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Rachael M. Huff  
rachael.huff@maine.edu

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Running head: A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

**A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING: THE ROLE  
OF COGNITIVE RESTRAINT, NEGATIVE AFFECT, AND IMPULSIVITY  
IN BINGE EATING AS A RESPONSE TO STRESS**

By

Rachael M. Huff

B.A., Michigan Technological University, 2014

M.A., University of Maine, 2016

A DISSERTATION

Submitted in Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

(in Clinical Psychology)

The Graduate School

The University of Maine

August 2020

Advisory Committee:

Shannon K. McCoy, Associate Professor of Psychology, Chair

Emily A. P. Haigh, Assistant Professor of Psychology

Shawn W. Ell, Associate Professor of Psychology

Fayeza S. Ahmed, Assistant Professor of Psychology

Mollie A. Ruben, Assistant Professor of Psychology

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

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Dissertation Advisor: Dr. Shannon McCoy

An Abstract of the Dissertation Presented  
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As obesity-related health issues account for the leading number of deaths in America, and these illnesses are even more prevalent in those that binge eat, treating obese individuals with Binge Eating Disorder (BED) has become a matter of life and death. Stress, negative affect, cognitive restraint, and impulsivity have all been implicated in binge eating symptomatology, suggesting that these may be key variables to consider when developing a comprehensive model (Groesz et. al., 2012; Friese et. al., 2015; Galanti et. al., 2007; Gay et al., 2008; Racine et. al., 2015; Leehr et. al., 2015). This project aims to further examine the relationships between these factors in college-age participants and will not be limited to obese participants or those that meet criteria for BED, which has been a limitation of previous research.

Electrocardiograph, impedance cardiography, and blood pressure equipment were used in this project to characterize a maladaptive stress response during a social-evaluative stressor and subsequent cognitive task measuring impulsivity. Self-report measures were administered throughout the lab session, and negative affect and drive to eat were assessed at the end. Information from the laboratory experiment (Study 2) study was sought to build on anonymous

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

self-report data (Study 1) which supported these factors as significant predictors in a proposed comprehensive model of stress-induced binge eating, especially impulsivity. Specifically, Study 2 aimed to replicate Study 1 and to examine whether women higher in binge eating react differently to maladaptive stress than those who are not (i.e., with increased impulsivity, negative affect, and drive to eat).

Results of the self-report data showed that once again, perceived stress predicted increased binge eating through increased impulsivity. Further, the laboratory task demonstrated that for those who are higher (+ *1SD*) in binge eating, more maladaptive stress (i.e., lower HF-HRV) was actually associated with lower negative affect while no relationship was present for others, suggesting a disconnect between conscious emotions and physical reactivity for binge eaters. This project enhances understanding of the etiology of binge eating, allowing clinicians to develop more efficacious treatments and target key factors that will lead to the greatest improvement

## ACKNOWLEDGEMENTS

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**LIST OF ABBREVIATIONS**

ACTH	Corticotropin
ADHD	Attention-Deficit/Hyperactivity Disorder
AN	Anorexia Nervosa
ANS	Autonomic Nervous System
APA	American Psychiatric Association
BCa CI	Bias Corrected and Accelerated Confidence Interval
BDI	Beck Depression Inventory
BDI-II	Beck Depression Inventory – Second Edition
BED	Binge Eating Disorder
BES	Binge Eating Scale
BMI	Body Mass Index
BN	Bulimia Nervosa
BP	Blood Pressure
CBT	Cognitive-behavioral Therapy
CES-D	Center for Epidemiological Studies Depression Scale
CIDI	Composite International Diagnostic Interview
CO	Cardiac Output

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CRH	Corticotropin-releasing Hormone
DSM	Diagnostic and Statistical Manual of Mental Disorders
DSM-5	Diagnostic and Statistical Manual – Fifth Edition
DSM-IV	Diagnostic and Statistical Manual – Fourth Edition
ECG	Electrocardiography
EDE	Eating Disorder Examination
EDE-Q	Eating Disorder Examination-Questionnaire
EEG	Electroencephalogram
EMA	Ecological Momentary Assessment
fMRI	functional Magnetic Resonance Imaging
fNIRS	functional Near-infrared Spectroscopy
FOBES	Functional Assessment of Binge Eating
G-FCQ-S	General Food Cravings Questionnaire-State
HbO	Oxygenated Hemoglobin
HbR	Deoxygenated Hemoglobin
HF-HRV	High-frequency Heart Rate Variability
HP	Heart period



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HPA	Hypothalamic-Pituitary-Adrenocortical
HR	Heart Rate
HRV	Heart Rate Variability
ICG	Impedance Cardiography
ICD	International Classification of Diseases
IRB	Institutional Review Board for the Protection of Human Subjects
K-SADS	Schedule for Affective Disorders and Schizophrenia for School-age Children
LOC	Loss of control eating
MAP	Mean Arterial Pressure
MDD	Major Depressive Disorder
PANAS	Positive and Negative Affect Schedule
PANAS-X	Positive and Negative Affect Schedule – Expanded Form (PANAS-X)
PET	Positron Emission Tomography
PFC	Prefrontal Cortex
PNS	Parasympathetic Nervous System
PSS	Perceived Stress Scale
QEWP	Questionnaire on Eating and Weight Patterns
RSA	Respiratory Sinus Arrhythmia

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RSI	Response-to-stimulus Interval
RT	Reaction Time
SCID-I	Structured Clinical Interview for DSM-IV
SAM	Sympathetic-adrenomedullary
SES	Socioeconomic Status
SNS	Sympathetic Nervous System
SSD	Stop Signal Delay
SSRT	Stop-signal Reaction Time
TFEQ	Three Factor Eating Questionnaire
UPPS	UPPS Impulsive Behavior Scale
UPPS-P	UPPS-P Impulsive Behavior Scale
VAS	Visual Analog Scale
WHO	World Health Organization

**CHAPTER ONE**

**INTRODUCTION**

**1.1. Research Goals and Broader Impacts**

The strong relationship between obesity and BED provides emphasis for the importance of effective treatments for BED as a means of preventing and mitigating obesity-related illnesses. Information from the present study will inform researchers of the relationships between threat-appraised stress, negative affect, cognitive resources, impulsivity and binge eating, building on previously collected self-report data which supported a comprehensive model of stress-induced binge eating. This dissertation will further explore the relationships between these factors in college-age participants and will not be limited to obese participants or those that meet criteria for BED, providing more generalizable findings for those who suffer from subthreshold binge eating. This comprehensive model would impact understanding of the etiology of eating disorders, allowing clinicians to develop more efficacious treatments and target key factors that will lead to the greatest improvement. Studies have shown that when compared to obese individuals without BED, those with BED have an increased risk for obesity-related health problems (Mitchell et. al., 2015; Kessler et. al., 2013). Thus, finding evidence-based treatments for binge eating could mitigate the occurrence of obesity-related illnesses and deaths

## 1.2. Binge Eating Disorder Overview

“As I searched for food perfection, and as I gained weight, I began to realize that the race for perfection in anything was the path to destruction.”

-Rachael Rose Steil, Author of *Running in Silence: My Drive for Perfection and the Eating Disorder That Fed It*.

### 1.2.1. Diagnostic Criteria

Binge Eating Disorder (BED) was recently adopted in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association [APA], 2013) as a distinct eating disorder characterized by overeating and loss of control (LOC) eating. Overeating is defined as eating, in a discrete period of time (typically less than 2 hours), more than would be consumed by the average person in a comparable amount of time. This can be difficult to operationalize as different individuals tend to have varied patterns of calorie consumption, but many researchers have used a threshold of 1,000 calories in one sitting to quantify objective overeating (Racine et. al., 2015). Data on the largest amount of food that women typically eat before classifying it as unusually large, which ranges from 413-1,074 calories, support this cut-off. However, individuals that expend a large number of calories throughout the day (i.e., athletes) may need to eat more in order to maintain their weight, and this could be incorrectly characterized as binge-eating using only the identified threshold. In contrast, distress associated with eating an amount that is subjectively large, but does not meet this threshold, is common when the individual experiences loss of control eating (Colles, Dixon, & O’Brien, 2008). Loss of control eating is a sense that one cannot stop eating or control the amount of food they are eating. This has been shown to be associated with higher emotional

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distress, symptoms of depression, and greater dissatisfaction with appearance, suggesting that subthreshold subjective binge eating can be harmful to one's overall well-being, despite not meeting full criteria for BED.

Both overeating and LOC eating must be present to meet criteria for a binge-eating episode, and individuals with BED typically experience one binge-eating episode per week for at least three months to meet criteria for the disorder (APA, 2013). Other characteristics of BED include eating rapidly, eating in the absence of hunger, eating until uncomfortably full, eating alone due to embarrassment about how much one is eating, and feeling disgusted with oneself, depressed, or very guilty afterward. Individuals must have at least three of these occur in association with their binge eating episodes in order to be diagnosed with BED. Lastly, the individual must experience marked distress regarding their binge-eating, and some describe a dissociative experience during the episode. Although binge-eating includes losing control over one's eating, binge-eating episodes can be planned, and are typically carried out inconspicuously or in secret due to the significant shame associated with this behavior.

Although individuals who only experience loss of control over eating do not meet full criteria for BED, recent studies have shown that LOC eating is more strongly related to psychological distress than binge frequency or size (Colles et al., 2008). Therefore, future research of binge eating etiology and severity may benefit from including samples that do not meet full BED criteria, or that only exhibit subjective LOC eating. As clinical research typically looks at groups with and without clinical disorders and differences among these groups, this approach would create a unique opportunity to take a social psychology perspective, looking at various relevant constructs and behaviors in all individuals, without being confined by diagnostic criteria.

### **1.2.2. Occurrence**

While BED is a newly recognized disorder, it is thought to be more common than other eating disorders, in both men and women (Hudson, Hiripi, Pope, & Kessler, 2007). Notably, prevalence rates of BED vary significantly based on a number of demographic factors, and so one must consider the occurrence of BED overall, as well as for specific groups of individuals. Further, examining the presence of new BED cases can help researchers understand the course of BED within individuals and how its occurrence might change over time. As such, this section aims to detail the occurrence of this newly diagnosed eating disorder in order to more thoroughly understand its impact.

**1.2.2.1. Prevalence.** Prevalence is defined as the number of people that have a disorder in a population at a given point in time, and is typically expressed as point prevalence, lifetime prevalence, or one-year prevalence (Smink, Van Hoeken, & Hoek, 2012). Point prevalence is the number of people with a disorder in a population at a specific point in time. The 12-month prevalence rate is the point prevalence, plus the incidence rate, or number of new cases of the disorder, in the following year. Lastly, the lifetime prevalence is the number of people that had the disorder at any point in their life. The standard procedure for estimating prevalence of eating disorders is a two-stage screening approach in which the first stage identifies at-risk individuals using a self-report questionnaire. In the second stage, definite cases are established via personal diagnostic interview. The World Health Organization Composite International Diagnostic Interview (CIDI) is typically used, as it is a semi-structured, lay-person interview designed to provide International Classification of Diseases (ICD) and Diagnostic and Statistical Manual of Mental Disorders (DSM) diagnoses.

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Prevalence of BED has been assessed in a variety of population samples. In a large nationally representative population sample ( $N = 9282$ ) in the U.S., lifetime prevalence was 3.5% among adult women and 2.0% among adult men based on proposed Diagnostic and Statistical Manual-Fourth Edition (DSM-IV) criteria (Hudson et. al., 2007). Prevalence rates for BED (defined using new proposed criteria for the DSM-5) and subthreshold BED have been collected in 496 U.S. adolescent girls (age 12-15 at baseline) in an 8-year longitudinal study using the Eating Disorder Diagnostic Interview (Stice, Marti, & Rohde, 2013). Subthreshold BED was defined as having at least 2 binge eating episodes per month on average over at least a 3-month period or having at least 6 binge eating episodes over a shorter period of time. Lifetime prevalence was referred to as lifetime prevalence at age 20, as this was the average age of participants at the 8-year follow-up interview, which included participants who met criteria at baseline or exhibited onset of BED or subthreshold BED over the 8-year period. Lifetime prevalence at age 20 was found to be 3.0% ( $N = 15$ ) for BED and 3.6% ( $N = 18$ ) for subthreshold BED. In contrast, among 2,825 Finnish twin women (surveyed from age 16 through mid-thirties), a lifetime prevalence rate of 0.7% was established using the Structured Clinical Interview for DSM-IV (SCID-I), coding for DSM-5 BED criteria based on audio recordings (Mustelin, Raevuori, Hoek, Kaprio, & Keski-Rahkonen, 2015). Further, lifetime prevalence rates for BED, subthreshold BED, and any binge eating were 1.12%, .72%, and 2.15%, respectively among the general adult population in six European countries (Belgium, France, Spain, Germany, Italy and the Netherlands; Preti et. al., 2009). Notably, these rates excluded individuals younger than age 18, and are therefore likely an underestimation. Thus, it is evident that prevalence rates for BED differ significantly based on demographic variables for the surveyed sample (e.g., race, gender, age, etc.).

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In order to assess the prevalence of BED in a broader, more generalizable population, the World Health Organization (WHO) conducted community-based mental health surveys among 14 countries (Total  $N = 24,124$ ) for lifetime and 12-month prevalence rates using the CIDI (Kessler et. al., 2013). This study found that country-specific lifetime prevalence rates were consistently higher for BED than for Bulimia Nervosa (BN), with a median of 1.4% and a mean of 1.9% across surveys. The 12-month prevalence rates averaged .8% for BED. One limitation for this survey is that it used proposed research BED criteria from the DSM-IV, as the DSM-5 was not yet published. However, they used presence of binge eating episodes for at least three months (as opposed to six months as the DSM-IV required) to meet criteria for the disorder; this is the threshold proposed at that time for the DSM-5 and was later included in the published version. Furthermore, both versions of the DSM require loss of control over eating and marked distress as a result of binge eating behaviors. These were not directly assessed using the CIDI; rather, interview questions asked about attitudes and behaviors that are indicative of these features. These included feeling upset at out-of-control feelings, eating until uncomfortably full, and feeling guilty, upset or depressed after bingeing. Thus, newer large-scale surveys must be conducted using strict DSM-5 criteria in order to assess the true prevalence of the current BED construct in a generalizable sample. Considering the difficulty assessing for BED diagnoses using self-report questionnaires and the similar distress of subthreshold binge eaters (Colles et. al., 2008), it is important in future research to examine the full range of binge eating behavior. This would allow additional research not constrained by thoroughly assessing DSM-5 diagnoses to grow, while addressing binge-related distress for all affected by it.

**1.2.2.2. Incidence.** The incidence rate is defined as the number of new cases of a disorder in a population over a specified period of time (Smink et. al., 2012). In eating disorders this is



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commonly expressed in terms of per 100,000 people per year (person-years). To date, studies examining the incidence of Binge Eating Disorder are sparse due to the novel recognition of the disorder in the DSM-5 (APA, 2013). Despite this, some recent investigations of incidence rates for BED can shed light on the etiology and course of this distinct disorder.

In a study of Finnish twin women, the incidence rate of BED in women age 10-24 was 35 per 100,000 person-years as assessed by the SCID-I via telephone (Mustelin et. al., 2015). Among those that screened positive ( $N = 292$ ) for eating disorder symptoms via self-report questionnaire, the incidence rate was 165; the incidence was 18 among those that screened negative ( $N = 340$ ), suggesting that onset of BED may be missed for some individuals when measured only with self-report questionnaires. In 2013, Stice and colleagues collected incidence rates of BED (using proposed DSM-5 criteria) and subthreshold BED (see prevalence for criteria used) longitudinally for 496 adolescent (12-15 years of age at baseline) females using the Eating Disorder Diagnostic Interview over an 8-year period. Cumulative incidence rates over the 8-year follow-up were 2.7% ( $N = 13$ ) for BED and 3.5% ( $N = 17$ ) for subthreshold BED. Incidence per 100,000 person-years was 343 for BED and 447 for subthreshold BED. Given that both of these studies occurred in female populations only, more incidence studies are needed across a range of demographic variables, particularly gender.

Incidence rates are also available for binge eating behavior, defined as eating a very large amount of food in a short amount of time at least monthly and feeling out of control during the eating episode, that does not necessarily meet criteria for BED (Field et. al., 2008). This was assessed in a study of U.S. children and adolescents ( $N = 11,087$ ), aged 9-15 years, over a 7-year period using questions adapted from the Youth Risk Behavior Surveillance System questionnaire. Resulting cumulative incidence rates were for 4.3% for females and 2.1% for

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males. Stratified by age, older adolescent (age 14 and older) females evidenced incidence rates of 1010 per 100,000 person-years while older adolescent males had an incidence of 660. In contrast, for younger adolescent (younger than 14 years old) females the incidence was 500 per 100,000 person years, while the incidence for younger adolescent males was 150. These data suggest that binge eating behavior is more common later in adolescence than in childhood or early adolescence and occurs more frequently among females than males.

**1.2.2.3. Gender.** It has been well-established that eating disorders occur more frequently in women than in men (APA, 2013). However, the gender ratio for BED is far less skewed than other eating disorders, with 12-month prevalence rates of 1.6% for females and .8% for males among U.S. adults according to the DSM-5; this is similar to the aforementioned prevalence studies. Despite this, much of the research conducted on binge eating has included samples of only women, with men excluded from the study. Thus, less is known about binge eating processes in male populations and this requires further investigation in future studies.

A study of female college students showed that 44% reported severe binge symptoms while 8.8% met full criteria for BED after removing those who engaged in compensatory behaviors, suggesting that binge eating is especially problematic in female college populations (Napolitano & Himes, 2011). Similarly, a study of community adults in six European countries found lifetime prevalence rates of .26% ( $N = 12$ ) for men and 1.92% for women ( $N = 64$ ; Preti et. al., 2009) using proposed DSM-IV criteria. Another study of adult health maintenance organization members found that more women than men met criteria for BED using proposed DSM-IV criteria, as well as subthreshold binge eating at a lesser frequency (at least once per week; Striegel-Moore et. al., 2009).

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Predictors of frequent binge eating also appear to vary somewhat by gender, as shown in a large 7-year longitudinal study of 11,087 U.S. children and adolescents (ages 9-15 at baseline; Field et. al., 2008). Onset of binge eating (at least monthly) was assessed with 2 questions adapted from the Youth Risk Behavior Surveillance System questionnaire, which asked about the frequency during the past year of eating a very large amount of food, and whether they felt out of control during these episodes (i.e., like they could not stop eating even if they wanted to stop). Notably, 4.3% of females and 2.3% of males experienced onset of binge eating episodes based on these criteria. For females, binge eating frequently (at least weekly) was predicted significantly by frequent dieting (odds ratio = 2.2), trying to look like persons in the media (odds ratio = 2.2), and high level of concern about weight (odds ratio = 2.7). High level of concern about weight also predicted frequent binge eating for males (odds ratio = 3.0), in addition to negative comments about weight made by their fathers (odds ratio = 2.3). Interestingly, maternal history of eating disorders was not predictive of frequent binge eating for males or females.

Binge eating symptoms and correlates have also been shown to differ among men and women, but not to the extent that one might expect based on the focus in the literature on women alone. Health maintenance organization members (ages 18-35) were assessed via self-report questionnaires regarding health and eating behaviors in order to determine gender differences in symptoms and correlates of various eating disorders (Striegel-Moore et. al., 2009). Interestingly, although women reported significantly more severe loss of control over eating, men were more likely to report overeating than women. While these gender differences were significant, it is worth noting that effect sizes were small. Specifically, for every 50 women studied, one more case that meets criteria for BED would be expected than for every 50 men studied. Thus, emphasis in the literature on statistical significance without attending to effect sizes may lead to

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an underestimation of the extent and clinical significance of disordered eating in men. In contrast, a moderate effect was observed for gender differences in checking body shape and weight, with 20% of women and 10% of men checking their shape and weight at least "very often" over the 3 months prior. A weak effect was observed for conscious avoidance of checking body shape and weight, with women being significantly more likely to report this behavior than men. These behaviors converge to evidence overvaluation of body weight and shape, which has been indicated as a correlate of BED. In contrast, a study of 182 adult men and women with BED (based on proposed DSM-IV criteria) showed fewer differences in BED correlates by gender (Barry, Grilo, & Masheb, 2002). BED criteria were assessed using the SCID-I and a clinical interview conducted by trained and monitored Ph.D.-level clinicians. Men and women did not differ significantly on measures of binge eating, eating concerns, or shape and weight concerns; however, women reported significantly greater drive for thinness, cognitive restraint (non-significant after Bonferroni corrections were applied for multiple comparisons) and body image dissatisfaction than men. Further, men and women did not differ significantly on current depression or self-esteem, but men reported a greater frequency of past drug abuse problems, a behavior related to increased impulsivity.

Further, there appear to be differences in the demographic determinants of binge eating frequency among men and women (Reagan & Hersch, 2005). In an urban community sample of 933 adult participants (ages 18-97), binge eating frequency was assessed using a single question that asked participants how often they have "an eating binge in which [they] eat a lot of food within a few hours;" options ranged from "never" to "most days." Notably, the item did not specifically ask about loss of control. Results showed that for women, binge eating frequency was negatively associated with age and family income, while being positively associated with

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marriage, depression and time spent living in polluted areas. In contrast, binge eating frequency was negatively associated with age alone for men. A study of adult men and women ( $N = 182$ ) diagnosed with BED using proposed DSM-IV criteria found that men had significantly higher current Body Mass Index (BMI), highest adulthood BMI, and were more likely to be classified as obese (BMI greater than or equal to 27; Barry et. al., 2002). In contrast, there were no differences in age of first being overweight, age at first diet, age at onset of binge eating, or number of weight cycles (i.e. 20-lb weight loss-regain cycles) between men and women.

Taken together, these findings indicate that in addition to overall prevalence, gender differences exist in the experience of symptoms and correlates of BED, as well as demographic determinants. This further supports the need to investigate BED in male and female populations, which will be addressed in the present dissertation project.

**1.2.2.4. Body mass index.** Body Mass Index (BMI) is a measure of body fat that allows categorization of weight status based on the height and weight of an individual and is commonly expressed as  $\text{kg}/\text{m}^2$  (NIH, n.d.). BMI categories include underweight (less than 18.5), normal weight (18.5-24.9), overweight (25-29.9), and obesity (30 or more). The higher one's BMI, the higher the risk of obesity-related illnesses such as heart disease, high blood pressure, type 2 diabetes, gallstones, breathing problems, and certain cancers. There are some limitations to BMI; it can overestimate body fat for athletes or individuals with high muscle content and can underestimate body fat for elderly individuals or others who have lost muscle. Despite this, it is widely used as a measure of obesity and has frequently been used to examine the relationship between BED and weight status.

It is important to distinguish between obesity and BED, although the two have reliability been shown to be related. One study of female college students found that BMI was positively

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associated with binge eating severity, while being overweight or obese was associated with greater likelihood of BED (Napolitano & Himes, 2011). In a study of Finnish twin women, two-thirds of those who met criteria for BED were in the highest weight quartile at age 16 (Mustelin et. al., 2015). The mean BMI for these individuals from ages 22-27 was 26.2 kg/m<sup>2</sup>, which falls in the overweight range. In a nationally representative sample of U.S. community-based adults, 42.4% of those with BED were categorized as obese (BMI greater than or equal to 30; Hudson et. al., 2007). BED was shown to be associated with a significantly higher prevalence of BMI greater than or equal to 40, representing severe obesity, when compared to respondents with no eating disorder. Further, any presence of binge eating was shown to be associated with severe obesity; however, this was entirely attributable to cases of BED. Lifetime BED was also associated with higher BMI (obese) when compared to those without an eating disorder in a large-scale WHO mental health survey of 14 countries (Kessler et. al., 2013). Specifically, 36.2% of those with lifetime prevalence of BED and 41.7% of those with 12-month prevalence of BED were classified as obese (BMI greater than or equal to 30). Notably, those with lifetime and 12-month BED were also significantly more likely than non-eating disordered individuals to be severely obese (BMI greater than or equal to 40), with odds ratios of 6.6 and 10.2, respectively. The association between BMI and BED may also differ somewhat by gender; in a study of adult BED patients, men evidenced higher current BMI, highest adult BMI, and were more likely to be classified as obese (BMI greater than or equal to 27; Barry et. al., 2002).

In contrast, Stice and colleagues (2013) conducted an 8-year longitudinal study and found that among adolescent community-based females (ages 12-15 at baseline), those with BED did not differ significantly in BMI from those without disordered eating. This finding suggests that although higher BMI has been reliably linked with increased rates of BED, the two are not

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always closely associated. In fact, most obese individuals do not engage in recurrent binge eating, and binge eating does indeed occur in normal-weight samples (APA, 2013). Further, compared with weight-matched obese individuals without BED, those with the disorder have greater functional impairment, lower quality of life, more subjective distress, and greater psychiatric comorbidity. BED individuals appear to represent a distinct group; a limitation of the literature is that many previous studies have focused on obese BED participants only, excluding those with a healthy BMI. This will be addressed in this dissertation by including participants with a range of BMI status.

**1.2.2.5. Race, ethnicity, and national origin.** To date, research has been mixed regarding the occurrence of BED among racial groups. Binge eating frequency has been shown to be unrelated to race (among African American and White participants) based on self-report data from 573 women and 360 men in the community, holding constant gender and obesity status (Reagan & Hersch, 2005). The frequency of binge eating also did not differ by race in an ethnically diverse, college-based sample based on self-report data (Franko, Becker, Thomas, & Herzog, 2007). However, binge correlates (e.g., eating until uncomfortably full) were more frequent in White than African American participants. Further, binge eating was the greatest predictor of distress among White, African American, and Latino participants, suggesting that the impact of binge eating may differ by race even if frequency of episodes does not.

In contrast, Napolitano and Himes (2011) found that White participants were more likely to have BED than African American participants in a college sample of women, and they also evidenced more severe binge eating symptomatology. Further, White women had higher dietary cognitive restraint, body image dissatisfaction, drive for thinness, and depressed mood, which in concert with BMI accounted for significant variance in binge eating symptomatology for both

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racess. When examining these correlates across the whole sample, body image dissatisfaction and dietary cognitive restraint no longer contributed significantly to binge eating symptomatology, but race did. Individually, body image dissatisfaction contributed significantly to binge eating symptomatology for African American women but not for White women, while the opposite was true for lower dietary cognitive restraint. Thus, body image dissatisfaction may put African American women at risk for binge eating, perhaps through negative affect as opposed to dietary cognitive restraint. Consistent with the findings of Franko and colleagues (2007), it appears that binge correlates more reliably differ by race, suggesting that individualized treatments for binge eating should address these disparities to more effectively mitigate binge eating severity.

In fact, treatment response for individuals with BED has been shown to differ by race in a meta-analytic study including 11 randomized, controlled trials of psychosocial treatments (Thompson-Brenner et. al., 2013). Specifically, African American participants were more likely to drop out of treatment than White participants (odds ratio = 2.28); however, African Americans showed lower Eating Disorder Examination (EDE) scores posttreatment, evidencing better treatment outcome on cognitive measures than White participants. The EDE is a semi-structured clinical interview of eating disorder symptoms. In contrast, Hispanic/Latino participants did not differ significantly on treatment outcome or retention measures compared to White individuals.

Although there are inconsistent findings in the literature of the impact of race/ethnicity on frequency of binge eating and BED, it appears that binge correlates and treatment response more consistently differ, at least for African Americans compared with White individuals. Further research is needed to assess the impact of these disparities, as well as additional investigations comparing a more diverse set of racial/ethnic groups on these factors.



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**1.2.2.6. Socioeconomic status.** Socioeconomic status (SES) is defined as the social standing or class of an individual or group, and is often represented as a combination of education, occupation, and income (APA, n.d.). Examinations of SES often reveal inequities in access to resources, which can significantly impact physical and psychological well-being. Thus, it is not surprising that SES factors have been indicated in binge eating symptomatology.

In an adult community-based sample, Reagan & Hersch (2005) found that binge eating frequency (sample and assessment as described above) was significantly negatively associated with family income for women alone, with an adjusted odds ratio of .958 for a 10% increase in family income. Notably, the sample size for men in the study was only two-thirds that of women, and it is possible that the difference in statistical significance is attributable to the smaller sample size for men. Binge eating frequency was also positively associated with time spent residing in polluted areas, such that increased time spent in an unhealthy area increased the frequency of binge eating; this living situation is more common for lower SES individuals. This was assessed by asking participants if they had ever lived in an area “where residents have problems such as skin or eye irritation, breathing difficulties, or even cancers because of exposure to things like pollution, hazardous chemicals or waste incinerators” and if so, for how long. The number of years spent in such a neighborhood was then divided by the participants age in order to represent a fraction of their life spent in polluted/disadvantaged residences. An adjusted odds ratio of 1.11 was observed if a woman lived 10% more of her life in an unhealthy area, suggesting that these women were more likely to experience higher binge eating frequency.

Further, socioeconomic status may have a detrimental impact on treatment response in individuals with BED (Thompson-Brenner et. al., 2013). A meta-analysis of 11 randomized, controlled trials of psychosocial treatments for 1,073 individuals with BED across the U.S. was

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conducted to determine the effects of education on treatment response. The EDE was used across treatment trials to assess objective bulimic episodes, defined as eating an unusually large amount of food while experiencing a loss of control over one's eating. Psychosocial treatments included cognitive behavioral therapy (CBT), interpersonal therapy, couples therapy, dialectical behavior therapy, mindfulness therapy and others; all included an element of identifying antecedents and psychological triggers for binge eating, and strategies to avoid these. Results showed that lower levels of education predicted greater post-treatment objective bulimic episodes. Specifically, having more than a high school education was associated with significantly fewer objective bulimic episodes after treatment, and a significantly higher likelihood of remission (odds ratio = 1.79) after treatment, defined as no objective bulimic episodes in the past 28 days.

**1.2.2.7. Age.** Binge eating symptomatology appears to differ significantly as a function of age, with younger individuals (adolescence through middle adulthood) consistently showing higher rates of binge eating than older adults. Specifically, binge eating frequency was highest in an adult community sample among individuals younger than 40 years old (Reagan & Hersch, 2005). Further, a 7-year longitudinal study of children and adolescents (ages 9-15 years at baseline) showed that rates of binge eating onset differ amongst younger populations as well (Field et. al., 2008). Interestingly, both male and female participants younger than 14 years of age were significantly less likely to experience an onset of binge eating at least weekly than those age 14 and older. Incidence rates for older adolescents were 1010 per 100,000 person-years for females and 660 for males, while rates for younger adolescents were 500 per 100,000 person-years for females and 150 for males.

