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The Role Of Expectancies In Binge Eating Behavior

Jessica LaRose
University of Central Florida

THE ROLE OF EXPECTANCIES IN BINGE EATING BEHAVIOR

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JESSICA GOKEE LAROSE
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Major Professor: Stacey Tantleff Dunn, Ph.D.

ABSTRACT

The central aim of the present study was to examine the role of expectancies in binge eating behavior. Two distinct statistical techniques were used to accomplish this goal. First, regression analyses were conducted using variables previously identified in the literature, as well as eating expectancies as measured by the Eating Expectancy Inventory (EEI). For both females and males, regression equations including expectancies accounted for a substantial portion of the variance in binge eating behavior. Second, memory modeling techniques were used to model the probable organization of eating expectancies. Memory modeling of hypothetical expectancy networks has lead to successful interventions in alcohol use, and preliminary work in eating revealed a fundamental difference in the way that individuals with high levels of pathology activate and store eating related messages. In the present study, Individual Differences Scaling was used to model the two-dimensional organization of an eating expectancy memory network in relation to binge eating. INDSCAL weights indicated that participants with higher levels of binge eating placed more emphasis on the positive-negative dimension, and examination of group means revealed that high binge eaters expected more change in mood in response to eating. All findings are discussed in terms of implications for enhancing assessment, treatment, and prevention strategies.

This work is dedicated to the memory of my beloved grandparents, Ernestine and Maximillion Pawlik (Mom Mom and Pop Pop). Their unconditional love, hard work, and determination have been sources of inspiration throughout my life. They are with me daily in my heart and in my memories.

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INTRODUCTION

Ironically coexisting with hunger and malnutrition, obesity has become a global epidemic that is evident even in developing countries (International Obesity Task Force, 2006). Recently the World Health Organization termed obesity "one of the greatest public health challenges of the 21st century." Its' prevalence has tripled in many European countries over the last two decades, resulting in almost 400 million overweight individuals throughout Europe (International Association for the Study of Obesity, 2006). Notably, even in China the percentage of people who are overweight has risen from less than 10% to 15% in just three years (International Association for the Study of Obesity, 2006). In the United States, obesity is now the second leading cause of preventable death (American Obesity Association, 2006). Currently, about 127 million Americans are categorized as overweight or obese, and each year this results in at least 300,000 excess deaths in the U.S. alone, as well as exorbitant healthcare costs for adults nearing $100 billion annually (Allison, Fontaine, Manson, Stevens, & VanItellie, 1999; American Obesity Association, 2006). It is a pernicious problem, and prevalence rates have continued to rise over the last several decades (Flegal, Carroll, Kuczmarski, & Johnson, (1998); World Health Organization [WHO], 1998). In 2000 the prevalence rates for obesity had more than doubled from the prevalence rates in 1980 (American Obesity Association, 2006; CDC, National Center for Health Statistics, 2002). Obesity has become the single most expensive health problem in the United States, surpassing smoking and alcohol in both its medical and financial impact (Henderson & Brownell, 2004).

The present study pertains specifically to a disorder that is highly related to obesity – binge eating disorder. Extant research indicates that there are considerable physical and mental

health consequences of binge eating disorder, and even the best established treatments still fail to result in the cessation of symptoms for many people. A variety of factors are associated with binge eating disorder, yet the question of *how* these variables are related to binge eating remains relatively unanswered. The central aim of the current research is to examine the role of expectancies in binge eating behavior. In order to fully grasp the intent and purpose of this research, a thorough review of the relevant background and ramifications of the disorder is presented. This review first will focus on the consequences of obesity, and then will move to a discussion of the relationship between obesity and binge eating disorder. The consequences of binge eating disorder will be established, and the existing research on associated factors and treatment will be reviewed. Finally, background on expectancies and a rationale for their potential role in binge eating will be provided.

Obesity and Medical Morbidity

Excessive body fat in adults substantially increases the risk of illness from almost 30 serious medical conditions, all of which contribute to poor quality of life, poor health, and premature death. According to recent data, more than 75% of hypertension cases are directly attributed to obesity, and the risk of developing hypertension for obese adults in the U.S. ages 20-45 is five to six times greater than the risk posed to non-obese individuals of the same age (American Obesity Association, 2006; Must et al., 1999; Witteman et al., 1990). In many instances successful treatment of obesity will result in a reduction in blood pressure to normal levels (VanItallie & Lew, 1992). Researchers also have demonstrated that a maintained weight loss of 10%-15% of initial body weight can result in a sustained lowering of blood pressure and an improvement in other cardiovascular disease risk factors (Wing & Jeffrey, 1995).

Overweight and obese individuals also are at substantially greater risk for developing Type 2 diabetes (Colditz et al., 1990; Must et al, 1999). It is estimated that the percentage of individuals with Type 2 diabetes who are reported to be overweight or obese is as high as 90% (American Obesity Association, 2006), and that up to 60% of overweight individuals eventually develop Type 2 diabetes (VanItallie & Lew, 1992). According to data from the National Health and Nutrition Examination Survey (NHANES III), recent prevalence rates for diabetes for overweight individuals are twice those of non-overweight individuals (National Center for Health Statistics, 2002). Furthermore, for men those rates double yet again when comparing overweight men to obese men (National Center for Health Statistics, 2002; American Obesity Association, 2006). Leading researchers in the field have stated that weight loss is the treatment of choice for obese patients with Type 2 diabetes (Wing, 1992). Therefore, efforts to address obesity and related conditions that contribute to obesity (e.g., binge eating disorder) are important steps toward fighting diabetes.

A plethora of studies have demonstrated that overweight individuals also are at increased risk for developing heart disease (Graves & Miller, 2003; Hubert, Feinleib, McNamara, & Castelli, 1983; Manson et al., 1990; Must et al., 1999). Results from the Framingham Heart Study indicate that even a modest degree of overweight in early adult life can substantially increase one's risk for developing heart disease later in life (Hubert et al., 1983). The impact of obesity on lipoprotein lipid levels is well established, placing individuals at greater risk for a variety of cardiac-related diseases, including myocardial infarction and stroke (American Obesity Association, 2006). Research also has demonstrated the perilous relationship between obesity and a host of other diseases, such as osteoarthritis, deep vein thrombosis, various forms

of cancer, end stage renal disease, musculoskeletal pain, respiratory disease, and gallbladder disease (American Obesity Association, 2006; Must et al, 1999).

In addition to these and other physical health problems, researchers have posited that obesity may be associated with higher rates of mental health problems (see Faith & Allison, 1996). However, extant findings have yielded mixed results as to the actual prevalence of psychological symptoms among obese individuals in comparison to the population as a whole (Faith & Allison, 1996; Friedman & Brownell, 1995). While some studies have found higher rates of depression and anxiety among the obese, others have not found the same differences (Faith & Allison, 1996; Friedman & Brownell, 1995). Research clearly has indicated, however, that a substantial subset of the obese population is suffering from an eating disorder – namely, binge-eating disorder (Bulik, Sullivan, & Kendler, 2002; Spitzer et al, 1992; Stunkard et al., 1996; Yanovski, 1993).

Obesity and Binge Eating Disorder

The phenomenon of binge eating in the absence of compensatory behavior was first described in the late 1950s (Stunkard, 1959). However, it was not until it was listed as a provisional diagnosis warranting further investigation in the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) that binge eating disorder (BED) began to receive considerable attention. Since that time, researchers have reached a consensus as to the critical features and characteristics associated with BED (Streigel-Moore, Wilson, Wilfley, Elder, & Brownell, 1998; Stunkard, 2002). The initial criteria set forth by Spitzer and colleagues (1992) continue to be used today to characterize this disorder. The hallmark features of BED are recurrent episodes of binge eating (i.e., eating what would be considered a much larger amount

of food than normal) in the absence of compensatory behavior (i.e., vomiting, abusing laxatives, over exercise). The individual experiences a lack of control over eating during the binge, and often feels guilty and depressed after bingeing. Despite the consistency of these diagnostic criteria over the last decade, we still do not fully understand BED.

Researchers have suggested that a characteristic of BED that is particularly relevant to obesity is that individuals with this disorder show a general tendency to overeat in addition to their binges. That is, their binges occur within a context of constant overeating, whereas for individuals with bulimia nervosa, binges occur within a background of dietary restraint (Stunkard, 2002). A wealth of studies indicate that BED is more common in overweight individuals than in non-overweight individuals (Spitzer et al, 1992; 1993; Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998), and that a substantial proportion of overweight individuals engage in binge eating (Bulik, Sullivan, & Kendler, 2002). One recent study reported that 37% of obese participants endorsed a lifetime history of binge eating (Bulik et al., 2002), suggesting that a substantial subset of the obese population suffers from characteristics associated with BED.

Epidemiological studies have yielded somewhat inconsistent results in terms of actual prevalence rates of BED, depending upon the population sampled, as well as how BED was determined. One of the seminal field studies in the area examined prevalence rates in several different samples using self-report measures, and found that in a college student sample, 2.8% of women and 1.9% of men met the criteria; in a community sample, 5.3% of women and 3.1% of men met the criteria. No significant gender differences in prevalence were noted for either population (Spitzer et al, 1993). Results of that same study indicated that in a sample collected through weight control programs, 29.7% of female and 21.8% of male patients met criteria for

BED (Spitzer et al, 1993). Another large population-based study conducted using self-report measures found an overall prevalence rate of 1.5%, and noted that unlike other traditional eating disorders, BED appeared to be present in both males and females (Smith et al., 1998). Smith and colleagues (1998) also noted that rates of BED among the overweight were almost double the rates observed in the overall sample. Prevalence studies using interview methods as opposed to self-report have found that rates may be as high as 3.3%, and that rates were highest in obese participants (8.5%), when compared to overweight (4.1%) and normal weight (2.9%) participants (Kinzl, Trawegar, Trefalt, Mangweith, & Biebl, 1999). It is important to note, however, that these differences between weight groups were not statistically significant, and that this study was conducted with females only (Kinzl et al., 1999).

A recent review of the epidemiology of BED noted that research in this area has not progressed much over the last decade, but the authors reviewed several points to be taken into account given the current state of knowledge (Streigel-Moore & Franko, 2003). First, existing studies suggest that BED is more common than bulimia nervosa (BN). To date, prevalence rates have indicated that BN was the most common of the eating disorders, and has received a great deal of research attention. Noting that BED has now surpassed BN in terms of prevalence reiterates the importance of continued research in this area. Second, the gender difference in prevalence seems to be much less pronounced in BED than in other eating disorders. This suggests that BED is not a disorder only affecting females, but males as well. Therefore, unlike traditional eating disorders research, that typically samples females, studies should seek to include both males and females in order to further our understanding of the disorder. Finally, more minority women meet the criteria for BED than for other eating disorders (Streigel-Moore

& Franko, 2003). Therefore, continued efforts should be made to access diverse samples when conducting research in this area.

Clinical Significance of BED

There has been considerable debate in the field regarding the merit and clinical significance of including BED as a disorder. That is, does the scientific evidence support the inclusion of BED as a separate diagnostic category, and is the diagnosis clinically relevant and significant above and beyond the issue of weight? While clinic, community, and population-based studies have demonstrated the association of BED with being overweight, it is imperative to highlight that obese individuals with BED differ substantially from obese persons without BED (Wilfley, Wilson, & Agras, 2003).

Studies repeatedly have found greater levels of psychiatric morbidity, particularly depression, among obese individuals with BED than obese persons who do not binge eat (Kuehnel & Wadden, 1994; Mitchell & Mussell, 1995; Vendetti, Wing, Jakicic, Butler, & Marcus, 1996; Yanovski, Nelson, Dubbert, & Spitzer, 1993). One recent population-based study found that binge eating was associated with a higher lifetime prevalence of major depression, panic disorder, phobias, and alcohol dependence (Bulik et al., 2002). Still other studies have shown that relative to an overweight, non-eating disordered sample, individuals with BED are three times more likely to suffer from a current major depressive disorder (Telch & Stice, 1998). Individuals with BED also possess the extreme concern and preoccupation with weight and shape that is characteristic of eating disorders, and these concerns typically have a negative impact on overall self-esteem and self-worth (Crow, Agras, Halmi, Mitchell, & Kramer, 2002; Masheb & Grilo, 2000; Streigel-Moore et al., 2001; Wilfley et al., 2000). Other critical features

present in individuals with BED include low self-esteem, impairment in work and social functioning, and poorer quality of life than in non-BED obese individuals (Hsu et al., 2002; Spitzer et al., 1993; Wilfley et al., 2003). Research also has demonstrated that this population evidences more chaotic eating habits and higher levels of eating disinhibition (i.e., eating in response to emotional states) than obese individuals who do not binge eat (Eldredge & Agras, 1996; Hsu et al., 2002; Wilfley, Schwartz, Spurrell, & Fairburn, 2000; Yanovski, et al., 1992).

In addition to high rates of psychiatric morbidity, there is a considerable amount of data to suggest that BED may be associated with a host of medical and physical consequences. As noted previously, BED is often linked with being overweight, the detrimental consequences of which are well established and have been discussed previously. Research findings also have suggested that BED is linked with poor physical health (Bulik et al., 2002; Dohm et al., 2001; Johnson, Spitzer, & Williams, 2001). In a sample of women in obstetric gynecology clinics and primary care, patients with BED reported higher levels of disability and more health problems than women without eating disorders, even after statistically controlling for comorbid psychiatric disorders (Johnson et al., 2001). A recent population-based study indicated that obese women who binge eat reported greater health dissatisfaction and higher rates of major medical disorders than obese women who do not binge eat (Bulik et al., 2002). Recent findings also indicated that women who were suffering from BED were significantly more likely to have accessed healthcare services for the treatment of emotional and physical problems than healthy controls, and did so at a rate that was comparable to psychiatric controls (Dohm et al., 2001). Researchers have maintained that these increased rates of treatment utilization among individuals with BED underscore the clinical significance and potential economic impact of BED (Wilfley et al, 2003).

