DEPRESSIVE SYMPTOMS AND EATING BEHAVIORS: DO ATYPICAL SYMPTOMS DRIVE ASSOCIATIONS WITH FOOD ATTENTIONAL BIAS, EMOTIONAL EATING, AND EXTERNAL EATING?

by

Aubrey Lynn Shell

A Thesis

Submitted to the Faculty of Purdue University

In Partial Fulfillment of the Requirements for the degree of

Master of Science

Department of Psychological Sciences

Indianapolis, Indiana

May 2019

THE PURDUE UNIVERSITY GRADUATE SCHOOL
STATEMENT OF COMMITTEE APPROVAL

Dr. Jesse C. Stewart, Chair
Department of Psychology
Dr. Adam T. Hirsh
Department of Psychology
Dr. Melissa A. Cyders
Department of Psychology
Dr. John C. Guare
Department of Psychology

Approved by:

Dr. Jesse C. Stewart
Head of the Graduate Program

ACKNOWLEDGMENTS

I am grateful to many people who have supported me throughout the various stages of this thesis. I would like to first thank my committee chair and academic mentor, Dr. Jesse Stewart, who has instilled in me a vision for innovation, mentored me through complex scientific decisions, and bolstered my drive to bring our ideas to completion. Second, many thanks to my committee members, Drs. Adam Hirsh, Melissa Cyders, and John Guare, for their helpful suggestions that point me toward critical thinking and high quality science. Third, I would like to thank my family and friends who have cared for my well-being and have motivated me to stay steady. Finally, I am grateful to my husband, Isaac, for his faithful encouragement through all my endeavors.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Author: Shell, Aubrey Lynn,. MS
Institution: Purdue University
Degree Received: May 2019
Title: Depressive Symptoms and Eating Behaviors: Do Atypical Symptoms Drive Associations With Food Attentional Bias, Emotional Eating, and External Eating?
Committee Chair: Jesse C. Stewart

Depression is an emerging risk factor for obesity; however, it is unclear whether certain depressive symptoms drive this relationship. Recent evidence suggests that atypical major depressive disorder (MDD) – whose key features include the reversed somatic-vegetative symptoms of hyperphagia (increased appetite) and hypersomnia (increased sleep) – is a stronger predictor of future obesity than other MDD subtypes. The present study sought to examine food attentional bias (increased attention to food cues), emotional eating (eating in response to negative emotions), and external eating (eating in response to external food cues) as candidate mechanisms of the depression-to-obesity relationship. This cross-sectional laboratory study hypothesized that total depressive symptom severity, hyperphagia severity, and hypersomnia severity would all be positively associated with measures of food attentional bias, emotional eating, and external eating. Data were collected from a sample of 95 undergraduate students. Depressive symptom severity was measured using the Hopkins Symptom Checklist (SCL-20); two measures of food attentional bias were obtained from eye tracking with high calorie food images: direction bias and duration bias; and emotional eating and external eating were assessed using the Dutch Eating Behavior Questionnaire. Simultaneous regression models (adjusted for age, sex, race/ethnicity, body mass index, and subjective hunger) revealed total depressive symptom severity and hypersomnia severity were not associated with measures of food attentional bias, while hyperphagia severity was negatively associated with direction bias but not

associated with duration bias for high and low calorie food images. Findings related to emotional and external eating are consistent with previous literature: total depressive symptom severity and hyperphagia severity were positively associated with both emotional eating and external eating, and the pattern of results suggests that hyperphagia may be driving relationships between depressive symptoms and these eating behaviors. Hypersomnia severity was not associated with emotional eating and external eating, suggesting this symptom does not play an important role in the relationships between depressive symptoms and these eating behaviors. Future studies should examine prospective associations of hyperphagia severity with food attentional bias, emotional eating, and external eating in larger, more representative samples.

INTRODUCTION

Overview

Because obesity jeopardizes the health of more than one-third of U.S. adults, researchers strive to identify biopsychosocial risk factors for this chronic disease. In addition to traditional risk factors, considerable evidence indicates that depression predicts future obesity. However, it is unclear whether certain depression subtypes or depressive symptoms drive the overall depression-to-obesity relationship. Recent evidence suggests that atypical major depressive disorder (MDD) – whose key features include the reversed somatic-vegetative symptoms of hyperphagia (increased appetite) and hypersomnia (increased sleep) – is a stronger predictor of future obesity than other MDD subtypes. Other research suggests that food attentional bias (increased attention to food cues), emotional eating (eating in response to negative emotions), and external eating (eating in response to external food cues) may be candidate mechanisms underlying the depression-to-obesity relationship. Thus, the present study's objective is to examine associations of total depressive symptom severity, hyperphagia severity, and hypersomnia severity with food attentional bias, emotional eating, and external eating.

Obesity

Definition and Assessment

Obesity is a common condition that has been recognized as a chronic disease by the American Medical Association (AMA) since 2013 (AMA, 2013). It is characterized by excess adiposity resulting from an imbalance of energy intake and energy expenditure (Rosenbaum, Leibel, & Hirsch, 1997). The most widely used measure of adiposity is body mass index (BMI),

which is computed from body weight and height (kg/m^2; Roche, Sievogel, Chumlea, & Webb, 1981). According to the World Health Organization (2000), people with a BMI of 18.5-24.9 kg/m^2 are classified as having normal body mass, 25.0-29.9 kg/m^2 are classified as overweight, and 30.0 kg/m^2 and above are classified as obese. Although BMI is an internationally accepted measure of adiposity due to its accuracy and ease of measurement, it does not distinguish healthy body mass from adipose tissue, and it does not account for demographic variables (Luecken & Gallo, 2007). Other adiposity measures – such as waist circumference, body fat percent, and dual x-ray absorptiometry (Neeland, Poirier, & Després, 2018) – address these limitations to some extent, although they are more difficult to assess and some require special equipment. Thus, BMI was used to assess adiposity in the present study.

Significance

Obesity is highly prevalent, has serious health ramifications, and is associated with high costs. The prevalence of obesity in U.S. adults is 40%, with the highest rates observed in racial/ethnic minorities, particularly non-Hispanic Blacks (47%) and Hispanics/Latinos (47%) (Hales, Carroll, Fryar, & Ogden, 2017). Obesity is a predictor of all-cause mortality (Jensen et al., 2013) and a risk factor for several leading causes of death in the U.S., including cardiovascular disease, some cancers, type 2 diabetes, and dementia (Benjamin et al., 2017; Jensen et al., 2013). People with obesity have annual healthcare costs that are $1,429 (42%) higher than normal-weight people, and the estimated annual cost of obesity, including medical expenses and lost productivity, is $147 billion (Benjamin et al., 2017). Given its prevalence, ramifications, and costs, prevention of obesity is a top public health priority.

Etiology and Traditional Risk Factors

In the most basic terms, obesity results from a chronic imbalance between energy intake and expenditure (Rosenbaum et al., 1997). Energy intake occurs through the consumption of food and beverages; energy expenditure occurs through the execution of bodily functions (e.g., basal metabolism and thermogenesis), lifestyle activities (e.g., walking up stairs), and formal exercise (e.g., running). Extra energy consumed but not expended is converted into triglycerides, which are fat to be stored in adipocytes (Racette, Deusinger, & Deusinger, 2003). A chronic imbalance between energy intake and expenditure can lead to perpetual triglyceride synthesis, resulting in increased adiposity (Barness, Opitz, & Gilbert-Barness, 2007).