Age also appears to significantly impact treatment response, as shown in a meta-analysis of BED individuals receiving a range of psychosocial treatments (Thompson-Brenner et. al.,

2013). This review of 11 randomized, controlled trials found that age was a significant predictor of objective bulimic episodes posttreatment, objective bulimic episode cessation, and treatment dropout. Older age (greater than or equal to 46 years old) predicted greater reduction in objective bulimic episodes at posttreatment, greater rates of cessation (odd ratio = 1.26), and lower dropout rates (odds ratio = 1.35) compared with younger individuals (less than 46 years old). These results suggest that younger individuals evidence poorer response to treatment, and in fact, poorer engagement in treatment with higher likelihood of dropout once beginning therapy.

These findings have important implications for the identification and treatment of BED among individuals of various age groups. It appears that individuals in late adolescence through middle adulthood experience greater binge eating symptomatology and poorer response to treatment than older adults. Thus, it is especially vital to understand binge eating in these younger populations, in order to design more effective treatments with more positive outcomes.

**1.2.2.8. Course.** Although little is known currently about the development of BED, binge eating and loss of control over eating (LOC) can be observed in children and are associated with increased weight gain, body fat, and psychological symptoms in this population (APA, 2013). BED typically begins in adolescence or young adulthood but can also begin in later adulthood with less frequency. In fact, Reagan & Hersch (2005) found that binge eating frequency was negatively associated with age for men and women in a sample of community-based adults, suggesting that binge eating is mitigated as individuals age. Interestingly, the decline in frequency began at an earlier age for men than women. In a study of Finnish twin women, the mean age of onset of BED was 19 years old (Mustelin et. al., 2015). Similarly, a longitudinal study of U.S. adolescent girls found that peak age of onset for BED and subthreshold BED was between ages 18-20 (Stice et. al., 2013). A WHO mental health survey conducted across 14

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countries found a mean age of onset for BED of 23.3, which was consistently later than that of BN (20.6; Kessler et. al., 2013). The age of onset curve evidenced a steeper slope for BN than BED, suggesting that onset decreases more rapidly as age increases for BN than for BED. All of these studies converge to suggest that emerging adults and college-age individuals are particularly vulnerable for onset of binge eating.

Persistence of disordered eating was also significantly lower in BED than BN in a large international survey, with median years spent in episode of 4.3 for BED and 6.5 for BN; however, the interquartile range for prevalence, an indirect measure of persistence, was lower for BN (Kessler et. al., 2013). It is possible that this was partially due to the rapid recovery of BN in France and Portugal where BED recovery was much slower; recovery speeds in Italy and the Netherlands were comparatively slow for BED and BN. This disparity suggests that recovery from BN differs greatly based on ethnicity/race, while BED recovery may be more consistently slow. This has important implications for the negative impacts of binge eating behavior in the absence of compensatory behaviors, and for developing effective treatments to aid recovery. In contrast, Stice and colleagues (2013) found that average episode duration was 3.3 months for BED and 3.0 months for subthreshold BED among adolescent girls followed over a period of 8 years. It appears that persistence of BED symptoms varies greatly depending on the population assessed, and more studies are needed in a number of settings to more accurately understand the course of binge eating remission.

Further, Stice and colleagues (2013) found that of the 18 individuals with subthreshold BED, five (28%) progressed to BED later while 33% of those with BED evidenced crossover to subthreshold BED over time; these findings suggest that individuals with binge eating behavior may move in and out of diagnosable levels over the course of BED. In fact, of the 15 participants

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with BED, 14 experienced remission within 1 year but five of them experienced two or more episodes over the follow-up period, producing an overall recurrence rate of 33%. Of the 18 participants with subthreshold BED, all showed remission within 1 year but six experienced two or more episodes, again producing an overall recurrence rate of 33%.

Taken together, these findings suggest that remission and relapse rates of BED vary greatly depending on the populations assessed, but overall it appears that the course of BED is dynamic and individuals who binge eat likely experience a continuum of distress and frequency over the course of their life. This provides more evidence for the need to include individuals with a range of symptom severity in future investigations of BED, and not be confined by diagnostic criteria alone. Further, although BED is present in children and adolescents, as well as older adults, it is most commonly beginning in emerging adulthood. Thus, college-age populations are especially vulnerable to onset of binge eating behavior, and this vulnerability may converge with a range of college-related life stressors to cause more severe disordered eating and a range of associated psychological and physical health problems.

### **1.2.3. Comorbidity**

**1.2.3.1. Psychiatric comorbidity.** Comorbidity is defined as the presence of one or more additional disorders co-occurring with a primary disorder. There is a plethora of data to suggest that BED occurs comorbidly with a number of other mental health disorders. The vast majority of respondents (79%) with BED in a large WHO mental health survey sample collected from 14 countries met criteria for another DSM-IV/CIDI disorder (Kessler et. al., 2013). Odds ratios for BED were positive and significant for each of the other disorders, with a median of 2.8. Retrospective age of onset reports were used to examine odds ratios for BED predicting other mental health disorders as well as odds ratios for other mental health disorders predicting onset

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of BED. Interestingly, these were mostly elevated and significant in both directions, but were more so for other disorders predicting onset of BED than the reverse, with median odds ratios of 3.4 and 2.3, respectively. In a large, nationally representative sample of U.S. adults, 78.9% of those with BED met criteria for at least one lifetime additional DSM-IV disorder (Hudson et. al., 2007). The median odds ratio for BED predicting the likelihood of other lifetime DSM-IV disorders was 3.2, with a median odds ratio of 2.2 for subthreshold BED. All disorders evidenced statistically significant odds ratios for BED, with the highest being observed for lifetime agoraphobia without panic (5.2); this was also the highest for subthreshold BED (5.8). Shockingly, the likelihood of having three or more additional lifetime psychiatric disorders was 7.6 (odds ratio) for those with BED compared to those without.

Similarly, 73.8% of treatment-seeking patients with BED ( $N = 404$ ; based on DSM-IV research criteria) were shown to have at least one additional lifetime DSM-IV disorder and 42.8% had at least one current DSM-IV disorder in a study of respondents to media advertisements for treatment studies at an urban medical school (Grilo, White, & Masheb, 2009). Participants were administered the SCID-I to assess for BED and other mental health disorders, as well as the Eating Disorder Examination (EDE) to confirm BED diagnoses and assess for eating disorder symptoms. Among psychiatric comorbidities, lifetime mood disorders were most common (54.2%), with Major Depressive Disorder (MDD) being the most common specific disorders (46.8%). Lifetime anxiety disorders were also common (37.1%), with panic disorder being the most prevalent (15.3%). Lifetime substance use disorders were also common (24.8%); rates for alcohol use disorders were 20.3% and drug use disorders were 14.6%. Interestingly, male BED participants were significantly more likely to have a history of substance use disorders. Regarding current psychiatric comorbidities, 26% of BED patients were found to have

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a mood disorder, with MDD being the most common (18%). 24.5% had current anxiety disorders; generalized anxiety disorder was the most prevalent specific anxiety disorder, being present in 8.9% of patients with BED. Notably, current substance use disorders were uncommon (2.7%). One gender difference existed among current comorbidities, with males (5.3%) being more likely to meet criteria for obsessive compulsive disorder than females (1.3%). Further, presence of overall comorbidity in this sample did not differ significantly based on gender, ethnicity, age, current BMI or education; however, highest lifetime BMI was significantly higher in participants with current comorbidity than participants without. Participants with lifetime or current comorbidity had earlier onset of first diets than those without; age of onset of obesity, binge eating and BED did not differ. Patients with current comorbidity had higher binge eating frequency than those without, as well as higher eating concerns, shape concerns, weight concerns and global EDE scores than both those with lifetime comorbidity and no comorbidity.

Symptoms of depression appear to be especially highly comorbid with BED; in a study of Finnish twin women, six had lifetime major depressive disorder of the 16 who met criteria for BED (Mustelin et. al., 2015). Suicidality, as assessed by the depression module of the Schedule for Affective Disorders and Schizophrenia for School-age Children (K-SADS), was also shown to be higher in BED and subthreshold BED participants than non-eating disordered participants in an 8-year longitudinal study of adolescent females (Stice et. al., 2013). Emotional distress was also measured using the depression module of the K-SADS, with an item asking whether respondents had a period of time in the past 12 months where they felt sad, bad, unhappy, empty, or like crying for most of the day nearly every day. Results showed that emotional distress was greater in those with BED and subthreshold BED compared to non-eating disordered respondents. These findings suggest that regardless of whether binge-eaters meet full criteria for

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the disorder, binge eating is associated with increased depressive symptoms such as suicidality and emotional distress. Notably, the operationalization of emotional distress in this study is analogous to the depressed mood criteria for Major Depressive Disorder (MDD). Additionally, among treatment-seeking BED patients, those with current comorbidity of at least one additional DSM-IV disorder had higher levels of depression, assessed using the Beck Depression Inventory (BDI), and lower self-esteem, assessed using the Rosenberg Self-Esteem Scale, than those with lifetime comorbidity and no comorbidity (Grilo et. al., 2009).

Dieting often follows the development of BED but crossover to other eating disorders is uncommon according to the DSM-5 (APA, 2013). This is consistent with findings by Grilo and colleagues (2009), which showed that lifetime prevalence of other eating disorders was uncommon (6.4%) for treatment-seeking patients with BED. Specifically, only 5.9% met criteria for past bulimia nervosa and only 1.5% met criteria for past Anorexia Nervosa (AN). In contrast, Stice and colleagues (2013) found that 23% of the 22 participants with subthreshold BN later developed BED in a sample of community-based adolescent females followed over 8 years. Further, of the 18 participants with subthreshold BED, 22% later developed BN and 22% later developed subthreshold BN. It appears that although diagnostic crossover between eating disorders is uncommon, this is less rare when considering both threshold and subthreshold levels of BN and BED. This is not surprising given the overlap in binge eating behavior exhibited between these disorders. In fact, these findings suggest that perhaps individuals who binge eat in general experience periods where they are more likely to cope with their binge eating by attempting to compensate physically.

Overall, BED is associated with increased rates of comorbid psychiatric disorders, with anxiety, mood and substance use disorders being most common. Crossover among different



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eating disorders is uncommon; however, BN and BED appear to be more closely related than AN given the presence of binge eating behavior for both. Understanding comorbid psychiatric disorders for BED and subthreshold BED has important implications for treatment, as effective interventions must address and manage an individual's co-occurring psychological problem areas in order to create lasting and meaningful change in eating behavior. Further, depressive symptoms appear to be especially prevalent among those who binge eat; increased rates of suicidality suggest that risk assessment and safety planning could be a crucial part of treating BED.

**1.2.3.2. Medical comorbidity.** Given the aforementioned association between binge eating and obesity, it is not surprising that BED is associated with a number of obesity-related illnesses. Data from a WHO survey in 14 countries showed that BED was associated with increased risk of chronic back/neck pain (odds ratio = 1.5), diabetes (odds ratio = 2.4) and hypertension (odds ratio = 1.8), even after controlling for other mental health disorders (Kessler et. al., 2013). Interestingly, BED was not associated with increased risk of stroke or heart attack, while BN was (but only before controlling for other mental health disorders). In a sample of 2,225 adult bariatric surgery candidates from 6 clinical centers around the U.S., BED was associated with increased likelihood of impaired glucose levels (odds ratio = 1.45), high triglycerides (odds ratio = 1.28), and urinary incontinence (odds ratio = 1.30) compared with non-BED patients after controlling for BMI, age, gender, and education (Mitchell et. al., 2015). In contrast, severe walking limitations were significantly less common in the BED sample (odds ratio = .53). BED diagnoses were made using questions adapted from the Questionnaire for Eating and Weight Patterns-Revised; however, binge eating episodes for at least 6 months (rather than 3) were required, which is inconsistent with DSM-5 criteria. Importantly, these results were

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significantly altered when analyses were conducted while controlling for a number of psychological factors known to be related to BED. These included psychiatric medication use, depressive symptoms, self-esteem, and alcohol use symptoms. After controlling for these variables, impaired glucose levels were more common among those with BED (odds ratio = 1.36), while cardiovascular disease (odds ratio = .50) and severe walking limitations (odds ratio = .38) were less common among those with BED. Interestingly, it does not appear that BED is associated with increased risk of heart disease (Mitchell et. al., 2015; Kessler et. al., 2013), despite being comorbid with a number of other obesity-related health problems.

Further, a variety of other problematic medical conditions are prevalent among those with BED. Data from 14 countries evidenced increased risk of chronic headaches (odds ratio = 1.8) and other chronic pain conditions (odd ratio = 1.8) for those with BED, even after controlling for other mental health disorders (Kessler et. al., 2013). Notably, BED predicted increased risk of ulcers (odds ratio = 1.9) and arthritis (odds ratio = 1.7), but only before controlling for other mental health disorders; this suggests that psychological comorbidity of BED plays a role in associated medical problems.

Overall, it appears that BED is highly comorbid with medical illness; 81% of Finnish twin women with BED were currently ill when interviewed (condition not specified; Mustelin et. al., 2015). This has implications for the importance of developing effective treatments for BED, as those with the disorder appear to be particularly at-risk for medical problems; treatment of BED is vital not just to increase psychological and emotional wellbeing, but physical wellbeing as well.

### **1.3. Stress and Binge Eating**

#### **1.3.1. Overview of Stress**

Stress, a pervasive experience in today's society, is linked to a number of negative health outcomes, including heart disease, cancer, and cerebrovascular disorders (Sapolsky, 2004). A stressor can be any stimuli that disturbs the body's homeostatic balance, or the state in which physiological measures (i.e., oxygen, acidity, temperature etc.) are kept at optimal levels, either real or imagined. Thus, stress can involve the anticipation of a stressor; the stress-response can be mobilized in response to physical or psychological disturbances, as well as the expectation of one. Lazarus and Folkman (1984) discussed the distinction of psychological stress from physiological stress as dependent on personal meaning. They posited that psychological stress is bidirectional; it is composed of the relationship between the person and the stressor they appraise as taxing or exceeding resources and endangering their well-being. Thus, individual differences, such as how one appraises a stressor, can have a significant impact on the stress response. Appraisal is defined here as a universal process in which people constantly evaluate the significance of what is happening for their personal well-being (Lazarus, 1993). A harm appraisal takes place when psychological damage has already taken place, while threat is the anticipation of harm. In contrast, challenge involves difficult demands that one feels confident in their ability to overcome. These appraisals vary based on the environment and internal stimuli and, as such, stress cannot be considered in terms of simple activation. Indeed, psychological stress may be chronic and persistent (e.g., permanent disabilities or job stress), which is described as being exposed to a stressor for a prolonged time and appraising it as a threat to a fluctuating degree over time (Lazarus & Folkman, 1984). In contrast, chronic intermittent stress (e.g., financial problems or weather) provides time without the exposure to the stressor while

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acute stress (e.g., parachute jump or encountering a rattlesnake) refers to an acute event/external stimulus that results in stress reactivity and a perception of immediate threat.

Appraisals of stress can affect adaptational outcomes and coping abilities, including somatic health, morale, and social functioning (Lazarus & Folkman, 1984). Competence in social interactions is impacted by inappropriate appraisals, such as when a threat is perceived when there is none, or vice versa. This mismatch between appraisal and the actual flow of events can lead to social behavior that is not appropriate for the setting, and the tendency for this mismatch can lead to long-term social functioning problems. Morale, or the way people feel about themselves and their conditions of life, is harmed when appraisals involve the judgment that desired goals were not successfully achieved (i.e., harm). Somatic health is known to be impacted by strong and negative emotions, such as fear, so threat is associated with short and long-term illness.

This highlights the importance of assessing an individual's perception of stress. Perceived stress is defined as the degree to which one appraises their life situations as stressful (Cohen, Kamarck, & Mermelstein, 1983). This has reliably been measured using a 10-item scale that asks about an individual's perception of having the resources to cope with their stressful life situations over the past month (Cohen, Kamarck, & Mermelstein, 1994). Examining both perceived stress and physiological reactivity to a stressor can provide a full picture of an individual's experience of stress; it is important to consider both when examining the effects of stress on eating in this project (Study 1 examines perceived stress; Study 2 examines physiological reactivity).

**1.3.1.1. Impact of stress on eating behavior.** Stress has long been implicated in overeating in both animal and human studies; both tend to eat more in response to stress, as long as the stressor is not severe (Torres & Nowson, 2007-11). This suggests that more moderate,

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daily stressors may put an individual at particular risk for binge eating. In fact, daily diaries (kept for 30 days) completed by 46 binge-eating college women revealed that higher stress was associated with increased risk of same-day binge eating, regardless of depressed mood (Freeman & Gil, 2004). Stress was identified by having the participant indicate the most stressful event or moment of the day, and then this was rated using a visual analog scale (VAS) from 0-100. Binge eating episodes were self-identified, with the largest identified along with foods eaten.

Interestingly, psychological stress did not predict next-day binge eating but did predict binge-eating that same day. Similarly, another study using daily diaries to examine precursors of binge in 66 college women found that increased stress was marginally (approached significance) associated with increased likelihood of binge eating symptoms (measured by questions adapted from the Eating Disorders Inventory) the same day (Barker, Williams, & Galambos, 2006). In this case, stress was measured using the Daily Inventory of Stressful Events, which asks participants about specific stressful events that may have occurred in the past 24 hours. Elevated weight concerns predicted same and next-day binge eating, suggesting that body-focused stressors are particularly powerful. Crowther, Sanftner, Bonifazi, and Shepherd (2001) used a self-report measure called The Hassles Scale, completed by 34 normal-weight women each night for 2 weeks, to investigate the role of daily hassles. Daily hassles are defined as repetitive, chronic strains of everyday life. Participants were identified as binge eating or non-binge eating based on their responses to the Binge Eating Scale (BES) and the Eating/Dieting Questionnaire; those who binge eat showed significantly higher ratings of stress intensity on average from daily hassles than those who do not. The BES is a commonly used self-report measure of binge eating severity. Notably, the number of hassles did not differ between these groups, suggesting that binge eating women experience more stress in response to similar amounts of daily hassle.

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Further, the binge eating group ate significantly more calories on days with many hassles compared to days with few; however, number of binge episodes did not differ on these days. These findings suggest that even when frequency of stressors and binge eating episodes does not differ, intensity of eating and perception of stress does. Overall, daily, chronic stressors reliably predict binge eating behavior, but the perception of these experiences as stressful, and by how much, may be important to investigate further.

In addition to chronic stress, perceived stress has also been shown to be related to increased drive to eat, lack of control over eating, and binge eating frequency in a community sample of women with BMI's ranging from normal to obese ( $N = 561$ ; Groesz et al., 2012). This cross-sectional study investigated the role of stress exposure on eating via online self-report questionnaires measuring perceived stress (Perceived Stress Scale [PSS]), exposure to chronic stressors related to work, family and relationships, binge eating behavior (measured using the BES), dietary restraint, disinhibition and hunger. Participants were also presented with a list of various food items and asked to indicate how often they eat each item. This was used to create two reliable subscales; nutritious foods and palatable/non-nutritious foods. Partial correlations (controlling for age, BMI, income and education) among these variables revealed that increased perceived stress was related to higher palatable/non-nutritious food intake, lower nutritious food intake, increased lack of control over eating, greater hunger, and more frequent binge eating. Controlling for flexible restraint, greater levels of rigid restraint ("I avoid some foods on principle even though I like them") were associated with higher perceived stress. Conversely, greater flexible restraint was associated with lower perceived stress after controlling for rigid restraint. This finding was equivalent for exposure to stress. Increased exposure to stress was also related to higher palatable/non-nutritious food intake, lack of control over eating, hunger,

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and binge eating. Interestingly, neither stress exposure nor perceived stress differed based on participant's BMI. Additionally, Torres and Nowson (2007-11) reviewed a number of studies that showed perceived control impacting general food intake, as well as intake of fatty and intensely sweet foods. Thus, perceived control, which can impact the appraisal of one's ability to cope with stress, appears to play an important role in overeating, especially foods that are palatable as well as energy and nutrient-dense. These foods are associated with binge eating episodes and increased weight gain, suggesting that this kind of stress may play a key role in obesity for binge eating individuals.

Interestingly, stress appears to impact binge eating behavior differently depending on the coping styles engaged. Freeman and Gil (2004) showed that distraction coping was associated with increased future (the next day) binge eating in a sample of 46 college women completing daily diaries, while social support was negatively associated with binge eating. Notably, among those high in restraint, distraction coping had no impact on the relationship between stress and same-day binge eating. Acceptance coping, another form of more passive coping, was associated with increased binge eating on the same day as stress increases, for all levels of dietary restraint, while acceptance did not directly impact binge eating. Similarly, a study of 174 college females found that binge eating (measured using the BES) was positively associated with avoidant and emotional coping styles, while being unrelated to rational or detached styles (Sulkowski, Dempsey, J., & Dempsey, A.G., 2011). Stress was measured using a checklist of common life stressors, which does not account for how they perceived the stressor and assumes that all individuals would experience stress in response. Avoidant coping is described as pretending that nothing is wrong and daydreaming; this is typically associated with mood regulation problems and suicidality. Emotional coping, such as rumination and blaming oneself, is generally less

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effective than rational coping, or planning to solve a problem and thinking of alternative ways to view it. Lastly, detached coping is characterized by viewing a problem as separate from oneself and not taking things personally. These were assessed using the Coping Styles Questionnaire, which assesses the frequency that participants engage in specific behaviors in response to stress. The relationship between increased binge eating behavior and maladaptive coping strategies (i.e., emotional and avoidant) suggests that stress may lead to binge eating through poor emotion regulation. Further, emotional coping partially mediated the relationship between stress and binge eating, while avoidant coping was not significantly related to binge eating after controlling for emotional coping. Thus, engaging in emotion-focused coping strategies may put someone at particular risk for binge eating; negative affect in the context of stress and eating needs to be further investigated. Dietary restraint is also implicated in the usefulness of coping strategies, suggesting that this is something to consider in future studies as well.

**1.3.1.2. Physiological correlates of stress and binge eating.** Studies have also shown differences in physiological responses to stress for those with BED. Specifically, sustained Hypothalamic-Pituitary-Adrenocortical (HPA) axis activation after a stressor has been shown in individuals with BED, as well as increased cortisol (an index of HPA activation) levels in response to a stressor when compared with healthy controls (Lo Sauro, Ravaldi, Cabras, Faravelli, & Ricca, 2008). HPA axis activation has been hypothesized to impact reward systems in the brain through its release of cortisol (Adam & Epel, 2007). Repeated and sustained release of cortisol is thought to impact reward sensitivity through the intake of highly palatable foods and lead to neurobiological adaptations that lead to the compulsive nature of overeating and increased drive to overeat. Further, endogenous opioids are released in response to intake of highly palatable foods, and sustained levels of opioids are associated with increased intake of



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highly palatable foods, suggesting a somewhat cyclical nature of opioid release. Opioids are also stimulated by activation of the HPA axis, and act as a negative feedback mechanism for the HPA axis, and therefore decrease the stress response. Thus, opioids released in response to the intake of palatable foods may also lead to HPA axis reduction and may play a role in highly palatable food acquiring an “addictive” quality.

These findings suggest that BED individuals not only experience sustained sympathetic activation in response to stressors, but also evidence depressed parasympathetic cardiac control during mental stress tasks. These patterns of autonomic activity are associated with increased cardiac risk and even morbidity, suggesting the importance of looking at these factors and how they are impacted by and related to binge eating in response to stress.

**1.3.1.3. Purpose of proposed dissertation.** The role of stress in eating, specifically binge eating, has been well established. As such, this project proposes that stress directly impacts binge eating, with increased stress being associated with increased binge eating (See Figure 1). However, the exact mechanisms of this process are still unknown. I propose a comprehensive model (See Figure 5) of stress-induced binge eating via a number of variables shown to be related to both stress and eating including negative affect, cognitive restraint, and impulsivity.

Figure 1. Stress Directly Impacts Binge Eating



### 1.3.2. Negative Affect

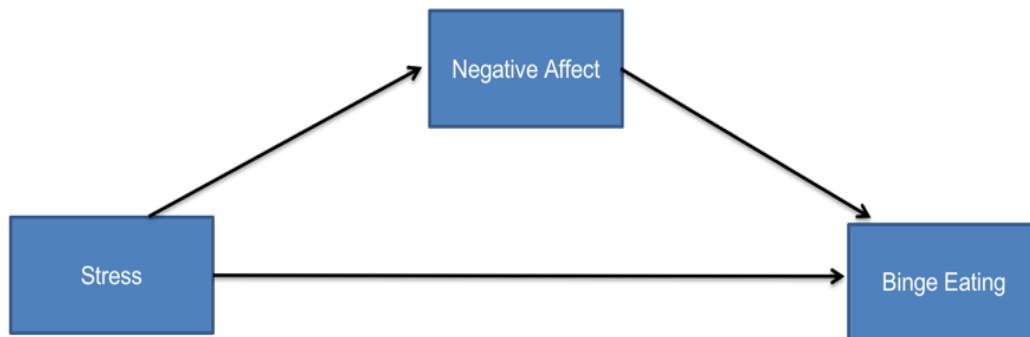
It has been suggested that obese individuals with BED represent a distinct neurobiological phenotype within the obesity spectrum that is characterized by an emotion regulation deficit (Leehr et al., 2015). There has been much debate regarding the definitions of emotion, affect, and mood, as well as the distinctions between them (Lane & Terry, 2000; Parkinson, Totterdell, Briner, & Reynolds, 1996). It has been suggested that affect is a broader construct that encompasses the others and involves evaluative feelings. Negative affect is a heterogeneous construct with many facets (i.e., anger, shame, anxiety), but it is often represented in the literature as a global construct with all facets combined. Emotion is thought to be relatively brief but intense, based on cognitive appraisals of the situation. Mood is thought to relate to the individual more than the situation, and in contrast to emotions is more prolonged but less intense. While there are contrasting definitions in the literature, all seem to hinge on evaluating duration and intensity. For the purposes of this research, valence is of interest, as negative valence may be particularly relevant for BED. Thus, distinctions will not be made regarding intensity or duration, and so the terms “emotion,” “mood,” and “affect” will be used interchangeably--this has been done often in the literature.

There are a variety of different theories related to emotion regulation; escape theory posits the alleviation of negative affect during binge eating, while emotional eating theory posits that eating is a coping mechanism used in the face of negative emotions (Leehr et al., 2015). Affect regulation theory assumes an improvement in negative affect after binge eating and emotional arousal theory assumes a decrease in emotional arousal. These theories have small distinctions but can be combined to represent a comprehensive emotion regulation model

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characterized by two key pieces. First, negative affect is implicated as a trigger for binge eating, and second, negative affect is alleviated and mood is improved as a result of binge eating.

Figure 2. Stress Impacts Binge Eating Through Increased Negative Affect



**1.3.2.1. Negative affect as a trigger for binge eating.** In a recent literature review, nine out of 15 studies included showed evidence for negative affect as a precursor for binge eating in BED samples, with five showing higher food intake and loss of control after a negative mood induction (Leehr et. al., 2015). While their inclusion criteria were rigorous (only experimental studies with group comparisons were included) and resulted in small sample size, this process has been well-supported by a number of other studies. Negative affect is the most common retrospectively reported trigger of binge eating among college-age women ( $N = 715$ ; Napolitano & Himes, 2011). The Functional Assessment of Binge Eating (FOBES) measure was completed in order to ascertain antecedents to binge eating via online survey, as was the Three Factor Eating Questionnaire (TFEQ) and the Binge Eating Scale (BES). Those meeting criteria (based on Eating Disorder Diagnostic Scale scores) for BED were more likely to endorse 14 out of 15 items on the FOBES, all of which were facets of negative affect (e.g., self-anger, worry, guilt), than those who endorsed some binge eating but did not meet criteria for the disorder. Hunger was the only antecedent not significantly more common in BED participants. Notably, among all

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participants, African Americans reported lower levels of anxiety prior to binge eating compared to White participants. Another study of college men and women ( $N = 780$ ) used online survey data to examine the interaction of rumination and body dissatisfaction as a precursor for binge eating and found that those with high body dissatisfaction were more likely to binge eat when they also were high in rumination, even after controlling for depressive symptoms (Gordon, Holm-Denoma, Troop-Gordon, & Sand, 2012). Interestingly, this model did not predict problematic drinking, suggesting that body-focused negative affect is specific as a precursor to binge eating. Similarly, daily diary analyses in a sample of 66 freshman females revealed that negative affect (measured using the Positive and Negative Affect Schedule [PANAS]) and weight concerns were associated with increased likelihood of reporting binge eating symptoms the same day, while weight concerns predicted next day binge eating as well (Barker et. al., 2006).

Further, a meta-analysis of 36 studies of women (89% White) by Haedt-Matt and Keel (2011) examined affect before and after binge eating using ecological momentary assessment (EMA) methods, which examine an individual's behavior and experiences in their natural setting. As expected, negative affect before binge eating was higher than average ratings of negative affect across the day, and the magnitude of this was greater for BED participants than those with BN. Negative affect was also higher before binge eating compared with affect experienced before regular eating, suggesting that binge eating is uniquely related to negative affect. Also, those with BED experienced more negative mood prior to binge eating than those with BN, suggesting that binge eating in the absence of compensatory behavior appears to be particularly impacted by negative affect as a precursor. In contrast, ratings of cues for binge eating did not differ between BED and BN patients from 2 CBT treatment studies (data collected at or close to

baseline), but type of negative affect experienced most frequently did (Mitchell et. al., 1999). In particular, BN patients most frequently endorsed ‘anxious to binge eat’ and ‘anxious about something else,’ while the most frequent precipitants for BED patients were ‘anxious about something else’, ‘depressed mood’ and ‘feeling bored.’ Experiences before, during and after binge eating were assessed using a measure developed for the study, the Eating Hedonics Questionnaire. Interestingly, those with BED were also more likely to report that they enjoyed the food, including taste, smell, and texture, while binge eating than those with BN. This suggests that in response to negative affect as a precursor, those with BED may experience an alleviation of negative affect from binge eating through enjoyment of food and thus, reduction in boredom/depressed mood.

**1.3.2.2. Alleviation of negative affect after binge eating.** There has been extensive support for the role of negative affect as a trigger for binge eating, while findings regarding the alleviation of negative affect after binge eating have been more mixed. For instance, Leehr and colleagues (2015) conducted a literature review and found only 2 studies that examined mood after food intake; both showed fewer negative emotions after intake compared to before, for both BED and obese participants. Similarly, Berg and colleagues (2015) found that in obese adults, global negative affect and guilt (as a specific facet of negative affect) increased in the 4 hours prior and decreased in the 4 hours following a binge eating episode. It has also been shown that BED patients ( $N = 49$ ) experience greater decreases in anxiety and physical discomfort as a result of binge eating compared with BN patients ( $N = 29$ ), as well as more relaxation (Mitchell et. al., 1999).

