Summer 8-17-2018

The Role of Perceived Control and Cardiac Function among Individuals with Binge Eating Symptomatology

Rachel E. Goetze
University of Maine, regoetze@gmail.com

THE ROLE OF PERCEIVED CONTROL AND CARDIAC FUNCTION AMONG INDIVIDUALS WITH BINGE EATING SYMPTOMATOLOGY

By

Rachel E. Goetze

B.A. University of Maine, 2005

M.A. University of Maine, 2015

A DISSERTATION

Submitted in Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

(in Psychology)

The Graduate School

University of Maine

August 2018

Advisory Committee:

Emily A. P. Haigh, Ph.D., Assistant Professor of Psychology, Advisor

Shannon K. McCoy, Ph.D., Associate Professor of Psychology

Michael A. Robbins, Ph.D., Research Professor and Chair of Psychology Department

Sandra T. Sigmon, Ph.D., Emerita Professor of Psychology

Stacy Whitcomb-Smith, Ph.D., Adjunct Faculty of Psychology; Clinical Psychologist at Health Psych Maine

THE ROLE OF PERCEIVED CONTROL AND CARDIAC FUNCTION AMONG INDIVIDUALS WITH BINGE EATING SYMPTOMATOLOGY

By Rachel E. Goetze

Dissertation Advisor: Dr. Emily A. P. Haigh

An Abstract of the Dissertation Presented
in Partial Fulfillment of the Requirements for the
Degree of Doctor of Philosophy
(in Psychology)
August 2018

The central aim of this study was to investigate the predictive role of perceived control in binge eating severity, mood reactivity, and possible concomitants with reduced cardiovascular function as measured by high frequency heart rate variability (HF-HRV/RSA). Participants (N = 75) included normal to overweight men and women who completed self-report measures assessing perceived control, binge eating severity, perceived stress, negative affect, and depressive symptom severity prior to a structured clinical interview and second experimental laboratory session. During this second experimental lab session, noninvasive electrical sensors were placed for physiological recordings to measure fluctuations in HF-HRV/RSA in participants randomized to a negative or neutral mood induction task. In addition to physiological data, participants completed self-report measures of mood and stress during baseline assessment, post-mood induction, and following a recovery period.

Results indicated that perceived control was predictive of binge eating severity such that higher self-reported perceived control was associated with less severe binge eating symptoms. This association was significantly mediated by perceived stress and depressive symptoms, such that those with greater perceived control also experienced less perceived stress and reduced depressive symptoms, which then significantly predicted less binge eating severity.

These associations remained significant across sex and history of major depressive disorder (MDD). No significant associations were observed between perceived control, binge eating severity, and mood, stress, or HF-HRV/RSA reactivity.

Results from the current investigation suggest that perceived control may buffer individuals from stress and depressive symptoms and predict less severe binge eating symptoms. Importantly, perceived control is an adaptive variable that can be modified through experience (Surtees et al., 2010). In line with prior research, which suggests that perceived control may be a malleable treatment target and predictive of positive outcomes following CBT for anxiety and mood disorders (Doering et al., 2015), the current results propose that perceived control may be a universal treatment target across various binge eating populations.

DEDICATION

I dedicate this dissertation to my parents who instilled in me the joy of learning and the value of giving my best effort, no matter the task. Thank you for your unwavering support, encouragement, and love.

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to the chair of my dissertation committee, my research advisor, and my mentor, Dr. Emily Haigh. You never fail to inspire me to think a little harder, to try in the face of potential risk, and to feel confident in who I am and what I have learned so far. It is with your guidance that I have begun to develop into the clinician and scientist I hope to become. Thank you for your unfailing support through good times and bad; you will be my mentor until I turn 85 years old… and beyond.

I would like to thank my dissertation committee members for their time and energy throughout this process. Without your expertise and support, this investigation would not have been possible. Thank you to the psychology faculty: you helped me discover corners of this field I never believed I would enjoy learning… yes, even statistics. To Dr. Doug Nangle: thank you for not only pushing me to do better, but for supporting and encouraging me from day one. A special thank you to Drs. Shannon McCoy, Sandy Sigmon, and April O'Grady who believed in me before I believed in myself. To my exceptional lab mates Olivia Bogucki, Victoria Quinones, and Colin Bosma: thank you for your hard work, your ongoing encouragement, and constant good humor (even in the face of yet another Mindware or E-Prime malfunction). Without your teamwork and friendship, I would have never completed this study. To the undergraduate research assistants (Meaghan Delcourt, Amber Stacy, Liv LiaBraaten, Ian Raugh, Chris Carey, Ryan Boucher, Natasha Bourgoin, Charlee Noble, Daniele Gold, Marisa Tandy, Maliyan Binette, McKenna Martin, Bryanna Plummer, Katie Willis, Erin Nason, and Kim Herron) who helped me complete this experiment and brought the MMDL to life: thank you for your willingness to learn and ask questions, for bringing your ideas to the table, and for devoting your time and energy to this project. To Shannon Brothers and Helen Day: I do not have the

literary skill to adequately describe what you two mean to me. Thank you quite simply for being who you are and loving me for who I am. You are family. To all my friends in the graduate program: thank you for making these years about more than just classroom assignments and stressful deadlines. Each of you is well-equipped to touch this field in unique and amazing ways; don't forget that. A special thank you to my newest friends and mentors in Boston: you have helped me better understand who I am as a clinician and what I hope to gain during the next phase of my training and career. To those amazing providers I had the luck to work with before beginning this journey; without you, I would never have submitted my application to this wonderful program. Thank you.

To my family (Mom, Daddy-O, Gram and Gramp, and the world's best uncles, aunts, and cousins): our family may be small, but the patience you've demonstrated and support you've provided throughout this process has been immeasurable. Thank you for celebrating my victories and helping me find my footing after defeat. I love you all more than words can express. To my second family (Robin, Walt, Sam, Shelby, Donny, and many more): thank you for your constant encouragement and confidence in me. This would not have been possible without you. Lastly, thank you to Nicholas Clukey. No one has been more patient and supportive during this time than you. I am so happy that we've traveled this path together despite unexpected bumps, and I simply cannot wait to see what is around the next bend. Let's adventure.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

Recently designated a distinct diagnosis, binge eating disorder (BED) is characterized by the consumption of a larger amount of food than most would consume in similar contexts and discrete time periods, and experiencing a loss of control during these binges [American Psychiatric Association (APA), 2013]. As the most prevalent eating disorder worldwide, it has been projected that 5% of the United States population will experience symptoms of BED within their lifetime, making this a significant public health concern (Mathes, Brownley, Mo & Bulik, 2009; Mitchell, 2016). Individuals who meet diagnostic criteria for BED typically report lower life satisfaction, greater emotional distress, more functional impairment, and experience higher rates of psychiatric and medical comorbidity than individuals without this disorder (APA, 2013; Grucza, Przybeck, & Cloninger, 2007; Mitchell & Mussell, 1995; Smith & Robbins, 2013). For example, medical research on cardiac function has found that obese women with BED are at greater risk for cardiovascular disease than obese women without BED (Friederich et al., 2006). However, the majority of individuals who meet diagnostic criteria for BED are not obese (i.e., approximately 65%), yet report levels of eating pathology and distress that are comparable to obese women with BED (Didie & Fitzgibbon, 2005). Additionally, research suggests that subthreshold binge eating, or experiencing a loss of control while consuming objectively small amounts of food or binging less than once a week for three months as required for BED diagnosis, is more prevalent than threshold BED (Hudson, Kiripi, Pope, & Kessler, 2007). Furthermore, subclinical binge eating is commonly reported in the general population, and similar levels of functional impairment and emotional distress are routinely expressed in both individuals experiencing subthreshold and those reporting clinical-range binge behaviors

(Hudson et a., 2007; Stice, Marti, Shaw & Jaconis, 2009). Taken together, these findings suggest a possible relation between cardiovascular disease and binge eating among non-obese women with subthreshold binge eating symptomatology that has not been addressed in the literature.

Theoretical models and research on the etiology and maintenance of BED have largely focused on identifying triggers for binge eating, such as stress and negative affective states (Groesz et al., 2012; Heatherton & Baumeister, 1991; Kenardy, Arnow, & Agras, 1996; Selby et al., 2008). Laboratory studies have demonstrated that when stressed, obese individuals with BED consume significantly more calories, eat faster, and change food preferences from healthy to less healthy options than obese individuals without BED (Laessle & Schultz, 2009; Zellner et al., 2006). Additionally, studies employing real-time assessment have found that increases in negative affect are associated with binge eating episodes in obese women with BED (Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007). However, research examining whether stress and negative affect consistently trigger binge eating has yielded mixed results, including the findings that stress did not predict binge eating in African American women (Napolitano & Himes, 2011), and that increases in negative mood did not lead to binge eating in a female college sample with subclinical binge behaviors (Wegner et al., 2002). One explanation for these mixed results may be related to the role of perceived control.

Despite the putative role of control over eating in BED diagnosis and treatment, the construct of overall perceived control has received little research attention to date. Perceived control refers to perceptions of one's ability to impact his or her behavior and environment to reach desired goals (Wallston, Wallston, Smith & Dobbins, 1987). Research has shown that increased levels of perceived control can buffer individuals from stress and predict improved recovery in cardiac surgery patients (Bollini, Walker, Hamann, & Kestler, 2004; Dracup et al.,

2003). In a study investigating the relation between perceived control, perceived stress, and binge eating severity in a community sample of racially diverse women, higher levels of perceived control, rather than higher levels of perceived stress, were consistently associated with lower binge eating severity in all racial groups (Goetze, Huff, Saslow, Epel, & McCoy, in preparation, 2018a). Furthermore, a follow-up study found that perceived control significantly predicted binge eating severity in part due to the mediating impact of both perceived stress and negative affect (Goetze, Huff, Bogucki, Haigh, & McCoy, in preparation, 2018b). These findings provide initial evidence for the importance of perceived control as a predictor of binge eating. Given the connection between BED and cardiovascular function in obese women, research that examines the association between perceived control, cardiovascular function, and binge eating symptomatology in normal to overweight populations is warranted.

Binge Eating Overview

Binge eating was first identified as a distinct behavior pattern over a half-century ago (Marcus et al., 1990; Stunkard, 1959). Initially proposed to be an eating style unique to obese individuals, early accounts noted that binge eating was characterized by the episodic consumption of large amounts of food followed by distress and feelings of extreme guilt, which often prompted intermittent attempts at restrictive dieting. Scientific interest in this pattern of behavior increased further when BED was introduced in the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) as a provisional diagnosis distinct from bulimia nervosa and representing a specific example of eating disorder not otherwise specified (EDNOS; Striegel-Moore & Franko, 2003). Binge eating is now formally defined as the consumption of larger amounts of food than most would eat within discrete time periods (e.g., 2 hours), coupled with a loss of control over eating, and is not

restricted to obese individuals (APA, 2013). Recognized as a prevalent problem, binge eating is a key symptom across eating disorders, including bulimia nervosa, anorexia nervosa of the binge/purge subtype, and BED (APA, 2013; Aubie & Jarry, 2009).

While research suggests that binge eating is unique from other forms of overeating, such as food addiction and emotional eating, there appears to be significant overlap and interaction between these eating patterns (Salamone & Correa, 2013). Whereas binge eating is defined as the uncontrollable consumption of large quantities of food, food addiction is defined by the excessive ingestion of palatable foods high in sugar, fat, and/or salt (Davis, 2015). Similar to how repeated substance use can lead to behavioral and neurophysiological changes in the brain, researchers have suggested that individuals can become dependent on highly palatable food (Davis, 2013). In contrast, the definition of emotional eating is the intake of food in response to negative emotions (Bongers, van den Akker, Havermans, & Jansen, 2015). Given these non-exclusive definitions, research findings have been confounded by the overlap in these eating patterns. For example, in a recent study investigating food addiction, of 120 adult men and women reporting symptoms of food addiction determined by a self-report measure, only 24% met diagnostic criteria for BED via structured clinical interview (Davis et al., 2013). Those who met criteria for BED reported more severe binge eating, greater food cravings, hedonically driven eating, and more depressive symptoms. Similarly, emotional eating may contribute to eating disorders as evidenced by the observation that negative emotions often prompt overeating in those reporting binge behavior. In Chua, Touyz, and Hill's (2004) study of obese females diagnosed with BED via self-report questionnaire, those exposed to a sad film clip designed to induce transient negative mood ate significantly more food in a subsequent taste-task than those in a neutral mood condition, suggesting a link between binge eating and emotional eating.

Despite the overlap that may occur between these eating patterns, increased rates of distress and impairment have been linked to the loss of control over eating characteristic of binge eating, but not food addiction or emotional eating styles (Telch, Pratt, & Niego, 1998; Mond, Latner, Hay, Owen, & Rodgers, 2010). Therefore, the unique symptom cluster associated with binge eating has been of special interest to researchers investigating maladaptive eating patterns and associated functional impairment.

Among the constellation of symptoms associated with clinical BED, research suggests that subjective loss of control over eating appears to be most closely linked to maladaptive outcomes (Latner, Vallance, & Buckett, 2008; Mond et al., 2010). Self-reported levels of functional impairment and emotional distress in those with subthreshold binge eating are similar to the reports of individuals meeting full diagnostic criteria for BED (Stice et al., 2009). Subthreshold binge eating is used to describe the experience of a loss of control while consuming objectively small or average amounts of food or binging less than once a week for three months as required for BED diagnosis. In a study examining health-related quality of life in 53 women with various eating disorders that shared binge eating as a common symptom (i.e., bulimia nervosa, anorexia nervosa of the binge/purge subtype, BED, and eating disorder not otherwise specified), results suggested that reports of subjective loss of control over eating may be more closely linked to diminished quality of life and psychiatric distress than the consumption of objectively large amounts of food (Latner et al., 2008). In this study, participants completed measures of health-related quality of life, eating-related psychopathology, and depressive symptoms. Compared to general population norms, participants reported a high level of eating- and depression-related psychopathology, and reports of diminished mental-health quality of life (e.g., frequent feelings of nervousness and depression, problems with work or daily activities,

feeling tired due to emotional or physical health problems). Additionally, it was found that the frequency of subjective binge eating (i.e., the intake of small or average amounts of food while still experiencing a loss of control over eating) was a significantly better predictor of overall quality of life (e.g., physical, mental, social functioning) than objective binge eating episodes, which are required to meet diagnostic criteria for formal eating disorders such as BED. These findings led researchers to propose that both subjective and objective binge eating may be independently associated with impairment in health-related quality of life, and therefore should not be disregarded in clinical and research settings.

The potential negative impact of subclinical binge eating was further illustrated by Mond and colleagues (2010) in their investigation of differences in eating disorder psychopathology, general psychological distress, impairment in role functioning, and healthcare service utilization in individuals reporting objective and subjective binge eating. A sample of 89 women meeting criteria for bulimia nervosa, BED, or subthreshold binge eating as determined by a semi-structured clinical interview, were divided into two groups based on reports of experiencing only objective binge eating or only subjective binges. Findings revealed that significant differences between groups were limited to weight status, as those who regularly ate objectively large amounts of food had significantly greater body mass index's (BMI's) than those who reported only subjective binge eating. Otherwise, findings indicated that both groups reported similar elevated rates of psychopathology and functional impairment. Results such as these strongly suggest that subclinical binge eating may not differ in a clinically meaningful way from diagnostic-level binge eating, and that the perceived loss of control over eating shared by these conditions may be especially relevant to associated distress and impairment, and therefore requires further attention.

Binge Eating Disorder

Binge eating disorder and bulimia nervosa are both characterized by recurrent episodes of binge eating, but differ in compensatory behaviors. A key component of bulimia is the regular use of self-induced vomiting, misuse of laxatives, or excessive exercise in an attempt to control weight. However, individuals with BED binge eat, on average, once weekly for at least three months without the use of compensatory strategies (APA, 2013; Messerli-Bürgy, Engesser, Lemmenmeier, Steptoe, & Laederach-Hofmann, 2010). Those who meet diagnostic criteria for BED report distress in regard to this behavior, and binge episodes are frequently associated with rapid eating until uncomfortably full, eating in the absence of physical hunger, eating alone due to embarrassment about the amount of food consumed, and marked feelings of disgust, depression, and guilt following a binge (APA, 2013).

Prevalence

Despite being a relatively recent addition to the Diagnostic and Statistical Manual of Mental Disorders (5th edition; APA, 2013), BED represents a significant public health concern. Considered the most widespread eating disorder with a lifetime prevalence rate of 3% to 8% in the United States (Davis, 2015; Voon, 2015), it has been projected that approximately 5% of the U.S. population will experience symptoms consistent with BED within their lifetime (Mathes et al., 2009). Individuals with BED have increased healthcare utilization, and annual direct healthcare costs per patient range between $2,000 and $4,000 more than those without these symptoms (Ágh et al., 2015). Although the economic burden and health service use of BED has been largely unstudied (Simon, Schmidt, & Pilling, 2005), researchers cite the $147 billion annual cost of obesity and obesity-related health conditions in the U.S. as additional evidence of BED's role in a growing public health problem (Klatzkin, Gaffney, Cyrus, Bigus & Brownley,

2015). BED is not unique to the U.S., as the World Health Organization Mental Health Survey Study of over 24,000 community-dwelling adults found a lifetime prevalence rate averaging 1.4%, making BED the most common occurring eating disorder worldwide (Mitchell, 2016). Despite these rates, fewer than half of those meeting BED diagnostic criteria will seek and receive treatment (Kessler et al., 2013), indicating that there is ample need for further research and psychoeducation of this widespread problem area.

Sex. Epidemiological studies of BED have indicated that unlike previously recognized eating disorders, prevalence rates of binge eating in women and men are more equivalent (2:1, respectively; APA, 2013; Hudson et al., 2007; Mitchell, 2016), with one community study indicating that adult men are as likely to screen positive for BED symptoms as adult women (Grucza et al., 2007). Utilizing data from the National Comorbidity Replication, a nationally representative face-to-face household survey conducted between 2001 and 2003, lifetime prevalence estimates of BED were 3.5% in women, and 2.0% in men. Across sex, researchers found that there was an increased risk for severe obesity, psychopathology, role impairment, and under-treatment in those with BED, leading authors to conclude that BED is a growing public health concern (Hudson et al., 2007). This 2:1, female-to-male ratio in incidence rates has been observed worldwide, with an elevated lifetime risk for binge eating in women and more recent cohorts (i.e., women younger than 40 years of age; Kessler et al., 2013; Mitchell, 2016; Reagan & Hersch, 2005). Due to these prevalence trends, much of the research to date has focused on female samples.

Despite the focus on women in binge eating research, Shingleton and colleagues (2015) completed a recent analysis of data pooled from 11 randomized controlled psychosocial treatment studies for BED to investigate baseline and outcome characteristics by sex. Once

aggregated, data for 208 males and 1,117 females were reviewed. The data indicated that men tended to report lower shape, weight, and eating concerns at baseline than women, but there was no significant main effect of sex on symptom severity following treatment completion. However, findings did reveal a significant interaction between sex, treatment length, and shape/weight concern. Men with lower baseline shape and weight concern had a significant reduction in binge eating following shorter treatments, whereas women and men with higher baseline concerns regarding shape and weight benefited most from longer interventions. These findings not only underscore the importance of considering sex when treating BED, but that recruitment and inclusion of males for treatment research should be targeted. Despite a more equivalent BED prevalence across sex than is observed in other eating disorders, the sex divide among participants in treatment studies is not surprising. Women are more likely to seek and receive treatment for BED (Kessler et al., 2013), and although the reasons for this are not completely understood, it has been posited that increased distress associated with sociocultural expectations of diet and weight for women plays a significant role (Davis, 2015).

Age. The median age of onset for both bulimia nervosa and BED is in the late teens to early 20's, although it is generally accepted that BED onset begins later in development than other eating disorders, with the first presentation of diagnostic symptoms generally occurring in early adulthood (APA, 2103; Kessler et al., 2013). In part due to this typical age at onset, much of the research on this relatively new diagnosis has been conducted with college samples aged 18 to 25 (McCabe & Ricciardelli, 2004; Tiggemann & Lynch, 2001). The transition to college includes unique challenges related to role transitions and increased social, academic, and life stressors that likely play a significant role in BED development.

Course. A study following 186 female college freshmen over two months found that most disordered eating patterns were likely to remain stable or decrease over time, with the exception of binge eating (Berg, Frazier, & Sherr, 2009), leading researchers to hypothesize that this maladaptive pattern of eating may be especially prevalent throughout young adulthood. In keeping with these results, in a large sample of 969 racially diverse undergraduate women, 8.4% of participants reported symptoms consistent with BED, while a surprising 44% reported severe, but subclinical, binge eating behaviors (Napolitano & Himes, 2011). Moreover, epidemiological research has traditionally indicated that eating disorders impact adolescent and young adult women more than other cohorts, but emerging data suggests that binge eating occurs across the lifespan (Brandsma, 2007).

Recent research has begun to examine binge eating in pediatric and older adult samples. Few children under the age of twelve meet current diagnostic criteria for BED, however, it has been suggested that BED symptoms may present differently in childhood than adulthood (Tanofsky-Kraff, Marcu, Yanovski, & Yanovski, 2008). For example, in studies focusing on loss of control eating instead of clinical-range BED behaviors, 2% to 10% of non-treatment seeking children aged 6 to 14 report feeling they have little control over eating (Morgan et al., 2002; Tanofsky-Kraff et al., 2008). In adulthood, data suggests that nearly half of women with BED seeking treatment do not begin experiencing clinical symptoms until middle-adulthood (Brandsma, 2007). Women who seek treatment for BED are typically significantly older than women seeking treatment for other eating disorders. Similarly, remission rates for BED are typically lower than those for anorexia and bulimia, suggesting that this persistent disorder can continue throughout various periods of adult development (APA, 2013). Relapse rates for individuals meeting diagnostic criteria for BED are often over 50% in spite of instances of

prolonged clinical treatment (Bello & Hajnal, 2010), and therefore, binge eating behavior can be a chronic disorder. However, an analysis of self-reported data from a community sample of 573 women and 360 men aged 18 to 97 revealed that BED is most common in adults younger than 40 years of age (Reagan & Hersch, 2005), which suggests that although research on BED in younger and older populations is needed, early adulthood represents the age period BED is most prevalent.

Race/ethnicity. Similar to epidemiological findings related to sex, studies on binge eating and BED find less disparity across racial and ethnic groups than other eating disorders (APA, 2013; Harrington, Crowther, Henrickson, & Mickelson, 2006; Marques et al., 2011). Overall, there is general research consensus that African Americans, Asian Americans, and Hispanic Americans experience binge eating and BED at comparable rates relative to European Americans (Chao, Grilo, & Sinha, 2016; Hudson et al., 2007). Additionally, the World Health Organization recently determined that BED prevalence is similar across countries (Kessler et al., 2013). However, there is some debate as to whether BED symptomatology presents similarly across racial groups.

Community-based studies have indicated that the frequency and overall distress associated with binge eating may be equivalent in African American and European American samples. Reagan and Hersch (2005) noted in their large sample of 933 adult men and women that frequency of binge eating was not significantly impacted by race, with similar self-reports in both African American and European American participants. Correspondingly, data from the National Institute of Mental Health Collaborative Psychiatric Epidemiological Studies has indicated that African American and Hispanic American individuals report similar levels of impairment due to BED as European Americans (Marques et al., 2011). However, it was also

noted that European American participants are more likely to seek BED treatment than other racial groups. This aligns with findings from Pike and colleagues (2001) who found that in a group of 150 African American and European American women meeting diagnostic criteria for BED via semi-structured clinical interview, European American women reported significantly higher levels of psychiatric distress and eating, shape, and weight concerns. Although European American women in this sample were more than twice as likely as African American women to meet criteria for two or more current psychiatric diagnoses, it was determined that both racial groups were equally likely to have one additional comorbid mental health disorder, and rates of major depression and social anxiety were consistent across groups.

Racial and ethnic group characteristics in those seeking BED treatment are less well understood, but preliminary findings suggest there may be significant differences in individuals reporting BED in the community versus those seeking treatment. A study investigating these differences in African American and European American women by comparing clinical characteristics of a recruited clinical sample with data from a previously collected community sample found significant differences between community and treatment seeking African American individuals (Grilo, Lozano, & Masheb, 2005). Results revealed that treatment seeking African American women reported significantly higher BMI's, lower binge eating frequency, and greater dietary and shape concerns than community samples of African American women not seeking treatment. These findings led authors to hypothesize that African American women may wait to seek treatment until they are particularly distressed about their bodies and eating habits. Additionally, it was noted that caution should be used when comparing community and treatment-seeking samples as there may be significant clinical differences between groups due to this type of sampling bias.

To learn more about treatment group characteristics, Franko and colleagues (2012) collected data from 11 research sites conducting psychosocial intervention clinical trials for adults with BED. There was no significant difference in binge eating frequency or severity between African American, Hispanic American, and European American women, and this finding remained non-significant after adjusting for weight, education, and socioeconomic status (SES). However, African American women were again noted to have significantly greater BMI's, and Hispanic American participants reported greater eating disorder psychopathology (e.g., concerns about weight, shape, and eating behavior) than European Americans, leading authors to suggest that culturally relevant variables such as attitudes toward weight gain should be considered when treating Hispanic American individuals with BED. Later research which investigated racial similarities and differences in men and women seeking BED treatment was unable to replicate this finding, but did note that in their sample of 755 African American, Hispanic American, and European American participants with BED determined by semi-structured clinical interview, African Americans again had significantly greater BMI's (Lydecker & Grilo, 2015). Unique to this sample, African Americans were found to report significantly more frequent binge eating episodes than European American participants, but significantly fewer symptoms of depression. Eating disorder distress and pathology did not significantly differ across racial groups before or after adjusting for age, education, sex, and weight, consistent with Franko et al.'s findings (2012). Although prevalence rates of binge eating and BED appear consistent across racial and ethnic groups, preliminary research suggests that there may be some differences in presentation such that BMI and distress regarding shape and weight may vary by racial group (e.g., Franko et al., 2012). Additional research has found significantly different

reports of distress and binge frequency between community and clinical African American samples (Grilo et al., 2005).

Socioeconomic status. To date, little is known about the impact of SES on binge eating. Although lower SES has been observed as a risk factor for BED across racial and ethnic groups (Franko et al., 2012), etiological and treatment studies of binge eating have rarely examined questions related to SES. Overall, epidemiological research suggests that there are complex relations between race, ethnicity, SES, BMI, and health status, and studies of medical comorbidity have noted that differences between racial or ethnic groups are often better accounted for by differences in SES than race (Farmer & Ferraro, 2005). For instance, in a review of 144 published studies examining the association between obesity and SES, there was a significant inverse relation found in women such that those reporting lower SES had significantly greater BMIs, although this pattern was inconsistent in men and children (Sobal & Stunkard, 1989). Moreover, findings from the U.S. National Health and Nutrition Examination Survey data revealed significant interactions between race, education, and employment status on health outcomes, indicating that SES may be an important variable to consider in future health-related research (Farmer & Ferraro, 2005). Although preliminary epidemiological and community studies specifically focused on binge eating have found that low SES may be associated with elevated risk for BED across racial groups (Alegria et al., 2007; Franko et al., 2012; Reagan & Hersch, 2005), the investigation into SES and interactions between various demographic variables requires further research.

Body mass index. Excess adiposity, or obesity, is generally defined as a BMI greater than or equal to 30 [i.e., weight (kg)/height $(m)^2$; Marcus & Wildes, 2014]. In the United States, rates of obesity have doubled in the last three decades, while reports of morbid obesity (i.e., BMI

greater than 40) have increased by four-fold (Davis, 2015). Approximately 70% of adults in the U.S. are currently overweight or obese, and over 15% of children and adolescents are overweight with an additional 30% at risk of becoming overweight (Wang & Beydoun, 2007). Although obesity is typically conceptualized as a condition caused by both environmental and genetic factors, behavioral influences such as disordered eating significantly impact development and maintenance of increased weight status (Marcus & Wildes, 2014). Given that binge eating disorder is not associated with compensatory behaviors, it is unsurprising that elevated BMI is positively correlated with BED and repeated binge eating. One population-based survey investigating the prevalence and correlates of eating disorders in the U.S. found binge eating was significantly associated with obesity in individuals with BED (Hudson et al., 2007). In fact, the rate of BED has been shown to be consistently elevated among individuals with obesity, with community prevalence rates ranging from 3.3% to 5.5%, but as high as 30% in weight-loss treatment seeking samples (Mitchell, 2016).

Although the prevalence of BED increases with increased BMI, obesity is not a diagnostic feature of this disorder and only approximately 35% of those who regularly binge fall in the obese weight range (APA, 2013; Corwin, Avena, & Boggiano, 2011). In their 2000 study investigating the course of binge eating disorder over a 5-year period in a community sample, Fairburn and colleagues found that of 48 individuals who met diagnostic criteria for BED, only 21% were obese at study recruitment, and only 39% were obese after 5-years of naturalistic study. Similar rates were reported by Carrard, Van der Linden, and Golay (2012), who found that among 74 women recruited from the community who met criteria for BED, only 40% were obese.

Despite findings that BED and binge eating is prevalent among normal and overweight individuals, clinical research investigating BED has historically been limited to obese, female samples (Carrard, Van der Linden, & Golay, 2012; Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). In their 2005 survey study, Didie and Fitzgibbon found that regardless of weight status, individuals with BED have comparable levels of psychological distress and eating pathology. In fact, additional research has suggested that individuals with BED not only report lower quality of life and greater rates of psychiatric comorbidity such as mood disorders, but that these rates are significantly linked to severity of binge eating and not degree of obesity (Carrard, et al., 2012; Mitchell & Mussell, 1995; Grucza et al., 2007). Additionally, there is data to suggest that loss of control eating is correlated with distress and functional impairment, with the only significant difference between individuals with objective and subjective binge eating being BMI (Mond et al., 2010). Taken together, it appears that the distress and impairment associated with binge eating is equivalent across weight status. Therefore, research limited to only obese individuals with BED has likely obscured potential differences in symptoms, etiological factors, and maintenance elements, negatively impacting the generalizability of findings.

Comorbidity

Psychiatric comorbidity. Research investigating the relation between binge eating and psychological comorbidity have found significantly higher levels of eating related pathology in obese individuals who regularly binge eat than obese non-binge eaters (de Zwaan, 2001). BED is significantly associated with psychiatric comorbidity, namely depressive disorders, and individuals who regularly binge eat report elevated rates of psychological distress and impaired self-esteem (Mitchell & Mussell, 1995). Moreover, individuals with BED report social role adjustment problems and indicate lower scores on measures of general health and mental health-

related quality of life, independent of sex, age, education, marital status, and race when compared to those without BED (Grucza et al., 2007).

Eating-related pathology. Research investigating eating-related pathology has found greater body image distress and weight and shape concern in obese individuals who binge eat than weight-matched individuals who do not binge eat (Colles, Dixon & O'Brien, 2008). Utilizing semi-structured clinical interviews and self-report questionnaires, Wilfley, Schwartz, Spurrell, and Fairburn (2000) found that overweight and obese women with BED reported significantly greater distress about eating, shape, and weight than both overweight and normal-weight individuals without binge eating symptomatology. Interestingly, in a study comparing questionnaire data for individuals with BED to both high- and low-weight EDNOS individuals who did not meet full criteria for BED or bulimia, it was found that self-reported levels of weight and shape concern significantly correlated with BED, but not weight status (Eldredge & Agras, 1996). Additionally, BED participants reported a significantly greater tendency to overeat in response to negative mood states than low-weight EDNOS individuals and control participants. However, responses to overeating due to negative mood were similar between BED and high-weight EDNOS participants, which researchers interpreted as evidence that subclinical binge eating may still lead to greater eating related distress than those who do not report repeated binge eating behavior.

Psychiatric symptomatology. Compared to weight-matched, non-binge eating individuals, those with BED have been found to report elevated rates of psychiatric comorbidity and distress (Bulik, Sullivan & Kendler, 2002; Javaras et al., 2008). Approximately 30% to 80% of individuals with BED meet criteria for lifetime comorbid mood or anxiety disorder (Dingemans, Visser, Paul & van Furth, 2015; Sheehan & Herman, 2015). For example, in a

study utilizing semi-structured clinical interviews to compare prevalence of psychiatric disorders among obese binge eaters and obese non-binge eaters of similar age and weight, researchers found that 60% of those reporting regular binge eating behavior met criteria for one or more psychiatric disorders compared to only 28% of obese individuals without binge eating behaviors (Marcus et al., 1990). In a later study investigating the prevalence of BED in a sample of obese females seeking weight-loss treatment, semi-structured clinical interviews revealed that those meeting diagnostic criteria for BED had significantly higher lifetime rates of Axis I diagnoses and Axis II, primarily cluster B and C diagnoses (Specker, de Zwaan, Raymond, & James, 1994). Of these comorbid conditions, major depression appeared to be the most prevalent. Similar results were observed in a large face-to-face U.S. household community survey (N = 5,692), where researchers found that 78.9% of individuals with BED met criteria for at least one additional psychiatric disorder, whereas 63.6% of individuals reporting subthreshold binge eating reported clinical levels of mood, anxiety, impulse-control, or substance use disorders (Hudson et al., 2007). Additionally, the majority of individuals with BED and subthreshold binge eating reported significant role impairment due to their symptoms. In keeping with these findings, a community sample self-report questionnaire study found that there were significant associations between BED diagnosis and comorbid major depression, generalized anxiety disorder, panic attacks, alcohol use, and a history of one or more suicide attempts (Grucza et al., 2007). Interestingly, these correlations were not significant for obese non-binge eaters, suggesting that comorbidity was linked to the severity of binge eating and not to the degree of obesity, despite obesity being a major risk factor of BED (APA, 2013; Grucza et al., 2007).

Depression. Noted to be the single most common psychiatric disorder, major depressive disorder (MDD) is characterized by five or more symptoms, including experiencing at least a

two-week period of sad mood and/or a diminished interest and pleasure in activities (APA, 2013; Beidel, Bulik, & Stanley, 2012). Individuals meeting diagnostic criteria for MDD may also experience changes in appetite, weight, sleep and energy levels, report feelings of worthlessness and guilt, experience difficulty concentrating, and/or have recurrent thoughts of death or suicidal ideation. The most prevalent comorbid lifetime diagnosis for individuals with BED is MDD, and researchers have found that severity of binge eating is positively related to severity of depressive symptomatology (Dingemans, Martijin, Jansen, & van Furth, 2009), with higher levels of depression associated with more severe binge eating (Dingemans et al., 2015). This finding is unsurprising when the literature investigating potential links between obesity and depression is reviewed. For example, there appears to be a significant relation between depression and obesity in women, such that higher BMI is associated with more severe depressive symptoms (Stunkard, Faith, & Allison, 2003). In a weight-loss treatment seeking sample, it was observed that those with mild to moderate depressive symptom severity not only had significantly greater BMI, but also reported lower self-esteem and more shape, weight, and eating concerns than those without depressive symptoms (Werrij, Mulkens, Hospers, & Jansen, 2006). This trend has also been found in community samples, such as the Simon and colleagues 2008 study that included structured telephone interviews with 4,641 women in the U.S. to evaluate weight status and depression symptomatology. Interestingly, there were significantly greater moderate to severe depression symptom levels (i.e., from 6.5% to 25.9%) reported from normal-weight versus obese individuals, respectively. In addition, the prevalence of obesity was lower (i.e., 25.4%) in those reporting no depressive symptomatology than among those with moderate to severe levels of depression (i.e., 57.8%). Overall, meta-analytic summaries of research investigating the relation between obesity and depression have suggested a significant positive association between

depression and weight status (de Wit et al., 2010), but how binge eating may fit into this correlation is not fully understood.

Although there appears to be a significant positive relation between obesity and depression symptomatology, it is less clear how this impacts individuals with BED (Barry, Grilo, & Masheb, 2003). A 2015 literature review investigating the association between BED and impaired mental health (Sheehan & Herman) found that the presence of comorbid depressive symptoms in individuals with BED did not appear to be solely related to weight status. Findings revealed that both obese and non-obese individuals with BED reported similar levels of depression severity. Collectively, research suggests that there are likely complex interactions between binge eating, obesity, and comorbid depression (Hughes et al., 2013). For example, in a sample of 113 women enrolled in a weight loss treatment program, researchers found that individuals meeting diagnostic criteria for BED reported significantly more severe depressive symptoms (Bittencourt, Lucena-Santos, Moraes, & Oliveira, 2012). However, in this sample, degree of depression and binge eating severity were not significantly related to BMI. To further investigate this connection in a treatment seeking sample, Dingemans and van Furth (2012) recruited 174 obese and non-obese individuals diagnosed with BED via clinical interview from a randomized controlled treatment trial sample. Primary findings indicated there were more similarities than differences between groups, with severity of self-reported depression symptoms falling in the mild to moderate range for both obese and non-obese individuals with BED. The authors concluded that not only did their results suggest that severity of psychopathology may be linked more closely to BED diagnosis than weight status, but that there should be increased research targeting non-obese individuals who binge eat as this is a largely ignored study population despite similar rates of depression and distress.

It is currently unclear if BED is an antecedent to depression and other psychiatric disorders, a complication associated with comorbid conditions, or an unrelated set of symptoms that occurs concurrently with other problem areas. Although the relations between depression, weight status, and binge eating may not be fully understood, it appears that depression can significantly influence BED expression and treatment outcomes (Sheehan & Herman, 2015). In a sample of 131 obese individuals enrolled in weight-loss treatment, 17% met criteria for MDD only, 13% diagnosed with BED only, and 17% met criteria for both MDD and BED (Pagoto et al., 2007). Although all three groups demonstrated significantly less weight-loss than obese individuals without psychiatric complaints, fewer individuals with both MDD and BED achieved clinically significant weight loss than those in other groups, illustrating that this psychiatric combination may be associated with worse treatment outcomes. Similarly, in a three-year follow-up study to evaluate the relation between psychiatric variables and binge eating episodes following cognitive behavioral therapy (CBT), researchers found that individuals with BED reported greater depression prior to treatment than other treatment-seeking individuals who binge ate (i.e., those meeting criteria for bulimia; Castellini et al., 2011). During treatment, reductions in binge eating in the BED group were significantly associated with reductions in depressive symptoms, which was again observed three-years post-treatment before and after controlling for BMI. Due to these findings, the authors concluded that given the role of different psychiatric variables in binge eating expression, treatment approaches may be more effective if these variables are included as targets for intervention. Similarly, research investigating constructs related to binge eating should be aware of the rates and possible influence of depressive symptoms on results prior to interpreting findings.

Medical comorbidity. Epidemiological investigations have revealed that binge eating is associated with obesity and overweight status, and that the prevalence rate of BED increases with increased BMI (APA, 2013). Coupled with this, researchers have found a sharp increase in average BMI rates over the last three decades, and obesity-related maladies such as type II diabetes, hypertension, and liver disease have replaced smoking as the leading cause of preventable death in adults (Bulik & Reichborn-Kjennerud, 2003; Smith & Robbins, 2013). Given the comorbidity between BED and obesity, interest in the medical health risks associated with binge eating has become a growing area of research. Interestingly, it has been observed that obese individuals with BED are at an increased risk for medical morbidity and mortality, and have greater health-care utilization compared to BMI-matched individuals without BED (APA, 2013; Smith & Robbins, 2013). This finding illustrates that BED may significantly and negatively impact health beyond the effects of obesity alone. However, despite data indicating that the minority of individuals with BED are obese (Didie & Fitzgibbon, 2005), little has been done to explore the potential maladaptive health impacts BED may have in normal to overweight individuals with binge eating behaviors (Messerli-Bürgy et al., 2010). Research that focuses on the health impact of repeated binge eating among those without comorbid obesity is of utmost importance and will fill a gap in the understanding of binge eating.

Physical wellbeing and quality of life. Largely dependent on self-report questionnaires, several studies have found that individuals with subthreshold binge eating or meeting diagnostic criteria for BED report significantly greater health dissatisfaction (e.g., Bulik et al., 2002). For example, in a large self-report questionnaire study (N = 4,654 female participants) investigating the prevalence of binge eating, psychiatric comorbidity, physical illness, and functional limitations in primary care and obstetric gynecology clinics in the U.S., women who reported

symptoms consistent with BED also reported significantly greater limitation in daily activities, poorer overall health, and impaired functioning when compared to individuals without BED (Johnson, Spitzer & Williams, 2001). It is noteworthy that despite significant psychiatric comorbidity in those with BED, the relation between binge eating symptoms, poor health and impaired functioning remained significant after controlling for comorbid conditions such as MDD. Bulik, Sullivan, and Kendler (2002) utilized a semi-structured clinical interview design and found similar results in their population-based, five-year longitudinal study investigating the prevalence of obesity and binge eating in female twins. The researchers found that obese binge eaters not only reported higher rates of major medical disorders than obese non-binge eaters, but also reported significantly greater overall health dissatisfaction. Similarly, in a community study aimed at identifying correlates of BED, self-report data indicated that obese individuals who regularly binge ate reported significantly lower health-related quality of life than obese individuals without BED (Grucza et al., 2007).

Due to findings that suggest diminished health-related quality of life in those with BED, an association between BED and poor health independent of psychiatric, medical, and BMI comorbidity has been hypothesized. In Marchesini and colleagues (2002) treatment study investigating the impact of CBT on health-related quality of life in obese participants with and without BED, individuals with BED treated with CBT for both weight loss and eating pathology experienced less weight loss than individuals without BED who underwent weight loss CBT only. However, individuals in the BED group reported significantly greater increases in health-related quality of life when compared to the non-BED group. In fact, individuals with BED reported improvements in physical and emotional role limitation after treatment and importantly noted significant improvements in perceived health status independent of changes in body

weight. Findings such as these suggest that the relation between BED and poor health may extend beyond medical comorbidity and obesity alone (Sheehan & Herman, 2015), although the direction and mechanisms of this connection are not yet well understood.

Medical symptomatology. Investigating medical morbidity in individuals with BED may be especially important due to the high rates of obesity and related risks of hypertension, diabetes, coronary heart disease, osteoarthritis, sleep apnea, stroke, and some forms of cancer (Bulik & Reichborn-Kjennerud, 2003). There is a growing literature investigating physical health and prevalence of medical conditions in those who binge eat. Overall findings indicate that individuals with BED may be at greater risk for medical morbidity and mortality than those who do not engage in binge eating behaviors (Mitchell, 2016). In a study exploring the association between binge eating and health problems in female participants from primary care and obstetric clinics, women reporting recurrent binge eating behavior (e.g., BED or bulimia) had greater rates of diabetes and reported significantly greater physical symptoms (e.g., joint pain, headache, chest pain, shortness of breath, and gastrointestinal problems; Johnson et al., 2001). Importantly, these rates of physical complaints remained significantly higher in individuals with BED after controlling for co-occurring alcohol use, anxiety, and mood disorders. While the authors suggested that their findings indicated that BED may be associated with greater rates of physical morbidity, the study suffered from a major limitation that would obscure such an interpretation. Specifically, BMI was not controlled for in this investigation and could have contributed to the observed group differences.

To address the potential confounding impact of BMI in investigations of BED and medical comorbidity, researchers have attempted to control for the impact of obesity. Preliminary results from these investigations suggest that binge eating is associated with greater

physical health impairment beyond the impact of elevated BMI alone (Smith & Robbins, 2013). For example, in their female twin study investigating obesity, binge eating, and rates of major medical conditions, Bulik and colleagues (2002) found that despite no significant differences in BMI or age between those reporting regular binge eating and those without BED, a significantly larger percentage of obese individuals who binge ate also reported higher rates of major medical conditions (e.g., hypertension, respiratory illness, diabetes, osteoarthritis, and cardiac problems). A later meta-analysis that examined the correlation between BED, physical health, and weight status concluded that although research was in its infancy, binge eating may be associated with medical morbidity independent of the effect of comorbid psychiatric symptoms and obesity (Bulik & Reichborn-Kjennerud, 2003). Later studies supported these conclusions, finding that BED independent of BMI status was associated with higher rates of insomnia (e.g., Reichborn-Kjennerud, Bulik, Sullivan, Tambs & Harris, 2004), greater risk for metabolic syndrome conditions such as hypertension and type 2 diabetes (e.g., Hudson et al., 2010), chronic pain (e.g., Kessler et al., 2013), gastrointestinal complaints (e.g., Sheehan & Herman, 2015), and cardiac risk factors (e.g., Grilo, 2015). Surprisingly, despite these numerous findings, a recent literature review of medical complications associated with binge eating cautioned medical professionals to exercise great care regarding potential increased health risks in patients with obesity and BED; however, stated that there was currently no reason to suggest extra caution with non-obese patients with BED (Mitchell, 2016). It would seem a more prudent conclusion might be to suggest that medical risk in those who are not obese and binge eat is an area in great need of further study rather than an area requiring little concern.

Cardiovascular health. An area emerging as especially important for further research is the association between BED and cardiovascular health and disease risk. Cardiovascular disease

is the leading cause of death and disability worldwide (e.g., Thayer, Yamamoto, & Brosschot, 2010). Research examining medical risks linked with BED indicate that binge eating is significantly associated with cardiovascular problems, including coronary heart disease, heart failure, and hypertension (Mitchell, 2016; Sheehan & Herman, 2015). Although preliminary, researchers focusing on cardiac wellness has found that women with BED may be at greater risk for cardiovascular disease independent of obese weight status (Friederich et al., 2006). To further investigate the relation between binge eating, weight, and cardiac wellness, research on binge eating is beginning to address physiological measures of cardiovascular function (e.g., Friederich et al., 2006; Ranzenhofer et al., 2016).