Traditional risk factors for obesity are genetic factors, neuroendocrine dysregulation, demographic factors, and lifestyle behaviors (Aronne, Nelinson, & Lillo, 2009). Genetic factors account for 25-40% of individual differences in BMI and may result in obesity when combined with detrimental lifestyle behaviors (Ravussin & Bouchard, 2000). Neuroendocrine dysregulation, especially pertaining to the hormones leptin and insulin, may also contribute to obesity. Leptin, synthesized by adipose tissue, communicates satiety and adiposity signals to the hypothalamus (Barness et al., 2007). People with greater adiposity have higher leptin levels, and excess leptin can result in reduced communication of satiety (Barness et al., 2007), which may promote increased energy intake (Caro, Sinha, Kolaczynski, Zhang, & Considine, 1996). Insulin, secreted by the pancreas following food consumption, promotes energy conversion into triglycerides for storage in adipocytes and inhibits the breakdown of triglycerides for energy consumption (Barness et al., 2007). Thus, excess insulin may contribute to obesity development (Barness et al., 2007).

In addition to genetic and neuroendocrine factors, demographic factors put people at increased risk of obesity (Labarthe, 1998). For example, older adults are at greater risk than younger adults (Ogden, Carroll, Kit, & Flegal, 2014). While women previously had higher risk of obesity, recent trends suggest equal risk among men and women (Flegal, Carroll, Kit, & Ogden, 2012). Furthermore, some racial/ethnic groups have an elevated risk of obesity, with non-Hispanic Blacks and Hispanics/Latinos having the highest prevalence rates (Ogden et al., 2014). Socioeconomic status (SES) is also a potential risk factor for obesity (McLaren, 2007; Sobal & Stunkard, 1989). In highly developed countries like the U.S., people with lower SES are more likely to have a larger body size, particularly among women (McLaren, 2007).

Lifestyle behaviors related to energy intake (e.g., eating behaviors and dietary factors) and expenditure (e.g., physical activity level) also affect obesity risk, and environmental and cultural factors affect these lifestyle behaviors. Eating behaviors have changed over time. According to the United States Department of Agriculture (USDA) and the United States Department of Health and Human Services (USDHHS) (2010), between 1970 and 2008, average daily energy intake increased by 600 calories. In addition, the American diet is replete with saturated fats, trans fats, carbohydrates, and processed sugars, all of which contribute to excess calorie intake (USDA & USDHHS, 2010). Furthermore, in recent decades, food became more readily accessible, and portion sizes increased (USDA & USDHHS, 2010). Experts also propose that decreased physical activity is a risk factor for obesity (USDA & USDHHS, 2010). However, energy expenditure occurs beyond physical activity, and overall energy expenditure has remained relatively stable over time. Hence, it is likely that the dramatic increase in obesity over the last two decades is largely due to increased energy intake (Neeland, Poirier, & Després, 2018; Westerterp & Speakman, 2008).

Depression and Obesity Risk

Increasing evidence indicates that depression is an emerging risk factor for obesity (Luppino et al., 2010). According to the American Psychiatric Association (APA), major depressive disorder (MDD) is characterized by five or more depressive symptoms, one of which must be either depressed mood or loss of interest or pleasure (APA, 2013). Other symptoms are significant weight gain or weight loss, increased or decreased appetite, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feelings of worthlessness or guilt, diminished concentration, and thoughts of death (APA, 2013). The 12-month prevalence of MDD in the U.S. is 7%; this varies by age such that adults aged 18-29 years (the population represented in the present study), have rates of MDD three times higher than that of adults over 60 years (APA, 2013). A recent meta-analysis by Luppino et al. (2010) confirmed a longitudinal relationship between depression and future obesity. In the nine included studies, depression was defined as either a depressive disorder diagnosis or elevated depressive symptoms. The overall effect of depression predicting future obesity (*OR* = 1.58, 95% *CI*: 1.33-1.87) indicated that people with versus without depression had a 58% greater odds of developing obesity over time. A similar but nonsignificant trend for depression predicting future overweight (*OR* = 1.20, 95% *CI*: 0.87-1.66) was also observed.

Atypical Depression, Hyperphagia, Hypersomnia, and Obesity Risk

Recent evidence suggests obesity risk may vary across depressive disorder subtypes, with the highest risk among people with atypical MDD (Chou & Yu, 2013). Atypical MDD is characterized by mood reactivity, hyperphagia (increased appetite) or significant weight gain, hypersomnia (increased sleep), leaden paralysis, and interpersonal rejection sensitivity (APA, 2013). In contrast, melancholic MDD – another common subtype – is characterized by loss of

pleasure or lack of reactivity to pleasurable stimuli, depressed mood that is worse in the morning, lack of appetite or significant weight loss, early morning awakening, psychomotor changes, and guilt (APA, 2013).

Atypical MDD has been found to be more strongly associated with obesity-related outcomes. To illustrate, in a large sample of U.S. adults, Polanka et al. (2017) found that atypical MDD was a stronger predictor of 3-year obesity incidence and 3-year BMI increases than nonatypical MDD, defined as all other MDD subtypes. Similarly, Lasserre et al. (2014) found that atypical MDD, but not the other MDD subtypes, predicted increases in BMI, waist circumference, fat mass, and incident obesity over a 5-year follow-up period. Atypical MDD has also been associated with biological pathways thought to contribute to obesity development, including systemic inflammation and leptin regulation (Penninx, Milaneschi, Lamers, & Vogelzangs, 2013; Scarpace & Zhang, 2009). Penninx and colleagues (2013) found that people with atypical MDD have higher levels of the inflammatory markers interleukin-6, tumor necrosis factor-α, and C-reactive protein (CRP) than those with melancholic MDD or no MDD. Likewise, Hickman, Khambaty, and Stewart (2014) reported that people with atypical MDD have higher levels of CRP than those with nonatypical MDD or no MDD. Milaneschi et al. (2017) observed that atypical MDD was associated with higher leptin levels, while the same association was not detected for nonatypical MDD.

Hyperphagia and hypersomnia are of particular interest here because of their centrality to the atypical MDD subtype and their potential relevance to obesity risk. In combination with a major depressive episode, these two symptoms alone have been used to define atypical MDD (Benazzi, 2002; Polanka et al., 2017). This approach to defining atypical MDD is valid, as it is associated with the remaining diagnostic criteria and is predictive of an atypical MDD diagnosis

using the full criteria (Benazzi, 2002). Hyperphagia and hypersomnia are also plausible candidates for increasing obesity risk over time. Hyperphagia could lead to increased food/beverage consumption and, thus, energy intake, and this symptom has been associated with increased BMI and waist circumference (Lamers, Milaneschi, de Jonge, Giltay, & Penninx, 2017). Similarly, hypersomnia has been associated with being overweight or obese and having a worse course of depressive symptoms (Murru et al., 2019) and could lead to decreased energy expenditure due to increased time sleeping and daytime sleepiness. In addition to these explanations, characteristics of people with atypical MDD are also relevant. First, people with atypical MDD tend to have poorer diet quality than those with melancholic MDD and, consequently, may consume more energy (Rahe et al., 2015). Second, people with atypical MDD have higher rates of disability days than those with other MDD subtypes (Matza, Revicki, Davidson, & Stewart, 2003), which could result in decreased energy expenditure (Polanka et al., 2017). Thus, both the symptoms and characteristics of people with atypical MDD may put them at increased risk of future obesity.

Candidate Mechanisms Underlying the Depression-to-Obesity Relationship

Although depression is an emerging risk factor for obesity, the mechanisms underlying the depression-to-obesity relationship have not yet been identified. Traditional candidate mechanisms of this relationship fall into two broad domains: biological pathways and psychosocial pathways (see Figure 1). Biological pathways include hypothalamic-pituitary-adrenal (HPA) dysregulation, neurotransmitter imbalance, neuropeptide imbalance, and increased inflammation (Bornstein, Schuppenies, Wong, & Licinio, 2006; Markowitz, Friedman, & Arent, 2008; Milaneschi, Simmons, van Rossum, & Penninx, 2018). Psychosocial pathways include increased food consumption, decreased physical activity, psychotropic medication use,

cigarette use, and alcohol use (Markowitz et al., 2008; McCarty et al., 2009; Shi, An, & Meijgaard, 2013). There are also important third factors – genetic factors, socioeconomic status, age, sex, race/ethnicity, disability, and chronic disease – that may predict both depression and obesity and lead to a spurious depression-to-obesity association.

