ significantly between groups, but those who experienced higher levels of negative affect were more likely to binge later in the day. However, the relationship between pre-binge anxiety and hostility and binge occurrences was stronger among individuals with purging-type BN than binge eaters. Again, these findings support the tenet that binge eating is preceded by increases in negative affect. However, the findings suggest that in the presence of increased anxiety and hostility, it is more likely that individuals will binge if they also have learned a habitual response to the binge (i.e., purging). Thus, it is possible that purging serves to reduce their initial heightened negative emotions. These findings are consistent with Rosen and Leitenberg's (1982) assertion that the anticipation of purging may increase severity (or frequency) of binge episodes. Furthermore, the anticipation of purging may suppress the increase in post-binge negative affect due to a belief that purging will offset the caloric consequences of the binge, as suggested by previous findings (Abraham & Beaumont, 1982; Leitenberg, Gross, Peterson, & Rosen, 1984). These results suggest that individuals who binge but are prevented from purging may experience higher post-binge negative affect compared to those who binge and purge, or purge and do not binge. These findings may have important implications for affective changes observed in both BN and PD.

Based on aforementioned evidence, it seems reasonable that individuals with PD restrict their dietary intake and purge in response to the consumption of normal amounts of food (i.e., SBEs) due to a fear of eating, gaining weight, and/or appearing overweight. As stated by Rosen and Leitenberg (1982, p. 167), "in the usual progression of (BN), once self-induced vomiting is learned, binge-eating typically becomes more severe and more frequent." Given that it is possible that individuals with PD experience SBEs, with continued use of self-induced vomiting they may eventually engage in OBEs and cross the diagnostic threshold to BN.

If PD exists on a spectrum of bulimic symptomatology, the anxiety reduction model suggests that individuals with PD, like those with BN, may anticipate engaging in compensatory behavior as a means to regulate their negative affect throughout the day. Similar to BN, it would be expected that purging behavior would be particularly effective in reducing anxiety. Thus, there is a need to assess the daily affective changes and behaviors of individuals with PD and BN in order to determine whether the anxiety reduction model is equally applicable to BN and PD.

Cognitive Behavioral and Transdiagnostic Theory

The leading evidence-based treatment for eating disorders is an adapted form of Cognitive Behavioral Therapy, termed Enhanced Cognitive Behavior Therapy for Eating Disorders (CBT-E; Fairburn, 2008), which is based on the cognitive behavioral theory of BN (Fairburn, 1986). Although the CBT-E model was developed for BN, it can be applied to other eating disorders, including Anorexia Nervosa (AN) and EDNOS or OSFED. Because of the broad applicability of the CBT-E model, Fairburn and colleagues (e.g., Fairburn & Bohn, 2005; Fairburn, Cooper, & Shafran, 2003) have developed a "transdiagnostic" model of eating disorders, which posits that all eating disorders share the same core psychopathology (i.e., a dysfunctional scheme for self-evaluation). According to this theory, the core psychopathology

underlying all eating disorders is a dysfunctional scheme for self-evaluation, which results in dietary restraint, negative body image, and other forms of weight control behavior. Fairburn et al. (2003) suggest that specific individual factors influence the maintenance of eating pathology: perfectionism, low self-esteem, mood intolerance (i.e., an inability to cope with or accept certain emotions), and interpersonal difficulties. The specific combination of these factors determines the unique patterns of eating disturbances in an individual (e.g., AN is associated with higher levels of perfectionism, whereas BN is associated with more mood intolerance). Binge eating, according to this model, is a result of dietary restraint. However, when experiencing negative moods or stressful life events, it is difficult for individuals to maintain adherence to unrealistic and overly restrictive diets. At such times, individuals with BN and subclinical BN temporarily abandon all dietary rules, which results in binge eating. This model suggests that binging temporarily distracts individuals from their negative mood states, yet they subsequently feel guilt and shame about binging. In turn, many individuals engage in compensatory behaviors (e.g., self-induced vomiting, excessive exercise, laxative abuse, diuretic use) to minimize the caloric effects of the binge and/or alleviate their guilt and shame about binging. However, the transdiagnostic model also posits that such compensatory behavior may lead to further guilt, shame, and mood dysregulation. Thus, according to this model, mood intolerance is both a cause and consequence of binging and purging, which thereby serves to maintain this cycle of behavior.

Evidence for restraint theory, the affect regulation model, and the anxiety reduction model has lent insight into the mechanisms underlying the binge-purge cycle in BN. If individuals with PD evidence mechanisms that are consistent with these theories of BN, then it is arguable that PD exists along a spectrum of bulimic symptomatology. However, if individuals

with PD do not exhibit similar mechanisms as those with BN, then it is reasonable to suggest PD is a distinct eating disorder. Thus, the present study aims to assess these mechanisms that have not been examined in PD.

The Present Study

The present study examined the daily cognitions, emotions, and eating behaviors of individuals with bulimic spectrum disorders, including BN and PD, as well as healthy controls in the naturalistic environment, as no study has done so thus far. The following research questions and hypotheses were examined in the present study.

1. What is the frequency of SBEs compared to OBEs among individuals with bulimic spectrum disorders, and how does the type and frequency of eating episodes differ from healthy controls? Furthermore, do PD and BN groups differ in the frequency of SBEs? It was hypothesized that individuals with PD would report more SBEs than controls but would not differ from those with BN in the frequency of SBEs.
2. What are the antecedents and consequences of dietary restraint? Although it is well-established that dietary restraint is associated with bulimic symptomatology, to our knowledge no study has examined antecedents and consequences of dietary restraint using Ecological Momentary Assessment (EMA) methods, which may yield valuable information for treatment interventions. The following two questions will address this issue:
    - First, what predicts dietary restraint among individuals with bulimic spectrum disorders? It was hypothesized that negative affect and body image dissatisfaction would predict restraint, and that this relationship would be stronger among those in the bulimic spectrum group.

- Second, what is the relationship between dietary restriction and different types of eating psychopathology among individuals with bulimic spectrum disorders? It was hypothesized that dietary restriction (i.e., independent variable, as measured by the time lapsed until the first eating episode each day) would be predictive of the occurrence of (1) subjective binge episodes and (2) objective binge episodes; and (3) contemplation of purging behavior.

3. It was hypothesized that individuals with bulimic spectrum disorders anticipate compensatory behaviors (i.e., purging, exercise, and restriction), as demonstrated by preoccupation with thoughts of these behaviors. This phenomenon has been documented among individuals with bulimic symptomatology, which is consistent with the anxiety reduction model.
4. What are the antecedents and consequences of the loss of control over eating among individuals with bulimic spectrum disorders? More specifically, what are the antecedents and consequences of SBEs (which involve a loss of control but not the consumption of large amounts of food) and OBEs (which involve both a loss of control and the consumption of large amounts of food) compared to normal eating episodes? Based on previous literature, it was hypothesized that SBEs and OBEs have similar affective and behavioral antecedents and consequences compared to normal episodes. If this hypothesis is supported, this may have implications for future diagnostic criteria, which currently require OBEs to be present for a diagnosis of Bulimia Nervosa (which overlooks the significance of SBEs). Furthermore, thus far no study has used EMA to assess the antecedents and consequences of loss of control over eating behavior. The following hypotheses addressed this question:

- Regarding antecedents, it was hypothesized that compared to normal eating episodes, the loss of control over eating (operationalized by the occurrence of both SBEs and OBEs) would be predicted by greater levels of overall negative affect, guilt, and severity of negative events. Second, it was hypothesized that these independent variables would not discriminate between SBEs and OBEs.
- Regarding consequences, it was hypothesized that compared to normal eating episodes, the loss of control over eating (operationalized by the occurrence of SBEs and OBEs) would predict contemplation of subsequent compensatory behaviors (i.e., dietary restriction, excessive exercise, purging), body image dissatisfaction, overall negative affect, and guilt.

In contrast to the methods used in the existing research, EMA methods are needed to examine eating behavior in participants' natural environments. EMA methods allows for the measurement of participants' current psychological states and behaviors, which decreases the problems of recall and memory biases encountered with retrospective reporting (Stone & Shiffman, 2002). Participants are assessed in their natural environment rather than laboratory settings, which also serves to increase the ecological validity and generalizability of findings. Lastly, the repeated measurements obtained via EMA studies allows for the temporal sequencing of variables, which allows for assessment of variability and possible causal relationships. As it is not feasible to replicate all of the possible contextual factors that influence individuals' binge and purge behavior in a laboratory setting, EMA provides the best methodology to assess the antecedents and consequences of bulimic symptomatology. Thus, the research aims of the present study are best addressed using EMA methodology.

CHAPTER II

METHOD

Participants

Participants were recruited at Kent State University through online mass testing procedures, which are administered as one means through which participants can complete their research participation requirements for undergraduate psychology courses. The mass testing protocol included the Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000), which was used to screen participants for the present study. Female individuals were invited to participate in the present study if they met current diagnostic criteria for BN and PD (i.e., bulimic spectrum disorders), or if they did not endorse any disordered eating symptomatology (i.e., healthy controls).

Group membership (i.e., BN, PD, or Control) was determined based on individuals' scores on the EDDS. That is, the scoring algorithm for the EDDS identified potential BN participants based on *DSM-5* criteria. Based on previous findings regarding the definition of PD (e.g., Keel & Striegel-Moore, 2009), participants were assigned to the PD group if they (1) did not meet criteria for AN or BN according to the EDDS algorithm, (2) reported at least one episode of self-induced vomiting and/or laxative abuse per week over the past three months, and (3) reported an undue influence of weight and/or shape on self-evaluation. Lastly, Control group membership was determined based on the lack of endorsement of symptomatology on the EDDS.

Procedure

Individuals who met criteria for the above diagnostic groups were invited to complete the initial assessment in the researcher's laboratory at Kent State University. Upon informed consent, a graduate student confirmed participants' group membership by reviewing their responses on the EDDS with them, and each participant completed a battery of self-report questionnaires in a private room in the researcher's laboratory. Participants were given a Palm Centro to complete their daily assessments and trained on its use. Participants carried the Palm Centro for 10 days. At the end of the 10-day data collection period, participants returned the Palm Centro to the lab and were paid for their participation. Participants received $90 for completing the study. In addition, to improve compliance to the protocol, participants received up to an additional $25 based upon their compliance rates. We have found that using this additional incentive yields acceptable compliance rates (70-80%).