Heart Rate Variability

In order to advance a more comprehensive understanding of binge eating pathology, it is necessary to conduct research that incorporates multiple modes of analysis. An emerging area of research that expands methods used to study BED is the investigation of physiological correlates of binge eating, including cardiovascular function. The autonomic nervous system (ANS) is primarily responsible for the internal regulation of body functions in an attempt to maintain homeostasis and is integral to cardiovascular function and response (e.g., Porges, 1995). The ANS is composed of two subsystems: the sympathetic and parasympathetic nervous systems (SNS and PSNS, respectively). The SNS promotes activation and increased metabolic output in response to external environmental challenges, whereas the PSNS has the primary role of restoring and maintaining baseline levels of body functioning. Therefore, these systems have an antagonistic association such that when one is activated, the other is suppressed. Together the coordinated responses of the SNS and PSNS work to maintain appropriate internal physiological states capable of adapting to changes in both internal and external demands (e.g., Porges, 1995).

In addition to the branches of the ANS, the central autonomic network (CAN) made up by cortical, limbic, and brainstem regions, allows the body to adjust to changing environmental demands by regulating physiological arousal (e.g., Appelhans & Luecken, 2006). Due to these interacting networks, the cardiovascular system is highly sensitive to neurobehavioral processes and is impacted by psychological factors such as stress, depression, and emotional arousal (Berntson, Quoigley, & Lozan, 2007). Utilizing internal and environmental information from the CAN, the PSNS and SNS regulate cardiac activity by adjusting heart rate and length of time between consecutive beats (i.e., interbeat interval).

Heart rate variability (HRV), or the ability of the heart to adjust to changing demands, represents the interplay between the SNS and the PSNS, overall autonomic flexibility, and cardiovascular health (Appelhans & Luecken, 2006). The HRV of healthy individuals naturally decreases under situations of emotional or physical stress but increases during times of rest, illustrating adaptive ANS function (Dekker et al., 2000). However, significantly reduced HRV, or the inability for components of the ANS to quickly and adaptively respond to changes in the environment, is a known risk factor for mortality in both patient and healthy populations (Nunan, Sandercock, & Brodi, 2010; Thayer & Brosschot, 2005). Derived from estimating the variation of interbeat intervals, determining HRV requires continuous heart rate measurement, typically through electrocardiography (ECG). ECG is a non-invasive measure of cardiac and ANS function that has increasingly been utilized in research investigating potential health risks associated with both obesity and psychiatric disorders.

There is evidence that HRV represents a useful tool for the assessment of changes in cardiac autonomic modulation such that high HRV indicates adaptively high vagal tone and the ability to quickly adjust to environmental demands (Stein & Kleiger, 1999). Physiological data

collected to investigate HRV can be analyzed in numerous ways (e.g., Berntson, Quigley, & Lozano, 2007). Time domain methods of HRV analysis are derived directly from interbeat intervals or from differences between successive intervals. Although simplest to calculate, time domain methods come at the expense of frequency resolution, which is necessary to obtain a detailed view of cardiac autonomic modulation (Stein & Kleiger, 1999). Frequency domain methods of analysis, although more mathematically complex, separate overall heart period variance into specifiable frequency bands that may be differentially impacted by components of the ANS. While there is some controversy over low-frequency HRV (i.e., LF-HRV; 0.05 – 0.15 Hz) being modulated by primarily SNS activity or both SNS and PSNS input, high-frequency HRV (i.e., HF-HRV; 0.15 – 0.4 Hz) is thought to be largely attributable to variations in parasympathetic control. Expert consensus suggests that HF-HRV serves as a proxy for underlying cardiovascular disease processes and deficits in adaptive ANS response to changing internal and external demands resulting from reduced PSNS regulation (Appelhans & Luecken, 2006; Berntson et al., 2007). HF-HRV generally corresponds with respiratory sinus arrhythmia (RSA) such that respiration rate and depth can accelerate or decelerate heart rate and impact HRV. Although respiratory depth (i.e., the amount of air that moves in and out of lungs with each respiration) may be less likely to impact overall HRV, HF-HRV is best calculated with respiratory rate as a covariate so that significant changes in respiration can be statistically controlled for (i.e., HF-HRV/RSA; Berntson et al., 2007).

Obesity. Due to the significant comorbidity of obesity and BED, and evidence to suggest that obesity significantly impacts HRV (e.g., Laederach-Hofmann, Mussgay, & Ruddel, 2000), research investigating HRV in obese individuals without BED will be considered first. Obesity status alone has been consistently linked with numerous health conditions and mortality,

including cardiovascular disease (e.g., Carroll, Phillips, & Der 2008). Physiological investigations of obese individuals have demonstrated blunted HRV both at rest and under mental challenge (Laederach-Hofmann et al., 2000). Utilizing 24-hour Holter recordings, a study investigating the effects of obesity and weight loss on cardiovascular functioning found that at baseline, obese individuals demonstrated significantly lower HRV than normal-weight individuals (Karason, Mølgaard, Wikstrand, & Sjöström, 1999). Interestingly, following one-year of weight loss, previously obese individuals exhibited a significant increase in HRV compared to baseline values, indicating that HRV can adjust over time. Taken together, research suggests that obesity is significantly associated with blunted HRV and should be considered when investigating cardiac function in obese BED populations.

Depression. Just as obesity may impact research examining HRV in those with BED, the influence of depression on cardiac function must also be considered. MDD is the psychiatric condition most frequently comorbid with BED (e.g., Dingemans et al., 2009) and has been consistently connected with cardiovascular disease and mortality (York et al., 2007). Investigations of mental stress and negative affect have indicated that blunted heart rate reactivity occurs in those reporting more severe depressive symptomology (Jin, Steding, & Webb, 2015; Phillips, 2011; York et al., 2007). A 2010 meta-analysis of HRV in individuals with MDD compared with healthy control participants concluded that depression (without comorbid cardiovascular disease) appears to be significantly associated with blunted HRV at rest and during 24-hour Holter monitoring (Kemp et al., 2010). Findings also indicated that individuals with more severe depressive symptoms demonstrate greater reductions in HRV. Given the frequent comorbidity of MDD and BED, and research indicating that depressive symptoms can significantly impact HRV, research aimed at investigating cardiac function in

those with BED should consider evaluating and controlling for individual differences in depression severity.

Binge eating disorder. Examining cardiac function in BED populations is an emerging area of research. Earlier studies including women with self-reported eating disorder tendencies or meeting diagnostic criteria for bulimia nervosa found that these groups demonstrated significantly lower HRV in response to laboratory psychological stress (e.g., auditory serial addition, mirror task, interpersonal speech task) than non-eating disordered individuals (Ginty, Phillips, Higgs, Heaney, & Carroll, 2012, Koo-Loeb, Pedersen, & Girdler, 1998; Koo-Loeb, Costello, Light, & Girdler, 2000). More recent investigations have begun to focus specifically on binge eating and BED.

Although initial studies of binge eating and cardiac function indicate that individuals with BED may have impaired autonomic stress reactivity and recovery, these few investigations relied upon small sample sizes, which impacts interpretation and generalizability of findings (Messerli-Bürgy et al., 2010). Friederich and colleagues (2006) attempted to address these limitations in their study investigating the effect of physical and social stress on HRV in obese women with BED (n = 38) and age and weight matched women without BED (n = 34). Participants completed two social stress tasks (i.e., Stroop color-word interference test, and reading aloud with delayed acoustic feedback used to evoke stuttering while being evaluated) and Head-up Tilt Testing (i.e., a method of physical stress placing the body at 75 degrees via a motorized tilt table). Researchers found that while there were no group differences in physical stress cardiac functioning, HRV during social stress tasks was significantly blunted in individuals with BED despite both groups falling in the obese weight range. Additionally, this finding remained significant when controlling for comorbid depression across individuals. Results such as these

indicate that a BED diagnosis may be associated with parasympathetic depression during mental stress greater than obesity status alone, potentially placing these individuals at greater risk for cardiac morbidity and mortality. Klatzkin and colleagues (2015) investigated cardiovascular reactivity in obese BED, obese non-BED, and normal weight non-BED women in response to the Trier Social Stress Test, and found that differences in HRV between groups were better accounted for by higher rates of depression in BED individuals. However, while Freiderich et al. did not observe any baseline differences in HRV while participants relaxed in a supine position in a quiet environment following psychophysiology hook-up, Klatzkin and colleagues found significant differences in blood pressure, depressive symptoms, and perceived stress between obese women with BED and both obese and normal-weight women without BED during a 10-minute relaxation period following physiological hook-up. Researchers concluded that there was evidence that obese women with BED had heightened physiological and psychological dysfunction independent of obesity and acute mental status, contradicting earlier findings by Freiderich and colleagues (2006). It is difficult to directly compare these investigations due to differences in methodology, but these studies illustrate the importance of investigating the role that this common psychiatric comorbidity may play (Klatzkin et al., 2015). Although unable to replicate Freiderich et al.'s findings, Klatzkin and colleagues recommended that future research investigate healthy weight individuals with BED given that there has been no empirical study of potential cardiac health risks in this group.

To date, only one additional study has specifically targeted HRV and symptoms characteristic of BED. Ranzenhofer and colleagues (2016) recently completed a pilot investigation of real-time heart rate, HRV, and loss of control eating in the natural environment of 17 adolescent girls assessed via semi-structured clinical interview. Importantly, all

participants were at or above the 85th weight percentile, and none met diagnostic criteria for MDD. Through Holter monitoring, participant cardiac functioning was recorded over two days in addition to instances of loss of control eating. The researchers found that lower HRV was associated with loss of control eating, and when examined categorically, HRV was significantly lower prior to high-loss of control eating episodes than low-loss of control eating periods. Although not investigating BED specifically, this study indicates that a key component of binge eating (i.e., experiencing a loss of control over eating) is significantly associated with maladaptive HRV in a sample without comorbid MDD. However, due to a lack of a normal-weight control group, it is difficult to know how these findings may have been influenced by BMI. Overall, the authors concluded that loss of control eating may involve physiological mechanisms that are in need of further research as findings may increase understanding of etiological and maintenance variables of disordered eating (Ranzenhofer et al., 2016).

It remains unclear if the association between BED and cardiovascular function in obese BED groups applies to non-obese individuals who binge eat. Investigating normal and overweight individuals with recurrent binge eating for impaired HRV could have a significant impact on our general understanding of binge eating behaviors, and influence treatment recruitment as health risks may not only be associated with weight status. Initial research examining the impact of 16-sessions of CBT for individuals with depression found significant short-term improvements in HRV post treatment (Carney et al., 2000), indicating that psychological interventions are able to impact this important physiological variable. Additionally, following six months of behavioral weight loss and BED-specific CBT, researchers found that obese individuals with BED reported significant improvements in binge eating symptomatology and exhibited improvements in cardiovascular disease risk factors (e.g., lipid

profiles; Grilo, 2015), suggesting that therapeutic BED intervention may positively impact cardiovascular functioning. To date, HRV in normal to overweight individuals reporting binge eating symptomology has not been examined. This represents a significant a gap in our understanding, particularly given the majority of individuals who binge eat may not be obese, and that increased cardiac risk may not be linked solely to weight status. Clearly the role of HRV in non-obese individuals who engage in binge eating is an area in need of investigation.

Maintenance of Binge Eating

Binge eating disorder is characterized as having low remission rates and a high prevalence of relapse following treatment (APA, 2013; Bello & Hajnal, 2010). Due to the chronic course of BED, several factors have been investigated related to the maintenance of binge eating behaviors. The association between binge eating symptomatology and negative mental and physical health has prompted increased interest to better understand, treat, and prevent these symptoms. Several theoretical models originally proposed to account for maladaptive eating patterns (i.e., bulimia nervosa, obesity) have been modified to capture hypothesized factors that may trigger and maintain binge eating (e.g., le Grange, Gorin, Catley, & Stone, 2001). Special attention has been given to variables that may precede binge eating episodes, and cognitive models have focused specifically on affective antecedents that may be predictive of binge behaviors (e.g., Haedt-Matt & Keel, 2011). Through both retrospective and real-time assessment, individuals have consistently reported that binge eating episodes are frequently preceded by stress and/or negative affective states (Aubie & Jarry, 2009). Additionally, there has been ample empirical support for the role of stress and negative affect as proximal triggers for binge eating (e.g., Greeno, Wing & Shiffman, 2000; Hilbert & Tuschen-

Caffier, 2007; Laessle & Schulz, 2009). Thus, several theoretical models have been proposed that emphasize stress and negative affective states in the maintenance of binge eating.

Theoretical Models

Two conceptual models of binge eating have dominated much of the literature to date: the affect regulation model and the escape from awareness model (Aubie & Jarry, 2009). Although distinct, it is noteworthy that both of these theoretical frameworks posit that binge eating is maintained through attempts to avoid negative affective states. In affect regulation models, it is proposed that increases in negative emotions trigger binge eating episodes. Using this framework, binge eating serves the function of reducing negative affect by using food as both a source of comfort and distraction. Binge eating is suspected to reduce negative mood states, and therefore, is hypothesized to be maintained through negative reinforcement (Haedt-Matt & Keel, 2011). One example of an affect regulation model of binge eating is the Trade-Off Hypothesis. According to this model, binging allows individuals to reduce intolerable negative emotions (e.g., sadness) by shifting focus to less aversive negative states (e.g., guilt following a binge; Kenardy et al., 1996). Although empirical support for a reduction in negative emotions following a binge is largely mixed (e.g., Haedt-Matt & Keel, 2011), there is ample evidence that negative affect may indeed be a proximal antecedent to binge eating. Researchers have found similar results despite varied methodology, including open-ended questions regarding reasons for binge behavior (e.g., Lynch, Everingham, Dubitizky, Hartman, & Kasser, 2000), self-report questionnaires assessing variables preceding binge eating (e.g., Davis & Jamieson, 2005), real-time self-report assessment (e.g., Deaver, Miltenberger, Smyth & Meidinger, 2003), and laboratory based procedures aimed at inducing transient negative mood prior to meal exposure (e.g., Chua et al., 2004).

The escape from awareness model has been also been influential in binge eating theory and research (Aubie & Jarry, 2009). Affect regulation models target negative affect more broadly, but the escape from awareness framework focuses on negative affect resulting from aversive self-awareness, or an emotional experience precipitated by awareness of one's failure to reach a valued standard (Heatherton & Baumeister, 1991). In this model, individuals who binge eat are characterized as having unrealistically high demands and expectations for themselves (including weight and shape expectations), a desire to be perceived favorably by others, and report high levels of self-awareness. When unachievable expectations are not met, this results in emotional distress, fear of negative evaluation from others, and increased negative self-awareness. To cope with this negative emotional state, individuals narrow their attention on immediate stimuli in the environment, and may use food and overeating as a method to distract themselves. Perhaps not as widely studied as affect regulation models, research supporting the escape from awareness theory has found that individuals who binge eat have higher negative self-awareness than non-binge eaters as assessed through self-report questionnaires (Paxton & Diggens, 1997), in addition to reporting more avoidance coping strategies (Schwarze, Oliver, & Handal, 2003). More recently, a study that evaluated research findings using both an affect regulation model and the escape from awareness model found that when binge eaters and non-binge eaters were exposed to vignettes of weight-related teasing, both groups reported increased negative affect, but only binge eating individuals proceeded to eat significantly more (Aubie & Jarry, 2009). Based on these findings, the authors suggested that the escape from awareness model may best illustrate the observed group differences, suggesting that weight-related teasing may have had a stronger impact on the binge eating group due to the importance and higher

personal expectations of weight and shape for these individuals, a key component of the escape from awareness model.

A relatively new maintenance model of binge eating with similar focus on negative affect has recently been introduced. The emotional cascade theory suggests that individuals with BED routinely ruminate, or repetitively think about causes and consequences of negative emotional experiences in an attempt to manage them (Selby, Anestis, Joiner, 2008). According to this model, repetitive thinking results in a cycle that increases negative affect, which then leads to more rumination. To break this sequence, it is hypothesized that individuals engage in behaviorally impulsive actions, such as binge eating, to narrow focus on external stimuli instead of unpleasant emotional states. Cross-sectional and longitudinal study designs investigating the relation between rumination and binge eating have suggested that there is a significant association between rumination and dysregulated behaviors (i.e., binge eating), and that rumination may proceed binge eating behaviors (Selby et al., 2008). Additionally, survey research suggests that those reporting higher trait rumination also report greater binge eating symptomatology (Harrell & Jackson, 2008), and that rumination and binge eating may have a reciprocal connection, where rumination is predictive of increased binge eating, and vice versa (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007).

Despite numerous maintenance models of binge eating, it is noteworthy that the three most frequently cited frameworks each include negative emotional states as a key trigger for binge behaviors. In their 2015 literature review of experimental studies utilizing an array of cognitive emotion regulation models of binge eating, Leehr and colleagues concluded there was evidence that stress and negative affect are triggers for binge eating in BED groups, but not

obese non-BED groups. Given findings such as these, it is of little surprise that the majority of BED research has focused on stress and negative emotions as triggers for binge eating behavior.

Binge Eating Research

Stress. One of the most widely studied variables hypothesized to impact binge eating is stress. Stress has historically been viewed as a multi-dimensional construct that is composed of daily hassles (e.g., misplacing keys) as well as major life events (e.g., divorce), which both have been found to be significantly associated with binge eating (Degortes et al., 2014; Woods, Racine & Klump, 2010). For example, Woods and colleagues (2010) examined the relation between binge eating, dietary restraint, major stressors, and daily hassles, and found that both major and minor stressors accounted for a significant proportion of the variance observed in binge eating behavior. Utilizing self-report questionnaires in a sample of 497 undergraduate females, the researchers found that restraint-binge eating associations were strongest when daily hassles occurred during significant life events. The authors concluded that the effects of these different forms of stress may be multiplicative rather than additive. Findings such as these illustrate the important role different forms of stress may play in the maintenance of binge eating.

Although measures of objective stress are useful due to easy administration and minimized risk of subjective biases by limiting questions to a dichotomous format (e.g., Have you been divorced? Yes/No), these types of questions do not account for the interaction of person and environment during stressful situations. When faced with a stressor, cognitive appraisals of a situation will impact how it is viewed (e.g., threatening or demanding), directly influencing how stressful circumstances are perceived (Lazarus & Folkman, 1984). Additional appraisals of ability to cope and availability of resources to handle a situation will also impact how stressors are perceived and experienced (Cohen, Kamarck & Mermelstein, 1983).

Therefore, in addition to assessing daily hassles and major life events, perceived stress, which incorporates an individual's cognitive and emotional response to the environment (Harney, Fitzsimmons-Craft, Maldonado, & Bardone-Cone, 2014), is an important factor to consider in empirical studies of binge eating.

In addition to self-report questionnaires requiring retrospective recall of stress severity and food intake, stress has also been investigated in laboratory studies examining potential changes in food intake in those with and without binge eating symptoms. Common stress induction methods (e.g., social evaluative tasks, challenging mental tasks) have demonstrated that when stressed, individuals with obesity who endorse binge eating symptoms consume significantly more calories, eat significantly faster, and change food preferences from healthy to less healthy options than obese individuals who report no binge eating symptoms (Laessle & Schultz, 2009; Zellner et al., 2006). In addition to these eating tasks, laboratory induced stress studies without food exposure have found that obese individuals with clinical and subclinical binge eating symptoms self-report greater hunger and desire to binge eat following stress inductions (e.g., cold pressor test, cognitive challenge, speech task; Cattanach, Malley & Rodin, 1988; Gluck et al., 2004). Complimenting these lab findings, in vivo monitoring of food intake and stress through daily diary methods have found that individuals who binge eat are more likely to perceive situations as stressful and report a lower tolerance for stress independent of depressed mood when compared to those who do not binge eat (Freeman & Gil, 2004; Wolff, Crosby, Roberts, & Wittrocks, 2000).

Research with a greater focus on perceived stress via self-report questionnaires has indicated that higher ratings of perceived stress are associated with a greater loss of control when eating (Groesz et al., 2012). Researchers comparing self-reported binge eating female college

students to non-binge eating students over a 21-day period found that daily stressors were significantly higher in the binge eating group. Perhaps more interesting was the finding that stress experienced on binge days was perceived to be much greater than stress on non-binge days, despite no significant differences in the number of stressful events each day (Wolff et al., 2000). This result is similar to the results of a later study of 62 obese women meeting diagnostic criteria for BED via a semi-structured clinical interview (Pendleton et al., 2001). Assessment of stress over a 16-month treatment period revealed that high perceived stress was associated with three times greater binge eating frequency than low perceived stress. However, given the correlational design of this study, the authors were unable to determine if greater perceived stress led to increases in binge eating, or if increases in binge behavior preceded increased perceived stress.

In an attempt to further investigate the temporal association between perceived stress and binge eating, Striegel-Moore and colleagues (2007) examined childhood risk factors for binge-eating disorders (e.g., perceived stress, eating-related concerns, family cohesion) collected as part of a 10-year longitudinal National Heart, Lung, and Blood Institute Growth and Health Study. Ten years following childhood self-reports, participants completed follow-up diagnostic clinical interviews to identify current or past symptoms of various psychological and medical conditions, including eating disorders. Using this data, researchers determined that participants who reported elevated perceived stress prior to 14 years of age were at significantly greater risk for developing binge eating behaviors consistent with BED, bulimia, and subclinical binge eating. Although the causal association between perceived stress and binge eating severity remains in question, this study is the first to suggest elevated levels of perceived stress may precede the onset of binge eating symptomatology (Striegel-Moore et al., 2007).

Building upon these previous stress findings, subsequent research sought to further investigate individual differences in stress perceptions in those with current and remitted eating disorders (Harney et al., 2014). In a clinical sample comprising three groups [i.e., individuals currently meeting diagnostic criteria for an eating disorder, partly recovered individuals with a remittance of behavioral symptoms but remaining psychological symptoms of disordered eating (e.g., elevated weight and shape concern), and individuals who were fully recovered and not reporting symptoms consistent with disordered eating], findings revealed that when compared to a control group (i.e., a community sample with no history of disordered eating), individuals in the remitted eating disorder group reported similar levels of perceived stress. However, individuals in the partially remitted and active eating disorder groups reported statistically similar ratings of perceived stress that were significantly greater than individuals in the control and remitted eating disorder groups. Given these findings, researchers hypothesized that continued elevated perceptions of stress may remain after the remission of eating disorder behaviors (as observed in the partially remitted group), and that this variable may increase the risk of treatment failure or future relapse.

Although there is increasing empirical support for the significant role stress plays in maintaining binge behavior, this research base is not without mixed findings. For example, researchers using a laboratory stress task asked females with and without binge eating symptoms to deliver a video-recorded speech about their negative qualities while being evaluated. According to the authors, there were no significant differences in the amount of food consumed between groups following this task (Levine & Marcus, 1997). Additionally, there is preliminary evidence that stress does not trigger binge eating across racial groups. Researchers have found that self-reported perceptions of stress are predictive of binge eating severity in European

American women, but not in African American women (Harrington et al., 2006; Goetze et al., in preparation, 2018a). Similarly, researchers examining correlates of binge eating, race, and weight among college-aged female students found that African American women retrospectively reported less stress than European American women, independent of the severity of reported binge behavior (Napolitano & Himes, 2011). According to authors, self-reported perceived stress may not be a universal trigger for binge eating. Mixed findings such as these have led some researchers to suggest that in addition to self-report measures of different forms of stress, physiological methods aimed at detecting differences in stress reactivity (e.g., HRV as a method for evaluating autonomic flexibility) are increasingly important (e.g., Gluck, 2006). Including these additional variables may lead to a more sophisticated understanding of the complex and interacting mechanisms that work to maintain binge behaviors.

Negative affect. In addition to stress, negative affect is frequently cited as an antecedent to binge eating (e.g., Wolff et al., 2000). Briefly stated, negative affect has been described as subjective distress and mood states that include anger, contempt, disgust, guilt, fear, and nervousness (Watson, Clark, & Tellegen, 1988). However, it is important to note that whereas high positive affect indicates high energy, pleasurable engagement, and complete concentration, low positive affect is characterized by sadness and lethargy. Therefore, when studying the impact of negative affect on binge eating, it may be equally important to assess low positive affect to capture sad mood and fatigue. For example, Munsch and colleauges (2012) found that reductions in positive affect frequently preceded binge eating episodes over a 7-day period in women with BED. However, much of the binge eating and mood literature focuses primarily on the role of negative affect.

Self-reports from individuals with BED and subclinical binge eating suggest that those who binge have a significantly greater tendency to eat in response to negative mood states than those who do not engage in binge eating behaviors (Deaver et al., 2003; Eldredge & Agras, 1996; Henderson & Huon, 2002; Stice, Akutagawa, Gaggar, & Agras, 2000). Similarly, a large community questionnaire-based study investigating behavioral risk factors of binge eating found that negative affect was significantly associated with binge eating behaviors in both men and women (Womble et al., 2001). In keeping with these results, a study of 147 female undergraduates found that rumination, avoidance of negative emotions, and increased stress predicted binge eating (Sulkowski, Dempsey, & Dempsey, 2011). Similar findings were observed in a large sample of 695 undergraduate students in which poor emotion identification and low ability to modulate negative moods accounted for significantly more variance in binge eating presentation and severity than sex, dietary restraint, and over-evaluation of weight and shape (Whiteside et al., 2007). Such findings are consistent with previous semi-structured interview-based results indicating that negative mood is a frequently reported antecedent to binge behaviors (Arnow, Kenardy, & Agras, 1992).

Although self-report is commonly used to assess emotional states, it is dependent on retrospective recall. Ecological momentary assessment (EMA) does not have this confound as individuals record target behaviors and variables in vivo, as they are occurring. EMA has been a frequently utilized tool for investigating affect and binge eating, and using this methodology, it has consistently been observed that increases in negative affect frequently precede binge eating episodes in overweight and obese women meeting diagnostic criteria for BED (Greeno, Wing, & Shiffman, 2000; Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007). Additionally, results from a 7-day EMA study with obese women with and without BED (determined by semi-structured

clinical interview) indicated that loss of control over eating was associated with negative affect independent of the amount of food consumed in the BED group (Goldschmidt et al., 2012). Given the suggestions that negative mood may be an antecedent of both objective and subjective binge eating, some researchers have argued that the link between negative emotions and binge eating is not a question of *if* negative emotions trigger binge behaviors, but *how* negative affect leads to binge eating (Dingemans et al., 2015; Tice, Bratslavsky, & Baumeister, 2001).

Similar to stress research, negative affect and binge eating has been investigated in laboratory settings to minimize reliance on less-objective measures of food consumption. The first laboratory study to investigate the impact of negative mood induction on women with and without BED utilized a 15-minute audio-guided autobiographical recall of either a negative or neutral life event prior to presenting participants with a multi-item food buffet (Telch & Agras, 1996). Findings revealed negative mood was significantly correlated with a greater sense of lack of control over eating, and higher subjective reports of binge eating regardless of the amount of food consumed in the BED group. These results indicate that both objective and subjective binge eating episodes may be correlated with negative affect. In this study, participants were asked to report their emotions following mood induction via an adjective checklist of different affective states (i.e., Multiple Affect Adjective Checklist). Participants with BED who labeled their eating as a binge reported significantly more depression-related emotions (e.g., gloomy, unhappy, blue) than participants with BED who described their laboratory consumption as overeating. However, the BED group's reports of high perceived loss of control were not better explained by depression-related adjective ratings. An important limitation of this study is that participants were not formally assessed for MDD. Although this first laboratory study did not observe the anticipated objective increase in amount of food eaten by those with binge eating

symptoms, later negative mood induction and food studies did support this hypothesis (e.g., Svaldi et al., 2014). A 2003 literature review of published laboratory studies of BED concluded that these individuals eat significantly more than weight-matched participants without binge eating behaviors when asked to eat until very full, or following conditions designed to increase the likelihood of inducing a binge episode (e.g., negative mood induction; Walsh & Boudreau, 2003). More recently, negative mood induced by written vignettes describing weight-related teasing (Aubie & Jarry, 2009), and film clips depicting sad scenes (Chua et al., 2004; Dingemans et al., 2009; Svaldi et al., 2014) have resulted in those with binge eating and induced negative mood to eat significantly more than individuals with neutral mood or non-binge eating comparison groups.

It has been suggested that distinguishing features of depression are both state and trait low positive affect and high negative affect (Watson et al., 1988). Due to this, several studies have explored the relation between depression, state negative affect, and binge eating. An early study of negative mood induction in obese women with BED (determined by clinical interview) found no significant associations between self-reported depressive symptoms, negative affect, and binge eating (Agras & Telch, 1998). Findings revealed that greater negative mood in response to guided autobiographical negative mood induction increased reports of loss of control and subjective binge eating. This finding led authors to hypothesize that acute, state negative affect rather than stable, trait negative mood (i.e., depression) may lead to binge eating. However, a later study of 66 overweight and obese women with clinical and subclinical binge eating (determined by structured clinical interview) found that following negative mood induction, individuals with higher baseline trait depressive symptoms experienced greater sadness and ate significantly more calories during a subsequent taste-task (Dingemans et al.,

2009). This finding was later replicated in a negative mood induction study with 75 overweight and obese women diagnosed with BED. This result suggests there may be a significant interaction between both state and trait features of negative mood in the maintenance of binge eating behaviors (Dingemans et al., 2015). In general, these findings indicate that baseline levels of depression should be considered in mood induction investigations as they may impact state negative affect and associated symptoms of binge eating.

The binge eating and negative affect literature is not without mixed findings. Although Wegner and colleagues (2002) observed that negative mood was greater overall on binge days in their sample of college women with subclinical binge eating symptoms, 2-week EMA results indicated that increased transient negative affect did not correlate with discrete binge eating episodes. Similarly, in a later laboratory study utilizing guided imagery to induce negative or neutral mood in overweight and obese women with BED, there was no significant difference between groups in amount of food consumed in a subsequent taste-task (Munsch, Michael, Biedert, Meyer, & Margraf, 2008). These results were unexpected as there were significant increases in negative affect in those with binge eating symptoms. Despite these mixed findings which may be partly attributable to methodological differences, a recent meta-analysis investigating the impact of negative mood induction on eating behaviors found that overall, research supports a causal relation between negative mood and greater food intake (Cardi, Leppanen, & Treasure, 2015). Results such as this indicate that negative mood may be an important variable to target in future maintenance research.

Stress and negative affect. Interestingly, little has been done to investigate the impact that both stress and negative emotions may concurrently have on binge eating despite research suggesting that perceived stress is positively associated with negative affect (Watson, 1988).

Smyth and colleagues (2007) utilized EMA to record stress, negative affect, and binge episodes in a sample of women diagnosed with bulimia nervosa (determined by semi-structured clinical interview). Findings indicated that both stress and negative mood were significantly elevated on binge days compared to non-binge days, and significantly higher leading up to a binge eating episode. This preliminary finding suggests a possible association between stress, negative affect, and binge behavior, highlighting the importance of further investigation.

Generalizability. Increased research attention has led to a greater understanding of the antecedents of binge eating behavior, however, this body of work is limited by methodological flaws and generalizability issues. A quick survey of the research literature indicates a pronounced underrepresentation of men (Grucza et al., 2007) as well as a lack of diverse racial and ethnic groups (Marques et al., 2011; Reagan & Hersch, 2005). In fact, studies of binge eating have often restricted recruitment to women meeting diagnostic criteria for BED, despite research indicating that individuals with subclinical binge behaviors report similar rates of functional impairment, emotional distress (Grucza et al., 2007; Mond et al., 2010; Stice et al., 2009), and reduced health-related quality of life (Latner et al., 2008). Similarly, research samples are often selected from obese, treatment seeking individuals despite evidence to suggest that more than half of those meeting criteria for BED are not obese (Carrard et al., 2012; Corwin et al., 2011) and treatment seeking populations may differ from non-treatment seeking individuals (Grilo et al., 2005). Typically binge eating research does not include normal-weight control groups, making it difficult to interpret significant findings (Svaldi, Tuschen-Caffier, Trentowska & Naumann, 2014). In spite of these limitations in the generalizability of findings, there is an expanding body of literature that suggests that both stress and negative affect significantly influence binge eating episodes and may play a significant role in the maintenance

of binge behaviors. However, it is likely that these inconsistent findings suggest that additional constructs may be impacting the effect stress and negative affective states have on binge eating.

Perceived Control

Perceptions of control have been identified as robust predictors of physical health, mental wellbeing, and positive outcomes across the lifespan (Skinner, 1996). Skinner (1996) proposed a four-part framework to be used to classify various forms of control. Using this method, the term *perceived control* should be utilized when describing subjective perceptions of control that are prospective, global in nature, and involve the self as the agent of control (Roepke & Grant, 2011). Although this construct may also reflect beliefs about controllability of the surrounding environment, it does not speak to beliefs regarding competence. This is an important distinction given that the level of success or failure an individual anticipates (i.e., self-efficacy) has been found to be a distinct construct that may be more predictive of behavioral intentions (e.g., motivation to engage in a behavior) whereas perceived control may be a significant predictor of actual behavior change (Armitage & Conner, 1999; Terry & O'Leary, 1995; White, Terry, & Hogg, 1994).

Perceived control, or one's perception of their ability to impact his or her behavior and environment to reach desired goals, has been identified as a psychological factor that may be associated with reduced risk for cardiovascular diseases (Roepke & Grant, 2011) and increased adherence to preventative health behaviors (McCaul, Sandgren, O'Neill, & Hinsz, 1993). In addition, perceived control may buffer individuals from stress exposure (Bollini et al., 2004; Wallston et al., 1987) and be associated with better overall physical health (Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000). Although perceived control has been largely ignored in binge eating research, studies investigating common comorbid conditions, such as cardiovascular

disease and depression, have included this variable. Results from these studies offer initial evidence that perceived control may not only be linked to the expression of these associated conditions but may positively impact health and mental wellbeing.

Medical

Although no published research has directly explored potential links between perceived control and HRV in a binge eating sample, perceived control has been cited as a significant factor associated with reduced risk for cardiovascular disease. Hypothesized to attenuate the physiological impact of stress, it has been demonstrated that perceived control has an inverse connection with cardiac disease and related death (Roepke & Grant, 2011). For example, in a prospective population-based study of over 19,000 men and women, aged 41 to 80 with no previous heart disease, it was found that low self-reported perceived control at initial assessment was associated with increased risk of cardiovascular disease related mortality over longitudinal follow-up (Surtees et al., 2010). This association remained significant after controlling for biological (e.g., diabetes), lifestyle (e.g., BMI), socioeconomic (e.g., social class), and psychosocial risk factors (e.g., MDD episode within the past year), such that there was over a 10% increased risk of cardiovascular disease mortality with each standard deviation decrease in baseline self-reports of perceived control. Startlingly, those reporting the bottom quintile of perceived control were 60% more likely to have died from cardiovascular disease, leading the authors to urge future researchers to continue investigations into psychological variables that may be linked to heart disease and mortality.

Further illustrating the potential protective role perceived control may play in cardiac wellness, research investigating perceived control in patients recovering from myocardial infarct, bypass surgery, or a history of cardiovascular disease has found that those with higher self-

reported perceptions of control demonstrate significantly lower psychological distress (e.g., depression, anxiety, hostility), better adaptive functioning (e.g., 6-minute walk test), and improved overall recovery 6-months following surgical intervention when compared to those with lower perceived control (Dracup et al., 2003; Moser & Dracup, 1995). In addition, Roepke and Grant's (2011) literature review of 32 empirical investigations of perceived control and cardiometabolic health concluded that there was ample evidence that higher self-reported perceived control is associated with better cardiovascular outcomes and may play a protective role in health. Due to this evidence base, the authors suggested that future investigators should instead focus on clarifying the mediators and moderators most relevant to the association between perceived control and later disease. In fact, whereas some studies have conceptualized perceived control as a mediator or moderator of the association between stress and cardiovascular function, there has been a lack of investigation into variables that may impact the relations between perceived control and health status (Roepke & Grant, 2011). One such area that requires this attention is the potential links between perceived control, binge eating severity, and possible associated deficits in HRV.

Psychiatric

Low perceived control is also associated with psychiatric symptoms such as anxiety and depression (Chaney et al., 1996; Langer, 1975; Rivard & Cappeliez, 2007; Taylor & Brown, 1988), and has been found to mediate relations between stress and psychological distress (Rosenbaum et al., 2012). For example, in a recent meta-analysis of 51 investigations of perceived control and anxiety disorders, findings suggested that greater deficits in perceived control were associated with more severe symptoms across anxiety disorders (Gallagher, Bentley, & Barlow, 2014). However, the psychiatric literature focusing on perceived control

most valuable to future investigations of binge eating may be the growing body of evidence suggesting that perceived control is a modifiable treatment target.

Perceived control is not conceptualized as a fixed personality trait, but rather an adaptive variable that can be modified through experience (Surtees et al., 2010), a claim supported by findings from empirical investigations of treatment for depression and anxiety. In a recent study examining the impact of eight weeks of CBT targeting depression following cardiac surgery, self-report measures pre- and post-intervention revealed that treatment was associated with increased perceived control (Doering et al., 2015). Self-report measures also indicated that treatment was related to reduced depressive symptomology, pain interference, and pain severity when compared to cardiac surgery patients receiving care as usual and after controlling for baseline differences in weight and sex between groups. Treatment trials for depressive and anxiety symptoms via cognitive-based online intervention have illustrated a similar relation between symptom change and perceived control (van der Zanden, Galindo-Garre, Curie, Kramer & Cuijpers, 2014). A recent 2-year longitudinal investigation of CBT's impact on anxiety symptoms and perceived control indicated that the adaptive gains in perception of control through treatment were significantly associated with recovery from anxiety disorders (Gallagher, Naragon-Gainey, & Brown, 2014). In fact, researchers hypothesized that perceived control may be a transdiagnostic mechanism of change in anxiety disorders, and predictive of positive outcomes and behavior change following CBT. Although further study is required to determine what elements of CBT are associated with positive changes in perceived control (Doering et al., 2015), these investigations suggest that perceived control is not only associated with common comorbid psychiatric conditions of binge eating but is amenable to change through therapeutic intervention.

Binge Eating

It has been found that loss of control over eating is not only a diagnostic variable of BED, but is also associated with greater binge eating severity, higher depressive symptoms, greater dissatisfaction with appearance, and poorer overall mental health in both clinical and subclinical binge eaters (Colles, Dixon & O'Brien, 2008). Despite the role control over eating plays in BED, little has been done to investigate how overall perceived control may be related to binge eating severity. A 2001 literature review found that individuals with anorexia or bulimia self-report less perceived control over events in the world than individuals without eating pathology (Dalgleish et al., 2001). Similarly, research has found that individuals with anorexia or bulimia self-report lower perceptions of control over feelings and events than those without eating disorder diagnoses (Sassaroli, Gallucci, & Ruggiero, 2008). Findings such as these have led some researchers to hypothesize that perceived control may have important explanatory power for understanding eating disorders and may serve as a possible target for therapeutic interventions.

To date, several studies have investigated perceived control among individuals reporting binge eating symptoms. In a recent study utilizing self-reports of eating disorder severity and perceptions of control in a community sample of 175 adults, perceived control was negatively correlated with symptoms indicative of anorexia, bulimia, and BED (Froreich, Vartanian, Grisham, & Touyz, 2016). Results also revealed that alternate forms of control (i.e., lack of control over one's life and fear of losing self-control) were most strongly associated with the variance in eating disorder severity scores, suggesting an attenuated role of perceived control in binge eating severity. However, the generalizability of these findings were limited by the small sample size of individuals reporting diagnostic binge eating symptoms assessed by self-report

questionnaire (n = 14). Furthermore, a study investigating self-reported control in individuals with anorexia, bulimia, and BED as determined by a structured clinical interview found significant differences in reported control deficits (Tomba, Offidani, Tecuta, Schumann, & Ballardini, 2014). For example, while individuals with BED reported significantly lower levels of environmental control (i.e., a sense of control over external activities/environment, and effective use of surroundings) than healthy controls, those with anorexia described similar ratings as individuals in the control group. These findings caution against including all individuals who report eating pathology together as this may mask important disorder-specific group differences in level of perceived control.

To date, one published experimental study has provided preliminary evidence suggesting that thoughts of control may significantly impact eating behaviors. Vartanian, Kernan, and Wansink (2016) investigated the impact of a disorganized environment and autobiographical recall of a time in life when one felt out of control on subsequent eating behavior. Ninety-eight female undergraduate students were placed in either a chaotic, disorganized kitchen or a tidy kitchen environment. In each condition, participants were either asked to write about a time when they felt out of control, or to describe the last lecture they attended as a neutral condition prior to completing a taste-task. It was found that those in the chaotic kitchen with the out-of-control writing task ate significantly more cookies than those in all other conditions, suggesting that one's mind-set in environments may either trigger or buffer against behavioral vulnerability to binge eat. Researchers suggested that their preliminary findings supported the significant impact both environment and mind-set can have on food intake, and that further research is needed to explore the influence thoughts of control may have on eating related behavior. Although not with a binge eating sample or directly assessing the construct of perceived control,

this preliminary study suggests there may be an association between cognitive perceptions of control and binge eating behaviors.

Goetze, Huff, Saslow, Epel, and McCoy (in preparation, 2018a) investigated the relation between self-reported perceived control, perceived stress, and binge eating severity in a diverse community sample of 575 women. The researchers found that perceived control significantly predicted binge eating severity, with those having higher perceived control reporting less binge behavior. Additionally, mediated by perceived stress, higher perceived control predicted lower perceived stress, leading to lower binge eating severity. Interestingly, but in keeping with previous race-related findings (Harrington et al., 2006; Napolitano & Himes, 2011), this model was significant for European American, Latina American, and Asian American women, but not for African American women. Although perceived control was a significant predictor of binge eating severity, perceived stress did not mediate this connection in African American women, even when controlling for group differences in BMI and SES. Researchers suggested that perceived stress may not be a reliable trigger for binge eating across racial groups and hypothesized that perceived control may be a more universal predictor of binge eating severity than perceived stress.

A follow up study was conducted to examine the mediating role of both perceived stress and negative affect in the association between perceived control and binge eating severity (Goetze et al., in preparation, 2018b). As expected, higher perceived control continued to be predictive of lower binge eating severity in part due to the mediating effects of perceived stress and negative mood. Additionally, analyses suggested that greater reports of perceived stress were associated with increases in negative mood, and that this relation also significantly mediated the impact of perceived control on binge eating severity. It is noteworthy that in both

of these investigations, perceived stress and negative affect did not prove to be better predictors of binge eating severity with perceived control as a mediating variable. In fact, when tested, these alternative models were not statistically significant. Overall, the authors suggested several mechanisms through which perceived control is a protective variable against binge eating symptomatology, and therefore, a psychological factor requiring future investigation.

Overview and Statement Purpose

Binge eating, the uncontrollable consumption of larger amounts of food than most would eat under similar contexts and discrete time periods, is a key symptom in clinical eating disordered samples (APA, 2013; Aubie & Jarry, 2009) and subclinical binge eating populations (Hudson et al., 2007). Both clinical and subthreshold binge eating is associated with elevated rates of functional impairment, psychiatric and medical comorbidity, and emotional distress (Didie & Fitzgibbon, 2005; Hudson et a., 2007; Stice et al., 2009). Although binge eating appears to be more equivalent across sex and race than other eating disorders (APA, 2013), research suggests that binge eating may occur most often in women under the age of 40 (Reagan & Hersch, 2005). Additionally, while the prevalence of BED increases with increased BMI, obesity is not a diagnostic feature of this disorder (APA, 2013). Only approximately 35% of those who regularly engage in binge eating fall into the obese weight range (Corwin et al., 2011), yet historically, research has been mostly limited to obese, female samples meeting BED diagnostic criteria. This narrow focus negatively impacts the generalizability of findings and highlights a significant gap in our understanding of binge eating among non-obese individuals (Carrard et al., 2012; Fairburn et al., 2000).

A growing area of investigation has focused on the role of binge eating in increased cardiovascular health and disease risk. Research findings suggest that binge eating is

significantly associated with cardiovascular problems, including coronary heart disease, heart failure, and hypertension (Mitchell, 2016; Sheehan & Herman, 2015). Although preliminary, research focusing on cardiac wellness has demonstrated that women with BED may be at greater risk for cardiovascular disease independent of obese weight status, with significantly blunted HRV (Friederich et al., 2006; Ranzenhofer et al., 2016). However, such investigations have depended on obese samples, which have previously been identified as exhibiting less adaptive HRV both at rest and under mental challenge (Karason et al., 1999; Laederach-Hofmann et al., 2000), potentially confounding obese binge eating findings. Additionally, physiological studies have neglected to consistently consider the potential impact depressive symptoms may have on HRV. As the most prevalent comorbid lifetime diagnosis for individuals with BED (Dingemans et al., 2009), MDD has also been found to reduce cardiovascular reactivity (Kemp et al., 2010), further complicating HRV findings related to obese individuals with binge eating behaviors. At this time, there has been no investigation into HRV in normal to overweight individuals reporting binge eating symptomology. This represents a significant gap in the current literature, given that the majority of individuals who binge eat may not be overweight, and that increased cardiac risk may not be linked solely to weight status. Research with careful consideration of potential confounding variables such as depressive symptoms is needed.

Theoretical models and subsequent research into the maintenance of binge behaviors have focused primarily on the impact of stress and negative affect. Although both variables have proved to precede binge behaviors in both clinical (Munsch et al., 2012; Pendleton et al., 2001) and community populations (Wolff et al., 2000; Womble et al., 2001), research indicates that these antecedents may not consistently trigger binge eating (Harrington et al.,2006; Levine & Marcus, 1997; Munsch et al., 2008). However, preliminary findings suggest that perceived

control may not only predict binge eating severity in diverse populations (Goetze et al., in preparation, 2018a), but may be protective in part due to buffering against commonly cited antecedents to binge eating behavior; both stress and negative affect (Goetze et al., in preparation, 2018b). Although higher perceived control has been linked to decreased cardiovascular disease and mortality (Surtees et al., 2010), less severe anxiety and depressive symptoms (Gallagher et al. 2014) and is amenable to therapeutic intervention (van der Zanden et al. 2014), there has been a lack of research on variables that may impact the relation between perceived control and health status (Roepke & Grant, 2011).