Moving Toward a Better Understanding of BED

Considering the background, prevalence rates, and incidence of both psychiatric and medical morbidity among this population, it is evident that BED poses a significant public health threat, both as it relates to obesity, and as an eating disorder independent of obesity. While our current treatments offer moderate success in symptom reduction, the growing prevalence rates and incidence of relapse indicate that we still lack the knowledge necessary to effectively treat and prevent this disorder. Given the substantial risks associated with BED, we have an obligation to do more. In order to move toward a better understanding of BED, and to more effectively prevent and treat this disorder, it is essential that we systematically investigate the factors that are believed to contribute to the development and maintenance of the disorder. Numerous factors are related to BED, and this study sought to extend the existing knowledge base by examining the role of expectancies in binge eating behavior. Specifically, the current research was concerned with whether eating expectancies account for a significant portion of variance in binge eating, and whether expectancies mediate the relationship between binge eating and other associated variables.

Most researchers support a multifaceted model of the development of disordered eating behaviors. To date, numerous factors have been identified as playing a role in the development of disordered eating, among them genetic predisposition, body image disturbance, social comparison, mass media and other sociocultural influences (Stice, 2001; Thompson, 1996; Thompson et al., 1999). Three distinct theoretical models of binge eating have been proposed in the literature: 1) an addiction model in which the eating disorder is conceptualized as a disease; 2) an affective disorders model which suggests that the eating disorder is secondary to an underlying affective illness; and 3) a biopsychosocial model, in which a variety of factors are

believed to intersect to produce binge eating (Wilson, 1993). Leading researchers in the area have argued that not only does the addictions model fail to account for the core characteristics of eating disorders, but conceptualizing binge eating as a disease could actually serve to delay the process of returning to normal eating (Wardle, 1987; Wilson, 1991; 1993). While the positive association between depression and binge eating is well established, the predictive nature of this relationship has not been demonstrated. Although it is plausible that there are other variables that may mediate this relationship, no such mediating variables have been identified. Several researchers have provided arguments for the application of a biopsychosocial model to binge eating, but they have acknowledged that additional studies are necessary to elucidate the relationship between the variables at play in binge eating (Waller, 2000; Wilson, 1993). The central aim of the present study was to examine key biopsychosocial variables in combination with expectancies to determine if expectancies play a role in binge eating behavior. Before discussing expectancies in more detail, it is important to review the extant research regarding biological and psychosocial variables that are related to binge eating.

Biological Factors

To a certain degree, the literature on risk factors for eating disorders points toward a biological predisposition for disturbance. Genetically influenced risk factors such as higher rates of eating disorders among first-degree relatives, as well as family history of psychiatric disorders have been found among individuals with eating disorders (Keel, Leon, & Fulkerson, 2001). Twin studies have revealed a moderate genetic correlation between BN and major depression, suggesting the presence of some shared genetic effect (Walters et al., 1992). In fact, leading researchers stated that additive genetic factors consistently have emerged as contributors to

liability to BN (Bulik, 2000 p.17), and have suggested that the degree to which the environment and sociocultural influences matter is related to one's genetic predisposition (Bulik, 2000).

Research also suggests that obesity is in large part genetically determined (Brownell & Wadden, 1992; Miller, Gold, & Silverstein, 2003; Stunkard et al., 1990). Twin and adoption studies have consistently indicated that human obesity has a substantial genetic component (Price, Cadoret, Stunkard, & Troughton, 1987; Stunkard, Harris, Pederson & McClearn, 1990). In one study genetic factors accounted for 66 – 70% of the variance in the BMI of identical twins that were reared apart (Stunkard et al., 1990), and several others have found that 60 – 80% of relative weight in twins was accounted for by genetic factors (Allison, Heshka, Neale, Lykken, & Heymsfield, 1994; Meyer & Stunkard, 1993).

There also is considerable data specific to BED indicating that biological factors play a role in the development of the disorder. Researchers have demonstrated that when compared to healthy controls, individuals with BED showed higher rates of parental depression as well as greater vulnerability to obesity (Fairburn et al, 1998). When compared to individuals with psychiatric disorders, those participants with BED more often reported a history of childhood obesity (Fairburn et al., 1998). Additional data have indicated a higher prevalence of eating, mood, and anxiety disorders in families of BED women versus non-BED controls (Fairburn et al., 1998; Fowler & Bulik, 1997; Streigel-Moore, 1999). Given these findings, researchers have posited that BED might share a common genetic diathesis with these other disorders (Wilfley et al., 2003).

Psychosocial Factors

In addition to biological factors, there is considerable research to support the notion that psychological and social factors are significantly associated with the development of eating disturbance in general, and with binge eating in particular (e.g., Devlin et al., 2003; Streigel-Moore, Wilson, Wilfley, Elder, & Brownell, 1998; Womble et al., 2001; Yanovski et al., 1993). Given the shared characteristics of BN and BED, it makes sense to examine factors that are related to BN. Early research in this area demonstrated that depression was a precursor of bulimic behavior (Shatford, 1986). The Stice "dual pathway" model of bulimia proposed and found that negative affect and restrained eating predicted future bulimic symptomatology, and those two factors mediated the effects of perceived pressure to be thin, body dissatisfaction, body mass, and internalization on future bulimic symptomatology (Stice, Nemeroff, & Shaw, 1996; Stice, Shaw & Nemeroff, 1998). In a follow-up longitudinal study, dieting and negative affect predicted bulimic symptoms (Stice, 2001). More recently, researchers tested an extended version of the Stice model that hypothesized that negative affect and overeating were related through emotional eating (VanStrien, Engels, VanLeewe, & Snoek, 2005). They found support for the affective pathway in this model, but not for the restraint pathway. In addition, emotional eating appeared to explain some of the association between negative affect and overeating (VanStrien et al., 2005). Research findings to date point to the central role of negative affect in bulimia, thus it is an important variable to examine in order to enhance our understanding of BED.

In terms of research findings specific to BED, one recent study found that in a sample of obese men and women, participants with BED differed significantly from non-BED individuals on several measures. Participants with BED had greater negative affect and weight

dissatisfaction, and lower self-esteem (Streigel-Moore, et al., 1998). As noted previously, a wealth of research also has revealed substantial rates of depression in this population (Bulik et al., 2002; Kuehnel & Wadden, 1994; Mitchell & Mussell, 1995; Telch & Stice, 1998; Yanovski, Nelson, Dubbert, & Spitzer, 1993). Findings also have indicated significant differences for history of weight fluctuation and amount of time spent dieting (Brody, Walsh, & Devlin, 1995; Yanovski, 1993). Given these findings in conjunction with those for BN, it is clear that negative affect and weight cycling/dieting are key factors to consider in a comprehensive model of binge eating.

In addition to depression and weight cycling, research has demonstrated differences on other variables as well. There are significant differences between BED versus non-BED individuals in terms of weight and appearance-related teasing (Grilo, et al., 1994), as well as history of negative weight, shape and eating-related feedback from family members (Fairburn et al., 1998). In one study in particular, when compared to healthy control subjects, individuals with BED noted repeated exposure to negative comments about shape, weight, and eating, and negative self-evaluation, and compared to subjects with other psychiatric disorders, they more often reported negative comments about shape, weight, and eating (Fairburn et al., 1998).

Recognizing the important role of psychosocial factors in the development and maintenance of binge eating, Womble and colleagues (2001) used path analysis to evaluate a preliminary psychosocial model of binge eating in a sample of overweight men and women. They included many of the predictors described in the literature, including weight cycling, body dissatisfaction, teasing, negative affect, and dietary restraint. Their proposed model accounted for a considerable portion of the variance for binge eating, and all paths were shown to be significant for both males and females in the original samples. In the cross-validation samples,

only two paths were non-significant: teasing to binge eating in females, and weight cycling to binge eating in males.

Based on the current state of research on BED and the findings presented thus far, there are a number of important conclusions: 1) individuals with BED typically are either overweight or obese, or likely will become overweight or obese with continued binge eating; 2) they are susceptible to the physical and health risks and consequences of obesity; 3) they may be more dissatisfied and at risk of poor physical health that goes beyond that which is created by obesity; 4) they have high rates of psychiatric morbidity and often suffer from depression, history of weight cycling, low self-esteem, and a preoccupation with their weight and shape; and 5) they report poor social functioning and poorer quality of life. Recognizing the severity of the disorder and the ramifications should it go untreated, treatment efforts have increased throughout the last decade.

Treatment of Binge Eating Disorder

In terms of treatment efforts, Behavioral Weight Loss (BWL) seems to work in the short-term, but Cognitive-Behavioral Therapy (CBT) and Interpersonal Therapy (IPT) target the core beliefs related to shape and weight and may have a longer term impact in terms of symptom reduction (Wilfley et al., 2003). Although early studies noted no significant differences in terms of weight loss at post-treatment for obese binge eaters versus non-obese binge eaters, there were important differences between the two groups that were not mitigated by behavioral treatment of obesity (Marcus, Wing, & Hopkins, 1988). Specifically, pre-treatment differences in negative affect and cognitions were not ameliorated by the behavioral treatment, and although both groups reported improvements in mood throughout the course of treatment, improvements in mood for

binge eaters did not occur as a function of weight loss (Marcus et al., 1988). Further, binge eaters were more likely to drop out of treatment (Marcus et al., 1988). More recently, researchers have suggested that because individuals with BED may be more likely to drop out of BWL treatments and regain weight, more studies are required to determine whether obese individuals with BED would be better served by having distinct treatments from obese non-BED individuals (Wilfley et al., 2003).

To date, CBT is considered the best established treatment for BED (National Institute for Clinical Excellence [NICE], 2004), although recent studies have shown that in group format, IPT is producing recovery rates comparable to those of group CBT (Wilfley et al., 2002). However, even among CBT studies with the most impressive outcomes (e.g., Grilo, Masheb, & Wilson, 2005, Wilfley et al., 2002), a substantial portion of the patients did not achieve abstinence from binge eating. Such findings hint at the resilience of eating pathology once developed and suggest that there still is not a comprehensive understanding of all contributing factors.

Summary: The Current State of Knowledge

Clearly, there is evidence to suggest that there are grave consequences of BED. Research also indicates that biological, psychological, and social factors all play a substantial role in the development and maintenance of binge eating. However, what also is evident is that no single factor is acting to produce BED, and that we are still lacking a comprehensive understanding of how the identified factors interrelate to develop and maintain BED. An individual only inherits the *tendency* toward obesity (Brownell & Wadden, 1992). Similarly, not all individuals with a family history of an eating disorder develop eating disorders themselves. Moreover, most individuals are exposed to the sociocultural messages in our society related to shape and weight,

but not all develop eating disorders. While it is apparent that all of these factors play a role in binge eating, it remains unclear *how* they are related to binge eating. Such gaps in the existing knowledge base are noticeable considering even the best established treatments are still lacking.

To expand our knowledge of BED, it may be beneficial to look to other psychological literature to inform theory development and research. Many researchers have likened eating disorders to substance use, and binge eating clearly shares many of the same characteristics of binge or heavy drinking (Cooper, 1989; Gold, Frost-Pineda, & Jacobs, 2003). Specifically, substance use and eating disorders are often comorbid and have been associated with impulsive behaviors and mood disorders. Therefore, it may prove valuable to examine recent findings and the methodology used in alcohol research to aid in our understanding of the variables that influence binge eating. One area that holds particular promise is memory modeling. Identifying memory processes may help to identify the common pathway by which these variables serve to influence behavior. The application of expectancy theory has provided useful information about the relationship between antecedents and behaviors in a variety of areas, particularly in alcohol research. Examining expectancies in relation to eating may be useful in terms of identifying memory content to facilitate understanding of a possible pathway for decisions and urges that influence binge eating behavior.

Expectancy Theory and Binge Eating

Tolman (1932) proposed that our past experiences are stored in our memory as mental representations that impact our behavior. He described "expectancies" as learned relations between behaviors and their consequences that are stored in memory and then influence future behavior – an "if, then" relationship. Researchers have argued that expectancy is not a narrow

theoretical process, but "is best viewed as an umbrella term for processes that influence *all* behavior" (Goldman, delBoca, & Darkes, 1999, p.206). Given this interpretation, researchers more recently have applied expectancies to various forms of behavior. One of the areas in which this application is most evident is alcohol research. There is a wealth of data on the role of expectancies in drinking behavior, and they are beginning to emerge as a potential causal factor in the explanation of alcohol use.

Expectancies have been found to develop before the onset of drinking behavior (e.g., Dunn & Goldman, 1996, 1998, 2000; Miller, Smith, & Goldman, 1990), predict future drinking behavior (e.g., Smith, Goldman, Greenbaum, & Christiansen, 1995), change in parallel with drinking habits (Darkes & Goldman, 1993, 1998; Dunn, Lau, & Cruz, 2000), and mediate the effects of a wide range of antecedent variables (Sher, Walitzer, Wood, & Brent, 1991; Stacy, Newcomb, & Bentler, 1991). In addition, challenging certain key expectancies has led to significant decreases in alcohol consumption among high-risk college students (Darkes & Goldman, 1993, 1998; Dunn et al., 2000). Despite the remarkable implications for treatment and prevention evidenced within alcohol research, surprisingly little work has investigated the role of expectancies in eating behavior.

In a seminal effort to understand the potential relationship between expectancies and decisions related to eating, Hohlstein, Smith, and Atlas (1998) used factor analytic strategies to develop and validate an eating expectancy questionnaire that measures learned expectancies for positive and negative reinforcement from eating. Their work yielded five factors, or subscales (1. eating helps manage negative affect, 2. eating is pleasurable and useful, 3. eating leads to feeling out of control, 4. eating enhances cognitive competence, and 5. eating alleviates boredom). The researchers also administered the measure to a clinical sample and found that

expectancy profiles differentiated between individuals with anorexia and those with bulimia, as well as between eating disorder patients and controls. In addition, they found that expectancies did not differentiate psychiatric controls from normal controls. Further, they tested the ability of the expectancy scales to correctly classify participants into groups, and the expectancy profiles were able to do so for 94% of the cases (58 of 63 controls, 8 of 9 patients with anorexia, and all 22 patients with bulimia). Factor analysis distinguished between expectancies for positive and negative reinforcement from eating, and negative reinforcement expectancies (i.e., eating helps manage negative affect, eating alleviates boredom) was associated with binge eating, and with some dieting behavior, but not with successful dieting (Hohlstein et al., 1998).