As is noted above, the atypical depressive symptoms of hyperphagia and hypersomnia may increase obesity risk through the psychosocial pathway of increased food consumption. Novel candidate mechanisms that may promote increased food consumption include food attentional bias, emotional eating, and external eating. These candidate mechanisms are the outcome variables of the present study.

Food Attentional Bias

Attentional bias is a cognitive tendency to pay more attention to certain stimuli in the environment than to other stimuli (Faunce, 2002). Such stimuli are often associated with rewards or punishments over time. Researchers have studied attentional bias to various cues among people who use alcohol or other substances, people with eating disorders, and people with mood or anxiety disorders (Copersino et al., 2004; Dobson & Dozois, 2004; Field & Cox, 2008; Williams, Mathews, & MacLeod, 1996). Studying attentional bias to food cues is an area of recent interest (Field et al., 2016; Hawkins et al., 2018; Hou et al., 2011; Kemps & Tiggemann, 2009; Nathan et al., 2012). An example of food attentional bias in daily life is that a person with stronger food attentional bias would be more likely to notice an open bag of chips in a commercial than one with weaker food attentional bias.

Some researchers conceptualize food attentional bias by considering reward processes and their impact on attention to rewarding stimuli. The mesocorticolimbic dopamine system is the site of neurological reward processing: increased levels of dopamine in this brain area lead to

a sense of pleasure and reward (Nathan et al., 2012). The Incentive-Sensitization Theory (IST) of addiction (Robinson & Berridge, 1993) is a useful theoretical framework to explain the relationship between food attentional bias and obesity (Field et al., 2016). When the mesocorticolimbic system is activated, people may pay more attention to highly rewarding foods. In turn, increased attention may lead to more "behavioral wanting" and additional consumption of rewarding foods (Berridge, 2009, p. 547; Nijs & Franken, 2012). This theory is supported by recent findings that women with symptoms of food addiction exhibit higher food attentional bias under a negative mood induction than controls (Frayn, Sears, & von Ranson, 2016).

Three approaches for measuring food attentional bias are modified Stroop tasks, visual dot-probe tasks, and eye tracking paradigms. The original Stroop task involves presenting a series of cards with the name of a color written in a different color ink, and participants are asked to name the color of the ink (Stroop, 1992). In food attentional bias research, the modified Stroop task involves comparing response latencies in color naming for food-related ("cake") versus neutral ("pencil") words (Williams et al., 1996). A delay in color naming for food-related words suggests cognitive interference due to the word content attracting attention (de Ruiter & Brosschot, 1994; Phelan et al., 2011). Therefore, longer delays may be interpreted as being indicative of stronger food attentional bias (Phelan et al., 2011).

The visual dot-probe task involves displaying a series of paired cues (images or words) on a computer screen. The paired cues consist of a food cue (image of a pizza) and a neutral cue (image of a ball) presented simultaneously. A dot-probe then appears in the same location as one of the cues, and participants are asked to identify the location of the dot-probe by pressing a key. Reaction time latencies for dot-probes presented in the same location as food cues are compared to those for neutral cues. Faster reaction times to food cues are interpreted as indicating stronger

food attentional bias (Mogg, Bradley, Hyare, & Lee, 1998). A notable limitation of visual dot-probe tasks is that they may assess different aspects of attentional bias depending on the duration of the image presentation. For example, a short duration (<100 ms) is intended to measure the automatic response but may be measuring where the participant happened to be looking at the time of picture onset (Nijs, Muris, Euser, & Franken, 2010). In contrast, a long duration (>1250 ms) is intended to assess sustained attentional processes (Bradley, Mogg, Wright, & Field, 2003). An intermediate duration may result in participants shifting their gaze, and reaction times may reflect the image at which they happened to be gazing at picture offset (Nijs & Franken, 2012).

A newer approach for measuring attentional bias is eye tracking. Eye tracking equipment records eye movements while content-specific and neutral cues are presented simultaneously on a computer screen. Eye tracking devices use reflections of light – from the computer monitor to the cornea and retina – to determine where the participant is gazing, and the location maps onto the computer screen. In food attentional bias research, eye tracking devices record (1) the image (food or non-food) at which the participant first looked (gaze direction) and (2) the length for which the participant looked at the food or non-food image (gaze duration). Due to eye tracking devices' continuous monitoring capabilities, this assessment approach may be more comprehensive and accurate than dot-probe tasks (Nijs & Franken, 2012). One possible limitation of eye tracking measurement is that it assumes that gazing at an image reflects attention (Hermans et al., 2013).

Recent evidence suggests that depressive symptoms may be associated with food attentional bias. In a study of 89 normal weight or overweight young adults, Hawkins and colleagues (2018) found that the somatic depressive symptom cluster – which includes appetite and sleep disturbance – was associated with greater food attentional bias as measured by reaction

time during a dot-probe task similar to that of the present study. Comparable though nonsignificant trends were observed for gaze direction and duration measured by eye tracking. Neither total depressive symptoms nor the cognitive-affective depressive symptom cluster were related to the food attentional bias measures. Unfortunately, the appetite and sleep items of the measure used in that study (Patient Health Questionnaire-8) are double-barreled and are unable to differentiate between increased/decreased appetite or increased/decreased sleep. Thus, the present study seeks to examine the specific role of the atypical depressive symptoms of hyperphagia and hypersomnia in the depression-to-food attentional bias relationship.

In addition to depression, food attentional bias has been associated with obesity (Nijs & Franken, 2012). First, food attentional bias is higher in people with versus without obesity (Hendrikse et al., 2015). Second, food attentional bias has been linked with increased food cravings and hunger (Field et al., 2016). Experimentally-induced food attentional bias leads to increased food cravings and food consumption, and reducing food attentional bias leads to decreased food cravings (Field et al., 2016). Third, increased food cravings and energy intake have been proposed as candidate explanations for the possible food attentional bias-to-obesity relationship (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Altogether, these findings provide support for a conceptual model in which food attentional bias leads to increased food cravings, hunger, energy intake, and ultimately obesity onset.

Emotional Eating

Emotional eating is eating in response to negative emotions (van Strien, Konttinen, Homberg, Engels, & Winkens, 2016) and is commonly assessed by the Dutch Eating Behavior Questionnaire (DEBQ) (van Strien, Frijters, Bergers, & Defares, 1986). Evidence indicates that depression is positively associated with emotional eating (Antoniou, Bongers, & Jansen, 2017;

Lazarevich, Irigoyen Camacho, Velazquez-Alva, & Zepeda Zepeda, 2016; Ouwens, van Strien, & van Leeuwe, 2009). History, severity, and duration of MDD all predict increased emotional eating (Paans et al., 2018a). Of particular relevance, the depressive symptoms of increased appetite and increased weight gain are more strongly associated with emotional eating than the other depressive symptoms (Paans et al., 2018a). Depressive symptom severity is positively associated with emotional eating among patients with binge eating disorder (Masheb & Grilo, 2006) and among bariatric surgery candidates (Sevinçer et al., 2017).

Evidence supports a plausible association between emotional eating and obesity (van Strien, Herman, & Verheijden, 2009). Theoretically, a person high in emotional eating experiencing the same negative emotions as a person low in emotional eating would be more likely to eat in response to these emotions, likely leading to increased food consumption over time. Despite limited evidence for emotional eating as a predictor of future obesity, higher levels of emotional eating are associated with weight gain (van Strien et al., 2016) and with less successful weight loss (Braden et al., 2016).