In contrast, a meta-analysis of 36 studies using EMA methods showed that negative affect was higher before binge eating than general affect, but that affect increased further after

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binge eating compared with pre-binge levels (average effect size = .50; Haedt-Matt & Keel, 2011). Consistently, Racine and colleagues (2015) found that 91% of individuals meeting full criteria for an objective binge eating episode reported an increase in shame, guilt and depression immediately after a binge eating episode. Further, it has been shown that shame and guilt are associated with binge eating, and that individuals typically express these emotions in relation to having binged previously (Duarte, Pinto-Gouveia, & Ferreira, 2014). Binge eating has also been shown to predict next-day negative affect in female college students through daily diary ratings of the PANAS, even when controlling for previous-day negative affect (Barker et. al., 2006). Hsu (1990) examined changes in affect over the course of a binge episode in 50 BN patients using a self-report questionnaire and found that many types of negative affect increased over the course of binge eating (before vomiting); these included guilt, depression, anger, frustration, feeling dirty, and exhaustion. However, anxiety was found to decrease over the course of binge eating, while being reported by 36 of the participants as a precursor for binge eating.

These inconsistent findings highlight the need for further research regarding the role of binge eating in alleviating negative affect as emotion regulation models suggest, as it may also be that negative affect is an eventual consequence of binge eating. Specifically, binge eating is associated with higher levels of depression (Grilo et. al., 2009) and it is possible that this is an eventual consequence of frequent binge eating. If so, it would be beneficial to understand the course of negative affect after binge eating, to more effectively develop interventions for this period of distress.

**1.3.2.3. Stress, depression, and negative affect.** There appears to be a strong relationship between binge eating behavior and psychological distress, particularly depression, given the aforementioned comorbidity findings. In fact, one study of 350 overweight (BMI >85th

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percentile for age and sex) treatment seeking children and adolescents (ages 8-18) showed that depressive symptoms directly predicted loss of control over eating using a structured interview and self-report questionnaires (Goossens, Braet, & Bosmans, 2010). Further, depressive symptoms were correlated with loss of control over eating as well as over-evaluation of eating, weight, and shape. Similarly, Napolitano and Himes (2011) found that depressive symptoms (measured with the Center for Epidemiological Studies Depression Scale [CES-D]) contributed significantly to binge eating symptomatology (BES scores) for both African American and White college students.

Depressive symptoms at baseline have also been shown to longitudinally predict future increases in binge eating symptomatology in a community sample of Dutch females (Spoor et al., 2006). Binge eating behavior was assessed by modifying the Bulimia subscale of the Dutch version of the Eating Disorder Inventory; the compensatory behavior item was removed. Interestingly, the opposite was not true; binge eating did not predict future increases in depressive symptoms. Another longitudinal study examined the role of depression (BDI) in binge eating (BES) for 89 obese adult women (ages in a CBT treatment program (Wardle, Waller, & Rapoport, 2001). At baseline, depressed mood was positively and significantly associated with both body dissatisfaction and binge eating. Depressed mood was also shown to be a significant mediator of the relationship between body dissatisfaction and binge eating at baseline. After treatment, improved body satisfaction significantly predicted reduced binge eating, even after controlling for weight loss, and reduced depressive symptoms was a significant mediator of this effect. Further, a greater reduction of depression was significantly associated with improved body dissatisfaction and reduced binge eating; this provides additional support for the relationship between binge eating, depression and body-focused negative affect.

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Overall, binge eating appears to be closely associated with depression and overall psychological distress, highlighting the importance of stress and negative affect for those with BED, particularly negative affect resulting from body and weight-related stressors. Given the aforementioned studies, stress may impact eating behavior for those with BED by leading to increased negative affect which triggers binge eating (See Figure 2).

**1.3.2.4. Negative affect and self-regulation.** One possibility is that negative affect plays a key role in the relationship between stress (particularly body-focused stressors) and loss of control over eating through decreased self-control, or the ability to inhibit a dominant response or activate a subdominant response (Nigg, 2017). Leehr and colleagues (2015) posit that if emotion regulation fails, other areas of self-regulation, such as control over eating, may fail as well. Emotion regulation can involve the increase, decrease or maintenance of positive or negative emotions; most emotion regulation theories involve the decrease of negative emotion through maladaptive behavior (Haedt-Matt & Keel, 2011). Thus, binge eating behavior may trace back to a failure in self-regulation that is caused by intense emotions. Similarly, Hofmann, Friese, and Roefs (2009) found that among female college students ( $N = 122$ ), automatic affective reactions to tempting food stimuli (assessed using Implicit Association Test for candy) impacted candy consumption less when inhibitory control (measured using a stop-signal paradigm), affect regulation, and executive attention were better (above average), indicating improved impulse control. Notably, all three factors moderated this relationship independently and were unrelated to each other.

One particularly important form of self-regulation, dietary restraint, is thought to be related to negative affect, and research in subthreshold and full-BED women ( $N = 92$ ) showed that those who experience both are particularly likely to experience more severe eating disorders

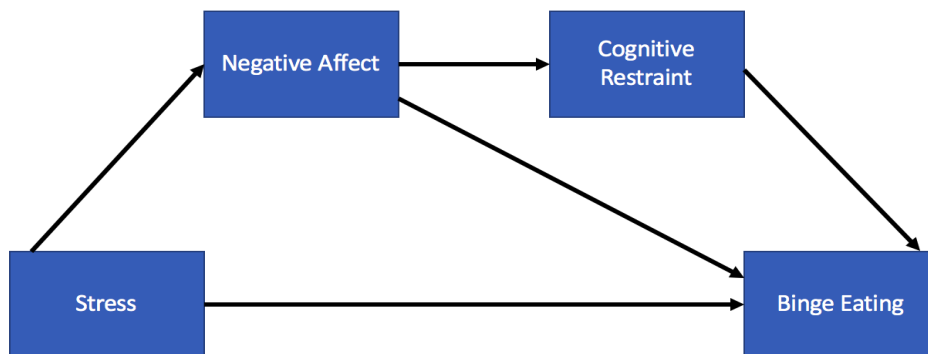


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and higher frequency of bingeing compared to those who experience only dietary restraint (Carrard, Crepin, Ceschi, Golay, & Van der Linden, 2012). These groups were created using BDI, Eating Disorder Examination-Questionnaire (EDE-Q), TFEQ Cognitive Restraint Scale, and Rosenberg Self Esteem Scale scores. The dietary restraint/negative affect group also evidenced a higher dropout rate during treatment compared with the dietary restraint-only group. Goossens and colleagues (2010) investigated the role of dietary restraint and depression in loss of control over eating in treatment-seeking overweight children and adolescents; they found that over-evaluation of eating, weight, and shape predicted dietary restraint, which in turn had a direct effect on loss of control over eating. Surprisingly, restraint and depression were not significantly correlated at all, providing further evidence that body-focused stress and negative affect may be particularly powerful in impacting cognitive restraint and in turn, binge eating.

Overall, these studies highlight the role of cognitive restraint in binge eating, and the interaction of negative affect with cognitive restraint, suggesting that this relationship should be further investigated. These cognitive processes appear to play an important role in the relationship between affect and eating, suggesting that those with BED may experience impairment of these key resources.

Figure 3. Stress Leads to Binge Through Negative Affect and Depletion of Restraint



### 1.3.3. Impairment of Cognitive Resources

Binge eating increases when individuals are under stress, and individuals that engage in self-control and exert cognitive restraint over a self-regulatory depletion task are more likely to binge later on unhealthy food, suggesting that stress and cognitive resources are key factors in binge eating (Groesz et. al., 2012; Friese, Engeler, & Florack, 2015). In fact, adult women with BED ( $N = 31$ ) have been shown to experience impairment on a cognitive control task compared to overweight healthy controls ( $N = 36$ ) when exposed to food stimuli (Svaldi et. al., 2014). An n-back task was used to measure participants ability to identify presence of a target stimuli a specified number of trials prior, indicating cognitive interference when performance on difficult (i.e., stimuli were presented recently) trials versus low difficulty trials is compared. A recent-probes task was also used to assess cognitive interference from previous information processing that has lost its relevance; participants indicated whether a target item was presented in a set of stimuli provided prior. Binge-related food items were included (i.e., chips, hamburger, butter) in both tasks. Results indicated that cognitive interference was greater for those with BED than controls in both tasks, as evidenced by response time and % errors, and this effect was significant in the n-back task. Further, the interference effect on response times in the recent-probes tasks was stronger for BED participants only when food-related items were presented compared with neutral items. Interference on the n-back task was associated with symptoms severity (i.e., dietary restraint, weight concerns, eating concerns, and shape concerns) assessed with the EDE-Q (questionnaire version of the EDE), as was the food-bias score based on recent-probes response times. Taken together these findings suggest that those with BED experience unique cognitive impairment when presented with food images, and these deficits are associated with increased binge eating severity overall, further supporting the role of cognition in binge eating.

**1.3.3.1. Cognitive restraint and eating.** One form of cognitive impairment that appears to play an important role in eating behavior is lack of self-control, or cognitive restraint (Johnson, Pratt, & Wardle, 2012). It is important to distinguish between dietary cognitive restraint and dieting; restraint reflects a perceived cognitive deprivation of highly palatable foods while dieting involves decreasing caloric intake for weight loss (Schulte, Grilo, & Gearhardt, 2016). Thus, cognitive restraint has been conceptualized as a corollary of hedonic hunger, which is denied through cognitive deprivation of palatable food intake. Dieting and cognitive restraint are typically related, but this behavior depends heavily on whether the individual is dieting currently. Individuals with BED experience frequent dieting and weight fluctuations, while about half identify dieting as a precursor for binge eating, or vice versa. Because individuals often engage in cognitive restraint over eating to maintain or lose weight, body shape and weight stress may predict restrained eating. Indeed, psychological and emotional stress appear to be related to general restraint; it has been reliably shown that increasing levels of psychological stress is associated with a decrease in behavioral self-control (Sinha, 2008). Additionally, those high in dietary cognitive restraint evidence stress-induced binge eating similarly regardless of distraction coping strategies, suggesting that restraint makes it harder to manage stress with these strategies (Freeman & Gil, 2004). Thus, there is support for lowered restraint in the face of stress, and stress may be even more impactful in the face of strict dietary restraint. As such, the relationship between stress and restraint may be especially important in the context of those who binge eat.

Restraint theory has been well-established in the clinical eating disorder literature, which posits that an eating style that is under cognitive, and not physiological, control induces counter-regulatory responses, lowers sensitivity to satiety signals, and can result in disinhibited, binge-like eating patterns when the cognitive control is undermined (Johnson et al., 2012). As early as

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the 1970's, research has shown that in response to attempts at reducing energy intake for weight control, individuals increase their intake of palatable, non-nutritious foods (Johnson et. al., 2012). Laboratory studies have shown that counter-regulation can be evoked through a high-restraint diet and binge eating severity (measured using the BES) has been shown to be related to increased overall habitual dietary restraint, or a chronic tendency to restrict food intake. (Johnson et. al., 2012; Marcus, Wing, & Lamparski, 1985). Further, a study of 1,678 same-sex female twins (ages 11-29) revealed that the impact of genetic and environmental risk factors on binge eating increases at higher levels of dietary restraint, as assessed using the Restraint subscale of the Eating Disorder Examination self-report questionnaire (Racine et. al., 2015). Binge eating was measured using the Binge Eating subscale of the Minnesota Eating Behavior Survey, a self-report questionnaire. The moderating role of restraint in this relationship suggests that those who may be vulnerable to developing the disorder are even more so when they engage in high restraint.

Along with the increasing evidence for the role of cognitive restraint through numerous studies and growing societal influence on dieting and restraint, dietary cognitive restraint has long been thought of as a precursor and causal factor for development of eating disorders (Johnson et. al., 2012; Polivy & Herman, 1985; Howard & Porzelius, 1999). However, evidence from naturalistic and longitudinal studies has been mixed. For example, a longitudinal study of 143 community-based Dutch women found that dietary restraint (measured using the Restrained Eating subscale of the Dutch Eating Behavior Questionnaire) did not predict future increase in binge eating, and binge eating did not predict future increases in dietary restraint (Spoor et. al., 2006). Similarly, dietary restraint was not shown to mediate the relationship between body dissatisfaction and binge eating (measured using the BES) in a sample of obese women ( $N = 89$

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at baseline) in a CBT-treatment study (Wardle et. al., 2001). Dietary restraint was assessed using the Cognitive Restraint subscale of the Three Factor Eating Questionnaire-revised; this score was not correlated with binge eating or body dissatisfaction at baseline. However, interactions revealed that high restraint was associated with greater binge eating only in the highly depressed group (BDI score in highest tertile), with decreased binge eating in the group with low depression (BDI score in lowest tertile). This effect approached significance in longitudinal data, with the greatest reduction in binge eating occurring for those who show decreased depression and increased restraint over time. Increased restraint over time was not associated with a reduction in binge eating, while it was associated with weight loss.

The relationship between dietary restraint and binge eating appears to be complex, and one important factor may be the degree and nature of stress experienced (Woods, Racine, & Klump, 2010). Online survey data from college females ( $N = 497$ ) indicated that restraint (the Restraint Scale), daily stress, and major life stress were positively associated with binge eating. Interestingly, binge eating was more strongly related to daily stress than major life stress. Further, daily stress moderated the relationship between dietary restraint and binge eating under high life stress only. Specifically, under conditions of high major life stress, high daily stress was associated with a stronger impact of restraint on binge eating. Under low life stress, daily stress and dietary restraint both independently predict increased binge eating. Thus, daily life stressors impact binge eating in the face of restraint differently under varying conditions of major life stress, providing further support for the role of stress and dietary restraint in the development of binge eating behavior.

Taken together, these findings suggest that dietary restraint does not invariably lead to increases in disinhibited eating, and this effect depends on a number of variables (i.e., depressive

symptoms and life stress). Consistent with this, the impact of restraint on eating behavior also appears to vary significantly when the restraint is more rigid and not engaged flexibly.

**1.3.3.2. Flexible versus rigid cognitive restraint.** Cognitive restraint has different effects on binge eating depending upon whether it is more disciplined, or “rigid” (e.g., I will never eat cake), as opposed to more lenient, or “flexible” (e.g., I will eat this cake now and eat healthier tomorrow). In a longitudinal treatment study of 50 obese (BMI = 30 or greater) BED patients, only increased flexible restraint predicted binge abstinence post-treatment and at a 3-month follow-up and predicted 5% weight loss post-treatment (Blomquist & Grilo, 2011). Specifically, higher flexible restraint scores over treatment increased binge abstinence likelihood by 3.53 times. Restraint subscales were measured using the Three Factor Eating Questionnaire (TFEQ) and binge abstinence was defined as having had no binge episodes for the past month.

Similarly, treatment outcomes for BED and obese individuals may vary somewhat based on the rigidity of cognitive restraint (Downe, Goldfein, & Devlin, 2009). 116 obese men and women (ages 18-75) diagnosed with BED were given group behavioral weight control treatment, as well as a randomized combination of CBT, placebo, and fluoxetine. Binge abstinence, defined as having no binge episodes for the previous 28 days, was associated with higher total, flexible, and rigid restraint scores than non-abstinence after treatment. 5 months after treatment, flexible restraint was marginally related to binge abstinence while rigid was not, and increased flexible restraint was associated with greater weight loss over treatment. Similarly, higher dieting success in 616 community-based, online survey participants was predicted by flexible control, while rigid control predicted poorer dieting success through increased food cravings (Meule, Westenhöfer, & Kübler, 2011). Weight loss and maintenance intervention for 49 obese individuals was also shown to be more successful when flexible restraint increased over

treatment; weight loss was greater and psychological distress was decreased (Sairanen, Lappalainen, Lapveteläinen, Tolvanen, & Karhunen, 2014). Reduction of rigid restraint over follow-up periods was associated with better maintenance of psychological improvement.

These findings converge to indicate that engaging in rigid restraint, instead of more flexibly approaching food intake, may put BED individuals particularly at risk of binge eating; however, it remains unclear precisely how this process unfolds. One possibility is that when control over eating is characterized by rigidity and strict self-deprivation, one's self-regulation is over-exerted, like a muscle. As a result, it may tire and fail on subsequent tasks, such as resisting tempting foods. This effect should be examined in this context further in order to better understand the role of cognitive control on binge eating behavior.

**1.3.3.3. Depletion of cognitive restraint.** The potentially exacerbating effects of rigid cognitive restraint on binge eating are consistent with the idea of ego-depletion, which has been proposed as a form of cognitive impairment from previous self-regulation (Baumeister, 2014). In this theory, self-regulation, or self-control, is a finite resource that is utilized when controlling impulses (i.e., inhibitory control); thus, use of this resource on one task may impair self-control on a subsequent task, such as control over eating, because the resource has been depleted. Ego-depletion has been well-researched, including a meta-analysis evidencing moderate effects (Hagger, Wood, Stiff, & Chatzisarantis, 2010), yet there is mixed support in the literature with a recent multi-lab registered replication yielding no significant effect (Hagger et. al., 2016). However, some have suggested methodological concerns (i.e., conceptual problems) for replication attempts (Lurquin & Miyake, 2017), supporting a need for additional research and clarity on this construct.

Although we should interpret these findings with caution, there have been some

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promising studies that have examined ego-depletion and its impact on eating. In fact, Vohs and Heatherton (2000) found that female dieters ( $N = 39$ ) ate more ice cream in a taste test after completing an emotion-regulation task, depleting cognitive restraint, than those who did not control their affect. Notably, those who were asked to suppress their emotions did not rate their mood as more dysphoric after the task, suggesting that negative affect does not fully account for the increase in consumption. In another study of community-based adult females ( $N = 67$ ), participants who completed a task depleting self-control subsequently ate more cookies in a taste test than those who did not (Friese et. al., 2015). Another study of college participants ( $N = 51$ ) showed similar results for the impact of ego-depletion on consumption of a sugary beverage, but found that positive mood induction negated this (Tice, Baumeister, Shmueli, & Muraven, 2007). Thus, the effects of ego-depletion may be particularly impacting in the absence of positive affect, suggesting that perhaps negative affect could play a key role in the consequences of ego-depletion.

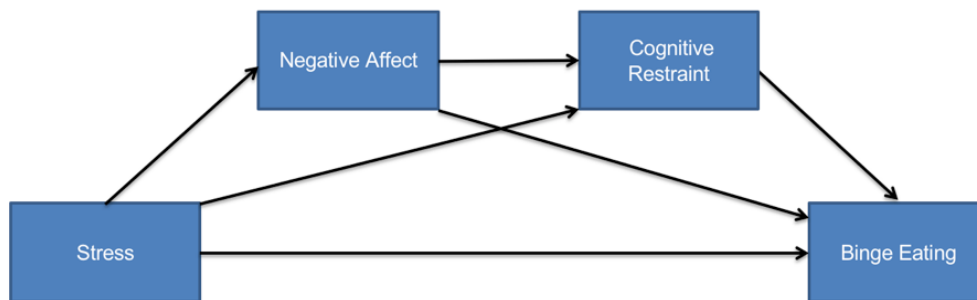
There appears to be some support for food-related cognitive restraint leading to ego-depletion effects on subsequent eating, suggesting that ego-depletion is especially relevant for control over food intake, a pervasive problem for binge eating individuals. In a sample of college females ( $N = 100$ ) classified as chronic dieters vs. non-dieters based on their dietary restraint levels, Vohs and Heatherton (2000) examined the impact of exposure to tempting food (i.e., chips, candy) and resisting consumption on later eating behavior. They found that dieters ate more ice cream in a taste-test paradigm after having been seated closer to tempting foods compared to sitting farther away. Interestingly, this interacted with the availability of the snacks, as participants were either told that they could eat the snacks or that they should not be touched. Dieters who were close to the snacks and told to help themselves ate more ice cream later than



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dieters who were seated far away and told to help themselves, suggesting that when palatable food is more available, those who are high in restraint evidence stronger effects of ego-depletion. They also found that 31 female dieters persisted for less time on an unsolvable puzzle after being seated closer to an overflowing bowl of M&M's than when seated further away. These findings emphasize the role of dietary restraint (as a trait) and cognitive control over eating tempting foods in ego-depletion effects, and further support the impact of this control on non-eating tasks. Taken together, the literature appears to suggest that the association between restrained eating and binge eating may be characterized by depletion of a key resource, cognitive restraint, which is impacted heavily by affect and stress (See Figure 4).

Figure 4. Negative Affect and Stress Lead to Binge Through Depletion of Restraint



Overall, these findings suggest that the relationships between stress, negative affect, cognitive resources and restraint are more complex than previously known, and the present research aims to more clearly elucidate this process. The implications for depletion of restraint resources on binge eating are especially important, and this mechanism remains unclear and debated in the literature. Indeed, it may be that this process leads to binge eating through increased impulsive behavior. Thus, when one can no longer exert cognitive inhibitory control after managing increased stress and negative affect (and perhaps engaging in dietary restraint), their impulsivity is enhanced, and out-of-control eating occurs.

#### **1.3.4. Impulsivity**

Stress and impulsivity are closely related, and both have been implicated together in addictive disorders, such as alcoholism and cigarette smoking (Helen, Keri, Peihua, & Rajita, 2010; Ansell, Gu, Tuit, & Sinha, 2012). Adolescents who have experienced several significant stressors (e.g., loss of a parent, sexual abuse) and are considered at risk for substance use disorders evidence decreased behavioral control and greater impulsivity (Sinha, 2008). Indeed, the impact of stress on inhibitory control suggests that subsequent impulsive behavior could be considered a reaction to stress. Further, children exposed to economic stress have been shown to grow up exhibiting more impulsive behaviors than those who were not exposed to stress in the family (Takeuchi, Williams, & Adair, 1991). Thus, there appears to be a strong relationship between stress and impulsivity, and this has important implications for BED given the key role of these factors in binge eating behavior.

Because of the proposed role of reward systems in eating behavior and the established role of these systems in impulsivity research, impulsivity has long been implicated in the development of binge eating disorder (see Schag, Schönleber, Teufel, Zipfel, & Giel [2013] for a review). Impulsivity, defined as responding to internal and external stimuli in an unplanned manner without regard to the consequences that might occur, is thought to underlie both addiction disorders and BED (Nigg, 2017; Schulte et. al., 2016). In substance use disorders, impulsivity is thought to explain maladaptive decision-making regarding their addiction; individuals engage in behavior that is rewarding in the short-term but detrimental in the long-term (i.e., health and interpersonal problems). As reviewed by Schulte and colleagues (2016), BED individuals have been shown to be more impulsive through results indicating that they make risky decisions, have trouble focusing attention, and are poor at utilizing feedback to guide

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future behavior. It is also thought to perhaps initiate binge cravings. Thus, the same maladaptive decision-making process appears to unfold for those who engage in binge eating despite the negative long-term consequences (i.e., health problems, guilt/shame). Consistent with comorbidity findings discussed above, BED has been shown in a number of studies to be related to trait impulsivity and disorders characterized by impulsive behavior, such as substance use disorders and attention-related disorders (Schag et. al., 2013). It has been suggested that increased food intake can develop when one puts a higher reward value on food and cannot inhibit their increased drive to eat. Thus, reward sensitivity and, as previously mentioned, inhibitory control may converge to support impulsive eating, and this process may occur more frequently in those with BED due to the characterization of binge eating as lacking in control.

As reviewed by Schag and colleagues ( $N = 51$  studies; 2013), observed natural food intake appears to vary for individuals based on weight and binge eating behavior. These observational findings suggest that increased food intake is observed for obese individuals, and this is even more pronounced for binge eaters, especially when told to eat how they like or when given multi-item meals. Further, test meal intake among 79 overweight participants has been shown to be greater for BED and subthreshold BED participants following an 8-hour fast than for controls (Galanti, Gluck, & Geliebter, 2007). Impulsivity (Barratt Impulsiveness Scale-11) and depressive symptoms (Zung Depression Scale) were also significantly higher in BED and subthreshold BED individuals, and impulsivity was related to test meal intake. It is worth noting that BED and subthreshold BED participants did not differ on test meal intake, further supporting the need to include these individuals in future research.

In addition to impulsive food intake, Schag and colleagues (2013) have shown that more cognitive-based spontaneous behavior may be especially salient for those with BED, compared

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to obese controls, based on a systematic review of the literature. They found that 10 studies examined the role of rash behavior using neuropsychological tests, eye-tracking methods, or electroencephalogram (EEG). In these studies, rash behavior was operationalized by ability to inhibit one's response or automatic preference to food-related stimuli (i.e., dot-probe task, go/no-go task etc.). For those studies using participants who are obese and have not been evaluated for BED, results were mixed. Most studies indicated no difference in inhibition for obese participants when compared to normal weight controls, with some sporadic positive findings (only when in a satiated state or early in the task). In contrast, the two studies that examined BED-diagnosed individuals in order to parse apart the role of disinhibition for BED, separate from obesity, were more promising. Higher EEG activation in response to food stimuli supports increased disinhibition toward food for those with obesity and BED compared to non-BED obese controls. Another study (Mobbs, Iglesias, Golay, & Van der Linden, 2011) used an inhibitory control task (go/no-go task) with food and body-related stimuli to examine differences between obese participants with ( $N = 16$ ) and without BED ( $N = 16$ ), as well as healthy (no BED) normal weight controls ( $N = 16$ ). Both obese BED participants and obese controls made more errors (pressing a key when a distractor is displayed) and omissions (failing to respond to a target stimuli) when presented with food and body-related stimuli (as opposed to neutral section of the task) than healthy controls did. However, BED participants made more errors and omissions than those without BED, suggesting that lack of inhibitory control in response to food and body-related stimuli is more specific to BED than obesity-status alone. Taken together, these findings suggest that those with BED show a stronger tendency toward impulsivity than obese individuals without BED, for which results have been inconsistent.

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Despite this evidence for the importance of impulsivity for those who binge eat, there have been some mixed results. A systematic review ( $N = 62$  studies) by Bartholdy, Dalton, O'Daly, Campbell, & Schmidt (2016) found that restrained eaters generally evidenced poorer inhibitory control on a stop-signal task than non-restrained eaters, but research in those with BED was sparse and inconsistent. A stop-signal task is a behavioral measure of reactive inhibition that measures an individual's ability to inhibit an already initiated response. The participant is asked to respond to a target stimulus and must inhibit their response when a "stop" stimuli appears after the target stimuli has already been presented. Many studies found no evidence of impaired performance for binge eaters, and samples often cut across diagnoses. Thus, there was not enough evidence to support impulsive behavior for BED individuals based on this widely used measure of inhibitory control. In contrast, Nasser, Gluck and Geliebter (2004) found that obese individuals with BED had higher motor impulsivity, measured with the Barratt Impulsiveness Scale, compared to those without ( $N = 33$ ). Interestingly, total impulsivity was positively correlated with some BED criteria: loss of control when eating, eating when not physically hungry, and eating alone due to embarrassment. BED criteria were measured using the Questionnaire on Eating and Weight Patterns (QEWP), a self-report questionnaire that assesses the individual criteria for BED, and participants also completed the BES. Impulsivity was also positively correlated with depressed mood rated before consuming the test meal when controlling for BED status, suggesting that negative affect before eating is related to overall impulsivity for all individuals. These inconsistencies highlight the complexity of the relationship between impulsivity and binge eating, suggesting that a comprehensive model including other relevant variables such as negative affect is needed to start to illuminate this process.

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Overall, the research regarding impulsivity and BED is mixed but promising, and further research is needed in this crucial area. Many studies evidenced higher impulsivity using diverse methodology in BED and obese participants; however, there were inconsistent findings as well and the role of impulsivity remains unclear. This is potentially due to the recognition now that impulsivity is not a heterogeneous construct, and it may be better understood as a compilation of unique constructs (Whiteside & Lynam, 2001).

**1.3.4.1. Facets of impulsivity.** A possible explanation for inconsistent results in impulsivity research could be the heterogeneous nature of impulsivity, which is now thought to reflect 4 distinct facets: urgency, sensation seeking, lack of planning, and lack of premeditation (Whiteside & Lynam, 2001; Gay, Rochat, Billieux, d'Acremont, & Van der Linden, 2008; Cyders & Smith, 2007). Premeditation, or the tendency to take into account the consequences for an action before engaging in it, is the most frequent conceptualization of impulsivity assessed by other measures. High scorers for premeditation are thoughtful and deliberate while low scorers act on spur of the moment and without regard to consequences. The next facet is urgency, which is rash behavior in response to distress, including the inability to resist cravings, bingeing, and acting rashly while upset. High scorers on urgency are likely to engage in impulsive behaviors in order to alleviate negative emotions despite the long-term negative consequences. Rash-based action may occur in response to positive emotions as well, as extreme positive emotion is sometimes a predecessor for risky behavior (Cyders & Smith, 2007). This was examined and supported using a multi-trait, multi-method matrix study. Positive urgency (rash behavior in response to positive affect) was later recognized as a distinct facet and included in commonly-used measures (Lynam, Smith, Whiteside, & Cyders, 2006); however, negative urgency is thought to more likely play a role in binge eating behavior due to the strong relationship shown

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between negative affect and binge eating. Negative urgency, measured using the UPPS Impulsive Behavior Scale (UPPS), has been shown to significantly predict errors on a task that involves inhibiting previously initiated responses on a go/no-go task in a sample of community-based adults ( $N = 126$ ; Gay et. al., 2008). The UPPS is a commonly used self-report measure of general impulsivity, as well as the distinct facets (with the exception of positive urgency).

The next identified facet is sensation-seeking, or a tendency to enjoy or pursue activities that are exciting; high scorers enjoy taking risks and engaging in dangerous activities, while low scorers avoid risk. Lastly, perseverance, or the ability to remain focused on a task that may be difficult or boring was identified. High scores for perseverance would indicate someone who is able to complete projects and work under conditions with distracting stimuli. Lack of perseverance has been shown to predict higher proactive interference in working memory on a recent-negative task (Gay et. al., 2008). It was also associated with more task-unrelated thoughts, an index of mind-wandering; however, it does not significantly predict them. Cyders and Smith (2007) have proposed through a series of factor analyses that lack of premeditation and lack of perseverance may reflect two components of one broader trait reflecting deficits in consciousness. Notably, Whiteside and Lynam (2001) did find these to map onto a common factor with other facets of consciousness when joint factor analyses were conducted with all impulsivity scales and all personality factors.

Thus, it may be that some facets are more closely related to binge eating than others, possibly accounting for some previously inconsistent results. In order to understand the role of impulsive behavior in stress-induced binge eating, these individual constructs must be considered independently in the context of other key variables (i.e., restraint and negative affect).