The central aim of this study is to investigate the predictive role of perceived control and binge eating severity on mood reactivity, and possible concomitants with reduced cardiovascular function as measured by HF-HRV/RSA. This study will advance the existing literature in several important ways. First, this investigation will focus on a sample including both normal and overweight individuals reporting a range of binge eating severity, but not necessarily diagnostic-level symptoms. Research on binge eating has traditionally focused on obese BED samples, and therefore may not generalize to a significant proportion of individuals with binge eating behaviors and related distress. Second, this study will contribute to a growing body of evidence suggesting that binge eating may be related to increased risk of cardiovascular dysfunction. Past research suggests a connection between BED and HRV that is not explained by obesity; however, it remains unclear whether this association is also found in non-obese individuals with BED. The current proposal will examine whether the link between cardiovascular function in obese women with binge eating symptomatology applies to non-obese women. Third, this study will provide research examining whether perceived control is protective against negative mood reactivity in response to a negative mood induction in

individuals reporting a range of binge eating severity. Despite theoretical connections between maladaptive negative mood reactivity and increased binge eating severity (e.g., Agras & Telch, 1998; Dingemans et al., 2009), psychophysiological research with this population has been neglected to date. The design of this study is structured to test for evidence that perceived control impacts BED symptoms and significantly influences comorbid medical risks, thereby implicating it as an important treatment target.

Research Hypotheses

This study will contribute new knowledge about the relation between perceived control, binge eating severity, and cardiovascular function in the context of negative mood manipulation. Based on pilot data, a review of the existing research, and the specific manipulations being tested in the current study, the following hypotheses are proposed:

1. Perceived control will significantly predict binge eating severity with an inverse relation, such that individuals with higher levels of perceived control will report less severe binge eating symptoms. The relation between perceived control and binge eating severity will be mediated by self-reported perceived stress, depressive symptom severity, and negative affect during the previous two weeks, such that part of perceived control's relation to binge eating severity will be explained by associations with stress, mood, and affect.
2. Perceived control will significantly predict resting HF-HRV/RSA, such that individuals with higher levels of perceived control will have more adaptive (i.e., higher) resting HF-HRV/RSA. The relation will be mediated by self-reported perceived stress, depressive symptom severity, and negative affect during the

previous two weeks, such that part of perceived control's relation to resting HF-HRV/RSA will be explained by associations with stress, mood, and affect.

3. Individuals within a negative mood induction group will demonstrate significantly greater negative affect and stress reactivity than those in a neutral mood induction group.
4. Individuals within a negative mood induction group who report higher perceived control will demonstrate significantly less severe negative affect and stress reactivity than those who report lower perceived control.
5. Participants in a negative mood induction group will demonstrate significantly greater changes in HF-HRV/RSA reactivity when compared to individuals undergoing neutral mood induction.
6. Individuals with higher binge eating severity within a negative mood induction group will demonstrate significantly less adaptive (i.e., lower) HR-HRV/RSA reactivity than those reporting lower binge eating severity.
7. Perceived control will be associated with HF-HRV/RSA, such that higher levels of perceived control will be associated with more adaptive (i.e., higher) HF-HRV/RSA reactivity during a negative mood induction. The relation between perceived control and HF-HRV/RSA reactivity will be mediated by reports of negative affect and stress during a negative mood induction, such that part of perceived control's relation to HF-HRV/RSA reactivity will be explained by associations with affect and stress.
8. It is expected that the relation between negative affect and HF-HRV/RSA, and stress and HF-HRV/RSA will be significantly moderated by binge eating severity, such that

higher binge severity will be associated less adaptive (i.e., lower) HF-HRV/RSA reactivity in response to a negative mood induction.

CHAPTER TWO

METHODS AND PROCEDURES

Participant Recruitment

Participants were 217 individuals, 18 to 60 years of age, enrolled as undergraduate students at the University of Maine in Orono or residing in the surrounding communities. Participants were randomized into two mood induction groups (i.e., negative mood induction and neutral mood induction) and matched for binge eating severity such that an equal number of self-reported non-binge behaviors and moderate/severe binge eating behaviors were represented in each experimental group. Power analysis using the G*Power 3.1.2 program (Faul, Erdfelder, Lang, & Buchner, 2007) revealed that a total sample size of 107 participants would result in an 80% chance of detecting a medium effect between groups for the experimental task. A sample of 217 individuals were recruited to account for exclusionary criteria, possible equipment problems, and participants who did not complete all study procedures.

Undergraduate subject pool recruitment. Participants included undergraduate students recruited from the University of Maine Psychology Department subject pool. Participants were recruited as part of an ongoing study (Attention and Elaboration IRB 2015-09-04) in the Maine Mood Disorders Lab (MMDL). Individuals who participated in the subject pool completed screening questionnaires to determine initial eligibility for Session 1 study participation via Qualtrics (2016), an online survey software system (Appendix A). Student participants were compensated with research participation credits for time spent in the laboratory, and received up to four credits toward their subject pool research experience for completing both Sessions 1 and 2 of this investigation.

Community recruitment. Participants also included individuals recruited from the larger community around the University of Maine in Orono via email announcement boards (e.g., University of Maine Announcements listserv, which is visible to faculty and staff of the University; Appendix C), craigslist and Facebook advertisements, and flyers placed in surrounding public areas (Appendix D).[1] Interested community members contacted the MMDL and answered brief screening questions over the phone to determine preliminary eligibility for the study (Appendix E; see Footnote 1). Community members who met screening criteria were scheduled for Session 1 clinical interview and questionnaires in the MMDL located in Corbett Hall on the University of Maine campus. Community participants were paid for their time, earning $30 for Session 1 and $15 for Session 2. For community members who did not complete the entire study, payment was prorated to reflect the number of hours they participated (Appendix F).

Study Criteria

General criteria required participants be between 18 and 60 years of age. Exclusionary criteria included current symptoms consistent with MDD, drug or alcohol abuse in the past 6 months, current or past diagnoses of substance or alcohol dependence, bipolar disorder, psychotic disorder, acute suicidal ideation, or mood episodes secondary to general medical conditions. These conditions represent common exclusionary criteria in studies of binge eating due to the confounding impact such comorbidities may have on self-reported symptom severity and behaviors (e.g., Svaldi, Caffier, Blechert, & Tuschen-Caffier, 2009). Participants were also excluded if they had a self-reported history of medication-dependent diabetes, heart disease,

[1] Community listserv emails, advertisements, flyers, and phone screening questions reflected study criteria for an ongoing investigation in the MMDL (Attention and Elaboration IRB 2015-09-04). However, due to similar inclusion/exclusion criteria, community members may have also be qualified for the current investigation.

hypertension, medical conditions specific to the central nervous system, or head trauma resulting in a loss of consciousness for more than one hour. Similar exclusion criteria have been utilized in studies investigating associations between psychopathology and psychophysiological reactions to minimize the risk of confounding variables that may significantly impact physiological responding (e.g., Friederich et al., 2006; Jin et al., 2015). Medical history was screened for exclusionary criteria using a general health screen included in the subject pool initial screening questionnaires (Appendix A) or during brief phone screenings for interested community members (Appendix E). Mental health history was collected during structured clinical interviews during Session 1 to further determine study eligibility.

Experimenters

The primary author was the primary experimenter for this study. Clinical psychology graduate students and undergraduate research assistants who completed the required training for the Institutional Review Board for the Protection of Human Subjects (IRB) at the University of Maine assisted during both Session 1 and Session 2 of this investigation. Graduate students conducted diagnostic clinical interviews during Session 1. Undergraduate research assistants were trained on study procedures to assist in participant recruitment (e.g., contacting study participants, administering phone surveys), questionnaire administration, physiological equipment hookup and monitoring, data cleaning, and data entry.

Interview Measure

The Structured Clinical Interview for DSM-IV-TR-Research Version (SCID-IV-RV). The SCID-IV-RV (First, Gibbon, Spitzer, & Williams, 1995) was administered by trained graduate students in the clinical psychology doctoral program and the MMDL. The SCID-IV-RV is a semi-structured interview designed to assess current and past major DSM-IV clinical

diagnoses based on criteria outlined in the DSM-IV-TR and was utilized to assess for exclusionary criteria (e.g., psychosis), and make appropriate diagnostic designations (e.g., remitted major depressive disorder). Interviews were audio-recorded for future fidelity checks and estimating inter-rater reliability.

Questionnaire Measures

Beck Depression Inventory- II Edition (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996b; Appendix G) was used to evaluate severity of depressive symptoms which are commonly associated with binge eating and have significantly influenced physiological findings in previous research with this population (Klatzkin et al., 2015). This 21-item self-report questionnaire assesses several dimensions of depressive symptoms, such as fatigue, low mood, irritability, and loss of pleasure. Respondents rate their experience over the past two weeks on a scale from 0 to 3, with 0 indicating no presence of a symptom and 3 suggesting that a symptom is present and severe. Total scores can range from 0 to 63, with higher scores indicating greater levels of depressive symptom severity. Research on the BDI-II indicates high internal consistency (a = .91; Beck, Steer, Ball, & Ranieri, 1996a) and adequate test-retest reliability (Beck et al., 1996b) and construct validity (Dozois, Dobson, & Ahnberg, 1998). Research also has demonstrated that the BDI-II has adequate convergent validity with specific measures of depression, self-esteem, anxiety, stress, and perceived mental health (Arnau, Meagher, Norris, & Bramson, 2001; Osman et al., 1997). Scores on the BDI-II were utilized as a moderator in Hypotheses 1 and 2, and to control for significant findings related to variations in depressive symptoms instead of variables of primary interest (e.g., binge eating symptom severity) in exploratory analyses.

Binge Eating Scale (BES). The BES (Gormally, Black, Datson, & Rardin, 1982; Appendix G) was used to evaluate severity of binge eating symptoms. The BES is a widely used

measure to assess cognitive and emotional features associated with binge eating episodes (e.g., experiencing guilt following a binge) and behavioral manifestations of binge eating (e.g., consuming food to the point of physical discomfort). This 16-item self-report questionnaire is composed of 62 statements that assess the severity of binge eating symptoms. Each BES item includes four statements that increase in symptom severity and are scored on a 0 to 3 scale. Responders select one statement from each item that best describes their perceptions and beliefs regarding current eating behaviors. For example, one item ranges from "I don't feel guilt or self-hate after I overeat" to "Almost all the time I experience strong guilt or self-hate after I overeat."

The BES provides a total binge eating severity score calculated by summing the 16 participant selected statements, with total scores ranging from 0 to 46. The BES is not designed as a diagnostic screening of BED, but rather provides a measure of binge eating severity such that higher total scores indicate greater binge eating symptom severity. This measure can be interpreted continuously or categorically. When utilizing categorical scoring, three groups have been cited in the literature: non-binge or minimal binge eaters (i.e., total score less than or equal to 17), moderate binge eaters (i.e., total score between 18 and 26), and severe binge eaters (i.e., total score greater than or equal to 27; Marcus, Wing, & Hopkins, 1988; Timmerman, 1999). For this study, efforts were made to include an equal number of individuals reporting no binge eating (i.e., scores equal to or less than 17) and moderate/severe binge eating, defined as a total score of 18 or higher. This procedure has previously been utilized in research to categorize binge eating status as an independent variable (Lourenço et al., 2008).

The BES has high internal consistency (a = .85) and adequate test-retest reliability (r = .87, p = <.001; Gormally et al., 1982; Timmerman, 1999). Studies investigating the convergent validity of the BES with alternate measures of disordered eating such as the Questionnaire on

Eating and Weight Patterns (QEWP) and Eating Disorder Examination (EDE) have found only fair agreement (Kappa's ranging from .31 to .50; Brody, Walsh, & Devlin, 1994; Gladis, Wadden, Foster, Vogt, & Wingate, 1998; Greeno, Marcus, & Wing, 1995). The discordance between these measures is likely due to the intended utility of each tool. While the QEWP and EDE are designed as diagnostic measures to determine the presence or absence of BED, the BES is better suited to assess symptom severity and psychopathology associated with binge eating. The BES has also demonstrated good concurrent validity when compared to additional measures of binge eating (Celio, Wilfley, Crow, Mitchell, & Walsh, 2004; Timmerman, 1999). In the current investigation, the BES was the primary measure of binge eating severity, utilized as an outcome variable in Hypotheses 1 and 2, a predictor variable in Hypothesis 6, and a hypothesized moderator in Hypothesis 8.

General Health Screen (GHS). As part of the subject pool prescreening and community phone screening, every participant completed a health measure created for psychophysiological studies in the MMDL to assess for confounding variables that may significantly impact physiological responses (Appendix A). Participants reported their primary language, and were assessed for history of or current health conditions that were excluded from the study. This survey included 9 *Yes* or *No* questions.

Pearlin's Perceived Mastery Scale. Pearlin's Perceived Mastery Scale (Pearlin & Schooler, 1978; Appendix G) was administered to evaluate perceptions of general control. This 7-item self-report questionnaire assesses an individual's perceived ability to impact behavior and life outcomes, including items such as, "I have little control over the things that happen to me." Responses ranging from 0 (*Strongly Disagree*) to 6 (*Strongly Agree*) are summed to create an overall perceived control score, with total scores ranging from 0 to 42 after reverse scoring

appropriate items (i.e., items 6 and 7). Higher total scores indicate greater perceived control. Research on the Mastery Scale has indicated adequate internal consistency (a = .70; Turner & Noh, 1988) and good construct validity, evidenced by significant correlations with measures of optimism and depression (Marshall & Lang, 1990; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin & Schooler, 1978). This measure was used to quantify participant's perceptions of control, the primary predictor variable in Hypotheses 1, 2, 4, 7, and 8.

Perceived Stress Scale – 10 (PSS-10). Perceived stress was assessed with the PSS-10 (Cohen & Williamson, 1988; Appendix G), a 10-item self-report scale evaluating the degree to which one has perceived life as unpredictable, overloading, and uncontrollable during the previous month. The PSS-10 is an updated version of the original 14-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). Respondents rate each item using a 5-point Likert scale ranging from 0 (*Never*) to 4 (*Very Often*). A total perceived stress score is calculated by summing responses. Total scores can range from 0 to 40, with higher scores indicating greater perceived stress. The PSS-10 possesses high internal consistency (a = .89) and adequate construct validity, evidenced by significant correlations with measures of anxiety and depression, but no correlations with measures of sensation seeking, religious faith, or overt aggression (Roberti, Harrington, & Storch, 2006). The PSS-10 was used to calculate participant's ratings of perceived stress, a hypothesized mediator in Hypotheses 1 and 2.

Positive and Negative Affect Schedule – Expanded Form (PANAS-X). The PANAS-X (Watson & Clark, 1994; Appendix G) is a 60-item self-report questionnaire that assesses positive and negative affect over the past two weeks. The PANAS-X is an expanded version of the original Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) designed to measure both general dimensions of affect (i.e., negative and positive), and 11 specific affective

states (e.g., fear, sadness, fatigue, serenity, etc.). Respondents are asked to indicate how much they have experienced 60 affective adjectives on a Likert scale of 1 (V*ery Slightly or Not at All*) to 5 (*Extremely*). Scores yield a total rating for positive affect and negative affect by summing the 10 adjectives composing each of these general dimension scales. Additionally, the 11 specific affective states are calculated by summing the 3 to 6 adjectives that make up each affective state scale.

Research has demonstrated adequate internal consistency for the general dimension scales (a = .83 to .90) and specific affect state scales (a = .76 to .94). The PANAS-X has also been shown to have adequate test-retest reliability (r = .51 to .71, p = <.01) and construct validity when compared to alternate measures of short term, affective states (e.g., Profile of Mood States), illustrating that it is an appropriate measure of state affect (Watson & Clark, 1994). In this investigation, the PANAS-X was utilized to assess affective states in the two weeks prior to Session 1. This measure was selected due to evidence that binge eating severity may be linked not only to greater negative affect, but lower positive affect (Wolff et al., 2000). Therefore, although the negative general dimension was used as the hypothesized negative affect mediator in Hypotheses 1 and 2, the PANAS-X also allowed for additional exploratory analysis of different affective states that may be associated with binge eating severity (e.g., positive affect).

Visual Analog Scales for Mood (VAS). Three 100-mm visual analogue scales (Appendix H) were used to assess ratings and changes in current affect and stress pre- and post-mood induction and post-recovery. Participants were presented with a 100-mm line to rank current ratings of sadness, happiness, and stress. VAS lines were presented in a randomized order and labeled with polar statements at the 0- and 100-mm points (i.e., "*extremely*" and "*not at all*"). Use of such scales has evidence suggesting they are valid and sensitive to change in

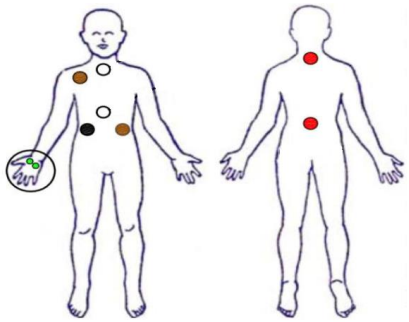
emotion and stress states when standardized measures are not obtainable because of limited time or experimental constraints (Cella & Perry, 1986; Munsch et al., 2008). Due to research suggesting that tasks requiring attention and concentration can augment and lower HRV (Hansen, Helge Johnsen, & Thayer, 2003), VAS ratings were used to assess changes in affect and stress during physiological procedures due to their efficiency. Ratings represent the negative affect and stress variables used as hypothesized outcomes in Hypothesis 4, and mediators in Hypotheses 7 and 8. Change in VAS scores also serve as a manipulation check for negative and neutral mood inductions.

Physiological Measures

Electrocardiography (ECG). Noninvasive disposable sensors were placed on participants to measure electrical activity of the heart and collect data for high-frequency heart rate variability/respiratory sinus arrhythmia (HF-HRV/RSA) calculations. MindWare Technologies Ltd. (2009) hardware and Biolab 3.1 analysis software set to collect ECG data falling within -5 and 5 volts with a sampling rate of 1000 Hz was utilized in conjunction with noninvasive self-sticking sensors located on the participants' right collarbone, bottom left rib, bottom right rib, jugular notch, and sternum (Figure 1). Participants were also equipped with noninvasive disposable sensors to collect impedance and galvanic skin response as part of an ongoing MMDL investigation and for use in calculating HF-HRV/RSA (between 0.15 – 0.4 Hz) reactivity via a Fast Fourier Transform. Therefore, additional self-sticking sensors were located on the participants' mid-back and upper-back, parallel within 1.5 inches of the jugular notch and sternum sensors, and on the heal of the non-dominant hand (Figure 1). All sensors were placed by a female graduate or research assistant, and participants were asked to sit upright in front of a

computer during physiological data collection. HF-HRV/RSA was used as the outcome variable in Hypotheses 5, 6, 7, and 8.

Figure 1. *Sensor Placement*



Note. Session 2 sensor placement for physiological data collection.

Height and weight. Participants were assessed for height, weight, waist circumference and hip circumference. Height in inches was assessed using a wall mounted height chart. Weight in pounds was measured using a non-digital scale. Waist circumference in centimeters was assessed at the level of the umbilicus with a flexible measuring tape after large outer layers (e.g., jackets) were removed. Hip circumference in centimeters was assessed at the widest portion of the buttocks with a flexible measuring tape after large outer layers (e.g., jackets) were removed. All measurements were completed by a female graduate or research assistant. Values were used to calculate BMI [i.e., weight (kg)/height (m)2] for use as an exclusionary variable in data analyses (i.e., analyses restricted to those with BMI falling in average to overweight range).

Experimental Tasks

Negative mood induction. The negative mood induction methodology combines music and autobiographical recall to create a mild, transient sad mood. This induction method has been validated by previous research (Martin, 1990; Segal, Gemar & Williams, 1999; Segal et al., 2006). Participants listened to a digitally re-mastered, half-speed, non-lyrical piece of classical

music by Prokofiev entitled “Russia under the Mongolian Yoke” with written instructions to recall a time in their lives when they felt sad. The approximately 8-minute long taped segment was played to participants through a subject computer via E-Prime (2015) software and over-ear headphones. Prior to the negative mood induction, participants completed a negative 40-word emotional Stroop task (e.g., doomed, crying, hurt) employed to elicit a more pronounced response to the mood induction (Ben-Haim, Mama, Icht, & Algom, 2014). Words appeared individually on the computer screen, printed in red, green, yellow, or blue and participants were asked to select the matching color key on the keyboard (i.e., f for red, g for green, h for yellow, j for blue). Before each word was presented, a fixation cross (i.e., +) appeared on the screen for 700 ms to help participants focus their attention.

Neutral mood induction. The neutral mood induction methodology combines music and autobiographical recall to act as a control condition. This induction method has been validated by previous research, with results suggesting no significant change in mood (Green, Sedikides, Saltzberg, Wood, & Forzano, 2003; Wood, Saltzberg, & Goldsamt, 1990). Participants listened to digitally re-mastered, non-lyrical pieces of classical music from a selection of Chopin Waltzes (i.e., No. 11 in G flat, Op. 70, No. 1 and No. 12 in F minor, Op. 70, No. 2) played at half-speed with the written instruction to recall an uneventful day in their life that was neither especially happy nor sad. The approximately 8-minute long taped segment was played to participants through a subject computer via E-Prime (2015) software and over-ear headphones. Prior to neutral mood induction, participants completed a neutral 40-word Stroop task (e.g., parking, dial, walk) to compare with negative emotional Stroop task used during negative mood induction. The procedure for the neutral Stroop task is identical to that used in the negative emotional Stroop task, with the exception of different stimulus words.

Procedure

Session 1. Eligible participants came to the MMDL located in Corbett Hall on the University of Maine campus in Orono. Participants were greeted by a research assistant trained in standardized study procedure. The laboratory personnel introduced the study with the following statement: *"The purpose of the research is to learn about the emotional and physiological responses related to sad mood."* The purpose of deception (e.g., not disclosing the focus on binge eating, control, or stress) was because revealing target variables may significantly influence self-report, leading to over- or underestimates of behaviors and perceptions important to this study.

Shortly after participants arrived at their scheduled appointment, they reviewed an informed consent document in the presence of laboratory personnel, who also reviewed this form with each subject to ensure comprehension and answer questions (Appendix I). Importantly, voluntary study involvement, the option to leave at any time without penalty, and confidentiality of all data was discussed in detail at this time. For example, participants were informed that all data was stored via a secure server and ID numbers were assigned to further protect information. Additionally, the subject key housing participant names and ID numbers was saved on an alternate device with an encrypted password using Bit Locker. Despite these precautions, potential risks of the study were included in the consent review (e.g., loss of privacy, potential for emotional discomfort), along with benefits (e.g., assistance in helping to better understand study variables).

Following informed consent, participants were asked to complete a battery of questionnaires via Qualtrics (2016) on an electronic tablet. Questionnaires were administered in a randomized order and assessed binge eating symptom severity (i.e., BES), perceived control

(i.e., Pearlin's Perceived Mastery Scale), affective states (i.e., BDI-II, PANAS-X), and perceived stress (i.e., PSS-10; Appendix G). Once measures were completed, a trained graduate assistant conducted the SCID-IV-RV with each participant to determine study eligibility based on inclusion and exclusion criteria, and record diagnoses. Graduate assistants administering the SCID-IV-RV were trained to determine if a participant endorsed an exclusionary criteria diagnosis (e.g., bipolar disorder, psychosis), and discontinued the interview at the end of that module. Participants were thanked for their participation, and told that the study was recruiting individuals who had a very specific mental health profile, and they did not qualify for Session 2. Study credit was awarded despite meeting exclusionary criteria (e.g., subject pool participants received 1 credit for each hour spent in the lab; community participants received $30).

Once a participant either discontinued due to exclusion criteria, or completed the interview, they were given a referral list to community counseling services as a potential resource (Appendix J). This was presented as an information source, and not something that must be followed, with the statement: *"This referral list is provided for your information. If/when you would like counseling for distressing issues, these are some of the available options in this area. The list includes a variety of resources, some of which are low cost while others vary based on an hourly rate."*

In the unlikely event that a participant endorsed current suicidal ideation, the graduate student interviewer was equipped to complete a suicide risk assessment and consult with a licensed psychologist affiliated with the University to discuss a course of action. If hospitalization was deemed necessary, all steps were to be taken to encourage the participant to voluntarily go to the emergency department for an evaluation. The interviewer would accompany the individual to the hospital, following in their own vehicle. If self-admission was

not a viable option and there was imminent risk to the participants' safety, the interviewer was to call law enforcement to escort the participant to the emergency department.

Following the completion of the SCID-IV-RV, height, weight, waist circumference and hip circumference were measured and recorded in Qualtrics (2016) by a female graduate or research assistant. Eligible participants were then invited to participate in Session 2 within the following two weeks. Participants were thanked for their time and awarded research credits/$30 (i.e., subject pool/community sample) for their Session 1 participation lasting approximately 2 hours.

Session 2. Eligible participants returned to the MMDL in Corbett Hall for Session 2. Laboratory personnel greeted participants, thanked them for volunteering, and provided a brief overview of the Session with the following statement: *"Thank you for returning for Session 2 of this study. We are interested in investigating the physiological effects of different mood states, so today we will measure your physiological responses to video and audio clips. There will also be some additional questionnaires for you to complete."* Participants were reminded that their participation was entirely voluntary and that they could discontinue at any time without penalty prior to obtaining written and verbal consent for Session 2 participation (Appendix K).

Following consent, a female graduate or research assistant placed noninvasive electrical sensors on participants for physiological recordings. Participants were asked to remove jewelry and place this with other personal belongings (e.g., cell phones), asked about skin sensitivity and allergies, and prompted to use the restroom if necessary. During physiological hookup, participants were briefed on electrode placement and verbally alerted before placing each sensor. While attaching sensors, small talk (e.g., about classes, weather, hometown) was made in an attempt to increase participant comfort levels. Areas for sensor attachment were cleaned with an

abrasive alcohol swab. Self-sticking sensors were placed on each participant's right collarbone, bottom left and right ribs, jugular notch, sternum, heal of non-dominant hand, and upper- and mid-back, parallel within 1.5 inches of the jugular notch and sternum electrodes. During sensor placement, a second research assistant was monitoring MindWare Biolab 3.1 (2009) software to ensure accurate electrode placement via recorded waveforms. Once hookup was completed, participants were asked to sit in a comfortable chair in front of a computer with uncrossed legs for the remainder of physiological monitoring.

To ensure procedural standardization, directions, video, and audio was presented on a computer using E-Prime (2015) computer software. To acclimate participants to electrode placement and allow physiological responses to normalize and generate baseline HRV data, participants watched a 10-minute neutral travel video about Alaska's Denali National Park (Kolbeinsson, 2016). After this baseline period, participants were instructed to complete VAS measures of affect and stress via electronic tablet prior to mood induction (Appendix H).

Participants were randomized to either negative or neutral mood induction. Participants read mood induction instructions on a computer while wearing over-ear headphones. Following negative emotional Stroop task, participants in the negative mood induction group were prompted to think of a time in their lives when they felt sad as an 8-minute non-lyrical piece of classical music by Prokofiev entitled "Russia under the Mongolian Yoke" was played a half-speed. Those randomized to the neutral mood induction group received instructions to think of an uneventful day in their lives that was neither especially happy nor sad as an 8-minute non-lyrical piece of classical music from a selection of Chopin Waltzes (i.e., No. 11 in G flat, Op. 70, No. 1 and No. 12 in F minor, Op. 70, No. 2) played at half-speed following the neutral Stroop task. Physiological recordings continued during this time.

Following the negative or neutral mood induction, participants were given instructions on the computer to repeat VAS measures of affect and stress to assess for changes from pre-mood induction. They then were prompted to sit quietly for a 10-minute physiological recovery period while viewing a neutral video titled, *Alaska Last Frontier*. Following recovery, participants were instructed to repeat VAS measures of affect and stress to assess for changes from post-mood induction. A female graduate or research assistant then assisted with electrode removal. Participants were given a debriefing form (Appendix L) and then had an opportunity to ask laboratory personnel questions about the study prior to being awarding research credits/$15 (i.e., subject pool/community sample) for their Session 2 participation lasting approximately 1 hour.

Table 1. *Study Procedure Chart*

Time	Task	Operationalization	Purpose
Prescreen	Questionnaire	General Health Screen	Exclusion criteria
Session 1	Questionnaires	Demographics: race, sex, age	Covariate/control for baseline differences
		BDI-II: depressive symptom severity (DS)	H1, H2 Mediator
		BES: binge eating severity (BE)	H1, H2 Outcome H6 Predictor H8 Moderator
		PPMS: perceived control (PC)	H1, H2, H4, H7, H8 Predictor
		PSS-10: perceived stress (PS)	H2 Mediator
		PANAS-X: negative mood states (NM)	H2 Mediator
	Interview	SCID-IV-RV: psychiatric diagnoses	Exclusion criteria
	Height and Weight	BMI	Exclusion criteria
Session 2	Baseline	Physiological recording (ECG and Impedance) while viewing Alaska Denali Park Video	H2, H5, H6, H7, H8 Outcome
	Negative or Neutral Mood Induction (MI)	Pre/Post MI VAS: sad, happy, stress rating	H3, H4 Outcome H7, H8 Mediators
		Listen to sad/neutral music and think about sad/neutral autobiographical memory	H6, H7 Outcome
	Recovery	Physiological recording (ECG and Impedance) while viewing Alaska Wilderness Video	Exploratory analysis
	Debriefing	Debriefing form provided	None

Note.

H1: Higher PC will be predictive of less severe BE and this association will be significantly mediated by PS, DS, and NM

H2: Higher PC will be predictive of more adaptive (i.e., higher) resting HF-HRV/RSA and this association will be significantly mediated by PS, DS, and NM

H3: The negative MI group will demonstrate greater sad mood and stress reactivity than the neutral MI group

H4: Participants reporting greater PC undergoing negative MI will demonstrate less sad affect and stress reactivity than those reporting lower PC

H5: Participants in negative MI group will demonstrate greater changes in HF-HRV/RSA reactivity than those in the neutral MI group

H6: Within the negative MI group, those reporting greater BE will demonstrate less adaptive (i.e., lower) HF-HRV/RSA reactivity than those with less severe BE

H7: Within the negative MI group, those reporting greater PC will demonstrate more adaptive (i.e., higher) HF-HRV/RSA reactivity that those with lower PC and this association will be significantly mediated by sad affect and stress reactivity

H8: Greater BE will moderate the association between sad affect reactivity and HF-HRV/RSA, and stress reactivity and HF-HRV/RSA, resulting is less adaptive (i.e., lower) HF-HRV/RSA

CHAPTER THREE

RESULTS

The central aim of this study was to investigate the predictive role of perceived control and binge eating severity on mood reactivity, and possible concomitants with reduced cardiovascular function as measured by high-frequency heart rate variability/respiratory sinus arrhythmia (HF-HRV/RSA). Self-report questionnaires were used to test study hypotheses regarding the relation between perceived control, perceived stress, depressive mood, negative affect, and binge eating severity (i.e., Primary Hypothesis 1). An experimental negative mood induction task and additional self-report measures were utilized to further investigate hypotheses regarding stress and mood reactivity, and HF-HRV/RSA (i.e., Primary Hypothesis 2, Exploratory Hypotheses 3 through 8). Physiological data was collected and amplified with Mindware hardware and Biolab 3.1 (2009) acquisition software at a sampling rate of 1000 Hz. All analyses were conducted using IBM SPSS Version 24.0.0.0 (IBM Corp., 2016).

Hypothesis One

Preliminary Analyses

Demographic information. A sample of 217 participants was collected from both the University of Maine Psychology subject pool and the surrounding community. Following Session 1 questionnaires and clinical interview, 97 (45.33%) participants met inclusion criteria to participate in Session 2. These data were utilized for Hypothesis 1 analyses. Once cleaned, outliers addressed, and those with missing measures excluded, 92 participants remained. In keeping with hypotheses targeting normal to overweight individuals, data were further sorted to exclude participants with a body mass index (BMI) falling below the average range (i.e., < 18.5) or greater than the overweight range (i.e., ≥ 30). After this additional sorting, 75 participants

(81.52% of 92 participant sample) remained for further analyses. This sample was predominately female (*n* = 49, 65.3%), European American (*n* = 65, 86.7%), never married/single (*n* = 69, 92.0%) and high school educated (*n* = 34, 45.3%) with a mean age of 21.01 years (*SD* = 6.79, range = 18-60), height of 67.73 inches (*SD* = 3.49, range = 61.00-78.00), weight of 154.47 pounds (*SD* = 24.55, range = 109.00-210.00) and BMI of 23.61 (*SD* = 2.83, range = 18.60-29.11). Subject pool participants made up 65.3% (*n* = 59) of the sample, and 22 participants (29.3%) had a history of major depressive disorder (MDD) with sub-clinical symptoms for at least 6 months before study participation. Despite efforts to collect a larger sample than initially proposed, the population was largely characterized as reporting minimal binge eating symptoms both before (*n* = 84, 86.6%) and after excluding obese participants (*n* = 69, 92.0%). Due to this, binge eating symptom severity was utilized as a continuous variable and not investigated dichotomously in analyses. Sample descriptive statistics are presented in Table 2.

Table 2. *Hypothesis One Sample: Descriptive Statistics*

Characteristic	*N*	%ile	*M*	*SD*	Range
Sex					
Female	49	65.3			
Male	26	34.7			
Race					
European American	65	86.7			
Asian	3	4.0			
African American	2	2.7			
Multiple Races	4	5.3			
Omitted Responses	1	1.3			
Marital Status					
Never Married/Single	69	92.0			
Married	5	6.7			
Divorced	1	1.3			
Education					
High School	34	45.3			
1 Year College or Technical School	17	22.7			
2+ Years College without Degree	14	18.7			
4 Years College with Degree	6	8.0			
Postgraduate MD, Ph.D.	4	5.3			
Age in Years			21.01	6.79	18-60
Height in Inches			67.73	3.49	61.0-78.0
Weight in Pounds			154.47	24.55	109.0-210.0
BMI			23.61	2.83	18.60-29.11
Binge Eating Symptom Severity					
None/Minimal (BES[a] score <18)	69	92.0			
Moderate/Severe (BES score ≥18)	6	8.0			
Recruitment Group					
Subject Pool	59	65.3			
Community	26	34.7			
History of Major Depressive Disorder					
No History	53	70.7			
Positive History ≥ 6 months from testing	22	29.3			

[a]Binge Eating Scale

Independent *t*-tests were utilized to investigate differences by recruitment sample. Results revealed that individuals recruited from the community were significantly older than participants from the University of Maine Psychology subject pool [$t(73) = -4.89$, $p < .001$, Cohen's $d = 0.99$]. However, there was no significant difference between groups for height [$t(73) = 1.26$, $p = .212$, Cohen's $d = 0.27$], weight [$t(73) = .78$, $p = .446$, Cohen's $d = 0.19$], or BMI [$t(73) = -.33$, $p = .981$, Cohen's $d = 0.01$]. Means and standard deviations are presented in Table 3.

Table 3. *Hypothesis One Sample: Mean Age, Height, Weight, and BMI by Recruitment Group*

	Subject Pool (n = 49)	Community (n = 26)
Characteristic	*M* (*SD*)	*M* (*SD*)
Age in Years	18.57_a (.79)	25.62_b (10.09)
Height in Inches	68.10 (3.36)	67.04 (3.69)
Weight in Pounds	156.08 (23.93)	151.42 (25.87)
BMI	23.62 (2.85)	23.59 (2.85)

Note. Means with different subscripts are statistically significant at $p < .01$.

Differences by history of MDD were also investigated using independent *t*-tests. Those with a history of MDD were significantly older than individuals without a history of MDD [$t(73) = 3.15$, $p = .002$, Cohen's $d = 0.65$], and there were again no significant differences by group for height [$t(73) = -1.21$, $p = .238$, Cohen's $d = 0.31$], weight [$t(73) = -1.50$, $p = .143$, Cohen's $d = 0.38$], or BMI [$t(73) = -1.06$, $p = .292$, Cohen's $d = 0.27$]. Table 4 presents means and standard deviations by MDD history.

Table 4. *Hypothesis One Sample: Mean Age, Height, Weight, and BMI by MDD History*

Characteristic	Positive MDD History (n = 22) *M* (*SD*)	No MDD History (n = 53) *M* (*SD*)
Age in Years	14.64_a (10.63)	19.51_b (4.49)
Height in Inches	66.98 (3.42)	68.05 (3.51)
Weight in Pounds	147.91 (25.62)	157.19 (22.80)
BMI	23.07 (2.78)	23.83 (2.85)

Note. Means with different subscripts are statistically significant at $p < .01$.

A Chi-Square analysis was used to compare race/ethnicity of participants across recruitment groups and history of MDD. Given the predominately European American sample (n = 65, 86.7%), race/ethnicity categories were collapsed into two dichotomous groups (i.e., European American, non-European American) to meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5). Results revealed that the community group had significantly more self-identified racial/ethnic diversity (i.e., non-European American) than participants recruited from the University of Maine Psychology subject pool [χ^2 (1) = 8.18, p = .004]. Race/ethnicity information within each recruitment group is presented in Table 5. There was no significant difference between groups when investigated by MDD history [χ^2 (1) = .28, p = .592; Table 6].

Table 5. *Hypothesis One Sample: Race/Ethnicity by Recruitment Group*

Race/Ethnicity	Subject Pool (n = 49)	Community (n = 26)
European American	46_a (93.9%)	19_b (73.1%)
Asian	1 (2.0%)	2 (7.7%)
African American	0 (0.0%)	2 (7.7%)
Multiple Races	1 (2.0%)	3 (11.5%)
Omitted Responses	1 (2.0%)	0 (0.0%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Table 6. *Hypothesis One Sample: Race/Ethnicity by MDD History*

Race/Ethnicity	MDD History (n = 22)	No MDD History (n = 53)
European American	20 (90.9%)	45 (84.9%)
Asian	1 (4.5%)	2 (3.8%)
African American	0 (0.0%)	2 (3.8%)
Multiple Races	1 (4.5%)	3 (5.7%)
Omitted Responses	0 (0.0%)	1 (1.9%)

Chi-Square analyses were also utilized to compare marital status of participants across recruitment groups and MDD history. Given the predominately never married/single sample (n = 69, 92.0%) marital status categories were collapsed into two dichotomous groups [i.e., never married/single, other (i.e., married, divorced)] to meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5). Results indicated that the community group had significantly more marital status diversity (i.e., married, divorced) than participants recruited from the subject pool who were predominately never married/single [χ^2 (1) = 12.29, p < .001; Table 7]. This significant difference was also observed by MDD history, with those having a history of MDD reporting greater marital status diversity (i.e., married, divorced) than those without a history of MDD [χ^2 (1) = 4.39, p = .043; Table 8].

Table 7. *Hypothesis One Sample: Marital Status by Recruitment Group*

Marital Status	Subject Pool (n =49)	Community (n = 26)
Never Married/Single	49_a (100.0%)	20_b (76.9%)
Married	0 (0.0%)	5 (19.2%)
Divorced	0 (0.0%)	1 (3.8%)

Note. Values with different subscripts are statistically significant at p < .05.

Table 8. *Hypothesis One Sample: Marital Status by MDD History*

Marital Status	MDD History (n =22)	No MDD History (n = 53)
Never Married/Single	18_a (81.8%)	51_b (96.2%)
Married	3 (13.6%)	2 (3.8%)
Divorced	1 (4.5%)	0 (0.0%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Chi-Square analyses were used to compare educational level of participants across recruitment groups. To meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5), educational level was collapsed into two groups (i.e., high school, greater than high school). Perhaps not surprising given that the University of Maine Psychology subject pool is largely made up by students enrolled in introductory psychology courses, results suggest that community members had significantly higher education levels beyond high school than subject pool participants [χ^2 (1) = 27.64, $p <$ .001; Table 9]. However, there was no significant difference in educational levels between those with a history of MDD and those who had never experienced MDD [χ^2 (1) = 2.29, p = .137; Table 10].

Table 9. *Hypothesis One Sample: Education Level by Recruitment Group*

Education	Subject Pool (n = 49)	Community (n = 26)
High School	33_a (67.3%)	1_b (3.8%)
1 Year College or Technical School	12 (24.5%)	5 (19.2%)
2+ Years College without Degree	4 (8.2%)	10 (38.5%)
4 Years College with Degree	0 (0.0%)	6 (23.1%)
Postgraduate MD, Ph.D.	0 (0.0%)	4 (15.4%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Table 10. *Hypothesis One Sample: Education Level by MDD History*

Education	MDD History (n = 22)	No MDD History (n = 53)
High School	7 (31.8%)	27 (50.9%)
1 Year College or Technical School	3 (13.6%)	14 (26.4%)
2+ Years College without Degree	5 (22.7%)	9 (17.0%)
4 Years College with Degree	5 (22.7%)	1 (1.9%)
Postgraduate MD, Ph.D.	2 (9.1%)	2 (3.8%)

Questionnaires. During Session 1, participants completed five self-report measures for Hypothesis 1 analyses. The Beck Depression Inventory-II Edition (BDI-II; Beck et al., 1996b; Appendix G) collects ratings of depressive symptoms experienced during the previous two weeks. Severity of 21 symptoms consistent with depression were rated on a 0 (*no presence of a symptom*) to 3 (*symptom is present and severe*) scale with greater scores indicating more severe depressive symptoms (total score range = 0-63). To assess current binge eating severity, participants completed the Binge Eating Scale (BES; Gormally et al., 1982; Appendix G). Each of the 16 items consistent with cognitive and behavioral symptoms associated with binge eating were rated on a 0 (*no presence of a symptom*) to 3 (*symptom is present and severe*) scale with greater scores indicating more severe binge eating symptoms (total score range = 0-42). Negative affect over the previous two weeks was rated on the Positive and Negative Affect Schedule – Expanded Form (PANAS-X; Watson & Clark, 1994; Appendix G), a 60-item self-report questionnaire where various affective adjectives are ranked on a Likert scale of 1 (*Very Slightly or Not at All*) to 5 (*Extremely*). Higher scores indicate greater presence of an affective state (e.g., negative affect total score range = 5-50). Ratings of perceived control (i.e., perceived ability to impact behavior and life outcomes) were collected via Pearlin's Perceived Mastery Scale (PC; Pearlin & Schooler, 1978; Appendix G). Seven items were rated on a 0 (*Strongly*

Disagree) to 6 (*Strongly Agree*) Likert scale with higher scores indicating greater perceptions of control (total score range = 0-42). Perceived stress (i.e., degree to which one has perceived life as unpredictable, overloading, and uncontrollable during the previous month) was assessed by the Perceived Stress Scale – 10 (PSS-10; Cohen & Williamson, 1988; Appendix G). Participants rated 10 items on a 0 (*Never*) to 4 (*Very Often*) Likert scale, with higher scores indicating greater perceived stress (total score range = 0-40).

Prior to analyses, data were inspected for potential univariate outliers, defined as z-scores exceeding ± 3.0 (Daszykowski, Kaczmarek, Heyden, & Walczak, 2007). Winsorizing, a data transformation procedure to manage outliers by changing extreme values to the next most non-outlier extreme value, was utilized. Winsorization reduces skew of the distribution caused by outliers while relatively preserving overall data variation (Field, 2009). Outlier data for binge eating severity (n = 1), negative affect (n = 1), and depressive mood (n = 2) were winsorized to address extreme values.

Descriptive statistics for Hypothesis 1 questionnaires are presented in Table 11. Univariate analyses of variance (ANOVA) indicated that questionnaire means did not represent ceiling or floor values that could restrict regression analyses. Additionally, standard deviations suggested adequate variability for hypothesis testing. All questionnaires demonstrated adequate internal consistency (α = .791 to .901) in this study sample.

Table 11. *Hypothesis One Sample: Questionnaire Descriptive Statistics*

Questionnaire	*M* (*SD*) (*N* = 75)	Sample Range	Sample α
BDI-II	5.16 (5.72)	0-23	.901
BES	7.59 (6.19)	0-26	.889
PANAS-X Negative Affect Subscale	16.11 (5.42)	10-32	.852
PC	32.98 (6.03)	16-42	.791
PSS-10	12.48 (6.38)	0-27	.856

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.

Questionnaires by recruitment group. Independent *t*-tests revealed that there was no difference in depressive symptom severity between participants recruited from the University of Maine Psychology subject pool and those from the surrounding community [$t(71) = -1.44$, $p = .152$, Cohen's $d = 0.34$]. Similarly, there was no difference between recruitment groups for ratings of perceived control [$t(71) = 1.92$, $p = .064$, Cohen's $d = 0.44$], perceived stress [$t(68) = .09$, $p = .933$, Cohen's $d = 0.02$], or negative affect [$t(69) = -1.93$, $p = .062$, Cohen's $d = 0.44$]. However, those recruited from the community reported higher levels of binge eating severity than individuals recruited from the Psychology subject pool [$t(68) = -4.62$, $p < .001$, Cohen's $d = 1.09$]. Means and standard deviations by recruitment group are presented in Table 12.

Table 12. *Hypothesis One Sample: Questionnaire Means and Standard Deviations by Recruitment Group*

Questionnaire	Subject Pool *M* (*SD*) (*n* = 49)	Community *M* (*SD*) (*n* = 26)
BDI-II	4.18 (4.56)	6.04 (6.39)
BES	4.66_a (3.55)	9.46_b (5.14)
PANAS-X Negative Affect Subscale	14.93 (3.73)	17.15 (6.00)
PC	34.21 (4.49)	31.68 (6.69)
PSS-10	12.25 (5.54)	12.11 (6.24)

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
Means with different subscripts are statistically significant at $p < .001$.