Additional evidence suggests emotional eating may be a mechanism in the relationship between depression and increased adiposity. In a five-year longitudinal study of Dutch parents, van Strien et al. (2016) found that emotional eating mediated the relationship between depression and BMI change over time in mothers, but had no significant effect in fathers. Among the mothers, depression was positively associated with emotional eating, and emotional eating was positively associated with greater increases in BMI. Among the fathers, depression was positively associated with emotional eating, although emotional eating was not associated with changes in BMI. Examining results of cross-sectional studies, other researchers concur that

emotional eating may mediate the relationship between depression and BMI (Antoniou et al., 2017; Lazarevich et al., 2016).

External Eating

External eating is the tendency to eat in response to external food cues (Hou et al., 2011) and is often assessed by the Dutch Eating Behavior Questionnaire (DEBQ) (van Strien et al., 1986). Evidence indicates that depression is associated with external eating (Paans et al., 2018a). Current and remitted MDD are both associated with external eating, as is duration of depressive symptoms (Paans et al., 2018a). Of particular relevance, the depressive symptoms of increased appetite and weight gain were stronger predictors of external eating than the other symptoms (Paans et al., 2018b). Depressive symptom severity correlates with increased external eating among bariatric surgery candidates (Sevinçer et al., 2017), although this association is inconsistent among adult women (Ouwens et al., 2009). Depressive symptom severity also predicts external eating among Korean adults at 3-year follow-up (Song, Lee, & Sung, 2017). Additionally, external eating statistically mediates the relationship between depressive symptom severity and consumption of fast food and savory snacks (Paans et al., 2019).

External eating is also associated with obesity (Vainik, Dagher, Dubé, & Fellows, 2013). Theoretically, a person high in external eating encountering the same environmental food cues as a person low in external eating would be more likely to eat in response to these cues, likely leading to increased energy intake. External eating has been associated with increased food consumption (Paans et al., 2019; van Strien, Herman, & Anschutz, 2012), although the findings have been inconsistent (Jansen et al., 2011). Despite limited evidence for external eating as a predictor of obesity, a plausible connection exists between external eating and future obesity.

THE PRESENT STUDY

This study's objective is to examine associations of total depressive symptom severity, hyperphagia severity, and hypersomnia severity with food attentional bias (eye tracking direction bias and duration bias), emotional eating (DEBQ), and external eating (DEBQ) in a sample of young adults. The associations of depressive symptoms (total depressive symptom severity, hyperphagia severity, and hypersomnia severity) to these candidate mechanisms of the depression-to-obesity relationship remain unknown. Knowledge of these associations could increase understanding of the obesity-promoting symptoms of depression and the mechanisms through which these depressive symptoms contribute to obesity onset. The present study's emphasis on reversed somatic-vegetative symptoms may address a limitation of Hawkins and colleagues' (2018) study, in which hyperphagia and hypersomnia were not separately measured. To achieve my objective, I tested the following nine hypotheses in a cross-sectional, laboratory study:

Primary Hypotheses

Hypothesis 1: Total depressive symptom severity is positively associated with the two food attentional bias measures.

Hypothesis 2: Hyperphagia severity is positively associated with the two food attentional bias measures.

Hypothesis 3: Hypersomnia severity is positively associated with the two food attentional bias measures.

Secondary Hypotheses

Hypothesis 4: Total depressive symptom severity is positively associated with emotional eating.

Hypothesis 5: Hyperphagia severity is positively associated with emotional eating.

Hypothesis 6: Hypersomnia severity is positively associated with emotional eating.

Hypothesis 7: Total depressive symptom severity is positively associated with external eating.

Hypothesis 8: Hyperphagia severity is positively associated with external eating.

Hypothesis 9: Hypersomnia severity is positively associated with external eating.

METHODS

Participants

This study was approved by the Indiana University-Purdue University Indianapolis (IUPUI) Institutional Review Board. Participants are 95 undergraduate students at IUPUI who sought research credit for a psychology course. An undergraduate sample is appropriate for the present study, given that young adulthood is a time of increasing autonomy regarding food choices and eating behaviors (Han & Lee, 2017). In addition, young adults may develop disordered eating in response to the stress of separation from relatives or adjustment to their new autonomy in college (Han & Lee, 2017). Consistent with this point, eating behaviors are a concern for many undergraduate students, as eating behaviors were a topic of therapy for 14% of college students receiving mental health services in the 2015-2016 academic year (Center for Collegiate Mental Health, 2017).

Students elected to participate through SONA, the Department of Psychology's undergraduate research recruitment website. The SONA advertisement stated: "The purpose of this study is to understand the relationships between psychosocial factors (e.g., cognitive and emotional factors) and eating behaviors." I withheld the details of the hypotheses due to concern that knowledge may influence participant responses on the surveys and the food attentional bias assessment. Participants were provided debriefing information regarding the purpose of the study at the end of their laboratory session.

Students were eligible if they were 18 years or older, not pregnant at the time of their study visit, and had a BMI in the healthy weight (18.5-24.9 kg/m^2) or overweight (25.0-29.9 kg/m^2) categories. I did not recruit pregnant women because pregnancy has been associated with

changes in depressive symptoms (McMahon, Arms-Chavez, Harper, & LoBello, 2017), food cravings (Belzer, Smulian, Lu, & Tepper, 2010), and eating behaviors (Pope, Skinner, & Carruth, 1992). I also did not recruit students who had a BMI in the obese (≥ 30 kg/m^2) or underweight (< 18.5 kg/m^2) categories because the depression-to-food attentional bias relationship may not be present at either end of the BMI spectrum. With the development of obesity, eating behaviors may be tied to negative experiences, leading to avoidance of certain foods (Kenardy, Arnow, & Agras, 1996; Werthmann et al., 2011). Similarly, those in the underweight category may have anorexia nervosa or symptoms of this disorder, which include food-avoidant behaviors (Hudson, Hiripi, Pope, & Kessler, 2007). Seven students completed the study session but did not meet BMI eligibility criteria; I excluded their data prior to analysis.

A total of 103 participants met eligibility criteria for the present study. I excluded participants who did not have sufficient eye tracking data ($n = 3$) and participants missing data on key variables ($n = 5$), leaving a final sample of 95.

Measures

Please see the appendix for the self-report measures.

Depressive Symptoms

Total depressive symptom severity was measured using the Hopkins Symptom Checklist-20 (SCL-20; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). The SCL-20 is a 20-item self-report questionnaire. Participants were asked to rate their level of distress regarding each depressive symptom over the past week using a 0-4 scale, with response options ranging from "not at all" to "extremely." The SCL-20 total score – computed by averaging the responses to the items – ranges from 0-4, with higher scores indicating more severe depressive symptoms

(Derogatis et al., 1974). In an evaluation of the reliability and validity of depressive symptom measures, Johns et al. (2013) observed good internal consistency ($\alpha = 0.85$) and convergent validity (strong correlations with other validated depression measures) for the SCL-20.

The SCL-20 is well suited for assessing the atypical depressive symptoms of interest. It is beneficial for detecting hyperphagia because there are two appetite items ("poor appetite" and "overeating"), unlike other common depression measures, which only assess one direction of appetite disturbance (Kroenke, Spitzer, & Williams, 2001; Radloff, 1977). Additionally, both directions of sleep disturbance can be assessed. The SCL-20 sleep items ask participants to rate their distress about having "trouble falling asleep," "sleep that is restless or disturbed," and "awakening in the early morning;" I added a supplemental sleep question (Item 21: "sleeping too much") that Dr. Stewart is using in an ongoing NIH-funded clinical trial. The SCL-20 total score is my measure of total depressive symptom severity. Hyperphagia severity and hypersomnia severity were computed by the participant's responses to the "overeating" item and the "sleeping too much" item, respectively. Three other SCL-20 total scores were computed for models that also included the hyperphagia or hypersomnia variable. The SCL-19 score was computed by averaging the SCL-20 item responses excluding the "overeating" item. The SCL-20 total minus appetite score was computed by averaging the SCL-20 item responses excluding the two appetite items, and the SCL-20 total minus sleep score was computed by averaging the SCL-20 item responses excluding the three insomnia items.