The EMA protocol involved two types of self-report methods: single-contingent responding and fixed event responding. First, participants were signaled at five semi-random times throughout the day. Each day (from 8:30 a.m. to 11:30 p.m.) was divided into five roughly equivalent time blocks, with one signal occurring randomly within each time block, and at least two hours since the last random signal. When the semi-random signals occur, participants completed several measures either created or adapted for use in EMA protocols, which are described below ("Random Sampling Measures"). Previous experience with similar protocols indicates it takes approximately 2-3 minutes to complete these items.

Second, participants used fixed event responding to indicate the occurrence and consumption of meals and snacks. The *DSM-5* (APA, 2013) defines an objective binge episode as "eating, in a discrete period of time (for example, within any 2-hour period), an amount of food

that is definitely larger than most people would eat in a similar period of time under similar circumstances;" and experiencing a "sense of lack of control over eating during the episode (for example, a feeling that one cannot stop eating or control what or how much one is eating)." Participants indicated whether they consumed food and/or beverages, including the number of alcoholic drinks (if any). The occurrence of OBEs and SBEs was assessed via event sampling data that was recorded immediately after eating episodes, in which participants indicate whether they (a) experienced a subjective sense of a loss of control over their eating (i.e., "During this eating episode, did you feel you couldn't stop eating or controlling the type or quantity of food you were eating?"); and (b) ate an objectively large amount of food (i.e., "During this eating episode, did you eat more food than others might eat in similar circumstances or a similar period of time?"). Thus, eating episodes were defined as SBEs if participants endorsed (a) but not (b), whereas episodes were defined as OBEs if both (a) and (b) were endorsed. Contemplation of purging behavior was assessed via random sampling data, in which participants indicated whether the degree to which they had thought of using compensatory behaviors such as vomiting, laxative, or diuretic use. In addition to these behavioral ratings, affect was measured using the PANAS-X (Watson & Clark, 1994). All protocol responses were time-stamped, and the delay between signal and response was measured. The data were collected by graduate students in clinical psychology.

Screening Measures

Eating Disorder Diagnostic Scale

Eating Disorder Diagnostic Scale (EDDS) (Stice, Telch, & Rizvi, 2000). This 22-item self-report measure was used as a screener to identify participants who reported symptoms of eating pathology during mass testing sessions. The EDDS also was reviewed with participants at

the time participants complete the study. The EDDS is both a diagnostic measure of eating disorders and a continuous measure of eating pathology. The symptom composite score can be used as an overall assessment of eating pathology, with higher scores indicating more severe pathology. Stice, Fisher, and Martinez. (2004) also specified scoring algorithms to identify individuals who meet criteria for AN, BN, and BED diagnoses. The EDDS has demonstrated good reliability and validity. The convergent validity of the EDDS was evidenced by significant correlations between the EDDS symptom composite score and scores on other validated measures of eating disturbances (e.g., Eating Disorder Examination subscales, Three-Factor Eating Questionnaire), and criterion validity was demonstrated by the agreement between the eating disorder diagnoses from the EDDS and those from structured interviews (99% for AN, 96% for BN, and 93% for BED) (Stice et al., 2000, 2004). In regards to temporal stability, Stice et al. (2000) reported one week test-retest kappa coefficients ranging from .71 to .98 for specific diagnoses, and a one-week test-retest correlation coefficient of .87 for the symptom composite score. Stice et al. (2004) also demonstrated that the internal consistency of the EDDS symptom composite score is adequate (mean α =.89). In the present study the internal consistency (α) of the EDDS symptom composite score was .95.

Baseline Measures

Demographics Questionnaire

This measure assesses various demographic characteristics of participants. Such items assessed age, year in school, occupation, personal and household income, and ethnicity.

Random Sampling Measures

Appearance Evaluation Subscale of the State Self-Esteem Scale

Appearance Evaluation Subscale of the State Self-Esteem Scale (SSES) (Heatherton & Polivy, 1991) is a 6-item subscale of the SSES measures state appearance self-esteem. Each random sampling included these items in order to assess participants' thoughts about their appearance "at this moment." Responses are rated on a 5-point Likert-type scale ranging from 1 ("never") to 5 ("extremely"), with higher scores indicating greater body dissatisfaction. The SSES is sensitive in detecting fluctuations in self-esteem following manipulations (Heatherton & Polivy, 1991). Heatherton and Polivy (1991) found that the SSES demonstrated adequate discriminant and construct validity with other measures of self-esteem and affect, as well as adequate reliability and internal consistency (α=.92). In the present study the internal consistency (α) of the SSES score was .91, which was measured by the first completed SSES during the EMA protocol. Among women, lower levels of appearance self-esteem have been associated with exposure to thin ideal images (Tiggeman, Polivy, & Hargreaves, 2009), the endorsement of thin ideals, comparison to others' appearance, and others' evaluation of one's appearance (Darlow & Lobel, 2010).

Positive and Negative Affect Schedule-Expanded Form

Positive and Negative Affect Schedule-Expanded Form (PANAS-X) (Watson, Clark, & Tellegen, 1994). The PANAS-X is a 60-item measure that assesses the intensity of 11 specific affective states on a five-point Likert scale ranging from 1 ("very slightly or not at all") to 5 ("extremely"). For the present study, the Negative Affect (NA) and Guilt scales were used. Among undergraduates who rated their affect at the present moment, the PANAS-X demonstrated good internal consistency for each of these scales (NA: α=.85, Guilt: α=.86;

Serenity: α=.74; Attentiveness: α=.72), as well as adequate convergent and discriminant validity, as demonstrated by correlations with external measures of distress and psychopathology (Watson & Clark, 1994). In the present study, the internal consistencies (α) of the NA and Guilt scales (as measured by the first completed measure during the EMA protocol) were .91 and .96, respectively.

Severity of Negative Events. Six possible questions were asked at each random sampling that assessed participants' experience of and the impact of negative events since the last random sampling. Participants were asked if they experienced a negative since the last alarm; if so, they were asked to indicate the type of each event (i.e., interpersonal-family, interpersonal-friends, work/school, financial, health, or other) and how negative the event was (rated on a 5-point Likert-type scale ranging from "very slightly" to "extremely").

Contemplation of compensatory behavior. Three questions were asked at each random sampling to assess participants' thoughts about compensatory behaviors since the last random sampling. Participants were asked (1) "Since the last alarm sounded, have you thought about trying to restrict the amount of food you eat as a way to control your weight or alter your shape?"; (2) "Since the last alarm sounded, have you thought about exercising as a way to control your weight, alter your shape, or burn off calories?"; and (3) "Since the last alarm sounded, have you thought about using any other means (other than restricting your food or exercising) as a way to control your weight or alter your shape?" Each item is rated on a 5-point Likert-type scale ranging from "not at all" (1) to "very much" (5).

Analyses

Preliminary Screening

The database was constructed by the principal investigator and other graduate students involved in data collection. Data from baseline assessments were entered by undergraduate

research assistants and later examined for accuracy. The daily EMA data was downloaded directly from the Palm PDAs and merged with participants' baseline assessments. Frequencies and correlations were used to assess normality of distributions, possible floor or ceiling effects, and multicollinearity.

Analyses for Hypotheses

Hypothesis 1

To examine the type and frequency of eating episodes among the bulimic spectrum and control groups, descriptive information about normal eating episodes, SBEs, and OBEs was assessed from event sampling data recorded immediately after eating episodes, in which participants indicated whether they (a) experienced a subjective sense of a loss of control over their eating; and (b) ate an objectively large amount of food. Eating episodes were defined as SBEs if participants endorsed (a) but not (b), whereas episodes were defined as OBEs if both (a) and (b) were endorsed. Eating episodes were defined as normal eating episodes if neither (a) nor (b) were endorsed.

Differences in the mean number of OBE, SBE, and normal eating episodes per day between the Control and Bulimic Spectrum groups were compared using separate t-tests. Additionally, a second set of t-tests compared the mean number of OBE, SBE, and normal eating episodes per day between the PD and BN groups to assess possible differences within the Bulimic Spectrum group.

Hypothesis 2-4

Hypotheses 2, 3, and 4 involved examination of temporal relationships between data at random and event sampling data. Hierarchical linear modeling (HLM; Bryk & Raudenbush, 2002) was chosen to analyze this EMA data, as HLM is preferred for data in which time is

nested within person. HLM models between-subject moderators of within-subject relationships; that is, HLM assesses whether intra-individual variables (e.g., contemplation of purging) are related to between-subject characteristics (e.g., group membership), while maintaining statistical power and ecological validity. Level 1 equations are used to model within-subject relationships (i.e., repeated measures nested within persons). Separate regression equations are estimated for each person at Level 1. Level 2 equations use between-subject predictors to model inter-individual differences in the Level 1 relationships. The variability of the slopes and intercepts at Level 1 are modeled with the predictors at Level 2.

HLM requires that five assumptions be met. First, HLM assumes that observations are not independent of one another; that is, a subject's score on Day 4 may be influenced by her score on Day 2. The EMA data collected in present study met this assumption; for example, it is possible that a participant's responses on one day may influence her responses at a subsequent time. Second, HLM requires an adequate sample size in order to achieve satisfactory statistical power. For longitudinal data, it is advised that at least three time points of data are collected for each participant (Kreft, 1996); the EMA data collected in present study met this assumption study, as participants could be randomly signaled five times per day for ten days, resulting in a minimum of 50 possible time points of data per person, in addition to eating event data entered by participants. Third, HLM requires random grouping; that is, for levels above the bottom level, there should be a random sampling of individuals for all groups. Although this study did not meet this assumption due to recruitment of subjects, HLM is fairly robust to violations of this assumption. Fourth, HLM assumes normally distributed variables. Thus, the distributions of all dependent variables were examined to confirm this assumption was met. Last, HLM assumes that all level-1 errors have equal variance, which was also confirmed by exploratory analyses.

First, an unconditional model (i.e., with no predictors) was run for each outcome variable to assess the variance components and confirm that it was appropriate to add predictor variables to explain the variance in the intercepts of these variables (Bryk & Raudenbush, 2002). Bernoulli models were used when outcome variables were dichotomous (e.g., whether an OBE occurred). Level 1 variance components were significant (all *p*'s <.01), which confirmed the need for additional HLM analyses. All continuous predictor variables were grand mean centered.[1]

As described below, data were examined for missing data prior to running HLM analyses. HLM analyses handles missing data at Level 1 but not at Level 2 (Bryk & Raudenbush, 2002). Therefore, if participants did not complete random or eating event signals, this data was left missing at Level 1. In the Level 2 data, all participants had complete data (i.e., group membership, EDDS score) and were therefore included in all analyses.