In the only other eating expectancy study to date, Gokee-LaRose, Tantleff-Dunn, and Dunn (2003) applied alcohol expectancy research to eating, and examined eating expectancies from a semantic network perspective. Using Individual Differences Scaling (INDSCAL), they modeled the two-dimensional organization of an eating expectancy memory network, and using Property Fitting (PROFIT), they empirically labeled the two resulting dimensions as positive-negative and satisfied-unsatisfied. Findings suggested that there is a fundamental difference in the way that individuals with higher scores on measures of restriction and bulimia activate and store eating-related messages compared to individuals with low levels of disturbance. Specifically, analyses revealed that females with higher restriction scores and higher bulimia scores placed more emphasis on a satisfied-unsatisfied dimension, and were more likely than males and lower scoring females to activate negative expectancies about eating earlier on in their path of activation (Gokee-LaRose et al., 2003). As evidenced by the promising findings of these two initial studies, continued research on the role of expectancies in eating behavior holds the potential to substantially enhance our understanding of eating disorders, and may serve to inform

valuable interventions similar to those demonstrated with alcohol. The possibility of ultimately producing changes in disordered eating behavior by targeting expectancies warrants additional research in this area.

The Current Study

The present study sought to extend previous work in this are in several ways. First, the fundamental research question was whether expectancies play a role in binge eating behavior. Both of the previous studies examining eating expectancies have done so specifically in relation to restriction and bulimic behavior, but not for binge eating in the absence of compensatory behavior. Second, although Womble and colleagues (2001) provided confirmation regarding the association between various psychological and social variables and binge eating, they did not incorporate any biological or family history variables into their model. As noted previously, leading researchers have stated that additive genetic factors consistently have emerged as contributors to liability to BN (Bulik, 2000), and BN shares the core characteristic of binge eating with BED. Family history of obesity and family history of psychiatric disorders are associated with BED (Fairburn et al, 1998), and individuals with eating disorders have higher rates of eating disorders among first-degree relatives (Keel, Leon, & Fulkerson, 2001). Further, twin and adoption studies consistently have indicated that human obesity has a substantial genetic component (Price, Cadoret, Stunkard, & Troughton, 1987; Stunkard, Harris, Pederson & McClearn, 1990). Therefore, the current study included family history variables in the prediction model. Third, there may be other variables (e.g., emotional eating) that mediate the relationship between negative affect and binge eating (VanStrien et al., 2005), yet the Womble study did not test for any potential moderator or mediator effects on binge eating. Doing so could provide

valuable information about how the identified risk factors serve to produce binge and substantially enhance our understanding of the disorder. Thus, it is important not only to investigate the role of expectancies in binge eating behavior, but to assess the potential mediational effects of expectancies. BED and obesity are more common in men than other eating disorders. Therefore, it is important to include both male and female participants and to assess potential gender effects.

Based on the associated variables described in the literature and presented previously, eight predictor variables were identified that together were hypothesized to significantly predict binge eating behavior and account for a substantial portion of the overall variance. Following the progression of research for BN, a regression model was selected to examine the data in the current study. Regression allows for the combination of all hypothesized predictor variables in one model, while maintaining a certain level of parsimony. It does not overstep the current state of research and theory development for BED by creating a complex model that is not theoretically justified. Regression analysis identifies which predictor variables account for significant portions of unique variance in the model, and it allows for tests of the mediating effects of expectancies.

Hypotheses

1. Consistent with previous research demonstrating the positive relationship between binge eating and depression, weight cycling, and teasing, it was expected that each of these variables would be significant predictor variables for binge eating in overweight males and females.

2. A regression equation that included expectancies with the biopsychosocial variables previously outlined would be statistically significant and would account for a substantial portion of the variance in binge eating for overweight females and males.
3. The first subscale of the EEI (i.e., eating helps manage negative affect) would be a significant predictor of binge eating in the regression model for overweight females and males.
4. A mediational model (see *Figure 1*) was proposed in which the relationship between depression and binge eating would be mediated by negative reinforcement expectancies regarding eating, as measured by the first subscale of the EEI.

METHOD

Participants

Much of the research done with BED has been done with clinical samples, and such samples may be influenced by sampling bias, and therefore not representative of individuals with BED in the general population (Reichborn-Kjennerud, Bulik, Sullivan, Tambs, & Harris, 2004). This study recruited a sample that was a mixture of undergraduates as well as individuals participating in a self-help group for overeating, but persons presenting for treatment for BED were deliberately not recruited in order to have a sample that was representative of binge eating behavior in the general population. Participants of all weights were recruited and BMI was used as a grouping variable in the analyses such that overweight and non-overweight participants were analyzed separately.

The sample consisted of 726 adults, but a portion of the original sample was not included for data analysis. Because diagnostic criteria dictate that BED cannot be better accounted for by another eating disorder, and in order to adequately measure the construct of interest, participants who reported engaging in behaviors consistent with Anorexia Nervosa (AN) or Bulimia Nervosa (BN) were not desirable for the purposes of the study. Therefore, a screening measure (i.e., Eating Disorder Diagnostic Screening) was included in all participants' packets, and any participant who endorsed symptoms associated with anorexia nervosa (e.g., severe restriction) or bulimia nervosa (e.g., regular compensatory behavior) was not used for the analyses (n = 32). The resulting sample consisted of 694 participants (193 male and 501 female), recruited from a large open-enrollment university in the southeast (n = 618), as well as through local meetings of

Overeaters Anonymous (n = 76) in a metropolitan city in the southeast. Sample characteristics are displayed in Table 1.

Measures

Eating Expectancy Inventory (EEI)

Expectancies were assessed using the factor-based Eating Expectancy Inventory (EEI), which is a 34-item, Likert format measure designed to assess cognitive expectations for eating. The five subscales of the EEI include: 1) eating helps manage negative affect; 2) eating is pleasurable and useful; 3) eating leads to feeling out of control; 4) eating enhances cognitive competence; and 5) eating alleviates boredom. Internal consistency for the subscales ranged from .78 to .94; and construct validity through the ability to differentiate between eating disordered individuals and controls has been well established (Hohlstein et al., 1998). A copy of this measure is provided in Appendix A.

Memory Model Based Eating Expectancy Questionnaire (MMBEEQ)

The rationale and specific use of this instrument is included in the exploratory analyses section of the results. Eating expectancies were assessed with a memory model-based inventory that consists of 23 expectancy words and that can be readily mapped into a network format with MDS procedures (Gokee-LaRose et al., 2003). It differentiated between individuals based on BMI, as well as levels of restriction and bulimic symptomatology (Gokee-LaRose et al., 2003). Participants were asked to respond on a 4-point Likert scale regarding how often they experienced each expectancy word (effect) when they ate food. A copy of this measure is provided in Appendix B.

Binge Eating Scale (BES)

The BES is used consistently for the purposes of classifying individuals according to binge eating severity (e.g., LaPorte, 1992; Marcus, Wing, & Hopkins, 1988). The scale consists of 16 items describing both behavioral (e.g., eating large amounts of food) and affective and cognitive (e.g., guilt) symptoms associated with binge eating. Participants respond by marking the statement that is most reflective of them. It has acceptable internal consistency, and is able to successfully discriminate between individuals with no, moderate, or severe binge eating symptoms (Gormally, Black, Daston, & Rardin, 1982). A copy of this measure is provided in Appendix C.

Eating Disorder Diagnostic Screening (EDDS)

This measure was designed to diagnose anorexia nervosa, bulimia nervosa, and binge eating disorder based on DSM-IV criteria. The 22-item measure had an internal consistency reliability of .89, test-retest reliability of .87, and criterion validity with interview diagnoses, as well as convergent validity with other eating pathology scales (Stice, Telch, & Rizvi, 2000). A copy of this measure is provided in Appendix D.

Beck Depression Inventory (BDI-II)

Depressive symptomatology was assessed with the BDI, a questionnaire designed to assess the cognitive, behavioral, affective, and somatic symptoms associated with depression. Each of the 21 items asks participants to respond on a 4-point scale from 0 to 3, with total scores ranging from 0 (minimal) to 63 (severe). Previous studies have reported internal consistency coefficients ranging from .92 to .93 and a test-retest reliability of .93. The BDI-II has correlated positively with other measures that are used widely to assess depression, hopelessness, and

suicidal ideation. It has been validated as a measure of depression (Beck, Steer, & Garbin, 1988), and is commonly used for clinical and research purposes (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). A copy of this measure is provided in Appendix E.

Beck Anxiety Inventory (BAI)

Anxiety-related symptomatology was assessed with the BAI, a 21-item self-report questionnaire. The BAI has good internal consistency (alpha = .92) and test-retest reliability over one week was .75 (Beck, Epstein, Brown, & Steer, 1988). The BAI also has discriminant validity (Beck et al., 1988). A copy of this measure is provided in Appendix F.

Rosenberg Self-Esteem Inventory

Self-esteem was assessed with a 10-item scale designed to measure general self-esteem. Validity was evidenced by significant correlations with peer reports of self-esteem; test-retest reliability was .85 (Demos, 1985). The RSEI is commonly used as a measure of self-esteem for research purposes (Rosenberg, 1979). A copy of this measure is provided in Appendix G.

Eating Disorders Inventory – Body Dissatisfaction Subscale

This measure contains assesses the degree to which one is dissatisfied with one's body. Individuals respond to the 9 items using a 6-point Likert rating scale (never – always) according to how often the stated attitudes, feelings, and behaviors are true of them. The measure was standardized on both eating disorder patients as well as nonpatient female controls. Chronbach's Alpha for the scale is .92 and .90 respectively (Garner & Olmstead, 1984). A copy of this measure is provided in Appendix H.

Perception of Teasing Scale – Weight Related Subscale (POTS-WR)

This subscale contains six appearance-related items about teasing due to body weight. Individuals respond on a 5-point Likert rating scale (never – always) based on how often they have encountered a variety of weight-related teasing experiences, and how upset they were by the experiences. Internal consistency and test-retest reliability have been well established (Thompson, Cattarin, Fowler & Fisher, 1995). A copy of the measure is provided in Appendix I.

Sociocultural Attitudes Towards Appearance Questionnaire (SATAQ)

The SATAQ assesses the extent to which women are aware of and internalize societal standards of beauty. Participants respond to 14 items on a 5-point Likert scale (completely disagree – completely agree). The SATAQ includes two subscales, Internalization (alpha = .88) and Awareness (alpha = .71), and has convergent validity (Heinberg, Thompson, & Stormer, 1995). A copy of this measure is provided in Appendix J.

Weight Cycling Questionnaire

This measure contains items taken from previous work designed to assess weight cycling (Foreyt et al., 1995; Friedman, Schwartz, & Brownell, 1998), and is routinely used to assess weight cycling for research purposes (Womble et al., 2001). All questions are included on the Weight Cycling Questionnaire provided in Appendix K.

Family History Questionnaire

A self-report measure was created for this study to assess family history of obesity, eating disorder, and other psychological diagnoses. A copy of the questionnaire is provided in Appendix L.

Demographics Sheet

Participants were asked to for self-report information including height and weight (to calculate BMI), race, gender, and age. A copy of the questionnaire is provided in Appendix M.

Procedure

This study was approved by the university's Institutional Review Board (IRB). All participants were tested in settings similar to small classrooms. Each participant was asked to read and sign an informed consent form detailing the content of the questionnaires and purpose of the study, and was informed of his/her right to decline or withdraw from the study at any time. Additionally, written instructions were provided for each questionnaire. Participants' names did not appear on any other part of the survey, and the informed consent forms were immediately separated from the survey in order to ensure anonymity. For the purposes of data entry, sequential ID numbers were used on the surveys and in the data base, but these numbers were not placed on the informed consent forms and could not be linked to the participants names in any way. Following their participation, participants were completely debriefed as to the nature of the study.

RESULTS

Assignment to Groups

Participants were grouped at several points during the analyses in order to more effectively examine the data. Although the prevalence rates for BED are much higher for males than traditional eating disorders, the specific variables that are related and act together to produce binge eating may be distinct for males and females. Therefore, as in previous studies, males and females were examined separately by using sex as a grouping variable.

Although BMI can be viewed as a continuous variable because its calculation is based on height and weight, the degree of overweight is clinically relevant in terms of associated risk factors (e.g., hypertension, diabetes, heart disease). Further, as both malnutrition and obesity became increasing problems throughout the world, the need for a standard way of categorizing individuals based on degree of risk became apparent. In 1998, the World Health Organization (WHO) established BMI cut points that were the first such cut points at the International level. Currently, the established cut points are: <18.5 = Underweight, 18.5 – 24.9 = Normal weight, 25.0 – 29.9 = Pre-obese or overweight, 30.0 – 34.9 = Obese I, 35.0 – 39.9 = Obese II, and $\geq$ 40 = Obese III (WHO, 1998). In order to effectively conduct research that will have meaningful clinical applications for this population, leading researchers and organizations have adopted these uniform standards by which an individual in considered overweight. Given that the majority of the previous research on BED has focused on overweight participants and the high rates of association between BED and overweight, BMI also was used to group participants for analysis. In order to identify differences between overweight and non-overweight participants, the majority of analyses were conducted separately for the overweight and non-overweight

groups. Classification as non-overweight was a BMI < 25, and classification as overweight was a BMI ≥ 25.

Despite the established cut points for binge eating severity based on the BES [i.e., no binge eating ≤ 17, moderate binge eating > 17, and severe binge eating ≥ 27 (Gormally et al., 1982; LaPorte, 1992; Marcus et al., 1988)], there is considerable evidence to suggest that applying these cut points to continuous variables can lead to inaccurate and misleading results (see MacCallum, Zhang, Preacher, & Rucker, 2002). Therefore, these cut points were used only to establish binge eating and no binge eating groups for the purposes of reporting characteristics of the sample in percentages and means (see Tables 1 and 2). All subsequent statistical analyses treated binge eating as a continuous variable.

Descriptive Analyses

Descriptive statistics were calculated for the sample broken down by groups (i.e., entire sample, overweight group, non-overweight group, overweight females, non-overweight females, overweight males, non-overweight males, binge eating group, no binge eating group, college group, and OA group). Because the entire sample was broken down in multiple ways to examine the data, it is important to note that there is some overlap between the groups displayed. For example, overweight and non-overweight groups sum to the N for the entire sample, but the overweight group includes both males and females, who also are represented separately. Means and standard deviations for all of the dependent variables of interest are displayed by group in Table 2.

Females: To establish the relationship between BMI and binge eating in the current sample, a simple correlation was computed between the two variables. For females, there was a

significant positive relationship between BMI and binge eating scores (r = .351, p < .001). Correlational analyses were used to examine the relationship between binge eating and the other variables of interest. Because multiple correlations were computed, a Bonferroni correction was applied to protect against the threat of Type 1 error. The resulting p value required for these correlations to be considered statistically significant was p <.005. All results are displayed in Table 3.