Questionnaires by MDD history. Independent *t*-tests revealed that participants without a history of MDD reported higher levels of perceived control than those with a history of MDD [$t(71) = -4.95$, $p < .001$, Cohen's $d = 1.21$]. Individuals with a history of MDD reported more severe depressive symptoms [$t(72) = 4.86$, $p < .001$, Cohen's $d = 1.08$], negative affect [$t(69) = 4.83$, $p < .001$, Cohen's $d = 1.11$], perceived stress [$t(68) = 3.40$, $p = .001$, Cohen's $d = 0.87$], and binge eating symptom severity [$t(68) = 4.85$, $p < .001$, Cohen's $d = 1.24$]. Means and standard deviations by history of MDD are presented in Table 13.

Table 13. *Hypothesis One Sample: Questionnaire Means and Standard Deviations by MDD History*

Questionnaire	MDD History *M* (*SD*) (*n* = 22)	No MDD History *M* (*SD*) (*n* = 53)
BDI-II	8.82_a (6.55)	3.12_b (3.52)
BES	10.25_a (4.63)	4.92_b (3.95)
PANAS-X Negative Affect Subscale	19.55_a (5.95)	14.25_b (3.22)
PC	29.18_a (5.45)	35.14_b (4.37)
PSS-10	15.65_a (6.01)	10.82_b (5.09)

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
Means with different subscripts are statistically significant at $p \leq .001$.

Primary Analyses

Primary Hypothesis 1 states that perceived control will significantly predict binge eating symptoms severity such that higher perceived control will be associated with lower binge eating severity. If data are consistent with Hypothesis 1, this will be due in part to higher perceived control being predictive of reduced perceived stress, decreased depressive symptomatology, and less negative affect, which will then be predictive of reduced binge eating symptom severity. Questionnaire data from Session 1 were utilized to investigate these associations.

In keeping with the directional relations predicted in Hypothesis 1, Pearson correlation analyses revealed a significant negative correlation between binge eating severity (BES) and perceived control (PC; $r = -.37$, $p < .001$, Table 14), such that greater reports of perceived control were associated with lower binge eating severity. Also consistent with hypotheses, perceived stress (PSS-10), depressive symptoms (BDI-II), and negative mood (PANAS-X Negative Affect Subscale) were significantly and positively correlated with binge eating severity (all r's > .31, all p's < .008), indicating that higher reports of stress, depressive symptoms, and negative affect were related to greater reports of binge eating symptom severity. Perceived stress, depressive

symptoms, and negative affect shared significant positive correlations (all *r*'s > .54, all *p*'s < .001), such that more symptom severity in one stress or mood area was associated with greater symptom severity in all stress and mood domains. Significant negative correlations were observed between perceived control and perceived stress ($r = -.54$, $p < .001$), depressive symptoms ($r = -.42$, $p < .001$), and negative mood ($r = -.49$, $p < .001$), indicating that consistent with hypotheses, those reporting greater perceived control experienced less severe perceived stress, depressive symptoms, and negative affect.

Table 14. *Hypothesis One Sample: Correlations Between Self-Report Questionnaires*

Questionnaire	1.	2.	3.	4.	5.
1. BDI-II	-				
2. BES	.55**	-			
3. PANAS-X Negative Affect Subscale	.59**	.31*	-		
4. PC	-.42**	-.37*	-.49**	-	
5. PSS-10	.62**	.32*	.60**	-.54**	-

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
* $p < .01$, ** $p < .001$.

In addition to Pearson correlation analyses, questionnaire data tests of multicollinearity were performed to investigate variance inflation factors (VIF) and tolerance to ensure that variables were not too closely related or redundant for regression analyses. It is generally accepted that a tolerance value less than 0.1 indicates redundancy of independent variables (IVs), and that a VIF greater than 3 is indicative of IVs being too closely related to assess their independent effects on dependent variables (DVs). A VIF greater than 5 indicates there is likely too much overlap in IVs, and a VIF greater than 10 is indicative of significant problems with multicollinearity (Tabachnick & Fidell, 2013). Total scores for IVs of depressive symptoms (BDI-II), negative affect (PANAS-X Negative Affect Subscale), perceived control (PC), and

perceived stress (PSS-10) were examined with binge eating symptoms (BES) as the DV. Results suggest no evidence of multicollinearity (all VIF's < 2.06, all tolerance >.49; Table 15).

Table 15. *Hypothesis One Sample: Tests of Multicollinearity for Self-Report Questionnaires*

Questionnaire	VIF	Tolerance
BDI-II	1.72	.58
PANAS-X Negative Affect Subscale	1.76	.57
PC	1.51	.66
PSS-10	2.06	.49

Note. Binge Eating Scale (BES) entered as dependent variable in all multicollinearity analyses. BDI-II = Beck Depression Inventory-II, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10, VIF = Variance Inflation Factors.

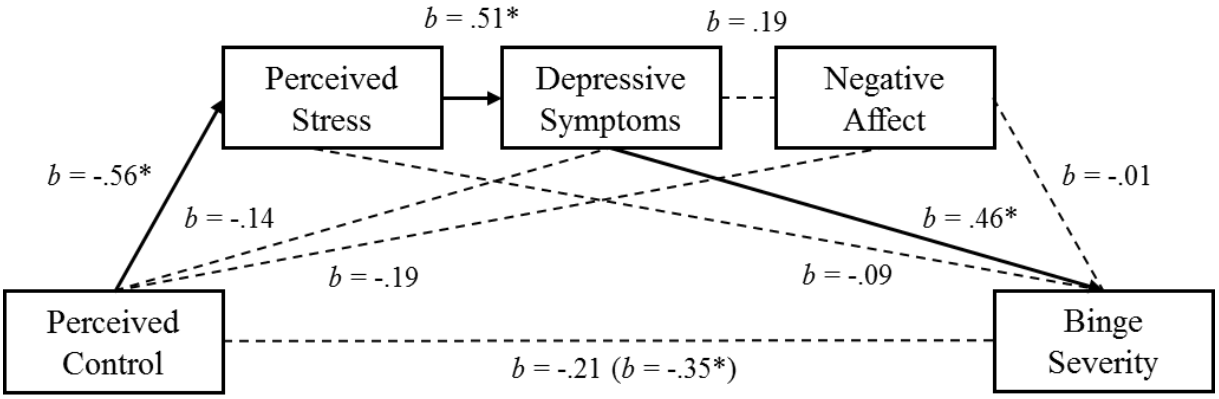
Serial mediated regression. Serial multiple mediator regression with bootstrap estimation (5000 samples) with PROCESS for SPSS (Hayes, 2014) was utilized to test the hypothesis that greater self-reported perceptions of control would predict less severe binge eating symptomatology, and that this relation would be significantly mediated by perceived stress, depressive symptoms, and negative mood.

In line with expectations, participants reporting higher perceived control also reported less binge eating behavior (total effect b = -.35; BC 95% CI: -.55, -.15; Figure 2 for full model), indicating that higher perceived control was predictive of less severe binge eating symptoms. Additionally, higher rates of perceived control predicted less perceived stress (b = -.56, p < .001). However, higher rates of perceived control were not significantly predictive of lower depressive symptoms (b = -.14, p = .214) or negative affect (b = -.19, p = .055). Once entered in the serial multiple mediator model [R^2 = .35, $F(4,63) = 8.31$, p < .001], the link between perceived control and binge eating severity was no longer significant (direct effect b = -.21; BC 95% CI: -.43, .01), and higher rates of depressive symptoms were predictive of greater binge

eating severity (b = .46, p < .001). However, greater perceived stress (b = -.09, p = .463) and negative affect (b = -.01, p = .924) did not significantly predict binge eating severity.

Serial multiple mediator modeling is best selected for analyses when it is assumed that mediator variables are significantly correlated. This assumption was partially supported as results revealed that greater perceived stress predicted increased depressive symptomatology (b = .51, p < .001), but depressive symptoms did not predict negative mood as anticipated (b = .19, p = .065). However, the significant association between perceived stress and depressive symptoms were found to significantly mediate the relation between perceived control and binge eating severity (specific indirect effect in serial b = -.13; BC 95% CI: -.28, -.04; Figure 2 for full model), suggesting that high perceptions of control predict reduced perceived stress. Lower perceived stress then is predictive of reductions in depressive symptoms, and this is predictive of less severe binge eating symptomatology.

Figure 2. *Hypothesis One Serial Multiple Mediation of Perceived Control and Binge Eating Severity*



Note. Bold arrows represent the significant specific serial mediation of perceived stress and depressive symptoms on the relation between perceived control and binge eating severity (specific indirect effect in serial b = -.13; Bias Corrected (BC) 95% Confidence Interval (CI): -.28, -.04). Significant hypothesized multiple mediator model [R^2 = .35, F(4,63) = 8.31, p < .001].
*p < .05.

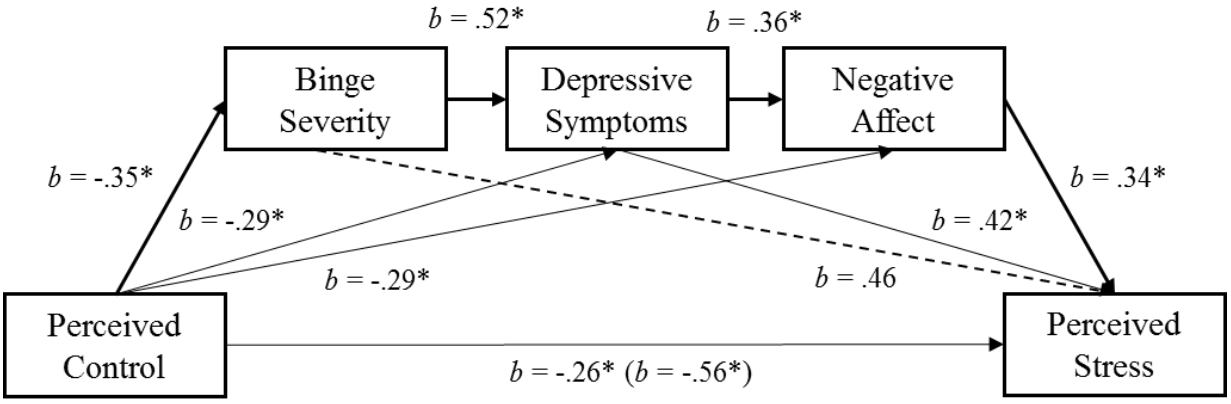
Sex moderated serial mediated regression. Hypothesis 1 serial multiple mediated regression was further investigated to examine possible moderating effects of sex. Results revealed there was no significant impact of sex on the associations between perceived control and perceived stress [difference between the two conditional indirect effects of a dichotomous moderator (index) = .00, BC 95% CI: -.04, .12], perceived control and depressive symptoms (index = .04, 95% CI: -.15, .22), or perceived control and negative affect (index = .00, BC 95% CI: -.07, .07). Similarly, sex did not significantly moderate relations between perceived stress and binge eating severity (index = -.03, BC 95% CI: -.23, .22), depressive symptoms and severity of binge eating (index = .28, BC 95% CI: -.31, .38), or negative affect and binge eating severity (index = -.17, BC 95% CI: -.45, .07). Results suggest that the associations between perceived control, perceived stress, depressive symptoms, negative affect, and binge eating severity do not significantly differ between males and females.

History of MDD moderated serial mediated regression. A follow-up analysis was conducted to better understand if a history of MDD moderated Hypothesis 1 serial multiple mediated regression. Results indicate that history of MDD does not significantly moderate associations between perceived control and mediator variables (all index's <.04, all BC 95% CI's between -.37, .24), or mediator variables and binge eating severity (all index's <.00, all BC 95% CI's between -.44, .28). Findings propose that a history of MDD does not significantly impact the relations between perceived control, perceived stress, depressive symptoms, negative affect, and binge eating severity.

Alternative serial mediated regression. In mediated regression analyses, outcomes can cause mediators and therefore, it is advisable to provide additional evidence for model fit by investigating reverse casual mediation by interchanging mediator and outcomes variables (Judd

& Kenny, 2010). Serial multiple mediator modeling was repeated to investigate mediators as outcome variables. When entered as an outcome variable, perceived stress was significantly predicted by perceived control (total effect b = -.56; BC 95% CI: -.77, .34; Figure 3 for full model), such that higher perceived control was predictive of less perceived stress. There was significant serial multiple mediator model fit [R^2 = .52, F(4,63) = 17.00, p < .001] and the relation between perceived control and perceived stress remained significant (total effect b = -.26; BC 95% CI: -.48, -.04). Unlike the hypothesized model, significant associations between all mediators significantly impacted the association between perceived control and perceived stress (specific indirect effect in serial b = -.02; BC 95% CI: -.08, -.00), suggesting that higher perceived control was predictive of lower binge eating severity and that reductions in binge eating severity was then predictive of less depressive symptomatology, which then predicted reductions in negative affect. These relations then significantly predicted less perceived stress.

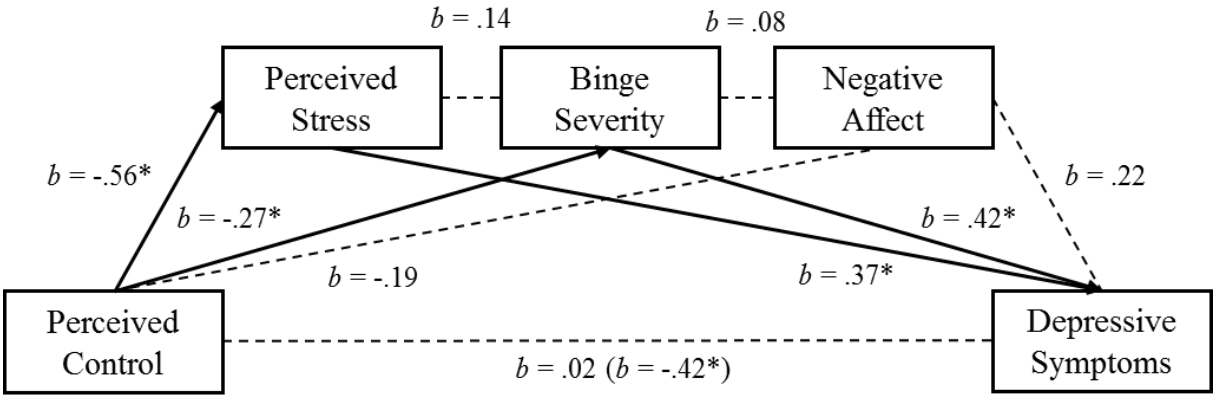
Figure 3. *Hypothesis One Alternative Model: Serial Multiple Mediation of Perceived Control and Perceived Stress*



Note. Bold arrows represent the significant specific serial mediation of binge eating severity, depressive symptoms, and negative affect on the relation between perceived control and perceived stress (specific indirect effect in serial b = -.22; BC 95% CI: -.08, -.00). Significant alternative multiple mediator model [R^2 = .52, F(4,63) = 17.00, p < .001].
*p < .05.

Depressive symptoms were then investigated as an outcome variable. Perceived control significantly predicted depressive symptoms (total effect *b* = -.56; BC 95% CI: -.77, -.34; Figure 4 for full model), such that higher perceived control predicted less depressive symptoms severity. There was significant serial multiple mediator model fit [R^2 = .53, $F(4,63) = 18.01$, $p < .001$] and the relation between perceived control and perceived stress did not remain significant (total effect *b* = .02; BC 95% CI: -.19, .24). Unlike the hypothesized model, there were no significant serial indirect effects, but rather the relation between perceived control and depressive symptoms was significantly mediated by perceived stress (specific indirect effect *b* = -.20; BC 95% CI -.45, -.08) and binge eating symptom severity (specific indirect effect *b* = -.12; BC 95% CI -.25, -.03). This suggests that greater perceived control is predictive of less perceived stress and less severe binge eating, and that these reductions are predictive of reduced depressive symptom severity. However, in this model, mediators (i.e., perceived stress and binge eating severity) were not significantly associated and changes in one variable did not appear to significantly impact the other variable.

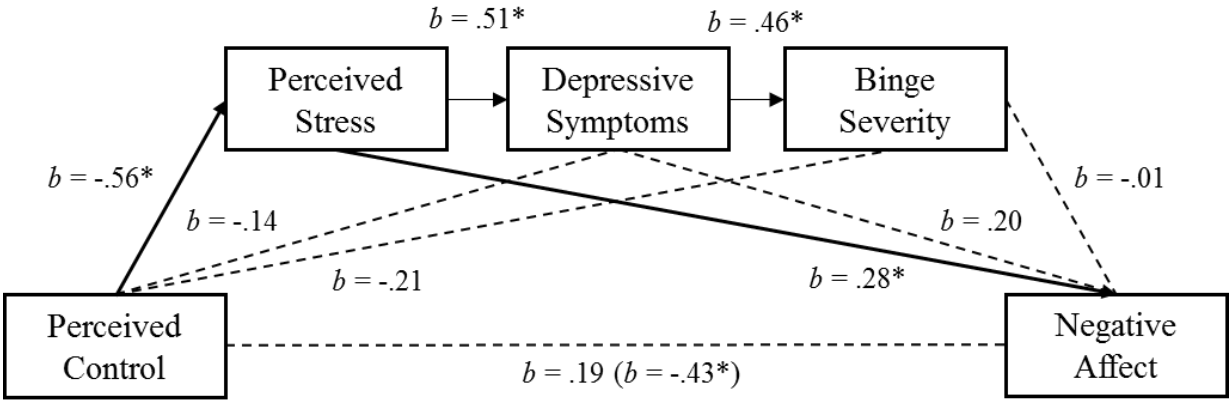
Figure 4. *Hypothesis One Alternative Model: Multiple Mediation of Perceived Control and Depressive Symptoms*



Note. Bold arrows represent the significant specific mediation of perceived stress and binge eating severity on the relation between perceived control and depressive symptoms (specific indirect effect of perceived stress b = -.20; BC 95% CI: -.45, -.08; specific indirect effect of binge eating severity b = -.12; BC 95% CI -.25, -.03). Significant alternative multiple mediator model [R^2 = .53, $F(4,63) = 18.01$, $p < .001$].
*$p < .05$.

The final mediator explored as an outcome variable was negative affect. Perceived control significantly predicted negative affect (total effect b = -.43; BC 95% CI: -.62, -.25; Figure 5 for full model), such that higher perceived control predicted less negative affect. There was significant serial multiple mediator model fit [R^2 = .66, $F(4,63) = 11.92$, $p < .001$] and the relation between perceived control and perceived stress did not remain significant (total effect b = -.19; BC 95% CI: -.39, .09). Unlike the hypothesized model, there were no significant serial indirect effects, and only perceived stress significantly mediated the association between perceived control and negative affect (specific indirect effect b = -.16; BC 95% CI -.36, -.03), suggesting that higher ratings of perceived control are predictive of less perceived stress which then predicts reduced negative affect.

Figure 5. *Hypothesis One Alternative Model: Multiple Mediation of Perceived Control and Negative Affect*



Note. Bold arrows represent significant specific mediation of perceived stress on the relation between perceived control and negative affect (specific indirect effect b = -.16; BC 95% CI: -.36, -.03). Significant alternative multiple mediator model [R^2 = .66, $F(4,63) = 11.92$, $p < .001$]. *$p < .05$.

Hypothesis Two

Preliminary Analyses

Demographic information. Hypothesis 1 had a sample of 97 participants who met inclusion criteria to proceed to Session 2. Once cleaned, outliers addressed, and those with missing measures excluded, 92 participants remained. Data were further sorted to exclude participants with BMI's falling below the average range (i.e., < 18.5) or greater than the overweight range (i.e., ≥ 30). After this additional sorting, 75 participants (81.52% of 92 participant sample) remained for further Hypothesis 1 analyses. However, of this sample, 20 participants who proceeded to Session 2 had incomplete physiological data due to equipment malfunction and could not be utilized for Primary Hypothesis 2 analyses or Exploratory Hypotheses 3-8 analyses. Due to this significant change in the sample size (i.e., 19.78% of 92 participant sample), demographic information was reanalyzed prior to proceeding with Hypothesis 2 testing.

Of the participants who completed Session 2 physiological data collection (*N* = 72), 70 participants remained after data was cleaned, outliers addressed, and those with missing measures excluded. In keeping with hypotheses targeting normal to overweight individuals, data were further sorted to exclude participants with BMI's falling below the average range (i.e., < 18.5) or greater than the overweight range (i.e., ≥ 30). After this additional sorting, 55 participants (78.57% of 70 participant sample) remained for further Hypothesis 2 analyses. Consistent with the Hypothesis 1 sample, these participants were predominately female (*n* = 34, 61.8%), European American (*n* = 50, 90.9%), never married/single (*n* = 53, 96.4%) and high school educated (*n* = 34, 61.8%) with a mean age of 20.22 years (*SD* = 6.43, range = 18-60), height of 68.18 inches (*SD* = 3.30, range = 61.00-78.00), weight of 155.31 pounds (*SD* = 22.63, range = 117.00-210.00) and BMI of 23.47 (*SD* = 2.77, range = 18.60-29.11). Subject pool participants made up 83.6% (*n* = 46) of the sample, and 13 participants (23.6%) had a history of an MDD episode at least 6 months before study participation. Despite efforts to collect a larger sample than initially proposed, the population was largely characterized as having minimal binge eating symptoms both before (*n* = 63, 85.1%) and after excluding obese participants (*n* = 53, 96.4%). Due to this, binge eating symptom severity was utilized as a continuous variable and not investigated dichotomously in analyses. Table 16 presents sample descriptive statistics.

Table 16. *Hypothesis Two and Exploratory Hypotheses Sample: Sample Descriptive Statistics*

Characteristic	*N*	%ile	*M*	*SD*	Range
Sex					
Female	34	61.8			
Male	21	38.2			
Race					
European American	50	90.9			
Asian	1	1.8			
African American	1	1.8			
Multiple Races	2	3.6			
Omitted Responses	1	1.8			
Marital Status					
Never Married/Single	53	96.4			
Married	1	1.8			
Divorced	1	1.8			
Education					
High School	34	61.8			
1 Year College or Technical School	12	21.8			
2+ Years College without Degree	5	9.1			
4 Years College with Degree	2	3.6			
Postgraduate MD, Ph.D.	2	3.6			
Age in Years			20.22	6.43	18-60
Height in Inches			68.17	3.30	61.0-78.0
Weight in Pounds			155.31	22.63	117.0-210.0
BMI			23.47	2.77	18.60-29.11
Binge Eating Symptom Severity					
None/Minimal (BES[a] score <18)	53	96.4			
Moderate/Severe (BES score ≥18)	2	3.6			
Recruitment Group					
Subject Pool	46	83.6			
Community	9	16.4			
History of Major Depressive Disorder					
No History	42	83.6			
Positive History ≥ 6 months from testing	13	23.6			

[a]Binge Eating Scale

Independent *t*-tests were utilized to investigate differences by recruitment sample. As seen in the sample used to test Hypothesis 1, results revealed that individuals recruited from the community were significantly older than participants from the University of Maine Psychology subject pool [$t(53) = -5.173$, $p < .001$, Cohen's $d = 1.05$]. However, there was no significant difference between groups for height [$t(53) = .28$, $p = .785$, Cohen's $d = 0.09$], weight [$t(53) = -.44$, $p = .673$, Cohen's $d = 0.03$], or BMI [$t(53) = .791$, $p = .43$, Cohen's $d = 0.29$]. Means and standard deviations are presented below in Table 17.

Table 17. *Hypothesis Two and Exploratory Hypotheses Sample: Mean Age, Height, Weight, and BMI by Recruitment Group*

	Subject Pool (n = 46)	Community (n = 9)
Characteristic	*M* (*SD*)	*M* (*SD*)
Age in Years	18.59_a (.83)	28.56_b (13.46)
Height in Inches	68.23 (3.10)	67.89 (4.40)
Weight in Pounds	157.72 (22.62)	158.33 (23.80)
BMI	23.33 (2.78)	24.13 (2.77)

Note. Means with different subscripts are statistically significant at $p < .01$.

Differences by history of MDD were also investigated using independent *t*-tests. As observed in the Hypothesis 1 sample, those with a history of MDD were significantly older than individuals without a history of MDD [$t(53) = 3.09$, $p = .003$, Cohen's $d = 0.67$], and there were again no significant differences by group for height [$t(53) = -1.23$, $p = .237$, Cohen's $d = 0.37$], weight [$t(53) = -1.15$, $p = .253$, Cohen's $d = 0.35$], or BMI [$t(53) = -.48$, $p = .639$, Cohen's $d = 0.15$]. Table 18 presents means and standard deviations by MDD history.

Table 18. *Hypothesis Two and Exploratory Hypotheses Sample: Mean Age, Height, Weight, and BMI by MDD History*

Characteristic	MDD History (n = 13) *M* (*SD*)	No MDD History (n = 42) *M* (*SD*)
Age in Years	24.69_a (12.37)	18.83_b (1.12)
Height in Inches	67.19 (3.81)	68.48 (3.12)
Weight in Pounds	149.00 (24.92)	157.26 (21.82)
BMI	23.14 (2.89)	23.57 (2.76)

Note. Means with different subscripts are statistically significant at $p < .01$.

A Chi-Square analysis was used to compare race/ethnicity of participants across recruitment groups and history of MDD. Given the predominately European American sample (n = 50, 90.9%), race/ethnicity categories were collapsed into two dichotomous groups (i.e., European American, non-European American) to meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5). Results revealed there was no significant difference in race/ethnicity between recruitment groups [χ^2 (1) = .864, p = .353; Table 19]. There was also no significant difference between groups when investigated by MDD history [χ^2 (1) = 1.37, p = .242; Table 20].

Table 19. *Hypothesis Two and Exploratory Hypotheses Sample: Race/Ethnicity by Recruitment Group*

Race/Ethnicity	Subject Pool (n = 46)	Community (n = 9)
European American	41 (89.1%)	9 (100%)
Asian	1 (2.2%)	0 (0.0%)
African American	1 (2.2%)	0 (0.0%)
Multiple Races	2 (4.3%)	0 (0.0%)
Omitted Responses	1 (2.2%)	0 (0.0%)

Table 20. *Hypothesis Two and Exploratory Hypotheses Sample: Race/Ethnicity by MDD History*

Race/Ethnicity	MDD History (n = 13)	No MDD History (n = 42)
European American	13 (100%)	37 (88.1%)
Asian	0 (0.0%)	1 (2.4%)
African American	0 (0.0%)	1 (2.4%)
Multiple Races	0 (0.0%)	2 (4.8%)
Omitted Responses	0 (0.0%)	1 (2.4%)

Chi-Square analyses were also utilized to compare marital status of participants across recruitment groups and MDD history. Given the predominately never married/single sample (n = 53, 96.4%), marital status categories were collapsed into two dichotomous groups [i.e., never married/single, other (i.e., married, divorced)] to meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5). Results indicated that the community group had significantly more marital status diversity (i.e., married, divorced) than participants recruited from the subject pool who were predominately never married/single [χ^2 (1) = 10.61, p = .001; Table 21]. This significant difference was also observed by MDD history, with those having a history of MDD reporting greater marital status diversity (i.e., married, divorced) than those without a history of MDD [χ^2 (1) = 6.71, p = .010; Table 22].

Table 21. *Hypothesis Two and Exploratory Hypotheses Sample: Marital Status by Recruitment Group*

Marital Status	Subject Pool (n =46)	Community (n = 9)
Never Married/Single	46_a (100.0%)	7_b (77.8%)
Married	0 (0.0%)	1 (11.1%)
Divorced	0 (0.0%)	1 (11.1%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Table 22. *Hypothesis Two and Exploratory Hypotheses Sample: Marital Status by MDD History*

Marital Status	MDD History (n =13)	No MDD History (n = 42)
Never Married/Single	11_a (84.6%)	42_b (100%)
Married	1 (7.7%)	0 (0.0%)
Divorced	1 (7.7%)	0 (0.0%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Chi-Square analyses were used to compare educational level of participants across recruitment groups. To meet the Chi-square assumption of expected frequencies (i.e., frequencies in each cell should be greater than 5), educational level was collapsed into two groups (i.e., high school, greater than high school). As seen in the sample used to test Hypothesis 1, results suggested that community members had significantly higher education levels beyond high school than Psychology subject pool participants [χ^2 (1) = 11.72, p = .001; Table 23]. However, there again was no significant difference in educational levels between those with and without a history of MDD [χ^2 (1) = 1.77, p = .183; Table 24].

Table 23. *Hypothesis Two and Exploratory Hypotheses Sample: Education Level by Recruitment Group*

Education	Subject Pool (n = 46)	Community (n = 9)
High School	33_a (71.7%)	1_b (11.1%)
1 Year College or Technical School	10 (21.7%)	2 (22.2%)
2+ Years College without Degree	3 (8.5%)	2 (22.2%)
4 Years College with Degree	0 (0.0%)	2 (22.2%)
Postgraduate MD, Ph.D.	0 (0.0%)	2 (22.2%)

Note. Values with different subscripts are statistically significant at $p < .05$.

Table 24. *Hypothesis Two and Exploratory Hypotheses Sample: Education Level by MDD History*

Education	MDD History (n = 13)	No MDD History (n = 42)
High School	6 (46.2%)	28 (66.7%)
1 Year College or Technical School	2 (15.4%)	10 (23.8%)
2+ Years College without Degree	1 (7.7%)	4 (9.5%)
4 Years College with Degree	2 (15.4%)	0 (0.0%)
Postgraduate MD, Ph.D.	2 (15.4%)	0 (0.0%)

Questionnaire and physiological data. During Session 1, participants completed five self-report measures for Hypothesis 2 analyses: the BDI-II (Beck et al., 1996b; Appendix G) to evaluate depressive symptoms experienced during the previous two weeks (total score range = 0-63), the BES (Gormally et al., 1982; Appendix G) to assess current binge eating severity (total score range = 0-42), the PANAS-X (Watson & Clark, 1994; Appendix G) to evaluate negative affect over the previous two weeks (total score range = 5-50), Pearlin's Perceived Mastery Scale (PC; Pearlin & Schooler, 1978; Appendix G) to assess perceived control (total score range = 0-42), and the PSS-10 (Cohen & Williamson, 1988; Appendix G) to evaluate perceived stress over the past month (total score range = 0-40). During Session 2, electrocardiography (ECG) and impedance cardiography data was collected and amplified with Mindware hardware and Biolab 3.1 (2009) acquisition software at a sampling rate of 1000 Hz. Following electrode placement for physiological data collection, participants watched a 10-minute neutral travel video to become acclimated to electrode placement and allow physiological responses to normalize and establish baseline physiological functioning (Kolbeinsson, 2016).

Prior to analyses, data were inspected for potential univariate outliers, defined as z-scores exceeding ± 3.0 (Daszykowski et al., 2007). Outlier data for binge eating severity (n = 1), negative affect (n = 1), and depressive mood (n = 1) were winsorized to address extreme values.

Biolab software was utilized to clean and calculate HR-HRV/RSA data and parameters. HF-HRV/RSA was derived utilizing Mindware's HRV module following manual artifact editing of the digital recording of inter-beat intervals. A Fast Fourier Transform was then used to derive HF-HRV frequency band distribution within 0.15 and 0.4 Hz. Due to the inclusion of impedance cardiography, the impact of respiration rate was assessed to generate HF-HRV/RSA values. Baseline HF-HRV/RSA was calculated by averaging the final 5 minutes of HF-HRV/RSA data during the 10-minute neutral travel video following electrode placement.

Descriptive statistics for Hypothesis 2 questionnaires and physiological data are presented in Table 25. Univariate analyses of variance (ANOVA) indicated that means did not represent ceiling or floor values that could restrict regression analyses. Additionally, standard deviations suggested adequate variability for hypothesis testing. All questionnaires demonstrated adequate internal consistency (α = .710 to .897) in this study sample.

Table 25. *Hypothesis Two Sample: Questionnaire and Baseline HF-HRV/RSA Descriptive Statistics*

Measure	*M* (*SD*) (*N* = 55)	Sample Range	Sample α
Baseline HF-HRV/RSA	5.93 (1.08)	3.62-9.44	-
BDI-II	4.94 (5.64)	0-23	.892
BES	7.03 (6.28)	0-26	.897
PANAS-X Negative Affect Subscale	15.96 (5.04)	10-31	.838
PC	33.13 (5.22)	18-42	.710
PSS-10	12.94 (5.99)	1-27	.814

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.

Questionnaires and baseline HF-HRV/RSA by recruitment group. Independent *t*-tests revealed that there was no difference in perceived stress between participants recruited from the University of Maine Psychology subject pool and those from the surrounding community [*t*(52)

= .553, p = .283, Cohen's d = 0.41]. However, community members reported significantly more severe depressive symptoms [$t(53)$ = -2.69, p = .010, Cohen's d = 0.85], binge eating severity [$t(52)$ = -4.21, p < .001, Cohen's d = 1.23], and negative affect [$t(53)$ = -3.03, p = .004, Cohen's d = 0.92], and significantly less perceived control [$t(52)$ = 2.12, p = .039, Cohen's d = 0.65] than subject pool participants. Despite these findings, there was no significant difference in mean baseline HF-HRV/RSA [$t(53)$ = -.32, p = .749, Cohen's d = 0.11]. Means and standard deviations by recruitment group are presented in Table 26.

Table 26. *Hypothesis Two Sample: Questionnaire and Baseline HF-HRV/RSA Means and Standard Deviations by Recruitment Group*

Measure	Subject Pool *M* (*SD*) (*n* = 46)	Community *M* (*SD*) (*n* = 9)
Baseline HF-HRV/RSA	5.81 (1.04)	5.94 (1.31)
BDI-II	3.67_a (4.70)	8.67_b (6.87)
BES	4.33_a (3.48)	10.56_b (6.29)
PANAS-X Negative Affect Subscale	14.67_a (3.72)	19.22_b (5.91)
PC	34.04_a (4.23)	30.44_b (6.56)
PSS-10	11.93 (5.51)	14.11 (5.09)

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
Means with different subscripts are statistically significant at p < .05.

Questionnaires and baseline HF-HRV/RSA by MDD history. Independent *t*-tests revealed that participants without a history of MDD reported higher levels of perceived control than those with a history of MDD [$t(52)$ = -4.14, p < .001, Cohen's d = 1.24]. Those with a history of MDD reported more severe depressive symptoms [$t(53)$ = 5.23, p < .001, Cohen's d = 1.32], negative affect [$t(53)$ = 4.47, p < .001, Cohen's d = 1.23], perceived stress [$t(52)$ = 3.88, p < .001, Cohen's d = 1.26], and binge eating symptom severity [$t(52)$ = 5.89, p < .001, Cohen's d = 1.62] than those without a history of MDD. However, there was no significant difference in

mean baseline HF-HRV/RSA [$t(53) = -.59$, $p = .556$, Cohen's $d = 0.18$] between participants with and without a history of MDD. Means and standard deviations by history of MDD are presented in Table 27.

Table 27. *Hypothesis Two Sample: Questionnaire and Baseline HF-HRV/RSA Means and Standard Deviations by MDD History*

Measure	MDD History *M* (*SD*) (*n* = 13)	No MDD History *M* (*SD*) (*n* = 42)
Baseline HF-HRV/RSA	5.67 (1.26)	5.88 (1.03)
BDI-II	10.08_a (7.16)	2.76_b (3.18)
BES	10.54_a (5.08)	3.73_b (3.06)
PANAS-X Negative Affect Subscale	19.54_a (5.30)	14.14_b (3.24)
PC	29.23_a (4.92)	34.78_b (3.97)
PSS-10	16.85_a (4.59)	10.85_b (4.92)

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
Means with different subscripts are statistically significant at $p \leq .001$.

Primary Analyses

Primary Hypothesis 2 states that perceived control will significantly predict HF-HRV/RSA such that higher perceived control will be associated with more adaptive (i.e., higher) baseline HF-HRV/RSA. If data are consistent with Hypothesis 2, this will be due in part to higher perceived control being predictive of reduced perceived stress, decreased depressive symptomatology, and less negative affect, which will then be predictive of higher baseline HF-HRV/RSA. Questionnaire data from Session 1, and baseline HF-HRV/RSA from Session 2 were utilized to investigate these associations.

In keeping with the directional relations predicted in Hypothesis 2, Pearson correlation analyses revealed that perceived stress (PSS-10), depressive symptoms (BDI-II), and negative mood (PANAS-X Negative Affect Subscale) were significantly and positively correlated with

binge eating severity (all *r*'s > .35, all *p*'s < .009; Table 28), indicating that higher reports of stress, depressive symptoms, and negative affect were related to greater reports of binge eating symptom severity. Perceived stress, depressive symptoms, and negative affect shared significant positive correlations (all *r*'s > .43, all *p*'s ≤ .001), suggesting that more symptom severity in one stress or mood area was associated with greater symptom severity in all stress and mood domains. Significant negative correlations were observed between perceived control and perceived stress (r = -.54, p < .001), depressive symptoms (r = -.36, p = .008), and negative mood (r = -.43, p = .001), suggesting that consistent with hypotheses, those reporting greater perceived control experienced less severe perceived stress, depressive symptoms, and negative affect. Perceived control and binge eating severity were significantly and negatively associated (r = -.43, p = .001), again suggesting that those reporting higher rates of perceived control also reported less severe binge eating severity. However, perceived control was not significantly associated with baseline HF-HRV/RSA as anticipated (r = .08, p = .558). In fact, baseline HF-HRV/RSA was not significantly correlated with any Hypotheses 2 variables (all *r*'s < .08, all *p*'s > .558), including binge eating severity (r = .00, p = .98). This finding contradicts expectations and earlier research suggesting that individuals with binge eating symptomatology may have significantly less adaptive baseline physiological function (Klatzkin et al., 2015).

Table 28. *Hypothesis Two Sample: Correlations Between Self-Report Questionnaires and Baseline HF-HRV/RSA*

Measure	1.	2.	3.	4.	5.	6.
1. Baseline HF-HRV/RSA	-					
2. BDI-II	-.03	-				
3. BES	.00	.61**	-			
4. PANAS-X Negative Affect Subscale	-.08	.43*	.35*	-		
5. PC	.08	-.36*	-.43*	-.43*	-	
6. PSS-10	.01	.59**	.48**	.51**	-.54**	-

Note. BDI-II = Beck Depression Inventory-II, BES = Binge Eating Scale, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10.
* $p < .01$, ** $p < .001$.

In addition to Pearson correlation analyses, questionnaire data tests of multicollinearity were performed to investigate VIF and tolerance to ensure that variables were not too closely related or redundant for regression analyses. It is generally accepted that a tolerance value less than 0.1 indicates redundancy of IVs, and that a VIF greater than 3 is indicative IVs being too closely related to assess their independent effects on DVs (Tabachnick & Fidell, 2013). Total scores for IVs of depressive symptoms (BDI-II), negative affect (PANAS-X Negative Affect Subscale), perceived control (PC), and perceived stress (PSS-10) were examined with baseline HF-HRV/RSA as the DV. There was no evidence of multicollinearity (all VIF's < 2.02, all tolerance >.49; Table 29).

Table 29. *Hypothesis Two Sample: Tests of Multicollinearity for Self-Report Questionnaires and Baseline HF-HRV/RSA*

Questionnaire	VIF	Tolerance
BDI-II	1.58	.63
PANAS-X Negative Affect Subscale	1.46	.69
PC	1.47	.68
PSS-10	2.02	.49

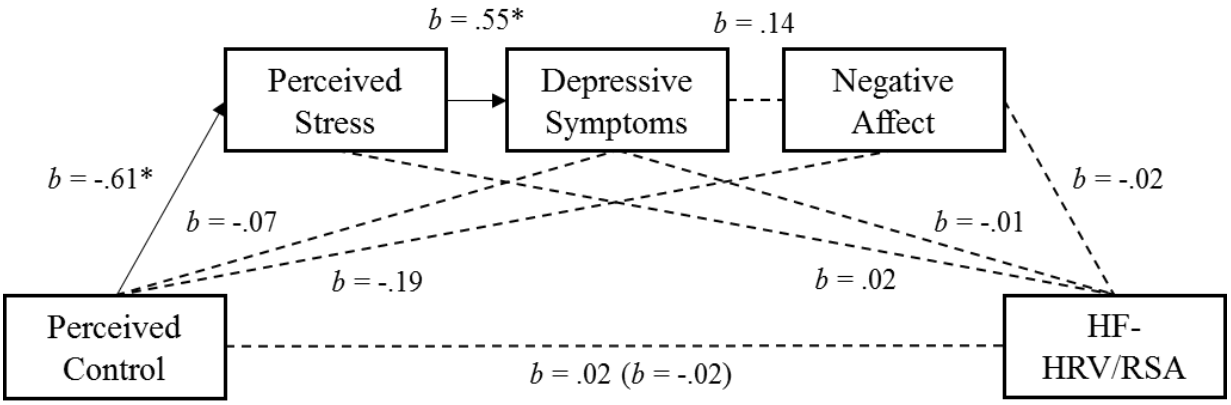
Note. Baseline HF-HRV/RSA entered as dependent variable in all multicollinearity analyses. BDI-II = Beck Depression Inventory-II, PANAS-X = Positive and Negative Affect Schedule – Expanded Form, PC = Pearlin's Perceived Mastery Scale, PSS-10 = Perceived Stress Scale-10, VIF = Variance Inflation Factors.

Serial mediated regression. Serial multiple mediator regression with bootstrap estimation (5000 samples) with PROCESS for SPSS (Hayes, 2014) was utilized to test the hypothesis that greater self-reported perceptions of control would predict more adaptive (i.e., higher) baseline HF-HRV/RSA, and that this connection would be significantly mediated by perceived stress, depressive symptoms, and negative mood.

Contrary to expectations, the association between perceived control and baseline HF-HRV/RSA was not significant (total effect b = -.02; BC 95% CI: -.04, .08; Figure 6 for full model), indicating that higher perceived control was not predictive of more adaptive (i.e., higher) baseline HF-HRV/RSA. As observed in Hypothesis 1 analyses, higher rates of perceived control predicted less perceived stress (b = -.61, p < .001). However, higher rates of perceived control were again not significantly predictive of lower depressive symptoms (b = -.07, p = .662) or less negative affect (b = -.19, p = .145). Once entered in the serial multiple mediator model [R^2 = .02, $F(4,49)$ = .20, p = .936], the relation between perceived control and baseline HF-HRV/RSA continued to be not significant (direct effect b = .02; BC 95% CI: -.06, .10), and depressive symptoms (b = -.01, p = .894), perceived stress (b = .02, p = .570), and negative affect (b = -.02, p = .611) did not significantly predict baseline HF-HRV/RSA.

Serial multiple mediator modeling is best selected for analyses when it is assumed that mediator variables are significantly correlated. This assumption was partially supported as results revealed that greater perceived stress predicted increased depressive symptomatology (b = .55, p < .001), but depressive symptoms did not predict negative mood as anticipated (b = .14, p = .256). However, the serial mediation of perceived stress and depressive symptoms was not significantly associated with HF-HRV/RSA (specific indirect effect in serial b = .00; BC 95% CI: -.02, .03, Figure 6 for full model).

Figure 6. *Hypothesis Two Serial Multiple Mediation of Perceived Control and HF-HRV/RSA*



Note. Solid arrows represent significant relations between variables. Hypothesized multiple mediator model is not significant [R^2 = .02, F(4,49) = .20, p = .936].
*p < .001.

Post-hoc power analyses. Given the unexpected reduction in sample size due to psychophysiological equipment malfunction (resulting in N = 55), *post-hoc* power analyses were performed utilizing observed results to determine the sample size needed to reach an 80% chance of detecting a medium effect size (i.e., R^2 = .13 or f^2 = .15). Power analyses using G*Power 3.1.2 program (Faul et al., 2007) revealed that the current model only had a power of .09, suggesting there was only a 9% chance of detecting a medium effect size. Further analyses suggested a sample of at least 92 participants would be required to achieve 80% power in

detecting a medium sized effect. If the most complex variation of this model is considered (i.e., 4 IVs and 6 possible interactions), a sample of 119 would be necessary for detecting a medium effect size with 80% power. However, the current findings suggest there may be a small effect in the current model (i.e., $R^2 = .02$). When the prior power analyses were repeated with this small effect size value, a sample of 635 or 806 participants would be required to achieve 80% power, respectively.