**1.3.4.2. The key role of negative urgency in binge eating.** Given the support for rash behavior and negative affect both occurring more frequently in those with BED, it follows that negative urgency, or the tendency to act rashly in response to negative emotion, is particularly prevalent for those with BED. This construct has been thoroughly researched in relation to binge eating and has shown promising results. It has been shown in same-sex female twins (ages 15-25) that women with full objective binge episodes (LOC and overeating), women with LOC only, and women with overeating only all experience higher levels of negative urgency than women with no pathological eating present (after controlling for BMI), suggesting that this construct may be relevant to both full and subthreshold BED ( $N = 612$ ; Racine et al., 2015). Eating behavior was assessed using the SCID-I and negative urgency was measured using the UPPS-P Impulsive Behavior Scale (UPPS-P), which is congruent with the UPPS with the addition of the positive urgency construct. These findings indicate that both the loss of control and objective overeating components of binge eating are related to increased levels of negative urgency, individually and in combination.

Further, negative urgency predicted binge eating frequency in an undergraduate female sample that endorsed binge eating in the previous 4 weeks ( $N = 186$ ; after controlling for BMI and other facets of impulsivity), while distress tolerance did not; NU is a distinct construct, despite their interrelatedness (Kelly, Cotter, & Mazzeo, 2014). Distress tolerance refers to one's ability to experience and accept negative affect; if one is not able to tolerate distress than it could be suggested that they may be more likely to act rashly in response to distress (i.e., negative urgency). Negative urgency was measured using the original UPPS scale, while distress tolerance was measured using the Distress Tolerance Scale and eating behavior was assessed using the EDE-Q. Notably, increased negative urgency predicted higher binge eating frequency



above and beyond disordered eating cognitions and depressive symptoms, and negative urgency was not a significant moderator of the relationship between these variables and binge eating frequency. Distress tolerance, in contrast, was significantly negatively correlated with binge eating frequency, but was not a significant predictor or moderator of binge eating. Overall, it appears that negative urgency is uniquely related to frequency of binge eating episodes, separate from related constructs such as distress tolerance, disordered eating attitudes and depressive symptoms. Interestingly, negative urgency (measured using the UPPS scale) has been shown to be even more strongly related to increased binge eating when emotional awareness, or the extent to which one attends to emotions and is able to identify and describe them, is low in college females ( $N = 249$ ; Manjrekar, Berenbaum, & Bhayani, 2015). Much like the aforementioned broader impulsivity research, BMI has been shown to predict higher UPPS-P scale negative urgency as well as cognitive inflexibility (measured using a Stroop task) in obese, overweight or normal weight adolescents ( $N = 63$ ; Delgado-Rico, Río-Valle, González-Jiménez, Campoy, & Verdejo-García, 2012).

**1.3.4.3. Negative urgency and cognitive control.** Negative urgency has also been shown to moderate the relationship between dietary restraint and binge eating disorder in a female first-year undergraduate study ( $N = 460$ ), and each of these constructs were independent significant predictors of binge eating, evidencing a relationship between cognitive restraint, negative affect and impulsivity (Emery, King, Fischer, & Davis, 2013). Negative urgency was assessed using the UPPS and the other variables were evaluated with the EDE-Q. After controlling for baseline binge eating frequency, both restraint and negative urgency led to incremental increases in binge eating frequency. When negative urgency was high, binge eating was not impacted greatly by changes in restraint. In contrast, at low to medium levels of negative urgency, increased restraint

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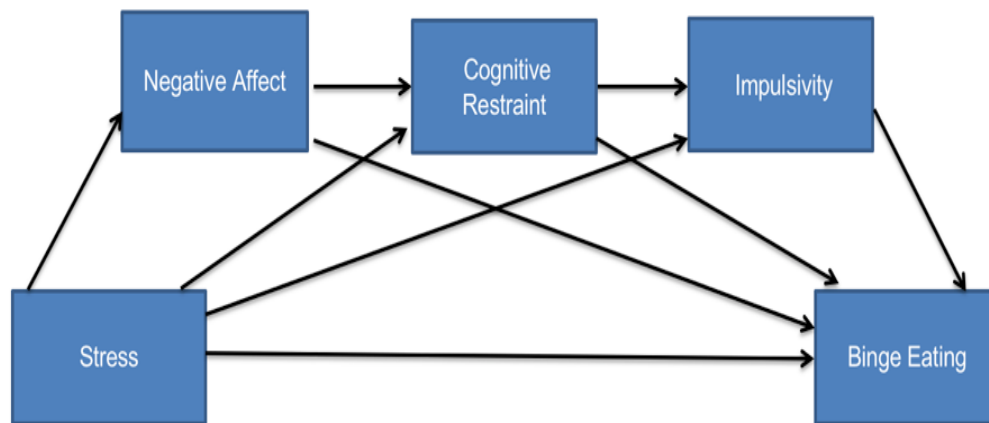
led to sharp increases in binge frequency. Similarly, in a study of 92 adult women meeting criteria for threshold or subthreshold BED, those who endorsed higher negative affect and dietary restraint evidenced higher negative urgency (measured with the UPPS) compared with those who only endorsed high dietary restraint (Carrard et. al., 2012). They also showed higher self-reported sensitivity to punishment. Thus, negative urgency and restraint appear to be closely related and may converge as an antecedent for binge eating behavior in those with BED, for whom these variables are particularly present.

Overall, while findings regarding the role of impulsivity have been somewhat mixed due to the heterogeneous nature of the construct and its measurements, it appears that those that binge eat are more impulsive overall, evidenced by both behavioral and neurophysiological studies. Further, there is some evidence for rash-behavior as a result of negative affect acting as an antecedent for binge eating behavior, especially in the context of dietary cognitive restraint. As reviewed by Schulte and colleagues (2016), it has even been implicated in (lack of) treatment adherence, which is vital to effective intervention. Thus, understanding the potential impacts of impulsivity for those who binge eat is important for developing treatments that will adequately address antecedents to binge episodes and encourage treatment engagement.

### 1.4. Conclusion

Taken together, the literature indicates that while negative affect, restraint, and impulsivity appear to independently play key roles in binge eating symptomatology, they may also combine, particularly in the face of stress, to precipitate binge eating episodes (See Figure 5). The process by which this occurs has yet to be illuminated; doing so would allow researchers and clinicians to develop more efficacious and targeted BED interventions. Thus, this project aims to examine these relationships in college-age participants and build a comprehensive model for stress-related binge eating, evaluating the role of these key variables using robust methodology.

Figure 5. A Comprehensive Conceptual Model for Stress-induced Binge Eating



## CHAPTER TWO

### STUDY ONE

#### 2.1. Hypotheses

Given the aforementioned relationships evidenced in the literature, the following hypotheses were tested in an initial cross-sectional study:

1. Stress directly predicts increased binge eating severity for both men and women.
2. Stress predicts increased binge eating severity indirectly through increased negative affect, dietary cognitive restraint, and impulsivity. The majority of research shows some gender differences for binge eating correlates; accordingly, we will examine this hypothesis among both men and women.

#### 2.2. Study Design & Methods

##### 2.2.1. Participants

Participants were 819 male and female psychology department subject pool participants ages 17-39 ( $M = 18.79$ ,  $SD = 1.48$ ). The majority of the sample identified themselves as White (89.3%), while 1.6% were African American, .2% were Pacific Islander, .9% were Native American, 1.7% were Asian, 1.7% were Hispanic/Latino, .1% were Brazilian, .1% were Jamaican, .1% were Haitian American, .1% were Dominican, .1% were Filipino, .8% were mixed race and 3.3% did not respond or indicated something other than a race/ethnicity (e.g., Catholic). BMI ranged from 16.69 to 48.46, while the average BMI was 24.71 ( $SD = 4.98$ ), or normal weight. Participants were directed to take a Qualtrics survey from the Sona Systems website and awarded credit toward introductory psychology course requirements for completion. Participants were only included in the main analyses if they completed the majority of scale

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items for each mean variable and all scale items for sum scores; this was done in an attempt to ensure unbiased variables. The remaining sample consisted of 735 participants (455 women and 280 men), evidencing a 10.3% dropout rate. The majority of the remaining sample was female (61.9%).

### **2.2.2. Procedure**

Study 1 was an online Qualtrics survey (Appendix F) composed of a variety of self-report measures for the hypothesized variables. Participants read a description of the study (Appendix A) on the Sona Systems website. Upon clicking on the study link, the participant was taken to a page to review the informed consent (Appendix C). It was explained that this study is about “eating behaviors and other psychological variables of college-age students, including stress and negative affect.” Participants were then given directions for each individual measure and asked to complete all questions honestly; however, they were informed that they can skip questions that might make them uncomfortable.

### **2.2.3. Measures**

The following self-report questionnaires were included in the online survey and administered, in randomized order, to each participant:

**2.2.3.1. Three Factor Eating Questionnaire-R18.** Binge eating behaviors have been shown to increase when individuals engage in cognitive restraint as a means of dietary control. Further, negative emotions have been linked to increased binge eating both before and after a binge eating episode. This 18-item measure is adapted from the original 51-item questionnaire, and is designed to assess various eating behaviors, specifically cognitive restraint, emotional eating and uncontrolled eating (Stunkard & Messick, 1985; de Lauzon et al., 2004). Separate mean scores were computed for each of these scales. On the cognitive restraint scale, data ranged

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from 1.00-4.67 ( $M = 2.50$ ,  $SD = .73$ ) with a Cronbach's alpha of .762 ( $N = 776$ ). On the emotional eating scale, data ranged from 1.00-4.00 ( $M = 1.97$ ,  $SD = .83$ ) with a Cronbach's alpha of .832 ( $N = 787$ ). On the uncontrolled eating scale, data ranged from 1.00-4.00 ( $M = 2.08$ ,  $SD = .60$ ) with a Cronbach's alpha of .848 ( $N = 755$ ). Other studies have shown reliability above .70 for all three subscales (de Lauzon-Guillain et. al., 2006). Sample items include "When I feel anxious, I find myself eating" and "I consciously hold back at meals in order not to gain weight."

**2.2.3.2. Binge Eating Scale.** This measure assesses severity of binge eating symptoms including both behavioral manifestations and thoughts/feelings surrounding a binge eating episode, and evidences good internal consistency reliability; Cronbach's alpha was .85 (Gormally, Black, Daston, Rardin, 1982). In this sample, a sum score was computed to reflect severity, which ranged from 0-45.00 ( $M = 10.49$ ,  $SD = 7.89$ ), and Cronbach's alpha was .914 ( $N = 778$ ). Scores equal to or greater than 27 are used to indicate severe binge eating symptomatology, while scores equal to or less than 17 indicate non-binge eaters (Celio, Wilfley, Crow, Mitchell, & Walsh, 2004). Sample item: "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."

**2.2.3.3. Positive and Negative Affect Schedule.** Negative affect is often associated with binge eating behavior, and studies have shown that individuals can experience increased negative affect both before and after a binge eating episode. This measure is designed to examine the severity of various kinds of positive and negative affect over the past week (Watson, Clark, & Tellegan, 1988). This information will allow us to examine global negative affect and how this relates to binge eating. In this sample, a negative affect scale was created by computing a sum score of negative affect items to reflect severity, which ranged from 10.00-50.00 ( $M = 24.00$ ,  $SD$

= 8.27), and Cronbach's alpha was .885 ( $N = 795$ ). Previous studies have found negative affect scale reliability of .85 (Crawford & Henry, 2004). Sample items include "excited," "upset" and "guilty."

**2.2.3.4. UPPS-P Impulsive Behavior Scale (revised version).** Previous research has shown a relationship between impulsivity, as well as individual facets of impulsivity, such as negative urgency. This scale is designed to assess overall impulsivity, as well as provide an index for the individual facets; urgency (positive and negative), sensation seeking, (lack of) premeditation, and (lack of) perseverance (Lynam et. al., 2006), allowing us to examine the relationship between individual facets of impulsivity and binge eating behavior. In this sample, a mean score was computed which ranged from 1.19-3.75 ( $M = 2.25$ ,  $SD = .38$ ), and Cronbach's alpha was .929 ( $N = 600$ ). Previous research has also supported good internal consistency, with Cronbach alphas of .87, .89, .85, and .83 for (lack of) Premeditation, Urgency, Sensation Seeking, and (lack of) Perseverance, respectively (Whiteside & Lynam, 2003). Sample items include "I always keep my feelings under control" and "Sometimes I do impulsive things that I later regret."

**2.2.3.5. Perceived Stress Scale.** Previous research suggests that stress is closely related to eating behavior, especially binge eating episodes. This scale is a measure of the degree to which situations in one's life are appraised as stressful (Cohen & Williamson, 1988). Items were designed to tap how unpredictable, uncontrollable, and overloaded respondents find their lives. This will allow us to examine the strength of the relationship between perceived stress and binge eating. A mean score was computed and ranged from 1.00-5.00 ( $M = 2.90$ ,  $SD = .75$ ); Cronbach's alpha was .878 ( $N = 796$ ). Previous studies have shown adequate internal consistency reliability as well, with a Cronbach's alpha of .89 (Roberti, Harrington, & Storch,

2006). Sample items include “In the last month, how often have you felt that things were going your way?” and “In the last month, how often have you been upset because of something that happened unexpectedly?”

**2.2.3.6. Demographics.** Demographic items (i.e., race, gender, age) were collected.

## 2.3. Results

### 2.3.1. Statistical Analyses

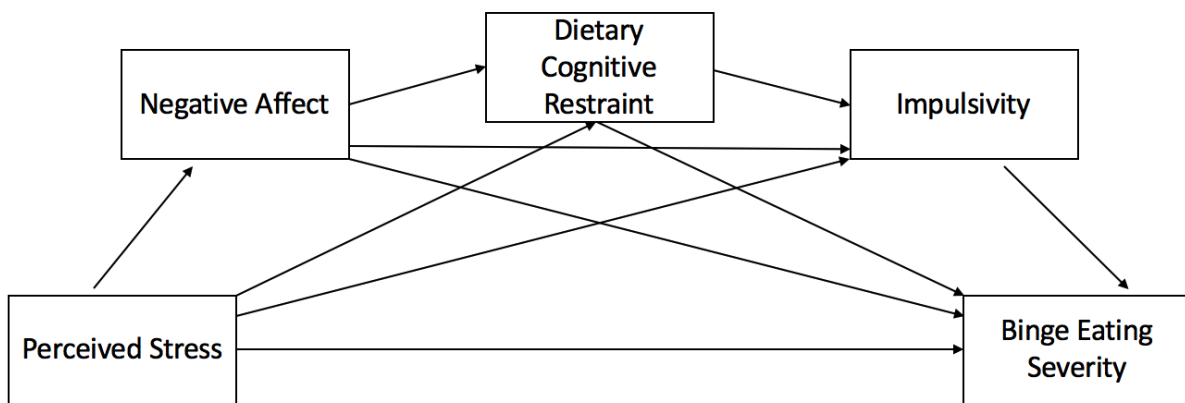
IBM SPSS V24 software was used to complete all statistical analyses (IBM Corp., 2016). All scales were computed and univariate (univariate outliers were windzorised; skewness and kurtosis values with Z score >1.96 were considered significant) and multivariate assumptions (multicollinearity:  $r$ 's > |.80| among variables and tolerance > .10, VIF < 10; multivariate outliers: studentized deleted residuals > +/- 2 and leverage >  $(2k+2)/n$ ,  $dfbeta$  > +/-  $2/\sqrt{[n]}$ ) were investigated. Variables with significant skewness were appropriately transformed (log transformation for substantial positive skew and square root transformation for moderate positive skew; Tabachnick & Fidell, 2007) and models were tested on the transformed data. As the hypothesized indirect effects remained the same, and log transformed variables are not as easily interpreted as the original scaled values, we present statistics and models utilizing the untransformed data. Six participants were identified as multivariate outliers with undue influence and were excluded from analyses. Descriptive statistics were obtained for each proposed variable and relationships among these factors were investigated through correlational analyses (See Table 1). Andrew F. Hayes' PROCESS mediation program was used in SPSS to analyze the proposed model (Figure 5; Hayes, 2017; IBM Corp., 2016). This regression-based analysis tests direct and indirect pathways between a proposed predictor variable (perceived stress) and outcome variable (binge eating severity); the indirect pathways occur through mediator variables



## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

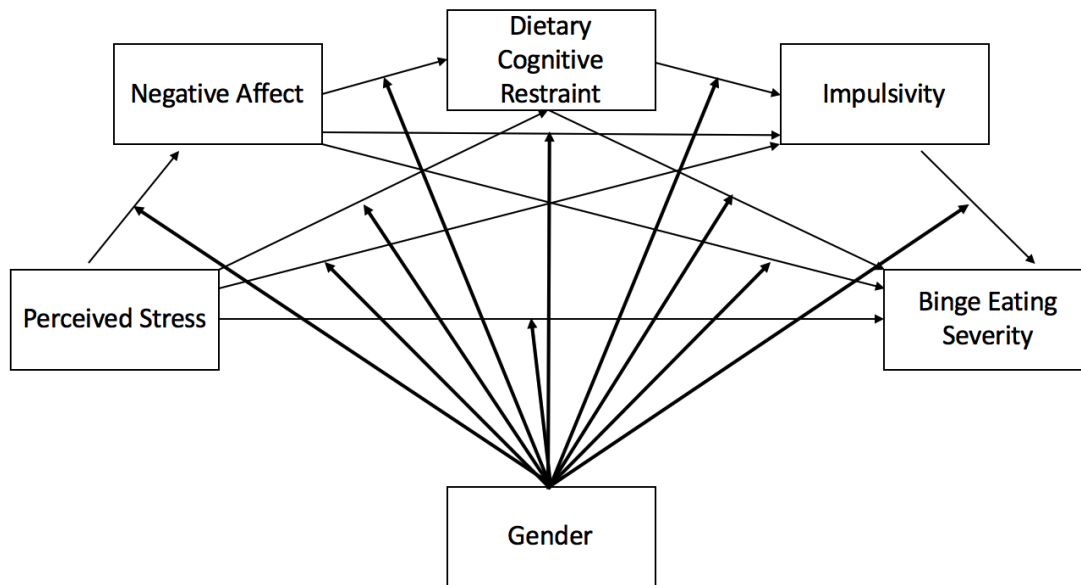
(negative affect, cognitive restraint, and impulsivity). Direct pathways between all variables are analyzed in order to better understand the impact of each factor on the others. Significant effects are identified by bootstrap estimation (based on 5,000 samples) of the 95% bias corrected confidence interval that does not contain 0. In order to further examine the gender differences for binge eating behavior, gender was included as a moderator for the mediation model. Thus, the statistical model investigated in this study used regression-based analyses to examine the direct and indirect effects of stress on binge eating for both men and women (See Figure 6).

Figure 6. All Pathways Tested for Study 1



Gender was examined as a moderator (See Figure 7) to determine whether any of these pathways differ for men and women. The effect size of the proposed model is determined by obtaining  $R^2$ , or the measure of the proportion of the variance of binge eating severity accounted for by the model.

Figure 7. All Pathways Tested with Moderation by Gender



### 2.3.2. Correlations and Descriptives

Consistent with the reviewed and hypothesized interconnectedness of the proposed variables, all correlations were significant at the .05 level (or lower) with only two exceptions (Table 1). Interestingly, cognitive restraint and impulsivity were not related for both men and women ( $r = -.04$  and  $r = .00$ , respectively), and restraint was not associated with negative affect for men ( $r = .10$ ). The latter was the only distinction between these relationships for men and women, indicating that these variables are connected in similar ways for both genders. Overall, effect sizes for significant correlations ranged from .014-.462, indicating small to large effects. Descriptive statistics and independent samples t-tests revealed that women were significantly ( $p < .01$ ) higher than men in perceived stress, negative affect, cognitive restraint, and binge eating severity, while men were higher in impulsivity. Cohen's  $d$  was calculated as a measure of effect size (.20 = small, .50 = medium, .80 = large; Lakens, 2013); effects ranged from small (negative affect and impulsivity) to medium (binge eating severity, perceived stress, and cognitive

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restraint). Notably, binge eating differences showed medium effects and this is consistent with previous binge eating literature (Striegel-Moore et. al., 2009).

Binge Eating Scale averages ( $M = 7.79$  for men and  $M = 11.82$  for women) were below the threshold indicating little to no binge eating (less than or equal to 17), suggesting that overall the sample represents minimal binge eating behavior; 645 (83.7%) participants fell in this range (Celio et. al., 2004). 99 participants (12.8%) fell in the subthreshold range (18-26) while 27 (3.5%) had scores that indicate the possible presence of significant binge eating symptomatology (equal to or greater than 27). Interestingly, 5% of women and 1% of men in the sample fell in this range, which is somewhat consistent with the aforementioned prevalence literature.

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Table 1. Study 1 Descriptive Statistics and Correlations by Sex

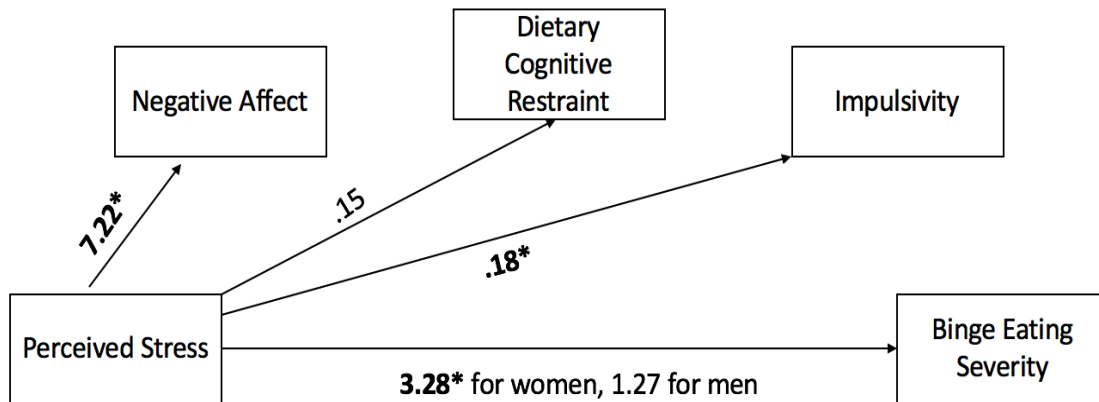
<b>Correlations</b>	1.	2.	3.	4.	5.
1. Perceived Stress	---	.67***	.12**	.42***	.41***
2. Negative Affect	.68***	---	.16***	.37***	.33***
3. Cognitive Restraint	.15**	.10	---	.00	.16***
4. Impulsivity	.42***	.33***	-.04	---	.31***
5. Binge Eating Severity	.34***	.33***	.15*	.32***	---
<b>Descriptives</b>					
<b><i>M(SD)</i></b>					
Men	2.70(.70) <sub>a</sub>	22.79(7.77) <sub>a</sub>	2.26(.67) <sub>a</sub>	2.30(.33) <sub>a</sub>	7.79(6.42) <sub>a</sub>
Women	3.00(.73) <sub>b</sub>	24.49(8.28) <sub>b</sub>	2.65(.72) <sub>b</sub>	2.20(.39) <sub>b</sub>	11.82(7.83) <sub>b</sub>
Cohen's d	0.562	0.212	0.561	0.277	0.562

Note: \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ . Pearson  $r$  linear correlations for women ( $n = 455$ ) above the diagonal, men ( $n = 280$ ) below. Means, followed by standard deviations within parentheses, are presented for each dependent variable by gender at the bottom of the table. Means with different subscripts in the same column are significantly different at  $p < .01$ .

**2.3.3. Mediation Models**

The overall model accounted for 25.4% of the variance ( $R^2$ ) in binge eating symptomatology. As predicted, PROCESS serial mediation analyses revealed that for women, stress directly predicted increased binge eating severity ( $b = 3.28$ ; 95% BCa CI [2.15, 4.40]) such that higher perceived stress leads to greater binge eating severity (Figure 8). Stress also directly predicted greater negative affect ( $b = 7.22$ ; 95% BCa CI [6.22, 8.21]) and impulsivity ( $b = .18$ ; 95% BCa CI [.10, .25]) for both men and women. Contrary to predictions, no direct path was observed for perceived stress to dietary restraint ( $b = .15$ ; 95% BCa CI [-.01, .31]) for either gender.

Figure 8. Direct Paths from Perceived Stress to Mediators and Binge Eating Severity



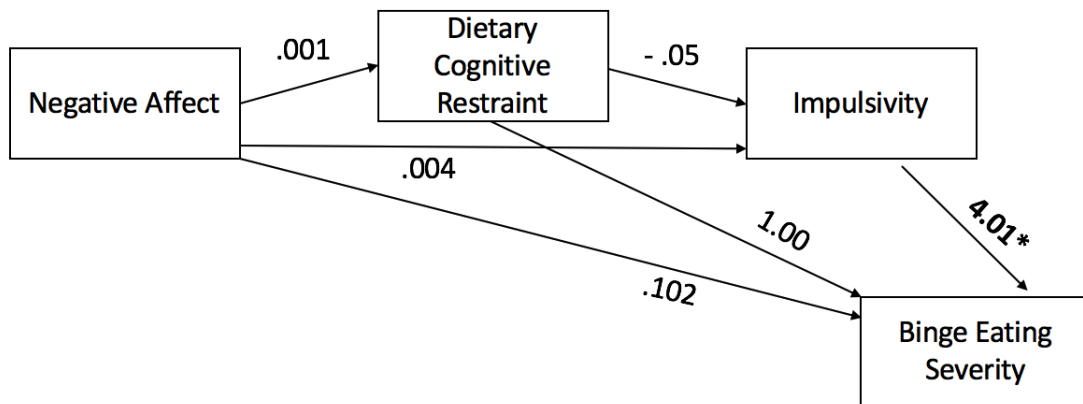
\*\*Note. Unless indicated, coefficients do not differ by gender

Next we consider the significance of paths between each of the mediators, and between the mediators and binge eating severity (Figure 9). For both men and women, no significant relationships were observed for the effect of negative affect on dietary restraint ( $b = .001$ , 95%

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BCa CI [-.01, .02]), impulsivity ( $b = .004$ ; 95% BCa CI [-.003, .011]) or binge eating severity ( $b = .102$ ; 95% BCa CI [-.036, .239]). Similarly, restraint did not evidence a significant effect on impulsivity ( $b = -.05$ ; 95% BCa CI [-.11, .01]) or binge eating severity ( $b = 1.00$ ; 95% BCa CI [-.17, 2.17]) for either gender. Consistent with predictions, greater impulsivity did predict greater binge eating severity ( $b = 4.01$ ; 95% BCa CI [1.48, 6.54]).

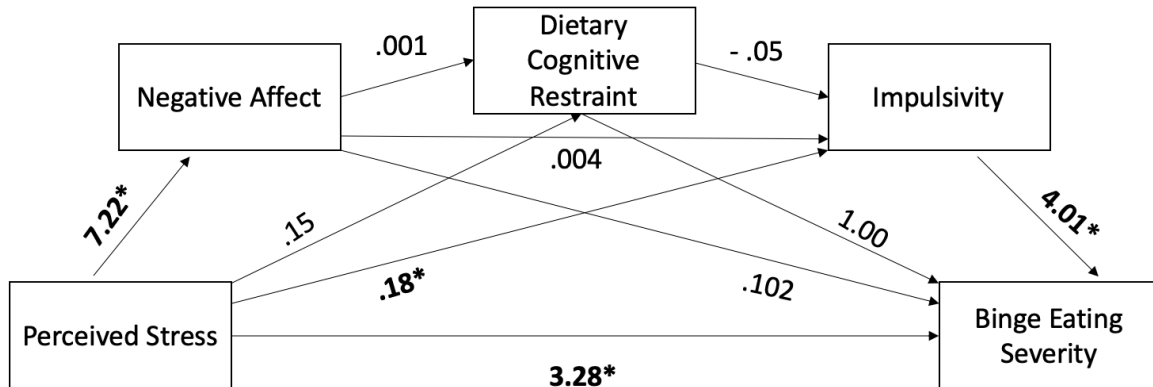
Figure 9. Direct Paths Between Mediators and Binge Eating Severity



Finally, we examine the hypothesized indirect effects. Does perceived stress impact binge eating severity through the proposed intervening variables? Of note, the test of the indirect effect examines the significance of each combined pathway from stress to binge eating (e.g. stress → negative affect → binge eating severity) and these indirect effects can be significant even if one of the component direct pathways is not, particularly for marginal direct effects (e.g. stress → negative affect, or negative affect → binge eating severity). The full model for women is presented in Figure 10.

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Figure 10. Full Model of Direct Effects of Stress on Binge Eating Severity for Women

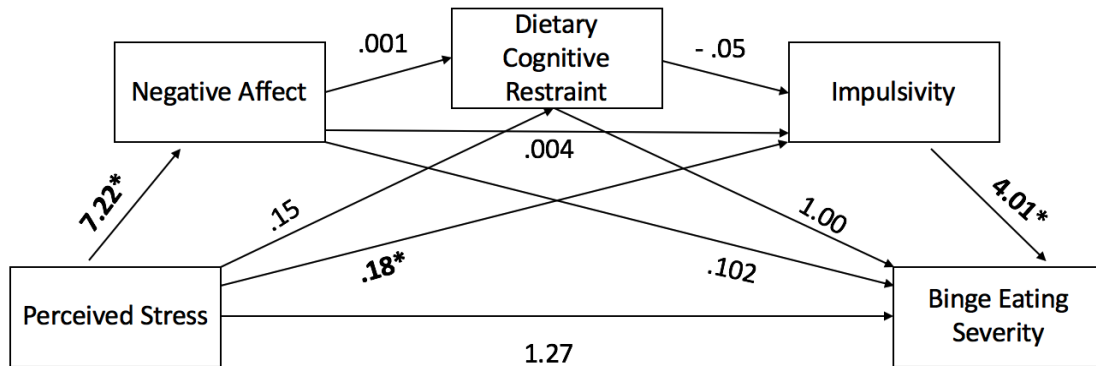


The largest indirect effects for women involved impulsivity. Stress was directly related to impulsivity which in turn was positively related to binge eating severity (stress → impulsivity → binge eating severity;  $b = .485$ , 95% BCa CI [.169, .891]). Stress also predicted greater negative affect which in turn predicted greater impulsivity leading to greater binge eating severity (stress → negative affect → impulsivity → binge eating severity;  $b = .217$ , 95% BCa CI [.060, .414]). Additionally, there was a small but significant effect of stress on binge eating severity through greater negative affect and cognitive restraint (stress → negative affect → restraint → binge eating severity;  $b = .112$ , 95% BCa CI [.005, .299]).