Food Attentional Bias

Food attentional bias was measured using a visual dot-probe task paired with an eye tracking assessment. I used a computerized task modified from Castellanos et al. (2009). A fixation cross was presented in the middle of the computer screen for 1,000 ms. Next, a pair of

images was presented, one on the left and one on the right, for 2,000 ms. A total of 50 image pairs were presented. Forty of the image pairs were experimental images, with each pair consisting of a food image and a non-food image. Of these 40 experimental food images, 20 images were of high calorie food (e.g., pastries or French fries) and 20 images were of low calorie food (e.g., fruits or vegetables; see Figures 2 and 3, respectively, for samples). Image pairs were matched for color, shape, and size. The remaining 10 image pairs were control images, which were nature scenes matched for color, shape, and size (see Figure 4 for samples). These control images were randomly dispersed with the food- and non-food image pairs to vary the task. All image pairs were presented twice, each image appearing once on the left and once on the right, for a total of 100 trials.

After the presentation of each image pair, a dot-probe appeared in the location of one of the two images and remained on the screen until the participants pressed either a "1" or "2" on the keyboard. Participants were instructed to respond quickly and accurately to these dot-probes by pressing a "1" for dots that were up and down (":") or a "2" for dots that were side-by-side (".."). Although participants were aware that their eye gazes were being tracked, they were told that their goal was to correctly identify the dot-probe. The dot-probes appeared equally often in the spatial location of the food and non-food images. There was an equal number of each type of dot-probe. I decided not to assess reaction time because I have a more direct measure of where the participants looked the longest (i.e., duration bias). Despite omitting the reaction time calculation, I retained the dot-probe task so the participants would have a context within which the eye tracking paradigm could occur.

I collected eye tracking data using a Tobii Pro X3-120 Eye Tracker. This eye tracker captures gaze data at 120 Hz. To ensure proper recording, each participant's eye movements

were calibrated using a standardized 5-point procedure recommended by the manufacturer (Tobii, 2012). Using Tobii Studio 3.X™ software, I identified areas of interest for each food and non-food image within the dot-probe task. For each experimental pair, Tobii Studio 3.X™ determined the number and duration of gaze fixations (gazes lasting > 0.1 ms) within the food and non-food image areas of interest.

I calculated two indices of food attentional bias: direction bias and duration bias. These two dependent variables were calculated for the high calorie and low calorie image pairs separately. Direction bias was calculated as the proportion of the experimental image presentations in which the participant's first gaze in an area of interest was directed at the food image. Values further above 0.5 indicate stronger direction bias for food images over non-food images. Duration bias was calculated as the proportion of time that the participant looked at food images divided by the total amount of time spent looking at either the food or non-food images across all experimental trials. Values further above 0.5 indicate stronger duration bias.

Emotional Eating and External Eating

Emotional eating and external eating were measured using the Dutch Eating Behavior Questionnaire (DEBQ) (van Strien et al., 1986), which has been translated to English (Wardle, 1987). This 33-item self-report questionnaire asks participants to rate their general frequency of particular eating behaviors (e.g., "Do you have a desire to eat when somebody lets you down?" and "If you see others eating, do you also want to eat?") on a 1-5 scale, with response options ranging from "never" to "very often." The questionnaire has a three-factor structure consisting of restrained eating, emotional eating, and external eating subscales. For the purposes of the present study, I did not examine associations of depressive symptoms with restrained eating. The subscales of the DEBQ demonstrate internal consistency, with a Cronbach's α of 0.94 for

emotional eating and 0.80 for external eating (van Strien et al., 1986). The emotional eating subscale demonstrates construct validity (van Strien, Herman, Anschutz, Engels, & de Weerth, 2012). In research attempting to establish the concurrent validity of the emotional eating subscale with food consumption in laboratory and naturalistic settings, findings have been mixed (Domoff, Meers, Koball, & Musher-Eizenman, 2014). It appears that type of food, type of mood induction, and sample characteristics may influence this relationship (Domoff et al., 2014). Likewise, the external eating subscale demonstrates construct validity with other self-report measures of food cue reactivity, although its prediction of food intake has produced mixed findings (Jansen et al., 2011; van Strien et al., 2012). Mean scores for emotional eating (13 items) and external eating (10 items) were computed, and higher scores indicate greater levels of the construct.

Covariates

I measured several covariates – i.e., demographic factors, BMI, physical activity, alcohol and cannabis use, state hunger, prescription antidepressant medication use, and food-related conditions. Covariates were coded into continuous and dummy-coded variables.

Data regarding age, sex, race/ethnicity, and residential status were collected using standard questions. Age was computed as the time in years from the participant's date of birth to the date of the study visit; the age variable was winsorized to the 5^{th} and 95^{th} percentiles because the original distribution was positively skewed (skewness = 4.14, kurtosis = 20.41). Sex was coded as 0 = male and 1 = female. Due to low frequency counts for individual racial/ethnic minority groups, race/ethnicity was coded as 0 = non-Hispanic White and 1 = non-White. I assessed residential status due to the possibility that this factor could affect participants' access to healthy foods and their autonomy in making food choices. Residential status was coded as 0 =

living at home with parents/family and 1 = not living at home with parents/family. Height and weight were measured with a standard medical scale, and BMI was calculated by dividing weight by height squared (kg/m^2).

Self-reported physical activity was measured using the International Physical Activity Questionnaire (IPAQ), a quantity-frequency measure of weekly physical activity in work and recreational activities (Craig et al., 2003). Participants reported the number of days in the past week and the number of hours per day that they engaged in vigorous activity, moderate activity, walking, and sedentary activity. For each activity level, a metabolic equivalent (MET) constant was multiplied by the time spent in that activity level times the number of days per week engaged in that activity level. Values for each activity level were summed to create an overall MET value, which is an estimate of metabolic intensity for the past week. In a multisite validation study, the IPAQ was found to have moderate test-retest reliability (Craig et al., 2003). The IPAQ demonstrated criterion validity, with fair to moderate agreement between the IPAQ and an accelerometer measure of physical activity (Craig et al., 2003). The IPAQ overall MET value is my measure of physical activity level.

I assessed alcohol use and cannabis use because they have been associated with depression (Foulds, Adamson, Boden, Williman, & Mulder, 2015; Lev-Ran et al., 2014), food cravings (Han, Lyool, Sung, Lee, & Renshaw, 2008; Hutchison, McGeary, Smolen, & Wooden, 2001; Sobik, Hutchison, & Craighead, 2005), and disordered eating (Sinha & O'Malley, 2000), and they could operate as confounders, leading to spurious relationships between my independent and dependent variables. Alcohol use was measured using the Alcohol Use Disorder Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1989). This 10-item questionnaire assesses the quantity, frequency, and consequences of alcohol consumption. Items

are on a 0-4 rating scale ranging from "never" to "daily or almost daily." Total scores range from 0-40, with scores at 8 or above indicating hazardous alcohol use (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). The AUDIT demonstrates internal consistency in college samples (Cronbach's α = .80) as well as high test-retest reliability (r=.86) (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Fleming, Barry, & MacDonald, 1991). The AUDIT total score is my measure of past-year alcohol use. Additionally, cannabis use was assessed with the cannabis item from the National Institute on Drug Abuse (NIDA) Quick Screen (NIDA, 2012). Of note, I changed the timeframe from "in your lifetime" to "in the past year" to align with that of the AUDIT. Past-year cannabis use was coded as 0 = no and 1 = yes based on responses to the NIDA Quick Screen.