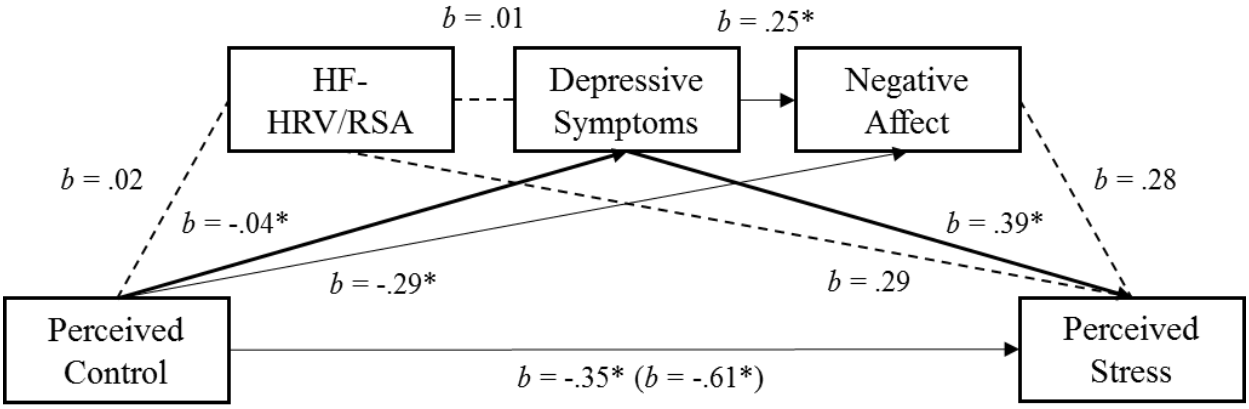
Sex moderated serial mediated regression. Hypothesis 2 serial multiple mediated regression was further investigated to examine possible moderating effects of sex. Results revealed there was no significant impact of sex on the associations between perceived control and perceived stress (index = .00, BC 95% CI: -.01, .04), perceived control and depressive symptoms (index = -.00, 95% CI: -.03, .02), or perceived control and negative affect (index = .00, BC 95% CI: -.03, .02). Similarly, sex did not significantly moderate relations between perceived stress and baseline HF-HRV/RSA (index = .01, BC 95% CI: -.08, .12), depressive symptoms and baseline HF-HRV/RSA (index = .03, BC 95% CI: -.09, .17), or negative affect and baseline HF-HRV/RSA (index = .05, BC 95% CI: -.03, .18). Results suggest that the associations between perceived control, perceived stress, depressive symptoms, negative affect, and HF-HRV/RSA do not significantly differ between males and females.

History of MDD moderated serial mediated regression. A follow-up analysis was conducted to better understand if a history of MDD moderated Hypothesis 2 serial multiple mediated regression. Results indicate that history of MDD does not significantly moderate associations between perceived control and mediator variables (all index's <.03, all BC 95% CI's between -.09, .06), or mediator variables and baseline HF-HRV/RSA (all index's <.05, all BC 95% CI's between -.15, .28). Results suggest that a history of MDD does not significantly

impact the relations between perceived control, perceived stress, depressive symptoms, negative affect, and HF-HRV/RSA.

Alternative serial mediated regression. In mediated regression analyses, outcomes can cause mediators and therefore, it is advisable to provide additional evidence for model fit by investigating reverse casual mediation by interchanging mediator and outcomes variables (Judd & Kenny, 2010). Serial multiple mediator modeling was repeated to investigate mediators as outcome variables. When entered as an outcome variable, perceived stress was significantly predicted by perceived control (total effect b = -.61; BC 95% CI: -.88, -.34; Figure 7 for full model), such that higher perceived control was predictive of less perceived stress. There was significant serial multiple mediator model fit [R^2 = .51, F(4,49) = 12.68, p < .001] and the relation between perceived control and perceived stress remained significant (total effect b = -.35; BC 95% CI: -.61, -.09). Although significant associations were observed between perceived control and depressive symptoms (b = -.02, p = .008) and negative affect (b = -.29, p = .019), the relation with HF-HRV/RSA remained not significant (b = .02, p = .558). In fact, HF-HRV/RSA was not significantly associated with depressive symptoms (b = .01, p = .984), negative affect (b = -.16, p = .708), or perceived stress (b = .29, p = .562; Figure 7 for full model).

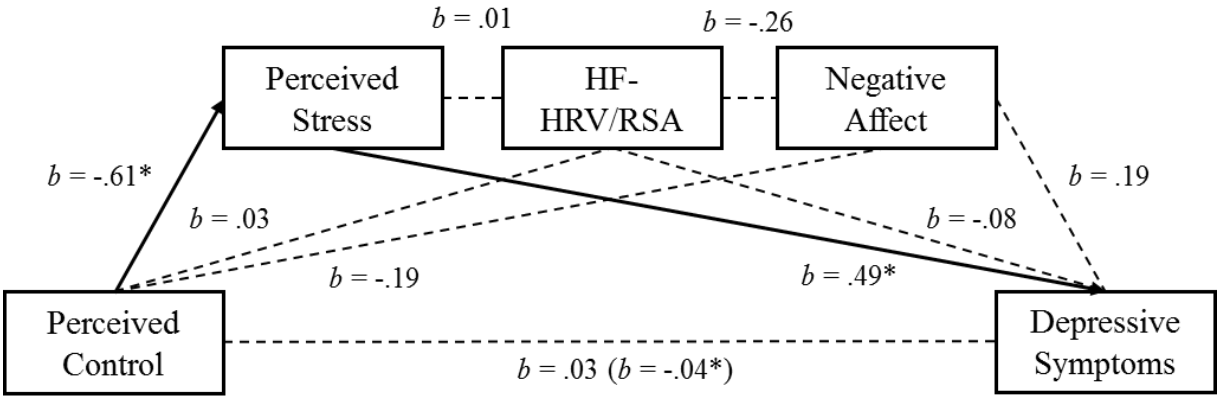
Figure 7. *Hypothesis Two Alternative Model: Serial Multiple Mediation of Perceived Control and Perceived Stress*



Note. Bold arrows represent the significant mediation of depressive symptoms on the relation between perceived control and perceived stress (specific indirect effect b = -.16; BC 95% CI: -.35, -.06). Significant alternative multiple mediator model [R^2 = .51, F(4,49) = 12.68, p < .001]. *p < .05.

Depressive symptoms were then investigated as an outcome variable. Perceived control significantly predicted depressive symptoms (total effect b = -.40; BC 95% CI: -.69, -.11; Figure 8 for full model), such that higher perceived control predicted less depressive symptoms severity. There was significant serial multiple mediator model fit [R^2 = .37, F(4,49) = 7.14, p < .001] and the association between perceived control and perceived stress did not remain significant (total effect b = -.02; BC 95% CI: -.34, .28). The relation between perceived control and depressive symptoms was significantly mediated by perceived stress (specific indirect effect b = -.30; BC 95% CI -.70, -.09), suggesting that greater perceived control is predictive of less perceived stress and that this reduction is predictive of reduced depressive symptom severity. In this model, HF-HRV/RSA again did not share significant associations with perceived control (b = .03, p = .475), perceived stress (b = .01, p = .676), negative affect (b = -.26, p = .587), or depressive symptoms (b = -.08, p = .894; Figure 8 for full model).

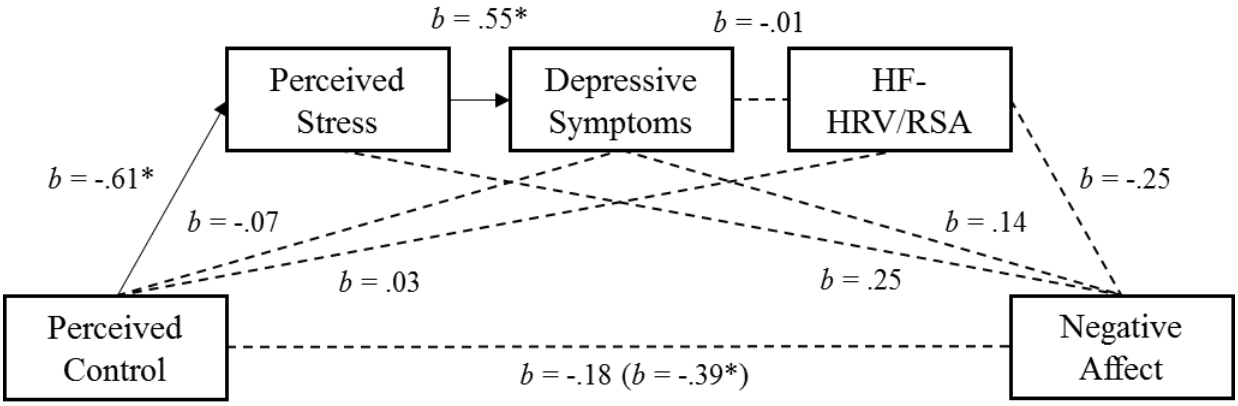
Figure 8. *Hypothesis Two Alternative Model: Multiple Mediation of Perceived Control and Depressive Symptoms*



Note. Bold arrows represent the significant mediation of perceived on the relation between perceived control and depressive symptoms (specific indirect effect b = -.30; BC 95% CI: -.70, -.09). Significant alternative multiple mediator model [R^2 = .37, F(4,49) = 7.14, p < .001]. *p < .05.

The final mediator explored as an outcome variable was negative affect. Perceived control significantly predicted negative affect (total effect b = -.39; BC 95% CI: -.62, -.16; Figure 9 for full model), such that higher perceived control predicted less negative affect. There was significant serial multiple mediator model fit [R^2 = .32, F(4,49) = 5.71, p = .001] and the relation between perceived control and perceived stress did not remain significant (total effect b = -.18; BC 95% CI: -.44, .07). There were no significant serial indirect effects, and HF-HRV/RSA was not significantly associated with perceived stress (b = .02, p = .640), depressive symptoms (b = -.01, p = .826), perceived control (b = .02, p = .489), or negative affect (b = -.25, p = .611; Figure 9 for full model).

Figure 9. *Hypothesis Two Alternative Model: Multiple Mediation of Perceived Control and Negative Affect*



Note. Solid arrows represent significant relations between variables. Significant alternative multiple mediator model [R^2 = .32, F(4,49) = 5.71, p = .001].
*p < .05.

Hypothesis Three

Preliminary Analyses

Hypothesis 3 through Hypothesis 8 are exploratory in nature. These hypotheses are designed to further investigate possible associations between perceived control, binge eating severity, mood reactivity, and cardiovascular function in individuals who undergo experimental neutral or negative mood induction. Sample characteristics are the same as those presented in Hypothesis 2 analyses (N = 55 participants). Participants were predominately female (n = 49, 65.3%), European American (n = 50, 90.9%), never married/single (n = 53, 96.4%) and high school educated (n = 34, 61.8%) with a mean age of 20.22 years (SD = 6.43, range = 18-60), height of 68.18 inches (SD = 3.30, range = 61.00-78.00), weight of 155.31 pounds (SD = 22.63, range = 117.00-210.00) and BMI of 23.47 (SD = 2.77, range = 18.60-29.11). Subject pool participants made up 83.6% (n = 46) of the sample, 13 participants (23.6%) had a history of an MDD episode at least 6 months before study participation, and 26 (47.3%) were randomized to

the negative mood induction group during Session 2 of the study. Descriptive statistics are presented in Table 16.

Questionnaire and physiological data. During Session 1, participants completed self-report measures for exploratory hypothesis analyses: the BES (Gormally et al., 1982; Appendix G) to assess current binge eating severity (total score range = 0-42) and Pearlin's Perceived Mastery Scale (PC; Pearlin & Schooler, 1978; Appendix G) to assess perceived control (total score range = 0-42). During Session 2, electrocardiography (ECG) and impedance cardiography data was collected and amplified with Mindware hardware and Biolab 3.1 (2009) acquisition software at a sampling rate of 1000 Hz. Following electrode placement for physiological data collection, participants watched a 10-minute neutral travel video to become acclimated to electrode placement and allow physiological responses to normalize and establish baseline physiological functioning (Kolbeinsson, 2016). After this baseline period, participants completed visual analog scales (VAS) of happy mood, sad mood, and stress (Appendix H). Importantly, for sad mood and stress VAS ratings, lower scores indicated greater levels of each mood state, while higher scores indicated less or no presence of sad mood or stress. Randomized participants then either completed experimental negative mood induction (i.e., negative emotional Stroop task followed by 8-minute non-lyrical piece of classical music by Prokofiev entitled "Russia under the Mongolian Yoke" played a half-speed while thinking of a particularly sad life event; e.g., Segal et al., 2006) or neutral mood induction (i.e., neutral Stroop task followed by 8-minute non-lyrical piece of classical music from a selection of Chopin played at half-speed while remembering an uneventful day; e.g., Green et al., 2003). Following mood induction, participants repeated VAS ratings before a 10-minute physiological recovery period

while viewing a second neutral travel video. VAS ratings were completed once more, and physiological recording continued throughout baseline, mood induction, and recovery periods.

Mood and stress reactivity and recovery were calculated using pre-mood induction, post-mood induction, and post recovery VAS scores for both negative and neutral mood induction groups. Prior to further analyses, these data were inspected for potential univariate outliers, defined as z-scores exceeding ± 3.0 (Daszykowski et al., 2007). Outlier data for sad mood recovery (n = 1), stress reactivity (n = 1), stress recovery (n = 2), and happy mood recovery (n = 1) were winsorized to address extreme values.

Biolab software was utilized to clean and calculate HR-HRV/RSA data and parameters. HF-HRV/RSA was derived utilizing Mindware's HRV module following manual artifact editing of the digital recording of inter-beat intervals. A Fast Fourier Transform was then used to derive HF-HRV frequency band distribution within 0.15 and 0.4 Hz. Due to the inclusion of impedance cardiography, the impact of respiration rate was assessed to generate HF-HRV/RSA values. Baseline HF-HRV/RSA was calculated by averaging the final 5 minutes of HF-HRV/RSA data during the 10-minute neutral travel video following electrode placement. Mood induction HF-HRV/RSA was calculated by averaging the final five minutes of HF-HRV/RSA data during 8-minute neutral or negative mood induction task. Recovery period HF-HRV/RSA was calculated by averaging the final 5 minutes of HF-HRV/RSA data during the 10-minute neutral travel video following mood induction. These values were utilized to calculate HF-HRV/RSA reactivity (i.e., the difference between baseline and mood induction mean values) and HF-HRV/RSA recovery (i.e., the difference between baseline and recovery period mean values). Outlier data for physiological reactivity (n = 1) and recovery (n = 1) were winsorized to address extreme values.

Descriptive statistics for exploratory hypothesis questionnaires and physiological data are presented in Table 30. Univariate analyses of variance (ANOVA) indicated that means did not represent ceiling or floor values that may restrict regression analyses. Additionally, standard deviations suggested adequate variability for hypothesis testing. All questionnaires demonstrated adequate internal consistency (α = .725 to .930) in this study sample.

Table 30. *Exploratory Hypotheses Sample: Questionnaire and Physiological Data Descriptive Statistics*

Measure	*M* (*SD*) (*n* = 55)	Sample Range	Sample α
BES	7.03 (6.28)	0-26	.897
PC	33.13 (5.22)	18-42	.710
VAS Sad Reactivity	13.00 (20.68)	-19-71	.903
VAS Stress Reactivity	5.71 (16.67)	-45-60	.903
VAS Happy Reactivity	-8.75 (18.88)	-60-29	.735
VAS Sad Recovery	-8.25 (18.16)	-67-38	.820
VAS Stress Recovery	-3.39 (13.30)	-37-40	.930
VAS Happy Recovery	3.91 (18.19)	-50-51	.725
HF-HRV/RSA Reactivity[a]	.05 (.56)	-1.70-1.50	-
HF-HRV/RSA Recovery[a]	.06 (.42)	-.77-1.30	-

Note. BES = Binge Eating Scale, PC = Pearlin's Perceived Mastery Scale.
[a] Across negative and neutral mood induction groups; means and standard deviations by group are presented in Table 34.

Questionnaire and physiological data by recruitment group. As previously reported, independent *t*-tests revealed that community members reported significantly more binge eating severity [$t(52) = -4.21$, $p < .001$, Cohen's $d = 1.23$], and significantly less perceived control [$t(52) = 2.12$, $p = .039$, Cohen's $d = 0.65$] than subject pool participants. Despite these findings, there was no significant difference in sad mood [$t(45) = .67$, $p = .507$, Cohen's $d = 0.27$], stress [$t(46) = -.87$, $p = .391$, Cohen's $d = 0.29$], or happy mood reactivity [$t(42) = -.89$, $p = .380$, Cohen's $d = 0.41$]. Similarly, there was no significant difference between groups for sad mood [$t(46) = -.87$, $p = .388$, Cohen's $d = 0.26$], stress [$t(44) = -.57$, $p = .569$, Cohen's $d = 0.22$], or

happy mood recovery scores [$t(43) = .13$, $p = .895$, Cohen's $d = 0.05$]. There was also no significant difference in HF-HRV/RSA reactivity [$t(53) = 1.72$, $p = .091$, Cohen's $d = 0.58$] or recovery [$t(52) = -.03$, $p = .980$, Cohen's $d = 0.43$] between community and Psychology subject pool participants. Means and standard deviations by recruitment group are presented in Table 31.

Table 31. *Exploratory Hypotheses Sample: Questionnaire and Physiological Data Means and Standard Deviations by Recruitment Group*

Measure	Subject Pool *M* (*SD*) (*n* = 46)	Community *M* (*SD*) (*n* = 9)
BES	4.33_a (3.48)	10.56_b (6.29)
PC	34.04_a (4.23)	30.44_b (6.56)
VAS Sad Reactivity	13.85 (20.64)	8.14 (21.85)
VAS Stress Reactivity	4.78 (15.28)	10.38 (23.08)
VAS Happy Reactivity	-9.94 (20.24)	-3.38 (10.08)
VAS Sad Recovery	-9.28 (14.97)	-3.13 (30.47)
VAS Stress Recovery	-3.87 (12.95)	-.71 (15.99)
VAS Happy Recovery	4.08 (18.63)	3.13 (17.18)
HF-HRV/RSA Reactivity[a]	.10 (.52)	-.24 (.65)
HF-HRV/RSA Recovery[a]	.08 (.43)	.09 (.45)

Note. BES = Binge Eating Scale, PC = Pearlin's Perceived Mastery Scale.
Means with different subscripts are statistically significant at $p < .05$.
[a] Across negative and neutral mood induction groups; means and standard deviations by group are presented in Table 34.

Questionnaire and physiological data by MDD history. As previously reported, independent *t*-tests revealed that participants with a history of MDD reported lower levels of perceived control [$t(52) = -4.14$, $p < .001$, Cohen's $d = 1.24$] and more severe binge eating symptomatology [$t(52) = 5.89$, $p < .001$, Cohen's $d = 1.62$] than those without a history of MDD. However, there was no significant difference in sad mood [$t(45) = 1.07$, $p = .291$, Cohen's $d = 0.34$], stress [$t(46) = 1.66$, $p = .103$, Cohen's $d = 0.49$], or happy mood reactivity [$t(42) = -.28$, $p = .780$, Cohen's $d = 0.09$]. Similarly, there was no significant difference between groups for sad

mood [$t(46) = .31$, $p = .759$, Cohen's $d = 0.09$], stress [$t(44) = .95$, $p = .347$, Cohen's $d = 0.32$], or happy mood recovery scores [$t(43) = -.49$, $p = .620$, Cohen's $d = 0.17$]. Results revealed that those with a history of MDD had significantly lower (i.e., less adaptive) HF-HRV/RSA reactivity [$t(53) = -2.74$, $p = .008$, Cohen's $d = 0.57$], but there was no difference between groups for HF-HRV/RSA recovery [$t(52) = -1.02$, $p = .315$, Cohen's $d = 0.18$]. Means and standard deviations by history of MDD are presented in Table 32.

Table 32. *Exploratory Hypotheses Sample: Questionnaire and Physiological Data Means and Standard Deviations by MDD History*

Measure	MDD History *M* (*SD*) (*n* = 13)	No MDD History *M* (*SD*) (*n* = 42)
BES	10.54_a (5.08)	3.73_b (3.06)
PC	29.23_a (4.92)	34.78_b (3.97)
VAS Sad Reactivity	18.82 (24.51)	11.22 (19.40)
VAS Stress Reactivity	12.15 (20.98)	3.31 (14.37)
VAS Happy Reactivity	-10.00 (17.19)	-8.23 (19.79)
VAS Sad Recovery	-6.83 (25.49)	-8.72 (15.41)
VAS Stress Recovery	-.25 (12.85)	-4.50 (13.47)
VAS Happy Recovery	1.77 (14.42)	4.78 (19.66)
HF-HRV/RSA Reactivity[a]	$-.30_a$ (.73)	$.15_b$ (.45)
HF-HRV/RSA Recovery[a]	-.03 (.42)	.12 (.43)

Note. BES = Binge Eating Scale, PC = Pearlin's Perceived Mastery Scale.
Means with different subscripts are statistically significant at $p < .01$.
[a] Across negative and neutral mood induction groups; means and standard deviations by group are presented in Table 34.

Questionnaire and physiological data correlations. Pearson correlation analyses revealed that binge eating severity and perceived control were significantly and negatively correlated ($r = -.43$, $p = .001$), suggesting that those reporting greater perceived control also reported less severe binge eating symptoms. Sad mood reactivity shared significant positive correlations with stress reactivity ($r = .38$, $p = .009$) and happy mood recovery ($r = .47$, $p = .001$) in both negative and neutral mood inductions, suggesting that those who demonstrated sad mood

change following experimental mood induction also experienced changes in stress levels following the experimental task, and changes in happy mood following 10-minute recovery. Greater changes in happy mood following negative and neutral mood induction had a significant negative correlation with sad mood after mood induction (r = -.66, p < .001), but a significant positive correlation with changes in happy mood following recovery (r = .41, p = .007). This suggests that those who experienced changes in happy mood after both negative and neutral mood induction also reported less change in sad mood following experimental tasks, and continued to experience happy mood state change following experimental task recovery. However, significant negative correlations between happy mood state post-recovery and happy mood reactivity (r = -.52, p < .001), sad mood recovery (r = -.67, p < .001), and stress recovery (r = -.49, p = .001) indicate that those who experienced greater changes in happy mood following recovery also reported less happy mood changes after mood induction, and less sad mood and stress change post-recovery. Those reporting greater stress recovery change also reported little stress reactivity to mood induction (r = -.39, p = .008), but indicated greater sad mood changes post-recovery (r = .37, p = .014). A significant negative correlation was observed between HF-HRV/RSA reactivity and sad mood reactivity (r = -.43, p = .002) and HF-HRV/RSA recovery (r = .48, p < .001), such that greater physiological reactivity was associated with less change in sad mood following mood induction and greater HF-HRV/RSA change post-recovery. Correlations for measures used in exploratory hypothesis analyses are presented in Table 33.

Table 33. *Exploratory Hypotheses Sample: Questionnaire and Physiological Data Correlations*

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. BES	-									
2. PC	-.43*	-								
3. VAS Sad Reactivity	.04	.00	-							
4. VAS Stress Reactivity	.04	-.04	.38*	-						
5. VAS Happy Reactivity	.08	-.06	-.66*	-.07	-					
6. VAS Sad Recovery	-.05	.04	-.73	-.24	.41*	-				
7. VAS Stress Recovery	.03	-.27	-.13	-.39*	.07	.37*	-			
8. VAS Happy Recovery	-.11	.13	.47*	.24	-.52*	-.67*	-.49*	-		
9. HRV Reactivity	-.03	-.00	-.18	-.43*	.08	.13	.06	-.14	-	
10. HRV Recovery	.07	-.07	.04	-.14	-.18	.17	.11	-.24	.48*	-

Note. BES = Binge Eating Scale, HRV = HF-HRV/RSA, PC = Pearlin's Perceived Mastery Scale.
* $p \leq .01$.

Primary Analyses

Exploratory Hypothesis 3 predicted that participants randomized to the negative mood induction group would demonstrate greater sad mood and stress reactivity during Session 2 than those assigned to the neutral mood induction group. Hypothesis 3 was partially supported as independent *t*-tests revealed that those in the negative mood induction group reported significantly greater sad mood reactivity following mood induction than those in the neutral condition [$t(45) = -3.28$, $p = .002$, Cohen's $d = 0.94$]. However, analyses indicated there was no significant difference in stress reactivity between groups [$t(46) = -.76$, $p = .454$, Cohen's $d = 0.22$].

Questionnaire and physiological data by experimental group. To better understand differences between experimental groups, additional independent *t*-tests were conducted. Analyses indicated there were no significant differences between groups in Session 1 ratings of perceived control [$t(52) = .31$, $p = .756$, Cohen's $d = 0.08$] or binge eating severity [$t(52) = .45$, $p = .659$, Cohen's $d = 0.12$]. Those in the negative experimental group reported greater reductions

in happy mood following mood induction [$t(42) = 2.30$, $p = .026$, Cohen's $d = 0.69$], and experienced greater reductions in sad mood [$t(46) = 3.44$, $p < .001$, Cohen's $d = 0.98$] and increases in happy mood [$t(43) = -3.95$, $p < .001$, Cohen's $d = 1.18$] following 10-minute recovery period than those in the neutral mood induction group. There were again no significant differences between mood induction groups for stress recovery [$t(44) = 1.81$, $p = .078$, Cohen's $d = 0.54$]. There was no significant difference observed between HF-HRV/RSA reactivity to mood induction [$t(53) = .44$, $p = .663$, Cohen's $d = 0.12$] or HF-HRV/RSA recovery after 10-minute rest period [$t(52) = -.27$, $p = .790$, Cohen's $d = 0.32$]. Means and standard deviations by experimental group are presented in Table 34.

Table 34. *Exploratory Hypotheses Sample: Questionnaire and Physiological Data Means and Standard Deviations by Experimental Mood Induction Group*

	Negative Mood Induction (n = 26)	Neutral Mood Induction (n = 29)
Measure	*M* (*SD*)	*M* (*SD*)
BES	5.64 (5.31)	5.08 (3.88)
PC	33.64 (4.95)	33.23 (4.75)
VAS Sad Reactivity	4.92_a (15.34)	23.00_b (22.35)
VAS Stress Reactivity	3.96 (21.18)	7.61 (9.82)
VAS Happy Reactivity	-2.50_a (16.03)	-15.00_b (19.77)
VAS Sad Recovery	$-.77_a$ (12.28)	-17.09_b (20.18)
VAS Stress Recovery	-.08 (14.41)	-7.00 (11.21)
VAS Happy Recovery	-5.17_a (14.69)	13.41_b (16.81)
HF-HRV/RSA Reactivity	.08 (.65)	.01 (.44)
HF-HRV/RSA Recovery	.10 (.44)	.07 (.43)

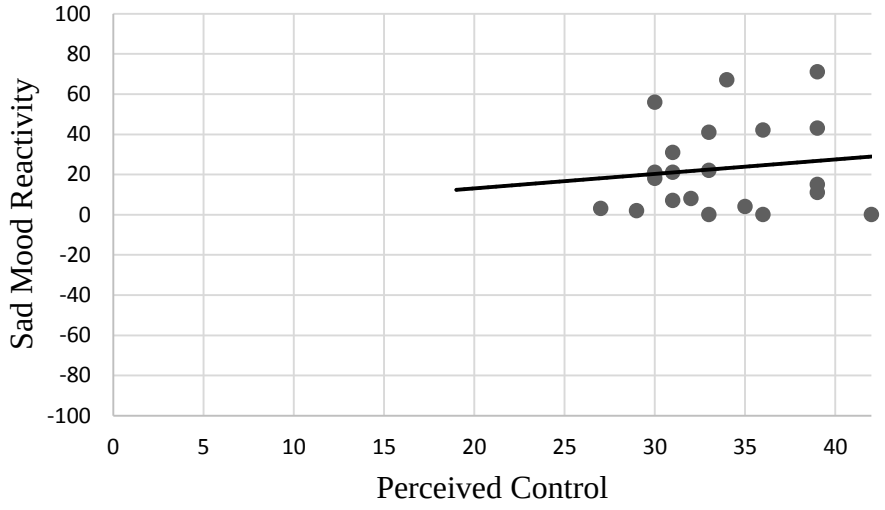
Note. BES = Binge Eating Scale, PC = Pearlin's Perceived Mastery Scale.
Means with different subscripts are statistically significant at $p < .01$.

Hypothesis Four

Primary Analyses

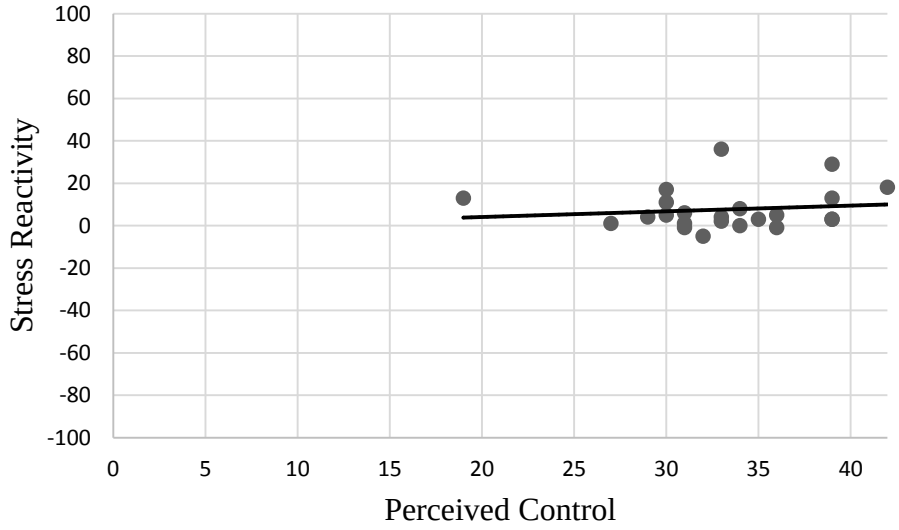
Exploratory Hypothesis 4 was intended to further investigate the relation between perceived control reported during Session 1 and state changes in sad mood and stress following negative mood induction during Session 2. It was expected that those reporting higher rates of perceived control would experience less mood and stress reactivity than individuals reporting lower rates of perceived control. Pearson correlation analyses did not support this hypothesis, and there were no significant correlations between perceived control and sad mood reactivity (r = .13, p = .573; Figure 10), or stress reactivity (r = .13, p = .541; Figure 11) in the negative mood induction group. Exploratory analyses were conducted to inspect the relation between perceived control and positive affect ratings. There was no significant association between perceived control and happy mood reactivity (r = -.33, p = .133; Figure 12) in the negative mood induction group. Further exploratory analyses indicated there was also no significant relations between perceived control and sad mood recovery (r = .18, p = .412), stress (r = -.12, p = .596), or happy mood recovery (r = -.09, p = .691) in the negative mood induction group after a 10-minute rest period.

Figure 10. *Correlation Between Perceived Control and Sad Mood Reactivity Following Negative Mood Induction*



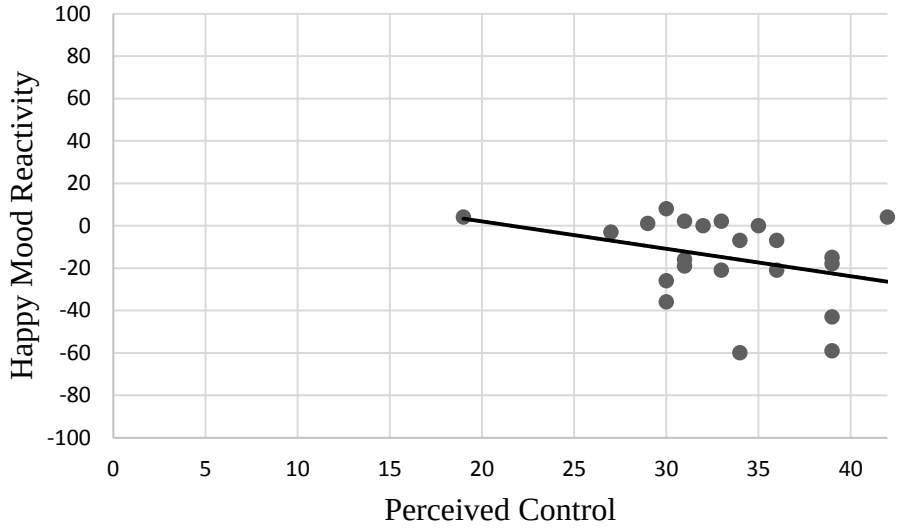
Note. No significant correlation between self-reported perceived control and changes in sad mood following negative mood induction (r = .13, p = .573).

Figure 11. *Correlation Between Perceived Control and Stress Reactivity Following Negative Mood Induction*



Note. No significant correlation between self-reported perceived control and changes in stress following negative mood induction (r = .13, p = .541).

Figure 12. *Correlation Between Perceived Control and Happy Mood Reactivity Following Negative Mood Induction*



Note. No significant correlation between self-reported perceived control and changes in happy mood following negative mood induction (r = -.33, p = .133).

Hypothesis Five

Primary Analyses

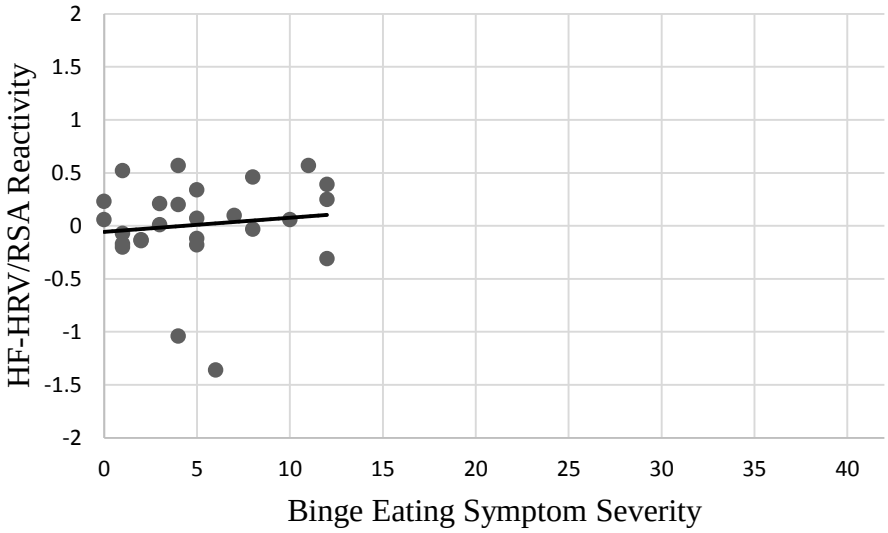
Changes in HF-HRV/RSA following negative mood induction were further investigated in exploratory Hypothesis 5. Greater HF-HRV/RSA reactivity was anticipated in the negative mood induction group than the neutral mood induction group. However, independent *t*-tests did not support this hypothesis, and there were no significant differences in HF-HRV/RSA reactivity between experimental mood induction conditions [$t(53)$ = .44, p = .663, Cohen's d = 0.12; means and standard deviations by experimental group are presented in Table 34].

Hypothesis Six

Primary Analyses

Exploratory Hypothesis 6 was intended to further explore the relation between binge eating severity reported during Session 1 and HF-HRV/RSA reactivity following negative mood induction during Session 2. It was expected that those reporting more severe binge eating symptoms would demonstrate less adaptive (i.e., lower) HF-HRV/RSA reactivity during Session 2 negative mood induction than participants reporting less severe binge eating symptoms. Pearson correlation analysis did not support this hypothesis, and there was no significant correlation between binge eating severity and HF-HRV/RSA reactivity following negative mood induction (r = .12, p = .565; Figure 13). Exploratory analyses were conducted to inspect the association between binge eating severity and HF-HRV/RSA recovery following 10-minute rest period post negative mood induction. There was no significant relation between binge eating severity and HF-HRV/RSA recovery (r = .07, p = .645) in the negative mood induction group.

Figure 13. *Correlation Between Binge Eating Severity and HF-HRV/RSA Reactivity Following Negative Mood Induction*



Note. No significant correlation between self-reported binge eating symptom severity and changes in HF-HRV/RSA following negative mood induction (r = .12, p = .565).

Hypothesis Seven

Primary Analyses

Exploratory Hypothesis 7 states that perceived control will significantly predict HF-HRV/RSA such that higher perceived control will be associated with more adaptive (i.e., higher) HF-HRV/RSA reactivity during negative mood induction. If data are consistent with Hypothesis 7, this will be due in part to higher perceived control being predictive of reduced stress reactivity and less sad mood reactivity, which will predict higher HF-HRV/RSA reactivity. Questionnaire data for perceived control from Session 1, and VAS stress, VAS sad mood, and HF-HRV/RSA reactivity following negative mood induction during Session 2 were utilized to investigate these associations.

Data underwent tests of multicollinearity to investigate VIF and tolerance to ensure that variables were not too closely related or redundant for regression analyses. It is generally accepted that a tolerance value less than 0.1 indicates redundancy of IVs, and that a VIF greater than 3 is indicative IVs being too closely related to assess their independent effects on DVs (Tabachnick & Fidell, 2013). Perceived control (PC), VAS stress reactivity, and VAS sad mood reactivity were examined with HF-HRV/RSA reactivity as the DV. Results indicated no evidence of multicollinearity (all VIF's < 1.25, all tolerance >.80; Table 35).

Table 35. *Hypothesis Seven Sample: Tests of Multicollinearity for Self-Report Measures and HF-HRV/RSA Reactivity*

Measure	VIF	Tolerance
PC	1.08	.93
VAS Stress Reactivity	1.25	.80
VAS Sad Reactivity	1.18	.85

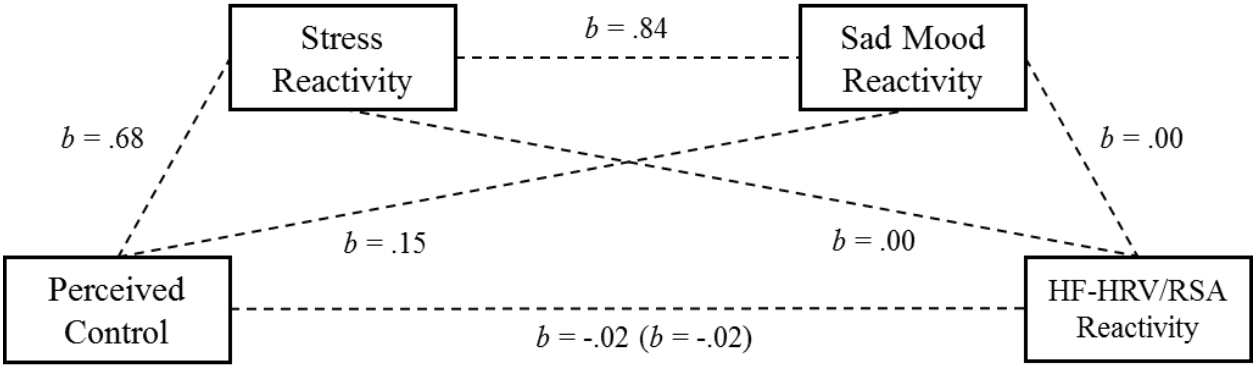
Note. HF-HRV/RSA reactivity entered as dependent variable in all multicollinearity analyses. PC = Pearlin's Perceived Mastery Scale, VIF = Variance Inflation Factors.

Serial mediated regression. Serial multiple mediator regression with bootstrap estimation (5000 samples) with PROCESS for SPSS (Hayes, 2014) was utilized to test the hypothesis that greater self-reported perceptions of control would predict more adaptive (i.e., higher) HF-HRV/RSA reactivity, and that this association would be significantly mediated by changes in stress and sad mood following negative mood induction.

Contrary to expectations, the association between perceived control and HF-HRV/RSA reactivity was not significant (total effect b = -.02; BC 95% CI: -.06, .03; Figure 14 for full model), indicating that higher perceived control was not predictive of more adaptive (i.e., higher) HF-HRV/RSA reactivity. In fact, self-reported perceived control was not significantly associated with stress reactivity (b = .68, p = .238) or sad mood reactivity (b = .15, p = .904) following negative mood induction. Once entered in the serial multiple mediator model [R^2 = .06, F(3,17) = .34, p = .798], the relation between perceived control and HF-HRV/RSA reactivity continued to be not significant (direct effect b = -.02; BC 95% CI: -.06, .03), and neither stress reactivity (b = .00, p = .749) nor sad mood reactivity (b = .00, p = .694) were predictive of HF-HRV/RSA reactivity.

Serial multiple mediator modeling is best selected for analyses when it is assumed that mediator variables are significantly correlated. This assumption was not supported as stress reactivity was not significantly associated with sad mood reactivity following negative mood induction (b = .84, p = .107). Therefore, the serial mediation of stress and sad mood reactivity was not significantly associated with HF-HRV/RSA reactivity (specific indirect effect in serial b = .00; BC 95% CI: -.00, .02, Figure 14 for full model).

Figure 14. *Hypothesis Seven Serial Multiple Mediation of Perceived Control and HF-HRV/RSA*



Note. Hypothesized multiple mediator model is not significant [R^2 = .06, F(3,17) = .34, p = .798].

Importantly, Hypothesis 7 model fit did not improve when baseline HF-HRV/RSA was entered as a covariate [R^2 = .13, F(4,22) = .83, p = .523], indicating that non-significant findings were not due to limited HF-HRV/RSA reactivity variability due to high initial baseline HF-HRV/RSA values. This was further investigated with baseline HF-HRV/RSA as a moderator between mediators and outcome variables, and again, model fit did not improve [R^2 = .13, F(3,23) = 1.15, p = .351].

Post-hoc power analyses. Due to psychophysiological equipment malfunction and associated reduction in sample size (resulting in N = 55, n = 26 in negative mood induction condition), *post-hoc* power analyses were performed utilizing observed results to determine the sample size needed to reach an 80% chance of detecting a medium effect size (i.e., R^2 = .13 or f^2 = .15). Power analyses using G*Power 3.1.2 program (Faul et al., 2007) revealed that the current model only had a power of .13, suggesting there was only a 13% chance of detecting a medium effect size. Further analyses suggested a sample of at least 85 participants would be required to achieve 80% power in detecting a medium sized effect. If the most complex variation of this model is considered (i.e., 3 IVs and 3 possible interactions), a sample of 98 would be necessary

for detecting a medium effect size with 80% power. However, the current findings suggest there may be a small effect in the current model (i.e., $R^2 = .06$). When the prior power analyses were repeated with this small effect size value, a sample of 192 or 220 participants would be required to achieve 80% power, respectively.

Sex moderated serial mediated regression. Hypothesis 7 serial multiple mediated regression was further investigated to examine possible moderating effects of sex. Results revealed there was no significant impact of sex on the associations between perceived control and stress reactivity (index = .00, BC 95% CI: -.01, .06) or sad mood reactivity (index = .00, BC 95% CI: -.06, .01) following negative mood induction. Similarly, sex did not significantly moderate relations between stress reactivity and HF-HRV/RSA reactivity (index = .00, BC 95% CI: -.02, .12) or sad mood reactivity and HF-HRV/RSA reactivity (index = .00, BC 95% CI: -.02, .14) following negative mood induction. Results suggest that the associations between perceived control, stress reactivity, sad mood reactivity, and HF-HRV/RSA reactivity do not significantly differ between males and females.

History of MDD moderated serial mediated regression. A follow-up analysis was conducted to better understand if a history of MDD moderated Hypothesis 7 serial multiple mediated regression. Results indicate that history of MDD does not significantly moderate associations between perceived control and mediator variables (all index's <.02, all BC 95% CI's between -.10, .26), or mediator variables and HF-HRV/RSA reactivity (all index's <.00, all BC 95% CI's between -.04, .10). These findings suggest that a history of MDD does not significantly impact the relations between perceived control, stress reactivity, sad mood reactivity, and HF-HRV/RSA.

Alternative serial mediated regression. In mediated regression analyses, outcomes can cause mediators and therefore, it is advisable to provide additional evidence for model fit by investigating reverse casual mediation by interchanging mediator and outcomes variables (Judd & Kenny, 2010). However, given that the hypothesized mediation model was not significant (Figure 14), correlations do not support significant associations between variables (Table 33), and changing the direction of possible relations of outcome and mediator variables is not supported by the literature (e.g., depression associated with lower HRV reactivity to negative mood induction, Kemp et al., 2010), alternative serial mediated regression models were not explored.

Hypothesis Eight

Primary Analyses

Exploratory Hypothesis 8 states that the hypothesized relations between perceived control, stress and sad mood reactivity, and HF-HRV/RSA reactivity following negative mood induction investigated in Hypothesis 7 will be moderated by binge eating severity. It was proposed that more severe binge eating symptoms would moderate the relation between stress and sad mood reactivity such that greater binge eating would be associated with reduced (i.e., less adaptive) HF-HRV/RSA reactivity. Questionnaire data for perceived control and binge eating severity from Session 1, and VAS stress, VAS sad mood, and HF-HRV/RSA reactivity following negative mood induction during Session 2 were utilized to investigate these associations.

Data underwent tests of multicollinearity to investigate VIF and tolerance to ensure that variables were not too closely related or redundant for regression analyses. It is generally accepted that a tolerance value less than 0.1 indicates redundancy of IVs, and that a VIF greater

than 3 is indicative IVs being too closely related to assess their independent effects on DVs (Tabachnick & Fidell, 2013). Perceived control (PC), binge eating severity (BES), VAS stress reactivity, and VAS sad mood reactivity were examined with HF-HRV/RSA reactivity as the DV. Results indicated no evidence of multicollinearity (all VIF's < 1.26, all tolerance >.79; Table 36).