The primary difference observed in the model for men (significant moderation by gender) was the lack of a direct effect of stress on binge eating severity ( $b = 1.27$ , 95% BCa CI [-.29, 2.82]; see Figure 11). Further, only the largest of the indirect effects observed for women was significant for men. Stress exerted a significant indirect effect on binge eating severity through impulsivity (stress → impulsivity → binge eating severity;  $b = .705$ , 95% BCa CI [.277, 1.24]).

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Figure 11. Full Model of Direct Effects of Stress on Binge Eating Severity for Men



### 2.4. Discussion

This initial cross-sectional dissertation study aimed to investigate the correlational relationships between the proposed variables and to provide preliminary evidence for the proposed model of stress-related binge eating using regression-type analyses. Undergraduate participants ( $N = 735$ ) completed an online survey composed of self-report questionnaires designed to assess perceived stress, negative affect, dietary cognitive restraint, impulsivity and binge eating. The proposed model was strong, accounting for 25.4% of the variance of binge eating severity. There is some evidence for the proposed processes of stress-induced binge eating in both men and women, particularly through impulsivity. Despite some differences in specific effects, results are mostly consistent between genders, suggesting that these are relevant variables for all binge-eating individuals who experience stress. Results indicate that these variables are closely related in this sample, with the notable exception of restraint and impulsivity for men and women. One possible explanation for this finding is the specifically dietary nature of the measure used to operationalize cognitive restraint; perhaps impulsivity is more closely related to more general cognitive control deficits. Further, results indicated that women were higher in stress, negative affect, cognitive restraint, and binge eating severity than



men, while men were more impulsive. These differences were not surprising given the literature on these variables.

### **2.4.1. Support for Hypotheses**

The hypothesized relationships were investigated using PROCESS serial mediation analyses, and this proposed model accounted for 25.4% of the variance for binge eating severity. Findings regarding the specific relationships were somewhat in line with hypotheses. First, stress directly predicted increased binge eating, but in contrast to predictions, this was only true for women. Further, all variables were significant mediators in an indirect pathway, suggesting that each mediator plays a key role in binge eating related to stress. Stress-related impulsivity appears to predict increased binge eating for both men and women, while women evidence unique indirect effects of stress through negative affect and restraint and through negative affect and impulsivity.

Findings suggest that for both men and women, stress indirectly impacted binge eating behavior through impulsivity; for women, indirect effects were also present through negative affect and cognitive restraint, and negative affect and impulsivity, suggesting that these variables are playing a role for women as well. Thus, the full proposed model appears to be especially relevant for women, but indicates a similar relationship between stress, impulsivity and binge eating for all participants regardless of gender. Although each variable played a role in the indirect effects of stress on binge eating for women, the largest pathway through each mediator (i.e., stress → negative affect → restraint → impulsivity → binge eating) was not significant for men or women. As such, it remains unclear exactly how these variables interact in the process of stress and binge eating. Despite this uncertainty, this preliminary evidence for the proposed variables is promising.

#### **2.4.2. Limitations**

One limitation of this study is that the data are cross-sectional and correlational; no relationship represented here is truly predictive and so causation cannot be inferred. Further, stress can vary markedly based on perception. As such, the use of only perceived stress in this study is a weakness because it is unclear whether participants are experiencing objective stress, or if individuals with BED are more vulnerable to stress. To address these methodological limitations, an experimental study using robust psychophysiological measures is proposed as a second study for this dissertation project.

**CHAPTER THREE**

**STUDY TWO**

**3.1. Social Evaluative Stress Response and Impulsivity**

The exciting findings from study 1 offer some insight into the possible structure of stress-related binge eating processes. In particular, Study 1 demonstrated the importance of stress related increases in impulsivity as a possible driver of binge eating. It could be the case, for example, that individuals with BED are more vulnerable to stressors leading to greater impulsivity.

To address this, a second study was conducted using correlational methodology across two time points to examine the impact of acute social-evaluative stress on impulsivity and drive to eat among individuals varying in binge eating severity. Robust measurements were collected using psychophysiological equipment to measure the stress response and examine moderation by binge eating severity. In addition, Study 2 examines high-frequency heart rate variability (HF-HRV) reactivity to stress which will allow this project to build on previous research showing that parasympathetic vagal tone is decreased in those with BED (Friederich et. al., 2006).

**3.1.1. Physiological Stress Reactivity**

The stress-response involves the body's attempts to restore homeostasis, an evolutionarily adaptive process that can become harmful when stress is chronic and elevated in response to perceived stressors (Sapolsky, 2004). The autonomic nervous system, the part of the nervous system involved with involuntary processes, plays an important role in the stress response. One half of the autonomic nervous system is activated during the stress-response, the other is suppressed. The sympathetic nervous system originates in the brain and branches to every organ, blood vessel, and gland in the body; it is in this system that the stress-response

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occurs. Termed the “fight or flight” response, it is the sympathetic nervous system that is responsible for releasing adrenaline and diverting blood to muscles to be ready for action. In contrast, the parasympathetic nervous system is involved with calm, vegetative activities like reproduction, digestion, and food intake; “rest and digest.” The two work in opposition to manage the body’s vital organs and when one is activated, the other is inhibited. When the stressor has passed and the body must return to a calm state, the parasympathetic system is activated through the vagus nerve.

As part of the sympathetic nervous system, two key axes are involved in the stress-response, the sympathetic-adrenomedullary (SAM) axis and the hypothalamic-pituitary-adrenocortical (HPA) axis (Adam & Epel, 2007). The control centers for the stress-response are located in the brainstem and the hypothalamus. SAM starts in the locus ceruleus and stimulates release of hormones called epinephrine, from the adrenal glands, and norepinephrine, from all other sympathetic nerve endings throughout the body. These hormones act within seconds to kick organs into gear (Sapolsky, 2004). The HPA axis originates in the hypothalamus, which secretes corticotropin-releasing hormone (CRH), stimulating the release of corticotropin (ACTH) from the anterior pituitary. This hormone then acts on the adrenal cortex to stimulate the release of glucocorticoids cortisol and corticosterone. Glucocorticoids are steroids that act similarly to epinephrine over the course of minutes or hours. These hormones are essential for mobilizing energy during stress, preparing the body for action.

The “harmfulness” of the stressor has implications for the physiological stress response; the SAM axis is high when a stressor is perceived as challenging, but manageable given one’s resources to cope, whereas both SAM and HPA are high when the stressor is deemed uncontrollable which is considered a distress or threat response (Adam & Epel, 2007). As such,

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the stress response depends heavily on the type of potential stressor (or pressure) used, as not all potential stressors lead to a “threat” response. Stressors likely to elicit a physiological response often combine both a self-relevant task (individual is motivated to perform well) and social evaluation (Tomaka, Blascovich, Kelsey, & Leitten, 1993; Denson, Spanovic, & Miller, 2009; Dickerson & Kemeny, 2004; McCoy, Hutchinson, Hawthorne, Cosley, & Ell, 2014). Yet not all individuals respond to a stressor with equivalent intensity or distress. This highlights the importance of measuring the intensity and type of stress reactivity, and this can be done using a number of physiological biomarkers derived from psychophysiological measures (Berntson, Quigley, & Lozano, 2007). Extensive research has been done in both physiological and psychophysiological fields regarding stress reactivity, or physiological response to a stressor (Linden, Earle, Gerin, & Christenfeld, 1997). In the current research, we focus on measurement of cardiovascular and hemodynamic measures that are likely to indicate a distress response: increased mean arterial pressure and decreased high frequency heart rate variability (e.g. Connell et. al., 1987; Thayer, Åhs, Fredrikson, Sollers III, & Wager, 2012).

### **3.1.1.1. Cardiovascular measures of stress.**

**3.1.1.1.1. Mean arterial pressure.** Blood pressure (BP) is defined as the pressure exerted on the walls of blood vessels in the body; in the current study it is measured using a finger cuff that applies modest pressure to the artery over bone, sensing the largest pulse wave amplitude (Berntson et al., 2007). Systolic blood pressure is the highest point as it is the blood pressure taken while the heart is pumping blood out into the body. Diastolic blood pressure is the lowest; it is taken when blood is filling the heart and is therefore not available to exert as much pressure on blood vessels in the body. The most commonly used marker of blood pressure is mean arterial pressure, which is calculated by finding one-third of the sum of systolic and diastolic pressure,

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where diastolic pressure is multiplied by 2 given the larger amount of time that the heart is in diastole. This gives an average index of blood pressure or mean arterial pressure (MAP). MAP increases with activation of SAM due to a stressor. Importantly for the current work, prolonged activation of MAP following an acute stressor is associated with a distress response (Juster, Perna, Marin, Sindi, & Lupien, 2012). Distress responses, which occur when one is overwhelmed by a stressor, involve co-activation of both SAM and HPA (cortisol release). Cortisol administration has been shown to elevate MAP (Connell, et al., 1987) and increased cortisol reactivity has been shown to increase risk for later development of hypertension (Hamer & Steptoe, 2012). While MAP reactivity during an acute stressor does not differentiate a distress response from a healthier challenged response, sustained MAP reactivity following a stressor is suggestive of distress and co-activation of SAM and HPA (Juster, et al., 2012). Accordingly, I propose that for those individuals higher in binge eating, greater MAP reactivity during recovery will be associated with greater impulsivity. Further, I will examine whether greater binge eating severity will be associated with greater MAP in recovery.

**3.1.1.1.2. High frequency heart rate variability.** Heart rate variability (HRV), or beat to beat fluctuations of the heart, can be used to index vagal cardiac control. Power spectral analysis of inter-beat interval time series is often used to calculate HRV, resulting in high-frequency (0.15 - 0.40 Hz) and low frequency (0.01 - 0.15 Hz) bands (Thayer et. al., 2012). HRV in the high frequency band (HF-HRV) generally corresponds to respiratory sinus arrhythmia (RSA) which reflects respiratory gating of autonomic control and vagal tone. This is important because the vagus nerve is a vital component of the parasympathetic nervous system and can be used as an index of overall parasympathetic functioning. As discussed above, the parasympathetic nervous system (PNS) is part of the autonomic nervous system (ANS), which includes nervous system

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components responsible for automatic, involuntary processes in the body, such as breathing or hormonal release. The parasympathetic nervous system is involved in maintaining homeostasis in the body and counteracting the sympathetic nervous system, which is involved in the stress response. Parasympathetic cardiac control is important as an index of recovery from stressors and the ability of the PNS to maintain control over the heartbeat, which is important for cardiovascular health. Thus, vagal tone represents parasympathetic cardiac control, and these can be indexed by HF-HRV. In contrast, lower frequencies represent a mixture of sympathetic and parasympathetic influences.

HF-HRV shows that an individual's cardiac regulation is responsive to external and internal stimuli, which is adaptive and thought to evidence healthy cardiac functioning (Thayer et. al., 2012). A dysregulated cardiac system would respond in a particular pattern that is "locked in" instead of being adaptable. Thus, it has been suggested that HF-HRV can serve as an index for the brain's "integrative" system, which regulates the activity of cognitive, perceptual, motor, and physiological systems to adaptively respond to signals from in and outside the body. This neural system would be activated by perceptions of stress, suggesting that HF-HRV can provide insight into both cardiac and neural processes involved in a stress response. Indeed, a meta-analysis of 8 neuroimaging studies (i.e., Positron Emission Tomography [PET] and functional Magnetic Resonance Imaging [fMRI]) showed HRV to be associated (direction not specified) with neural structures involved in the perception of threat and safety: the amygdala and medial prefrontal cortex (Thayer et. al., 2012). This supports HRV as a marker for cardiovascular health and stress, and also provides some insight regarding the neural processes that underlie the stress response. HF-HRV has been shown to be depressed in obese individuals, and one study in obese BED participants showed that these individuals experienced decreased HF-HRV during a mental

task, even when compared with weight-matched controls (Friederich et al., 2006). Accordingly, we hypothesize that individuals higher in binge eating severity will have lower HF-HRV during the stressor. Further, we will examine if greater decreases in HF-HRV in response to stress are related to impulsivity.

### **3.2. Hypotheses**

Based on the results of study 1 and the aforementioned literature, the following hypotheses will be tested for college-age individuals that experience social-evaluative stress:

1. Stress reactivity (MAP, HF-HRV) will interact with binge eating severity to predict impulsivity. Specifically, more maladaptive stress responses (increased MAP during recovery; decreased HF-HRV) will be associated with higher impulsivity for those who are higher in binge eating, while this relationship will be present but significantly weaker in those who are lower in binge eating.
2. Higher binge eating severity will be associated with higher MAP during recovery, and lower HF-HRV during the stressor task.

### **3.3. Study Design & Methods**

#### **3.3.1. Participants**

Participants ( $N = 115$ ; over the age of 18) were women recruited via the Psychology Department Sona Systems website. Participants reflect the demographic characteristics (race, age, SES) of the Introductory Psychology Subject Pool. They volunteered for participation using the Sona Systems website after reading a brief description and were compensated with up to 3 credits for completion of the study. The collected sample size exceeded the proposed samples size of 70, which was selected based on related effect sizes in the literature (small to medium). A sensitivity analysis with power of .80,  $\alpha = .05$ , and the proposed  $N$  of 70 suggested that the



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statistical test for the primary hypothesis (moderated regression, 2 predictors) will be sensitive to effect sizes for the increase in  $R^2$  as small as .11 (medium effect size = .15). Participants were only included in the main analyses if they completed the majority of scale items for each mean variable and all scale items for sum scores; this was done in an attempt to ensure unbiased variables. The remaining N for each model in the main analyses ranged from 76-92.

### 3.3.2. Procedure

For the full study, participants read a description of the study on the Sona Systems study page (Appendix B). There were two parts to this study. The first was an online survey which takes approximately 20 minutes to complete and included informed consent (See Figure 12; Appendix D).

Figure 12: Study 2 Online (Part 1) Survey Measures

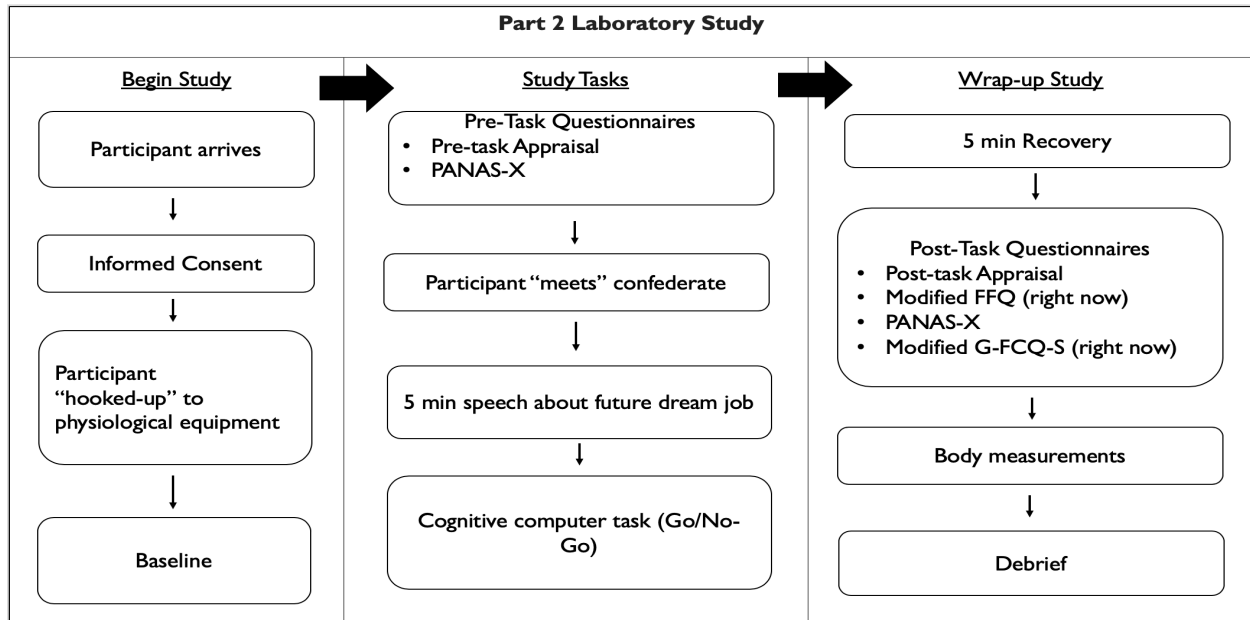
- 
- Part 1 Online Survey  
(completed  
independently)**
- Binge Eating Scale
  - UPPS-P Impulsive Behavior Scale
  - Self Control Scale
  - Perceived Stress Scale
  - Food Frequency Questionnaire
  - Weight Stigma Scale
  - PANAS-X
  - TFEQ-18
  - MASC-SF
  - Demographics

Participants were asked to complete this survey before taking part in the second portion, which took place in the laboratory. For a procedure timeline of the laboratory study see Figure 13. Participants first came into the lab and reviewed and signed a paper copy of the informed

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

consent. Participants were led to believe that the researchers were interested in first impressions and cognitive functioning. The purpose of this was to avoid priming participants with thoughts about food and eating before they completed the study tasks/questionnaires. Non-invasive sensors for psychophysiological equipment (i.e., electrocardiograph, impedance cardiograph and blood pressure) were applied by an experimenter, and participants then sat and rested for 10 minutes to collect baseline measures. They were then introduced to a fake confederate on a computer screen (a previously recorded video of a research assistant) and told that the person on the screen was another participant who would be evaluating the quality of their speech. Participants were asked to give a 5-minute speech about their future goals and plans. Participants were informed that the confederate was listening to the speech via intercom, but that video was turned off to avoid distraction. The speech task was video recorded. Before giving the speech, participants answered questions about their appraisal of the task (i.e., do they expect it to be difficult) and their current feelings. They then completed a cognitive stop-signal task on a computer monitor after the speech. Afterward, participants answered another questionnaire regarding appraisal of the task, thoughts/feelings and food cravings. Body measurements were then collected by a female experimenter. They were then fully debriefed (Appendix E) and awarded credit.

Figure 13: Part 2 Laboratory Study Timeline



### 3.3.3. Measures

**3.3.3.1. Three Factor Eating Questionnaire-R18.** This measure is the same as the measure used in Study 1.

**3.3.3.2. Binge Eating Scale.** This measure is the same as the measure used in Study 1. In this study ( $N = 115$ ), scores ranged from 0-32.00 ( $M = 10.81$ ,  $SD = 7.57$ ) and internal reliability was good with a Cronbach's alpha of .90.

**3.3.3.3. Positive and Negative Affect Schedule - Expanded Form.** Negative affect is often associated with binge eating behavior, and studies have shown that individuals can experience increased negative affect both before and after a binge eating episode. The original PANAS measure is designed to examine the severity of various kinds of positive and negative affect over the past week (Watson et al., 1988), and included basic forms of negative affect such as upset, guilty, and anxious. The extended form (PANAS-X) includes a number of additional facets of affect in order to assess a wider range, including self-focused negative affect (i.e., disgusted at self; Watson & Clark, 1999). This is more relevant to those with BED given their

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

body-focused affect. For the purposes of part 2, this form was completed at home and after the challenging tasks in order to assess changes in affect. This information will allow us to examine global negative affect and how this relates to binge eating, as well as key facets involved with binge eating behavior. The negative affect scale has evidenced good internal reliability, ranging from .85-.93 based on the population assessed. In this sample ( $N = 113$ ), a negative affect scale was created by computing a sum score of negative affect items to reflect severity, which ranged from 10.00-38.00 ( $M = 21.68$ ,  $SD = 6.77$ ), and Cronbach's alpha was .852.

**3.3.3.4. UPPS-P Impulsive Behavior Scale (revised version).** This measure is the same as the measure used in Study 1. In this study ( $N = 114$ ), scores ranged from 1.14-3.14 ( $M = 2.25$ ,  $SD = .42$ ) and internal reliability was good with a Cronbach's alpha of .95.

**3.3.3.5. Perceived Stress Scale.** This measure is the same as the measure used in Study 1. In this study ( $N = 119$ ), scores ranged from 1.10-4.90 ( $M = 3.06$ ,  $SD = .68$ ) and internal reliability was good with a Cronbach's alpha of .842.

**3.3.3.6. Food Frequency Questionnaire.** Participants were asked to indicate the frequency with which they eat a variety of foods using a measure adapted from the Block Food Frequency Questionnaire, a self-report measure used to assess food intake among adults (Boucher et. al., 2006). Reliability of different versions has been shown to be adequate, with Pearson correlations coefficients ranging from .57 to .90. Sample items include “cakes, cookies and brownies” and “sausage, burgers, pizza, hot dogs.” In study 1, a mean score for unhealthy foods was computed which ranged from 1.00-5.17 ( $M = 2.47$ ,  $SD = .61$ ), and Cronbach's alpha was .760 ( $N = 784$ ). A separate mean score for fast food items was computed and ranged from 1.00-5.50 ( $M = 2.49$ ,  $SD = .88$ ); Cronbach's alpha was .660 ( $N = 799$ ). This measure was modified for the survey given after completion of the tasks (post-task questionnaire; See

Appendix I); this survey asked participants to indicate the extent to which they wish to eat the listed foods “right now.”

**3.3.3.7. General Food Cravings Questionnaire-State.** The G-FCQ-S was designed to assess general food-related cravings and urges occurring in the moment (Nijs, Franken, & Muris, 2007). This was included in the post-task questionnaire to provide information about how the tasks impact eating and drive to eat. Because we are not excluding participants based on diagnostic criteria or BMI, this measure of drive to eat was chosen instead of a more realistic test-food intake paradigm in order to avoid inducing a stress-related binge episode in someone who has BED, as this could cause the participant harm. Food cravings measured with the original version of this measure have been shown to predict overeating of specific foods during ad libitum eating tasks (Martin, O’Neil, Tollefson, Greenway, & White, 2008). Further, general food cravings measured with the G-FCQ-S were shown to correspond with hunger and satiety suggesting that the construct measured with this instrument reflects an urge to eat that is connected with physiological hunger cues (Nijs et al., 2007). Thus, this measure appears to be an intriguing proxy for drive to eat that does not require risking participant’s psychological harm by encouraging harmful behaviors. Previous studies have shown good internal reliability findings (Nijs et. al., 2007), evidencing a Cronbach’s alpha of .93. Sample items include “I am craving tasty food” and “If I had something tasty to eat, I could not stop eating it.” This measure was modified for the survey given after completion of the tasks (See Appendix I); this survey asked participants to indicate the extent to which they felt this way “right now.”

**3.3.3.8. Mood and Anxiety Symptom Questionnaire-Short Form.** The MASQ-SF was designed to assess symptoms related to a range of anxiety and mood disorders over the past week (Watson et. al., 1995). This scale provides subscales that indicate general distress related to both

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

anxious and depressed symptoms, as well as subscales that more specifically represent arousal and anhedonia. This will allow us to further examine the complex relationship between binge eating severity and anxiety/mood-related problems, as both are thought to predict the other. Studies have evidenced good internal consistency reliability, with a Cronbach's alpha of .94 (Watson et. al., 1995; Yang et. al., 2009). Sample items include "blamed myself for a lot of things," "felt withdrawn from other people," and "felt like a failure."

**3.3.3.9. Demographics.** We asked participants to respond to demographic items (race, gender, age).

**3.3.3.10. Stop signal cognitive task.** Participants completed Gordon Logan's classic stop-signal task, which measures the ability to inhibit motor and cognitive responses (Logan et. al., 1984). This widely-used inhibitory control task has been frequently used as a measure of impulsivity in BED populations, as poorer performance indicates poorer response inhibition, or higher impulsivity. The stop-signal model is based on an independent horse-race model and can be described as a race between a go process, initiated by a go stimulus (i.e., white arrows), and a stop process, triggered by the stop signal (i.e., blue arrows; Verbruggen, Chambers and Logan, 2013). Participants were presented with different colored arrows on a computer screen and asked to indicate the direction by pressing analogous arrow-keys on a standard keyboard when white arrows are present. There are 2 phases: a practice phase with 32 trials and an experimental phase with three blocks of 64 trials each. There are 15 seconds between blocks, during which participants receive information about their performance for both signal and no-signal trials in the previous blocks. Each trial starts with the presentation of a fixation sign (white dot in center of screen), which is replaced with a task stimulus after 250 msec. The stimulus remains on the screen until the participant responds, or until 1.5 seconds have elapsed. After the participant

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responds during the practice phase, the word “correct” or “incorrect” appears on the screen to provide feedback for each trial. For experimental blocks, the fixation sign reappears on the screen after a response is given to each trial. The default response-to-stimulus interval (RSI) is independent of reaction time and is set to 500 msec for all trials. An HP ProDesk 600 G2 SFF computer and Matlab R2017a/Psychtoolbox software were used to run the task, while the stimuli were displayed on a 20-inch Dell monitor (Model No. 2007FPb).

On some trials, a stop signal is presented--the arrow turns blue after a variable latency period (stop signal delay; SSD), at which point the participants are required to inhibit their response; participants are instructed not to respond to the blue arrows. The SSD is initially set at 250 msec and is continuously adjusted based on the staircase-tracking procedure: SSD is increased by 50 msec when inhibition is successful and decreased by 50 msec when inhibition is unsuccessful.

At the start of the task, instructions appear on the screen. Participants are informed that their main task is to respond to white arrows to indicate direction with the arrow keys. They are told that some arrows will turn blue, and this indicates that they should withhold their response; the instructions specify that sometimes it will turn early and be easy to withhold responses and sometimes it will turn late and be more difficult. They are instructed not to wait until the arrow turns blue, and to respond as quickly and accurately as possible. If they wait for blue arrow, instructions state that the program adjusts and delays the stop signal—this leads to longer reaction times.

Performance is often measured by examining the participants stop-signal reaction time (SSRT), which reflects the average time (in ms) that an individual requires in order to

successfully inhibit a motor response approximately 50% of the time. Using the integration method for estimating, this is calculated by subtracting the average stop signal delay (SSD), or the variable time delay between go and stop stimuli, from the  $n$ th reaction time, or the estimate of the time at which the stopping process is finished relative to the onset of the go signal. Thus, SSRT is the time between presentation of the stop signal and completion of the process, which is estimated based on the reaction time (RT) of go-trials (i.e., arrow stays white) and the probability of a response given a signal to determine when inhibiting the response should be completed. As such, SSRT is thought to reflect impulsivity as it represents the process of inhibiting a motoric response in response to external stimuli. Thus, SSRT is higher when impulsivity is higher. In this study ( $N=105$ ), scores ranged from 1.51-3.08 ( $M=2.34$ ,  $SD=.21$ ).

**3.3.3.11. Self Control Scale.** Participants completed a 36-item measure of general self-control, designed to assess behaviors related to the self's capacity to inhibit its antisocial impulses and conform to the norms of group life, including control over thoughts, emotional control, impulse control, performance regulation and habit-breaking (Tangney, Baumeister, & Boone, 2018). This measure allows investigators to obtain an index for restraint that is more generalizable and less reliant on dietary restraint. This questionnaire has evidenced adequate internal reliability, with a Cronbach's alpha of .89. Sample items include "I say inappropriate things," "I spend too much money" and "I lose my temper too easily." In this study, scores ranged from 59-179 ( $M=113.09$ ,  $SD=20.76$ ) and internal reliability was good with a Cronbach's alpha of .92.

**3.3.3.12. Task appraisal.** Participants were asked questions about their appraisal of the demands of the task with an 11-item measure designed by the McCoy lab (See Appendices H and I). Sample items include "The task took a lot of effort to complete" and "The task was very



stressful.” Answers are made on a 0 to 6 Likert-type scale, ranging from Strongly Disagree (0) to Strongly Agree (6). For the purposes of this study, this measure was also modified to ask about appraisals before the task has been completed (e.g., “The task will be very demanding”). A threat-appraisal subscale will be created to assess the degree to which participants found the task threatening, using 4 items that ask about the extent to which they found it: stressful, effortful, distressing, and demanding (Ell, Cosley, & McCoy, 2011). This scale has been shown to have adequate reliability in prior studies, with a Cronbach’s alpha of .87. Post-task threat appraisal scores in this study ( $N=109$ ) ranged from 0.00-5.25 ( $M=2.46$ ,  $SD=1.54$ ) and internal reliability was good with a Cronbach's alpha of .822.

**3.3.3.13. Body measurements.** Participants were assessed for height (inches) and weight (lbs) with a fixed steel tape and an analog scale by a female graduate student at the completion of all experimental tasks. Waist circumference was measured in centimeters at the umbilicus level and hip measurements were measured in centimeters around the widest part of the buttocks. Abdominal sagittal diameter was measured in centimeters using a sagittometer.

**3.3.3.14. Psychophysiological recording.** Noninvasive disposable sensors were placed on the participant to measure electrical activity of the heart (i.e. electrocardiography (ECG) and impedance cardiography [ICG]). Participants were seated comfortably in a small recording room. The sensors were detached following the recovery period. This required psychophysiological equipment which the McCoy lab had already obtained. The lab also has acquisition hardware (Biopac-MP150) and Biopac software for data collection/display. Biopac collected the cardiovascular and BP signals and were cleaned for artifact and ensemble averaged over baseline (10 minutes), tasks, and recovery (5 minutes) periods using Mindware.

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**3.3.3.14.1. ECG.** Electrocardiography measures electrical impulses in the cardiovascular system through electrodes placed on each forearm and the left calf (Berntson et al., 2007). ECG is used to derive heart period (HP), which is the time between beats of the heart, and this is typically converted into heart rate (HR), which is expressed in beats of the heart per minute. Equipment obtained for this measure includes an electrocardiograph module (Biopac-NICO100C), disposable electrodes, and electrical leads to collect ECG data.

**3.3.3.14.2. ICG.** Impedance cardiography measures resistivity in the thoracic cavity by conducting electrical impulses between inner and outer electrode bands and measuring the resistance of electrical flow based on Ohm's law (law regarding flow of energy; Berntson et al., 2007). Thus, when the aorta is filled with blood, the signal would travel faster through the thoracic cavity so we can use impedance to determine when blood is leaving the heart. In the current study, impedance data is used to provide an estimation of respiration, allowing this to be accounted for when calculating HF-HRV. For the purposes of this study, HF-HRV and HRV will be used interchangeably to refer to HF-HRV, as this is the only frequency band we are interested in. A Biopac module (ECG100C) allows us to collect impedance data as well, using additional alligator leads and special conductive impedance tape.