For overweight females, there were significant positive relationships between binge eating and depression, body dissatisfaction, teasing history, weight cycling, anxiety, internalization of societal standards, and family history of obesity, and a significant negative relationship between binge eating and self-esteem. Similarly, for non-overweight females there was a significant positive association between binge eating and depression, body dissatisfaction, teasing history, weight cycling, anxiety, internalization of societal standards, and family history of an eating disorder. However, there was a significant negative relationship between binge eating and self-esteem (see Table 3 for graphic representation of findings and specific r values).

Males: Correlational analyses were conducted on data collected from males in the same manner as described for females. To establish the relationship between binge eating and BMI for males, a correlation was computed that yielded a significant positive association between the two variables (r = .325, p < .001). Correlational analyses were used to assess the relationship between binge eating and the other variables of interest for males. Since multiple correlations were computed, a Bonferroni correction was applied to protect against the threat of Type 1 error. The resulting p value required for these correlations to be considered significant was p <.005. All results are displayed in Table 3.

For overweight males, there was a significant positive relationship between binge eating and depression, body dissatisfaction, teasing history, weight cycling, anxiety, and internalization of societal standards, and a significant negative relationship was found between binge eating and self-esteem. In contrast, for non-overweight males, there were only significant positive associations between binge eating and anxiety, teasing history, and weight cycling. There was a significant negative relationship between binge eating and self-esteem.

Regression Analyses

The proposed regression model was evaluated using a series of simultaneous (i.e., enter) regression analyses. Simultaneous regression was chosen because it involves all predictor variables being entered into the equation at the same time, rather than at different steps, thereby removing any potential for the researcher's biases to influence the data. The regression coefficient for each predictor is estimated while holding the other predictor variables constant. It is still crucial that there be a solid reason for including each of the variables because if an irrelevant predictor variable is included power may be reduced depending on the extent to which the irrelevant variable is correlated with the other predictors.

Independent regression analyses predicting binge eating behavior were conducted for 1) overweight females, 2) non-overweight females, 3 overweight males, and 4) non-overweight males. In each regression analysis, binge eating behavior (as measured by the BES) served as the criterion variable. Based on the research findings to date and the hypotheses outlined for this study, the predictor variables were: family history of obesity, family history of an eating disorder, family history of a psychiatric disorder, depression, body dissatisfaction, teasing, SATAQ, weight cycling, and eating expectancies (as measured by each of the individual five

subscales of the EEI). Please see Tables 4 and 5 for a graphic representation of the regression results.

Females: For overweight females, a regression including all of the previously detailed variables significantly predicted binge eating behavior (F (13, 88) = 12.41, p <.001), and accounted for 65% of the total variance. Specifically, depression (p < .01) and weight cycling (p < .01) were significant predictors within the model. The subscales of the EEI reflecting eating helps manage negative affect and eating leads to feeling out of control approached significance (p = .058, p = .053, respectively).

For non-overweight females, a regression including all of the variables previously outlines significantly predicted binge eating behavior (F (13, 255) = 18.99, p <.001), and accounted for 49% of the variance. As with the overweight females, depression (p < .01) and weight cycling (p < .01) were significant predictors in this model. For non-overweight females body dissatisfaction (p < .05) and internalization of societal ideals (p < .01) also were significant predictor variables in the model. The first and third subscales of the EEI were not approaching significance with the non-overweight females as they were with the overweight females.

Males: For overweight males, a regression including all of the noted predictor variables did significantly predict binge eating behavior (F (13, 44) = 9.54, p <.001) and accounted for 74% of the variance. As with overweight females, depression (p < .01) and weight cycling (p < .01) were significant predictors within the model. The subscale of the EEI reflecting eating is pleasurable and useful approached significance (p = .057). In contrast, for non-overweight males the model did not significantly predict binge eating (F (13, 57) = .774, p =.683).

Post-Hoc Moderator Analyses: Based on the regression models presented above, depression and weight cycling both were significant predictors of binge eating for overweight

males and females. Therefore, follow-up analyses were conducted to examine whether sex serves as a moderator in the relationship between each of these variables and binge eating. In the first of the two regressions, depression and sex significantly predicted binge eating (F (2, 180) = 47.94, p <.001), but adding the interaction of these two variables did not contribute to the prediction model (F (3, 179) = 1.47, p <.227). In the second of the regressions, weight cycling and sex significantly predicted binge eating (F (2, 195) = 56.44, p <.001), but adding the interaction of these two variables did not contribute to the prediction model (F (3, 194) = 2.53, p <.115).

Regression Analyses for the Mediational Hypothesis

Based on the hypothesized mediational model, it was expected that depression would predict binge eating by way of eating expectancies (as measured by the first subscale of the EEI, eating helps to manage negative affect). This model was tested for females and males using a series of regression analyses. Based on Baron and Kenny (1986), evidence of a mediational model required several findings (see *Figure 1*). The first step was to estimate and test path c in order to establish that there was an effect that may be mediated. In the current study, the relationship between depression and binge eating would have to be significant. The second step was to show that the initial variable was correlated with the mediator, or estimate and test path a. In the current study, this meant that the relationship between depression and eating expectancies would have to be significant. The third step was to demonstrate that the mediator affects the outcome variable by estimating and testing path b. In the current study, this refers to the relationship between eating expectancies and binge eating. If all of these predictions held true in the hypothesized direction, then the final step would be to conduct a regression with X and Z

(i.e., depression and expectancies) predicting Y (i.e. binge eating). In this equation, mediation would be supported if the partial direct effect for path c was nonsignificantly different from zero and path b was significantly greater than zero. If c were nonsignificantly different from zero, results would be consistent with a *full* mediational model. If path b was significant after controlling for the direct effect of X (path c), but path c was still significant, the model would be consistent with *partial* mediation.

Females: For overweight females, depression was a significant predictor of binge eating behavior (F (1, 106) = 46.97, $p < .01$). In the second regression equation of the mediational model, depression was not a significant predictor of eating expectancies (F (1, 111) = .023, $p < .89$). In the third set of equations for the mediational model, expectancies did not significantly predict binge eating (F (1, 115) = 2.49, $p < .12$). Because two of the three paths estimated were not significant, a mediational model did not explain the relationship between depression and binge eating behavior problems when the EEI was used as a mediator. See Table 6 for a graphic presentation of these regression analyses.

For non-overweight females, depression predicted binge eating behavior (F (1, 293) = 101.25, $p < .01$). In the second regression equation of the mediational model, depression did not significantly predict eating expectancies (F (1, 310) = .268, $p < .61$). In the third equation, expectancies did not significantly predict binge eating (F (1, 306) = 2.64, $p < .12$). As with the overweight female group, two of the three paths were not significant for the non-overweight female group. Therefore, a mediational model did not explain the relationship between depression and binge eating when the EEI was used as a mediator. See Table 6 for all results.

Males: For overweight males, depression was a significant predictor of binge eating behavior (F (1, 64) = 43.30, $p < .01$). In the second regression equation of the mediational

model, depression was not a significant predictor of eating expectancies (F (1, 69) = .120, p < .731). In the third set of equations for the mediational model, expectancies did not significantly predict binge eating (F (1, 68) = .254, p < .62). Because two of the three paths estimated were not significant, a mediational model did not explain the relationship between depression and binge eating behavior problems when the EEI was used as a mediator. See Table 6 for a graphic presentation of these regression analyses.

For non-overweight males, depression was a significant predictor of binge eating behavior (F (1, 83) = 4.64, p < .05). In the second regression equation of the mediational model, depression was not a significant predictor of eating expectancies (F (1, 90) = .029, p < .865). In the third equation, expectancies did not significantly predict binge eating (F (1, 93) = 4.33, p < .08). As with the previous three groups, two of the three paths were not significant for the non-overweight male group. Therefore, a mediational model did not explain the relationship between depression and binge eating when the EEI was used as a mediator. See Table 6 for all results.

Exploratory Analyses

In all previous analyses, expectancies were measured by the EEI, a factor based measure that produces a score on individual subscales and a total score. This type of measure is very useful for regression analyses such as those conducted in this study, and has been shown to discriminate between individuals based on levels of eating pathology. However, examining expectancies in this way is only one of the possible ways to examine their potential role in binge eating behavior. To further examine the role of expectancies in binge eating behavior, exploratory analyses were conducted in which expectancies were examined from a memory modeling perspective. A brief background and rationale for this approach is provided below.

Memory modeling is an area that holds potential for explaining the processes by which variables impact behavior. Multidimensional scaling (MDS) techniques have been used to model the organization and activation of alcohol expectancies in memory (Cruz & Dunn, 2003; Dunn & Earleywine, 2001; Dunn & Goldman, 1996, 1998, 2000; Rather, Goldman, Roerich, & Brannick, 1992; Rather & Goldman, 1994). MDS solutions have been well validated as a proxy for network structure (e.g., Cooke, Durso, & Schvaneveldt, 1986; Dunn & Goldman, 2000). Such alcohol expectancy networks have been modeled for adults of varying drinking habits (Dunn & Earleywine, 2001; Rather & Goldman, 1994; Rather et al., 1992), as well as for children (Cruz & Dunn, 2003; Dunn & Goldman, 1996; 1998, 2000; Dunn & Yniguez, 1999). Further, researchers have found that expectancy challenge programs have altered paths of activation in children (Cruz & Dunn, 2003), and in college students, with subsequent changes in alcohol consumption (Darkes & Goldman, 1993, 1998; Dunn, et al., 2000).

In order to further our understanding of the storage and activation of eating related expectancies in memory, it may be beneficial to use these techniques in conjunction with a general theory of memory processes. Numerous conceptualizations for understanding memory processes have been proposed, one of which was offered by Estes (1991). He suggested that memory traces could be viewed as "vectors or lists, as nodes in a network, or as points in a multidimensional space" (p.12, Estes, 1991). Using this conceptualization of memory, and consistent with alcohol expectancy research (e.g., Rather, et al., 1992), a semantic memory network model was used in the current analyses. In semantic network theory, concepts are thought to be represented by nodes that are linked together on the basis of intrinsic meaning and learning history (Rather et al., 1992). The relationships between all such nodes are retained and mapped with MDS. A MDS stimulus configuration of expectancies can be thought of as a

snapshot of a cognitive process in which expectancies are represented in an associate network model (Goldman, Del Boca, & Darkes, 1999). Such a cognitive map has previously been constructed for eating expectancies for groups based on restriction and bulimia (Gokee-LaRose, et al., 2003), but not for binge eating. The present study used the methodology previously implemented in the area of alcohol expectancies (Cruz & Dunn, 2003; Dunn & Earleywine, 2001; Dunn & Goldman, 1996, 1998, 2000; Dunn et al., 2000; Dunn & Earlywine, 2001; Dunn & Yniguez, 1999; Rather et al., 1992; Rather & Goldman, 1994) and recently adapted for eating expectancies (Gokee-LaRose et.al., 2003, 2004) to model a semantic network of eating expectancies stored in memory in relation to binge eating.

Configuration of an Eating Expectancy Network

Distinct mathematical techniques were used in order to emphasize different aspects of a hypothetical memory network. In order to discriminate between participants, individuals were stratified into thirds based on their respective levels of binge eating as determined by the previously detailed measures (e.g., high binge eating score, moderate binge eating score, and low binge eating score). Stratifying into thirds was somewhat arbitrary, but thirds were chosen to limit the number of points and vectors in figures to a number that would present clear conclusions. As in previous studies within the realm of alcohol expectancies (Cruz & Dunn, 2003; Dunn & Earlywine, 2001; Dunn et al., 2000; Dunn & Yniguez, 1999; Dunn & Goldman, 1996, 1998; Rather & Goldman, 1994), and marijuana expectancies (Linkovich-Kyle & Dunn, 2001), nonmetric INDSCAL was used to empirically model a memory network of eating expectancies. Such network models are not entirely distinct from factor-models. Despite the mathematical similarities between factors and MDS solutions, a network model provides insight

into the process by which eating related expectancies may be stored and activated in memory. A more detailed discussion of the similarities and differences between factor and network models can be found in Goldman (1994).

As with other MDS techniques, INDSCAL involves applying a mathematical algorithm to a matrix of proximities. Each item appears on one column and one row of the matrix, and each possible pair-wise combination of items comprise the elements of the matrix. Similar to a correlation matrix, the halves separated by the diagonal are identical. However, the diagonal is composed of zeros (representing no difference) because each item is identical to itself. Consequently, the algorithm actually performs computations on a similarity (or dissimilarity) half-matrix in order to locate each item on the stimulus configuration. Group "weights" on each dimension are computed, ranging from zero to one, with higher weights indicating larger distances between stimuli on that particular dimension. Greater weight on a dimension essentially means that there is a greater emphasis placed on a dimension, meaning that people recognize much more distinction between items along that dimension. A squared group weight represents the proportion of variance in the group's data that is explained by the particular dimension (Wish & Carroll, 1974). In the present study, INDSCAL was used to map eating expectancies into network format and participant weights were used to explore the distinctions between dimensions for participants divided into groups based on binge eating scores.

Previous work has revealed a substantial gender effect for many of the variables in the present study, such that even when males and females were matched on physical appearance-related anxiety and drive for thinness scores, a pronounced gender effect still obscured differences between higher and lower scoring males and females (Gokee-LaRose, Dunn, & Tantleff-Dunn, 2004). This can occur because INDSCAL constrains participant weights to a

range of 0 to 1. Thus, when participants are grouped on more than one variable (e.g., binge eating and gender), the distance required to represent the variable with a greater impact may squeeze groups based on the other variable together, making it impossible to examine group differences. Therefore, males and females were analyzed together, and then separately in order to account for possible gender effects. Analyses also were conducted using only those participants meeting criteria for overweight (i.e., BMI≥25). In sum, 7 sets of INDSCAL analyses were completed: 1) entire sample grouped on binge eating scores; 2) overweight sample grouped on binge eating scores; 3) overweight sample grouped on BMI; 4) all females grouped on binge eating scores; 5) overweight females grouped by binge eating scores; 6) all males grouped on binge eating scores; and 7) overweight males grouped on binge eating scores. In each analysis, proximity matrices for each group were used as input for the INDSCAL analysis to produce a stimulus configuration reflective of all participants included in each analysis. Because a single proximity matrix was used as input for each group in each analysis, every group had an equal amount of influence in determining the final solution, regardless of unequal sample sizes. The mathematical properties of matrices are such that they are stable if based on 25 or more participants and highly stable when based on 100 or more participants (Linkovich-Kyle & Dunn, 2001). The grouping strategy used in the present study resulted in the smallest group containing 20 participants, and only 2 of the 21 groups being composed of fewer than 25 participants. Further, for the primary analysis of concern (i.e., entire sample group by binge eating scores), the grouping strategy resulted in all groups containing well over 100 participants. Therefore, the results of these analyses should be stable and generalizable to similar populations.