Table 36. *Hypothesis Eight Sample: Tests of Multicollinearity for Self-Report Measures and HF-HRV/RSA Reactivity*

Measure	VIF	Tolerance
BES	1.07	.94
PC	1.15	.87
VAS Stress Reactivity	1.26	.79
VAS Sad Reactivity	1.19	.84

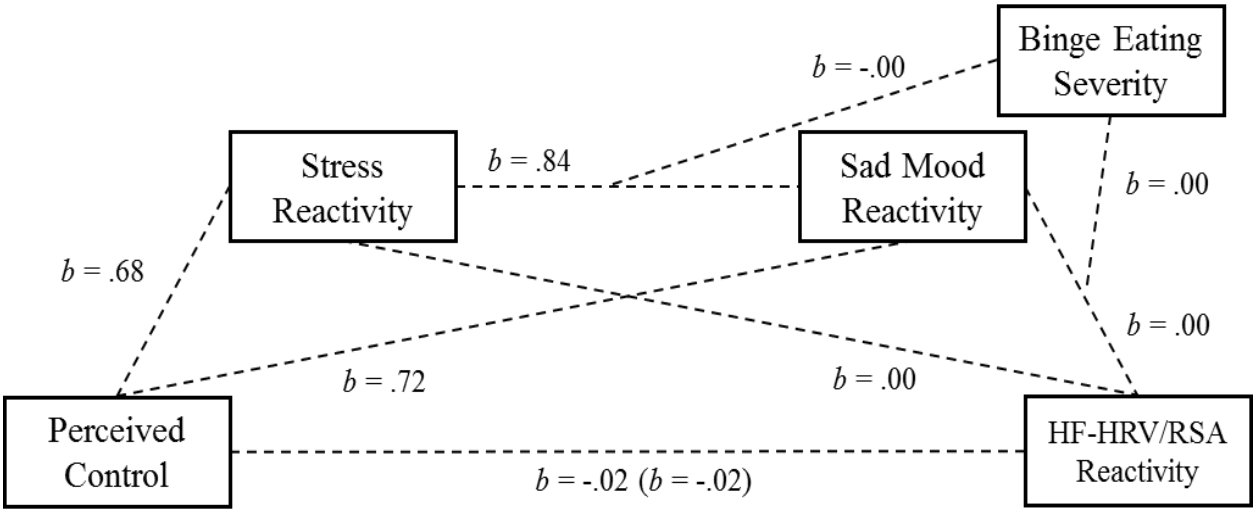
Note. HF-HRV/RSA reactivity entered as dependent variable in all multicollinearity analyses. BES = Binge Eating Scale, PC = Pearlin's Perceived Mastery Scale, VIF = Variance Inflation Factors.

Serial mediated regression. Serial multiple mediator regression with bootstrap estimation (5000 samples) with PROCESS for SPSS (Hayes, 2014) was utilized to test the hypothesis that greater self-reported perceptions of control would predict more adaptive (i.e., higher) HF-HRV/RSA reactivity, and that this connection would be significantly mediated by changes in stress and sad mood reactivity, and that this would be significantly moderated by binge eating severity.

Analyses revealed that the previous model tested in Hypothesis 7 remained not significant [R^2 = .18, $F(6,14)$ = .51, p = .788], and there were no significant associations between variables (Figure 15 for full model). Contrary to expectations, Session 1 self-reported binge eating severity did not significantly moderate the associations between stress reactivity and HF-HRV/RSA reactivity (index = -.00; BC 95% CI: -.02, .00) or sad mood reactivity and HF-HRV/RSA reactivity (index = .00; BC 95% CI: -.01, .01; Figure 15). Exploratory analyses were

conducted to investigate if binge eating severity significantly moderated relations between predictor and mediator variables. Results revealed that binge eating severity did not significantly impact the relation between perceived control and stress reactivity (index = -.00; BC 95% CI: -.01, .00) or mood reactivity (index = .00; BC 95% CI: -.00, .01).

Figure 15. *Hypothesis Eight Moderated Serial Multiple Mediation of Perceived Control and HF-HRV/RSA*



Note. Hypothesized multiple mediator model is not significant [R^2 = .18, F(6,14) = .51, p = .788].

Post-hoc power analyses. Given the reduction in sample size due to psychophysiological equipment malfunction (resulting in N = 55, n = 26 in negative mood induction condition), *post-hoc* power analyses were performed utilizing observed results to determine the sample size needed to reach an 80% chance of detecting a medium effect size (i.e., R^2 = .13 or f^2 = .15). Power analyses using G*Power 3.1.2 program (Faul et al., 2007) revealed that the current model had a power of .25, suggesting there was only a 25% chance of detecting a medium effect size. Further analyses suggest that a sample of at least 104 participants would be required to achieve 80% power in detecting a medium sized effect. If the most complex variation of this model is

considered (i.e., 4 IVs and 7 possible interactions), a sample of 123 would be necessary for detecting a medium effect size with 80% power.

CHAPTER FOUR

DISCUSSION

The present study set out to accomplish three primary aims. The first was to expand the current knowledge base on binge eating symptomatology to frequently neglected samples (i.e., subthreshold binge eating behaviors, males, normal to overweight populations). Both clinical and subthreshold binge eating is associated with elevated rates of functional impairment, psychiatric and medical comorbidity, and emotional distress (e.g., Stice et al., 2009). Unlike other eating disordered behaviors (e.g., restricting, purging), binge eating appears to be more equivalent across sex (APA, 2013). Additionally, while the prevalence of binge eating disorder (BED) increases with increased BMI, obesity is not a diagnostic feature of this disorder (APA, 2013). Approximately 35% of those who regularly engage in binge eating fall into the obese weight range (Corwin et al., 2011), yet historically, research has often been limited to obese, female samples meeting BED diagnostic criteria. This narrow focus negatively impacts the generalizability of findings and highlights a significant gap in our understanding of binge eating among subclinical, non-obese individuals (e.g., Carrard et al., 2012).

The second principal aim was to investigate the predictive role of perceived control in binge eating severity. Theoretical models and subsequent research on the maintenance of binge behaviors have focused primarily on the impact of stress and negative affect. Both variables have proved to precede binge behaviors in both clinical (e.g., Munsch et al., 2012) and community populations (e.g., Womble et al., 2001). However, research indicates that these antecedents may not consistently trigger binge eating (e.g., Munsch et al., 2008) and suggest that additional variables may be impacting these associations. Preliminary findings from earlier research indicate that perceived control may predict binge eating severity in diverse populations

(Goetze et al., in preparation, 2018a) and could serve as a protective buffer against commonly cited antecedents to binge eating behavior, including stress and negative affect (Goetze et al., in preparation, 2018b). Although higher perceived control has been linked to less severe anxiety and depressive symptoms (e.g., Gallagher et al. 2014) and is amenable to therapeutic intervention (e.g., van der Zanden et al. 2014), there has been a lack of research investigating the possible relations between perceived control, elevated perceived stress and negative mood states, and binge eating severity.

The third aim of this study was to explore the role of perceived control and binge eating severity on mood reactivity, and possible concomitants with reduced cardiovascular function as measured by high-frequency heart rate variability/respiratory sinus arrhythmia (HF-HRV/RSA). A growing area of investigation has focused on the role of binge eating in increased cardiovascular disease risk. Research findings suggest that binge eating is significantly associated with cardiovascular disease (e.g., Mitchell, 2016), and although preliminary, research focusing on cardiac wellness has found that women with BED may be at greater risk for cardiovascular disease independent of obese weight status, with significantly blunted HRV (Friederich et al., 2006; Ranzenhofer et al., 2016). One limitation is that these investigations have relied upon obese samples, which have previously been identified as exhibiting less adaptive HRV both at rest and under mental challenge (e.g., Laederach-Hofmann et al., 2000), potentially confounding obese binge eating findings. In addition, physiological studies have neglected to consistently consider the potential impact depressive symptoms may have on HRV. As the most prevalent comorbid lifetime diagnosis for individuals with binge eating disorder (Dingemans et al., 2009), major depressive disorder (MDD) has also been found to reduce cardiovascular reactivity (e.g., Kemp et al., 2010), and further complicates the interpretation of

HRV findings among obese individuals with binge eating behaviors. At this time, there has been no investigation into HRV in normal to overweight individuals reporting binge eating symptomology. Finally, although higher perceived control has been linked to decreased cardiovascular disease and mortality (Surtees et al., 2010), little research has examined the potential associations between perceived control, binge eating severity and cardiovascular health status (Roepke & Grant, 2011).

Perceived Control and Binge Eating Symptom Severity

As anticipated and consistent with prior preliminary findings (e.g., Goetze et al., in preparation, 2018a), perceived control was significantly associated with binge eating symptoms such that higher ratings of perceived control were predictive of less severe binge eating severity. This association was significantly mediated by perceived stress and depressive symptoms, which suggests that those with greater perceived control also experienced less perceived stress. This reduced rate of perceived stress was then associated with reduced depressive symptoms, which then significantly predicted less severe binge eating severity. These findings are in line with prior research suggesting that increased levels of perceived control can buffer individuals from stress (e.g., Bollini et al., 2004) and depressive symptoms (e.g., Gallagher et al., 2014); both of which have been posited as antecedents to binge eating behavior (e.g., Dingemans et al., 2009, Laessle & Schultz, 2009).

Surprisingly and contrary to expectations, when depressive symptoms were included in this model, ratings of negative mood states were not related to perceived control or predictive of binge eating severity as previously demonstrated (Goetze et al., in preparation, 2018b). Although prior research has suggested that higher rates of negative mood are associated with a greater loss of control when eating (e.g., Haedt-Matt & Keel, 2011), findings have been mixed

(e.g., Wegner et al., 2002), as has been the literature on the possible role of depressive symptoms. For example, in an early study of negative mood induction in obese women with BED (determined by clinical interview), no significant associations between self-reported depressive symptoms, negative affect, and binge eating were observed (Agras & Telch, 1998). Instead, findings revealed that greater negative mood in response to guided autobiographical negative mood induction increased reports of loss of control and subjective binge eating. These results lead authors to hypothesize that acute, state negative affect rather than stable, trait negative mood (i.e., depression) may lead to binge eating. However, a later study of 66 overweight and obese women with clinical and subclinical binge eating (determined by structured clinical interview) found that following negative mood induction, individuals with higher baseline trait depressive symptoms experienced greater sadness and ate significantly more calories during a subsequent taste-task (Dingemans et al., 2009). This finding was later replicated in a negative mood induction study with 75 overweight and obese women diagnosed with BED, leading authors to suggest there may be a significant interaction between both state and trait features of negative mood in the maintenance of binge eating behaviors (Dingemans et al., 2015). Findings from the current investigation suggest that in this sample, depressive symptoms better accounted for the relation between perceived control and binge eating and were more predictive of binge eating severity than ratings of negative mood states (e.g., afraid, scared, hostile, upset, distressed) over the past two weeks.

Interestingly, history of MDD did not significantly moderate the relations between perceived control, perceived stress, depressive symptoms severity, and binge eating severity. MDD is the most prevalent comorbid lifetime diagnosis in individuals with BED, and prior research has found that severity of binge eating is positively related to severity of depressive

symptomatology (Dingemans et al., 2009), with higher levels of depression associated with more severe binge eating (Dingemans et al., 2015). However, current study findings suggest that a history of clinically elevated depressive symptoms does not significantly impact the protective role of perceived control against perceived stress and depressive symptoms, although these findings warrant replication in a larger sample. The present study also suggests that symptoms of depression are predictive of binge eating, even at subclinical levels. These results highlight the importance of shifting research focus to subclinical populations, which may be especially important in binge eating samples as prior findings have suggested that subclinical binge eating is more prevalent than clinically elevated binge eating behaviors (e.g., Hudson et al., 2007). Research has also demonstrated that subclinical binge eating is associated with similar levels of functional impairment and emotional distress as symptoms consistent with BED (e.g., Stice et al., 2009), further emphasizing the need to study this population.

In addition to findings that support the importance of investigating subclinical binge eating populations, results also support the inclusion of male participants in future research. Epidemiological studies of BED have indicated that unlike previously recognized eating disorders, prevalence rates of binge eating in women and men are more equivalent (2:1, respectively; APA, 2013; Hudson et al., 2007; Mitchell, 2016), with one community study indicating that adult men are as likely to screen positive for BED symptoms as adult women (Grucza et al., 2007). Shingleton and colleagues (2015) pooled 11 randomized controlled psychosocial treatment studies for BED to investigate baseline and outcome characteristics by sex. The data indicated that men tended to report lower shape, weight, and eating concerns at baseline than women, but no significant main effect for sex on symptom severity following treatment completion was observed. However, findings revealed a significant interaction

between sex, treatment length, and shape/weight concern. Men with lower baseline shape and weight concern had a significant reduction in binge eating following shorter treatments, whereas women and men with higher baseline concerns regarding shape and weight benefited most from longer interventions. These results not only underscore the importance of considering sex when treating BED, but support increased recruitment and inclusion of men in treatment research. The present study findings that sex did not significantly mediate the significant associations between perceived control, perceived stress, depressive symptoms, and binge eating severity, suggest that these associations are universal across sex. Therefore, these relations may have utility as future treatment targets despite potential differences in baseline and treatment characteristics unique to each sex.

Interestingly, alternate model testing suggested significant serial mediation such that higher perceived control predicted less binge eating severity, which led to less severe depressive symptoms. These depressive symptoms were then linked to less negative affect, which was predictive of reduced perceived stress. Although this model does not significantly test any of the primary aims of this study (i.e., the predictive role of perceived control in binge eating severity), it does suggest a reciprocal association between variables supported by prior research. For example, low perceived control has not only been linked to psychiatric symptoms such as anxiety and depression (e.g., Rivard & Cappeliez, 2007), but has been found to mediate relations between stress and psychological distress (Rosenbaum et al., 2012). A study investigating stress over a 16-month treatment period revealed that high perceived stress was associated with three times greater binge eating frequency than low perceived stress (Pendleton et al., 2001). However, given the correlational design of this study, the authors were unable to determine if greater perceived stress led to increases in binge eating, or if increases in binge behavior

preceded increased perceived stress. A later study utilizing a ten-year longitudinal design determined that participants who reported elevated perceived stress prior to 14 years of age were at significantly greater risk for developing binge eating behaviors consistent with BED, bulimia, and subclinical binge eating (Striegel-Moore et al., 2007). These findings suggest that although the causal association between perceived stress and binge eating severity remain in question, preliminary evidence suggests that perceived stress may precede the onset of binge eating symptomatology (Striegel-Moore et al., 2007); a finding supported by the present study's model of perceived control predicting binge eating severity due in part to the mediating impact of perceived stress.

Perceived Control and Baseline HF-HRV/RSA

Contrary to expectations, the association between perceived control and baseline resting HF-HRV/RSA was not significant, indicating that higher perceived control was not predictive of more adaptive (i.e., higher) HF-HRV/RSA. This finding was somewhat unexpected given prior research suggesting that greater perceived control has been associated with improved recovery in cardiac surgery patients (e.g., Dracup et al., 2003) and demonstrated to have an inverse relation with cardiac disease and related death (Roepke & Grant, 2011). For example, in a prospective population-based study of over 19,000 men and women, aged 41 to 80 with no previous heart disease, it was found that low self-reported perceived control at initial assessment was associated with increased risk of cardiovascular disease related mortality over longitudinal follow-up (Surtees et al., 2010). Importantly, this association remained significant after controlling for biological-based conditions (e.g., diabetes), lifestyle (e.g., BMI), socioeconomic (e.g., social class), and psychosocial risk factors (e.g., MDD episode within the past year), such that there was over a 10% increased risk of cardiovascular disease mortality with each standard deviation

decrease in baseline self-reports of perceived control. Despite these past findings, there was no evidence of perceived control being significantly associated with baseline HF-HRV/RSA in the present study, indicating that the relation between perceived control and this marker of cardiovascular health remains unclear.

Results did not support the hypothesized mediating impact of perceived stress, depressive symptoms and negative affect on perceived control and baseline HF-HRV/RSA. In fact, none of these variables were significantly associated with resting HF-HRV/RSA despite prior research indicating significant differences in blood pressure, perceived stress and depressive symptoms between obese women with BED and both obese and normal-weight women without BED during a 10-minute relaxation period following physiological hookup (Klatzkin et al., 2015). These prior findings led researchers to conclude there was evidence that women with BED may have heightened resting physiological and psychological dysfunction independent of obesity. It may be that the present findings indicate that depressive symptoms and perceived stress are not related to physiological dysfunction in a subclinical, normal to overweight, binge eating sample; however, it is also important to note that null findings may also be the result of underpowered analyses (N = 55) and a sample size too small to detect these associations. Post-hoc power analyses revealed that the current model only had a 9% chance of detecting a medium effect size, supporting the likelihood of underpowered analyses. Furthermore, current model findings suggest a small effect size between variables (R^2 = .02), indicating a sample of at least 635 participants would be necessary to detect possible significant associations.

Relations between perceived control, perceived stress, depressive symptoms, negative affect, and HF-HRV/RSA were not significantly moderated by sex. This too is surprising given a recent meta-analysis investigating sex differences in the autonomic control of the heart,

specifically measured by HRV in healthy human participants (Koenig & Thayer, 2016). Results indicated that although females may have less power in LF-HRV bands, they typically demonstrate higher HF-HRV, indicating that autonomic control of the female heart may be characterized by greater parasympathetic activity. Therefore, it would be expected that in the present study, sex would potentially moderate the connections between HF-HRV/RSA and both predictive and mediating variables. However, it is important to note that the most recent evidence for the significant difference in HRV by sex was determined by pooling data on 63,612 participants (31,970 females), and therefore, the present study likely does not have the necessary power to detect this difference.

History of MDD did not significantly moderate associations between perceived control, perceived stress, depressive symptoms, negative affect, and HF-HRV/RSA. This finding is contrary to expectations given that MDD has been consistently connected with cardiovascular disease and mortality (e.g., York et al., 2007), and associated with blunted heart rate reactivity (e.g., Jin et al., 2015). Given the frequent comorbidity of MDD and BED, and meta-analytic findings that depression (without comorbid cardiovascular disease) appears to be significantly associated with blunted HRV both at rest and during 24-hour Holter monitoring with more severe depressive symptoms demonstrating greater reductions in HRV (Kemp et al., 2010), it would not be surprising to find a significant association between history of clinically elevated MDD and baseline HF-HRV/RSA. However, similar to earlier null findings of moderation by sex, it is likely that the present study is too underpowered to detect possible baseline HF-HRV/RSA differences by MDD history.

Binge Eating Severity and Baseline HF-HRV/RSA

In the present study, there was no significant correlation between binge eating severity and baseline resting HF-HRV/RSA. To date, preliminary investigations exploring the relation between binge eating severity and physiological functioning have resulted in mixed findings. Friederich and colleagues (2006) did not find baseline differences in HRV among obese women with BED and obese women without BED. However, when Klatzkin and colleagues (2015) investigated cardiovascular reactivity in obese BED, obese non-BED, and normal weight non-BED women in response to the Trier Social Stress Test, they found significant baseline differences in blood pressure, depressive symptoms, and perceived stress between obese women with BED and both obese and normal-weight women without BED. This study contradicted earlier findings by Freiderich and colleagues (2006) and concluded that obese women with BED had heightened physiological and psychological dysfunction independent of obesity and acute mental status. While binge eating symptoms were not associated with baseline HF-HRV/RSA in the present study, most participants reported binge eating severity falling within the "minimal" range. Despite participants reporting enough variability for analyses, the sample's skew towards the minimal binge eating severity range may have limited analyses. Therefore, the nature of the association between binge eating severity and baseline HF-HRV/RSA remains uncertain.

Stress and Mood Reactivity

Consistent with expectations and prior research findings, participants randomized to undergo transient negative mood induction utilizing music and autobiographic recall experienced greater sad mood reactivity than individuals randomized to a neutral mood induction condition (e.g., Segal et al., 2006). Results further suggested specificity in producing sad mood states following mood induction, as stress reactivity did not significantly differ between groups.

However, greater reductions in happy mood following negative mood induction were observed, which is consistent with literature suggesting that low positive affective states are characterized by sadness and lethargy, and is important to consider given that low positive mood may precede binge eating episodes (e.g., Munsch et al., 2012).

Perceived Control, Stress Reactivity, and Mood Reactivity

Despite significant associations between perceived control, perceived stress, and depressive symptoms in earlier analyses from the present study, there was no significant correlation between perceived control and sad mood or stress reactivity following negative mood induction. However, it is important to note that while prior analyses in this investigation included negative affect (i.e., subjective distress and mood states that include anger, contempt, disgust, guilt, fear, and nervousness; Watson, Clark, & Tellegen, 1988) as a potential mediator, this and subsequent analyses in the current study rely on reports of sad mood which may be better characterized as low positive affect. Due to this, additional exploratory analyses were conducted and indicated that there were no significant correlations between perceived control and happy mood reactivity. Further analyses revealed no significant associations between perceived control and sad mood, happy mood, or stress recovery following 10-minute rest period. These findings are contrary to research suggesting that perceived control buffers individuals from daily stress and negative affect, and has been linked to less severe anxiety and depressive symptoms (e.g., Drewelies et al., 2018; Gallagher et al., 2014). As such, this may be an artifact of low power (n = 26 randomized to negative mood induction group) associated with these exploratory analyses.

Negative Mood Induction and HF-HRV/RSA Reactivity

Results indicated that there were no significant differences in HF-HRV/RSA reactivity between participants randomized to undergo negative mood induction and individuals randomized to the neutral mood induction group. These findings are unexpected given that the cardiovascular system is highly sensitive to neurobehavioral processes and is impacted by psychological factors such as stress, depression, and emotional arousal consistent with transient mood induction techniques (Berntson et al., 2007), and that HRV of healthy individuals naturally decreases under situations of emotional or physical stress but increases during times of rest (Dekker et al., 2000). Given this study's earlier findings that sad mood significantly increased in the negative mood induction group when compared to individuals undergoing neutral mood induction, and that reductions in HF-HRV have been observed during transient negative mood inductions as short as 3-minutes in length (Strange, Hamilton, Fresco, & Alloy, 2017), it is unlikely that this null finding is due to an unsuccessful negative mood induction. These analyses were characterized as exploratory in nature *a priori* due to expected low sample sizes (e.g., n = 26 participants randomized to the negative mood induction group), and therefore, null findings are likely related to inadequate power rather than flawed study design or lack of significant differences in physiological reactivity between mood induction groups.

Binge Eating Symptom Severity and HF-HRV/RSA Reactivity

Exploratory hypotheses predicted that participants reporting higher binge eating severity randomized to the negative mood induction group would demonstrate significantly less adaptive (i.e., lower) HF-HRV/RSA reactivity than those reporting lower binge eating severity. This prediction was not supported as no significant correlations between self-reported binge eating severity and HF-HRV/RSA reactivity following negative mood induction were found.

Additional exploratory analyses revealed no significant correlations between binge eating severity and baseline resting HF-HRV/RSA or HF-HRV/RSA recovery following 10-minute rest period post negative mood induction. These findings are unexpected given prior preliminary research suggesting that individuals with BED demonstrate significantly blunted HF-HRV/RSA reactivity following experimental stress tasks (e.g., Freidich et al., 2006) and past literature suggesting that those with depressive symptoms (which are often comorbid with BED) demonstrate similarly attenuated HF-HRV/RSA in response to negative mood induction (e.g., Strange et al., 2017). As with previous exploratory analyses in this study, these null findings may be due in part to low sample size. However, it is also important to note that although self-reported binge eating severity did not represent floor or ceiling values, most participants reported binge eating severity falling within the "minimal" range. Despite participants reporting enough variability for analyses, the sample's skew towards the minimal binge eating severity range may be limiting these analyses.

Perceived Control and HF-HRV/RSA Reactivity

Contrary to expectations, the association between perceived control and HF-HRV/RSA reactivity to negative mood induction was not significant, indicating that higher perceived control was not predictive of more adaptive (i.e., higher) HF-HRV/RSA reactivity. This finding was somewhat unexpected given prior research suggesting that greater perceived control has been associated with improved recovery in cardiac surgery patients (e.g., Dracup et al., 2003) and demonstrated to have an inverse relation with cardiac disease and related death independent of BMI and recent history of MDD (e.g., Roepke & Grant, 2011; Surtees et al., 2010). These findings are in line with this study's prior analyses suggesting there were no significant associations between perceived control and baseline HF-HRV/RSA; however, it is unclear if

these null findings represent a true lack of significant association between variables or are better characterized as inconclusive due to their exploratory nature and low sample size. *Post-hoc* power analyses revealed that the current model only had a 13% chance of detecting a medium effect size, supporting the likelihood of underpowered analyses. Furthermore, current model findings suggest a small effect size between variables (R^2 = .06), indicating a sample of at least 192 participants would be necessary to detect possible significant associations.

Results did not support the hypothesized mediating impact of sad mood and stress reactivity during negative mood induction on perceived control and HF-HRV/RSA reactivity. In fact, neither of these variables were significantly related to HF-HRV/RSA reactivity despite prior research indicating that mental stress and negative affect have been associated with blunted heart rate reactivity following sad mood induction (e.g., Jin, Steding, & Webb, 2015). Also inconsistent with the literature, these relations did not significantly change when inspected by the possible moderating impact of sex. However, as previously discussed while evaluating the association between perceived control and baseline HF-HRV/RSA, the present study's small sample size is likely negatively impacting the ability to draw conclusions from these analyses.

History of MDD did not significantly moderate associations between perceived control, and stress, sad mood, or HF-HRV/RSA reactivity. This finding is contrary to expectations given that prior research investigating cardiovascular reactivity in obese BED, obese non-BED, and normal weight non-BED women in response to the Trier Social Stress Test found that differences in HRV between groups were better accounted for by higher rates of depression in BED individuals (Klatzkin et al., 2015). However, like earlier null findings of moderation by sex, it is likely that the present study is too underpowered to detect possible HF-HRV/RSA reactivity differences by MDD history.

Binge Eating, Perceived Control, and HF-HRV/RSA Reactivity

Contrary to expectations, exploratory analyses did not support the hypothesis that self-reported binge eating severity would significantly moderate associations between sad mood and stress reactivity and HF-HRV/RSA reactivity, such that those reporting greater binge eating severity would experience less adaptive (i.e., lower) HF-HRV/RSA reactivity in response to negative mood induction. This finding is inconsistent with prior research suggesting that individuals reporting clinical levels of binge eating symptoms demonstrate less HF-HRV/RSA reactivity than weight-matched individuals without binge eating symptoms (Freiderich et al., 2006; Klatzkin et al., 2015). Although this finding may indicate that only clinically elevated BED symptoms significantly impact markers of cardiovascular function (i.e., HF-HRV/RSA), it is also possible that the small sample size ($n = 26$) and the sample's skew towards self-reported "minimal" range of binge eating severity may be limiting these analyses. *Post-hoc* power analyses revealed that the current model only had a 25% chance of detecting a medium effect size, supporting the likelihood of underpowered analyses. Furthermore, additional analyses indicated a sample of at least 104 participants would be necessary to detect a medium effect size and possible significant associations between variables.

Implications

This study has several important implications for both research and treatment of binge eating symptomatology. Perhaps the most important finding in the current investigation is the significant predictive role of perceived control in binge eating severity, and potential treatment implications of perceived control for subclinical binge eating across BMI categories and sex. Perceived control, or one's perception of their ability to impact behavior and environment to reach desired goals, has been identified as a psychological factor that may be associated with

increased adherence to preventative health behaviors (McCaul et al., 1993), associated with better overall physical health (e.g., Taylor et al., 2000), may buffer individuals from stress exposure (e.g., Bollini et al., 2004) and is related to mental wellbeing (e.g. Skinner, 1996). The present study replicates earlier findings in a racially diverse, female, community sample suggesting that higher rates of perceived control may be predictive of less severe binge eating symptomatology in part due to the mediating role of stress and negative affective states (Goetze et al., manuscript in preparation, 2018a; Goetze et al., manuscript in preparation, 2018b), and expands these findings to include males and a normal to overweight population. The current investigation suggests that higher rates of perceived control are predictive of less severe binge eating symptom severity due to perceived control's association with less severe perceived stress and depressive symptoms, common triggers for binge eating behavior (e.g., Bollini et al., 2004; Gallagher et al., 2014).

Findings that perceived control may buffer individuals from stress and depressive symptoms and predict less severe binge eating symptoms is especially important given that perceived control is not conceptualized as a fixed personality trait, but rather an adaptive variable that can be modified through experience (Surtees et al., 2010). For example, adaptive gains in perception of control have been associated with recovery from anxiety disorders and depression following cardiac surgery (Gallagher, Naragon-Gainey, & Brown, 2014), leading past researchers to hypothesize that perceived control may be a transdiagnostic mechanism of change in anxiety and mood disorders, and predictive of positive outcomes and behavior change following CBT (Doering et al., 2015). Although further study is required to determine what elements of CBT are associated with positive change in perceived control, past findings combined with significant results from the current investigation suggest that perceived control is

not only associated with common comorbid psychiatric conditions of binge eating, but also predictive of subclinical binge eating symptoms. Additionally, perceived control may protect individuals from common affective triggers of binge eating, and has been shown to be amenable to change through therapeutic intervention.

The current investigation made efforts to include participants that are rarely recruited for studies of binge eating (e.g., normal to overweight, male, self-reported subclinical binge eating symptoms). Despite the inclusion of these new sample characteristics, predictive associations between self-reported perceived control and binge eating severity were replicated from previous research focused on female, community samples including individuals with BMI's falling in the obese range (e.g., Goetze et al., manuscript in preparation, 2018b). Results in the current investigation's sample are important for several reasons. Epidemiological studies of BED have indicated that prevalence rates of binge eating in women and men are more equivalent than found across other eating disorders (2:1, respectively; e.g., Mitchell, 2016), and prior research has suggested that lifetime prevalence estimates of BED are 3.5% in women, and 2.0% in men (Hudson et al., 2007). Although binge eating research samples are often restricted to female populations, there is evidence to suggest differences in binge eating symptoms and response to treatment across sex, suggesting that research must include males if mechanisms of binge eating and treatment approaches are to be applicable to both male and female populations (Shingleton et al., 2015). The present study's inclusion of males and the finding that sex did not moderate associations between perceived control and binge eating offers evidence that variables targeted in this study may have future treatment utility in both males and females presenting with binge eating symptoms.

The current investigation's sample is also unique in that it included only normal to overweight individuals reporting subclinical binge eating symptoms. Despite the fact that BED and binge eating is prevalent among normal and overweight populations (i.e., approximately 65% of individuals who regularly engage in binge eating are not obese; Corwin et al., 2011), clinical research investigating BED has historically been limited to obese, female samples (e.g., Carrard et al. 2012). Additionally, subclinical binge eating is commonly reported in the general population (i.e., 3% to 8% lifetime prevalence; e.g., Davis, 2015), and similar levels of functional impairment and emotional distress are routinely expressed in both individuals experiencing subthreshold and those reporting clinical-range binge behaviors (e.g., Stice et al., 2009). The current study's focus on binge eating symptoms in a non-obese, subclinical sample may be more representative of the majority of individuals reporting binge eating symptoms given that approximately 65% of those who routinely binge eat are not obese, yet report levels of eating pathology and distress that are comparable to obese women with BED (Didie & Fitzgibbon, 2005), and it is posited that subclinical binge behaviors are more prevalent than behaviors suggestive of BED (e.g., Hudson et al., 2007). The current finding that perceived control is predictive of binge eating severity in a normal to overweight subclinical sample highlights that predictors of binge eating behaviors have research and treatment utility in these otherwise understudied binge eating populations.

Although largely exploratory in nature, the current investigation highlights the importance of research on physiological markers of health in subclinical binge eating populations. Prior research has suggested that obese individuals with BED are at an increased risk for medical morbidity and mortality and have greater health-care utilization compared to BMI-matched individuals without BED (e.g., Smith & Robbins, 2013). Therefore, BED may

significantly and negatively impact health beyond the effects of obesity alone. However, despite data indicating that the minority of individuals with BED are obese (e.g., Didie & Fitzgibbon, 2005), little has been done to explore the potential maladaptive health impacts BED may have in normal to overweight individuals with binge eating behaviors (Messerli-Bürgy et al., 2010). Research examining medical risks linked with binge eating indicate that these symptoms are significantly associated with cardiovascular problems, including coronary heart disease, heart failure, and hypertension (e.g., Mitchell, 2016). Although preliminary, research focusing on cardiac wellness has found that women with BED may be at greater risk for cardiovascular disease independent of obese weight status (Friederich et al., 2006), though such findings have also been attributed to increased rates of depression symptoms in obese BED populations (Klatzkin et al., 2015). Although the present sample was likely not large enough to fully explore associations between perceived control, binge eating, and cardiovascular function as measured by HF-HRV/RSA, it took two important steps towards addressing gaps in the current literature. First, this study included only normal to overweight participants to address current recommendations that future research investigate healthy weight individuals with binge eating symptoms given that there has been no empirical study of potential cardiac health risks in this group (Klatzkin et al., 2015). Second, this study utilized structured clinical interviews to gather detailed information about current and past depressive symptoms consistent with MDD. Given the frequent comorbidity of MDD and binge eating symptoms (e.g., Dingemans et al., 2009), and research indicating that depressive symptoms can significantly impact HRV (e.g., Jin, Steding, & Webb, 2015), research aimed at investigating cardiac function in those reporting binge eating symptoms must evaluate individual differences in depression severity and investigate possible impacts on significant findings.

Study Limitations and Future Directions

Despite strengths of the current study, several limitations must be considered when interpreting results. Although attempts were made to address gaps in the current literature regarding often-neglected sample characteristics (e.g., inclusion of males, focus on non-obese, subclinical population), the participants in this study were primarily young adult (M_{age} = 21.01, *SD* = 6.79) European American college students despite efforts to recruit ages 18 to 60 from the surrounding community, and may not be representative of the U.S. population. In spite of these limitations, prior research has indicated that binge eating symptomatology may be most prevalent in adults younger than 40 years of age (Reagan & Hersch, 2005) with the first presentation of diagnostic binge eating symptoms generally occurring in early adulthood (e.g., Kessler et al., 2013). However, additional data suggests that nearly half of women with BED seeking treatment do not begin experiencing clinical symptoms until middle-adulthood (Brandsma, 2007). Similarly, relapse rates for individuals meeting diagnostic criteria for BED are often over 50% despite instances of prolonged clinical treatment (Bello & Hajnal, 2010), and therefore, binge eating behavior can be a chronic disorder throughout adulthood, suggesting that additional research on binge eating and BED in older populations is needed. Research has also found less disparity of binge eating symptoms across racial and ethnic groups than other eating disorders (e.g., Marques et al., 2011), and there is evidence that African Americans, Asian Americans, and Hispanic Americans experience binge eating and BED at comparable rates relative to European Americans (e.g., Chao et al., 2016). In an earlier study by Goetze and colleagues (in preparation, 2018a), perceived control was found to be a significant predictor of binge eating severity across groups of African American, Asian American, European American, and Hispanic American women, suggesting that the current investigation's primary predictive

variable may be a universal predictor of binge eating severity across racially diverse women. Nevertheless, preliminary research suggests that there may be differences in symptom presentation and distress regarding binge eating behaviors by racial group (e.g., Franko et al., 2012), and few efforts to investigate potential differences by sex and race/ethnicity (Grucza et al., 2007), which represents an area requiring future research attention.

Perhaps the largest limitation of the current investigation was the small sample size, specifically in exploratory analyses investigating possible associations between perceived control, binge eating severity, mood reactivity, and HF-HRV/RSA. Given that perceived control has been shown to attenuate the physiological impact of stress, been associated with lower rates of anxiety and depression, buffers against negative mood states, and shares an inverse association with cardiac disease and related death (e.g., Bollini et al., 2004; Dracup et al., 2003; Roepke &Grant, 2001), it was anticipated that it would be predictive of both baseline HF-HRV/RSA and physiological reactivity to negative mood induction. It was the exploratory aim of this investigation to address gaps in the current preliminary body of literature suggesting baseline and HF-HRV/RSA reactivity differences in obese BED populations when compared to weight-matched non-BED samples. Efforts were made to recruit normal to overweight, male and female participants who were thoroughly assessed for current and history of MDD symptoms that have historically impacted past physiological findings. However, it is unclear if this investigation's null findings represent a true lack of significant association between variables or is better characterized as inconclusive due to low sample sizes (n = 26 in negative mood induction condition). *Post-hoc* power analyses utilizing observed results suggested that these models were significantly underpowered, and therefore it is likely that the current findings are best considered inconclusive. Thus, although future research should continue to make efforts to

address gaps in the current binge eating HF-HRV/RSA literature base, special efforts should be made to ensure large sample sizes powerful enough to detect possible associations between predictive and mediating variables potentially linked to binge eating severity and cardiovascular function.

In addition to small sample size, the current study is limited by the cross-sectional design. Mediated regression, although suggestive of predictive relations between variables, is more accurately described as modeling simple correlations especially when utilizing data from a cross-sectional design. Cross sectional studies only provide a snapshot of links between variables at one moment in time, and therefore, temporal associations and causal claims cannot be inferred (Hayes, 2013). Furthermore, utilizing cross sectional data in mediated regression analyses indirectly suggests that the magnitude of relations between variables is static and unchanging; however, research has illustrated that this is untrue and effects between variables frequently change over time (Selig & Preacher, 2009). To further explore the relations and possible predictive role of perceived control in binge eating severity, future studies utilizing longitudinal design that measure perceived control, perceived stress, depressive symptoms, and binge eating severity over time may better illustrate temporal relations between these variables.

To limit confounding psychiatric and physiological variables that may have significantly impacted study findings (e.g., current MDD, current or past diagnoses of substance or alcohol dependence, bipolar disorder, heart disease, hypertension, medical conditions specific to the central nervous system), this study's exclusionary criteria may have inadvertently restricted the range of binge eating severity reported by recruited participants and limited the generalizability of findings in both clinical and subclinical binge eating populations. In fact, the current investigation's sample may be better characterized as "healthy" due to these exclusionary criteria

and may not adequately represent a subclinical binge eating population. Research investigating the relations between binge eating and psychological comorbidity has found elevated rates of psychiatric comorbidity and distress in this population (e.g., Javaras et al., 2008), with approximately 30% to 80% of individuals with BED meeting criteria for lifetime comorbid mood or anxiety disorder (e.g., Sheehan & Herman, 2015). Moreover, individuals with BED endorse lower scores on measures of general health and mental health-related quality of life, independent of sex, age, education, marital status, and race when compared to those without BED (Grucza et al., 2007). It is likely that excluding participants with comorbid psychiatric and physiological conditions skewed binge eating severity scores to the healthy or minimal range, which may not adequately represent subclinical binge eating samples in the general population. It is recommended that future studies increase focus on including variables such as current mood disorders as potential moderators in the study design rather than excluding these common comorbid conditions. This approach may increase generalizability of findings in both clinical and subclinical populations by facilitating the collection of a wider range of binge eating symptom severity.

Conclusions

A better understanding of the role of perceived control in binge eating symptom severity is an important step towards identifying protective, predictive, and malleable treatment variables for individuals experiencing both clinical and subclinical symptoms of BED. Results from the current investigation suggest that perceived control significantly predicts binge eating severity, such that individuals reporting greater perceived control experience less severe binge eating symptoms. This significant association was explained by the mediating impact of perceived stress and depressive symptoms, two common affective antecedents to binge eating behavior.

Findings revealed that higher self-reported perceived control was predictive of reduced perceived stress and depressive symptomatology, and that these reductions predicted less severe binge eating presentations. Interestingly, these associations were not significantly impacted by sex or history of MDD, suggesting that perceived control may be predictive of binge eating behavior and severity across sex and in frequently neglected yet widely prevalent subclinical binge eating populations. Although current results investigating associations between perceived control, binge eating severity, mood reactivity, and HF-HRV/RSA during transient negative mood induction suggest no significant relations between variables, due to the exploratory nature of these findings, it is unclear if this investigation's null results represent a true lack of significant association between variables or are better characterized as inconclusive. Given the potential protective and predictive role of perceived control in binge eating populations, and remaining questions regarding this population's potential risk for maladaptive cardiovascular function, additional research is needed to further understand the possible significant and predictive associations between these important variables and how they may impact future treatment of binge eating symptoms.

REFERENCES

Ágh, T., Kovács, G., Pawaskar, M., Supina, D., Inotai, A., & Vokó, Z. (2015). Epidemiology, health-related quality of life and economic burden of binge eating disorder: A systematic literature review. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 20*, 1-12. doi: 10.1007/s40519-014-0173-9

Agras, W. S., & Telch, C. F. (1998). The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. *Behavior Therapy, 29*, 491-503. doi: 10.1016/S0005-7894(98)80045-2

Alegria, M., Woo, M., Cao, Z., Torres, M., Meng, X. L., & Striegel-Moore, R. (2007). Prevalence and correlates of eating disorders in Latinos in the United States. *International Journal of Eating Disorders, 40*, S15-S21. doi: 10.1002/eat.20406

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.

Anderson, D. A., Williamson, D. A., Johnson, W. G., & Grieve, C. O. (2001). Validity of test meals for determining binge eating. *Eating Behaviors, 2*, 105-112. doi: 10 .1016/S1471-0153(01)00022-8

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology, 10*, 229-240. doi: 10.1037 /1089-2680.10.3.229

Armitage, C. J., & Conner, M. (1999). Distinguishing perceptions of control from self-efficacy: Predicting consumption of a low-fat diet using the theory of planned behavior. *Journal of Applied Social Psychology, 29*, 72-90. doi: 10.1111/j.1559-1816.1999.tb01375.x

Arnau, R. C., Meagher, M. W., Norris, M. P., & Bramson, R. (2001). Psychometric evaluation of the Beck Depression Inventory-II with primary care medical patients. *Health Psychology, 20*, 112-119. doi: 10.1037/0278-6133.20.2.112

Arnow, B., Kenardy, J., & Agras, W. S. (1992). Binge eating among the obese: A descriptive study. *Journal of Behavioral Medicine, 15*, 155-170. doi: 10.1007 /BF00848323

Arriaza, C. & Mann, T. (2001). Ethnic differences in eating disorder symptoms among college students: The confounding role of body mass index. *Journal of American College Health, 49*, 307-315. doi: 10.1008/07448480109196317

Aubie, C. D., & Jarry, J. L. (2009). Weight-related teasing increases eating in binge eaters. *Journal of Social and Clinical Psychology, 28*, 909-936. doi: 10.1521 /jscp.2009 .28.7.909

Barry, D. T., Grilo, C. M., & Masheb, R. M. (2003). Comparison of patients with bulimia nervosa, obese patients with binge eating disorder, and nonobese patients with binge eating disorder. *The Journal of Nervous and Mental Disease*, *191*, 589-594. doi: 10.1097 /01.nmd.0000087185.95446.65

Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. F. (1996a). Comparison of Beck Depression Inventories-IA and-II in psychiatric outpatients. *Journal of Personality Assessment*, *67*, 588-597. doi: 10.1207/s15327752jpa6703_13

Beck, A. T., Steer, R. A., & Brown, G. K. (1996b). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Beidel, D. C., Bulik, C. M., & Stanley, M. A. (2012). *Abnormal Psychology* (3rd ed.). Pearson Education, Inc.

Bello, N. T., & Hajnal, A. (2010). Dopamine and binge eating behaviors. *Pharmacology Biochemistry and Behavior*, *97*, 25-33. doi: 10.1016/j.pbb.2010.04.016

Ben-Haim, M. S., Mama, Y., Icht, M., & Algom, D. (2014). Is the emotional Stroop task a special case of mood induction? Evidence from sustained effects of attention under emotion. *Attention, Perception, & Psychophysics*, *76*, 81-97. doi: 10.3758/ s13414-013-0545-7

Berg, K. C., Frazier, P., & Sherr, L. (2009). Change in eating disorder attitudes and behavior in college women: Prevalence and predictors. *Eating Behaviors*, *10*, 137-142. doi: 10.1016 /j.eatbeh.2009.03.003

Berntson, G. G., Quigley, K. S., & Lozano, D. (2007). Cardiovascular psychophysiology. In J. Cacioppo, L. Tassinary, & G. Berntson (Eds.), *The handbook of psychophysiology (3rd ed.)* (pp. 182-210). New York: Cambridge University Press.

Bittencourt, S. A., Lucena-Santos, P., Moraes, J. F. D., & Oliveira, M. D. S. (2012). Anxiety and depression symptoms in women with and without binge eating disorder enrolled in weight loss programs. *Trends in Psychiatry and Psychotherapy*, *34*, 87-92. doi: 10.1590/S2237-60892012000200007

Bolger, N., DeLongis, A., Kessler, R. C., & Schilling, E. A. (1989). Effects of daily stress on negative mood. *Journal of Personality and Social Psychology*, *57*, 808-818. doi: 10.1037 /0022-3514.57.5.808

Bollini, A. M., Walker, E. F., Hamann, S., & Kestler, L. (2004). The influence of perceived control and locus of control on the cortisol and subjective response to stress. *Biological Psychology, 3,* 245-260. doi: 10.1016/j.biopsycho.2003.11.002

Bongers, P., Jansen, A., Havermans, R., Roefs, A., & Nederkoorn, C. (2013). Happy eating. The underestimated role of overeating in a positive mood. *Appetite, 67,* 74-80. doi: 10.1016 /j.appet.2013.03.017

Bongers, P., van den Akker, K., Havermans, R., & Jansen, A. (2015). Emotional eating and Pavlovian learning: Does negative mood facilitate appetitive conditioning?. *Appetite, 89,* 226-236. doi: 10.1016/j.appet.2015.02.018

Brandsma, L. (2007). Eating disorders across the life span. *Journal of Women & Aging*, *19*, 155-172. doi: 10.1300/J074v19n01_10

Brody, M. L., Walsh, B. T., & Devlin, M. J. (1994). Binge eating disorder: reliability and validity of a new diagnostic category. *Journal of Consulting and Clinical Psychology*, *62*, 381-386. doi: 10.1037/0022-006X.62.2.381

Bulik, C. M., & Reichborn-Kjennerud, T. (2003). Medical morbidity in binge eating disorder. *International Journal of Eating Disorders*, *34*, S39-S46. doi: 10.1002 /eat.10204

Bulik, C. M., Sullivan, P. F., & Kendler, K. S. (2002). Medical and psychiatric morbidity in obese women with and without binge eating. *International Journal of Eating Disorders*, *32*, 72-78. doi: 10.1002/eat.10072

Cardi, V., Leppanen, J., & Treasure, J. (2015). The effects of negative and positive mood induction on eating behaviour: A meta-analysis of laboratory studies in the healthy population and eating and weight disorders. *Neuroscience & Biobehavioral Reviews*, *57*, 299-309. doi: 10.1016/j.neubiorev.2015.08.011

Carney, R. M., Freedland, K. E., Stein, P. K., Skala, J. A., Hoffman, P., & Jaffe, A. S. (2000). Change in heart rate and heart rate variability during treatment for depression in patients with coronary heart disease. *Psychosomatic Medicine, 62*(5), 639-647. Retrieved from http://ovidsp.tx.ovid.com.prxy4.ursus.maine.edu /sp-3.20.0b/

Carrard, I., Linden, M. V., & Golay, A. (2012). Comparison of obese and nonobese individuals with binge eating disorder: delicate boundary between binge eating disorder and non-purging bulimia nervosa. *European Eating Disorders Review, 20*, 350-354. doi: 10.1002 /erv.2174

Carroll, D., Phillips, A. C., & Der, G. (2008). Body mass index, abdominal adiposity, obesity, and cardiovascular reactions to psychological stress in a large community sample. *Psychosomatic Medicine, 70*, 653-660. doi: 10.1001/jama.2009.2012

Castellini, G., Mannucci, E., Lo Sauro, C., Benni, L., Lazzeretti, L., Ravaldi, C., . . . Ricca, V. (2011). Different moderators of cognitive-behavioral therapy on subjective and objective binge eating in bulimia nervosa and binge eating disorder: A three-year follow-up study. *Psychotherapy and Psychosomatics*, *81*, 11-20. doi: 10.1159/000329358

Cattanach, L., Malley, R., & Rodin, J. (1988). Psychologic and physiologic reactivity to stressors in eating disordered individuals. *Psychosomatic Medicine*, *50*(6), 591-599.