**3.3.3.14.3. BP.** Blood pressure, or the pressure exerted on the walls of blood vessels in the body, can be measured continuously using a wrist or finger cuff that repeatedly applies modest pressure to the artery over bone, sensing the largest pulse wave amplitude (Berntson et al., 2007). This allows BP to be collected over an extended period of time after an initial calibration period (using a non-continuous blood pressure arm band) and allows changes in BP over time to be examined. MAP is calculated by finding one-third of the sum of systolic and diastolic pressure, where diastolic pressure is multiplied by 2 given the larger amount of time

that the body is in diastole. Higher MAP during recovery is used here to index a more maladaptive stress (threat) response (Connell et. al., 1987). The McCoy lab also has a noninvasive blood pressure monitor (CNAP Monitor 500/Biopac), and three sizes of finger cuffs (small, medium and large) to collect blood pressure data.

### **3.4. Online Survey Results: Replication of Study 1 (Part 1)**

#### **3.4.1. Statistical Analyses**

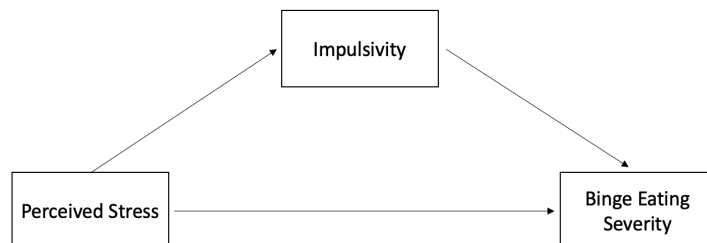
IBM SPSS V26 software was used to complete all statistical analyses (IBM Corp., 2019). All scales were computed and univariate (univariate outliers were windzorised; skewness and kurtosis values with Z score >1.96 were considered significant) and multivariate assumptions (multicollinearity:  $r$ 's > |.80| among variables and tolerance < .10, VIF > 10; multivariate outliers: studentized deleted residuals > +/- 2 and leverage >  $(2k+2)/n$ ,  $dfbeta > +/- 2/\sqrt{[n]}$ ) were investigated. Variables with significant skewness were appropriately transformed (log transformation for substantial positive skew; Tabachnick & Fidell, 2007) and models were tested on the transformed data. As the hypothesized effects differed with the transformed variable (BES), we present results below utilizing the transformed data. One participant was identified as a multivariate outlier with undue influence and was excluded from analyses.

Descriptive statistics were obtained for each proposed variable and relationships among these factors were investigated through correlational analyses (See Table 2). Due to the high correlation between restraint and impulsivity, the proposed model from Study 1 (Figure 5) could not be replicated as these two variables may not represent unique constructs. Additionally, the reduced number of participants compared to Study 1 also supports a more parsimonious model. Andrew F. Hayes' (2017) PROCESS mediation program was used in SPSS to investigate the strongest indirect effect from Study 1 (Stress → Impulsivity → Binge Eating), which was

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significant for both men and women. This regression-based analysis tests the direct pathway between a proposed predictor variable (perceived stress) and outcome variable (binge eating severity) as well as the indirect pathways that occur through mediator variables (impulsivity). Direct pathways between all variables are analyzed in order to better understand the impact of each factor on the others. Significant effects are identified by bootstrap estimation (based on 5,000 samples) of the 95% bias corrected confidence interval that does not contain 0. Thus, the statistical model investigated in this study used regression-based analyses to examine the direct and indirect effects of stress on binge eating for women (See Figure 14). The effect size of the proposed model is determined by obtaining  $R^2$ , or the measure of the proportion of the variance of binge eating severity accounted for by the model.

Figure 14. Mediation Model Tested for Part 1 of Study 2



### 3.4.2. Part 1 Correlations and Descriptives

Descriptive statistics revealed that this population endorsed similar levels of each construct compared to the population in Study 1, as similar means were observed across all variables (See Table 2). The average untransformed Binge Eating Scale score was 11.00, which falls well below the range of subthreshold binge eating severity (less than or equal to 17), suggesting that the overall sample represents minimal binge eating behavior; this is consistent

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with Study 1. Further, in this study the range of binge eating severity was 0 - 32.00. Only 7 people (6.1% of binge eating scores) had scores that indicated severe binge eating. Further, there were 12 participants (10.6% of binge eating scores) with subthreshold binge eating (scores of 18-26). The correlational analyses conducted in Study 1 were replicated to determine whether similar relationships exist between the variables in this sample of women. All correlations were significant at the  $<.01$  level (See Table 2), including the relationship between cognitive restraint and impulsivity, which was uncorrelated for women in study 1. This suggests that the new, non-food specific restraint measure is more closely related to the hypothesized variables. In fact, the correlation between restraint and impulsivity was  $-.79$ , suggesting that the constructs measured by each may not be distinct from each other and testing only one of these variables may be more meaningful. As discussed above, the two were not tested in the same model for this reason.

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Table 2. Online Study Descriptive Statistics and Correlations

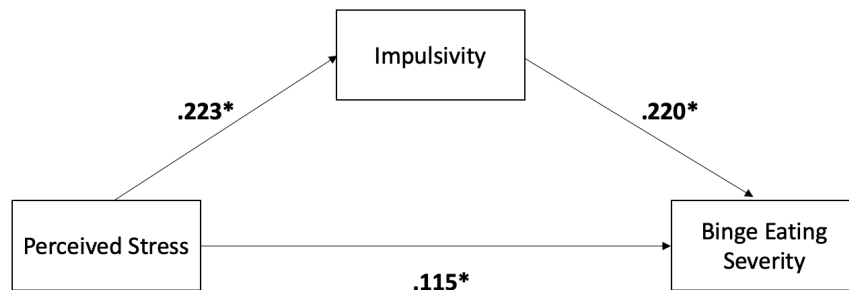
<b>Correlations</b>	1.	2.	3.	4.	5.
1. Perceived Stress	---	.63	-.37	.31	.31
2. Negative Affect	---	---	-.34	.32	.51
3. Restraint	---	---	---	-.79	-.46
4. Impulsivity	---	---	---	---	.32
5. Binge Eating Severity	---	---	---	---	---
<b>Descriptives M(SD)</b>	3.06(.68)	21.68(6.77)	113.01 (20.51)	2.25(.42)	10.81(7.57)

*Note: All correlations above are significant at the <.01 level. Pearson  $r$  linear correlations for women ( $N = 83$ ). Means, followed by standard deviations within parentheses, are presented for each dependent variable at the bottom of the table.*

**3.4.3. Part 1 Mediation Models**

PROCESS Mediation analyses were conducted using the simple mediation model (Model 4; Hayes, 2017) in Figure 14, which represents the strongest pathway demonstrated in Study 1 for both men and women. The overall model accounted for 11.49% of the variance ( $R^2$ ) in binge eating symptomatology and all direct effects were significant. The main direct effect of stress on binge eating was significant ( $b = .115$ ; 95% BCa CI [.015, .216]) such that greater stress predicted higher binge eating severity. Greater stress also predicted higher levels of impulsivity ( $b = .223$ ; 95% BCa CI [.099, .348]), while greater impulsivity predicted higher binge eating severity ( $b = .220$ ; 95% BCa CI [.067, .372]; See Figure 15)

Figure 15. Direct Effects of Hypothesized Model



Finally, we examine the hypothesized indirect effects. Does perceived stress impact binge eating severity through impulsivity? Of note, the test of the indirect effect examines the significance of the combined pathway from stress to binge eating (e.g. stress → impulsivity → binge eating severity). This indirect effect was significant suggesting that greater perceived stress does indeed increase binge eating severity through increased impulsivity ( $b=.049$ , 95% BCa CI [.006, .101]).

### 3.5. Laboratory Study Results (Part 2)

#### 3.5.1. Statistical Analyses

IBM SPSS V26 software was used to complete all statistical analyses (IBM Corp., 2019). All scales were computed and univariate (univariate outliers were windzorisied; skewness and kurtosis values with  $Z$  score  $> 1.96$  were considered significant) and multivariate assumptions (multicollinearity:  $r$ 's  $> |.80|$  among variables and tolerance  $< .10$ , VIF  $> 10$ ; multivariate outliers: studentized deleted residuals  $> +/- 2$  and leverage  $> (2k+2)/n$ ,  $dfbeta > +/- 2/\sqrt{n}$ ) were investigated. Variables with moderate skewness were appropriately transformed (log transformation for substantial positive skew; Tabachnick & Fidell, 2007) and models were tested on the transformed data. Two participants were identified as multivariate outliers with undue influence; one was excluded from analyses for one of the SSRT models (stress variable=MAP recovery; outcome=SSRT) and the second was excluded from all analyses involving negative affect.

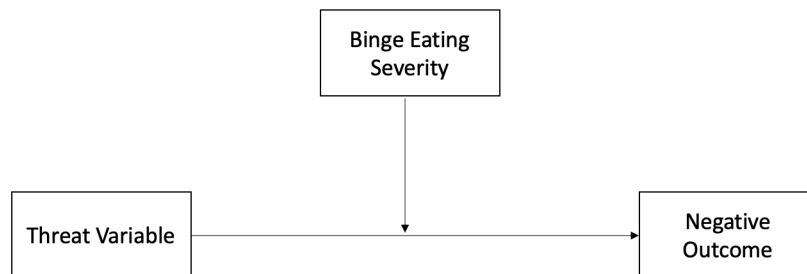
Descriptive statistics were obtained for each proposed variable and relationships among these factors were investigated through correlational analyses (See Table 3). Andrew F. Hayes' PROCESS moderated regression program was used in SPSS to analyze the proposed models (Figure 16; Hayes, 2017; IBM Corp., 2016). This regression-based analysis tests direct and pathways as well as interactions between predictor variables that may account for variance in the outcome variable. Direct pathways between all variables are analyzed in order to better understand the impact of each factor on the others. Significant effects are identified by bootstrap estimation (based on 5,000 samples) of the 95% bias corrected confidence interval that does not contain 0. Thus, the statistical model investigated in this study used regression-based analyses to examine the direct and combined effects of stress and binge eating on negative outcomes for



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women (See Figure 16). The effect size of the proposed model is determined by obtaining  $R^2$ , or the measure of the proportion of the variance of binge eating severity accounted for by the model. Physiological reactivity variables for MAP and HF-HRV were calculated by subtracting the mean baseline variable (averaged over the last 5 minutes of baseline) from the mean task variable. Because HRV and MAP habituate quickly to stressors (Kelsey, Blascovich, Leitten, Schneider, Tonaka, & Wiens, 2000), the speech task was averaged over only minutes 2 and 3. Recovery was computed by subtracting the baseline variable from the mean recovery (averaged over the last 4 minutes of recovery) variable.

Figure 16. Proposed Moderation Model for Laboratory Study



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3.5.2. Part 2 Correlations and Descriptives

Table 3. Laboratory Study Descriptive Statistics and Correlations

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. BMI	-	.02	-.04	-.12	.01	<b>-.24*</b>	.07	.01	-.01	<b>.25*</b>	-.10	-.01	-.14	<b>-.26*</b>	-.02
2. BES	-	-	<b>.41*</b>	<b>.35*</b>	.03	.19	.12	-.11	.02	.05	-.12	-.06	.07	<b>.22*</b>	<b>.46*</b>
3. Stress	-	-	-	<b>.29*</b>	.11	.04	.06	-.07	-.03	-.20	-.18	-.12	-.11	.09	<b>.51*</b>
4. Impulsivity	-	-	-	-	.19	.17	-.01	.10	.14	-.03	<b>-.23*</b>	-.06	.18	.15	<b>.36*</b>
5. Pre-TA	-	-	-	-	-	<b>.21*</b>	.05	-.02	-.00	-.06	.03	.03	.10	.03	.14
6. Post-TA	-	-	-	-	-	-	-.00	-.07	.04	-.05	.05	-.10	.10	.12	<b>.20*</b>
7. HRV Baseline	-	-	-	-	-	-	-	<b>-.54*</b>	<b>-.33*</b>	<b>-.29*</b>	.11	-.02	-.00	-.02	.10
8. HRV Reactivity	-	-	-	-	-	-	-	-	<b>.34*</b>	.03	-.18	.03	.12	.10	-.11
9. HRV Recovery	-	-	-	-	-	-	-	-	-	.17	<b>-.21*</b>	-.10	.02	-.02	.07
10. MAP Baseline	-	-	-	-	-	-	-	-	-	-	<b>-.36*</b>	<b>-.38*</b>	.04	-.12	-.07
11. MAP Reactivity	-	-	-	-	-	-	-	-	-	-	-	<b>.61*</b>	-.13	.04	-.05
12. MAP Recovery	-	-	-	-	-	-	-	-	-	-	-	-	.10	.03	-.02
13. SSRT	-	-	-	-	-	-	-	-	-	-	-	-	-	.17	-.06
14. Drive to Eat	-	-	-	-	-	-	-	-	-	-	-	-	-	-	<b>.21*</b>
15. Post-task NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

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<i>M(SD)</i>	23.75 (5.42)	<b>.94</b> (.33)	3.08 (.69)	2.25 (.42)	1.96 (1.16)	2.46 (1.54)	6.72 (1.15)	<b>-.38</b> (1.06)	<b>.06</b> (.60)	84.47 (10.02)	13.94 (13.73)	1.75 (10.23)	2.34 (.21)	29.21 (18.36)	18.24 (6.40)
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*Note:* BES and SSRT variables are log-transformed and their descriptive statistics reflects the log-transformed values. All **bolded\*** correlations are significant at the  $p < .05$  level. NA=Negative Affect; TA=Task Appraisal; BMI=Body Mass Index; BES=Binge Eating Scale; HRV=Heart Rate Variability; MAP=Mean Arterial Pressure; SSRT=Stop Signal Response Time

Table 3 Continued.

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Pearson's  $r$  correlations and descriptive statistics can be found in Table 3. BMI ranged from 15.06 - 41.20 ( $M = 23.75$ ,  $SD = 5.42$ ), indicating that the average score for this sample falls in the healthy weight range. Correlational analyses were conducted to examine the relationships among the hypothesized variables. The online survey measures were discussed in part 1, so here we review the correlations for the remainder of the variables. Notably, SSRT did not correlate with any variables. Negative affect measured after the challenging tasks correlated positively with impulsivity, perceived stress, binge eating severity, and both drive to eat and threat appraisal measured after the tasks. Higher drive to eat was associated with higher binge eating severity, while surprisingly, higher BMI was associated with lower drive to eat. All MAP variables were correlated with each other, as were HRV variables. Higher post-task threat appraisal was associated with lower BMI and higher pre-task threat appraisal. Baseline MAP was negatively correlated with baseline HRV and positively correlated with BMI. Higher trait impulsivity was associated with lower MAP reactivity, while lower MAP reactivity was associated with higher HRV recovery. Surprisingly, binge eating did not correlate with HRV reactivity or MAP recovery as predicted, nor did impulsivity or even perceived stress. It also is worth noting that the post-task threat appraisal measure did not correlate with the maladaptive stress response variables (HRV reactivity and MAP recovery) either. Unlike part 1, none of the variables here appear to share too much variance, so all can be included in further analysis.

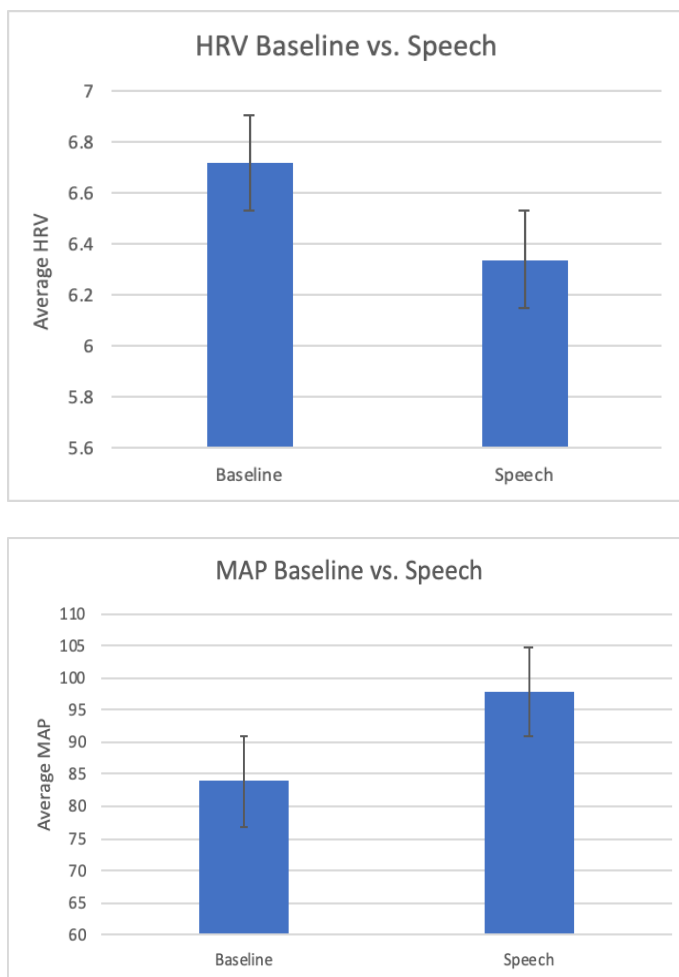
### **3.5.3. Were Participants Actually Stressed?**

Overall, the post-task appraisal showed an average threat appraisal of 2.46 ( $SD = 1.54$ ), which is slightly below the midpoint of 3.00. Interestingly, the average appraisal of threat collected after the stressful tasks was significantly higher than the appraisal collected before the tasks ( $t [108] = -2.90$ ,  $p < .01$ ), suggesting that the women in this study found the task more

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threatening to their resources to cope than they expected. Consistent with intentions, women had a significant physiological reaction to the stressor. Paired t-tests were conducted between the average baseline physiological variables (HRV and MAP) and those same variables during the speech task. Mean baseline HRV was significantly higher than mean HRV during the speech task ( $t [111] = 3.82, p < .001$ , Cohen's  $d = .36$ ; See Figure 17). Mean MAP was significantly higher during the speech task than mean MAP during baseline ( $t [95] = -9.95, p < .001$ , Cohen's  $d = 1.02$ ; Figure 17). Thus, women did evidence significant stress reactivity.

Figure 17. Comparison of Mean HRV and MAP During Baseline and Speech Task



### 3.5.4. Moderated Regression Analyses

Moderated regression models were tested to determine whether self-report and cardiovascular measures indicative of threat stress reactivity interact with binge eating severity to predict impulsivity, negative affect or drive to eat. For example, it is expected that for those higher in binge eating, lower HF-HRV will be associated with higher impulsivity more strongly than those low in binge eating. This is determined by examining coefficients (*b*; slope) of the interaction between binge eating and threat. As discussed previously, threat stress appraisal can be represented by lower HF-HRV reactivity and higher MAP recovery. Here we also examine HF-HRV recovery and MAP reactivity, as well as self-report post-task threat appraisal scores, as possible indicators of a maladaptive stress response. Three post-stressor outcome variables related to binge eating were tested in Part 2: impulsivity, negative affect, and drive to eat. Impulsivity was measured during the inhibitory control task (SSRT). In the post-task survey, negative affect was measured using the PANAS-X while drive to eat/food cravings was measured with the G-FCQ-S. Potential covariates (BMI, physiological baseline, UPPS for SSRT models) were tested individually as predictors of each outcome variable and included as covariates in the regression only if they significantly predicted the outcome variable (post-task NA, SSRT, drive to eat).

Although the proposed analyses focus on the moderation of the stress-impulsivity relationship by BES, it is possible that a mediation model is a better fit to the data. For example, BES may be associated with greater impulsivity due to increased stress reactivity (i.e. BES → Stress Reactivity → Impulsivity). These models were also tested in PROCESS with each of the three outcome variables (drive to eat, post-task NA, SSRT) and each of the 5 stress response variables (MAP recovery/reactivity, HRV recovery/reactivity, post-task threat appraisal).

**3.5.4.1. Did binge interact with stress to predict drive to eat?** We first examine the impact of stress response and binge eating on drive to eat (dependent variable). Moderated regressions were conducted to examine whether there is a significant interaction between binge eating severity (moderator) and stress, such that those higher in binge eating experience greater drive to eat in response to a more maladaptive stress response. BMI was a significant predictor of drive to eat independently, and as such was included as a covariate for each drive to eat model (See Table 4). Across all models, and consistent with predictions, binge eating significantly predicted drive to eat in this stressful context (See Table 4). Neither stress response, nor the interaction between stress response and binge eating severity, were significant for any of the models (See table 4).

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Table 4. Impact of Binge on Relationship Between Stress Response and Drive to Eat

Predictors of Drive to Eat	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			<b>.18*</b>
BMI	<b>-62.54*</b>	<b>[-100.68, -24.39]</b>	
Binge	<b>12.60*</b>	<b>[2.44, 22.76]</b>	
HRV reactivity	1.48	[-1.82, 4.78]	
Binge X HRV reactivity	-4.78	[-13.80, 4.25]	
<u>HRV RECOVERY MODEL</u>			<b>.17*</b>
BMI	<b>-69.44*</b>	<b>[-108.19, -30.69]</b>	
Binge	<b>11.99*</b>	<b>[1.78, 22.21]</b>	
HRV recovery	.54	[-5.59, 6.67]	
Binge X HRV recovery	-2.94	[-16.53, 10.65]	
<u>MAP REACTIVITY MODEL</u>			<b>.15*</b>
BMI	<b>-55.91*</b>	<b>[-97.69, -14.14]</b>	
Binge	<b>12.86*</b>	<b>[1.97, 23.74]</b>	
MAP reactivity	.03	[-.28, .33]	
Binge X MAP reactivity	.25	[-.91, 1.42]	
<u>MAP RECOVERY MODEL</u>			<b>.18*</b>
BMI	<b>-61.58*</b>	<b>[-102.23, -20.93]</b>	
Binge	<b>12.21*</b>	<b>[1.96, 22.45]</b>	
MAP recovery	.01	[-.37, .39]	
Binge X MAP recovery	-.81	[-1.89, .28]	
<u>POST APPRAISAL MODEL</u>			<b>.20*</b>
BMI	<b>-63.16*</b>	<b>[-102.00, -24.31]</b>	
Binge	<b>13.05*</b>	<b>[2.75, 23.35]</b>	
Post-task appraisal	.05	[-2.33, 2.43]	
Binge X Post-task appraisal	6.20	[-.06, 12.47]	

Note: **Bolded\*** values indicate significance at the p<.05 level.



**3.5.4.2. Did binge interact with stress to predict SSRT?** Next we consider whether these relationships might be present with regard to impulsivity, as measured by the cognitive task just after the stressor, as the outcome variable. Here we observe no significant direct effects, nor interactions, for any of the variables.

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Table 5. Impact of Binge on Relationship Between Stress Response and SSRT

Predictors of SSRT	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			.02
Binge	.05	[-.07, .17]	
HRV reactivity	.02	[-.02, .06]	
Binge X HRV reactivity	.00	[-.10, .10]	
<u>HRV RECOVERY MODEL</u>			.01
Binge	.04	[-.08, .16]	
HRV recovery	.02	[-.05, .10]	
Binge X HRV recovery	.04	[-.12, .20]	
<u>MAP REACTIVITY MODEL</u>			.02
Binge	.00	[-.11, .12]	
MAP reactivity	-.00	[-.01, .00]	
Binge X MAP reactivity	-.00	[-.01, .01]	
<u>MAP RECOVERY MODEL</u>			.03
Binge	.07	[-.05, .20]	
MAP recovery	.00	[-.00, .01]	
Binge X MAP recovery	.00	[-.01, .02]	
<u>POST APPRAISAL MODEL</u>			.03
Binge	.04	[-.09, .16]	
Post-task appraisal	.01	[-.02, .04]	
Binge X Post-task appraisal	.04	[-.03, .12]	

Note: **Bolded\*** values indicate significance at the  $p < .05$  level.

**3.5.4.3. Did binge interact with stress to predict negative affect?** Lastly, we explore whether binge eating moderates the relationship between stress response and negative affect measured after the challenging tasks. No covariates were significant predictors of negative affect.

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Table 6. Impact of Binge on Relationship Between Stress Response and Negative Affect

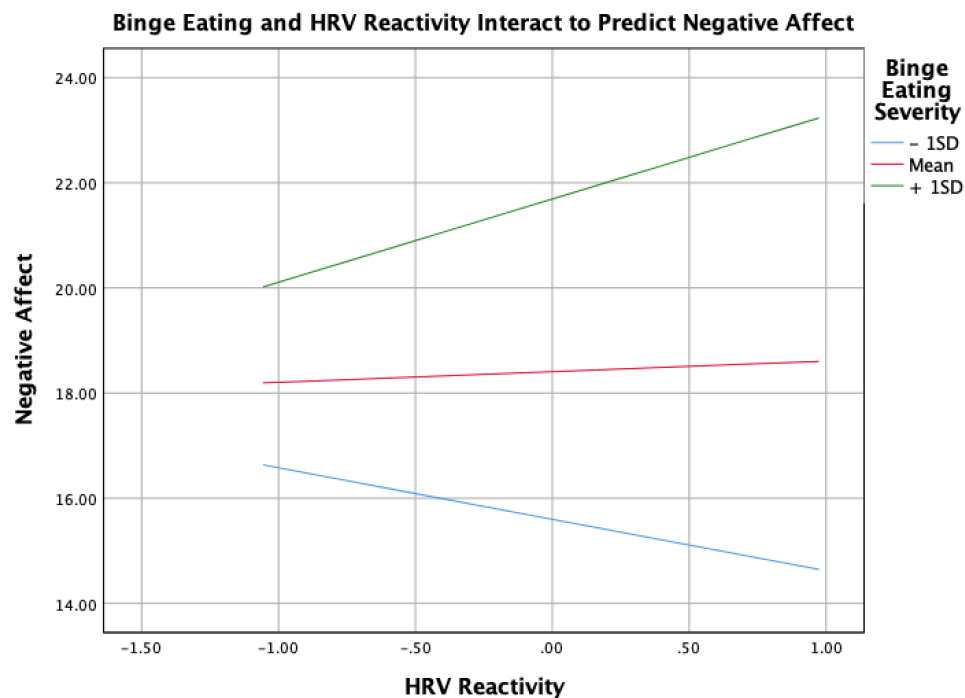
Predictors of Negative Affect	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			<b>.31*</b>
Binge	<b>9.33*</b>	<b>[5.74, 12.91]</b>	
HRV reactivity	.28	[-.80, 1.36]	
Binge X HRV reactivity	<b>3.92*</b>	<b> [.97, 6.88]</b>	
<u>HRV RECOVERY MODEL</u>			<b>.26*</b>
Binge	<b>9.54*</b>	<b>[5.84, 13.25]</b>	
HRV recovery	.61	[-1.47, 2.70]	
Binge X HRV recovery	2.79	[-1.94, 7.52]	
<u>MAP REACTIVITY MODEL</u>			<b>.29*</b>
Binge	<b>10.15*</b>	<b>[6.22, 14.08]</b>	
MAP reactivity	-.03	[-.13, .07]	
Binge X MAP reactivity	-.04	[-.43, .36]	
<u>MAP RECOVERY MODEL</u>			<b>.27*</b>
Binge	<b>9.69*</b>	<b>[5.90, 13.48]</b>	
MAP recovery	-.02	[-.15, .11]	
Binge X MAP recovery	.14	[-.27, .55]	
<u>POST APPRAISAL MODEL</u>			<b>.25*</b>
Binge	<b>9.27*</b>	<b>[5.39, 13.15]</b>	
Post-task appraisal	.14	[-.68, .96]	
Binge X Post-task appraisal	-.33	[-2.59, 1.93]	

Note: **Bolded\*** values indicate significance at the p<.05 level.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

For the model that examined HF-HRV reactivity as the stress variable, a significant interaction was observed (See Table 6). Although not significant, women lower in BES tended to report greater negative affect at lower levels of HRV ( $b = -.98$ ). Yet as binge eating increased from mean levels (at which there was no trend;  $b = .20$ ) to higher levels, this relationship tended to reverse. Intriguingly, among women who were higher in BES, decreased HRV (i.e., maladaptive threat response) was associated with reporting less negative affect ( $b = 1.58$ , 95% BCa CI [.02, 3.14]; Figure 18) and this model accounted for 31% of the variance in negative affect.

Figure 18. Binge Eating and HRV Reactivity Interact to Predict Negative Affect



### **3.5.5. Mediation Analyses**

Given that there was only one significant interaction for the moderation models tested, we consider whether a mediation effect may better represent the relationships between these variables. Specifically, we examine whether higher binge eating predicts increased drive to eat, impulsivity, or negative affect through more maladaptive stress. As none of the indirect effects were significant, we have no evidence to support that maladaptive stress is the mechanism or process that explains the outcome variables. See Tables 7, 8, and 9 for model details on these analyses.

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Table 7. Impact of Binge on Drive to Eat Through Stress Response

Pathways Tested	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			<b>.17*</b>
BMI → HRV reactivity	.06	[-2.37, 2.50]	
Binge → HRV reactivity	-.33	[-.98, .33]	
HRV reactivity → Eat	1.77	[-1.48, 5.03]	
BMI → Eat	<b>-65.67*</b>	<b>[-103.37, -27.97]</b>	
Binge → Eat	<b>12.51*</b>	<b>[2.35, 22.68]</b>	
Binge → HRV reac → Eat	-.58	[-2.86, 1.13]	
<u>HRV RECOVERY MODEL</u>			<b>.17*</b>
BMI → HRV recovery	.17	[-1.21, 1.56]	
Binge → HRV recovery	.01	[-.35, .38]	
HRV recovery → Eat	.88	[-5.02, 6.77]	
BMI → Eat	<b>-69.53*</b>	<b>[-108.09, -30.97]</b>	
Binge → Eat	<b>11.99*</b>	<b>[1.82, 22.15]</b>	
Binge → HRV recov → Eat	.01	[-1.59, 1.43]	
<u>MAP REACTIVITY MODEL</u>			<b>.15*</b>
BMI → MAP reactivity	-5.34	[-38.24, 27.56]	
Binge → MAP reactivity	-4.95	[-13.46, 3.56]	
MAP reactivity → Eat	.05	[-.23, .33]	
BMI → Eat	<b>-57.26*</b>	<b>[-98.34, -16.17]</b>	
Binge → Eat	<b>12.51*</b>	<b>[1.80, 23.21]</b>	
Binge → MAP reac → Eat	-.25	[-2.23, 1.40]	
<u>MAP RECOVERY MODEL</u>			<b>.15*</b>
BMI → MAP recovery	-2.05	[-25.77, 21.67]	
Binge → MAP recovery	-1.75	[-7.71, 4.22]	
MAP recovery → Eat	.00	[-.38, .38]	
BMI → Eat	<b>-60.33*</b>	<b>[-101.24, -19.42]</b>	
Binge → Eat	<b>12.65*</b>	<b>[2.35, 22.95]</b>	

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Binge → MAP recov → Eat	-.00	[-1.23, 1.50]	
<u>POST APPRAISAL MODEL</u>			<b>.16*</b>
BMI → Post	<b>-4.43*</b>	<b>[-7.73, -1.13]</b>	
Binge → Post	.86	[-.02, 1.75]	
Post → Eat	.15	[-2.27, 2.57]	
BMI → Eat	<b>-65.87*</b>	<b>[-104.30, -25.44]</b>	
Binge → Eat	<b>11.80*</b>	<b>[1.41, 22.18]</b>	
Binge → Post → Eat	.13	[-2.49, 2.55]	

Note: **Bolded\*** values indicate significance at the  $p < .05$  level.