Power Analysis

In order to appropriately plan for data collection and subsequent statistical analyses, it is necessary to determine the minimum sample size needed to achieve adequate statistical power. Cohen (1992) refers to statistical power as the probability of a statistical test to correctly reject the null hypothesis (p. 98).

A-priori power analysis is more complex with multilevel models, as power estimates depend on the extent to which model assumptions are met, the correlation between variables within levels, and the number of observations, individuals, and groupings in the model (Kreft & de Leeuw, 1998). However, Kreft and de Leeuw (1998) recommend that using maximum likelihood estimation generally yields the highest power and lowest probability of making a type

[1] Analyses were also run with group-centered continuous Level 1 predictors. There were no differences in the pattern of results or significance levels.

I error. In addition, it is recommended that the number of groups (i.e., persons) is larger than 20 to achieve sufficient power to detect cross-level effects (Kreft & de Leeuw; 1998). Kreft (1996) also advises that at least three time points of data are collected for each participant in longitudinal studies. It was anticipated that each participant would have a minimum possible 50 time points (five random signals for ten days), thereby meeting this recommendation. Taken together, in order to achieve sufficient power to detect the effects of interest in the present study, I aimed to achieve a sample size of at least 48 individuals.

CHAPTER III

RESULTS

Characteristics of the Sample

The sample (*N*=73) consisted of 33 individuals with BN, 9 individuals with PD, and 31 healthy controls. Of the participants who reported their ethnicity (*n*=23; 31.51%), 20 (86.96%) identified as Caucasian, and 3 (13.04%) identified as African American; the large amount of missing data reflects the fact that ethnicity data was inadvertently dropped mass testing data for some semesters. Table 1 displays means and standard deviations of baseline characteristics (i.e., age, BMI, and overall disordered eating symptomatology, as measured by the EDDS symptom composite score) for each of the three groups, and results of one-way ANOVAs that assessed group differences in these variables. Participants ranged in age from 18 to 33 (*M*=19.25 years; *SD*=2.45). There were no significant differences between the Bulimic Spectrum group (combined BN and PD groups; *M*=18.76, *SD*=1.10) and Controls (*M*=20.05, *SD*=3.63), *t*(53)=-1.58, *p*=.13, nor were there significant differences between the three groups (BN, PD, and Controls) in age (F[2,52]=1.83, *p*=.17). Participants' Body Mass Index (BMI) ranged from 17.07 to 30.17 (*M*=22.74, *SD*=3.02). There were no differences in BMI between the Bulimic Spectrum group (*M*=23.13, *SD*=2.72) and Controls (*M*=22.21, *SD*=3.38), *t*(70)=-1.28, *p*=.21. A one-way ANOVA indicated there was a significant effect of group for BMI (*F*[2,69]=3.60, *p*=.03). Student-Neuman-Keuls (SNK) post-hoc results indicated that individuals with BN had significantly higher BMIs than those with PD, though Controls did not differ significantly from

Table 1

Baseline Measures: Group Means, Standard Deviations, between-Subjects Effects, and Post-hoc Analyses

Dependent Variable	Bulimia Nervosa[1] n=33 *M (SD)*	Purging Disorder[2] n=9 *M (SD)*	Control[3] n=31 *M (SD)*	Between subjectseffects *F*	*p*	η_p^2	Student-Neuman-Keuls post-hoc results
Age	18.76 (1.15)	18.80 (0.84)	20.05 (3.63)	1.83	.17	.07	---
BMI	23.68 (2.60)	21.11 (2.21)	22.21 (3.38)	3.60	.03	.09	1>2
ED DS-sym	41.51 (10.38)	23.06 (7.86)	6.53 (6.34)	133.07	<.01	.79	1>2>3

Note. BMI=Body Mass Index; EDDS-sym=Eating Disorder Diagnostic Scale symptom composite score.

either the BN or PD groups. One individual (1.37%) did not report their BMI, and 18 individuals (24.66%) did not report their age.

Regarding disordered eating symptomatology, EDDS symptom composite scores were calculated for all participants. The Bulimic Spectrum group had significantly higher scores (M=37.56, SD=12.45) compared to Controls (M=6.53, SD=6.34), t(71)=-13.89, p<.001. A one-way ANOVA showed a significant group effect (F[2,70]=133.07, p<.01); SNK post-hoc results indicated that individuals with BN endorsed significantly more disordered eating symptomatology than those with PD, and both BN and PD groups endorsed more symptomatology than Controls.

Quality of EMA Data

Daily assessments were nested within each person (N=73). Of the 3,491 random signals, 2,383 were completed by participants, while 1,108 were skipped or ignored; of the 664 eating events, 643 were completed, while 21 were missing data. The mean length of participation in the study was 10.74 days (SD=1.40; range: 9 to 12 days).

Participants responded to an average of 33.64 random signals (SD=8.65) throughout the study, with the number of random signal responses ranging from 13 to 45. Participants recorded an average number of 8.81 eating events (SD=1.60) throughout the study, with the number of eating events ranging from 2 to 10. Participants did not report more than one eating episode per day. There was not a significant difference in the mean number of completed eating events between the Control (M=8.94, SD=1.39) and Bulimic Spectrum groups (M=8.71, SD=1.81), t(71)=.57, p=.57, nor was there a significant difference in the mean number of completed random signals (Control: M=31.94, SD=7.35; Bulimic Spectrum: M=33.17, SD=9.55), t(71)=-.60, p=.55.

Participants' compliance rates were calculated by dividing the number of completed entries by the total number of signals that were prompted (both random and event), resulting in an average compliance rate of 72.66%, with rates ranging from 35.00 to 95.00%.

Hypothesis 1

The mean frequencies of normal eating episodes, SBEs, and OBEs per day were examined within each group. Individuals with BN reported a total of 263 eating episodes, which consisted of 168 (63.9%) normal eating episodes, 34 SBEs (12.9%), and 61 (36.3%) OBEs. Individuals with PD reported 74 eating episodes, which consisted of 65 (87.8%) normal eating episodes, eight SBEs (10.8%), and one (1.4%) OBE. The Control group reported a total of 265 eating episodes, which consisted of 257 (97.0%) normal eating episodes, 4 SBEs (1.5%), and 4 (1.5%) OBEs. Independent samples t-tests compared the mean number of OBEs, SBEs, and normal eating episodes per day between the Bulimic Spectrum (n=33) and Control (n=31) groups. The Bulimic Spectrum group reported significantly more OBEs per day (M=.14, SD=.17) than the Control group (M=.01, SD=.04), $t(71)=-4.79$, $p<.001$, as well as significantly more SBEs per day than Controls (Bulmic Spectrum: M=.09, SD=.16; Control group: M=.01, SD=.03), $t(71)=-3.18$, $p=.003$. The Bulimic Spectrum group reported significantly fewer normal eating episodes (M=.52, SD=.28) per day compared to the Control group (M=.78, SD=.18), $t(71)=4.94$, $p<.001$.

To explore differences within the Bulimic Spectrum group, the BN (n=33) and PD (n=9) groups were compared on the mean frequencies of each type of eating episode per day. Individuals in the BN group reported significantly more OBEs per day (M=.17, SD=.17) compared to those in the PD group (M=.01, SD=.03), $t(40)=5.10$, $p<.001$, and fewer normal eating episodes per day (BN: M=.47, SD=.25; PD: M=.68, SD=.34), $t(40)=-2.05$, $p=.047$.

However, the frequency of SBEs per day in the BN group (M=.09, SD=.15) was not significantly different than that of those in the PD group (M=.09, SD=.20), t(40)=.13, p=.90.

Hypothesis 2

Antecedents of Dietary Restriction

Separate HLM analyses were used to examine negative affect (NA), guilt, and body dissatisfaction (i.e., SSES scores) as predictors of dietary restriction among the Bulimic Spectrum (n=33) and Control (n=31) groups. Dietary restriction (*EATTIME*) was operationalized as the time lapsed (in seconds) since 12:00 A.M. until the first eating episode recorded each day. The mean time lapsed until the eating episode of each day (M=12.37 hours, SD=2.36) did not differ significantly between the Control (M=12.03 hours, SD=2.15) and the Bulimic Spectrum groups (M=12.62 hours, SD=2.50), t(71)=-1.06, p=.29. Measurements of NA, guilt, and SSES were taken from the first random assessment of each day; if the first assessment of the day was an eating episode, these episodes (n=269; 35.63% of the first assessments of the day) were excluded from analyses due to the lack of measures preceding the eating episode. The following equations were used for these analyses:

$$EATTIME_{ti} = \pi_{00} + \pi_{10}*(First\_NA_{ti}) + r_{0i} + r_{1i}*(First\_NA_{ti}) + e_{ti}$$

$$EATTIME_{ti} = \pi_{00} + \pi_{10}*(First\_Guilt_{ti}) + r_{0i} + r_{1i}*(First\_Guilt_{ti}) + e_{ti}$$

$$EATTIME_{ti} = \pi_{00} + \pi_{10}*(First\_SSES_{ti}) + r_{0i} + r_{1i}*(First\_SSES_{ti}) + e_{ti}$$

As shown in Table 2, results indicated there was not a significant relationship between the first assessed NA (π_{10} = -433.973, SE = 415.57, t(64) =-1.044, p =.300), guilt (π_{10} = -248.720, SE =247.946, t(64) =-1.003, p = .320), or SSES (π_{10} = 161.425, SE = 219.938, t(72)= 0.734, p =.465) and the time lapsed to the first eating episode. To examine possible group differences in these relationships, group (0=Control; 1=Bulimic Spectrum) was subsequently entered at Level 2