Binge Eating Groups

As mentioned earlier, there is considerable evidence to suggest the applying cut points to continuous variables can lead to inaccurate and misleading results (see MacCallum, Zhang, Preacher, & Rucker, 2002). However, multidimensional scaling procedures necessitate that participants are broken into groups on the variable of interest in order for the analyses to be conducted. Unlike many other cases in which continuous variables are dichotomized, there are not comparable analyses that would allow the data to be analyzed in its continuous form and provide similar information as to the cognitive organization of eating expectancies. Therefore, it is believed that applications of MDS are one of the few exceptions in which using cut points is warranted.

Participants were stratified into thirds in order to discriminate between groups while maintaining the stability of the matrices (i.e., in order to ensure that there were an adequate number of participants in each group as defined by the previously noted criteria). Groups were created based on binge eating scores (low binge eating, moderate binge eating, and high binge eating), and a one-way ANOVA indicated significant differences between groups ($F = 1083.61$, $p < .001$).

Multidimensional Scaling Analyses: INDSCAL

For each of the INDSCAL analyses performed, a solution depicting the stimuli in two dimensions was retained because it was readily interpretable and accounted for a significant portion of the variance. For each variable, addition of a third dimension accounted for a minimal amount of variance. A good fit is typically defined as R^2 of .80 or greater, and stress of .15 or less. Excellent fit of the solution to the data is typically defined as R^2 of .90 or greater,

and stress of .10 or less. When using INDSCAL, however, stress is artificially inflated. Therefore, R^2 is the most appropriate fit index for INDSCAL (Davison, 1983).

The INDSCAL analyses including the entire sample based on the binge eating grouping variable yielded a solution with excellent fit indices (R^2 = .93, stress = .14). An INDSCAL analysis of the overweight sample (i.e., males and females meeting criteria for overweight) grouped by binge eating scores produced good fit indices as well (R^2 = .88, stress = .18), but not as strong as those including the entire sample.

Despite good fit indices, previous work in this area has indicated the presence of a pronounced gender effect for the cognitive organization of eating expectancies (Gokee, Dunn & Tantleff-Dunn, 2001; Tantleff-Dunn, Dunn, & Gokee, 2001). One potential explanation for the substantial gender effect in previous work is that there is a much lower incidence of traditional eating disorders among males than females. Given findings indicating that BED is more common among men than other forms of eating disorders, it is possible that such a gender effect may not obscure differences in the current study. However, in order to assess for this possibility, analyses also were conducted with males and females separately. An INDSCAL analysis of all females grouped by binge eating yielded a solution with good fit indices (R^2 = .90, stress = .16). A strong solution also was yielded for analyses of all males grouped by binge eating (R^2 = .80, stress = .21). Acceptable solutions were produced for overweight females grouped by binge eating (R^2 = .74, stress = .24), and overweight males grouped by binge eating (R^2 = .73, stress = .23). These findings suggest that including the entire sample (i.e., males and females, overweight and non-overweight) does not obscure potential differences, but rather, produces a solution that better represents and explains the data, as evidenced by the excellent fit indices.

Therefore, the most inclusive analysis, and that yielding the best fit indices, is what is presented in the remainder of this work.

INDSCAL solutions provide stimulus configurations that can be conceptualized as maps, in which the probability for activation of an expectancy node is a function of how close the respective node is to other expectancy nodes that have a high probability of activation. Thus, the expectancies most likely to be activated are those closest to the first expectancy nodes that are activated (Rather et al., 1992). The stimulus configuration of eating expectancies for participants grouped based on binge eating scores are displayed in Figure 2). The INDSCAL stimulus configurations are meant to serve as a reference for interpreting findings because they are an average across all groups in the analysis, and each stimulus configuration represents a participant who weights each of the dimensions equally (Linkovich-Kyle & Dunn, 2001). Group weights produced by INDSCAL represent the salience of each dimension to each group. Squared group weights indicate the amount of variance in the group data that is accounted for by the dimension (Wish & Carroll, 1974). Different weights on a dimension are indicative of disagreement between groups about the level of similarity of expectancy nodes. As weights increase, differences also increase between the expectancy nodes along that dimension (Linkovich-Kyle & Dunn, 2001).

Examination of the current dimension weights indicated that the positive-negative dimension was more salient (and accounted for the most variance) to participants with higher binge eating scores than it was to participants with low or moderate binge eating scores (see Figure 3). Thus, for participants with high levels of binge eating symptomatology, the stimulus configuration depicted in Figure 2 would be elongated along the positive-negative dimension and compressed along the satisfied-unsatisfied dimension. As a result, stimulus points along the

positive-negative dimension, such as "happy" and "fat," would be farther apart, and stimulus points along the satisfied-unsatisfied dimension, such as "comforted", and "relaxed," would be closer together. Expectancies in closer proximity to one another are more likely to activate together than expectancies that are farther apart (Rather & Goldman, 1994). Therefore, participants with severe binge eating may expect "comforted" and "good" to occur with similar frequency after eating, and would be less likely to activate "happy" and "hungry" together. Examination of the means for each of the groups lends support to the theory that expectancies are more likely to be activated in this fashion. For example, for the high binge eating group, the means for comforted (1.35) and good (1.39) are closer together, while the means for happy (1.76) and hungry (0.88) are farther apart. (See Table 7 for means for each expectancy word by group).

Participants with lower levels of binge eating placed very little emphasis on the positive-negative dimension and nearly all their emphasis on the satisfied-unsatisfied dimension (see Figure 3). This means that for these individuals, the stimulus configuration displayed in Figure 2 would be elongated along the satisfied-unsatisfied dimension and compressed along the positive-negative dimension. Thus, individuals with low levels of binge eating may expect that both positive and negative expectancies such as "strong" and "tired" occur with similar frequency after eating. Further, for these individuals, such expectancies may be likely to activate at the same time (See Table 7 for means).

DISCUSSION

Results of the present study confirmed much of the research to date and provided valuable new information about the variables that influence binge eating behavior. The initial hypothesis in the current study was supported. As in previous research, there was a significant positive relationship between depression and binge eating. Depression was a significant predictor variable for binge eating for overweight males and females, as well as non-overweight females. It was not a significant predictor for non-overweight males. However, it is important to note that the overall model was not significant for non-overweight males. These findings underscore one of the important distinctions between BED and other eating disorders. That is, in other forms of eating disorders there is a high incidence of anxiety disorders, and often the onset of the anxiety is prior to that of eating disturbance. It has been theorized that anxiety may serve as a vulnerability factor for the development of disordered eating (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). In fact, anxiety disorders predate the emergence of BN in the majority of cases (Bulik et al., 1996). For individuals with BED, it seems that negative affect, or depression, may play a much larger role than anxiety. Of participants in the severe binge eating group, 48.6% reported clinically significant levels of depression (i.e. BDI>13), whereas only 14% reported clinically significant levels of anxiety (i.e., BAI>21). Depression accounted for the largest portion of unique variance in the regression model for both overweight males and non-overweight females, and the second most for overweight females. This finding also lends some support to the affective theory of binge eating, although there are clearly other variables that contribute to the development and maintenance of BED as well. Future studies should

continue to examine the relationship between depression and binge eating, with particular emphasis on how this affective component can be most effectively addressed in treatment.

A second hypothesis for the present study was that there would be a significant positive association between weight cycling and binge eating scores, and that weight cycling would be a significant predictor variable for binge eating in overweight females and males. This hypothesis also was supported. Weight cycling was highly associated with binge eating for overweight males and females, as well as non-overweight females. It also accounted for the largest portion of unique variance in the regression model for overweight females (followed by depression). These findings reiterate those of much of the previous work, and provide additional support for the key role that history of weight fluctuation plays in binge eating behavior. Also of note, in the cross validation portion of the Womble path analysis, the path from weight cycling to binge eating was not significant for males. In the current study however, weight cycling was a significant predictor variable for overweight males. Future studies should seek to clarify the predictive nature of weight cycling for males. If in fact weight cycling is a predictor variable that is confirmed in future studies, that information could be used in prevention or primary intervention programs with children and their parents to deter children from dietary restraint and yo-yo dieting, and provide education regarding healthy eating habits and regular exercise.

The third hypothesis was that there would be a significant positive relationship between teasing history and binge eating, and that teasing would be a significant predictor variable for binge eating in overweight females and males. This prediction was partially supported. Specifically, teasing was shown to be significantly and positively associated with binge eating for all groups in the current study, even non-overweight females and males. However, it was not a statistically significant predictor variable in the regression equation for females or males,

although it did approach significance for overweight females (p = .063). It is of particular interest that this is one of only three variables that were significantly associated with binge eating for non-overweight males. Although the overall prediction model was not significant for non-overweight males, it is conceivable that teasing is a substantial vulnerability factor for males who may be perceived as underweight, and may binge eat in response to teasing. Such possibilities merit additional investigation.

Previous findings have indicated differences in teasing history between BED and non-BED individuals, but in the cross validation sample for the Womble path analysis, the path from teasing to binge eating was not significant for females. In the current study, teasing approached significance for overweight females, but not so for males. Additional research specifically targeting the role of teasing in this population is necessary to elucidate this relationship. If in fact history of teasing is a clinically significant predictor of future binge eating behavior, as theory and some research findings would indicate, then school-based intervention programs could target teasing early on as one way of preventing binge eating.

Additionally, it was hypothesized that a regression equation that included all of the biopsychosocial variables previously outlined, including expectancies, would be statistically significant and account for a substantial portion of the variance in binge eating for overweight females and males. This hypothesis was supported, but merits further discussion. The overall prediction model was statistically significant for overweight females and males, as well as non-overweight females. It accounted for an impressive 74% of the variance in binge eating for overweight males, 65% of the variance for overweight females, and 49% for non-overweight females. Two variables in particular accounted for a statistically significant portion of the variance within the model for overweight males and females – depression and weight cycling.

A central aim of the present work was to examine the role of expectancies in binge eating. It was predicted that the first subscale of the EEI would be a significant predictor of binge eating in the model described previously for overweight females and males (i.e., eating helps manage negative affect). This hypothesis was not supported. It is important to note, however, that the level approached significance for overweight females (p = .058). Considering that only two of the other variables in the equation met criteria for statistical significance within the model, it is important to consider the issue of clinical significance as opposed to statistical significance (for a detailed discussion see Kazdin, 2003). In short, given the applied nature of clinical psychology, and the nature of many of the constructs studied, behavior change cannot only be measured in terms of an arbitrary alpha level cut off. The present findings offer a prime example in support of this argument. There is evidence to suggest that eating expectancies differentiate between individuals with disordered eating and those without, and negative reinforcement expectancies were close to statistical significance in the present model. Therefore, it is plausible that expectancies are playing a clinically meaningful role in the onset and maintenance of this behavior, yet in the current study did not meet the arbitrary statistical cut off. To dismiss the clinical relevance of this finding would be negligent. Furthering this line of research may very well serve to inform treatment and prevention efforts, and is clearly warranted.

The final hypothesis proposed in the current study was related to the existence of a mediational model (see *Figure 1*) in which the relationship between depression and binge eating would be mediated by negative reinforcement expectancies regarding eating, as measured by the first subscale of the EEI. This hypothesis was not supported. Future studies should seek to test this hypothesis in a clinical sample. It also is important to note that the measure being used to

assess expectancies for this purpose (i.e., EEI) was developed and validated for restriction and bulimia. Although BED and bulimia share many of the same critical features/symptoms, there are several clear distinctions as well. For example, individuals with bulimia nervosa (BN) tend to be at or near normal weight, not overweight, and they also engage in compensatory behaviors of some sort (e.g., vomiting) after their binges. That is to say, whereas the binges in BED tend to occur within the background of constant overeating, those in BN occur within the context of dietary restraint and restriction. Therefore, it is possible that the EEI needs to be adapted and revised for use with the BED population in order to adequately assess the role of such negative reinforcement expectancies.

It is noteworthy that the same variables were positively associated with binge eating for overweight males and females, and the same regression model was significant for both groups as well. Moreover, for both groups, the same two predictor variables were found to be significant within the model – depression and weight cycling. This finding is consistent with the bulk of literature that indicates that BED is more common in men than traditional eating disorders, and would suggest that the same factors are at play for both males and females. The findings of this study support one potential explanation that individuals with BED experience significant symptoms of depression and sadness, and engage in binge eating behavior as a mechanism to manage their negative affect. This hypothesis is conceivable for both males and females. It is quite possible, however, that the reasons that depression drives binge eating could be distinct for males versus females. For example, a negative reinforcement expectancy subscale (eating helps to manage negative affect) approached significance for overweight females, whereas a positive reinforcement subscale (eating is pleasurable and useful) approached significance for males.

Future research should explore these potential differences and the role that endorsement of such expectancies may play in binge eating in the absence of compensatory behavior.

The exploratory scaling analyses in the current study offered promising results in terms of the role of expectancies in binge eating from a memory modeling perspective. Specifically, INDSCAL dimension weights differentiated between individuals based on binge eating scores (see Figure 3). These findings suggest that there is a fundamental difference in the way that individuals with higher levels of binge eating behavior activate and store eating related messages compared to individuals with low levels of binge eating behavior. Further, these distinctions are present for both males and females. Also of interest, participants in the high binge eating group seemed to expect eating to produce an affective change more so than those individuals in the low binge eating group (e.g., means for the expectancy word "less depressed" were significantly greater for high binge eaters when compared to low binge eaters). Additional studies are needed to replicate these initial findings, and to use Preference Mapping (PREFMAP) to model hypothetical paths of activation throughout the network. Such studies would clarify the order in which expectancies are activated for severe binge eaters when compared to no or low binge eaters.