Table 7. Continued



A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Table 8. Impact of Binge on SSRT Through Stress Response

Pathways Tested	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			.02
Binge → HRV reactivity	-.36	[-1.01, .29]	
HRV reactivity → SSRT	.02	[-.01, .06]	
Binge → SSRT	.05	[-.07, .17]	
Binge → HRV reac → SSRT	-.01	[-.05, .01]	
<u>HRV RECOVERY MODEL</u>			.01
Binge → HRV recovery	.05	[-.30, .40]	
HRV recovery → SSRT	.02	[-.05, .09]	
Binge → SSRT	.04	[-.08, .16]	
Binge → HRV recov → SSRT	.00	[-.02, .02]	
<u>MAP REACTIVITY MODEL</u>			.02
Binge → MAP reactivity	-4.51	[-12.83, 3.81]	
MAP reactivity → SSRT	-.00	[-.00, .00]	
Binge → SSRT	.00	[-.11, .12]	
Binge → MAP reac → SSRT	.01	[-.01, .04]	
<u>MAP RECOVERY MODEL</u>			.02
Binge → MAP recovery	-.22	[-6.03, 5.60]	
MAP recovery → SSRT	.00	[-.00, .01]	
Binge → SSRT	.07	[-.05, .19]	
Binge → MAP recov → SSRT	-.00	[-.02, .01]	
<u>POST APPRAISAL MODEL</u>			.01
Binge → Post	.84	[-.11, 1.78]	
Post → SSRT	.01	[-.02, .04]	
Binge → SSRT	.03	[-.09, .15]	
Binge → Post → SSRT	.01	[-.02, .04]	

Note: **Bolded\*** values indicate significance at the  $p < .05$  level.

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Table 9. Impact of Binge on Negative Affect Through Stress Response

Pathways Tested	b	CI	Model R <sup>2</sup>
<u>HRV REACTIVITY MODEL</u>			<b>.25*</b>
Binge → HRV reactivity	-.49	[-1.21, .24]	
HRV reactivity → NA	.08	[-1.03, 1.18]	
Binge → NA	<b>9.57*</b>	<b>[5.86, 13.28]</b>	
Binge → HRV reac → NA	-.04	[-1.47, .54]	
<u>HRV RECOVERY MODEL</u>			<b>.25*</b>
Binge → HRV recovery	.16	[-.24, .56]	
HRV recovery → NA	.30	[-1.72, 2.32]	
Binge → NA	<b>9.48*</b>	<b>[5.78, 13.19]</b>	
Binge → HRV recov → NA	.05	[-.44, .78]	
<u>MAP REACTIVITY MODEL</u>			<b>.29*</b>
Binge → MAP reactivity	-4.29	[-13.74, 5.15]	
MAP reactivity → NA	-.04	[-.13, .06]	
Binge → NA	<b>10.20*</b>	<b>[6.34, 14.06]</b>	
Binge → MAP reac → NA	.15	[-.37, .92]	
<u>MAP RECOVERY MODEL</u>			<b>.27*</b>
Binge → MAP recovery	-4.03	[-10.47, 2.41]	
MAP recovery → NA	-.02	[-.15, .12]	
Binge → NA	<b>9.70*</b>	<b>[5.92, 13.47]</b>	
Binge → MAP recov → NA	.07	[-.66, .70]	
<u>POST APPRAISAL MODEL</u>			<b>.25*</b>
Binge → Post	<b>1.25*</b>	<b> [.26, 2.23]</b>	
Post → NA	.14	[-.68, .95]	
Binge → NA	<b>9.36*</b>	<b>[5.56, 13.17]</b>	
Binge → Post → NA	.17	[-.84, 1.43]	

Note: **Bolded\*** values indicate significance at the  $p < .05$  level.

## CHAPTER FOUR

### DISCUSSION

#### 4.1. Study 2 Discussion and Limitations

##### 4.1.1. Study 2 Discussion of Findings

Two aims were pursued in Study 2. First, we sought to replicate the findings from Study 1 which indicated that stress leads to increased binge eating through increased impulsivity. Consistent with Study 1, analysis of the pretest survey results in the present study showed that one process through which stress impacts binge eating is through its influence on impulsivity. Specifically, higher stress led to greater impulsivity which in turn led to greater binge eating severity.

The second aim of Study 2 was to manipulate a stressful situation in which to examine the way that women higher in binge eating severity might react differently than women lower in binge eating. This time, impulsivity was measured using a stop-signal inhibitory control task. Consistent with a successful stress manipulation, physiological variables differed significantly from baseline, and self-reported threat appraisal was significantly higher after the task than pre-task, suggesting that women were indeed stressed as a result of the paradigm during the laboratory study.

Binge eating was once again correlated with negative affect, this time after a stressful task, which is consistent with Study 1. Direct effects also supported the literature indicating that higher binge eating predicted higher negative affect (Haedt-Matt & Keel, 2011). Consistent with the overlap between binge eating and drive to eat these were correlated as well, although not too strongly, suggesting they still measured unique constructs. Likewise, direct effects indicated that binge eating predicted increased food cravings and drive to eat within the context of stress which

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is consistent with prior findings (Schag et. al., 2013). Our findings indicated that BMI evidenced a separate pattern from that of BES and using BMI as a covariate did not impact results, suggesting that obesity alone is not responsible for this effect. Lastly, binge eating predicted post-task threat appraisal only when tested as a mediator of the impact of binge on negative affect. This is consistent with literature supporting a more maladaptive stress response for those who binge eat (Lo Sauro et. al., 2008), although this was only observed with self-reported threat.

Contrary to predictions, only one significant interaction was present for the moderation models tested. This interaction showed that more maladaptive stress (i.e., lower HF-HRV; Thayer et. al., 2012) was associated with women reporting less negative affect among those higher in BES. This suggests that for those higher in binge eating, more adaptive stress was actually associated with higher negative affect, which is contrary to what was expected based on the established relationships between increased binge eating, maladaptive stress, and increased negative affect (Haedt-Matt & Keel, 2011; Adam & Epel, 2007; Lee et al., 2015).

Interestingly, the level of BES that represents the higher level in this study falls at 18.79, which is in the subthreshold range of binge eating severity. Further, these findings suggest that in addition to the results of Study 1, which showed that these factors influence binge eating significantly, Study 2 shows that binge eating also impacts those factors; thus, a cyclical process may indeed be implicated.

These findings are strengthened by the measuring of constructs over multiple timepoints, in reaction to real-life stress related to social evaluation and outcome pressure. Binge eating severity was measured in a pre-session survey and it predicted outcomes measured later in the lab after a stressful situation. However, it is worth noting that these data still reflect a correlational design so no causal inferences can be made despite being measured over multiple

timepoints. Further, these relationships were demonstrated among a sample that was overall quite low in binge eating, with the average falling well below the subthreshold range, indicating that even low levels of binge eating predict negative outcomes (negative affect, drive to eat) at a later time point.

Hypothesis 1 sought to answer whether those with binge eating might react to maladaptive (threat) stress (i.e., lower HF-HRV during task and higher MAP during recovery) in a different way than those who are lower in binge eating, particularly with more impulsivity. As none of the moderation models tested with SSRT as the outcome variable showed significant interactions, Hypothesis 1, was not supported. Further, binge eating was not correlated with MAP during recovery or HRV during the stressor; as such, Hypothesis 2 was not supported either. Overall, the lack of interactions and indirect effects suggests that moderation and mediation models do not best represent the relationships between stress response, binge eating, and impulsivity/drive to eat.

### **4.1.2. Limitations**

There were several limitations to this study that may have impacted the results. The participants in this study were not screened based on binge eating severity and as such, the sample does not reflect a clinical population. This was done in an attempt to examine subthreshold binge eating as well, as this has been shown to cause similar distress to that caused by diagnosable binge eating (Colles et al., 2008). It may be helpful in future studies to examine these variables within groups identified as having Binge Eating Disorder (meeting criteria), subthreshold BED, and no BED symptoms. Recall that in Study 1, the BES score ranged from 0-45.00 ( $M = 10.49$ ,  $SD = 7.89$ ). In this study the range of binge eating severity was 0 - 32.00 ( $M = 10.81$ ,  $SD = 7.57$ ), suggesting that this present sample reflects an even smaller range of severity.

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Scores equal to or greater than 27 are used to indicate severe binge eating symptomatology, while scores equal to or less than 17 indicate non-binge eaters (Celio et al., 2004). As such, the average for this sample falls well below the threshold for binge eating symptomatology and only 7 people (6.1% of binge eating scores) had scores that indicated severe binge eating. Further, there were 12 participants (10.6% of binge eating scores) with subthreshold binge eating (scores of 18-26). Yet, relationships between binge and negative outcomes measured after the stressful task were observed even among this sample that is low in binge eating overall. In fact, the higher level of binge eating (that was observed to experience decreased negative affect in response to threat) fell in the subthreshold range of BES scores. Perhaps this non-clinical level of binge eating represents a subset of binge eaters who experience similar distress overall but react differently to stress. Future research should examine this in order to tailor individualized interventions for those experiencing various levels of binge eating.

Unfortunately, no effects were found using SSRT as an operationalization of impulsivity. Indeed, our trait measure of impulsivity was not significantly related to SSRT (see Table 3). One argument is that SSRT may not be an appropriate marker of impulsivity within this post-stressor context, as this has not been examined in prior studies. Many BED studies thus far have examined performance on go/no-go tasks with food or body-related stimuli specifically, and most often, omissions/commissions have been presented rather than SSRT (Shag et al., 2013; Mobbs et al., 2011). SSRT was chosen for this study because it was created specifically to represent impulsivity, as it reflects the average time it takes someone to stop a motor response in response to an external stimulus. That is, instead of representing the amount of times the participant cannot inhibit the response (i.e., error rate), SSRT considers how long on average the person takes to inhibit their response--this allows us to see when people are slower to think

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through the decision to stop and then follow-through which is thought to most closely represent impulsivity (Verbruggen, Chambers and Logan, 2013). Notably, this motor response differs from inhibitory control, or the cognitive process of inhibiting a response, which the task was originally designed to measure. This has not yet been fully examined in the context of binge eating. One reason provided for not using SSRT in a binge eating study was due to using a fixed SSD time (the easiest method for calculating SSRT requires mean SSD), whereas we chose to vary the onset of the stop signal in order to increase demands on the inhibitory process (Oliva, Morys, Horstmann, Castiello, & Begliomini, 2019). It is possible that impulsivity within the context of binge eating is not best represented by SSRT, as the task involves an external stimulus that is rarely present within a binge eating context, particularly as it typically involves eating alone due to embarrassment (APA, 2013). Perhaps reacting impulsively in the face of internal stimuli would be more relevant to investigating binge eating behavior and this may be a target for future research.

One study of 80 women did find that SSRT was higher, suggesting more impulsivity for those with BED (diagnosed by semi-structured interviews (Manasse, Goldstein, Wyckoff, Forman, Juarascio, Butryn, Ruocco, & Nederkoorn, 2016). Further, this was true for tasks that involved food stimuli as well as those that did not. Thus, there is prior support for using SSRT to characterize impulsivity in the context of binge eating, and yet we did not observe a relationship between SSRT and binge eating in this post stress context. It is unclear what is responsible for the lack of SSRT effects in our study. One possibility is that SSRT is inherently limited by the fact that unlike the finishing times involved in the “go” process, the “stop” process cannot be directly observed and must be inferred by lack of response (Teichert & Ferrera, 2015). Thus, the distribution of SSRT is an estimation and its true shape cannot be observed. To address this,

modified stop signal tasks (i.e., typing tasks provide a hard lower estimate and a soft upper limit due to the required key-presses) have been developed to provide a more narrow window for estimating SSRT and this method could be utilized in future studies in the context of a stressor. This could provide more accurate information about whether impulsivity, as characterized by SSRT, might appear different in the face of a social stressor, as it is expected based on the literature that SSRT would be higher in response to higher stress (Ansell et al., 2012). Further, perhaps using food-related stimuli like previous literature would illustrate a process that is more salient to binge eating.

### **4.2. General Discussion**

Overall, this project has aimed to outline a more comprehensive model of binge eating in reaction to stress, investigating the role of key variables implicated in the literature (i.e., negative affect, cognitive restraint, and impulsivity). This dissertation included a large sample of self-report data collected via online survey from both male and female participants (Study 1). Physiological and cognitive task data were later collected in the context of a manipulated stressor in order to more closely examine the process by which women in particular might react to stress differently based on their level of binge eating severity (Study 2). In Study 1 cognitive restraint was not significantly related to negative affect in men, nor was it related to impulsivity in either group, possibly due to the cognitive restraint measure being specific to dietary restraint. Further, Study 1 showed that only women experienced increased binge eating directly predicted by increased stress. Self-report data from both studies demonstrated relationships among almost all proposed variables, but overall, the findings of this dissertation offer the strongest support for negative affect and impulsivity being involved in the etiology and maintenance of stress-induced binge eating.



#### **4.2.1. Support for a Cyclical Process Involving Negative Affect**

This project demonstrated that one way that stress impacts binge eating is through negative affect (Study 1). In Study 1, stress predicted negative affect for everyone, and for women alone, negative affect was a mechanism by which stress led to binge eating. Specifically, only women were observed to experience higher binge eating in response to stress through negative affect/impulsivity, as well as through negative affect/cognitive restraint; this further indicates a distinct process for men and women. Within the context of a manipulated stressor (Study 2), negative affect post-stressor was also significantly related to higher perceived stress measured in the online survey and both higher drive to eat and self-reported threat appraisal measured after the challenging tasks. This is all consistent with literature indicating negative affect as a precursor for binge and as a result of high perceived stress/threat (Leehr et al., 2015; Lazarus & Folkman, 1984). Further, higher negative affect after a stressful task was predicted by higher pre-session levels of binge eating severity; this is consistent with literature indicating that those higher in binge eating tend to experience increased depressed mood and other facets of negative affect overall (Goossens et al., 2010).

Study 2 also sought to expand on the information gained about the etiology of binge eating by considering whether those at different levels of binge eating experience more negative outcomes in response to stress. This was true for the impact of HRV reactivity on negative affect measured post-stressor; those higher in binge eating evidenced lower negative affect as a result of more maladaptive stress. Specifically, those higher in binge eating evidenced higher negative affect overall, but it trended lower as HRV reactivity decreased. So how can we understand this seemingly better outcome in the face of more harmful stress?

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One possible answer is to consider whether lower affect in this context does indeed represent a better outcome. In particular, those at a higher level of binge eating may experience a disconnect between conscious emotions and physiological stress, suggesting that perhaps when threatened they practice more avoidance of their affective experience. In fact, research shows that BES scores are positively correlated with both avoidant (i.e., pretend that nothing is wrong) and emotional (i.e., rumination and blaming oneself) coping styles (Sulkowski et al., 2011). Could it be that binge eaters switch from an emotional to a more avoidant coping style in the face of threatening stress that exceeds their resources? Given that in our sample, those higher in binge eating fell at a subthreshold level, perhaps this could even be a mechanism by which those individuals stave off a binge eating episode, at least temporarily, as their ability to disconnect from a threatening experience precludes the need to binge eat as an emotion regulation process to alleviate negative affect (Leehr et al., 2015). Indeed, the same study showed that emotional coping mediated the relationship between stress and binge eating, while avoidant coping did not.

In addition, our finding that higher pre-session binge eating predicted higher post-stressor negative affect supports a more cyclical nature of binge eating, which has been suggested in the literature with regard to emotion (Leehr et al., 2015). Further research should examine this within the context of stress and observable food intake to determine whether binge eating in response to stress is both precipitated and followed by increased negative affect. If this were indeed the case, then it would indicate that understanding how binge eaters experience and react to negative affect is of crucial importance for effective intervention.

### **4.2.2. Impulsivity as a Pathway to Binge**

Another key mechanism for understanding stress-induced binge eating might be impulsive, rash behavior, as this project also supports this as an important precursor for binge

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eating as a result of stress. In particular, impulsivity evidenced the strongest support as a mechanism through which stress influenced binge eating in Study 1. Stress predicted impulsivity for everyone, while impulsivity in turn predicted binge eating. This is consistent with the established relationships between stress, impulsivity, and binge eating (Ansell et al., 2012; Helen et al., 2010; Schag et al., 2013). Indirectly, women showed that the strongest pathway by which stress predicted binge eating was impulsivity, and this was the only significant pathway for men. Study 2 successfully replicated the self-report finding that stress impacts binge eating through trait impulsivity.

As discussed above, women in Study 1 also showed that higher perceived stress predicted higher negative affect, which in turn predicted higher impulsivity and then higher binge eating. This supports the research previously indicating negative urgency as a key factor for women who binge eat at all levels (Racine et al., 2015). In particular, it has been shown to be higher in women with both loss of control over eating and objective overeating, as well as women who experience only one of these. Future studies should examine how impulsivity might be involved with a more avoidant and detached response to threat stress, as this could also shed light on our findings. Might it be that the avoidance itself is an impulsive reaction to distress? This could even be reinforced through the process of impulsively binge eating and experiencing some relief, as a switch to disengaging with the most threatening moments by eating uncontrollably would certainly serve a function. Given that this effect in our study was among a subthreshold BED level, perhaps this process of disengaging with emotions in the face of threat might even be a precursor for BED. Understanding more about the role of impulsivity in this potential disconnect between physiological stress and emotion will help to start illuminating whether this kind of response is associated with more or less binge eating.

In Study 2, our trait measure of impulsivity was significantly related to higher negative affect after a stressor and, interestingly, with lower MAP reactivity (i.e., less stress reactivity). The relationship between negative affect and impulsivity is well-established in the literature (Racine et al., 2015), but impulsivity correlating to lower stress during the task is intriguing. Again, it indicates that perhaps more impulsive individuals are disengaging from stressful experiences, possibly in such a way that impacts physiological responding. In our study, task-measured impulsivity after a stressful task showed no significant relationships with stress response, binge eating, or any other hypothesized variables. Future studies should seek to identify the most accurate way to characterize impulsivity, and neuroimaging and cerebral oxygenation measures may provide some guidance toward this.

### **4.2.3. Clinical Implications**

The clinical implications of this information are far-reaching and have been indicated throughout this discussion, as those with subclinical levels of binge eating may require intervention that is not currently being indicated based on a diagnostic model of care. Specifically, the findings discussed above suggest that there may be a distinct process of disengaging from negative affect when faced with threatening situations that might facilitate, or possibly even circumvent, binge eating. Understanding this process more could help therapists intervene by helping individuals become more mindful of their internal experience and develop adaptive ways to cope with this. For instance, perhaps the process by which subthreshold binge eaters switch to an avoidant pattern of coping under more threatening stress is protective in preventing binge eating episodes, or it could reinforce an “escape” response that might eventually involve binge eating as well. It might be that this way of coping is helpful for a time but eventually fails and contributes to an escalation to full BED. Gaining an understanding of

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these hypothesized processes could be key to prevention of binge eating disorder in individuals who endorse some binge eating. This is vital, as it has been shown that some individuals with subthreshold levels of binge eating progress to diagnosable BED later in life, as 28% ( $N = 5$ ) of the subthreshold sample from one study was later diagnosed with BED (Stice et al., 2013). This study followed adolescent girls over the course of 8 years, suggesting that this finding provides evidence for subthreshold BED being a precursor to BED in some women. Thus, understanding this early stage of binge eating development and tailoring clinical interventions based on this information could serve to help these individuals implement important changes and prevent future BED. As reviewed in the introduction, there are several medical and psychological comorbidities of BED suggesting that if one could prevent a client from developing from subthreshold to BED, there could be important implications for quality of life.

One implication of this is to consider the importance of negative affect and depression when treating those with BED. Recall that depressive symptoms are positively correlated with binge eating, and the two disorders are highly comorbid, indicating that integrated treatment of depression and binge eating might provide a more comprehensive model of care with more lasting impact (Goossens et al., 2010; Grilo et al., 2009). Indeed, it has been shown that among 72 adults with BED, depressive symptoms (measured with the Brief Symptoms Inventory) mediated the relationship between BED diagnosis and health-related quality of life, suggesting that depression is an important part of why binge eaters experience poor quality of life (Singleton, Kenny, Hallett, & Carter, 2019). Thus, addressing both simultaneously in an integrated approach could have more meaningful outcomes. Understanding the cyclical, complex nature of the relationship between negative affect and binge might provide important information

for developing such a treatment, and future studies should aim to examine this, particularly within the context of varying levels of stress.

It has also been suggested that negative urgency predicts binge eating frequency in college women while distress tolerance does not, and this may help us understand the present findings (Kelly et al., 2014). Perhaps what causes those higher in binge eating to disengage from their emotions in the face of threat is the need to avoid impulsivity that might lead to binge, rather than being unable to tolerate the emotions. Thus, interventions that target the process of reacting impulsively to negative emotions might be helpful for those who binge eat. For example, examining the thoughts that occur just prior to the impulsive behavior and identifying how they intersect with one's negative emotions could be indicated--what is it about those emotions that cause the client to be rash, what functions does the rashness serve? Examining these questions with clients might outline important needs that they are attempting to meet through these unhelpful actions.

#### **4.2.4. Limitations**

These findings may not generalize to clinical levels of binge, racially diverse populations, or non-female samples. Overall, this sample did not reflect a clinical population of binge eaters, and those who are diagnosed with BED may evidence different relationships with the proposed variables than this group of overall subthreshold binge eating. However, much of the research appears to indicate analogous processes for subthreshold and full BED, with some evidence that BED participants show more exaggerated effects (Galanti et al., 2007; Colles et al., 2008; Racine et al., 2015; Carrard et al., 2012; Stice et al., 2013; Mustelin et al., 2015; Hudson et. al., 2007). For instance, a sample of college women ( $N = 715$ ; Napolitano & Himes, 2011) demonstrated that those that met criteria for BED had higher levels of negative affect before a binge eating

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episode than those who endorsed some binge eating but did not meet criteria for BED. The purpose of this project was to gain a better understanding of subthreshold binge eating by including a sample with a range of binge eating severity. Although our sample was smaller in range than expected and represented a group that is relatively low in binge eating, preventing us from speaking to how these processes apply to clinical levels of BED, this has indeed allowed us to learn more about the subthreshold level. Notably, it is still possible that these same processes occur among those with BED but that we were unable to examine this due to the low number of participants indicating this level of severity, and we did not conduct clinical interviews to truly diagnose BED. These processes should be examined in future research with distinct clinical (diagnosed with interview), subthreshold, and obese controls in order to build on our findings indicating that subthreshold binge might reflect a unique process.

Further, one limitation of both studies is the lack of racial diversity present in this geographical region. In Study 2, 86.6% of the sample indicated their race as “Caucasian or White,” while 89.3% of the sample in Study 1 identified themselves as such. Some research suggests that White individuals evidence higher rates of BED with higher binge eating symptomatology, as well as higher levels of depressed mood, dietary cognitive restraint and body dissatisfaction in a college sample of women (Napolitano & Himes, 2011); this suggests that the proposed variables for this project may be more relevant for White women than other races/ethnicities. However, African American participants have also been shown to experience binge eating in a distinct way to that of White individuals. In particular, anxiety (a facet of negative affect) was lower prior to binge eating for African American participants than for White participants. Further research should be conducted to consider what clinical interventions might

be indicated within a multicultural framework, individualizing client care based on the influence of culture.

Further, some of the findings of this study provided information about how men and women might experience binge eating differently, and it was demonstrated that the process by which men binge eat in response to stress is tied more closely to impulsivity, while women showed other significant pathways. As such, the information gleaned in Study 2, which included only women, may not generalize to how men experience binge eating. Further, to our knowledge no research has examined the correlates of binge eating among non-binary or transgender individuals and this should be an important goal of current research given the aforementioned differences demonstrated between just men and women in this study.

### **4.2.5. Conclusion**

Overall, this project supports the hypothesized variables (particularly negative affect and impulsivity) as important factors to consider in understanding and developing interventions for binge eating, especially at the subclinical level. Future studies should aim to include individuals with a broader range of binge eating severity, conducting clinical interviews, in order to truly delineate the key differences between clinical and subthreshold levels that may provide crucial information for intervention. Negative affect is indicated not only in the etiology of binge eating, but it is related to somatic health and might represent a key reason for why BED significantly impacts quality of life (Lazarus & Folkman, 1984). Thus, further research should also aim to consider the impact of clinical interventions to address the specific impact that negative affect has on binge eating, particularly in the face of stress. Perhaps the most intriguing finding of this project is the indication that those who are higher in binge eating experience lower negative affect in response to a more threatening stress response. Understanding whether this might



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represent a blunted response that serves the function of disconnecting binge eaters with what their body is telling them could provide exciting information about whether this may be a protective process by allowing them to behave less rashly.

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**APPENDICES**

**Appendix A. Sona Systems Study 1 Description**

Study Name: College Student Eating Behavior Survey

Study Type: Web Study

Duration: 60 minutes

Credits: 1 credit

Description: We are interested in the eating behaviors and other psychological variables of college-age students, including stress and negative affect. You will be asked to respond to a number of questionnaires anonymously, and will receive 1 credit for this study.

**Appendix B. Sona Systems Study 2 Description**

Study Name: First Impressions and Cognition Study (Part 1 and 2)

Study Type: Web Study and Standard Laboratory Study

Duration: 180 minutes

Credits: up to 3 credits

Description: We are interested in cognitive functioning of college-age students when first impressions are formed, as well as other psychological variables. You will be asked to respond to a number of questionnaires in an online-survey (Part 1). Later, you will be asked to complete a speaking task and a computer-based cognitive task while psychophysiological measures are collected with non-invasive sensors (Part 2). You will receive up to 3 credits for this study.



### **Appendix C. Study 1 Informed Consent**

You are invited to participate in a research study being conducted by Rachael Huff and Shannon McCoy, PhD in the Department of Psychology. We are interested in factors related to eating behaviors in college-age populations, as well as the relationship to psychological variables for both men and women. Because you are age 18 (or over) in Introductory Psychology you are being invited to participate in this study. You will receive 1 research credit for your participation.

#### What you will be asked to do:

You will be asked to honestly answer a number of questions related to stress, eating, negative affect, and impulsivity. Sample statements include: “When I am really ecstatic, I tend to get out of control.” The survey is anonymous and should take about an hour to complete.

#### Risks

There is the risk that you may become uncomfortable answering the questions. You may opt to skip any questions you find stressful or uncomfortable. If you have concerns, you may contact the researchers (contact information is below). If you experience any discomfort and wish to talk with someone, please call the campus Counseling Center at (207) 581-1392.

#### Confidentiality

The survey responses will be anonymous so your name will not be associated with any of the research findings. Survey data will be kept indefinitely in accordance with guidelines of the American Psychological Association. Data will be kept on a password-protected hard drive in a locked office.

#### Benefits

While there is no direct benefit to you, it is hoped that the information gained from this study will help in understanding eating habits of college-age individuals and possible risk factors for maladaptive eating behavior such as binge eating.

#### Compensation

You will receive 1 hour of research credit for your participation.

#### Voluntary

Your participation is voluntary. You may skip any questions that you wish not to answer and you may stop participation at any time without the loss of credit.

#### Contact Information

If you have any questions about this study, please contact Rachael Huff ([rachael.huff@maine.edu](mailto:rachael.huff@maine.edu)) or Dr. Shannon McCoy ([shannon.mccoy@umit.maine.edu](mailto:shannon.mccoy@umit.maine.edu); 207-581-2029). 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 email [gayle.jones@umit.maine.edu](mailto:gayle.jones@umit.maine.edu)).

Please click “next” if you have read and understood this page and agree to participate in the study.

### **Appendix D. Study 2 Informed Consent**

The following research is being administered by Rachael Huff and Shannon McCoy, PhD in the Department of Psychology. We are interested in factors related to first impressions in college-age populations, as well as the relationship to other psychological variables, such as cognition, for both men and women. Because you are age 18 (or over) in Introductory Psychology and have completed the Psychology Department Prescreening, you are being invited to participate in this study. You will receive up to 3 research credits for your participation.

#### *What you will be asked to do*

In part 1, you will be asked to honestly answer a number of questions related to your health, mood, thoughts and beliefs. Sample statements include: “When I am really ecstatic, I tend to get out of control” and “I have a reserved and cautious attitude toward life.” The survey should take about half an hour to complete.

In part 2, you will come into the laboratory for approximately an hour and a half. You will be asked to complete challenging tasks (a brief speaking task followed by a computer-based cognitive task) while psychophysiological data (e.g., assessing your heart functioning via blood pressure and other cardiovascular measures) are collected via non-invasive sensors on your legs, arms, chest and head. This equipment is not medical-grade and measurements collected are not diagnostic. You will be administered brief self-report questionnaires before and after these tasks, in which you will be asked about your mood and thoughts. Sample items include “The task was/will be very demanding” and “I feel confident about my abilities.” Your height and weight will be measured.

#### *Risks*

The risks associated with this study are generally minimal and include any inconvenience caused by the time it takes to complete the survey. Additionally, you may feel uncomfortable while answering questions about yourself. You may opt to skip any questions you find stressful or uncomfortable, 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 and you will still receive credit for the time you have spent in the study. There are no risks to the psychophysiological monitoring and measurements other than possible skin irritation upon removal of the sensors (like removal of a large band-aid). This irritation may leave initial red marks which should go away a few hours after removal. The challenging tasks are generally without risk, but you may have strong emotional reactions to the tasks and you may find the tasks to be stressful or uncomfortable. In that case, one of the investigators will be available to help you, and you can opt to skip any questions or stop the experiment at any time. If you experience any psychological distress from participating in this study, please contact the counseling center at (207) 581-1392.

#### *Confidentiality*

Your name will not be associated with any of the research findings. If you agree to participate, you will be assigned a participant number which will be used to link prescreening responses, subsequent questionnaires and study data. Your name will appear only on this consent form, which will be kept apart from any other study information in a locked office accessible only to study personnel. A cross-index key will be created linking your name and participant number,

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and this will be stored using software that provides additional security (BitLocker). This key will be destroyed after main data analyses have been completed, which we anticipate will be done by December 2019. All data will be identified by participant number only and will be kept in a locked office. Only your participant number will appear on all study data. Data will be kept indefinitely in accordance with guidelines of the American Psychological Association. Data will be kept on a password-protected computer in a locked office.