Table 2

Antecedents of Restriction: Negative Affect, Guilt, and Body Dissatisfaction

	Restriction (time to first eating episode)			Restriction (mean intensity of thoughts per day)		
	π	*SE*	*t (df)*	π	*SE*	*t*
First NA						
Intercept (π_{00})	47398.707	1356.461	34.943 (64)**	1.487	0.145	10.286 (65)**
First NA (π_{10})	-433.973	415.57	-1.044 (64)	0.049	0.023	2.094 (65)*
Group effect:						
Intercept (π_{01})	110.881	2840.789	0.039 (63)	1.208	0.219	5.507 (64)**
First NA (π_{11})	-709.429	873.359	-0.812 (63)	0.075	0.035	2.139 (64)*
First Guilt						
Intercept (π_{00})	49293.403	1067.297	46.185 (64)**	1.408	0.132	10.691 (65)**
First Guilt (π_{10})	-248.720	247.964	-1.003 (64)	0.045	0.024	1.901 (65)
Group effect:						
Intercept (π_{01})	-1280.369	2269.237	-0.564 (63)	0.991	.252	3.923 (64)**
First Guilt (π_{11})	-1558.697	553.692	-2.815 (63)**	0.048	0.054	0.892 (64)
Table 2 (continued)						
First SSES						
Intercept (π_{00})	45632.939	1280.185	35.646 (72)**	1.392	0.121	11.490 (72)**
First SSES (π_{10})	161.425	219.938	0.734 (72)	0.021	0.014	1.490 (72)
Group effect:						
Intercept (π_{01})	1045.187	2667.153	0.392 (71)	0.891	0.220	4.045 (71)**
First SSES (π_{11})	35.832	460.071	0.078 (71)	0.001	0.028	0.019 (71)

Note. *$p < .05$, **$p < .001$. First NA= First randomly assessed Positive and Negative Affect Schedule-Expanded Form (PANAS-X) Negative Affect subscale score of the day. First Guilt=First randomly assessed PANAS-X guilt subscale score of the day. First SSES=First randomly assessed Appearance Evaluation subscale of the State Self-Esteem Scale of the day. Group: Bulimic Spectrum group=1; Controls=0. Level-1 variables were grand-centered; Group was uncentered at Level-2.

in each model as a predictor of both the intercept and the slope of NA, guilt, and SSES. The following equations were used for these analyses:

$$EATTIME_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_NA_{ti}) + \pi_{11}*(GROUP_i*First\_NA_{ti}) + r_{0i} + r_{1i}*(First\_NA_{ti}) + e_{ti}$$

$$EATTIME_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_Guilt_{ti}) + \pi_{11}*(GROUP_i*First\_Guilt_{ti}) + r_{0i} + r_{1i}*(First\_Guilt_{ti}) + e_{ti}$$

$$EATTIME_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_SSES_{ti}) + \pi_{11}*(GROUP_i*First\_SSES_{ti}) + r_{0i} + r_{1i}*(First\_SSES_{ti}) + e_{ti}$$

Results indicated that there was a significant cross-level interaction of group for guilt (π_{11}=-1558.697, *SE* = 553.692, *t*(63) =-2.815, *p* =.007), such that there was a negative relationship between guilt and time to first eating episode among individuals in the Bulimic spectrum group. No other group effects were significant (see Table 2).

Given the low frequency of eating episodes and the non-significant differences between groups in the time lapsed to first eating episode, a secondary analysis examined the relationship between first assessed negative affect, guilt, and body dissatisfaction and *thoughts of restriction* throughout the day. That is, it is possible that individuals in the Bulimic Spectrum group were more preoccupied with restriction despite being similar to the Control group in the time lapsed until eating, and therefore it would be useful to examine possible antecedents of such preoccupation. Participants' contemplation of thoughts of restriction at each random assessment were averaged throughout the day to serve as the dependent variable (the first assessment of the day was excluded from this average). Separate models were run with the first randomly assessed levels of NA, guilt, and body dissatisfaction of the day as Level 1 predictors of mean level of thoughts of restriction per day. Results indicated that higher levels of NA (π_{10} =0.049, *SE* =

0.023, t (65) = 2.094, p = .04) were associated with more intense thoughts of restricting, but there was not a significant effect for guilt or body dissatisfaction (see Table 2). Group (0=Control; 1=Bulimic Spectrum) was subsequently entered as a predictor of intercept at Level 2, as illustrated by the following equations:

$$Restriction\_thoughts_{ti}=\pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_NA_{ti}) + \pi_{11}*(GROUP_i*First\_NA_{ti}) + r_{0i} + r_{1i}*(First\_NA_{ti}) + e_{ti}$$

$$Restriction\_thoughts_{ti}=\pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_Guilt_{ti}) + \pi_{11}*(GROUP_i*First\_Guilt_{ti}) + r_{0i} + r_{1i}*(First\_Guilt_{ti}) + e_{ti}$$

$$Restriction\_thoughts_{ti}=\pi_{00} + \pi_{01}*(GROUP_i) + \pi_{10}*(First\_SSES_{ti}) + \pi_{11}*(GROUP_i*First\_SSES_{ti}) + r_{0i} + r_{1i}*(First\_SSES_{ti}) + e_{ti}$$

As shown in Table 2, there was a significant group effect on the intercept in each model, such that individuals in the Bulimic Spectrum group had more intense thoughts of restriction per day compared to Controls (First NA: π_{01}=1.208, SE = .219, t(64) =5.507, p<.001; First guilt: π_{01}=.991, SE = .252, t(64) =3.923, p =.001; First SSES: π_{01}=0.891, SE = .220, t(71) =4.045, p<.001). Furthermore, there was a significant cross-level interaction between group and the first assessed NA of the day (but not guilt or SSES), such that the first assessed NA predicted more intense thoughts of restriction for those in the Bulimic Spectrum group but not for those in the Control group (π_{11}=.077, SE = .035, t(64) =2.139, p =.036).

Consequences of Dietary Restriction

The consequences of dietary restraint were assessed using time lapsed until the first eating episode of the day (in seconds) since 12:00 A.M. as a Level 1 predictor of the occurrence of (1) subjective binge episodes (SBEs), (2) objective binge episodes (OBEs), and (3) contemplation of purging behavior (i.e., vomiting, laxative, or diuretic use) after eating episodes.

For these analyses, only individuals within the Bulimic Spectrum group (n=42) were included. The occurrence of OBEs and SBEs was assessed via event sampling data that was recorded immediately after eating episodes, and contemplation of purging behavior was assessed via the most proximal subsequent random sampling data after eating episodes, in which participants indicated whether the degree to which they had thought of using purging behaviors such as vomiting, laxative, or diuretic use. With the dichotomous dependent variables SBE (1=SBE, 0=normal eating episode) and OBE (i.e., 1=OBE, 0=normal eating episode), the following Bernoulli models were used:

OBE: $\log[P_{OBE}(1 - P_{OBE})] = \pi_{00} + \pi_{10}*(EATTIME_{ti}) + r_{0i}$

SBE: $\log[P_{SBE}(1 - P_{SBE})] = \pi_{00} + \pi_{10}*(EATTIME_{ti}) + r_{0i}$

For the dependent variable of contemplation of purging behaviors after eating episodes, the following equation was used (controlling for the time since the eating episode):

$$PURGE_{ti} = \pi_{00} + \pi_{10}*(EATTIME_{ti}) + \pi_{20}*(TIMESINCE_{ti}) + r_{0i} + e_{ti}$$

Results indicated that there was a significant relationship between the time lapsed until the first eating episode and the occurrence of a SBE (π_{10} =.000039, SE =.000017, t(232) =2.282, p =.023), such that longer time was associated with higher probabilities of a SBE. However, there was not a significant relationship between the time lapsed until first eating episode and the occurrence of OBEs (π_{10} =.000017, SE =.000012, t(252) =1.459, p =.146) or contemplation of purging behavior after eating (π_{10} =.000008, SE =.000005, t(147) =1.401, p=.163).

Additionally, the possible effect of the interaction of restraint and the occurrence of a binge (1=OBE or SBE; 0=normal eating episode) on contemplation of purging behavior after was investigated:

$$PURGE_{ti} = \pi_{00} + \pi_{10}*(EATTIME_{ti}) + \pi_{20}*(TIMESINCE_{ti}) + \pi_{30}*(BINGE_{ti}) +$$

$$\pi_{40}*(EATTIME_{ti} * BINGE_{ti}) + r_{0i} + e_{ti}$$

However, there was not a significant interaction between the time lapsed and the occurrence of an OBE or SBE on thoughts of purging after eating (π_{40} <.001, *SE* <.001, *t*(127) =0.578, *p*=.564); that is, the relationship between restraint and thoughts of purging did not differ based on an individual's loss of control over eating.

Hypothesis 3

To assess group differences in the anticipation of compensatory behaviors (i.e., restriction, exercise, purging behavior) among the Bulimic Spectrum (*n*=33) and Control (*n*=31) groups, separate models were run for thoughts of each type of compensatory behavior at random assessments. No predictors were entered at Level 1, and group membership was added as a predictor at Level 2 (0=Control, 1=Bulimic Spectrum). The following equations were used these analysis:

$$RESTRICT_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + r_{0i} + e_{ti}$$

$$EXERCISE_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + r_{0i} + e_{ti}$$

$$PURGE_{ti} = \pi_{00} + \pi_{01}*(GROUP_i) + r_{0i} + e_{ti}$$

Results indicated that there was a significant relationship between group membership and thoughts of compensatory strategies, such that individuals in the Bulimic Spectrum group reported more intense thoughts of restriction (π_{01} = 1.928, *SE* = .215, *t*(71) = 8.966, *p* <.001), exercise (π_{01} = 1.447, *SE* = .257, *t*(71) = 5.263, *p* <.001), and other compensatory behaviors (π_{01} = .994, *SE* = .224, *t*(71) = 4.438, *p* <.001) compared to Controls.

To examine possible differences between individuals within the Bulimic Spectrum group (*n*=42), Controls were excluded and group (0=PD, 1=BN) was entered as a Level 2 predictor of each outcome variable. Results were non-significant for restriction (π_{01} = .639, *SE* = .370, *t*(40) =

1.728, p=.092), exercise (π_{01} = .480, SE = .437, t(40) = 1.100, p=.278), and other compensatory behaviors (π_{01} = .374, SE = .467, t(40) = .801, p=.428).