Increased knowledge about expectancies may be very valuable in treatment as well as intervention programs, as we have seen with alcohol interventions (Darkes & Goldman, 1993, 1998; Dunn et al., 2000). For example, within the context of therapy, one could implement programs focused on the development of adaptive coping skills in response to depression for clients who hold expectancies that eating helps manage negative affect. Cognitive techniques such as restructuring could be used to challenge and reframe expectancies and beliefs about food. These techniques also could be used to promote negative expectancies for this population, such

as the realistic health consequences of binge eating and obesity. Similarly, expectancies could be incorporated into prevention programs for children. For example, school-based programs could promote healthy eating, provide education about how to effectively manage emotions in adaptive ways, and challenge beliefs that eating can lead to feeling out of control, or that eating can help to alleviate symptoms of sadness or anger. Increased emphasis on effectively combating the growing pediatric obesity crisis is crucial as well. Preventing youth from becoming overweight and obese at such an early age and aiding in the nutrition and exercise efforts both at school and at home would undoubtedly change the learning and experiences of children, which would impact the development of eating expectancies and subsequent eating behavior.

Although the present study added substantially to the current state of knowledge, there are several limitations that should be taken into consideration. First, binge eating was assessed using the BES rather than a structured clinical interview, which may have resulted in overestimates of binge eating behavior, and has the potential to make it more difficult to distinguish between BED and non-purging BN (which is characterized by recurrent episodes of binge eating and inappropriate compensatory behaviors such as fasting or excessive exercise). However, the BES is routinely used for this purpose, and in fact, the majority of studies use the BES to determine binge eating severity as opposed to clinical interview (Wilfley et al, 2003; Womble et al., 2001). Also, the EEDS was included as a second measure of binge eating, which allowed the identification of participants who engage in a variety of compensatory behaviors (e.g., excessive exercise, laxative use), and those participants were not used in the analyses. Second, the sample was somewhat homogeneous given that it was predominantly Caucasian and was drawn from a single metropolitan area. A more ethnically diverse sample drawn from various regions of the country may lend itself to greater generalizability, particularly given the

higher incidence of BED among minority women compared to other eating disorders. Further, given the growing incidence of obesity throughout the world, it may be beneficial to examine BED in cross-cultural populations as well. Third, the way in which family history variables were measured in the present study may have affected the degree to which they were related to binge eating. Future studies should seek to more directly measure genetic factors that may play an important role in BED. Finally, future studies in this area also should seek to investigate eating expectancies with a younger sample. Alcohol researchers have found differences in expectancies about the effects of alcohol in children as young as second grade that were related to their future alcohol use (e.g., Dunn & Goldman, 1996, 1998, 2000), and have shown that those memory processes can be changed in children to lower risk (see Cruz & Dunn, 2003). Examining expectancies in older samples also may prove valuable. It is conceivable that one's experiences and learned consequences vary with age. That is, the way that one experiences the world, particularly related to physical appearance and weight, may be quite different at 60 versus 30, and therefore expectancies may vary considerably as well.

In addition to the directions for future studies already noted, the addition of studies showing correlations between expectancies (as measured by the EEI or another scale) and binge eating behavior is important for providing a solid foundation for more advanced work in this area. Assessing expectancies in children prior to the onset of binge eating would provide valuable information about when eating expectancies develop, and how they relate to future eating behavior. Conducting longitudinal studies with a broad age range, such as following young children into adulthood, could be employed to track expectancies over time and assess what variables may influence potential changes in those beliefs. Finally, an essential step toward understanding the relationship between eating expectancies and binge eating behavior will

involve true experiments in which expectancies are primed and/or challenged and the impact on actual eating behavior is evaluated.

The present study offers new information about the way individuals process and store information related to binge eating, and provides solid preliminary support for the role of expectancies in binge eating behavior. Consistent with the biopsychosocial model, there are several overarching variables that may influence outcome expectancy formation, and potentially explain some of the differences observed in eating expectancies. Recognizing the essential role of biopsychosocial factors and the salient role that expectancies may play, the current study combined these variables and ultimately accounted for a substantial portion of the variance in binge eating for both males and females. Exploratory analyses further revealed that there is a fundamental difference in the way that high binge eaters store eating related messages when compared to low/no binge eaters. A plethora of research on alcohol expectancies has demonstrated that expectancies not only develop before use and predict future use, but also that it is possible to alter expectancies, producing notable changes in subsequent behavior patterns. In light of these accomplishments in the alcohol arena, the current findings hold promise as a springboard for future theoretical and applied studies of binge eating and obesity. Considering the substantial portion of the obese population that engage in binge eating, this line of research has the potential to substantially impact the obesity epidemic as a whole, thereby reducing one of the most significant public health threats of this generation.

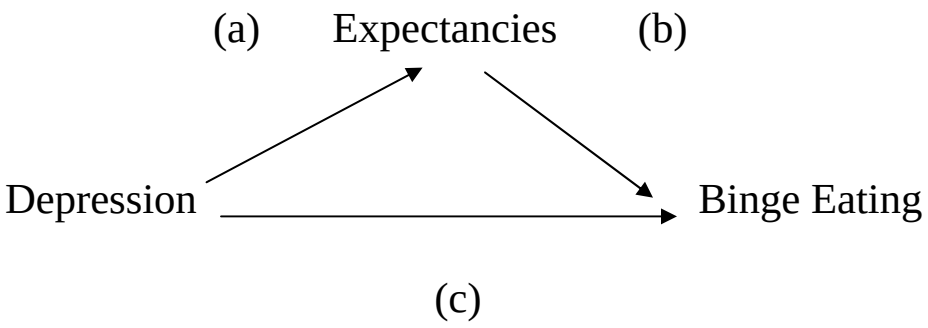

Figure 1. Hypothesized mediational model in which depression predicts binge eating by way of eating expectancies

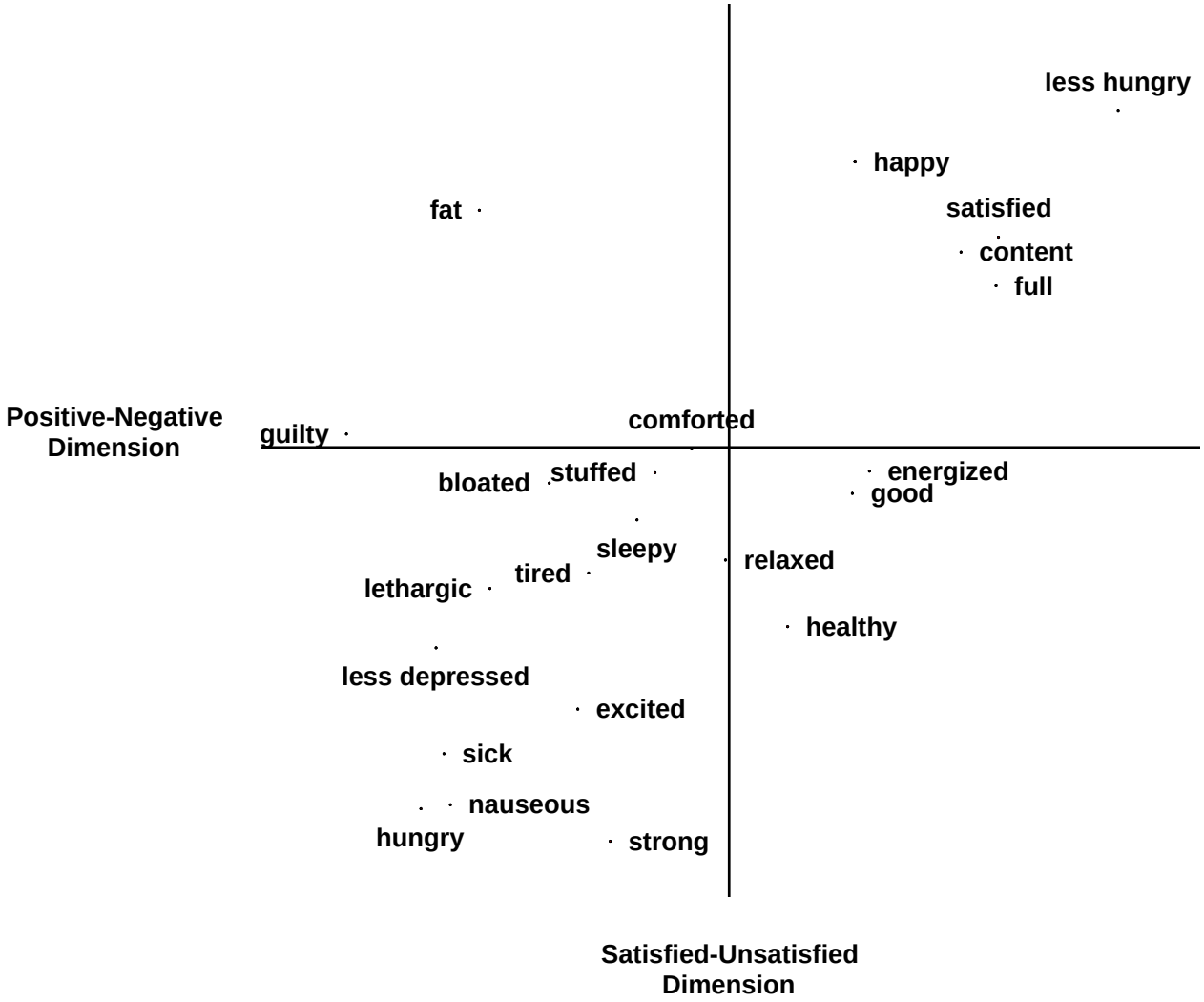

Figure 2. Stimulus configuration based on INDSCAL analyses for entire sample based on binge eating scores

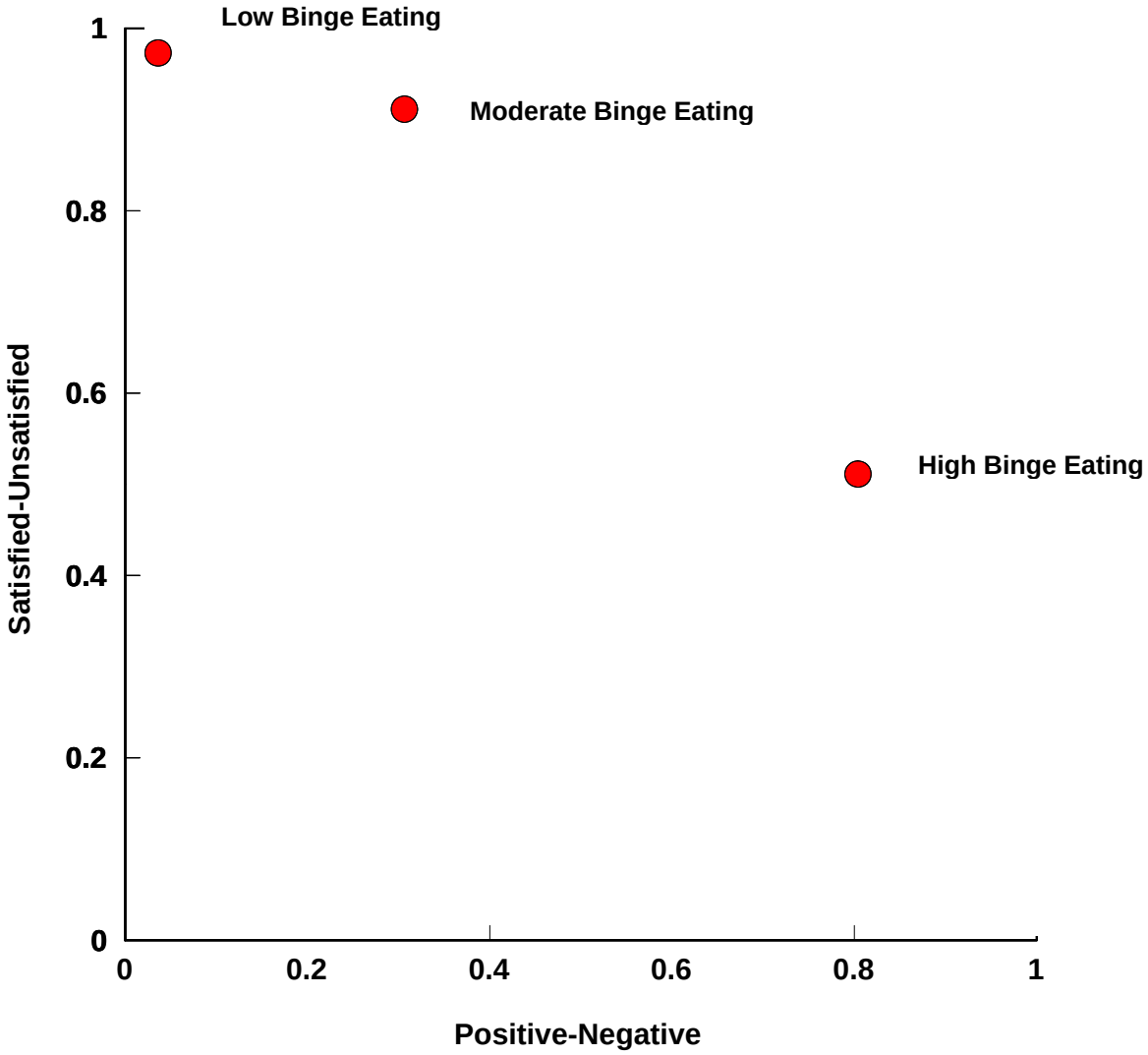

Figure 3. Individual-Differences scaling participant weights on the positive-negative dimension and the satisfied-unsatisfied dimension for participants with different levels of binge eating.

Table 1 Demographic Characteristics of the Sample by Group

Group	Mean Age (SD)	Males	Females	Mean BMI (SD)	Caucasian	Black/ African American	Latino/a	Asian American	Biracial/ Other
Entire Sample (N = 694)	24.2 (9.8)	193	501	24.03 (4.7)	71.9%	7.3%	13.5%	3.4%	3.8%
Overweight Group (n = 211)	28.3 (13.8)	80	131	29.59 (4.28)	69.6%	11.6%	13.5%	2.4%	2.9%
Non-overweight (n = 483)	22.4 (6.8)	111	372	21.5 (2.4)	73.1%	5.5%	13.1%	3.8%	4.2%
Binge Eating Group (n = 121)	25.5 (10.9)	17	104	26.8 (5.5)	72.3%	6.7%	14.3%	3.4%	3.4%
College Group (n = 618)	21.4 (4.4)	187	431	23.4 (4.4)	70.0%	7.5%	14.4%	3.7%	4.2%
OA Group (n = 76)	44.2 (15.5)	17	59	28.4 (5.5)	82.4%	6.8%	8.1%	1.4%	1.4%

Note: Overweight group defined as BMI $\geq$ 25, non-overweight as BMI <25. Binge eating group defined as BES score > 17, based on established cut points.