Because evidence supports a positive relationship between state hunger and food attentional bias (Castellanos et al., 2009; Mogg et al., 1998; Placanica, Faunce, & Soames Job, 2002), two 0-100 visual analogue scales (VAS) were administered to assess state hunger prior to the food attentional bias assessment. For both questions ("How hungry do you feel?" and "How strong is your urge to eat?"), participants rated their level of agreement along a line, ranging from "not very" to "very much." Evidence supports the use of a VAS in the assessment of state appetite and hunger, with correlations to subsequent energy intake ranging from 0.50-0.53 (Flint, Raben, Blundell, & Astrup, 2000). I computed a mean VAS score by averaging the values for the two state hunger questions.

Finally, participants were asked about their medications and food-related medical conditions. Participants who were currently taking prescription medications were asked to record the name and dosage of their medications. Participants were also asked if they had any food-related allergies or conditions. These data allowed me to conduct exploratory analyses examining

the influence of antidepressant medication use and food conditions on any observed relationships between my independent and dependent variables. Current antidepressant use was coded as 0 = no and 1 = yes, and food conditions were coded as 0 = absent and 1 = present.

Procedure

On the SONA advertisement, participants were informed that they needed to eat a full meal within 3 hours preceding their laboratory session. This restriction was in place to restrict variability in state hunger and minimize its influence on the assessments. Participants who did not adhere to this restriction had their session rescheduled.

Participants completed a 1-hour laboratory session consisting of informed consent, VAS items, an eye tracking computer task, body measurements, a battery of self-report questionnaires, and a debriefing statement. At the start of the session, participants provided written informed consent to all study procedures and were given a copy of the signed consent form to keep. Participants then completed the two VAS items to assess state hunger. Next, participants completed the food attentional bias assessment. Participants sat at a computer desk while the research assistant explained the order of events for the task and calibrated the eye tracking camera. After the research assistant calibrated the eye tracking camera (see Food Attentional Bias section for details), participants practiced the visual dot-probe task; this was identical to the experimental task, except that the stimuli were shapes instead of food and non-food images. The research assistant observed participants during the practice task, checking for accurate responses and providing a brief reminder of the task instructions if participants responded inaccurately. The research assistant then left the room while participants completed the visual dot-probe task.

After completing the food attentional bias assessment, participants had their height and weight measured and completed a battery of self-report questionnaires on SurveyMonkey, a

password-protected web-based research tool. The battery included the following measures: demographic questions, substance use and prescription medications, SCL-20 with supplemental sleep question, DEBQ, IPAQ, and AUDIT. Finally, the research assistant provided the participant with debriefing information regarding the study purpose.

Data Analytic Plan

Data Cleaning and Reduction

I ran frequencies on all variables to assess for out-of-range values on raw and computed scores. If out-of-range values were found, I checked for data entry errors (raw scores) and computational errors (computed scores). I assessed the distribution of all variables. For continuous variables, I assessed for normality using skewness and kurtosis estimates, with skewness >3.0 or kurtosis >10 indicating non-normality (Kline, 2005). For categorical variables, I checked to ensure that they were proportionately distributed. If an outlier category appeared (i.e., a category that is very small compared to the others), I considered collapsing it with another category.

I assessed the quality and usability of eye tracking data using two methods. First, I calculated the accuracy of participants' key presses in response to dot-probes and excluded those with <70% accuracy (n = 0). Second, I computed the proportion of usable trial scores across each participant and excluded those who looked at either the food or non-food areas of interest in <50% of the high and low calorie food trials (n = 3).

Tests of Hypotheses

I tested my nine hypotheses by running multiple linear regression models. In each model, I adjusted for age, sex, race/ethnicity, BMI, and state hunger. In the models where hyperphagia

or hypersomnia was an individual predictor, I further adjusted for SCL-20 total minus appetite score and SCL-20 total minus sleep score, respectively. In each model, I assessed for multicollinearity across the predictor variables by examining variance inflation factors (VIFs). All VIFs were <2.0 other than the models adjusted for residential status (VIF = 2.04 for the SCL-19 variable). Overall, this suggests that multicollinearity is not of concern among the variables despite the inclusion of multiple depressive symptom variables in the same models.

To test each of the primary hypotheses (Hypotheses 1-3), I ran two simultaneous multiple regression analyses, one with high calorie direction bias as the outcome variable and one with high calorie duration bias as the outcome variable. Specifically, I examined total depressive symptom severity (SCL-19), hyperphagia severity, and hypersomnia severity as simultaneous predictors of direction bias and duration bias computed from the high calorie food trials only. To further investigate the primary hypotheses, I also ran six individual regression analyses, two per hypothesis. Specifically, in separate models, I examined total depressive symptom severity, hyperphagia severity, and hypersomnia severity as predictors of high calorie direction and duration bias. For comparison, I reran the two simultaneous models and six individual models with direction bias and duration bias computed from the low calorie food trials only. To test the secondary hypotheses, I ran a parallel set of models (first simultaneous and then individual) with emotional eating (Hypotheses 4-6) and external eating (Hypotheses 7-9) as the outcome variables.

Finally, I reran all of the simultaneous models described above, further adjusting for residential status, physical activity, alcohol use, cannabis use, antidepressant use, and food conditions one at a time to assess whether any of these potential confounders attenuated the relationships of interest.

Power Analysis

After data collection, I conducted a power analysis using *G*Power* to determine the effect size the analyses were powered to detect. Using multiple linear regression (fixed model, R^2 increase) as my statistical test, I entered the number of predictors (3 tested, 8 total), as well as the desired alpha (0.05), power (0.80), and sample size (95). According to this analysis, the study was powered to detect an f^2 effect size as small as 0.12. This is considered a small-to-medium effect size (Cohen, 1988).

RESULTS

Participant Characteristics

For the final sample of 95 participants, descriptive statistics for the demographic, depressive symptom, food attentional bias, and eating behavior variables are presented in Table 1. Age ranged from 18 to 44 years, 81% were female, and 74% were White. Average BMI was in the normal weight category and ranged from 18.6 to 29.7 kg/m^2. Average subjective hunger was mild, and most participants lived independent from their families. Average physical activity was high, with great variability. Average past-year alcohol use fell in the low risk for harm category on the AUDIT (Babor et al., 2001), and 25% of participants reported using cannabis in the past year. Fourteen percent of participants reported current antidepressant use, and 10% reported having a food condition.

Average depressive symptom severity was mild (SCL-20: $M = 0.85$, $SD = 0.70$, range: 0.00-3.60), and 8.4% of participants scored above 2.0, which is indicative of clinically significant depression (Katon et al., 1995; Walker et al., 2000). I expected this mean and distribution in a sample of undergraduate students. Similarly, hyperphagia ($M = 0.68$, $SD = 1.10$, range: 0.00-4.00) and hypersomnia ($M = 0.83$, $SD = 1.10$, range: 0.00-4.00) were generally mild with good variability. On average, high calorie direction bias ($M = 0.41$, $SD = 0.10$, range: 0.08-0.70) and duration bias ($M = 0.49$, $SD = 0.07$, range: 0.21-0.67) fell below 0.50, indicating a slight bias away from food stimuli. These means, especially for direction bias, were lower than I expected considering the Incentive-Sensitization Theory that humans are inclined to notice cues that activate reward pathways in the brain (Berridge, 2009; Nijs & Franken, 2012). Nonetheless, it is worth noting that these variables had reasonable variability. Low calorie direction and

duration bias had similar descriptive statistics, again reflecting a slight bias away from food stimuli. On average, emotional eating (M = 2.08, SD = 0.90, range: 1.00-4.46) and external eating (M = 3.05, SD = 0.76, range: 1.20-4.90) were moderate. These mean scores are comparable to those reported in Castellanos et al. (2009) and Nijs et al. (2010), both of which also examined normal weight samples.