Hypothesis 4

In regards to antecedents of the loss of control over eating, separate models were run to assess whether increases in NA, guilt, and/or severity of negative events predicted the occurrence of SBEs and OBEs among individuals in the Bulimic Spectrum group (n=42). Ratings of the severity of negative events were taken from the random assessment prior to eating episodes and entered as Level 1 predictors. Increases in NA and guilt prior to eating were assessed by entering eating episode NA or guilt ratings as Level 1 predictors, while also controlling for pre-eating NA or guilt ratings (lagged variables) at Level 1. Separate Bernoulli models were run for both SBEs and OBEs as outcome variables for each Level 1 predictor:

$$\log[P_{OBE}(1 - P_{OBE})] = \pi_{00} + \pi_{10}*(Pre\_NA_{ti}) + \pi_{20}*(NA_{ti}) + r_{0i} + r_{1i}*(Pre\_NA_{ti}) + r_{2i}*(NA_{ti})$$

$$\log[P_{SBE}(1 - P_{SBE})] = \pi_{00} + \pi_{10}*(Pre\_NA_{ti}) + \pi_{20}*(NA_{ti}) + r_{0i} + r_{1i}*(Pre\_NA_{ti}) + r_{2i}*(NA_{ti})$$

$$\log[P_{OBE}(1 - P_{OBE})] = \pi_{00} + \pi_{10}*(Pre\_Guilt_{ti}) + \pi_{20}*(Guilt_{ti}) + r_{0i} + r_{1i}*(Pre\_Guilt_{ti}) + r_{2i}*(Guilt_{ti})$$

$$\log[P_{SBE}(1 - P_{SBE})] = \pi_{00} + \pi_{10}*(Pre\_Guilt_{ti}) + \pi_{20}*(Guilt_{ti}) + r_{0i} + r_{1i}*(Pre\_Guilt_{ti}) + r_{2i}*(Guilt_{ti})$$

$$\log[P_{OBE}(1 - P_{OBE})] = \pi_{00} + \pi_{10}*(Negativity_{ti}) + r_{0i} + r_{1i}*(Negativity_{ti})$$

$$\log[P_{SBE}(1 - P_{SBE})] = \pi_{00} + \pi_{10}*(Negativity_{ti}) + r_{0i} + r_{1i}*(Negativity_{ti})$$

Results indicated that there was a significant relationship between increases in NA (π_{20} = .233, SE = .066, t(37) = 3.532, p=.001) and increases in guilt (π_{20} = .586, SE = .117, t(37) = 5.017, p <.001), but not pre-eating negativity of events (π_{10} = .087, SE = .686, t(17) = .127, p=.900) and the occurrence of OBEs. There was not a significant relationship between increases

in NA (π_{20} = .053, *SE* = .067, *t*(37) =.792, *p*=.434), increases in guilt (π_{20} = .151, *SE* = .080, *t*(37) = 1.880, *p*=.068), or pre-eating ratings of negativity of events (π_{10} = .995, *SE* = .539, *t*(21) = -1.847, *p*=.079) and the occurrence of SBEs.

To assess possible consequences of the loss of control over eating among the Bulimic Spectrum group (*n*=42), the type of eating episode (i.e., OBE, SBE, normal eating episode) was assessed as predictors of post-eating episode contemplation of restriction, exercise, or purging behavior (i.e., use of vomiting, laxatives, and/or diuretics), as well as post-eating ratings of body image dissatisfaction and changes in overall negative affect and guilt. Separate models were run for each of the aforementioned variables as outcome variables, while controlling for the time elapsed since the eating episode. Each type of eating episode was dummy coded (i.e., "1" indicating the individual experienced the specific type of eating episode and "0" as the reference group) and entered as Level 1 predictors. The following analyses used normal eating episodes as the reference group:

$$Post\_Restriction_{ti} = \pi_{00} + \pi_{10}*(TIMESINCE_{ti}) + \pi_{20}*(OBE\_DUMMY_{ti}) + \pi_{30}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

$$Post\_Exercise_{ti} = \pi_{00} + \pi_{10}*(TIMESINCE_{ti}) + \pi_{20}*(OBE\_DUMMY_{ti}) + \pi_{30}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

$$Post\_Purging_{ti} = \pi_{00} + \pi_{10}*(TIMESINCE_{ti}) + \pi_{20}*(OBE\_DUMMY_{ti}) + \pi_{30}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

$$Post\_SSES_{ti} = \pi_{00} + \pi_{10}*(TIMESINCE_{ti}) + \pi_{20}*(OBE\_DUMMY_{ti}) + \pi_{30}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

$$Post\_NA_{ti} = \pi_{00} + \pi_{10}*(TIMESINCE_{ti}) + \pi_{20}*(Eating\_NA_{ti}) + \pi_{30}*(OBE\_DUMMY_{ti}) + \pi_{40}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

$$Post\_Guilt_{ti} = \pi_{00} + \pi_{10}*TIMESINCE_{ti} + \pi_{20}*(Eating\_Guilt_{ti}) + \pi_{30}*(OBE\_DUMMY_{ti}) + \pi_{40}*(SBE\_DUMMY_{ti}) + r_{0i} + e_{ti}$$

Results indicated that compared to normal eating episodes, the occurrence of OBEs predicted more intense thoughts of restriction after eating (π_{20} = .810, *SE* =.249, *t* (106) = 3.122, *p*=.002), whereas the results for the occurrence of SBEs were not significant (π_{30} = .641, *SE* =.338, *t* (106) = 1.895, *p*=.061).

Compared to normal eating episodes, there were no significant relationships between the occurrence of OBEs or SBEs and the intensity of thoughts of exercise (OBE: π_{20} = .183, *SE* =.272, *t* (103) = .673, *p*=.502; SBE: π_{30} = -.050, *SE* =.314, *t* (103) = -.157, *p*=.876), thoughts of purging behavior (OBE: π_{20} = .083, *SE* =.212, *t* (129) = .391, *p*=.696; SBE: π_{30} =.204, *SE* =.259, *t* (129) = .791, *p*=.430), body dissatisfaction (OBE: π_{20} = .947, *SE* =.631, *t* (176) = 1.493, *p*=0.137; SBE: π_{30} = 1.610, *SE* =.806, *t* (176) = 1.998, *p*=.05), increased NA (OBE: π_{30} = -.912, *SE* =.801, *t* (170) = -1.139, *p*=.256; SBE: π_{40} = .266, *SE* =.952, *t* (170) = .279, *p*=.780), or increased guilt (OBE: π_{30} = -1.289, *SE* =.740, *t* (170) = -1.741, *p*=.084; SBE: π_{40} = -.383, *SE* =811, *t* (170) = -.472, *p*=.638) after eating.

Exploratory Analyses

Given that many of the expected relationships between affect and eating behaviors were not observed, exploratory analyses were pursued in order to examine other processes that may have influenced participants' eating behaviors. As previous findings have demonstrated a relationship between affective lability and disordered eating (Lavender et al., 2013), the relationship between affective lability, binge episodes, and overall disordered eating symptomatology (as measured by the EDDS symptom composite score) was examined. Affective lability was assessed by calculating the Mean Squared Successive Difference (MSSD)

for negative affect and guilt scores for each individual. The MSSD is a measure of point-to-point variability in a time series and represents an individual's average variability in affect over time (Witte, Fitzpatrick, Joiner, & Smith, 2005; Woyshville, Lackamp, Eisengart, & Gilliland, 1999), where *n* is number of elements in the time series, the *i*th element of which is denoted x_i:

$$MSSD = \frac{\sum_{i=1}^{n-1}(x_{i+1} - x_i)^2}{n-1}$$

Two independent samples t-tests were conducted to assess group differences in the lability of NA and guilt. There were significant differences between groups in NA MSSD, $t(71)=-3.14$, $p=.002$, and guilt MSSD, $t(71)=-4.46$, $p<.001$, such that individuals in the Bulimic Spectrum group exhibited significantly greater lability in NA ($M=19.06$, $SD=13.85$) and guilt ($M=13.88$, $SD=12.05$) compared those in the Control group (NA MSSD: $M=8.89$, $SD=13.47$; guilt MSSD: $M=3.90$, $SD=6.96$).

Table 3 displays bivariate correlations between NA MSSD, guilt MSSD, OBE frequency, SBE frequency, and EDDS symptom composite scores. Higher NA MSSD and guilt MSSD scores were associated with higher frequencies of OBEs (NA MSSD: $r[71]=.28$, $p=.02$; guilt MSSD: $r[71]=.53$, $p<.001$) and higher levels of overall disordered eating psychopathology (NA MSSD: $r[71]=.29$, $p=.01$; guilt MSSD: $r[71]=.44$, $p<.001$). However, there was not a significant relationship between NA MSSD or guilt MSSD scores and the frequency of SBEs (NA MSSD: $r[71]=.07$, $p=.57$; guilt MSSD: $r[71]=.10$, $p=.38$).

As previously discussed, the affect regulation model is a commonly accepted conceptualization of the function of bulimic symptomatology, which posits that negative affect increases prior to binge episodes and decreases after binge episodes. Furthermore, it is often believed that individuals with eating disorders engage in binge episodes as a means to avoid or

Table 3

Bivariate Correlations between NA MSSD, Guilt MSSD, SBE Frequency, OBE Frequency, and EDDS-sym Scores (N=73)

	NA MSSD	Guilt MSSD	SBE frequency	OBE frequency	EDDS-sym
NA MSSD	---				
Guilt MSSD	.70**	---			
SBE frequency	.07	.10	---		
OBE frequency	.28*	.53**	.03	---	
EDDS-sym	.29*	.44**	.35**	.46**	---

Note. *$p < .05$, **$p < .01$. NA=Positive and Negative Affect Schedule-Expanded Form (PANAS-X) Negative Affect subscale score; Guilt= PANAS-X guilt subscale score; MSSD=Mean Squared Successive Difference; OBE=Objective Binge Episode; EDDS-sym=Eating Disorder Diagnostic Scale symptom composite score.

escape from adverse emotional states (Hayes et al., 2004; Lillis, Hayes, & Levin, 2011). If these assumptions are true, then the occurrence of binge episodes should result in a net decrease in negative affect from pre- to post-eating, thereby demonstrating effective affect regulation.