Table 2. Means and Standard Deviations for Dependent Variables by Group

Group	Depression	Self-Esteem	Body DS	Anxiety	Teasing	SATAQ	Binge Eating	Weight Cycling
Entire Sample (N = 694)	8.41 (8.8)	32.86 (6.2)	30.14 (11.2)	8.27 (9.1)	14.83 (9.6)	44.68 (10.5)	9.63 (7.8)	8.12 (3.9)
Overweight Group (n = 211)	9.30 (9.6)	31.93 (6.3)	35.59 (11.7)	8.41 (9.4)	19.29 (11.6)	44.90 (10.8)	12.59 (8.6)	10.11 (4.1)
Non-overweight Group (n = 483)	7.87 (8.3)	33.41 (5.9)	27.62 (10.2)	8.19 (8.9)	12.72 (7.6)	44.50 (10.5)	8.26 (6.9)	7.21 (3.4)
Overweight Females (n = 131)	10.77 (10.2)	31.40 (6.4)	40.04 (10.0)	9.45 (9.5)	20.3 (12.6)	47.65 (11.3)	14.15 (9.0)	10.91 (4.3)
Non-overweight Females (n = 369)	8.29 (8.6)	33.43 (5.9)	29.55 (10.1)	8.54 (8.6)	12.71 (7.6)	45.91 (10.5)	8.95 (7.1)	7.59 (3.5)
Overweight Males (n = 80)	6.95 (8.0)	32.76 (6.1)	28.23 (10.7)	6.74 (8.9)	17.68 (9.8)	40.17 (7.8)	9.96 (7.3)	8.8 (3.5)
Non-overweight Males (n = 114)	6.14 (6.8)	33.42 (6.1)	21.17 (7.7)	6.98 (10.1)	12.77 (7.5)	39.68 (8.8)	5.81 (5.9)	5.91 (2.7)
Binge Eating Group (n = 121)	16.82 (11.6)	28.39 (6.5)	40.41 (10.4)	13.88 (11.8)	21.69 (12.6)	52.80 (9.2)	22.69 (5.7)	12.13 (4.1)
No Binge Eating Group (n = 573)	6.80 (6.9)	33.90 (5.6)	28.04 (10.1)	7.24 (8.1)	13.21 (7.8)	42.99 (10.1)	6.67 (4.4)	7.23 (3.2)
College Group (n = 618)	8.63 (9.1)	32.86 (6.2)	29.20 (10.8)	8.43 (9.3)	14.50 (9.3)	44.59 (10.5)	9.26 (7.7)	7.74 (3.7)
OA Group (n = 76)	5.7 (5.6)	33.85 (5.7)	37.17 (12.6)	6.64 (7.2)	17.42 (11.2)	44.92 (11.0)	12.29 (8.4)	10.87 (4.6)

Note: Overweight group defined as BMI $\geq$ 25, non-overweight as BMI <25. Binge eating group defined as BES score > 17, based on established cut points.

Table 3. Pearson Correlations between Binge Eating and All Other Variables (by group)

	Depression	Self-Esteem	BDS	Anxiety	Teasing	SATAQ	Weight Cycling	FH of Obesity	FH of Eating D/O	FH of Psych
Entire Sample	.519*	-.453*	.573*	.326*	.457*	.454*	.606*	.262*	.224*	.113
Overweight Females	.554*	-.543*	.566*	.372*	.512*	.545*	.632*	.330*	.199	.168
Non-overweight Females	.507*	-.406*	.529*	..289*	.251*	.467*	.551*	.141	.308*	.061
Overweight Males	.635*	-.449*	.456*	.416*	.373*	.357*	.509*	.243	.019	.181
Non-overweight Males	.230	-.305*	.238	.341*	.384*	.251	.285	.009	-.010	-.051

*denotes significant correlation after Bonferroni correction ($p < .005$)

Table 4. Regression Analyses for Females

Variable	*ß*		*t*		*p*	
	Overweight	Non-overweight	Overweight	Non-overweight	Overweight	Non-overweight
FH of obesity	.084	-.023	1.15	-.473	.252	.637
FH of eating disorder	.069	.080	1.01	1.66	.320	.098
FH of psych disorder	-.094	-.024	-1.24	-.521	.219	.602
Depression	.209	.286	2.59	5.56	.011**	.000**
Teasing	.155	.050	1.89	1.02	.063	.31
Body Dissatisfaction	.119	.131	1.35	2.11	.180	.036*
SATAQ	.154	.200	1.74	3.52	.085	.001**
Weight Cycling	.441	.268	5.33	4.91	.000**	.000**
EEI 1	.197	-.064	1.92	-.857	.058	.392
EEI 2	-.076	.068	-.975	1.23	.332	.221
EEI 3	-.175	.074	-1.96	1.21	.053	.226
EEI 4	-.092	-.037	-1.20	-.707	.233	.480
EEI 5	.099	.077	1.07	1.35	.288	.179

Table 5. Regression Analyses for Males

Variable	*ß*		*t*		*p*	
	Overweight	Non-overweight	Overweight	Non-overweight	Overweight	Non-overweight
FH of obesity	*-.143*	-.118	-1.50	-.871	.140	.387
FH of eating disorder	-.045	-.040	-.487	-.271	.629	.787
FH of psych disorder	.067	.008	.714	.063	.479	.950
Depression	*.472*	.031	4.98	.223	.000**	.824
Teasing	.142	*.240*	1.59	1.71	.120	.093
Body Dissatisfaction	.098	.079	.869	.537	.389	.594
SATAQ	.103	*.148*	1.07	1.06	.292	.292
Weight Cycling	.411	.082	3.90	.560	.000**	.578
EEI 1	-.087	.010	-.727	.047	.471	.962
EEI 2	-.196	.021	-1.952	.131	.057	.896
EEI 3	.152	.050	1.45	.284	.154	.778
EEI 4	.090	-.059	.965	-.384	.340	.703
EEI 5	.028	.090	.300	.486	.765	.629

Table 6. Regression Analyses for Mediational Hypothesis

Path	ß	*t*	*p*
Path a: Depression predicting expectancies			
Overweight Females	.014	.150	.881
Non-overweight Females	.029	.518	.605
Overweight Males	.042	.346	.73
Non-overweight Males	-.018	-.171	.865
Path b: Expectancies predicting binge eating			
Overweight Females	.145	1.58	.118
Non-overweight Females	.092	1.62	.106
Overweight Males	.061	.504	.616
Non-overweight Males	.211	.228	.082
Path c: Depression predicting binge eating			
Overweight Females	.554	6.85	.000*
Non-overweight Females	.507	10.06	.000*
Overweight Males	.635	6.58	.000*
Non-overweight Males	.230	2.16	.034*

Table 7. Means for each expectancy word by binge eating group

Expectancy Word	*Low BE*	*Moderate BE*	*High BE*
Hungry	0.59	0.72	0.88
Full	2.00	1.91	1.73
Happy	1.62	1.76	1.76
Less Hungry	2.24	2.15	1.90
Satisfied	2.03	2.01	1.78
Content	1.91	1.87	1.72
Energized	1.64	1.52	1.39
Fat	0.71	1.02	1.58
Nauseous	0.65	0.65	0.75
Strong	1.02	0.90	0.84
Tired	1.00	1.07	1.18
Bloated	0.82	1.02	1.22
Sleepy	1.09	1.18	1.24
Healthy	1.52	1.31	1.16
Relaxed	1.37	1.30	1.27
Sick	0.64	0.64	0.83
Stuffed	1.15	1.21	1.30
Guilty	0.35	0.66	1.25
Lethargic	0.69	0.88	1.01
Less Depressed	0.58	0.82	0.92
Excited	0.92	0.91	0.96
Good	1.60	1.62	1.39
Comforted	1.20	1.31	1.35

APPENDIX A: EATING EXPECTANCY INVENTORY

EEI

Read each statement and circle the number of the response which most closely matches your level of agreement. Please respond to the items in terms of what the word "eating" means to you. There are no right or wrong answers. Choose only one response for each item. Do not leave any items blank.

1	2	3	4	5	6	7
completely disagree	mostly disagree	slightly disagree	neither agree nor disagree	slightly agree	mostly agree	completely agree

1. Eating makes me feel loved.	1 2 3 4 5 6 7
2. When I am feeling depressed or upset, eating can help me take my mind off my problems.	1 2 3 4 5 6 7
3. Eating makes me feel out of control.	1 2 3 4 5 6 7
4. Eating fills some emotional need.	1 2 3 4 5 6 7
5. When I am feeling anxious or tense, eating helps me relax.	1 2 3 4 5 6 7
6. I don't see eating as a pleasurable event.	1 2 3 4 5 6 7
7. Eating helps me deal with feelings of inadequacy about myself.	1 2 3 4 5 6 7
8. Eating doesn't help me deal with boredom.	1 2 3 4 5 6 7
9. When I have nothing to do, eating helps relieve the boredom.	1 2 3 4 5 6 7
10. When I eat, I often feel I am not in charge of my life.	1 2 3 4 5 6 7
11. When I am feeling anxious, eating does not make me feel calmer.	1 2 3 4 5 6 7
12. Eating serves as an emotional release.	1 2 3 4 5 6 7
13. Eating seems to decrease my level of anxiety if I am feeling tense or stressed.	1 2 3 4 5 6 7
14. Eating is a good way to celebrate.	1 2 3 4 5 6 7
15. When I do something good, eating is a way to reward myself.	1 2 3 4 5 6 7
16. Eating isn't useful as a reward for me.	1 2 3 4 5 6 7

17. I don't get a sense of security or safety from eating. 1 2 3 4 5 6 7

18. If I have nothing planned to do during the day, eating isn't something that would help me fill the time. 1 2 3 4 5 6 7

19. Eating helps me think and study better. 1 2 3 4 5 6 7

20. Eating is fun and enjoyable. 1 2 3 4 5 6 7

21. My eating behavior often results in a feeling that I am not in control. 1 2 3 4 5 6 7

22. When I work hard or accomplish something, eating doesn't serve as a good reward. 1 2 3 4 5 6 7

23. Eating is something to do when you feel bored. 1 2 3 4 5 6 7

24. Eating is a way to vent my anger. 1 2 3 4 5 6 7

25. Eating helps me avoid uncomfortable social situations. 1 2 3 4 5 6 7

26. When I am angry at my parents, spouse or friends, eating helps me get back at them. 1 2 3 4 5 6 7

27. When I am faced with difficult tasks, eating can help me avoid doing them. 1 2 3 4 5 6 7

28. Eating helps me forget or block out negative feelings, like depression, loneliness, or fear. 1 2 3 4 5 6 7

29. Eating calms me when I am feeling stressed, anxious, or tense. 1 2 3 4 5 6 7

30. Eating can help me bury my emotions when I don't want to feel them. 1 2 3 4 5 6 7

31. Eating helps me work better. 1 2 3 4 5 6 7

32. Eating helps me cope with negative emotions. 1 2 3 4 5 6 7

33. Eating does not make me feel out of control. 1 2 3 4 5 6 7

34. Eating helps me deal with sadness or emotional pain. 1 2 3 4 5 6 7

APPENDIX B: MEMORY MODEL BASED EATING EXPECTANCY QUESTIONNAIRE

MMBEEQ

The following pages contain words describing possible effects of eating. For each word, imagine it completing the sentence: "EATING FOOD MAKES ME _____." Then, for each word circle the word that indicates how often you think that this effect happens or could happen to you after eating.

There are no right or wrong answers. Answer each item quickly according to your first impression and according to your own personal beliefs about the effects of eating. Circle one answer for each question.

"EATING FOOD MAKES ME _____."

1.	Hungry	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
2.	Full	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
3.	Happy	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
4.	Less Hungry	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
5.	Satisfied	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
6.	Content	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
7.	Energized	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
8.	Fat	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
9.	Nauseous	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
10.	Strong	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
11.	Tired	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**

"EATING MAKES ME _____."

12.	Bloated	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
13.	Sleepy	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
14.	Healthy	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
15.	Relaxed	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
16.	Sick	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
17.	Stuffed	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
18.	Guilty	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
19.	Lethargic	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
20.	Less Depressed	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
21.	Excited	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
22.	Good	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**
23.	Comforted	**NEVER**	**SOMETIMES**	**USUALLY**	**ALWAYS**

APPENDIX C: BINGE EATING SCALE

EATING HABITS CHECKSLIST

Instructions: Below are groups of numbered statements. Read all of the statements in each group and mark on this sheet the one that best describes the way you feel about the problems you have controlling your eating behavior.

#1

1. I don't feel self-conscious about my weight or body size when I'm with others.
2. I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
3. I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
4. I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.

#2

1. I don't have any difficulty eating slowly in the proper manner.
2. Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
3. At times, I tend to eat quickly and then, I feel uncomfortably full afterwards.
4. I have the habit of bolting down my food, without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.

#3

1. I feel capable to control my eating urges when I want to.
2. I feel like I have failed to control my eating more than the average person.
3. I feel utterly helpless when it comes to feeling in control of my eating urges.
4. Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

#4

1. I don't have the habit of eating when I'm bored.
2. I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
3. I have a regular habit of eating when I'm bored, but occasionally, I can use some other activity to get my mind off eating.
4. I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

#5

1. I'm usually physically hungry when I eat something.
2. Occasionally, I eat something on impulse even though I really am not hungry.
3. I have the regular habit of eating foods, that I might not really enjoy, to satisfy a hungry feeling even though physically, I don't need the food.
4. Even though I'm not physically hungry, I get a hungry feeling in my mouth that only seems to be satisfied when I eat a food, like a sandwich, that fills my mouth. Sometimes, when I eat the food to satisfy my mouth hunger, I then spit the food out so I won't gain weight.

#6

1. I don't feel guilt or self-hate after I overeat.
2. After I overeat, occasionally I feel guilt or self-hate.
3. Almost all the time I experience strong guilt or self-hate after I overeat.