Correlations among measures of food attentional bias, eating behaviors, and depressive symptom severity are presented in Table 2. Both hyperphagia and hypersomnia were positively correlated with the SCL-19 (r = .55, p < .01 and r = .47, p < .01, respectively), but hyperphagia and hypersomnia did not correlate with one another (r = .13, p = 0.21), indicating that these two symptoms may not be as related to one another as the construct of atypical depression suggests. High calorie direction bias and duration bias did not correlate (r = .01, p = 0.93), demonstrating that there was not a relationship between the image participants looked at first and the image they looked at longest. This correlation is inconsistent with Nijs et al. (2010) and Hawkins et al. (2018), who both found a positive correlation between direction and duration bias (r = .54 and .56, respectively). Low calorie direction bias and duration bias also did not correlate (r = .14, p = 0.19). However, high calorie direction bias strongly correlated with low calorie direction bias (r = .72, p < .01), and the same was true for the duration bias variables (r = .63, p < .01). Total depressive symptom severity measured by the SCL-19 (the average of SCL-20 items excluding the hyperphagia item) was not correlated with high calorie direction or duration bias (rs = -.12 and .17, respectively), nor low calorie direction or duration bias (rs = .00 and .00, respectively). Hyperphagia was not significantly correlated with high calorie direction bias, low calorie direction bias, or low calorie duration bias (rs = -.17, -.12, and .17, respectively), but was

positively correlated with high calorie duration bias ($r = .22$, $p < .05$). Hypersomnia was not correlated with any of the food attentional bias measures (see Table 2).

Emotional eating was moderately correlated with external eating ($r = .47$, $p < .01$), which is to be expected based on past literature (Bongers & Jansen, 2016; van Strien, Schippers, & Cox, 1995). However, there was only one significant correlation between the four food attentional bias measures and self-reported eating behavior; namely, high calorie duration bias was positively correlated with external eating ($r = .31$, $p < .01$). Total depressive symptom severity measured by SCL-19 was positively correlated with emotional eating ($r = .30$, $p < .01$) and external eating ($r = .23$, $p < .05$), as was hyperphagia ($r = .54$, $p < .01$ and $r = .36$, $p < .01$, respectively). Hypersomnia was not correlated with emotional eating or external eating (rs = .03 and .03, respectively). Of the covariates, age correlated with hyperphagia ($r = .22$, $p < .05$), BMI ($r = .27$, $p < .05$), and external eating ($r = .24$, $p < .05$), and subjective hunger correlated with external eating ($r = .28$, $p < .01$). All other correlations involving the covariates were not significant (*rs* range from -0.20 to 0.19).

Associations of Depressive Symptoms with Food Attentional Bias

Total Depressive Symptom Severity and High Calorie Direction and Duration Bias (Hypothesis 1)

As is shown in Table 3, the primary simultaneous regression models (adjusted for age, sex, race/ethnicity, BMI, and subjective hunger) showed that total depressive symptom severity (SCL-19) was not significantly associated with direction bias ($\beta = 0.06$, $p = 0.69$) or duration bias ($\beta = 0.01$, $p = 0.43$) for high calorie food images. In the individual regression models (see Table 4), the relationships between total depressive symptom severity (SCL-20) and high calorie direction bias and duration bias remained nonsignificant ($\beta = -0.13$, $p = 0.21$ and $\beta = 0.18$, $p =$

0.08, respectively). It is noteworthy that inclusion of the hyperphagia item in the SCL-20 total pulled both associations toward significance, albeit in opposite directions. Adjusting the simultaneous models for additional covariates (residential status, physical activity, alcohol use, cannabis use, antidepressant use, and food conditions; see Table 5) one at a time also did not meaningfully change the associations between total depressive symptom severity (SCL-19) and high calorie direction bias or duration bias (all *p*s ≥ 0.49 and ≥ 0.29, respectively).

Hyperphagia Severity and High Calorie Direction and Duration Bias (Hypothesis 2)

The primary simultaneous regression models (see Table 3) showed that hyperphagia severity was negatively associated with high calorie direction bias (β = -0.26, p = 0.05) and was not significantly associated with duration bias (β = 0.10, p = 0.44). In the individual regression models (see Table 4), the relationship between hyperphagia severity and high calorie direction bias and duration bias did not change (β = -0.26, p = 0.05 and β = 0.10, p = 0.46, respectively). Adjusting the simultaneous model for additional covariates one at a time did not meaningfully change the association between hyperphagia severity and high calorie direction bias (all βs ≤ -0.22), although it did render some relationships nonsignificant (see Table 5). Adjusting for these covariates also did not meaningfully change the association between hyperphagia severity and high calorie duration bias (all *p*s > 0.40).

Hypersomnia Severity and High Calorie Direction and Duration Bias (Hypothesis 3)

As is shown in Table 3, the primary simultaneous regression models showed that hypersomnia severity was not significantly associated with high calorie direction bias (β = -0.08, p = 0.54) or duration bias (β = 0.02, p = 0.86). In the individual regression models (see Table 4), the relationship between hypersomnia severity and high calorie direction bias and duration bias

did not change (β = -0.03, p = 0.82 and β = 0.02, p = 0.90, respectively). Adjusting the simultaneous model for additional covariates one at a time also did not meaningfully change the relationships between hypersomnia severity and high calorie direction bias or duration bias (all ps > 0.50 and ≥ 0.75, respectively; see Table 5).

Depressive Symptoms and Low Calorie Direction and Duration Bias

Re-running the primary simultaneous regression models with direction bias and duration bias for low calorie food images as outcomes did not yield different results (see Table 6). Neither total depressive symptom severity nor hypersomnia were associated with low calorie direction bias (ps = 0.26 and 0.86, respectively); hyperphagia had a significant negative association with low calorie direction bias (p = 0.02). None of the depressive symptom variables was significantly associated with low calorie duration bias (all ps > 0.22). Furthermore, these relationships were similar in the individual regression models (see Table 7). As is shown in Table 8, adjusting for additional covariates one at a time did not meaningfully change the association between hyperphagia severity and low calorie direction bias (all βs ≤ -0.25), although it did render one relationship nonsignificant. The associations of total depressive symptom severity and hypersomnia severity with low calorie direction bias remained nonsignificant after adjusting for these covariates (all ps ≥ 0.18 and ≥ 0.80, respectively). Similarly, these adjustments did not meaningfully change the associations of total depressive symptom severity, hyperphagia severity, and hypersomnia severity with low calorie duration bias (all ps ≥ 0.75, ≥ 0.25, and ≥ 0.18, respectively).

Associations of Depressive Symptoms with Emotional Eating and External Eating

Total Depressive Symptom Severity and Emotional Eating and External Eating (Hypotheses 4 and 7)

As is shown in Table 9, the primary simultaneous regression models showed that total depressive symptom severity (SCL-19) was not significantly associated with emotional eating ($\beta = 0.08$, $p = 0.53$) or external eating ($\beta = 0.14$, $p = 0.30$). In the individual regression models (see Table 10), the relationship between total depressive symptom severity (SCL-20) and emotional eating was significant ($\beta = 0.32$, $p = 0.001$), as was the relationship between total depressive symptom severity and external eating ($\beta = 0.25$, $p = 0.01$). Of note, inclusion of the hyperphagia item in the SCL-20 total made both associations statistically significant. Adjusting the simultaneous models for additional covariates (see Table 11) one at a time did not meaningfully change the associations of total depressive symptom severity (SCL-19) with emotional eating or external eating (all ps ≥ 0.41 and ≥ 0.25, respectively).