Thus, in order to evaluate whether OBEs and/or SBEs *effectively* regulate affect, net changes in affect from pre- to post-eating episode were assessed among individuals in the Bulimic Spectrum group (n=42). Eating episodes (OBEs: 1=OBE; 0=normal eating episode; SBEs: 1=SBE; 0=normal eating episode) reported during the EMA protocol were entered as level-1 predictor variables of net changes in affect (i.e., post-eating episode affect level – pre-eating episode affect level), while controlling for the time elapsed from pre- to post-eating episodes (i.e., *TIME*). Difference scores were calculated by subtracting the pre-eating affect level from the post-eating affect level, so that positive values represented increases in affect levels. Separate HLM models were run with OBEs (1=OBE; 0=normal eating episode) and SBEs (1=SBE; 0=normal eating episode) as level-1 predictors of differences in negative affect (NA) and guilt from pre- to post-eating.

$$NA\_difference_{ti} = \pi_{00} + \pi_{10}*OBE_{ti} + \pi_{20}*TIME_{ti} + r_{0i} + e_{ti}$$

$$Guilt\_difference_{ti} = \pi_{00} + \pi_{10}*OBE_{ti} + \pi_{20}*TIME_{ti} + r_{0i} + e_{ti}$$

$$NA\_difference_{ti} = \pi_{00} + \pi_{10}*SBE_{ti} + \pi_{20}*TIME_{ti} + r_{0i} + e_{ti}$$

$$Guilt\_difference_{ti} = \pi_{00} + \pi_{10}*SBE_{ti} + \pi_{20}*TIME_{ti} + r_{0i} + e_{ti}$$

Results indicated that, compared to normal eating episodes, the occurrence of OBEs was not related to significant net changes in negative affect (π_{10} =.004, SE =1.032, t(103) = .004, p=.997) or guilt (π_{10}=.672, SE=.779, t(106) = .863, p=.390). The occurrence of SBEs was associated with significant decreases in negative affect (π_{10} = -2.243, SE = 1.096, t(100) = -

2.046, p=.043), but not significant changes in guilt (π_{10} = -.903, SE = .842, t(104) = -1.071, p =.286).

CHAPTER IV

DISCUSSION

The present study assessed daily cognitions, emotions, and eating behaviors of individuals with bulimic spectrum disorders (i.e., BN and PD) as compared to healthy controls. The present findings either fully (i.e., hypotheses 1 and 3) or partially (i.e., hypotheses 2 and 4) supported hypotheses and can be interpreted in light of existing theoretical models of disordered eating behavior. Results illustrated the distinction between cognitive and behavioral aspects of dietary restriction, as well as phenomenological similarities and differences between SBEs and OBEs among individuals with BN and PD. Analyses also demonstrated differential relationships between eating psychopathology, overall negative affect, and guilt and highlighted the potential importance of affective lability. Furthermore, the present study expands upon theoretical understandings of bulimic spectrum disorders and PD, and provides clinically useful data that could be applied in the treatment of such disorders.

When assessing the antecedents of a behavioral aspect of dietary restriction (i.e., time lapsed until the first eating episode), there were unexpected findings, in that there were not significant relationships between individuals' level of negative affect, guilt, or body dissatisfaction at the beginning of the day and subsequent delays until eating. Furthermore, there was a negative relationship between guilt and time to the first eating episode among individuals in the Bulimic Spectrum group, such that higher levels of initial guilt were associated with shorter times lapsed until eating. Although counter to what was expected, this result could

suggest that when individuals with BN or PD experience high levels of guilt they feel compelled to eat sooner, perhaps as a means of coping with this emotion.

However, the lack of expected relationships between affect, body image, and restriction may also be due to methodological concerns regarding the measurement of dietary restriction. First, no participant reported more than one eating episode per day; this raises questions regarding the fidelity of participants' reporting of eating episodes, as previous EMA research with disordered eating populations and healthy controls has observed more eating episodes among participants (e.g., Berg et al., 2013). Second, the time lapsed until the first eating episode of the day assessed the degree to which participants delayed eating, rather than the degree to which they limited their caloric intake; therefore, the time lapsed until the first eating episode may be an inaccurate proxy of restriction.

Despite the generally unexpected findings regarding behavioral aspects of dietary restriction, follow-up analyses demonstrated that individuals in the Bulimic Spectrum group reported more intense *thoughts* of restriction per day compared to Controls. Furthermore, higher levels of initial negative affect, but not guilt or body dissatisfaction, were predictive of more intense thoughts of restriction throughout the day among those in the Bulimic Spectrum group. This is a notable finding, given that Fairburn (2008, 2014) makes a distinction between dietary restriction and restraint. That is, restriction refers to a behavioral phenomenon in which individuals under-eat relative to their caloric needs, whereas restraint refers to a cognitive phenomenon in which individuals are preoccupied with attempting to limit their intake. According to Fairburn (2014), it is restraint, rather than actual restriction, that leads to a

vulnerability to binge, and therefore treatment of eating disorders (i.e., CBT-E) focuses on reducing cognitive preoccupation with restriction (i.e., restraint).

Furthermore, the present findings suggest that addressing negative affect may be particularly important in reducing individuals' cognitive efforts to restrict their intake. That is, it may be that individuals attempt to restrict their intake when they experience negative emotion, and interventions could target such maladaptive coping behaviors. It is notable that guilt may not precipitate restraint. Just as previous studies (e.g., Davis et al., 1988; Powell & Thelen, 1996; Rebert et al., 1991) have found that different facets of negative affect precipitate binge episodes, different facets of negative affect besides guilt (e.g., anger, distress, nervousness) should be further investigated as antecedents of restraint and possible targets for intervention. In addition, the lack of relationship between initial state body dissatisfaction and subsequent restraint was unexpected given previous research that has found that body dissatisfaction predicts dieting (e.g., Stice, Mazotti, Krebs, & Martin, 1998; Stice, Nemeroff, & Shaw, 1996). However, it is possible that state levels of body dissatisfaction become more variable throughout the day as events and stressors occur, including appearance comparisons (Leahey, Crowther, & Ciesla, 2011). Thus, the first assessment of state body dissatisfaction of each day may not have captured fluctuations that could influence subsequent restraint.

Just as NA predicted restraint among those in the Bulimic Spectrum group, increases in pre-eating NA as well as guilt predicted the occurrence of OBEs, but not SBEs. These findings partially support the first tenet of the Affect Regulation Model and illustrate contextual differences between OBEs and SBEs. That is, increases in individuals' level of negative affect,

including guilt, may be related to the severity and size of binge episodes and be a distinguishing factor in whether an individual engages in an SBE or OBE. Whereas increases in NA and guilt predicted OBEs, it is notable that restriction (as measured via time lapsed until eating) predicted SBEs, but not OBEs or levels of post-eating contemplation of purging. This is consistent with the findings of Kerzhnerman and Lowe (2002), who found that higher levels of dieting intensity were related to more frequent SBEs but not OBEs. Thus, it may be that SBEs, in which individuals do not consume objectively large amounts of food, but still feel a loss of control over their eating behavior, are associated with more successful efforts to delay and/or restrict intake, which may explain their perceptions of consuming normal portions as "binge" episodes. Although the present findings did not address this question, future investigation should investigate whether individuals who engage in SBEs are generally more successful with dietary restriction, either by delaying or limiting intake, and whether engaging in SBEs is indicative of a progression from restrictive to bulimic symptomatology. Such an investigation may be particularly informative in the conceptualization of PD.

Additionally, the occurrence of OBEs predicted more intense thoughts of restriction after eating compared to normal eating episodes, although this was not the case for SBEs. Thus, the quantity of food consumed during a binge episode may a particularly important maintaining factor in the cycle of bulimic symptomatology when individuals engage in OBEs, in that these individuals may attempt to compensate for the calories consumed by focusing on restriction and subsequently engaging in dietary restraint. This thereby increases the likelihood of future binge

episodes, which is consistent with Restraint Theory (Herman & Polivy, 1975, 1980, 1983; Polivy & Herman, 1985).

Given the aforementioned differences between OBEs and SBEs, interventions for individuals who engage in OBEs may benefit by directly addressing skills to regulate pre-eating emotions and post-eating urges to restrict; however, such interventions may not be as relevant for those with PD, who do not engage in OBEs. However, given that NA predicted cognitive restraint for all individuals in the Bulimic Spectrum group and restriction was associated with SBEs, individuals with PD (who report SBEs but not OBEs) may benefit more from interventions and emotion regulation strategies to address affective changes that precipitate dietary restriction and restraint.

The lack of relationship between pre-eating severity of negative events and binge episodes was unexpected given previous EMA research documenting relationships between stressful events and bulimic symptomatology (Goldschmidt et al., 2014; Smyth et al., 2007). It could be that individuals had difficulty remembering or appraising events that occurred since the last random signal. Moreover, selective attention and memory biases for schema-related (e.g., body or food-related) stimuli have been observed among individuals with eating disorders (Brooks, Prince, Stahl, Campbell, & Treasure, 2011; Cooper, Anastasiades, & Fairburn, 1992; King, Polivy, & Herman, 1991; Ruiz, del Consuelo Escoto Ponce de León, & Díaz, 2008; Sebastian, Williamson, & Blouin, 1996), which may have contributed to a lack of recall of other events during the EMA protocol. However, the low frequency of negative events reported throughout the EMA protocol among the participants in the Bulimic Spectrum group (*M*=5.21

events per participant, *SD*=5.03) may have accounted for the lack of relationship between the severity of negative events and eating behaviors.

According to Hypothesis 4, it was also expected that the occurrence of SBEs and OBEs would predict subsequent contemplation of purging behavior, body dissatisfaction, NA, and guilt. Contrary to expectations, OBEs or SBEs did not predict post-eating body dissatisfaction, increased levels of negative affect or guilt, or thoughts of exercise or purging. Despite some recent evidence demonstrating decreases in NA following binge episodes (e.g., Berg et al., 2013), a previous review of EMA studies of binge eating demonstrated increases in NA after binge episodes (Haedt-Matt & Keel, 2011). These inconsistencies suggest that binge eating may not be the maintaining factor of bulimic symptomatology, contrary to what is purported by the Affect Regulation Model. However, it is notable that individuals in the Bulimic Spectrum group reported more intense thoughts of restriction, exercise, and purging compared to Controls throughout the day, and there were no significant differences in the intensity of these thoughts between individuals with BN and those with PD, perhaps suggesting that these individuals anticipate compensatory behavior as a means of affect regulation, which also may reduce inhibitions toward binge eating. These results are consistent with the Anxiety Reduction Model, in that the affective change that results from compensatory behavior, rather than binging, may be a more powerful maintaining factor in bulimic symptomatology. Additionally, the lack of relationships between binging and post-eating affect, thoughts of purging, and body dissatisfaction also may be due to this study's methodology, in that the random assessment after eating episodes may have been too distal from the eating episode to capture changes in these

variables. Thus, it would be useful for future EMA assessments to implement assessments at shorter intervals after eating episodes. Furthermore, unreported eating episodes may have occurred, which could have impacted affective changes.