#7

1. I don't lose total control of my eating when dieting even after periods when I overeat.
2. Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
3. Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I overeat on a diet. When that happens I eat even more.
4. I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either a "feast" or "famine".

#8

1. I rarely eat so much food that I feel uncomfortably stuffed afterwards.
2. Usually about once a month, I eat such a quantity of food, I end up feeling very stuffed.
3. I have regular periods during the month when I eat large amounts of food, either at mealtime or at snacks.
4. I eat so much food that I regularly feel quite uncomfortable after eating and sometimes a bit nauseous.

#9

1. My level of calorie intake does not go up very high or go down very low on a regular basis.
2. Sometime after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten.
3. I have a regular habit of overeating during the night. It seems that my routine is not to be hungry in the morning but overeat in the evening.
4. In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I overeat. It seems I live a life of either "feast or famine."

#10

1. I usually am able to stop eating when I want to. I know when "enough is enough."
2. Every so often, I experience a compulsion to eat, which I can't seem to control.
3. Frequently, I experience strong urges to eat which I seem unable to control, but at other times I can control my eating urges.
4. I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.

#11

1. I don't have any problem stopping eating when I feel full.
2. I usually can stop eating when I feel full but occasionally overeat leaving me feeling uncomfortably stuffed.
3. I have a problem stopping eating once I start and usually I feel uncomfortably stuffed after I eat a meal.
4. Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling.

#12

1. I seem to eat just as much when I'm with others (family, social gatherings) as when I am by myself.
2. Sometimes, when I'm with other persons, I don't eat as much as I want to eat because I'm self-conscious about my eating.
3. Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.
4. I feel so ashamed about overeating that I pick times to overeat when I know no one will see me. I feel like a "closet eater."

#13

1. I eat three meals a day with only an occasional between meal snack.
2. I eat 3 meals a day, but I also normally snack between meals.
3. When I am snacking heavily, I get in the habit of skipping regular meals.
4. There are regular periods when I seem to be continually eating, with no planned meals.

#14

1. I don't think much about trying to control unwanted eating urges.
2. At least some of the time, I feel my thoughts are pre-occupied with trying to control my eating urges.
3. I feel that frequently I spend much time thinking about how much I ate or about trying not to eat anymore.
4. It seems to me that most of my waking hours are pre-occupied by thoughts about eating *or* not eating. I feel like I'm constantly struggling not to eat.

#15

1. I don't think about food a great deal.
2. I have strong cravings for food but they last only for brief periods of time.
3. I have days when I can't seem to think about anything else but food.
4. Most of my days seem to be pre-occupied with thoughts about food. I feel like I live to eat.

#16

1. I usually know whether or not I'm physically hungry. I take the right portion of food to satisfy me.
2. Occasionally, I feel uncertain about knowing whether or not I'm physically hungry. At these times it's hard to know how much food I should take to satisfy me.
3. Even though I might know how many calories I should eat, I don't have any idea what is a "normal" amount of food for me.

APPENDIX D: EATING DISORDER DIAGNOSTIC SCREENING (EDDS)

EDDS

Please carefully complete all questions.

Over the past 3 months…

	Not at all		Slightly		Moderately		Extremely
1. Have you felt fat?	0	1	2	3	4	5	6
2. Have you had a definite fear that you might gain weight or become fat?	0	1	2	3	4	5	6
3. Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
4. Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6

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 (e.g., 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

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. _____lbs

20. How tall are you? _____feet _____inches

21. Over the past 3 months, how many menstrual 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 E: BECK DEPRESSION INVENTORY, SECOND EDITION (BDI-II)

BDI – II

Date:_____

Name:_____ Marital Status:_____ Age:_____ Sex:_____

Occupation:_____ Education:_____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you are feeling in the past two weeks, including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number from that group. Be sure that you so not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
   - 0 I do not feel sad.
   - 1 I feel sad much of the time.
   - 2 I am sad all the time.
   - 3 I am so sad or unhappy that I can't stand it.

2. Pessimism
   - 0 I am not discouraged about my future.
   - 1 I feel more discouraged about my future
   - 2 I do not expect things to work out for me.
   - 3 I feel my future is hopeless and will only get worse.

3. Past Failure
   - 0 I do not feel like a failure.
   - 1 I have failed more than I should have.
   - 2 As I look back, I see a lot of failures.
   - 3 I feel I am a total failure as a person.

4. Loss of Pleasure
   - 0 I get as much pleasure as I ever did from the things I enjoy.
   - 1 I don't enjoy things as much as I used to.
   - 2 I get very little pleasure from the things I used to enjoy.
   - 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
   - 0 I don't feel particularly guilty.
   - 1 I feel guilty over many things I have done or should have done.
   - 2 I feel quite guilty most of the time.
   - 3 I feel guilty all of the time.

6. Punishment Feelings
   - 0 I don't feel I am being punished.
   - 1 I feel I may be punished.
   - 2 I expect to be punished.
   - 3 I feel I am being punished.

7. Self-Dislike
   - 0 I feel the same about myself as ever.
   - 1 I have lost confidence in myself.
   - 2 I am disappointed in myself.
   - 3 I dislike myself.

8. Self-Criticalness
   - 0 I don't criticize or blame myself more than usual.
   - 1 I am more critical of myself than I used to be.
   - 2 I criticize myself for all of my faults.
   - 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
   - 0 I don't have any thoughts of killing myself.
   - 1 I have thoughts of killing myself, but I would not carry them out.
   - 2 I would like to kill myself.
   - 3 I would kill myself if I had the chance.

10. Crying
    - 0 I don't cry any more than I used to.
    - 1 I cry more than I used to.
    - 2 I cry over every little thing.
    - 3 I feel like crying, but I can't.

_____Subtotal Page 1

Continued on Back ⇒

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated than it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to.
2 I don't have enough energy to do very much.
3 I can't get any pleasure from the things I used to enjoy.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite somewhat more than usual.
2a My appetite is much less than usual.
2b My appetite is much more than usual.
3a I have no appetite at all.
3b I crave food all the time.

.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

APPENDIX F: BECK ANXIETY INVENTORY (BAI)

BAI

Instructions: Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing and X in the corresponding space in the column next to each symptom.

	NOT AT ALL	MILDLY It didn't bother me much.	MODERATELY It was very uncomfortable but I could stand it.	SEVERELY I could barely stand it.
1. Numbness or tingling				
2. Feeling hot.				
3. Wobbliness in legs.				
4. Unable to relax.				
5. Fear of the worst happening.				
6. Dizzy or lightheaded.				
7. Heart pounding or racing.				
8. Unsteady.				
9. Terrified				
10. Nervous.				
11. Feelings of choking				
12. Hands trembling				
13. Shaky				
14. Fear of losing control.				
15. Difficulty breathing				
16. Fear of dying				
17. Scared				
18. Indigestion or discomfort in abdomen				
19. Faint				
20. Face flushed				
21. Sweating (not due to heat)				

APPENDIX G: ROSENBERG SELF-ESTEEM INVENTORY

ROSENBERG SEI

DIRECTIONS: Please circle the number of the response that you feel most represents how much or little you agree with the following statements. Do not skip any questions. Use the following scale:

1----------2----------3----------4
Strongly Disagree Disagree Agree Strongly Agree

		Strongly Disagree	Disagree	Agree	Strongly Agree
1.	I feel that I am a person of worth, or at least on an equal basis with others.	**1**	**2**	**3**	**4**
2.	I feel that I have a number of good qualities.	**1**	**2**	**3**	**4**
3.	All in all, I am inclined to feel that I am a failure.	**1**	**2**	**3**	**4**
4.	I am able to do things as well as most other people.	**1**	**2**	**3**	**4**
5.	I feel that I do not have much to be proud of.	**1**	**2**	**3**	**4**
6.	I take a positive attitude towards myself.	**1**	**2**	**3**	**4**
7.	On the whole, I am satisfied with myself.	**1**	**2**	**3**	**4**
8.	I wish I could have more respect for myself.	**1**	**2**	**3**	**4**
9.	I certainly feel useless at times.	**1**	**2**	**3**	**4**
10.	At times I think I'm no good at all.	**1**	**2**	**3**	**4**

APPENDIX H: EATING DISORDERS INVENTORY - BODY DISSATISFACTION SUBSCALE (EDI-BD)

EDI-BDS

Please read each statement and **CIRCLE** the number that best indicates how often you agree with the following statements:

1 = NEVER 2 = RARELY 3 = SOMETIMES 4 = OFTEN 5 = USUALLY 6 = ALWAYS

1.	I think my stomach is too big.	**1**	**2**	**3**	**4**	**5**	**6**
2.	I think that my thighs are too large.	**1**	**2**	**3**	**4**	**5**	**6**
3.	I think my stomach is just the right size.	**1**	**2**	**3**	**4**	**5**	**6**
4.	I feel satisfied with the shape of my body.	**1**	**2**	**3**	**4**	**5**	**6**
5.	I like the shape of my buttocks.	**1**	**2**	**3**	**4**	**5**	**6**
6.	I think my hips are too big.	**1**	**2**	**3**	**4**	**5**	**6**
7.	I think that my thighs are just the right size.	**1**	**2**	**3**	**4**	**5**	**6**
8.	I think my buttocks are too large.	**1**	**2**	**3**	**4**	**5**	**6**
9.	I think my hips are just the right size.	**1**	**2**	**3**	**4**	**5**	**6**

APPENDIX I: PERCEPTION OF TEASING SCALE – WEIGHT-RELATED SUBSCALE (POTS-WR)

POTS

We are interested in whether you have been teased and how this affected you.

First, for each question rate how often you think you were teased using the scale provided, "never" (1) to "always" (5).

1 2 3 4 5
Never----------Sometimes----------Very Often

Second, unless you responded "never" to the question, rate how upset you were by the teasing, "not upset" (1) to "very upset"(5).

1 2 3 4 5
Not Upset----------Somewhat Upset----------Very Upset

1.	People make fun of you because you were heavy.	1	2	3	4	5
	How upset were you?	1	2	3	4	5
2.	People made jokes about you being heavy.	1	2	3	4	5
	How upset were you?	1	2	3	4	5
3.	People laughed at you for trying out for sports because you were heavy.	1	2	3	4	5
	How upset were you?	1	2	3	4	5
4.	People called you names like "fatso."	1	2	3	4	5
	How upset were you?	1	2	3	4	5
5.	People pointed at you because you were overweight.	1	2	3	4	5
	How upset were you?	1	2	3	4	5

APPENDIX J: SOCIOCULTURAL ATTITUDES TOWARD APPEARANCE QUESTIONNAIRE (SATAQ)

Sociocultural Attitudes Toward Appearance Questionnaire (SATAQ)

Please read each of the following items and circle the number that best reflects your agreement with the statement.

1----------2----------3----------4----------5

Completely Disagree		Neither agree nor disagree		Completely agree

1. Women who appear in TV shows and movies project the type of appearance that I see as my goal.
   1 2 3 4 5

2. I believe clothes look better on thin models.
   1 2 3 4 5

3. Music videos that show thin women make me wish that I were thin.
   1 2 3 4 5

4. I do not wish to look like the models in the magazines.
   1 2 3 4 5

5. I tend to compare my body to people in magazines and on TV.
   1 2 3 4 5

6. In our society, fat people are not regarded as unattractive.
   1 2 3 4 5

7. Photographs of thin women make me wish that I were thin.
   1 2 3 4 5

8. Attractiveness is very important if you want to get ahead in our culture.
   1 2 3 4 5

9. It is important for people to work hard on their figures/physiques if they want to succeed in today's culture.
   1 2 3 4 5

10. Most people do not believe that the thinner you are, the better you look in clothes.
    1 2 3 4 5

11. People think the thinner you are, the better you look in clothes.
1 2 3 4 5

12. In today's society, it's not important to always look attractive.
1 2 3 4 5

13. I wish I looked like a swimsuit model.
1 2 3 4 5

14. I often read magazines like Cosmopolitan, Vogue, and Glamour and compare my appearance to the models.
1 2 3 4 5

APPENDIX K: WEIGHT CYCLING QUESTIONNAIRE

WCQ

Please respond to the following questions as honestly as possible:

1 = NEVER 2 = RARELY 3 = SOMETIMES 4 = OFTEN 5 = ALWAYS

1. How often do you lose and regain weight?	**1**	**2**	**3**	**4**	**5**
2. How often are you a yo-yo dieter?	**1**	**2**	**3**	**4**	**5**
3. How often do you start and diet and quit?	**1**	**2**	**3**	**4**	**5**
4. How often do you regain more weight than you lost on a diet?	**1**	**2**	**3**	**4**	**5**

APPENDIX L: FAMILY HISTORY QUESTIONNAIRE

FHQ

Please respond to the following items as honestly as possible:

1) Do you have a family history of obesity? YES NO

2) If so, who in your family is/was obese? (Please circle all that apply)

MOTHER FATHER BROTHER SISTER

GRANDMOTHER GRANDFATHER AUNT UNCLE

3) Has anyone in your family ever suffered from an eating disorder? YES NO

4) If so, which eating disorder?

ANOREXIA BULIMIA BINGE EATING DISORDER NOT SURE

5) If so, who in your family suffered from an eating disorder? (Please circle all that apply)

MOTHER FATHER BROTHER SISTER

GRANDMOTHER GRANDFATHER AUNT UNCLE

6) Has anyone in your family ever suffered from a psychological disorder other than an eating disorder? (For example, depression, anxiety, substance use)

YES NO

7) If so, what? _____

8) If so, who? (Please circle all that apply)

MOTHER FATHER BROTHER SISTER

GRANDMOTHER GRANDFATHER AUNT UNCLE

APPENDIX M: DEMOGRAPHICS SHEET

DEMOGRAPHIC INFORMATION

Please provide the researchers with the following information about yourself. All information will remain confidential and anonymous.

Age: _____

Year in school: _____

Race (Circle one):

a. African American
b. Asian or Pacific Islander
c. Caucasian
d. Hispanic
e. Other _____

Height: _____ feet _____ inches

Weight: _____ pounds

Are you currently on a diet? YES NO

Have you gained or lost any weight in the last month(s)? YES NO

If so, how much? _____ pounds in _____ weeks / months (please circle one)

Are you a vegetarian? YES NO

APPENDIX N: IRB APPROVAL DOCUMENT

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