### Benefits

While there is no direct benefit to you, it is hoped that the information gained from this study will help in understanding cognition and other psychological variables in college-age individuals.

### Compensation

You will receive up to 3 hours of research credit for your participation. If you choose not to schedule part 2 of this study, you will only receive 1 credit for your time in part 1. If you begin the part 2 laboratory session and withdraw within the first hour, you will receive only 1 credit for your laboratory participation.

### Voluntary

Your participation is voluntary. You may skip any questions that you wish not to answer and you may stop participation at any time without the loss of credit earned up to that point.

### Contact Information

If you have any questions about this study, please contact Rachael Huff (rachael.huff@maine.edu) or Dr. Shannon McCoy (shannon.mccoy@umit.maine.edu; 207-581-2029). 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 email gayle.jones@maine.edu).

For part 1, please click "next" if you have read and understood this page and agree to participate in the study. For part 2, you have already seen this consent form online. Please sign below to indicate your continued agreement to participate.

**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 E. Debriefing Script**

Full Study:

Thank you for participating in our research today. We are examining cardiovascular responses and cognitive functioning during potentially stressful situations. I don't know which condition you had today, but sometimes people who do this study are led to believe they are being evaluated by someone else while giving the speech. As you can imagine, doing this might impact heart rate and performance on the computer task, and this is something we are interested in looking at. You were informed that this study was investigating first impressions and cognitive functioning; however, the other participant you interacted with was a confederate from our lab. We worked really hard to fool you because it is so important for our research that you think they are a real participant who might be evaluating you. We couldn't do this work without your help, it's really appreciated! This cover story was created in order to give us unbiased information regarding your eating/food choices in the face of stress, as well as to support our stress condition. We are also interested in how answers to the survey questions relate to performance on the computer task. For example, one of the things we can measure from the computer task is impulsivity, so some of the answers given on the survey might be related to how impulsive someone is. Do you have any questions or concerns about our study? It is really important that other participants have a similar experience to what you had today, so we ask that you please refrain from talking to anyone about the study tasks. Thank you for your time today!

**Appendix F. Study 1 Questionnaire**

*UPPS-P Impulsive Behavior Scale (revised version)*

Directions: Below are a number of statements that describe ways in which people act and think. For each statement, please indicate how much you agree or disagree with the statement. If you **Agree Strongly** circle **1**, if you **Agree Somewhat** circle **2**, if you **Disagree somewhat** circle **3**, and if you **Disagree Strongly** circle **4**. Be sure to indicate your agreement or disagreement for every statement below.

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

1. I have a reserved and cautious attitude toward life.
2. I have trouble controlling my impulses.
3. I generally seek new and exciting experiences and sensations.
4. I generally like to see things through to the end.
5. When I am very happy, I can't seem to stop myself from doing things that can have bad consequences.
6. My thinking is usually careful and purposeful.
7. I have trouble resisting my cravings (for food, cigarettes, etc.).
8. I'll try anything once.
9. I tend to give up easily.
10. When I am in great mood, I tend to get into situations that could cause me problems.
11. I am not one of those people who blurt out things without thinking.
12. I often get involved in things I later wish I could get out of.
13. I like sports and games in which you have to choose your next move very quickly.
14. Unfinished tasks really bother me.
15. When I am very happy, I tend to do things that may cause problems in my life.
16. I like to stop and think things over before I do them.
17. When I feel bad, I will often do things I later regret in order to make myself feel better now.
18. I would enjoy water skiing.
19. Once I get going on something I hate to stop.
20. I tend to lose control when I am in a great mood.
21. I don't like to start a project until I know exactly how to proceed.

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22. Sometimes when I feel bad, I can't seem to stop what I am doing even though it is making me feel worse.
23. I quite enjoy taking risks.
24. I concentrate easily.
25. When I am really ecstatic, I tend to get out of control.
26. I would enjoy parachute jumping.
27. I finish what I start.
28. I tend to value and follow a rational, "sensible" approach to things.
29. When I am upset I often act without thinking.
30. Others would say I make bad choices when I am extremely happy about something.
31. I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional.
32. I am able to pace myself so as to get things done on time.
33. I usually make up my mind through careful reasoning.
34. When I feel rejected, I will often say things that I later regret.
35. Others are shocked or worried about the things I do when I am feeling very excited.
36. I would like to learn to fly an airplane.
37. I am a person who always gets the job done.
38. I am a cautious person.
39. It is hard for me to resist acting on my feelings.
40. When I get really happy about something, I tend to do things that can have bad consequences.
41. I sometimes like doing things that are a bit frightening.
42. I almost always finish projects that I start.
43. Before I get into a new situation I like to find out what to expect from it.
44. I often make matters worse because I act without thinking when I am upset.
45. When overjoyed, I feel like I can't stop myself from going overboard.
46. I would enjoy the sensation of skiing very fast down a high mountain slope.
47. Sometimes there are so many little things to be done that I just ignore them all.
48. I usually think carefully before doing anything.
49. When I am really excited, I tend not to think of the consequences of my actions.

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50. In the heat of an argument, I will often say things that I later regret.
51. I would like to go scuba diving.
52. I tend to act without thinking when I am really excited.
53. I always keep my feelings under control.
54. When I am really happy, I often find myself in situations that I normally wouldn't be comfortable with.
55. Before making up my mind, I consider all the advantages and disadvantages.
56. I would enjoy fast driving.
57. When I am very happy, I feel like it is ok to give in to cravings or overindulge.
58. Sometimes I do impulsive things that I later regret.
59. I am surprised at the things I do while in a great mood.

### Binge Eating Scale

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

#### #1

- 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.

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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 any 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.



## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### #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 food 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.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### #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 snack.
- 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.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### #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 preoccupied 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.

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

PANAS

Directions: This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word.

**Indicate to what extent you have felt this way over the past week.**

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

Very Slightly      A Little      Moderately      Quite a Bit      Extremely

Or Not at All

\_\_\_\_\_ 1. Interested

\_\_\_\_\_ 2. Distressed

\_\_\_\_\_ 3. Excited

\_\_\_\_\_ 4. Upset

\_\_\_\_\_ 5. Strong

\_\_\_\_\_ 6. Guilty

\_\_\_\_\_ 7. Scared

\_\_\_\_\_ 8. Hostile

\_\_\_\_\_ 9. Enthusiastic

\_\_\_\_\_ 10. Proud

\_\_\_\_\_ 11. Irritable

\_\_\_\_\_ 12. Alert

\_\_\_\_\_ 13. Ashamed

\_\_\_\_\_ 14. Inspired

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

\_\_\_\_\_ 15. Nervous

\_\_\_\_\_ 16. Determined

\_\_\_\_\_ 17. Attentive

\_\_\_\_\_ 18. Jittery

\_\_\_\_\_ 19. Active

\_\_\_\_\_ 20. Afraid

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Perceived Stress Scale

Directions: The questions in this scale ask you about your thoughts and feelings during the last month. In each case, please indicate how often you felt or thought a certain way by choosing the appropriate value from the scale below.

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

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?

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Three Factor Eating Questionnaire-R18

Directions: Please rate the extent to which each of the following statements is true for you. There are no right or wrong answers for any statement. The best answer is what you think is true for yourself.

1-----2-----3-----4  
Definitely False      Mostly False      Mostly True      Definitely True

1. When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.
2. I deliberately take small helpings as a means of controlling my weight.
3. When I feel anxious, I find myself eating.
4. Sometimes I start eating, I just can't seem to stop.
5. Being with someone who is eating often makes me hungry enough to eat too.
6. When I feel blue, I overeat.
7. When I see a real delicacy, I often get so hungry that I have to eat right away.
8. I get so hungry that my stomach often seems like a bottomless pit.
9. I am always hungry so it is hard for me to stop eating before I finish the food on my plate
10. When I feel lonely, I console myself by eating.
11. I consciously hold back at meals in order not to gain weight.
12. I do not eat some foods because they make me fat.
13. I am always hungry enough to eat at any time.

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

Only at meal times    Sometimes between meals    Often between meals    Almost Always

14. How often do you feel hungry?

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

Almost Never    Seldom    Usually    Almost Always

15. How frequently do you avoid 'stocking up' on tempting foods?

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

Unlikely    Slightly Likely    Moderately Likely    Very Likely

16. How likely are you to consciously eat less than you want?

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

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Never            Rarely            Sometimes            At least once a week

17. Do you go on eating binges though you are not hungry?

18. On a scale of 0 to 8, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 8 means total restraint (constantly limiting food intake and never 'giving in' ) what number would you give yourself?

Demographics

Please indicate how you identify your gender: Male, Female \_\_\_\_\_

Please indicate how you identify your race/ethnicity: \_\_\_\_\_

How old are you?

How tall are you? \_\_\_\_\_Feet; \_\_\_\_\_Inches

How much do you weigh? \_\_\_\_\_lbs



**Appendix G. Study 2 Online Questionnaire**

PANAS-X

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 few weeks. Use the following scale to record your answers:

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

Very Slightly    A Little    Moderately    Quite a Bit    Extremely  
Or Not at All

_____ 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

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### UPPS-P Impulsive Behavior Scale (revised version)

Directions: Below are a number of statements that describe ways in which people act and think. For each statement, please indicate how much you agree or disagree with the statement. If you **Agree Strongly** circle **1**, if you **Agree Somewhat** circle **2**, if you **Disagree somewhat** circle **3**, and if you **Disagree Strongly** circle **4**. Be sure to indicate your agreement or disagreement for every statement below.

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

1. I have a reserved and cautious attitude toward life.
2. I have trouble controlling my impulses.
3. I generally seek new and exciting experiences and sensations.
4. I generally like to see things through to the end.
5. When I am very happy, I can't seem to stop myself from doing things that can have bad consequences.
6. My thinking is usually careful and purposeful.
7. I have trouble resisting my cravings (for food, cigarettes, etc.).
8. I'll try anything once.
9. I tend to give up easily.
10. When I am in great mood, I tend to get into situations that could cause me problems.
11. I am not one of those people who blurt out things without thinking.
12. I often get involved in things I later wish I could get out of.
13. I like sports and games in which you have to choose your next move very quickly.
14. Unfinished tasks really bother me.
15. When I am very happy, I tend to do things that may cause problems in my life.
16. I like to stop and think things over before I do them.
17. When I feel bad, I will often do things I later regret in order to make myself feel better now.
18. I would enjoy water skiing.
19. Once I get going on something I hate to stop.
20. I tend to lose control when I am in a great mood.
21. I don't like to start a project until I know exactly how to proceed.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

22. Sometimes when I feel bad, I can't seem to stop what I am doing even though it is making me feel worse.
23. I quite enjoy taking risks.
24. I concentrate easily.
25. When I am really ecstatic, I tend to get out of control.
26. I would enjoy parachute jumping.
27. I finish what I start.
28. I tend to value and follow a rational, "sensible" approach to things.
29. When I am upset I often act without thinking.
30. Others would say I make bad choices when I am extremely happy about something.
31. I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional.
32. I am able to pace myself so as to get things done on time.
33. I usually make up my mind through careful reasoning.
34. When I feel rejected, I will often say things that I later regret.
35. Others are shocked or worried about the things I do when I am feeling very excited.
36. I would like to learn to fly an airplane.
37. I am a person who always gets the job done.
38. I am a cautious person.
39. It is hard for me to resist acting on my feelings.
40. When I get really happy about something, I tend to do things that can have bad consequences.
41. I sometimes like doing things that are a bit frightening.
42. I almost always finish projects that I start.
43. Before I get into a new situation I like to find out what to expect from it.
44. I often make matters worse because I act without thinking when I am upset.
45. When overjoyed, I feel like I can't stop myself from going overboard.
46. I would enjoy the sensation of skiing very fast down a high mountain slope.
47. Sometimes there are so many little things to be done that I just ignore them all.
48. I usually think carefully before doing anything.
49. When I am really excited, I tend not to think of the consequences of my actions.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

50. In the heat of an argument, I will often say things that I later regret.
51. I would like to go scuba diving.
52. I tend to act without thinking when I am really excited.
53. I always keep my feelings under control.
54. When I am really happy, I often find myself in situations that I normally wouldn't be comfortable with.
55. Before making up my mind, I consider all the advantages and disadvantages.
56. I would enjoy fast driving.
57. When I am very happy, I feel like it is ok to give in to cravings or overindulge.
58. Sometimes I do impulsive things that I later regret.
59. I am surprised at the things I do while in a great mood.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Binge Eating Scale

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

#### **#1**

- 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.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### #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 any 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 food 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.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### #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.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE 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 snack.
- 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 preoccupied 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.



A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Three Factor Eating Questionnaire-R18

Directions: Please rate the extent to which each of the following statements is true for you. There are no right or wrong answers for any statement. The best answer is what you think is true for yourself.

1-----2-----3-----4  
Definitely False      Mostly False      Mostly True      Definitely True

1. When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.
2. I deliberately take small helpings as a means of controlling my weight.
3. When I feel anxious, I find myself eating.
4. Sometimes I start eating, I just can't seem to stop.
5. Being with someone who is eating often makes me hungry enough to eat too.
6. When I feel blue, I overeat.
7. When I see a real delicacy, I often get so hungry that I have to eat right away.
8. I get so hungry that my stomach often seems like a bottomless pit.
9. I am always hungry so it is hard for me to stop eating before I finish the food on my plate
10. When I feel lonely, I console myself by eating.
11. I consciously hold back at meals in order not to gain weight.
12. I do not eat some foods because they make me fat.
13. I am always hungry enough to eat at any time.

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

Only at meal times    Sometimes between meals    Often between meals    Almost Always

14. How often do you feel hungry?

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

Almost Never    Seldom    Usually    Almost Always

15. How frequently do you avoid 'stocking up' on tempting foods?

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

Unlikely    Slightly Likely    Moderately Likely    Very Likely

16. How likely are you to consciously eat less than you want?

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

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Never            Rarely            Sometimes            At least once a week

17. Do you go on eating binges though you are not hungry?

18. On a scale of 0 to 8, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 8 means total restraint (constantly limiting food intake and never 'giving in' ) what number would you give yourself?

### Perceived Stress Scale

Directions: The questions in this scale ask you about your thoughts and feelings during the last month. In each case, please indicate how often you felt or thought a certain way by choosing the appropriate value from the scale below.

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

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?

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Self Control Scale

Using the scale provided, please indicate how much each of the following statements reflects how you typically are.

Not at all Very much  
1—————2—————3—————4—————5

1. I am good at resisting temptation.
2. I have a hard time breaking bad habits.
3. I am lazy.
4. I say inappropriate things.
5. I never allow myself to lose control.
6. I do certain things that are bad for me, if they are fun.
7. People can count on me to keep on schedule.
8. Getting up in the morning is hard for me.
9. I have trouble saying no.
10. I change my mind fairly often.
11. I blurt out whatever is on my mind.
12. People would describe me as impulsive.
13. I refuse things that are bad for me.
14. I spend too much money.
15. I keep everything neat.
16. I am self-indulgent at times.
17. I wish I had more self-discipline.
18. I am reliable.
19. I get carried away by my feelings.
20. I do many things on the spur of the moment.
21. I don't keep secrets very well.
22. People would say that I have iron self-discipline.
23. I have worked or studied all night at the last minute.
24. I'm not easily discouraged.
25. I'd be better off if I stopped to think before acting.
26. I engage in healthy practices.
27. I eat healthy foods.
28. Pleasure and fun sometimes keep me from getting work done.
29. I have trouble concentrating.
30. I am able to work effectively toward long-term goals.
31. Sometimes, I can't stop myself from doing something, even if I know it is wrong.
32. I often act without thinking through all the alternatives.
33. I lose my temper too easily.
34. I often interrupt people.
35. I sometimes drink or use drugs to excess.
36. I am always on time.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Food Frequency Questionnaire

How often do you eat the following foods?

(1 – Never; 2 – Less than once a week; 3 – Once or twice a week; 4 – Most days; 5 – Once a day;  
6 – More than once a day)

1. potato chips or similar snacks (such as Doritos or Cheetos)
2. salted nuts, corn chips
3. pretzels, rice cakes, unbuttered popcorn
4. chocolate and candy bars
5. cakes, cookies and brownies
6. other desserts such as fruit pies, tarts, flans
7. mousse, or milk pudding, ice cream
8. sorbet, hard candies, jello, frozen yogurt
9. white bread, pasta, white rice
10. whole grain bread, pasta and brown rice
11. potatoes, mashed or baked (not french fries)
12. any fried foods (such as french fries, fried chicken, onion rings)
13. beef, lamb, pork, ham or bacon
14. chicken or turkey
15. sausage, burgers, pizza, hot dogs
16. fish
17. all types of vegetables or salad (not including potatoes)
18. peas, beans, corn, lentils (including baked beans)
19. all types of fruit (canned, frozen or fresh)
20. cheese (such as cheddar, jack, colby, cream cheese, etc)
21. eggs
22. cream (including cream in coffee)
23. breakfast cereals (all types)
24. 100% fruit juice
25. regular soda or sweetened drinks (such as Hawaiian Punch, Nestea, Snapple)
26. diet soft drink (such as Diet Coke, Mountain Dew, Sprite, etc)
27. smoothies (such as Jamba Juice)
28. sweetened coffee drinks (such as Frappuccino, etc)

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### MASQ – SF

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then mark the appropriate choice in the space next to that item. Use the choice that best describes **how much** you have felt or experienced things this way **during the past week, including today**.

	Not at all	A little bit	Moder- ately	Quite a bit	Extre- mely
1. Felt sad	(1)	(2)	(3)	(4)	(5)
2. Startled easily	(1)	(2)	(3)	(4)	(5)
3. Felt cheerful	(1)	(2)	(3)	(4)	(5)
4. Felt afraid	(1)	(2)	(3)	(4)	(5)
5. Felt discouraged	(1)	(2)	(3)	(4)	(5)
6. Hands were shaky	(1)	(2)	(3)	(4)	(5)
7. Felt optimistic	(1)	(2)	(3)	(4)	(5)
8. Had diarrhea	(1)	(2)	(3)	(4)	(5)
9. Felt worthless	(1)	(2)	(3)	(4)	(5)
10. Felt really happy	(1)	(2)	(3)	(4)	(5)
11. Felt nervous	(1)	(2)	(3)	(4)	(5)
12. Felt depressed	(1)	(2)	(3)	(4)	(5)
13. Was short of breath	(1)	(2)	(3)	(4)	(5)
14. Felt uneasy	(1)	(2)	(3)	(4)	(5)
15. Was proud of myself	(1)	(2)	(3)	(4)	(5)
16. Had a lump in my throat	(1)	(2)	(3)	(4)	(5)
17. Felt faint	(1)	(2)	(3)	(4)	(5)
18. Felt unattractive	(1)	(2)	(3)	(4)	(5)
19. Had hot or cold spells	(1)	(2)	(3)	(4)	(5)
20. Had an upset stomach	(1)	(2)	(3)	(4)	(5)
21. Felt like a failure	(1)	(2)	(3)	(4)	(5)
22. Felt like I was having a lot of fun	(1)	(2)	(3)	(4)	(5)
23. Blamed myself for a lot of things	(1)	(2)	(3)	(4)	(5)
24. Hands were cold or sweaty	(1)	(2)	(3)	(4)	(5)
25. Felt withdrawn from other people	(1)	(2)	(3)	(4)	(5)
26. Felt keyed up, “on edge”	(1)	(2)	(3)	(4)	(5)
27. Felt like I had a lot of energy	(1)	(2)	(3)	(4)	(5)
28. Was trembling or shaking	(1)	(2)	(3)	(4)	(5)
29. Felt inferior to others	(1)	(2)	(3)	(4)	(5)
30. Had trouble swallowing	(1)	(2)	(3)	(4)	(5)
31. Felt like crying	(1)	(2)	(3)	(4)	(5)
32. Was unable to relax	(1)	(2)	(3)	(4)	(5)
33. Felt really slowed down	(1)	(2)	(3)	(4)	(5)
34. Was disappointed in myself	(1)	(2)	(3)	(4)	(5)

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

35. Felt nauseous	①	②	③	④	⑤
36. Felt hopeless	①	②	③	④	⑤
37. Felt dizzy or lightheaded	①	②	③	④	⑤
38. Felt sluggish or tired	①	②	③	④	⑤
39. Felt really “up” or lively	①	②	③	④	⑤
40. Had pain in my chest	①	②	③	④	⑤
41. Felt really good	①	②	③	④	⑤
42. Felt like I was choking	①	②	③	④	⑤
43. Looked forward to things with enjoyment	①	②	③	④	⑤
44. Muscles twitched or trembled	①	②	③	④	⑤
45. Felt pessimistic about the future	①	②	③	④	⑤
46. Had a very dry mouth	①	②	③	④	⑤
47. Felt like I had a lot of interesting things to do	①	②	③	④	⑤
48. Was afraid I was going to die	①	②	③	④	⑤
49. Felt like I had accomplished a lot	①	②	③	④	⑤
50. Felt like it took extra effort to get started	①	②	③	④	⑤
51. Felt like nothing was very enjoyable	①	②	③	④	⑤
52. Heart was racing or pounding	①	②	③	④	⑤
53. Felt like I had a lot to look forward to	①	②	③	④	⑤
54. Felt numbness or tingling in my body	①	②	③	④	⑤
55. Felt tense or “high-strung”	①	②	③	④	⑤
56. Felt hopeful about the future	①	②	③	④	⑤
57. Felt like there wasn’t anything interesting or fun to do	①	②	③	④	⑤
58. Seemed to move quickly and easily	①	②	③	④	⑤
59. Muscles were tense or sore	①	②	③	④	⑤
60. Felt really good about myself	①	②	③	④	⑤
61. Thought about death or suicide	①	②	③	④	⑤
62. Had to urinate frequently	①	②	③	④	⑤

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Demographics

Please indicate how you identify your gender: Male, Female \_\_\_\_\_

Please indicate how you identify your race/ethnicity: \_\_\_\_\_

How old are you?

How tall are you? \_\_\_\_\_ Feet; \_\_\_\_\_ Inches

How much do you weigh? \_\_\_\_\_ lbs

Appendix H. Study 2 Pre-task Questionnaire

PANAS-X

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 over the past few weeks.** Use the following scale to record your answers:

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

Very Slightly    A Little    Moderately    Quite a Bit    Extremely  
Or Not at All

_____ 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 with self	_____ shy	_____ drowsy	_____ dissatisfied with self



## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Task Appraisal

Please indicate by writing a number before each statement to indicate how you are feeling right now regarding the task you will complete.

0-----1-----2-----3-----4-----5-----6  
Strongly Disagree Strongly Agree

- \_\_\_\_\_ 1. The task will be very demanding.
- \_\_\_\_\_ 2. I am very uncertain about how I will perform during the task.
- \_\_\_\_\_ 3. The task will take a lot of effort to complete.
- \_\_\_\_\_ 4. The task will be very stressful.
- \_\_\_\_\_ 5. I will perform the task successfully.
- \_\_\_\_\_ 6. I will perform poorly on this task.
- \_\_\_\_\_ 7. I usually perform better in these types of situations than I will on this task.
- \_\_\_\_\_ 8. I will be distressed by my performance.
- \_\_\_\_\_ 9. I will perform about how I expect on the task.
- \_\_\_\_\_ 10. The task will be a positive challenge for me.
- \_\_\_\_\_ 11. The task will be threatening to me.

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

Appendix I. Study 2 Post-task Questionnaire

PANAS-X

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 over the past few weeks.** Use the following scale to record your answers:

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

Very Slightly    A Little    Moderately    Quite a Bit    Extremely  
Or Not at All

_____ 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 with self	_____ shy	_____ drowsy	_____ dissatisfied with self

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Self Esteem Scale

**Instructions:** Please rate the extent to which each of the following statements is true for you right now.

	0 Not at all	1	2	3	4	5	6 Very much
I feel that I am a person of worth, at least on an equal basis with others.							
I feel that I have a good number of qualities.							
All in all, I am inclined to think I am a failure.							
I feel satisfied with the way my body looks right now.							
I feel that others respect and admire me.							
I am able to do things as well as most people.							
I feel that I do not have much to be proud of.							
I am dissatisfied with my weight.							
I feel good about myself.							
I have a positive attitude toward myself.							
On the whole, I am satisfied with myself.							
I feel pleased about my appearance right now.							
I wish I could have more respect for myself.							
I feel useless at times.							
At times I feel I am no good at all.							

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

I feel unattractive.

I feel confident about my abilities.

I feel frustrated or rattled by my performance.

I feel that I am having trouble understanding things I read.

I feel as smart as others.

I feel like I am not doing well.

I feel confident that I understand things.

I feel I have less scholastic ability right now than others.

I am worried about whether I am regarded as a success or a failure.

I feel self-conscious.

I feel displeased with myself.

I am worried about what other people think of me.

I am worried about looking foolish.

I feel inferior to others at this moment.

I feel concerned about the impression I am making.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Task Appraisal

Please indicate by writing a number before each statement to indicate how you are feeling right now regarding the task you just completed.

0-----1-----2-----3-----4-----5-----6  
Strongly Disagree Strongly Agree

- \_\_\_\_\_ 1. The task was very demanding.
- \_\_\_\_\_ 2. I am very uncertain about how I performed during the task.
- \_\_\_\_\_ 3. The task took a lot of effort to complete
- \_\_\_\_\_ 4. The task was very stressful.
- \_\_\_\_\_ 5. I performed the task successfully.
- \_\_\_\_\_ 6. I performed poorly on this task.
- \_\_\_\_\_ 7. I usually perform better in these types of situations.
- \_\_\_\_\_ 8. I am distressed by my performance.
- \_\_\_\_\_ 9. I performed about how I expected on the task.
- \_\_\_\_\_ 10. The task was a positive challenge for me.
- \_\_\_\_\_ 11. The task was threatening to me.

## A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

### Food Frequency Questionnaire (modified)

Please rate the extent to which you desire to eat the following foods **right now**

(1 – Not at all; 2 – A little; 3 – Moderately; 4 – Quite a bit; 5 – Very Much; 6 - Extremely)

1. potato chips or similar snacks (such as Doritos or Cheetos)
2. salted nuts, corn chips
3. pretzels, rice cakes, unbuttered popcorn
4. chocolate and candy bars
5. cakes, cookies and brownies
6. other desserts such as fruit pies, tarts, flans
7. mousse, or milk pudding, ice cream
8. sorbet, hard candies, jello, frozen yogurt
9. white bread, pasta, white rice
10. whole grain bread, pasta and brown rice
11. potatoes, mashed or baked (not french fries)
12. any fried foods (such as french fries, fried chicken, onion rings)
13. beef, lamb, pork, ham or bacon
14. chicken or turkey
15. sausage, burgers, pizza, hot dogs
16. fish
- 17. all types of vegetables or salad (not including potatoes)
18. peas, beans, corn, lentils (including baked beans)
19. all types of fruit (canned, frozen or fresh)
20. cheese (such as cheddar, jack, colby, cream cheese, etc.)
- 21. eggs
22. cream (including cream in coffee)
23. breakfast cereals (all types)
24. 100% fruit juice
25. regular soda or sweetened drinks (such as Hawaiian Punch, Nestea, Snapple)
26. diet soft drink (such as Diet Coke, Mountain Dew, Sprite, etc.)
27. smoothies (such as Jamba Juice)
28. sweetened coffee drinks (such as Frappuccino, etc.)

A COMPREHENSIVE MODEL OF STRESS-INDUCED BINGE EATING

*General Food Cravings Questionnaire-State (G-CFQ-S)*

Indicate the extent to which the following statements are true for you **right now- in this moment**.

0-----1-----2-----3-----4-----5	
Strongly Disagree	Strongly Agree
_____	1. I'm craving tasty food.
_____	2. I have an urge for tasty food.
_____	3. I have an intense desire to eat something tasty.
_____	4. If I ate something, I wouldn't feel so sluggish and lethargic.
_____	5. Satisfying my appetite would make me feel less grouchy and irritable.
_____	6. I would feel more alert if I could satisfy my appetite.
_____	7. If I ate right now, my stomach wouldn't feel as empty.
_____	8. I am hungry.
_____	9. I feel weak because of not eating.
_____	10. My desire to eat something tasty seems overpowering.
_____	11. I know I am going to keep on thinking about tasty food until I actually have it.
_____	12. If I had something tasty to eat, I could not stop eating it.
_____	13. If I were to eat what I'm desiring, I am sure my mood would improve.
_____	14. Eating something tasty would feel wonderful.
_____	15. Eating something tasty would make things just perfect.

**Appendix J. Confederate Interaction Video Script for Stress Task**

Experimenter: “Hello can you two see and hear each other?”

Confederate: “Ya I can hear you.” (seated in chair with mock physio hook up)

*\*Participant answers*

E: “For the purposes of the experiment it’s important that this is your first meeting, do you two know each other?”

*Confederate shakes head*

*\*Participant answers*

E: “OK, we’re going to shut off your video now but you will still be able to hear everything via intercom, alright?”

C: “OK, sounds good!”

*Monitor is turned off and participant begins the speech task*



**BIOGRAPHY OF THE AUTHOR**

Rachael Huff was born in Battle Creek, MI and she was raised predominantly in Michigan. She graduated from Midland High School in 2010 and received her Bachelor's of Science in Psychology from Michigan Technological University in 2014. She joined the Psi Chi National Honors Society in 2013 and has been a member of many professional organizations; some of these include Association for Behavioral and Cognitive Therapies, Anxiety and Depression Association of America, and Maine Psychological Association--for which she was the student representative for the University of Maine's Clinical Psychology program. In addition to serving at the university training clinic for 4 years, she has worked clinically with Native American court systems, the Maine Department of Corrections, and Penobscot Job Corps Center. She has also completed evaluations referred by the Department of Health and Human Services. She completed her internship at Hutchings Psychiatric Center and the Sex Offender Treatment Program at Marcy Correctional Facility in New York. She has worked as a mentor and teacher for first year graduate students in the Clinical Psychology program for 3 years, and has published a chapter with Dr. Sue Righthand, Ph.D. on juvenile sexual assault recidivism, called "Assessing Risks and Needs." She has also published a manuscript with Dr. Righthand examining the predictive validity of tools used to examine sexual assault recidivism risk, titled "Long-term predictive validity of the Juvenile Sex Offender Assessment Protocol II: Research and practice implications." Rachael is a candidate for the Doctorate in Clinical Psychology from The University of Maine in August 2020.