Exploratory analyses yielded intriguing findings regarding affect lability and eating psychopathology, which were consistent with prior research (Lavender et al., 2013). Individuals in the Bulimic Spectrum group exhibited greater lability in negative affect and guilt compared to those in the Control group, and higher levels of lability in negative affect, but not guilt, were related to higher levels of eating psychopathology and OBEs, yet not SBEs. Such findings suggest that addressing affect variability via emotion regulation strategies may be particularly important in the treatment of disordered eating, particularly those who engage in OBEs. Furthermore, these findings may partially account for the success of interventions that employ such strategies (i.e., Dialectical Behavior Therapy) in in the treatment of binge eating (Safer, Telch, & Agras, 2001).

In addition, exploratory analyses demonstrated that OBEs do not serve as effective means of decreasing levels of negative affect or guilt from pre- to post-eating episodes. These findings may be particularly useful in providing psychoeducation for individuals with bulimic spectrum disorders, as they often believe that engaging in binging and/or purging will alleviate or reduce the intensity of aversive emotions. At the same time, it is notable that SBEs were associated with net decreases in NA, but not guilt, among individuals in the Bulimic Spectrum group. It may be that to some extent, eating normal quantities of food, even when an individual feels as though it is "too much" food or feels guilty for eating it, may play a role in reducing some

negative emotions. This net reduction of negative affect associated with SBEs may be a normative phenomenon, as demonstrated by the finding that four individuals in the Control group reported one SBE each during the EMA protocol. However, when individuals consume objectively large quantities, the ensuing negative emotion and guilt may outweigh this effect.

Though there were only nine individuals with PD in the present study, the results provide tentative evidence to inform the conceptualization and support the clinical significance of this syndrome. To date, there are no published studies that have examined the topography of PD symptomatology using EMA methods. Consistent with previous research (Binford & le Grange, 2005; Fink et al., 2009), individuals with BN evidenced more severe eating psychopathology than those with PD, though those with PD evidenced more severe eating psychopathology than healthy controls. In addition, there was not a significant difference in the frequency of SBEs between those with BN and those with PD, yet individuals with PD reported more SBEs than controls. However, it is also important to note that due to the small sample size these results are underpowered. Nevertheless, these findings are consistent with prior studies that suggest that the loss of control over eating, rather than the amount of food consumed, is the characteristic of binge eating that accounts for clinically significant levels of distress, and is arguably what leads individuals to engage in subsequent compensatory behaviors (Mond et al., 2006; Niego et al., 1997; Pratt et al., 1998). Furthermore, this finding replicates previous observations of loss of control over eating among individuals with PD (Forney, Haedt-Matt, & Keel, 2014). Furthermore, the finding that SBEs were associated with longer delays until eating could suggest that individuals with PD may adhere to rigid dietary rules (which also supports Restraint Theory)

and find normal amounts of food to be highly anxiety provoking due to a fear of weight gain. Future research should further investigate restraint in PD and whether these individuals progress to engaging in OBEs over time. This evidence would suggest PD is not a distinct disorder but rather lies along a continuum of bulimic symptomatology.

Although this study provides valuable information regarding the antecedents and consequences of restraint and losses of control over eating among individuals with bulimic spectrum disorders, it is not without limitations. First, all data in the study was self-reported by participants. Despite inherent problems with the use of self-report measures, this method is the most time and cost-efficient way of assessing internal states and behaviors, especially when using EMA protocols. Another limitation is participant compliance during the study. It is important to note the amount of missing random data, as well as the fact that individuals reported at most one eating episode per day. This may have resulted in inaccurate and/or incomplete representations of participants' thoughts, emotions, and behaviors throughout the study. However, the amount of missing data was comparable to other studies using EMA protocols (Jones, Crowther, & Ciesla, 2014).

As previously mentioned, the measurement and operationalization of dietary restriction may have limited the ability to find relationships with variables of interest. While the time lapsed until the first eating episode assesses the degree to which participants delayed eating, it does not provide information regarding their caloric intake, nor was their wake time assessed, which prevented the assessment of time since awakening. It would be more accurate for future studies to utilize methods that can provide estimates of caloric intake throughout the day.

Furthermore, it is important to note that four individuals in the Control group each reported one SBE, while another four individuals in the Control group reported one OBE each during the EMA protocol. While this may suggest that occasional binge eating is common among college-aged individuals, the use of the EDDS as a screening measure may have been insufficient to screen out individuals with low frequencies of disordered eating behaviors. It may be more effective for screening measures to explicitly define binge episodes and/or ask individuals to describe these episodes so as to reduce possible ambiguities about their eating behaviors.

Also, as noted previously, the intervals between random assessments may have been too long to adequately capture fluctuations in affect, particularly before and after eating episodes. If possible, it may be useful for future studies to implement shorter intervals. At the same time, the use of more frequent random sampling may raise concerns regarding potential participant burden and decreased compliance. Thus, perhaps future studies could utilize technology to implement more frequent assessments focusing particularly on the times surrounding eating episodes.

Lastly, while this study assessed participants' contemplation of compensatory behavior, it was not possible to assess whether they engaged in such behavior. Although contemplation of compensatory behavior was assumed to be related to participants' likelihood of engaging in these behaviors, contemplation may have been an inaccurate proxy for actual engagement in compensatory behavior.[2] Therefore, inferences regarding individuals' contemplation of compensatory behavior must be interpreted with caution.

[2] The correlation between baseline EDDS purging frequency and participants' mean levels of contemplation of purging correlation was calculated: r=.582, p<.001.

Despite the aforementioned limitations, the present study yielded important information regarding the cognitions, affect, and behaviors of individuals with BN and PD in a naturalistic environment. Results expand upon Restraint Theory in demonstrating the distinction between cognitive and behavioral aspects of restriction, and suggest that interventions that specifically target cognitive efforts to restrict, such as CBT-E, would be particularly applicable for bulimic spectrum disorders. Results also illustrated differences in the antecedents and consequences between SBEs and OBEs, and suggest that the occurrence of OBEs is associated with more problems with emotion regulation and eating psychopathology compared to SBEs.

The results partially supported the Affect Regulation Model, in that increases in negative affect and guilt precipitated OBEs, yet OBEs did not effectively regulate affect. It appears that affect lability may be a particularly salient characteristic of individuals who engage in OBEs, which suggests a continued need for interventions to address emotion regulation skills for individuals with bulimic spectrum disorders. In addition, as it may be that participants' anticipation of compensatory behavior provided a means of regulating affect throughout the day and/or reduced inhibitions for binge eating, there is a need to further examine the affective antecedents and consequences of purging behavior, which may lend additional support for the maintaining role of purging behavior in bulimic symptomatology, as suggested by the Anxiety Reduction Model (Rosen & Leitenberg, 1982).

This study also lends insight into the topography of symptoms among individuals with PD. Although engaging in OBEs (as reported by those with BN) was associated with the most severe psychopathology and emotion dysregulation, results suggested there were

phenomenological similarities (e.g., a subjective sense of loss of control over eating, dietary restraint, anticipation of compensatory behavior) between the symptoms of individuals with PD and those with BN, which is consistent with the transdiagnostic model of eating disorders (Fairburn et al., 2003). Therefore, it is reasonable to suggest that despite some differences in symptomatology between BN and PD, similar interventions could be applied to both populations, as demonstrated by a growing body of research (Fairburn, 2009; Tasca, Maxwell, Bone, Trinneer, Balfour, & Bissada, 2012). Thus, rather than focusing on the nosology and diagnostic boundaries of eating disorders, perhaps it would be more effective for future studies to continue to address the observed cognitions, emotions, and behaviors among those with eating disorders to develop and apply interventions that target the mechanisms that maintain a variety of forms of eating psychopathology.

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APPENDICES

APPENDIX A

CONSENT FORMS

Consent Form for participants with BN or PD

Study Title: Cognitive Rumination, Affect, and Body Image
Principal Investigator: Janis H. Crowther, Ph.D., Professor of Psychology

You are being invited to participate in a research study. This consent form will provide you with information on the research project, what you will need to do, and the associated risks and benefits of the research. Your participation is voluntary. Please read this form carefully. It is important that you ask questions and fully understand the research in order to make an informed decision. You will receive a copy of this document to take with you.

Purpose: We are interested in examining the role of several factors that may contribute to disordered eating in women, including cognitive rumination, affect, and thoughts and behaviors related to their body. This is of particular interest due to the prevalence of disordered eating among women and the negative impact it can have on many areas of life.

Procedures: If you decide to participate, you will be asked to complete a brief interview followed by a battery of paper-and-pencil questionnaires. The interview and questionnaires should take approximately 60 to 75 minutes. You will then be given a Palm Z22 and instructed on how to use it. For the next 10 days, your Palm Z22 will signal at five semi-random times per day between 8:30 am and 11:30 pm. Each time you receive a random signal, you will have a series of questions to answer that will take approximately 5-6 minutes to complete. During these 10 days, you will also be asked to respond to questions on the Palm Z22 after every beverage, snack, or meal that you consume. These questions will also take approximately 1-3 minutes to complete each time. At the end of the 10-day period, we will ask you to come to the research lab and return the Palm Z22.

Benefits: This research may not benefit you directly. However, your participation in this study will help us to better understand the day-to-day relationship between thoughts, mood, and body image among women who report some difficulties with eating. This study has the potential to add to our knowledge and help us to develop more effective strategies for treatment of disordered eating.

Risks and Discomfort: Because some of the questions asked in this study address sensitive issues and behaviors, it is possible that you may experience emotional distress during or after completing the study. If you experience more than mild discomfort, we encourage you to contact one of the facilities on the attached page. If you like, we can assist you with the referral.

Privacy and Confidentiality: The responses you provide during this study will be completely confidential. Your signed consent form will be kept separate from any date you provide. Your responses will be identified only by a unique ID number, not your name. Consent forms will be kept in a locked file cabinet in my faculty office, and data records will be kept in a locked file cabinet in my laboratory office. My laboratory office can only be accessed by me and my research assistants. Only the researchers involved in the current study will have access to these records.

Compensation: If you part in this project, you may receive up to $125.00 depending on your involvement, and you will be helping the researchers to better understand what contributes to the continuation of disordered eating among women. You will be compensated as follows:

- $5 for completing the interview
- $5 for completing the battery of self-report questionnaires
- Up to $90 ($9 per day) for completing the 10 days of data collection using the Palm Z22.
- Additional incentive for compliance: You may receive up to an additional $25.00 depending on your level of participation in answering the questions on the Palm Z22. Specifically, if you respond to 25 to 50% of the signals over the 10-day period, you will receive an additional $7; if you respond to 51 to 75% of the signals over the 10-day period, you will receive an additional $15, and if you respond to 76 to 100% of the signals over the 10-day period, you will receive an additional $25.