Celio, A. A., Wilfley, D. E., Crow, S. J., Mitchell, J., & Walsh, B. T. (2004). A comparison of the binge eating scale, questionnaire for eating and weight patterns-revised, and eating disorder examination questionnaire with instructions with the eating disorder examination in the assessment of binge eating disorder and its symptoms. *International Journal of Eating Disorders, 36*, 434-444. doi: 10.1002/eat.20057

Cella, D. F., & Perry, S. W. (1986). Reliability and concurrent validity of three visual-analogue mood scales. *Psychological Reports, 59*, 827-833. doi: 10.2466/pr0 .1986.59.2.827

Chaney, J. M., Mullins, L. L., Uretsky, D. L., Doppler, M. J., Palmer, W. R., Wees, S. J., . . . Reiss, M. J. (1996). Attributional style and depression in rheumatoid arthritis: The moderating role of perceived illness control. *Rehabilitation Psychology*, *41*, 205-223. doi: 10.1037/0090-5550.41.3.205

Chao, A. M., Grilo, C. M., & Sinha, R. (2016). Food cravings, binge eating, and eating disorder psychopathology: Exploring the moderating roles of gender and race. *Eating Behaviors, 21*, 41-47. doi: 10.1016/j.eatbeh.2015.12.007

Chua, J. L., Touyz, S., & Hill, A. J. (2004). Negative mood-induced overeating in obese binge eaters: an experimental study. *International Journal of Obesity, 28*, 606-610. doi: 10.1038/sj.ijo.080259

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396. Retrieved from http: //www.jstor .org.prxy4.ursus.maine.edu/stable/2136404?seq=1#page_scan_tab_contents

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the U.S. In S. Spacapam & S. Oskamp (Eds.), *The social psychology of health: Claremont Symposium on Applied Social Psychology*. Newbury Park, CA: Sage.

Colles, S. L., Dixon, J. B., & O'Brien, P. E. (2008). Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity, 16*, 608-614. doi: 10.1038 /oby.2007.99

Corwin, R. L., Avena, N. M., & Boggiano, M. M. (2011). Feeding and reward: perspectives from three rat models of binge eating. *Physiology & Behavior, 104*, 87-97. doi: 10.1016 /j.physbeh.2011.04.041

Dalgleish, T., Tchanturia, K., Serpell, L., Hems, S., de Silva, P., & Treasure, J. (2001). Perceived control over events in the world in patients with eating disorders: A preliminary study. *Personality and Individual Differences, 31*, 453-460. doi: 10.1016/S0191-8869(00) 00150-1

Daszykowski, M., Kaczmarek, K., Vander Heyden, Y., & Walczak, B. (2007). Robust statistics in data analysis—A review: Basic concepts. *Chemometrics and Intelligent Laboratory Systems, 85*, 203-219. doi: 10.1016/j.chemolab.2006 .06.016

Davis, C. (2013). From passive overeating to "food addiction": A spectrum of compulsion and severity. *ISRN Obesity, 1216*, 1-20. doi: 10.1155/2013/435027

Davis, C. (2015). The epidemiology and genetics of binge eating disorder (BED). *CNS Spectrums, 20*, 522-529. doi: 10.1017/S1092852915000462

Davis, R., & Jamieson, J. (2005). Assessing the functional nature of binge eating in the eating disorders. *Eating Behaviors, 6*, 345-354. doi: 10.1016/j.eatbeh.2005.02.001

Davis, C., Loxton, N. J., Levitan, R. D., Kaplan, A. S., Carter, J. C., & Kennedy, J. L. (2013). 'Food addiction and its association with a dopaminergic multilocus genetic profile. *Physiology & Behavior, 118*, 63-69. doi: 10.1016/j.physbeh .2013.05.014

Deaver, C. M., Miltenberger, R. G., Smyth, J., Meidinger, A. M. Y., & Crosby, R. (2003). An evaluation of affect and binge eating. *Behavior Modification, 27*, 578-599. doi: 10.1177 /0145445503255571

Degortes, D., Santonastaso, P., Zanetti, T., Tenconi, E., Veronese, A., & Favaro, A. (2014). Stressful life events and binge eating disorder. *European Eating Disorders Review, 22*, 378-382. doi: 10.1002/erv.2308

Dekker, J. M., Crow, R. S., Folsom, A. R., Hannan, P. J., Liao, D., Swenne, C. A., & Schouten, E. G. (2000). Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: The ARIC study. *Circulation, 102*, 1239-1244. doi: 10.1161/01.CIR.102.11.1239

De Wit, L., Luppino, F., van Straten, A., Penninx, B., Zitman, F., & Cuijpers, P. (2010). Depression and obesity: a meta-analysis of community-based studies. *Psychiatry Research, 178*, 230-235. doi: 10.1016/j.psychres.2009.04.015

De Zwaan, M. (2001). Binge eating disorder and obesity. *International Journal of Obesity & Related Metabolic Disorders, 25*(Suppl 1), S51-S55. Retrieved from http://web.a .ebscohost.com.prxy4.ursus.maine.edu/ehost/pdfviewer/pdfviewer?sid=c612f73a-8e95-42e7-9689-74397be5a829%40sessionmgr4003&vid=1&hid =4101

Didie, E. R., & Fitzgibbon, M. (2005). Binge eating and psychological distress: Is the degree of obesity a factor?. *Eating Behaviors, 6*, 35-41. doi: 10.1016/j.eatbeh .2004.08.007

Dingemans, A. E., Martijn, C., Jansen, A. T., & van Furth, E. F. (2009). The effect of suppressing negative emotions on eating behavior in binge eating disorder. *Appetite, 52*, 51-57. doi: 10.1016/j.appet.2008.08.004

Dingemans, A. E., & van Furth, E. F. (2012). Binge eating disorder psychopathology in normal weight and obese individuals. *International Journal of Eating Disorders*, *45*, 135-138. doi: 10.1002/eat.20905

Dingemans, A. E., Visser, H., Paul, L., & van Furth, E. F. (2015). Set-shifting abilities, mood and loss of control over eating in binge eating disorder: An experimental study. *Psychiatry Research, 230*, 242-248. doi: 10.1016/j.psychres.2015.09.001

Doering, L. V., McGuire, A., Eastwood, J. A., Chen, B., Bodán, R. C., Czer, L. S., & Irwin, M. R. (2015). Cognitive behavioral therapy for depression improves pain and perceived control in cardiac surgery patients. *European Journal of Cardiovascular Nursing*. Advance online publication. doi: 10.1177 /1474515115592292.

Dozois, D. J., Dobson, K. S., & Ahnberg, J. L. (1998). A psychometric evaluation of the Beck Depression Inventory–II. *Psychological Assessment, 10*, 83-89. doi: 10.1037 /1040-3590.10.2.83

Dracup, K., Westlake, C., Erickson, V. S., Moser, D. K., Caldwell, M. L., & Hamilton, M. A. (2003). Perceived control reduces emotional stress in patients with heart failure. *The Journal of Heart and Lung Transplantation, 22*, 90-93. doi: 10.1016 /S1053-2498(02) 00454-0

Drewelies, J., Schade, H., Hülür, G., Hoppmann, C. A., Ram, N., & Gerstorf, D. (2018). The more we are in control, the merrier? Partner Perceived Control and Negative Affect in the Daily Lives of Older Couples. *The Journals of Gerontology - Series B: Psychological and Social Sciences*. Advance online publication. doi: 10.1093/geronb/gby009

Eldredge, K. L., & Agras, W. S. (1996). Weight and shape overconcern and emotional eating in binge eating disorder. *International Journal of Eating Disorders*, *19*, 73-82. doi: 10. 1002/(SICI)1098-108X(199601)19:1<73::AID-EAT9>3.0.CO;2-T

E-Prime (Version 2). (2015). [Computer software]. Sharpsburg, PA: Psychology Software Tools, Inc.

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry, 57*, 659-665. doi: 10-1001/pubs.Arch Gen Psychiatry-ISSN-0003-990x-57-7-yoa9404.

Farmer, M. M., & Ferraro, K. F. (2005). Are racial disparities in health conditional on socioeconomic status?. *Social Science & Medicine*, *60*, 191-204. doi: 10.1016 /j.socscimed.2004.04.026

Faul, F., Erdfelder, E., Lang, A.-G. & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191. doi: 10.3758/BF03193146

Field, A. (2009). *Discovering statistics using SPSS: And sex and drugs and rock 'n' roll* (3rd ed.). Washington, DC: Sage Publications.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders: Patient Edition (SCID I/P), Version 2.0.* New York: Biometric Research.

Franko, D. L., Thompson-Brenner, H., Thompson, D. R., Boisseau, C. L., Davis, A., Forbush, K. T., . . . Devlin, M. J. (2012). Racial/ethnic differences in adults in randomized clinical trials of binge eating disorder. *Journal of Consulting and Clinical Psychology*, *80*, 186-195. doi: 10.1037/a0026700

Freeman, L. M. Y., & Gil, K. M. (2004). Daily stress, coping, and dietary restraint in binge eating. *International Journal of Eating Disorders, 36*, 204-212. doi: 10 .1002 /eat.20012

Friederich, H. C., Schild, S., Schellberg, D., Quenter, A., Bode, C., Herzog, W., . . . Zipfel, S. (2006). Cardiac parasympathetic regulation in obese women with binge eating disorder. *International Journal of Obesity, 30*, 534-542. doi: 10.1038 /sj.ijo.0803181

Froreich, F. V., Vartanian, L. R., Grisham, J. R., & Touyz, S. W. (2016). Dimensions of control and their relation to disordered eating behaviours and obsessive-compulsive symptoms. *Journal of Eating Disorders, 4*, 1419-1428. doi: 10.1186 /s40337-016-0104-4

Gallagher, M. W., Bentley, K. H., & Barlow, D. H. (2014). Perceived control and vulnerability to anxiety disorders: A meta-analytic review. *Cognitive Therapy and Research, 38*, 571-584. doi: 10.1007/s10608-014-9624-x

Gallagher, M. W., Naragon-Gainey, K., & Brown, T. A. (2014). Perceived control is a transdiagnostic predictor of cognitive-behavior therapy outcome for anxiety disorders. *Cognitive Therapy and Research, 38*, 10-22. doi: 10.1007/s10608-013-9587-3

Ginty, A. T., Phillips, A. C., Higgs, S., Heaney, J. L., & Carroll, D. (2012). Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress. *Psychoneuroendocrinology, 37*, 715-724. doi: 10.1016 /j.psyneuen.2011.09.004

Gladis, M. M., Wadden, T. A., Foster, G. D., Vogt, R. A., & Wingate, B. J. (1998). A comparison of two approaches to the assessment of binge eating in obesity. *International Journal of Eating Disorders, 23*, 17-26. doi: 10.1002/(SICI)1098-108X(199801)23:1 <17::AID-EAT3>3.0.CO;2-4

Gluck, M. E. (2006). Stress response and binge eating disorder. *Appetite*, *46*, 26-30. doi: 10.1016 /j.appet.2005.05.004

Gluck, M. E., Geliebter, A., Hung, J., & Yahav, E. (2004). Cortisol, hunger, and desire to binge eat following a cold stress test in obese women with binge eating disorder. *Psychosomatic Medicine*, *66*, 876-881. doi: 10.1097/01.psy.0000143637.63508.47

Goetze, R. E., Huff, R. M., Saslow, L. R., Epel, E., & McCoy, S. K. (In preparation, 2018a). Perceived control and perceived stress in predicting binge eating severity: Examining racial differences in a new model. Manuscript in preparation. University of Maine, Orono, Maine.

Goetze, R. E., Huff, R. M., Bogucki, O. E., Haigh, E. A. P., & McCoy, S. K. (in preparation, 2018b). Investigating the role of perceived control in women with binge eating symptomatology. Manuscript in preparation. University of Maine, Orono, Maine.

Goldschmidt, A. B., Engel, S. G., Wonderlich, S. A., Crosby, R. D., Peterson, C. B., Grange, D., . . . Mitchell, J. E. (2012). Momentary affect surrounding loss of control and overeating in obese adults with and without binge eating disorder. *Obesity*, *20*, 1206-1211. doi: 10.1038/oby.2011.286

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*, 47-55. doi: 10.1016 /0306-4603(82) 90024-7

Green, J. D., Sedikides, C., Saltzberg, J. A., Wood, J. V., & Forzano, L. A. B. (2003). Happy mood decreases self-focused attention. *British Journal of Social Psychology*, *42*, 147-157. doi: 10.1348/014466603763276171

Greeno, C. G., Marcus, M. D., & Wing, R. R. (1995). Diagnosis of binge eating disorder: Discrepancies between a questionnaire and clinical interview. *International Journal of Eating Disorders*, *17*, 153-160. doi: 10.1002/1098-108X(199503)17:2 <153::AID-EAT2260170208>3.0.CO;2-V

Greeno, C. G., Wing, R. R., & Shiffman, S. (2000). Binge antecedents in obese women with and without binge eating disorder. *Journal of Consulting and Clinical Psychology*, *68*, 95-102. doi: 10.1037/0022-006X.68.1.95

Grilo, C. (2015). Cardiovascular disease risk reduction in patients with binge eating disorder and obesity: Randomized controlled trial of stepped-care versus standard behavioral weight loss. *Atherosclerosis*, *241*, e19. doi: 10.1016/j.atherosclerosis .2015.04.081

Grilo, C. M., Lozano, C., & Masheb, R. M. (2005). Ethnicity and sampling bias in binge eating disorder: Black women who seek treatment have different characteristics than those who do not. *International Journal of Eating Disorders*, *38*, 257-262. doi: 10.1002/eat.20183

Groesz, L. M., McCoy, S., Carl, J. Saslow, L., Stewart, J., Adler, N., . . . Epel, E. (2012). What is eating you? Stress and drive to eat. *Appetite, 58*, 717-721. doi: 10.1016 /j.appet.2011 .11.028

Grucza, R. A., Przybeck, T. R., & Cloninger, C. R. (2007). Prevalence and correlates of binge eating disorder in a community sample. *Comprehensive Psychiatry, 48*, 124-131. doi: 10.1016 /j.comppsych.2006.08.002

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137*, 660-681. doi: 10.1037/a0023660

Hansen, A. L., Johnsen, B. H., & Thayer, J. F. (2003). Vagal influence on working memory and attention. *International Journal of Psychophysiology, 48*, 263-274. doi: 10.1016/S0167-8760(03)00073-4

Harney, M. B., Fitzsimmons-Craft, E. E., Maldonado, C. R., & Bardone-Cone, A. M. (2014). Negative affective experiences in relation to stages of eating disorder recovery. *Eating Behaviors, 15*, 24-30. doi: 10.1016/j.eatbeh.2013.10.016

Harrell, Z. A., & Jackson, B. (2008). Thinking fat and feeling blue: Eating behaviors, ruminative coping, and depressive symptoms in college women. *Sex Roles, 58*, 658-665. doi: 10.1007/s11199-007-9388-9

Harrington, E. F., Crowther, J. H., Henrickson, H. C. P., & Mickelson, K. D. (2006). The relationships among trauma, stress, ethnicity, and binge eating. *Cultural Diversity and Ethnic Minority Psychology, 12*, 212-229. doi: 10.1037/1099-9809.12.2.212

Hayes, A. F. (2014). *The PROCESS macro for SPSS and SAS* [Computer software]. Retrieved from http://www.processmacro.org/

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*, 86-108. doi: 10.1037/0033-2909. 110.1.86

Henderson, N. J., & Huon, G. F. (2002). Negative affect and binge eating in overweight women. *British Journal of Health Psychology, 7*, 77-87. doi: 10.1348 /135910702169376

Hilbert, A., & Tuschen-Caffier, B. (2007). Maintenance of binge eating through negative mood: A naturalistic comparison of binge eating disorder and bulimia nervosa. *International Journal of Eating Disorders, 40*, 521-530. doi: 10.1002/eat.20401

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry, 61*, 348-358. doi: 10.1016/j.biopsych.2006.03.040

Hudson, J. I., Lalonde, J. K., Coit, C. E., Tsuang, M. T., McElroy, S. L., Crow, S. J., . . . Pope, H. G. (2010). Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals with binge-eating disorder. *The American Journal of Clinical Nutrition, 91,* 1568-1573. doi: 10.3945/ajcn.2010.29203

Hughes, E. K., Goldschmidt, A. B., Labuschagne, Z., Loeb, K. L., Sawyer, S. M., & Grange, D. L. (2013). Eating disorders with and without comorbid depression and anxiety: similarities and differences in a clinical sample of children and adolescents. *European Eating Disorders Review, 21,* 386-394. doi: 10.1002 /erv.2234

IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.

Javaras, K. N., Pope, H. G., Lalonde, J. K., Roberts, J. L., Nillni, Y. I., Laird, N. M., . . . Walsh, B. T. (2008). Co-occurrence of binge eating disorder with psychiatric and medical disorders. *The Journal of Clinical Psychiatry, 69*(2), 266-273. Retrieved from http://www-psychiatrist-com.prxy4.ursus.maine.edu/JCP/article/Pages/2008 /v69n02/v69n0213.aspx

Jin, A. B., Steding, L. H., & Webb, A. K. (2015). Reduced emotional and cardiovascular reactivity to emotionally evocative stimuli in major depressive disorder. *International Journal of Psychophysiology, 97,* 66-74. doi: 10.1016/j.ijpsycho .2015.04.014

Johnson, J. G., Spitzer, R. L., & Williams, J. B. W. (2001). Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. *Psychological Medicine, 31,* 1455-1466. doi: 10.1017 /S0033291701004640

Judd, C. M., & Kenny, D. A. (2010). *Data analysis in social psychology: Recent and recurring issues*. John Wiley & Sons, Inc.

Karason, K., Mølgaard, H., Wikstrand, J., & Sjöström, L. (1999). Heart rate variability in obesity and the effect of weight loss. *The American Journal of Cardiology, 83,* 1242-1247. doi: 10.1016/S0002-9149(99)00066-1

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biological Psychiatry, 67,* 1067-1074. doi: 10.1016/j.biopsych.2009 .12.012

Kenardy, J., Arnow, B., & Agras, W. S. (1996). The aversiveness of specific emotional states associated with binge-eating in obese subjects. *Australian and New Zealand Journal of Psychiatry, 30,* 839-844. doi: 10.3109/00048679609065053

Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., . . . Bruffaerts, R. (2013). The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry, 73,* 904-914. doi: 10.1016/j.biopsych.2012.11.020

Klatzkin, R. R., Gaffney, S., Cyrus, K., Bigus, E., & Brownley, K. A. (2015). Binge eating disorder and obesity: Preliminary evidence for distinct cardiovascular and psychological phenotypes. *Physiology & Behavior, 142,* 20-27. doi: 10.1016 /j.physbeh.2015.01.018

Koeng, J. & Thayer, J. F. (2016). Sex differences in healthy human heart rate variability: A meta-analysis. *Neuroscience and Biobehavioral Reviews, 64,* 288-310. dio: 10.1016 /j.neubiorev.2016.03.007

Kolbeinsson, Þ. (2016). Vulnerabilities to depression: Cognitive reactivity, depressive rumination, and heart rate variability (Doctoral thesis, Sigillum Universitatis Islandiae, Heilbrigðisvísindasvið Háskóla Íslands, Iceland). Retrieved from http://skemman.is /item/view /1946/25100

Koo-Loeb, J. H., Costello, N., Light, K. C., & Girdler, S. S. (2000). Women with eating disorder tendencies display altered cardiovascular, neuroendocrine, and psychosocial profiles. *Psychosomatic Medicine, 62*(4), 539-548. Retrieved from http://ovidsp.tx.ovid.com.prxy4 .ursus.maine.edu/sp-3.20.0b/

Koo-Loeb, J. H., Pedersen, C., & Girdler, S. S. (1998). Blunted cardiovascular and catecholamine stress reactivity in women with bulimia nervosa. *Psychiatry Research, 80*(1), 13-27. doi: 10.1016/S0165-1781(98)00057-2

Laederach-Hofmann, K., Mussgay, L., & Ruddel, H. (2000). Autonomic cardiovascular regulation in obesity. *Journal of Endocrinology, 164*(1), 59-66. doi: 10.1677 /joe.0.1640059

Laessle, R. G., & Schultz, S. (2009). Stress-induced laboratory eating behavior in obese women with binge eating disorder. *Appetite, 58,* 457-461. doi: 10.1016 /j.appet.2011.12.007

Langer, E. J. (1975). The illusion of control. *Journal of Personality and Social Psychology, 32,* 311-328. doi: 10.1037/0022-3514.32.2.311

Latner, J. D., Vallance, J. K., & Buckett, G. (2008). Health-related quality of life in women with eating disorders: association with subjective and objective binge eating. *Journal of Clinical Psychology in Medical Settings, 15,* 148-153. doi: 10.1007/s10880-008-9111-1

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer Publishing Company, Inc.

Le Grange, D. L., Gorin, A., Catley, D., & Stone, A. A. (2001). Does momentary assessment detect binge eating in overweight women that is denied at interview?. *European Eating Disorders Review*, *9*, 309-324. doi: 10.1002/erv.409

Leehr, E. J., Krohmer, K., Schag, K., Dresler, T., Zipfel, S., & Giel, K. E. (2015). Emotion regulation model in binge eating disorder and obesity-A systematic review. *Neuroscience & Biobehavioral Reviews*, *49*, 125-134. doi: 0.1016 /j.neubiorev.2014.12.008

Levine, M. D., & Marcus, M. D. (1997). Eating behavior following stress in women with and without bulimic symptoms. *Annals of Behavioral Medicine*, *19*, 132-138. doi: 10.1007 /BF02883330

Lourenço, B. H., Arthur, T., Rodrigues, M. B., Guazzelli, I., Frazzatto, E., Deram, S., . . . Villares, S. F. (2008). Binge eating symptoms, diet composition and metabolic characteristics of obese children and adolescents. *Appetite*, *50*, 223-230. doi: 10.1016 /j.appet.2007.07.004

Lydecker, J. A., & Grilo, C. M. (2016). Different yet similar: Examining race and ethnicity in treatment-seeking adults with binge eating disorder. *Journal of Consulting and Clinical Psychology*, *84*, 88-94. doi: 10.1037/ccp0000048

Lynch, W. C., Everingham, A., Dubitzky, J., Hartman, M., & Kasser, T. (2000). Does binge eating play a role in the self-regulation of moods?. *Integrative Physiological and Behavioral Science*, *35*, 298-313. doi: 10.1007/BF02688792

Marchesini, G., Natale, S., Chierici, S., Manini, R., Besteghi, L., Di Domizio, S., . . . Melchionda, N. (2002). Effects of cognitive-behavioural therapy on health-related quality of life in obese subjects with and without binge eating disorder. *International Journal of Obesity & Related Metabolic Disorders*, *26*(9), 1261-1267. Retrieved from http://web.b .ebscohost.com.prxy4.ursus.maine.edu/ehost /pdfviewer/pdfviewer?sid=7ea6a006-aeb5-459c-9efb-411c9ae5bc1d%40 sessionmgr107&vid=0&hid=102

Marcus, M. D., & Wildes, J. E. (2014). Disordered eating in obese individuals. *Current Opinion in Psychiatry*, *27*, 443-447. doi: 10.1097/YCO.0000000000000103

Marcus, M. D., Wing, R. R., Ewing, L., Kern, E., Gooding, W., & McDermott, M. (1990). Psychiatric disorders among obese binge eaters. *International Journal of Eating Disorders*, *9*, 69-77. doi: 10.1002/1098-108X(199001)9:1<69::AID-EAT2260090108 >3.0.CO;2-K

Marcus, M. D., Wing, R. R., & Hopkins, J. (1988). Obese binge eaters: Affect, cognitions, and response to behavioral weight control. *Journal of Consulting and Clinical Psychology*, *56*, 433-439. doi: 10.1037 /0022-006X.56.3.433

Marques, L., Alegria, M., Becker, A. E., Chen, C. N., Fang, A., Chosak, A., & Diniz, J. B. (2011). Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups: Implications for reducing ethnic disparities in health care access for eating disorders. *International Journal of Eating Disorders*, *44*, 412-420. doi: 10.1002/eat.20787

Marshall, G. N., & Lang, E. L. (1990). Optimism, self-mastery, and symptoms of depression in women professionals. *Journal of Personality and Social Psychology*, *59*, 132-139. doi: 10.1037/0022-3514.59.1.132

Martin, M. (1990). On the induction of mood. *Clinical Psychology Review*, *10*, 669-697. doi: 10.1016/0272-7358(90)90075-L

Mathes, W. F., Brownley, K. A., Mo, X., & Bulik, C. M. (2009). The biology of binge eating. *Appetite*, *52*, 545-553. doi: 10.1016/j.appet.2009.03.005

McCabe, M. P., & Ricciardelli, L. A. (2004). Body image dissatisfaction among males across the lifespan: A review of past literature. *Journal of Psychosomatic Research*, *56*, 675-685. doi: 10.1016/S0022-3999(03)00129-6

McCaul, K. D., Sandgren, A. K., O'Neill, H. K., & Hinsz, V. B. (1993). The value of the theory of planned behavior, perceived control, and self-efficacy expectations for predicting health-protective behaviors. *Basic and Applied Social Psychology*, *14*, 231-252. doi: 10.1207/s15324834basp1402_7

Messerli-Bürgy, N., Engesser, C., Lemmenmeier, E., Steptoe, A., & Laederach-Hofmann, K. (2010). Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. *International Journal of Psychophysiology*, *78*, 163-168. doi: 10.1016/j.ijpsycho .2010.07.005

MindWare Technologies Ltd. (2009). [Computer software]. Gahanna, OH: Mindware Technologies Ltd.

Mitchell, J. E. (2016). Medical comorbidity and medical complications associated with binge-eating disorder. *International Journal of Eating Disorders*, *49*, 319-323. doi: 10.1002 /eat.22452

Mitchell, J. E., & Mussell, M. P. (1995). Comorbidity and binge eating disorder. *Addictive Behaviors*, *20*, 725-732. doi: 10.1016/0306-4603(95)00095-X

Mond, J. M., Latner, J. D., Hay, P. H., Owen, C., & Rodgers, B. (2010). Objective and subjective bulimic episodes in the classification of bulimic-type eating disorders: another nail in the coffin of a problematic distinction. *Behaviour Research and Therapy*, *48*, 661-669. doi: 10.1016/j.brat.2010.03.020

Morgan, C. M., Yanovski, S. Z., Nguyen, T. T., McDuffie, J., Sebring, N. G., Jorge, M. R., . . . Yanovski, J. A. (2002). Loss of control over eating, adiposity, and psychopathology in overweight children. *International Journal of Eating Disorders, 31,* 430-441. doi: 10.1002/eat.10038

Moser, D. K., & Dracup, K. (1995). Psychosocial recovery from a cardiac event: The influence of perceived control. *Heart & Lung: The Journal of Acute and Critical Care, 24*, 273-280. doi: 10.1016/S0147-9563(05)80070-6

Munsch, S., Meyer, A. H., Quartier, V., & Wilhelm, F. H. (2012). Binge eating in binge eating disorder: a breakdown of emotion regulatory process?. *Psychiatry Research, 195*, 118-124. doi: 10.1016/j.psychres.2011.07.016

Munsch, S., Michael, T., Biedert, E., Meyer, A. H., & Margraf, J. (2008). Negative mood induction and unbalanced nutrition style as possible triggers of binges in binge eating disorder (BED). *Eating and Weight Disorders: Studies on Anorexia, Bulimia and Obesity, 13*, 22-29. doi: 10.1007/BF03327781

Napolitano, M. A., & Himes, S. (2011). Race, weight, and correlates of binge eating in female college students. *Eating Behaviors*, *12*, 29-36. doi: 10.1016/j.eatbeh .2010.09.003

Nolen-Hoeksema, S., Stice, E., Wade, E., & Bohon, C. (2007). Reciprocal relations between rumination and bulimic, substance abuse, and depressive symptoms in female adolescents. *Journal of Abnormal Psychology, 116*, 198-207. doi: 10.1037/0021-843X.116.1.198

Nunan, D., Sandercock, G. R., & Brodie, D. A. (2010). A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. *Pacing and Clinical Electrophysiology*, *33*, 1407-1417. doi: 10.1111/j.1540-8159.2010.02841.x

Osman, A., Downs, W. R., Barrios, F. X., Kopper, B. A., Gutierrez, P. M., & Chiros, C. E. (1997). Factor structure and psychometric characteristics of the Beck Depression Inventory-II. *Journal of Psychopathology and Behavioral Assessment, 19*, 359-376. doi: 10.1007/BF02229026

Pagoto, S., Bodenlos, J. S., Kantor, L., Gitkind, M., Curtin, C., & Ma, Y. (2007). Association of major depression and binge eating disorder with weight loss in a clinical setting. *Obesity*, *15*, 2557-2559. doi: 10.1038/oby.2007.307

Paxton, S. J., & Diggens, J. (1997). Avoidance coping, binge eating, and depression: An examination of the escape theory of binge eating. *International Journal of Eating Disorders*, *22*, 83-87. doi: 10.1002/(SICI)1098-108X(199707)22:1<83::AID-EAT11>3.0.CO;2-J

Pearlin, L. I., Menaghan, E. G., Lieberman, M. A., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior, 22*(4), 337-356. Retrieved from http://www.jstor .org.prxy4.ursus.maine.edu/stable/2136676

Pearlin, L. I., & Schooler, C. (1979). The structure of coping. *Journal of Health and Social Behavior, 19*(1), 2-21. Retrieved from http://www.jstor.org.prxy4.ursus.maine.edu /stable/2136319

Pendleton, V. R., Willems, E., Swank, P., Poston, W. S. C., Goodrick, G. K., Reeves, R. S., . . . Foreyt, J. P. (2001). Negative stress and the outcome of treatment for binge eating. *Eating Disorders, 9,* 351-360. doi: 10.1080/106402601753454912

Phillips, A. C. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology, 86,* 106-113. doi: 10.1016 /j.biopsycho .2010.03.016

Pike, K. M., Dohm, F. A., Striegel-Moore, R. H., Wilfley, D. E., & Fairburn, C. G. (2001). A comparison of black and white women with binge eating disorder. *American Journal of Psychiatry, 158,* 1455-1460. doi: 10.1176/appi.ajp.158.9 .1455

Porges, S. W. (1995). Cardiac vagal tone: a physiological index of stress. *Neuroscience & Biobehavioral Reviews, 19*(2), 225-233. doi: 10.1016/0149-7634(94)00066-A

Qualtrics. (Version 7640326). (2016). [Online computer software]. Provo, Utah: Qualtrics.

Ranzenhofer, L. M., Engel, S. G., Crosby, R. D., Haigney, M., Anderson, M., McCaffery, J. M., & Tanofsky-Kraff, M. (2016). Real-time assessment of heart rate variability and loss of control eating in adolescent girls: A pilot study. *International Journal of Eating Disorders, 49,* 197-201. doi: 10.1002/eat.22464

Reagan, P., & Hersch, J. (2005). Influence of race, gender, and socioeconomic status on binge eating frequency in a population-based sample. *International Journal of Eating Disorders, 38,* 252-256. doi: 10.1002/eat.20177

Reichborn-Kjennerud, T., Bulik, C. M., Sullivan, P. F., Tambs, K., & Harris, J. R. (2004). Psychiatric and medical symptoms in binge eating in the absence of compensatory behaviors. *Obesity Research, 12,* 1445-1454. doi: 10.1038/oby.2004.181

Roberti, J. W., Harrington, L. N., & Storch, E. A. (2006). Further psychometric support for the 10-item version of the perceived stress scale. *Journal of College Counseling, 9,* 135-147. doi: 10.1002/j.2161-1882.2006.tb00100.x

Rivard, V., & Cappeliez, P. (2007). Perceived control and coping in women faced with activity restriction due to osteoarthritis: relations to anxious and depressive symptoms. *Canadian Journal on Aging, 26,* 241-253. doi: 10.3138/cja.26.3.241

Roepke, S. K., & Grant, I. (2011). Toward a more complete understanding of the effects of personal mastery on cardiometabolic health. *Health Psychology*, *30*, 615-632. doi: 0.1037 /a0023480

Rosenbaum, D. L., White, K. S., & Gervino, E. V. (2012). The impact of perceived stress and perceived control on anxiety and mood disorders in noncardiac chest pain. *Journal of Health Psychology*. Advance online publication. doi: 10.1177 /1359105311433906.

Salamone, J. D., & Correa, M. (2013). Dopamine and food addiction: lexicon badly needed. *Biological Psychiatry, 73*, e15-e24. doi: 10.1016/j.biopsych.2012.09.027

Sassaroli, S., Gallucci, M., & Ruggiero, G. M. (2008). Low perception of control as a cognitive factor of eating disorders. Its independent effects on measures of eating disorders and its interactive effects with perfectionism and self-esteem. *Journal of Behavior Therapy and Experimental Psychiatry, 39*, 467-488. doi: 10.1016 /j.jbtep.2007.11.005

Schwarze, N. J., Oliver, J. M., & Handal, P. J. (2003). Binge eating as related to negative self-awareness, depression, and avoidance coping in undergraduates. *Journal of College Student Development, 44*, 644-652. doi: 10.1353/csd.2003.0058

Segal, Z. V., Gemar, M., & Williams, S. (1999). Differential cognitive response to a mood challenge following successful cognitive therapy or pharmacotherapy for unipolar depression. *Journal of Abnormal Psychology*, *108*, 3-10. doi: 10.1037 /0021-843X .108.1.3

Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, *63*, 749-755. doi: 10.1001/archpsyc .63.7.749

Selby, E. A., Anestis, M. D., & Joiner, T. E. (2008). Understanding the relationship between emotional and behavioral dysregulation: Emotional cascades. *Behaviour Research and Therapy, 46*, 593-611. doi: 10.1016/j.brat.2008.02.002

Selig, J. P., & Preacher, K. J. (2009). Mediation models for longitudinal data in developmental research. *Research in Human Development, 6*, 144-164. doi: 10.1080 /15427600902911247

Sheehan, D. V., & Herman, B. K. (2015). The psychological and medical factors associated with untreated binge eating disorder. *The Primary Care Companion for CNS Disorders*, *17*(2). doi: 10.4088/PCC.14r01732

Shingleton, R. M., Thompson-Brenner, H., Thompson, D. R., Pratt, E. M., & Franko, D. L. (2015). Gender differences in clinical trials of binge eating disorder: An analysis of aggregated data. *Journal of Consulting and Clinical Psychology*, *83*, 382-386. doi: 10.1037/a0038849

Simon, G. E., Ludman, E. J., Linde, J. A., Operskalski, B. H., Ichikawa, L., Rohde, P., . . . Jeffery, R. W. (2008). Association between obesity and depression in middle-aged women. *General Hospital Psychiatry*, *30*, 32-39. doi: 10.1016 /j.genhosppsych .2007.09.001

Simon, J., Schmidt, U., & Pilling, S. (2005). The health service use and cost of eating disorders. *Psychological Medicine*, *35*, 1543-1551. doi: 10.1017 /S0033291705004708

Skinner, E. A. (1996). A guide to constructs of control. *Journal of Personality and Social Psychology*, *71*, 549-570. doi: 10.1037/0022-3514.71.3.549

Smith, D. G., & Robbins, T. W. (2013). The neurobiological underpinnings of obesity and binge eating: A rationale for adopting the food addiction model. *Biological Psychiatry*, *73*, 804-810. doi: 10.1016 /j.biopsych.2012.08.026

Smyth, J. M., Wonderlich, S. A., Heron, K. E., Sliwinski, M. J., Crosby, R. D., Mitchell, J. E., . . . Engel, S. G. (2007). Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. *Journal of Consulting and Clinical Psychology*, *75*, 629-638. doi: 10.1037/0022-006X.75.4.629

Sobal, J., & Stunkard, A. J. (1989). Socioeconomic status and obesity: a review of the literature. *Psychological Bulletin*, *105*, 260-275. doi: 10.1037/0033-2909.105 .2.260

Sona Systems. (2016). [Online computer software]. Bethesda, MD: Sona Systems.

Specker, S., de Zwaan, M., Raymond, N., & Mitchell, J. (1994). Psychopathology in subgroups of obese women with and without binge eating disorder. *Comprehensive Psychiatry*, *35*, 185-190. doi: 10.1016/0010-440X(94)90190-2

Stein, R. I., Kenardy, J., Wiseman, C. V., Dounchis, J. Z., Arnow, B. A., & Wilfley, D. E. (2007). What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *International Journal of Eating Disorders*, *40*, 195-203. doi: 10.1002/eat.20352

Stein, PhD, P. K., & Kleiger, MD, R. E. (1999). Insights from the study of heart rate variability. *Annual review of medicine*, *50*(1), 249-261. doi: 10.1146/annurev.med .50.1.249

Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *International Journal of Eating Disorders*, *27*, 218-229. doi: 10.1002/(SICI)1098-108X(200003)27:2<218::AID-EAT10>3.0.CO;2-1

Stice, E., Marti, C. N., Shaw, H., & Jaconis, M. (2009). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology*, *118*, 587-597. doi: 10.1037 /a0016481

Striegel-Moore, R. H., Dohm, F. A., Kraemer, H. C., Schreiber, G. B., Taylor, C. B., & Daniels, S. R. (2007). Risk factors for binge-eating disorders: An exploratory study. *International Journal of Eating Disorders*, *40*, 481-487. doi: 10.1002/eat

Striegel-Moore, R. H., & Franko, D. L. (2003). Epidemiology of binge eating disorder. *International Journal of Eating Disorders*, *34*(S1), S19-S29. doi: 10.1002 /eat.10202

Strange, J. P., Hamilton, J. L., Fresco, D. M., & Alloy, L. B. (2017). Flexible parasympathetic responses to sadness facilitate spontaneous affect regulation. *Psychophysiology*, 54, 1054-1069. doi: 10.1111/psyp.12856

Stunkard, A. J. (1959). Eating patterns and obesity. *Psychiatric Quarterly*, *33*(2), 284-295.

Stunkard, A. J., Faith, M. S., & Allison, K. C. (2003). Depression and obesity. *Biological Psychiatry*, *54*, 330-337. doi: 10.1016/S0006-3223(03)00608-5

Sulkowski, M. L., Dempsey, J., & Dempsey, A. G. (2011). Effects of stress and coping on binge eating in female college students. *Eating Behaviors*, *12*, 188-191. doi: 10.1016/j.eatbeh .2011.04.006

Surtees, P. G., Wainwright, N. W., Luben, R., Wareham, N. J., Bingham, S. A., & Khaw, K. T. (2010). Mastery is associated with cardiovascular disease mortality in men and women at apparently low risk. *Health Psychology*, *29*, 412-420. doi: 10.1037 /a0019432

Svaldi, J., Caffier, D., Blechert, J., & Tuschen-Caffier, B. (2009). Body-related film clip triggers desire to binge in women with binge eating disorder. *Behaviour Research and Therapy*, *47*, 790-796. doi: 10.1016/j.brat.2009.06.005

Svaldi, J., Tuschen-Caffier, B., Trentowska, M., Caffier, D., & Naumann, E. (2014). Differential caloric intake in overweight females with and without binge eating: effects of a laboratory-based emotion-regulation training. *Behaviour Research and Therapy*, *56*, 39-46. doi: 10.1016/j.brat.2014.02.008

Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (6th ed.). New York: Pearson.

Tanofsky-Kraff, M., Faden, D., Yanovski, S. Z., Wilfley, D. E., & Yanovski, J. A. (2005). The perceived onset of dieting and loss of control eating behaviors in overweight children. *International Journal of Eating Disorders*, *38*, 112-122. doi: 10.1002/eat.20158

Taylor, S. E., & Brown, J. D. (1988). Illusion and well-being: a social psychological perspective on mental health. *Psychological Bulletin*, *103*, 193-210. doi: 10.1037/ 0033-2909.103 .2.193

Taylor, S. E., Kemeny, M. E., Reed, G. M., Bower, J. E., & Gruenewald, T. L. (2000). Psychological resources, positive illusions, and health. *American Psychologist, 55*, 99-109. doi: 10.1037/0003-066X.55.1.99

Telch, C. F., & Agras, W. S. (1996). Do emotional states influence binge eating in the obese? *International Journal of Eating Disorders, 20*, 271-279. doi: 10.1002/ (SICI)1098-108X(199611)20:3<271::AID-EAT6>3.0.CO;2-L

Telch, C. F., Pratt, E. M., & Niego, S. H. (1998). Obese women with binge eating disorder define the term binge. *International Journal of Eating Disorders, 24*, 313-317. doi: 10.1002 /(SICI)1098-108X(199811)24:3<313::AID-EAT9>3 .0.CO;2-P

Terry, D. J., & O'Leary, J. E. (1995). The theory of planned behaviour: The effects of perceived behavioural control and self-efficacy. *British Journal of Social Psychology, 34*, 199-220. doi: 10.1111/j.2044-8309.1995.tb01058.x

Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoeneuroendocrinology, 30*, 1050-1058. doi: 10.1016 /j.psyneuen.2005.04 .014

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology, 141,* 122-131. doi: 10.1016/j.ijcard.2009 .09.543

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: if you feel bad, do it!. *Journal of Personality and Social Psychology, 80*, 53-67. doi: 10.1037//0022-3514.80.1.53

Tiggemann, M., & Lynch, J. E. (2001). Body image across the life span in adult women: the role of self-objectification. *Developmental Psychology, 37*, 243-253. doi: 10 .1037/0012-1649.37.2.243

Timmerman, G. M. (1999). Binge eating scale: Further assessment of validity and reliability. *Journal of Applied Biobehavioral Research, 4*, 1-12. doi: 10.1111 /j.1751-9861.1999 .tb00051.x

Tomba, E., Offidani, E., Tecuta, L., Schumann, R., & Ballardini, D. (2014). Psychological well-being in out-patients with eating disorders: A controlled study. *International Journal of Eating Disorders, 47*, 252-258. doi: 10.1002/eat.22197

Turner, R. J., & Noh, S. (1988). Physical disability and depression: A longitudinal analysis. *Journal of Health and Social Behavior, 29*(1), 23-37. Retrieved from http://www.jstor .org.prxy4.ursus.maine.edu/stable/2137178

Udo, T., Grilo, C. M., Brownell, K. D., Weinberger, A. H., DiLeone, R. J., & McKee, S. A. (2013). Modeling the effects of positive and negative mood on the ability to resist eating in obese and non-obese individuals. *Eating Behaviors, 14*, 40-46. doi: 10.1016 /j.eatbeh.2012.10.010

van der Zanden, R., Galindo-Garre, F., Curie, K., Kramer, J., & Cuijpers, P. (2014). Online cognitive-based intervention for depression: Exploring possible circularity in mechanisms of change. *Psychological Medicine, 44*, 1159-1170. doi: 10.1017 /S003329171300175X

Vartanian, L. R., Kernan, K. M., & Wansink, B. (2016). Clutter, chaos, and overconsumption: The role of mind-set in stressful and chaotic food environments. *Environment and Behavior*. Advance online publication. doi: 0013916516628178

Voon, V. (2015). Binge eating disorder: From bench to bedside. *CNS Spectrums, 20*, 520-521. doi: 10.1017/S109285291500067X

Wallston, K. A., Wallston, B. S., Smith, S., & Dobbins, C. J. (1987). Perceived control and health. *Current Psychology*, *6*(1), 5-25. Retrieved from http://www.vanderbilt.edu /nursing/kwallston/perceived%20control%20and%20health.pdf

Walsh, B. T., & Boudreau, G. (2003). Laboratory studies of binge eating disorder. *International Journal of Eating Disorders, 34*, S30-S38. doi: 10.1002/eat.10203

Wang, Y., & Beydoun, M. A. (2007). The obesity epidemic in the United States - gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiologic Reviews, 29*, 6-28. doi: 10.1093/epirev/mxm007

Watson, D. (1988). Intraindividual and interindividual analyses of positive and negative affect: Their relation to health complaints, perceived stress, and daily activities. *Journal of Personality and Social Psychology, 54*, 10201030. doi: 10.1037/0022-3514.54.6.1020

Watson, D., & Clark, L. A. (1994). *The PANAS-X: Manual for the Positive and Negative Affect Schedule-Expanded Form*. Ames: The University of Iowa.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063-1070. doi: 10.1037/0022-3514.54.6.1063

Wegner, K. E., Smyth, J. M., Crosby, R. D., Wittrock, D., Wonderlich, S. A., & Mitchell, J. E. (2002). An evaluation of the relationship between mood and binge eating in the natural environment using ecological momentary assessment. *International Journal of Eating Disorders, 32*, 352-361. doi: 10.1002/eat.10086

Werrij, M. Q., Mulkens, S., Hospers, H. J., & Jansen, A. (2006). Overweight and obesity: the significance of a depressed mood. *Patient Education and Counseling*, *62*, 126-131. doi: 10.1016/j.pec.2005.06.016

White, K. M., Terry, D. J., & Hogg, M. A. (1994). Safer sex behavior: The role of attitudes, norms, and control factors. *Journal of Applied Social Psychology*, *24*(24), 2164-2192. doi: 10.1111/j.1559-1816.1994.tb02378.x

Whiteside, U., Chen, E., Neighbors, C., Hunter, D., Lo, T., & Larimer, M. (2007). Difficulties regulating emotions: Do binge eaters have fewer strategies to modulate and tolerate negative affect? *Eating Behaviors, 8*, 162-169. doi: 10.1016/j.eatbeh.2006.04.001

Wilfley, D. E., Schwartz, M. B., Spurrell, E. B., & Fairburn, C. G. (2000). Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. *International Journal of Eating Disorders, 27*, 259-269. doi: 10.1002/(SICI)1098-108X (200004)27:3<259::AID-EAT2>3.0.CO;2-G

Wolff, G. E., Crosby, R. D., Roberts, J. A., & Wittrocks, D. A. (2000). Differences in daily stress, mood, coping, and eating behavior in binge eating and nonbinge eating college women. *Addictive Behaviors, 25*, 205-216. doi: 10.1016/S0306-4603(99)00049-0

Womble, L. G., Williamson, D. A., Martin, C. K., Zucker, N. L., Thaw, J. M., Netemeyer, R., . . . Greenway, F. L. (2001). Psychosocial variables associated with binge eating in obese males and females. *International Journal of Eating Disorders*, *30*, 217-221. doi: 10.1002 /eat.1076

Wood, J. V., Saltzberg, J. A., & Goldsamt, L. A. (1990). Does affect induce self-focused attention? *Journal of Personality and Social Psychology, 58*, 899-908. doi: 10.1037 /0022-3514.58.5.899

Woods, A. M., Racine, S. E., & Klump, K. L. (2010). Examining the relationship between dietary restraint and binge eating: Differential effects of major and minor stressors. *Eating Behaviors, 11*, 276-280. doi: 10.1016/j.eatbeh.2010.08.001

York, K. M., Hassan, M., Li, Q., Li, H., Fillingim, R. B., & Sheps, D. S. (2007). Coronary artery disease and depression: patients with more depressive symptoms have lower cardiovascular reactivity during laboratory-induced mental stress. *Psychosomatic Medicine, 69*, 521-528. doi: 10.1097/PSY.0b013e3180cc2601

Zellner, D. A., Loaiza, S., Gonzalez, Z., Pita, J., Morales, J., Pecora, D., . . . Wolf, A. (2006). Food selection changes under stress. *Physiology & Behavior, 87*, 789-793. doi: 10.1016 /j.physbeh.2006.01.014

APPENDICES

Appendix A: Prescreening Questionnaires

GHS

1. Do you speak and read English fluently? Y / N
2. Are you color-blind? Y / N
3. Have you ever been diagnosed with any learning disabilities that interfere with your ability to read or process visual information? Y / N
4. Have you ever lost consciousness for more than one hour? Y / N
5. Have you ever been diagnosed with any neurological disorder, such as Alzheimer's Disease, Parkinson's Disease, or Huntington's Disease? Y / N
6. Have you ever had a stroke, hemorrhage, or brain tumor? Y / N
7. Have you ever had brain/neural surgery or brain radiation treatment (e.g., for a brain tumor)? Y / N
8. Do you have multiple seizures or Epilepsy? Y / N
9. Have you ever been diagnosed with cardiovascular disease? Heart disease? Hypertension? Medication-dependent diabetes? Y / N
10. Comments:

Appendix B: Recruitment Email

Dear student, you are invited to participate in our study, Experiment ##.