Voluntary Participation: Taking part in this research study is entirely up to you. You may choose not to participate or you may discontinue your participation at any time without penalty or loss of benefits to which you are otherwise entitled. You will be informed of any new, relevant information that may affect your health, welfare, or willingness to continue your study participation.

Contact Information: If you have any questions or concerns about this research, you may contact Dr. Janis H. Crowther at 330-672-2090 or jcrowthe@kent.edu. This project has been approved by the Kent State University Institutional Review Board. If you have any questions about your rights as a research participant or complaints about the research, you may call the IRB at 330.672.2704.

Consent Statement and Signature: I have read this consent form and have had the opportunity to have my questions answered to my satisfaction. I voluntarily agree to participate in this study. I understand that I must return the Palm Z22 and I understand that a copy of this consent will be provided to me for future reference.

_____ _____

Participant Signature **Date**

Kent State University Campus Facilities

- Kent State University Psychological Clinic, Room 176 Kent Hall, (330) 672-2372
- Kent State Students Only: University Psychological Services, 2nd Floor DeWeese Health Center, (330) 672-2487

Other Facilities

- Coleman Professional Services, 5982 Rhodes Road, Kent, Ohio (330) 673-1347
- Town Hall II, 123 S. Water Street, Kent, Ohio 44240, Kent, Ohio (330) 678-4357
- Portage Path Community MHC, 340 S. Broadway, Akron, Ohio (330) 253-4118
- Behavioral Health Services, Summa Health System, St. Thomas Hospital, 444 N. Main Street, Akron, OH (330) 379-5906
- Nova Behavioral Health, Inc., Canton, 832 McKinley Avenue N.W., Canton, Ohio (330) 455-9407

Consent form for control group participants

Study Title: Cognitive Rumination, Affect, and Body Image
Principal Investigator: Janis H. Crowther, Ph.D., Professor of Psychology

You are being invited to participate in a research study. This consent form will provide you with information on the research project, what you will need to do, and the associated risks and benefits of the research. Yow participation is voluntary. Please read this form carefully. It is important that you ask questions and fully understand the research in order to make an informed decision. You will receive a copy of this document to take with you.

Purpose: We are interested in examining the role of several factors that may be related to eating in women, including cognitive rumination, affect, and thoughts and behaviors related to their body. This is of particular interest due to the prevalence of disordered eating among women and the negative impact it can have on many areas of life.

Procedures: If you decide to participate, you will be asked to complete a brief interview followed by a battery of paper-and-pencil questionnaires. The interview and questionnaires should take approximately 60 to 75 minutes. You will then be given a Palm Centro and instructed on how to use it. For the next 10 days, your Palm Centro will signal at five semi-random times per day between 8:30 am and 11:30 pm. Each time you receive a random signal, you will have a series of questions to answer that will take approximately 5-6 minutes to complete. During these 10 days, you will also be asked to respond to questions on the Palm Centro after every beverage, snack, or meal that you consume. These questions will also take approximately 1-3 minutes to complete each time. At the end of the 10-day period, we will ask you to come to the research lab and return the Palm Centro.

Benefits: This research may not benefit you directly. However, your participation in this study will help us to better understand the day-to-day relationship between thoughts, mood, and body image among women. This study has the potential to add to our knowledge and help us to develop more effective strategies for treatment of disordered eating.

Risks and Discomfort: Because some of the questions asked in this study address sensitive issues and behaviors, it is possible that you may experience emotional distress during or after completing the study. If you experience more than mild discomfort, we encourage you to contact one of the facilities on the attached page. If you like, we can assist you with the referral.

Privacy and Confidentiality: The responses you provide during this study will be completely confidential. Your signed consent form will be kept separate from any date you provide. Your responses will be identified only by a unique ID number, not your name. Consent forms will be kept in a locked file cabinet in my faculty office, and data records will be kept in a locked file cabinet in my laboratory office. My laboratory office can only be accessed by me and my research assistants. Only the researchers involved in the current study will have access to these records.

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Contact Information: If you have any questions or concerns about this research, you may contact Dr. Janis H. Crowther at 330-672-2090 or jcrowthe@kent.edu. This project has been approved by the Kent State University Institutional Review Board. If you have any questions about your rights as a research participant or complaints about the research, you may call the IRB at 330.672.2704.

Consent Statement and Signature: I have read this consent form and have had the opportunity to have my questions answered to my satisfaction. I voluntarily agree to participate in this study. I understand that I must return the Palm Centro and I understand that a copy of this consent will be provided to me for future reference.

_____ _____

Participant Signature **Date**

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- Kent State University Psychological Clinic, Room 176 Kent Hall, (330) 672-2372
- Kent State Students Only: University Psychological Services, 2nd Floor DeWeese Health Center, (330) 672-2487

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- Town Hall II, 123 S. Water Street, Kent, Ohio 44240, Kent, Ohio (330) 678-4357
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- Behavioral Health Services, Summa Health System, St. Thomas Hospital, 444 N. Main Street, Akron, OH (330) 379-5906
- Nova Behavioral Health, Inc., Canton, 832 McKinley Avenue N.W., Canton, Ohio (330) 455-9407

APPENDIX B

SCREENING AND BASELINE ASSESSMENTS

Eating Disorder Diagnostic Scale (EDDS)

OVER THE PAST *THREE MONTHS*:

0	1	2	3	4	5	6
Not at all		Slightly		Moderately		Extremely

___ 1. Have you felt fat?

___ 2. Have you had a definite fear that you might gain weight or become fat?

___ 3. Has your weight influenced how you think about (judge) yourself as a person?

___ 4. Has your shape influenced how you think about (judge) yourself as a person?

5. During the past 6 months, have there been times when you felt you have eaten what other people would regard as an unusually large amount of food (i.e., a quart of ice cream) given the circumstances?
YES NO

6. During the times when you ate an unusually large amount of food, did you experience a loss of control (feel you couldn't stop eating or control what or how much you were eating)?
YES NO

7. How many DAYS per week on average over the past 6 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?

0	1	2	3	4	5	6	7

8. How many TIMES per week on average over the past 3 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

During these episodes of overeating and loss of control did you…

9. Eat much more rapidly than normal? YES NO

10. Eat until you felt uncomfortably full? YES NO

11. Eat large amounts of food when you didn't feel physically hungry? YES NO

12. Eat alone because you were embarrassed by how much you were eating? YES NO

13. Feel disgusted with yourself, depressed, or very guilty after overeating? YES NO

14. Feel very upset about your uncontrollable overeating or resulting weight gain? YES NO

15. How many times per week on average over the past 3 months have you made yourself vomit to prevent weight gain or counteract the effects of eating?

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

16. How many times per week on average over the past 3 months have you used laxatives or diuretics to prevent weight gain or counteract the effects of eating?

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

17. How many times per week on average over the past 3 months have you fasted (skipped at least 2 meals in a row) to prevent weight gain or counteract the effects of eating?

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

18. How many times per week on average over the past 3 months have you engaged in excessive exercise specifically to counteract the effects of overeating episodes?

0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

19. How much do you weigh? If uncertain, please give your best estimate. _____ lb.

20. How tall are you? _____ ft. _____ in.

21. Over the past 3 months, how many periods have you missed? 1 2 3 4 *n/a*

22. Have you been taking birth control pills during the past 3 months? YES NO

APPENDIX C

RANDOM SAMPLING EVENT ASSESSMENTS INCLUDED IN THE PRESENT STUDY

Since the last alarm, have you experienced a negative event?

1. Yes
2. No

(Please Note: If the participant indicates that they have experienced a negative event, they will be asked the following questions.)

Which category best describes the event?

1. Interpersonal/family
2. Interpersonal/friends
3. Work/School
4. Financial
5. Health
6. Other

How negative was the event?

1. Very slightly
2. A little
3. Moderately
4. Quite a bit
5. Extremely

Positive and Negative Affect Schedule-Expanded Form (PANAS-X)

Rate the following words with regard to how you are currently feeling. (Please Note: Consistent with the following example, each of the words below will appear on the screen using the same scale.)

1. Very slightly or not at all
2. A little
3. Moderately
4. Quite a bit
5. Extremely

Afraid	Upset	Attentive
Scared	Distressed	Concentrating
Nervous	Blameworthy	Determined
Jittery	Angry at self	Relaxed
Irritable	Disgusted with self	At ease
Hostile	Dissatisfied with self	Hungry
Guilty	Alert	Calm
Ashamed		

Appearance Evaluation subscale of the State Self-Esteem Scale (SSES)

This is a questionnaire designed to measure what you are thinking at this moment. There is, of course, no right answer for any statement. The best answer is what you feel is true of yourself at this moment. Be sure to answer all of the items, even if you are not certain of the best answer. Again, answer these questions as they are true for you RIGHT NOW.

1	2	3	4	5
Not at all	A little	Somewhat	Very much	Extremely

__ 1. I feel satisfied with the way my body looks right now.
__ 2. I feel that others respect and admire me.
__ 3. I am dissatisfied with my weight.
__ 4. I feel good about myself.
__ 5. I am pleased with my appearance right now.
__ 6. I feel unattractive.

Contemplation of Compensatory Behavior

Since the last alarm sounded, have you thought about trying to restrict the amount of food you eat as a way to control your weight or alter your shape?

Not at all Some Very Much

---

1 2 3 4 5

Since the last alarm sounded, have you thought about exercising as a way to control your weight, alter your shape, or burn off calories?

Not at all Some Very Much

---

1 2 3 4 5

Since the last alarm sounded, have you thought about using any other means (other than restricting your food or exercising) as a way to control your weight or alter your shape?

Not at all Some Very Much

---

1 2 3 4 5

APPENDIX D

EATING EVENT ASSESSMENTS INCLUDED IN THE PRESENT STUDY

PANAS-X (administered both at random sampling events and eating events; see Appendix C)

During this eating episode, did you feel you couldn't stop eating or controlling the type or quantity of food you were eating?

1. Yes
2. No

During this eating episode, did you eat more food than others might eat in similar circumstances or a similar period of time?

1. Yes
2. No