WHAT IS THE STUDY ABOUT?

The study will help us understand how minor changes in mood impact risk for depression.

The study takes two hours, and you will receive two credits.

During the study, you will participate in an interview and complete questionnaires about symptoms of mental illness, like depression and anxiety.

YOU MAY QUALIFY TO COMPLETE SESSION 2 & 3

After the interview, ***you may qualify*** to complete session 2 (complete an attention task and listen to sad music, while we use sensors attached to your chest and back to measure changes in your physiology) and session 3 (complete an online survey).

HOW DO I PARTICIPATE?

Go to Sona and sign up for Experiment ##.

Enter CODE #####

NOTE Please do not share the code with other students.

Thank you, Dr. Emily Haigh

Appendix C: Listserv Emails

Healthy Control Email

Subject Line: Healthy Volunteers needed for Paid Research Study!

The Maine Mood Disorders Lab at the University of Maine, Orono is conducting a study to help us understand how changes in mood impact risk for depression.

WHAT WILL I BE ASKED TO DO?

Participate in an interview and complete questionnaires about your mood and past emotional experiences.

YOU MAY ALSO QUALIFY FOR SESSION 2 and 3:

For Session 2, you will complete an attention task and listen to sad music, while we use sensors attached to your body to measure changes in your physiology).

For Session 3, you will receive an email with a link to complete an online survey.

TO BE ELIGIBLE YOU **MUST:**

- ✓ **Healthy participants <u>WITHOUT</u> a history of depression, anxiety, or other emotional disorder**
- ✓ You are between 18 and 60 years of age
- ✓ speak and read English fluently
- ✓ have color vision
- ✓ not suffer from alcohol abuse or dependence have no known cardiovascular disease

HOW WILL I BE COMPENSATED FOR MY TIME?

- $30 for session 1 (approx. 2 hrs.)
- $15 for session 2 (approx. 1 hr.)
- 1 in 10 chance to win a $25 VISA Gift Card for session 3 (30 min.)

The study takes place at the Department of Psychology Maine Mood Disorders Lab located in Corbett Hall at the University of Maine, Orono.

INTERESTED? TAKE A PHOTO OF THE FLYER!

CALL or TEXT: 207-518-8089

Email: **mainemooddisorderslab@gmail.com**

Say you are interested in **STUDY # 1**

Remitted Depression Email

Subject Line: Volunteers needed for Paid Research Study!

CURRENTLY healthy *with* a PAST history of Depression?

Do you ***currently*** feel like your **normal self (e.g. NOT depressed)?**

In the ***past,*** have you felt down or **depressed for most of the day, nearly every day for at least two weeks?**

The Maine Mood Disorders Lab at the University of Maine, Orono is conducting a study to help us understand how changes in mood impact risk for depression.

WHAT WILL I BE ASKED TO DO?

Participate in an interview and complete questionnaires about your mood and past emotional experiences.

YOU MAY ALSO QUALIFY FOR SESSION 2 and 3:

For Session 2, you will complete an attention task and listen to sad music, while we use sensors attached to your body to measure changes in your physiology.

For Session 3, you will receive an email with a link to complete an online survey.

TO BE ELIGIBLE YOU **MUST:**

- ✓ Currently healthy participants WITH a history of depression
- ✓ You are between 18 and 60 years of age
- ✓ speak and read English fluently
- ✓ have color vision
- ✓ not suffer from alcohol abuse or dependence have no known cardiovascular disease

HOW WILL I BE COMPENSATED FOR MY TIME?

- $30 for session 1 (approx. 2 hrs.)
- $15 for session 2 (approx. 1 hr.)
- 1 in 10 chance to win a $25 VISA Gift Card for session 3 (30 min.)

INTERESTED? TAKE A PHOTO OF THE FLYER!

CALL or TEXT: 207-518-8089

Email: **mainemooddisorderslab@gmail.com**

Say you are interested in **STUDY # 2**

Appendix D: Community Flyers

Healthy Control Flyer

Healthy Volunteers needed for Paid Research Study

The Maine Mood Disorders Lab at the University of Maine, Orono is conducting a study to help us understand how changes in mood impact risk for depression.

WHAT WILL I BE ASKED TO DO?

Participate in an interview and complete questionnaires about your mood and past emotional experiences.

YOU MAY ALSO QUALIFY FOR SESSION 2 and 3:

For Session 2, you will complete an attention task and listen to sad music, while we use sensors attached to your body to measure changes in your physiology).

For Session 3, you will receive an email with a link to complete an online survey.

TO BE ELIGIBLE YOU **MUST:**

- ✓ Healthy participants WITHOUT a history of depression, anxiety, or other emotional disorder
- ✓ You are between 18 and 60 years of age
- ✓ speak and read English fluently
- ✓ have color vision
- ✓ not suffer from alcohol abuse or dependence have no known cardiovascular disease

HOW WILL I BE COMPENSATED FOR MY TIME?

- $30 for session 1 (approx. 2 hrs.)
- $15 for session 2 (approx. 1 hr.)
- 1 in 10 chance to win a $25 VISA Gift Card for session 3 (30 min.)

The study takes place at the Department of Psychology Maine Mood Disorders Lab located in Corbett Hall at the University of Maine, Orono.

INTERESTED? TAKE A PHOTO OF THE FLYER!

CALL or TEXT: 207-518-8089

Email: **mainemooddisorderslab@gmail.com**

Say you are interested in **STUDY # 1**

Remitted Depression Flyer

Volunteers needed for Paid Research Study

CURRENTLY healthy *with* a PAST history of Depression?

Do you ***currently*** feel like your **normal self (e.g. NOT depressed)?**

In the ***past,*** have you felt down or **depressed for most of the day, nearly every day for at least two weeks?**

The Maine Mood Disorders Lab at the University of Maine, Orono is conducting a study to help us understand how changes in mood impact risk for depression.

WHAT WILL I BE ASKED TO DO?

Participate in an interview and complete questionnaires about your mood and past emotional experiences.

YOU MAY ALSO QUALIFY FOR SESSION 2 and 3:

For Session 2, you will complete an attention task and listen to sad music, while we use sensors attached to your body to measure changes in your physiology.

For Session 3, you will receive an email with a link to complete an online survey.

TO BE ELIGIBLE YOU **MUST:**

- ✓ Currently healthy participants WITH a history of depression
- ✓ You are between 18 and 60 years of age
- ✓ speak and read English fluently
- ✓ have color vision
- ✓ not suffer from alcohol abuse or dependence have no known cardiovascular disease

HOW WILL I BE COMPENSATED FOR MY TIME?

- $30 for session 1 (approx. 2 hrs.)
- $15 for session 2 (approx. 1 hr.)
- 1 in 10 chance to win a $25 VISA Gift Card for session 3 (30 min.)

INTERESTED? TAKE A PHOTO OF THE FLYER!

CALL or TEXT: 207-518-8089

Email: **mainemooddisorderslab@gmail.com**

Say you are interested in **STUDY # 2**

Appendix E: Community Phone Screen

MMDL Lab Phone Screen

Thank you so much! The brief interview you are about to begin will be used solely for the purpose of determining if you are a good fit for this study. If you agree to participate in this phone interview, you will be asked detailed information about your mental health and alcohol and drug use. You don't have to answer anything you don't want to.

Your participation in this phone interview is entirely voluntary; there will be no payment for participating in this phone interview.

Following our conversation today, if you do not qualify for this study and you are not interested in participating in future research, your name and information will be destroyed. If you do not qualify for this particular study but are interested in possibly participating in future studies, we will ask you if we may keep your name and contact information on file, but will destroy all other information that you provide during the phone interview.

Do you have any questions before we begin?

Question	Answer	Eligibility Criterion
What gender do you identify with?	MALE FEMALE	
What is your date of birth?	/ /	Ages 18-60 (1949-1998)
Are you a native English speaker? [If NO: How long have you spoken English?]	YES NO	Native English speaker [If communicating in English over the phone is problematic, Exclude]
Are you color-blind?	YES NO	Not color-blind [If YES, EXCLUDE]
Have you ever been diagnosed with any learning disabilities? [if NO: Any difficulties reading a magazine? Any difficulties reading subtitle when watching a movie?]	YES NO YES NO	No dyslexia, other reading difficulties, or visual processing problems [If YES <u>and</u> it is severe enough to interfere with cognitive tasks or reading subtitles, EXCLUDE]

Have you ever received an injury or trauma to your head?	YES NO	If significant head injury, EXCLUDE
Have you ever lost consciousness? **[IF YES**: ask when it occurred, and duration of Loss of Consciousness]	YES NO	**INELIGIBLE** LOC > 1 hr if beyond 1 year ever.
Have you ever been diagnosed with any neurological disorder, such as Alzheimer's Disease, Parkinson's Disease, Huntington's Disease?	YES NO	**IF YES**: Exclude
Have you ever had a stroke, hemorrhage, or brain tumor?	YES NO	**IF YES**: Exclude
Have you ever had brain/neural surgery or brain radiation treatment (e.g. for brain tumor)?	YES NO	**IF YES**: Exclude
Do you have seizures or Epilepsy? [If YES, ask about severity, frequency and medication]	YES NO	If multiple seizures or have Epilepsy, exclude.
Have you ever been diagnosed with cardiovascular disease? Heart disease? Hypertension? Medication-dependent diabetes?	YES NO	**IF YES**: Exclude

If they meet EXCLUSION criteria:

[Go to page 12, and use the Ineligible script.]

If they do not meet any of the above exclusion criteria:

OK, great. Now, I'm going to ask you several questions that are a more sensitive and personal than the ones I've just asked. Again, everything you say will be kept strictly confidential and you may choose to skip questions.

OK, first I would like to ask you some questions about your general health.

Are you currently in treatment for any emotional problems? What about treatment for misuse of any substances (e.g., alcohol)? If yes to either one: Why did you seek treatment? Were you offered a diagnosis?	YES NO YES NO	Note: Listen for evidence of bipolar disorder, psychotic symptoms, schizophrenia, substance abuse in past 6 months Comorbidity of non-study diagnoses OK
Are you currently taking <u>any</u> medications on a <u>daily</u> basis?	Med 1 _____ Med 2 _____ Med 3 _____	Med 4 _____ Med 5 _____ Med 6 _____ Med 7 _____ *<u>Meds not exclusion criteria</u>, but note if medication is an antipsychotic, beta blocker, tricyclic antidepressants, antihistamines (see "Medications to Note" list below for common names of these medications)
Have you received treatment for any emotional problems? [If Yes] Were you offered a diagnosis? What about treatment for alcohol or substance abuse in the past?	YES NO YES NO	
How much alcohol do you drink, on average, per week?	Estimated amount & frequency:	
In the past six months, have you ever had five or more drinks on one occasion? [If YES] How many times?	YES NO YES NO	EXCLUDE if recurrent use resulting in failure to fulfill obligations, in legal problems or in social or interpersonal problems or use in physically

Did your drinking cause problems for you or did other people comment it?		hazardous situations.
In the past six months have you used any street drugs or have you gotten hooked on a prescribed medication?	YES NO	If excessive or causing impairments, EXCLUDE
Have you ever experienced a period of several days or more when you were feeling so good, "high," hyper, or excited that other people thought you were not your normal self? Did anyone say you were manic? Was that more than feeling good? If NO: What about a period of time when you were so irritable that you found yourself shouting at people or starting fights or arguments? (what about with people you didn't really know?) If YES for EITHER of above: How long did it last?	YES NO YES NO	-If 4 days, hypomania; if 7 days or hospitalization, mania; continue with following questions
If you suspect mania ask: During that time… 1. How did you feel about yourself? 2. Did you need less sleep than usual? [If YES] Did you still feel rested? 3. Were you much more talkative than usual? 4. Were your thoughts racing	YES NO YES NO YES NO YES NO YES NO	Inflated self-esteem; more self-confident than usual Decreased need for sleep (e.g. feel rested after only three hours sleep) Pressure to keep talking, talking fast

through your head?	YES	NO	Flight of ideas
5. Were you so easily distracted by things around you that you had trouble concentrating or staying on one track?	YES	NO	Attention too easily drawn to unimportant or irrelevant external stimuli
6. How did you spend your time? [if NO] Were you physically restless?			Increase in goal-directed activity or psychomotor agitation
7. Did you do anything that could have caused trouble for you or your family?			Excessive involvement in pleasurable activities that have a high potential for painful consequences

If they seem to be Bipolar I (i.e., unusually elevated mood or irritability for 1 week + 3 more Sx): Exclude. Use the "ineligible" script on page 12.

If they are NOT Bipolar I, continue the phone screen

In the last month…			
A1…has there been a period of time when you were feeling depressed or down most of the day nearly every day? (What was that like?) How long did it last? (As long as two weeks?)	YES	NO	**To be eligible to participate at a later time as remitted MDD, they need to have 4 YESs on this page (at least 1 YES needs to be from this box).** **If they have current MDD and otherwise eligible:** use "Unclear" script and ask for contact information to contact them at a later time.
A2…have you lost interest or pleasure in almost all of your daily activities? Was it nearly every day? As long as two weeks?	YES	NO	If YES to either of these questions, go through the next questions.
[If YES to either A1 or A2] In the last month, when was the			

worst two week period?			
During these two weeks:			
1. How was your appetite? ____ days out of these two weeks	YES	NO	Significant weight loss or gain or decreased/increased appetite nearly every day
2. How were you sleeping? ____ days out of these two weeks (quantify # hrs: "normal" vs. current)	YES	NO	insomnia or hypersomnia nearly every day
3. Were you fidgety or restless? [If NO] Were you talking or moving slowly? ____ days out of these two weeks	YES	NO	psychomotor agitation or retardation nearly every day
	YES	NO	fatigue or loss of energy nearly every day
	YES	NO	feelings of worthlessness or excessive or inappropriate guilt nearly every day
4. What was your energy like? ____ days out of these two weeks	YES	NO	trouble thinking or making decisions nearly everyday
5. How did you feel about yourself? [If NO] Did you feel guilty about things you've done or not done? ____ days out of these two weeks			
6. Did you have trouble			

concentrating? [If NO] Hard to make decisions about everyday things? ____ days out of these two weeks		

To be eligible to participate in studies as remitted MDD, they need to have 4 YESs on the symptoms listed under "Nearly every day" AND at least 1 YES needs to be from one of the first two questions on past depression.		
HAVE YOU EVER had a period of time when you were feeling depressed or down most of the day nearly every day? (When was this? What was that like?)	YES NO	NOTE: YES requires 2-week period of nearly continuous depressed mood.
How long did it last? (As long as two weeks?)	YES NO If YES to either one: Impairment/Distress?	NOTE: YES requires 2-week period of markedly diminished interest or pleasure.
HAVE YOU EVER had a period of time in which you lost interest or pleasure in almost all of your daily activities? Was it nearly every day? As long as two weeks?		If YES to either of these questions + a total of 5 symptoms: **<u>Nearly every day:</u>** Significant weight loss or gain or decreased/increased appetite; insomnia or hypersomnia; fatigue or loss of energy; psychomotor agitation or retardation; feelings of worthlessness or guilt; trouble thinking or making decisions

For possible CONTROLS ONLY: **[If "yes" for any of the following questions, ask for <u>impairment/distress</u> (e.g., In what ways does it interfere with your life? How much has the fact that (*e.g., you are afraid of spiders, you experience panic attacks, etc.)* bothered you?)]**		
Have you ever had a panic attack, when you suddenly felt frightened or anxious or suddenly developed a lot of physical symptoms?	YES NO If YES: Impairment/Distress?	Recurrent, unexpected attacks that peak within 10 minutes, and are accompanied by at least one month of worry (that there is something terribly wrong or about having another one) or behavioral change
Were you ever afraid of going out of the house alone, being in crowds, standing in a line, or traveling on buses or trains?	YES NO If YES: Impairment/Distress?	Avoidance of or marked distress in enduring situations where escape might be difficult or embarrassing, often accompanied by limited-symptom panic attacks or requiring presence of a companion
Are there any other things that you have been especially afraid of, like flying, seeing blood, getting a shot, heights, closed places, or certain kinds of animals or insects?	YES NO If YES: Impairment/Distress?	Marked and persistent fear cued by presence/anticipation of a specific object or situation; exposure invariably provokes anxiety; person must recognize fear is excessive/unreasonable
Have you ever been bothered by thoughts that didn't make any sense and kept coming back to you even when you tried not to have them?	YES NO If YES: Impairment/Distress?	Recurrent and persistent thoughts or impulses experienced as intrusive and inappropriate that cause marked anxiety or distress and are accompanied by attempts to suppress/ignore them

Was there ever anything that you had to do over and over again and couldn't resist doing , like washing your hands again and again, counting up to a certain number, or checking something several times to make sure that you'd done it right?	YES NO If YES: Impairment/Distress?	Repetitive behaviors or mental acts that person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly; behaviors are aimed at reducing distress or preventing a dreaded event, or are clearly obsessive; are time-consuming or cause marked interference with functioning
In the last six months, have you been particularly nervous or anxious?	YES NO If YES: Impairment/Distress?	Excessive anxiety/worry that is difficult to control and is accompanied by physical symptoms more days than not for at least six months
Have you ever had a time when you weighed much less than other people thought you ought to weigh?	YES NO If YES: Impairment/Distress?	Refusal to maintain body weight above a minimally normal weight, accompanied by intense fear of becoming fat; absence of at least three consecutive menstrual cycles
Have you often had times when your eating was out of control?	YES NO If YES: Impairment/Distress?	Recurrent episodes of binge eating accompanied by marked distress at least 2x/wk for 3-6 months; may include recurrent inappropriate compensatory behavior to prevent weight gain

FINISHING THE INTERVIEW:

"Thank you very much for answering all of these questions. I would like to invite you to come to the University of Maine to participate in Session 1. If you are interested, we can set up a time right now." If they are unsure of their schedule at the time: "*That's fine. We can call you later. When would be a good time for us to call to schedule you for Session 1?*" If participant would like to call back, give them our phone number, (207) 518-8089.

"Thank you very much for your time and for answering these questions. I am just looking over the interview now, and unfortunately it looks as though you are not going to be eligible for this particular study. However, would you like us to keep your contact information to contact you if we have any new studies in the future?"

____ Yes _____ No

If **Yes**:

- Get their full name, phone number(s), and email address, note in tracking spreadsheet.]

Name: _____

Phone number: _____

Email: _____

- Shred phone screen

If **No**, say "*Thank you again for your time*"

- Remove identifying info from tracking spreadsheet
- Shred the phone screen

<u>If participant wants more information about why they are not eligible:</u>

Explain that there is not any ONE thing that makes them ineligible. Say that we are looking for very specific profiles across a host of different criteria and unfortunately their profile is not a match with any of the detailed profiles that we are looking for. **If they are not satisfied, you can always tell them that you will refer them to your supervisor.** If you have questions about this phone interview, please call my supervisor Dr. Emily Haigh at (207) 581-2053.

I want to thank you VERY MUCH for your time and for answering all of these questions. I will have my supervisor go over the protocol, and then I will give you a call in the next few days to let you know whether or not you are eligible for this particular study.

****Note** **1. If you are concerned about someone, notify Dr. Haigh immediately.**

2. For any participants who may be interested in seeking treatment: Give them appropriate referrals from the referral list.

Appendix F: Prorated Community Payment

Session 1:

Up to ½ hour	$8.00
½ hour to 1 hour	$15.00
1 hour to 1 ½ hours	$23.00
1 ½ hours to 2 hours (or session completion)	$30.00

Session 2:

Up to ½ hour	$8.00
½ hour to 1 hour (or session completion)	$15.00

Appendix G: Session 1 Questionnaires

BDI-II

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 have been feeling during 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 for that group. Be sure that you do 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 than I used to be.
   - 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.

9. Suicidal Thoughts or Wishes
   - 0 I don't have any thoughts of killing

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 have.
   - 2 I don't have enough energy to do very much.
   - 3 I don't have enough energy to do anything.

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.

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.

11. Agitation
0 I am no more restless or would up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that 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.

18. Changes in Appetite

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

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater 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.

BES

INSTRUCTIONS: Below are groups of numbered statements. Read all of the statements in each group and circle the letter next to the statement that best describes the way you feel about the problems you have controlling your eating behavior.

#1

(a) I don't feel self-conscious about my weight or body size when I'm with others.
(b) I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
(c) I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
(d) 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

(a) I don't have any difficulty eating slowly in the proper manner.
(b) Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
(c) At times, I tend to eat quickly and then, I feel uncomfortably full afterwards.
(d) 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

(a) I feel capable to control my eating urges when I want to.
(b) I feel like I have failed to control my eating more than the average person.
(c) I feel utterly helpless when it comes to feeling in control of my eating urges.
(d) Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

#4

(a) I don't have the habit of eating when I'm bored.
(b) I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
(c) 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.
(d) I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

#5

(a) I'm usually physically hungry when I eat something.
(b) Occasionally, I eat something on impulse even though I really am not hungry.
(c) 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.
(d) 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

(a) I don't feel guilt or self-hate after I overeat.
(b) After I overeat, occasionally I feel guilt or self-hate.
(c) Almost all the time I experience strong guilt or self-hate after I overeat.

#7

(a) I don't lose total control of my eating when dieting even after periods when I overeat.
(b) Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
(c) 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.
(d) 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

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

#9

(a) My level of calorie intake does not go up very high or go down very low on a regular basis.
(b) Sometimes after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten.
(c) 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.
(d) 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

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

#11

(a) I don't have any problem stopping eating when I feel full.
(b) I usually can stop eating when I feel full but occasionally overeat leaving me feeling uncomfortably stuffed.
(c) I have a problem stopping eating once I start and usually I feel uncomfortably stuffed after I eat a meal.
(d) 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

(a) I seem to eat just as much when I'm with others (family, social gatherings) as when I'm by myself.
(b) 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.
(c) Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.
(d) 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

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

#14

(a) I don't think much about trying to control unwanted eating urges.
(b) At least some of the time, I feel my thoughts are pre-occupied with trying to control my eating urges.
(c) I feel that frequently I spend much time thinking about how much I ate or about trying not to eat anymore.
(d) 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

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

#16

(a) I usually know whether or not I'm physically hungry. I take the right portion of food to satisfy me.
(b) 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.
(c) 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.

Perceived Mastery Scale

INSTRUCTIONS: How strongly do you agree or disagree that:

0	1	2	3	4	5	6
Strongly Disagree			Neither Agree nor Disagree			Strongly Agree

____1. I have little control over the things that happen to me.

____2. There is really no way I can solve some of the problems I have.

____3. There is little I can do to change many of the important things in life.

____4. Often I feel helpless in dealing with the problems of life.

____5. Sometimes I feel like I am being pushed around in life.

____6. What happens to me in the future mostly depends on me.

____7. I can do about anything I really set my mind to do.

PSS-10

INSTRUCTIONS: The questions in this scale ask you about your feelings and thoughts during the last month. In each case, please indicate how often you felt or thought a certain way.

0----------1----------2----------3----------4
Never Almost Never Sometimes Fairly Often Very Often

____1. In the last month, how often have you been upset because of something that happened unexpectedly?

____2. In the last month, how often have you felt that you were unable to control the important things in your life?

____3. In the last month, how often have you felt nervous and "stressed"?

____4. In the last month, how often have you felt confident about your ability to handle your personal problems?

____5. In the last month, how often have you felt that things were going your way?

____6. In the last month, how often have you found that you could not cope with all the things that you had to do?

____7. In the last month, how often have you been able to control irritations in your life?

____8. In the last month, how often have you felt that you were on top of things?

____9. In the last month, how often have you been angered because of things that were outside of your control?

____10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

PANAS-X

INSTRUCTIONS: This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have felt this way during the past two weeks. Use the following scale to record your answers:

1	2	3	4	5
Very Slightly or Not at All	A Little	Moderately	Quite of Bit	Extremely

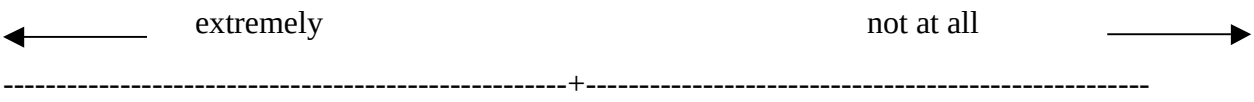
_____ cheerful	_____ sad	_____ active	_____ angry at self
_____ disgusted	_____ calm	_____ guilty	_____ enthusiastic
_____ attentive	_____ afraid	_____ joyful	_____ downhearted
_____ bashful	_____ tired	_____ nervous	_____ sheepish
_____ sluggish	_____ amazed	_____ lonely	_____ distressed
_____ daring	_____ shaky	_____ sleepy	_____ blameworthy
_____ surprised	_____ happy	_____ excited	_____ determined
_____ strong	_____ timid	_____ hostile	_____ frightened
_____ scornful	_____ alone	_____ proud	_____ astonished
_____ relaxed	_____ alert	_____ jittery	_____ interested
_____ irritable	_____ upset	_____ lively	_____ loathing
_____ delighted	_____ angry	_____ ashamed	_____ confident
_____ inspired	_____ bold	_____ at ease	_____ energetic
_____ fearless	_____ blue	_____ scared	_____ concentrating
_____ disgusted	_____ shy	_____ drowsy	_____ dissatisfied with self with self

Appendix H: Session 2 Questionnaires

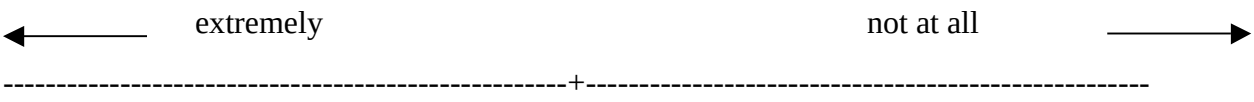
VAS

INSTRUCTIONS: We are interested in knowing about your current mood. Please mark an 'X' on the line below to indicate how you feel right now. Use the labels above the line to help you in your judgment.

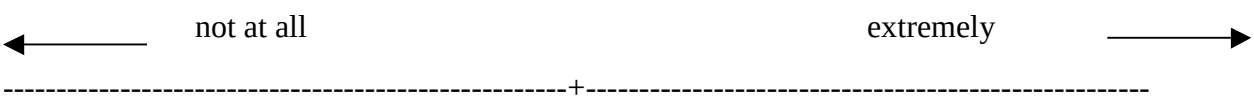
Sadness



Stressed



Happy



Appendix I: Session 1 Consent Forms

Attention and Elaboration Study: Session 1
The University of Maine at Orono

Informed Consent Document- (PSY 100, 212)

You are invited to participate in a research project being conducted by Dr. Emily Haigh, in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

If you decide to participate, you will complete an online survey and an interview in the lab. As part of the online survey you will answer questions about how you're feeling (e.g. *"After I overeat, occasionally I feel guilt or self-hate.")* and different types of thoughts that people sometimes have (e.g., *"I worry about making mistakes" or "I do not need the approval of other people in order to be happy*"). This portion of the study will take about 30-minutes total.

Next, you will participate in an interview. During the interview, you will be asked about your mood (e.g., "*In the past month, have you been feeling depressed or down?*") and different symptoms that are related to disorders like depression and anxiety (e.g., "*In the past month, have you had trouble sleeping?*") The interview will take about 1.5 hours. With your consent, we will audio-record the interview. The audio-record will be used to confirm that the interview was conducted properly by the researcher. Even if you agree to be audio-recorded, you may ask us to stop or destroy the audio file at any time during or after the study is completed. After the interview, a graduate student will measure your height and weight.

Based on information gathered during the interview and questionnaires, some participants will be asked to take part in a second part of the study. If you are eligible and decide to participate in the second part, you will be scheduled for another session that will take place on a different day.

During the second part of the study, you will be given a description of the study and asked to give consent for the procedures involved. Briefly, you will be asked to participate in physiological recording (sensors to detect electrical impulses will be attached to your chest and back) while you complete the following: self-report questionnaires, a computerized attention task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood.

Participants that complete the second portion of the study will be invited to complete a third and final portion of the study. For this part of the study, you will receive an email with a link to some questions about your mood and whether you have experienced any recent stressful events.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about how experiencing brief sad mood relates to depression.

Compensation

You will receive 1 research credit for each hour of participation. Since the interview is expected to take 1.5 hours and the survey is expected to take 30-minutes, it is likely that you will earn 2 credits today.

Confidentiality

Your name will not appear on any of the documents. A code number will be used to protect your identity. This code is stored on a file with software designed to provide added security. Data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh, Maine Mood Disorders Lab graduate students, and research assistants who have been trained to deal with sensitive material. Your name or other identifying information will not be reported in any publications. The key linking your name to the data will be destroyed two years after data analysis is complete, which we anticipate will be in 2018. All data, including audio recordings, will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer. You will earn 1 credit for each hour of participation with the possibility of earning 2 credits today.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 or via e-mail gayle.jones@umit.maine.edu.

Audiotaping
I agree to audio recording the interview.

☐ Yes ☐ No

Future Studies
Would you be interested in being contacted for future studies conducted in the lab for monetary compensation?

☐ Yes ☐ No

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____
Signature Date

Attention and Elaboration Study: Session 1
The University of Maine at Orono

Informed Consent Document- (Community Participants)

You are invited to participate in a research project being conducted by Dr. Emily Haigh, in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

If you decide to participate, you will complete an online survey and an interview in the lab. As part of the online survey you will answer questions about how you're feeling (e.g. "After I overeat, occasionally I feel guilt or self-hate.") and different types of thoughts that people sometimes have (e.g., *"I worry about making mistakes" or "I do not need the approval of other people in order to be happy").* This portion of the study will take about 30-minutes total.

Next, you will participate in an interview. During the interview, you will be asked about your mood (e.g., "*In the past month, have you been feeling depressed or down?*") and different symptoms that are related to disorders like depression and anxiety (e.g., "*In the past month, have you had trouble sleeping?*"). The interview will take about 1.5 to 2 hours. With your consent, we will audio-record the interview. The audio-record will be used to confirm that the interview was conducted properly by the researcher. Even if you agree to be audio-recorded, you may ask us to stop or destroy the audio file at any time during or after the study is completed. After the interview, a graduate student will measure your height and weight.

Based on information gathered during the interview and questionnaires, some participants will be asked to take part in a second part of the study. If you are eligible and decide to participate in the second part, you will be scheduled for another session that will take place on a different day.

During the second part of the study, you will be given a description of the study and asked to give consent for the procedures involved. Briefly, you will be asked to participate in physiological recording (sensors to detect electrical impulses will be attached to your chest and back) while you complete the following: self-report questionnaires, a computerized attention task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood.

Participants that complete the second portion of the study will be invited to complete a third and final portion of the study. For this part of the study, you will receive an email with a link to some questions about your mood and whether you have experienced any recent stressful events.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about experiencing how brief sad mood relates to depression.

Compensation

You will receive $30 for participating in this research session to compensate you for your time and travel expenses. If you do not complete the session you will receive compensation pro-rated to the nearest half hour.

Confidentiality

Your name will not appear on any of the documents. A code number will be used to protect your identity. This code is stored on a file with software designed to provide added security. Data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh, Maine Mood Disorders Lab graduate students and research assistants who have been trained to deal with sensitive material. Your name or other identifying information will not be reported in any publications. The key linking your name to the data will be destroyed in about two years after data analysis is complete, which we anticipate will be in 2018. All data, including audio recordings, will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 or via e-mail at gayle.jones@umit.maine.edu.

Audiotaping
I agree to audio recording the interview.

☐ Yes ☐ No

Future Studies
Would you be interested in being contacted for future studies conducted in the lab for monetary compensation?

☐ Yes ☐ No

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____
Signature Date

Appendix J: Counseling Resources

Counseling Resource List

If you feel upset after having completed the study or find that some questions or aspects of the study triggered distress, talking with a qualified clinician may help. The following represents a list of resources that you may contact. These resources are options and in no way do they reflect an endorsement by the University of Maine.

Counseling Services		
ON-CAMPUS RESOURCES Available for UMaine Faculty, Staff, and Students		
Counseling Center **Cutler Health Building (Gannet Hall side)** **(FREE to UMaine students)**	**207-581-1392** http://www.umaine.edu/counseling/	Weekdays 8:00 am-4:30 pm After business hours, call UMaine Police, 581-4040 or 911
Psychological Services Center **330 Corbett Hall** (Sliding fee scale; costs are your responsibility)	**207-581-2034** http://umaine.edu/clinicalpsychology/psychological-services-center/	Weekdays 8:00 am – 4:30 pm
COMMUNITY RESOURCES Available to Anyone		
Community Health & Counseling Services **42 Cedar Street** **Bangor, ME 04401** (Any costs are your responsibility)	**207-947-0366** http://www.chcs-me.org/	Weekdays 8:00 am-5:00 pm
Maine Warm Line (Any costs are your responsibility)	**1-888-771-9276** http://www.thecommunityconnector.org/directory/profile/maine-warm-line	7 days/week 5:00 pm – 8:00 am

Maine Suicide and Crisis Hotline (Any costs are your responsibility)	**1-888-568-1112** http://www.maine.gov/suicide/youth/index.htm	7 days/week 24 hours
Psychological Services Center **330 Corbett Hall** (sliding fee scale)	**207-581-2034** http://umaine.edu/clinicalpsychology/psychological-services-center/	Weekdays 8:00 am – 4:30 pm
Contact Your Primary Care Provider (Any costs are your responsibility)		

NATIONAL RESOURCES
Mental Health Services Locator http://store.samhsa.gov/mhlocator
National Suicide Prevention Lifeline, Toll-Free, 24-hour Hotline, **1-800-273-TALK (1800-273-8255)**

Appendix K: Session 2 Consent Forms

Attention and Elaboration Study: Session 2
The University of Maine at Orono

Informed Consent Document- (PSY 100, 212)

You are invited to participate in a research project being conducted by Dr. Emily Haigh in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

A trained female research assistant will place sensors on your body in order to record electrical activity of the heart, skin, and facial muscle groups. Once the sensors are placed on your body, you will be asked to sit comfortably in front of a computer in a small room. You will then be asked to complete the following tasks: watch a short video about Alaska's Denali Mountain, answer some questions about how you're feeling (e.g. check a box to indicate whether you are *interested, upset, nervous*), complete a short computer task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood. This portion of the study will take approximately 1-hour total.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. Upon completion of the session, all participants will be given a list of referrals which will include a variety of mental health resources in the area.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about how experiencing brief sad mood relates to depression.

Compensation
You will receive 1 research credit for your participation.

Confidentiality

The code number assigned during the interview will again be used to protect your identity. This code is stored on a file with software designed to provide additional security. All data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh and Maine Mood Disorders Lab graduate students and research assistants who have completed training in order to deal with sensitive material. Your name or other identifying information will

not be reported in any publications. As previously described, the key linking your name to the data will be destroyed in approximately two years after data analysis is complete, which we anticipate will be in 2018. All data will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time or skip any questions you do not wish to answer and still receive the 1 research credit.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 (or e-mail gayle.jones@umit.maine.edu).

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Attention and Elaboration Study: Session 2
The University of Maine at Orono

Informed Consent Document- (Community Participants)

You are invited to participate in a research project being conducted by Dr. Emily Haigh in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

A trained female research assistant will place sensors on your body in order to record electrical activity of the heart, skin, and facial muscle groups. Once the sensors are placed on your body, you will be asked to sit comfortably in front of a computer in a small room. You will then be asked to complete the following tasks: watch a short video about Alaska's Denali Mountain, answer some questions about how you're feeling (e.g. check a box to indicate whether you are *interested, upset, nervous*), complete a short computer task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood. This portion of the study will take approximately 1-hour total.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

This study will have no direct benefit to you, though it will help to better understand how individuals process emotional information and how this relates to risk for depression.

Compensation

You will receive $15 for your participation.

Confidentiality

The code number you have been assigned during session 1 will again be used to protect your identity. This code is stored on a file with software designed to provide additional security. All data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh and Maine Mood Disorders Lab graduate students and research assistants who have completed training in order to deal with sensitive material. Your name or other identifying information will not be reported in any publications. As previously described, the key linking

your name to the data will be destroyed in approximately two years after data analysis is complete, which we anticipate will be in 2018. All data will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary
Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer. You will receive $15 for participating in this research session to compensate you for your time and travel expenses. If you do not complete the session you will receive compensation pro-rated to the nearest half hour.

Contact Information
If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 (or e-mail gayle.jones@umit.maine.edu).

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Appendix L: Debriefing Form

Debriefing Form for Participation in a Research Study
University of Maine

Thank you for your participation in our study. Your participation is greatly appreciated.

Purpose of the Study:

The purpose of this study is to examine how the way you think and the way your body physiologically responds (e.g. heart rate) to emotional stimuli relates to depression. This study is important because it may help us understand how short periods of sad mood lead some individuals to develop lasting depressed mood.

In this study you completed an interview and several questionnaires about how you think and feel. You also completed an attention task (e.g. computer task) and using sensors to detect electrical impulses we measured physiological arousal (e.g. heart rate) as you listened to music designed to either make you feel sad or no change in your mood.
We expect to find that participants with a history of depression who completed an attention task with negative words and listened to the sad music will report more sad mood and have a stronger physiological response than individuals without a history of depression. Previous research has shown that individuals with depression have difficulty turning their attention away from negative stimuli and have negative repetitive thoughts in response to sad mood; however, little research has examined how these factors relate to physiological functioning.

Do you have any questions about the study? When you were doing the study what did you think the study was about? Was there any part of the study that was difficult? How is your mood now?

We realize that some of the questions asked may have provoked an emotional reaction. As researchers, we do not provide mental health services and we will not be following up with you after the study. However, we want to provide every participant in this study with a comprehensive and accurate list of clinical resources that are available, should you decide you need assistance at any time. Please see information pertaining to local resources at the end of this form.

Confidentiality:

You may decide that you do not want your data used in this research. If you would like your data removed from the study and permanently deleted please email your request to Principal Investigator, Dr. Emily Haigh @ Emily.a.haigh@maine.edu.
Whether you agree or do not agree to have your data used for this study, you will still receive compensation for your participation.

Final Report:

If you would like to learn about the results of the study, let the researcher know and we will email you a summary of the results at the end of the study.

Further Reading(s):

If you would like to learn more about cognitive vulnerability to depression please see the following references:

Farb, N. A. S., Irving, J. A., Anderson, A. K., & Segal, Z. V. (2015). A two-factor model of relapse/recurrence vulnerability in unipolar depression. *Journal of Abnormal Psychology*, *124*(1), 38–53. http://doi.org/10.1037/abn0000031

Key, B. L., Campbell, T. S., Bacon, S. L., & Gerin, W. (2008). The influence of trait and state rumination on cardiovascular recovery from a negative emotional stressor. *Journal of Behavioral Medicine*, *31*(3), 237–248. http://doi.org/10.1007/s10865-008-9152-9

Lethbridge, R., & Allen, N. B. (2008). Mood induced cognitive and emotional reactivity, life stress, and the prediction of depressive relapse. *Behaviour Research and Therapy*, *46*(10), 1142–1150. http://doi.org/10.1016/j.brat.2008.06.011

Useful Contact Information:

If you have any questions or concerns regarding this study, its purpose or procedures, or if you have a research-related problem, please feel free to contact the Principal Investigator, Dr. Emily Haigh at 207-581-2053. If you have other concerns about this study or would like to speak with someone not directly involved in the research study, you may contact the Chair of the Department of Psychology (Dr. Michael Robbins, Michael_Robbins@umit.maine.edu)

If you have any questions concerning your rights as a research subject, you may contact Gayle Jones at the University of Maine Institutional Review Board for the Protection of Human Subjects at (207) 581-1498 or gayle.jones@umit.maine.edu.

(Counseling Resource List Attached – see Appendix J)

BIOGRAPHY OF THE AUTHOR

Rachel E. Goetze was born in Bangor, Maine on July 23, 1983. She was raised in Exeter, Maine and graduated from Dexter Regional High School in 2001. Her undergraduate work was completed at the University of Maine, Orono, where she received her Bachelor of Arts degree in Psychology and Bachelor of Arts degree in Social Work. Prior to her graduate studies, Rachel was employed at Eastern Maine Medical Center as a psychometrist, administering neuropsychological evaluations in adult and pediatric populations. During this time, she also conducted research funded by the National Institute of Health (1-R21-CA-143619-01A1). Rachel entered the Clinical Psychology Doctoral Program at the University of Maine during the fall of 2013, with Emily A. P. Haigh, Ph.D. as her advisor. She was awarded her Master of Arts degree in Psychology in 2015 and moved on to doctoral candidacy. During her time at the University of Maine, Rachel co-authored one peer-reviewed article and one book chapter, and was first- or co-author on 21 research presentations at local and national conferences. Rachel was instrumental in establishing Dr. Haigh's research laboratory, the Maine Mood Disorders Lab (MMDL), participated in two experimental research projects (including her dissertation study), and was awarded the Chase Distinguished Research Assistantship for research productivity and academic achievement. During this time, Rachel was also the sole instructor for courses in Abnormal Psychology. She is a student member of Psi Chi National Honor Society in Psychology and an inductee of Phi Kappa Phi Honor Society and Phi Betta Kappa Society. Rachel is a student affiliate of the Association for Behavioral and Cognitive Therapies (ABCT), ABCT Obesity and Eating Disorders Special Interest Group, and the Maine Psychological Association.

Rachel will complete an APA-Accredited Predoctoral Internship in Clinical Psychology with an emphasis in rehabilitation psychology at VA Boston Healthcare System in Boston, Massachusetts in the summer of 2018. After receiving her degree, Rachel will begin a two-year APA-Accredited Postdoctoral Fellowship in Clinical Health Psychology at Mayo Clinic in Rochester, Minnesota with a major emphasis in obesity. Rachel is a candidate for the Doctor of Philosophy degree in Psychology with a concentration in Clinical Psychology from the University of Maine in August 2018.