Hyperphagia Severity and Emotional Eating and External Eating (Hypotheses 5 and 8)

The primary simultaneous regression models showed that hyperphagia severity had a significant positive association with emotional eating ($\beta = 0.47$, $p < 0.001$; see Table 9), but was not significantly associated with external eating, despite a potentially meaningful effect size ($\beta = 0.21$, $p = 0.09$). In the individual regression models (see Table 10), the relationship between hyperphagia severity and emotional eating and external eating did not change ($\beta = 0.46$, $p < 0.001$ and $\beta = 0.21$, $p = 0.09$, respectively). Adjusting the simultaneous model for additional covariates (see Table 11) one at a time also did not meaningfully change the associations between hyperphagia severity and emotional eating or external eating (all ps < 0.001 and ≥ 0.07, respectively).

Hypersomnia Severity and Emotional Eating and External Eating (Hypotheses 6 and 9)

As is shown in Table 9, the primary simultaneous regression models illustrate that hypersomnia severity was not significantly associated with emotional eating (β = -0.06, p = 0.57) or external eating (β = -0.03, p = 0.77). In the individual regression models (see Table 10), the associations of hypersomnia severity with emotional eating and external eating were similar (β = -0.12, p = 0.27 and β = -0.06, p = 0.62, respectively). Adjusting the simultaneous model for additional covariates (see Table 11) one at a time also did not meaningfully change the associations of hypersomnia severity with emotional eating and external eating (all ps $\geq$ 0.32 and $\geq$ 0.46, respectively).

DISCUSSION

Summary of Results

The objective of the present study was to examine associations of total depressive symptom severity, hyperphagia severity, and hypersomnia severity with measures of food attentional bias (eye tracking direction bias and duration bias), emotional eating (DEBQ), and external eating (DEBQ). Hypothesis 1, that total depressive symptom severity is positively associated with high calorie direction bias and duration bias, was not supported. Hypothesis 2, that hyperphagia severity is positively associated with high calorie direction bias and duration bias, was also not supported. Rather, regression models revealed that hyperphagia had a significant negative association with high calorie direction bias (higher hyperphagia was associated with a weaker direction bias for food over non-food images) and was not significantly associated with high calorie duration bias. Hypothesis 3 was not supported: hypersomnia severity was not associated with high calorie direction bias nor duration bias. Repeating these tests with low calorie food attentional bias measures as outcome variables did not meaningfully change these relationships, as total depressive symptom severity and hypersomnia severity were not associated with low calorie direction bias or duration bias, and hyperphagia severity was negatively associated with low calorie direction bias and was not associated with low calorie duration bias. Of note, additional adjustment for residential status, physical activity, alcohol use, cannabis use, antidepressant use, and food conditions generally did not meaningfully change the associations reported above.

Hypothesis 4, that total depressive symptom severity (SCL-19) is associated with emotional eating, was not supported in the simultaneous model. However, there is some evidence

of the hypothesized relationship: a significant positive association was detected between the SCL-20 and emotional eating in the individual model. It is noteworthy that hyperphagia is included in the SCL-20 and that this item seems to be strengthening the observed relationship. Consistent with this finding, Hypothesis 5 was supported, as hyperphagia was significantly and positively associated with emotional eating in the simultaneous model. In addition, this association was significant and positive in the individual model, and the bivariate correlation for this association was positive and large in size. Hypothesis 6 was not supported, given that hypersomnia was not significantly associated with emotional eating. Hypothesis 7, that total depressive symptom severity is associated with external eating, was not supported in the simultaneous model. However, this association was significant and positive in the individual model, and the bivariate correlation for this association was positive and moderate in size. Similar to Hypothesis 7, Hypothesis 8 was not supported, but there is some evidence of the hypothesized positive relationship between hyperphagia severity and external eating. Specifically, the standardized regression coefficients in both the simultaneous and individual models are positive and may be meaningful, and the bivariate correlation for this association is positive and moderate in size. Hypothesis 9 was not supported: hypersomnia severity was not associated with external eating. Additional adjustment for residential status, physical activity, alcohol use, cannabis use, antidepressant use, and food conditions did not meaningfully change the associations reported above.

To summarize, many of my hypotheses were not supported, especially the primary hypotheses involving the food attentional bias measures. I did observe some hypothesized associations of total depressive symptom severity and hyperphagia severity with emotional eating and external eating. The pattern of results suggests hyperphagia may be driving

relationships between total depressive symptom severity and emotional eating and external eating. In contrast, hypersomnia does not appear to play an important role in the associations of total depressive symptom severity with emotional eating or external eating.

Fit with Existing Literature

Food Attentional Bias

The present findings conflict with the only other study examining the relationship between depression and food attentional bias. Hawkins and colleagues (2018) detected a significant association between higher somatic depressive symptoms and greater food attentional bias as indicated by a reaction time measure, as well as similar associations with eye tracking measures of direction bias and duration bias. A potential explanation for these discrepant results is differences in the depressive symptom measures. Hawkins et al. (2018) examined the somatic depressive symptom cluster, which consists of fatigue, appetite changes (increased or decreased), and sleep changes (increased or decreased). In contrast, the present study examined only one direction (increased) of appetite and sleep changes (namely, hyperphagia and hypersomnia). This raises the possibility that the other somatic symptoms (namely, fatigue, poor appetite, and insomnia) may drive Hawkins et al.'s (2018) observed association between the somatic depressive symptom cluster and food attentional bias.

In addition to the null results for the analyses examining measures of food attentional bias, I detected one unanticipated relationship that was in the opposite direction of my hypothesis. Specifically, hyperphagia severity was negatively associated with high calorie direction bias, indicating that those with higher hyperphagia severity exhibited a weaker direction bias for food over non-food images than people with lower hyperphagia severity. In fact, people with higher hyperphagia severity showed a preference for non-food over food

images. This relationship was consistent across individual and simultaneous models and across measures of high and low calorie food attentional bias. This finding may indicate that people with elevated hyperphagia severity tend to avoid food cues, perhaps because they are aware of their increased appetite, eating behaviors, and/or weight gain. Of note, evidence suggests that people with obesity demonstrate a similar avoidance to food stimuli (Nijs & Franken, 2012). The authors who reported this finding speculated that this may be due to conflicting desires to eat and lose weight, and that avoidance may be a response to "'forbidden' substances" (Nijs & Franken, 2012, p. 107).

With respect to the null findings for the analyses examining measures of food attentional bias, there are two possible explanations: (1) there may be no true association between depressive symptom measures and food attentional bias or (2) true associations may exist but methodological issues impeded my ability to detect them (Kazdin, 2003). Regarding the first explanation, only one small study ($N = 89$) found a significant effect for one food attentional bias measure (a reaction time measure), and only trends were observed for eye tracking direction bias and duration bias (Hawkins et al., 2018). Evidence for these associations was not observed in the present study, which utilized the same attentional bias task but used updated eye tracking equipment that yielded more complete data from a larger and more diverse sample. Thus, one possible explanation is that Hawkins and colleagues' (2018) findings may have been observed due to type 1 error.

Concerning the second explanation, potential methodological issues that could have impeded my ability to detect true associations include inadequate measurement of key variables, limited variability in key variables, and insufficient statistical power. First, the present study may be affected by inadequate measurement of food attentional bias. In particular, the food images

may not have had sufficient ecological validity to result in the innate attentional bias one would expect based on the Incentive Sensitization Theory of "wanting" that which is rewarding (Berridge & Robinson, 2016; Robinson & Berridge, 1993). A more ecologically valid assessment could involve participants navigating a place where food cues (including visual, olfactory, and auditory cues) are naturally more salient, such as a food court, and assessing their attentional bias in that setting with eye tracking glasses. It is also possible that participants may have habituated to the food cues in the task and thus the stimuli may have decreased in salience over time. Evidence suggests that prolonged exposure to food cues can lead to habituation and decreased food consumption (Epstein, Carr, Cavanaugh, Paluch, & Bouton, 2011).

Second, the present study may have limited variability in key variables, which could lead to missing true associations. However, the total depressive symptom severity (SCL-20; *SD* = 0.70, range: 0.00-3.60), hyperphagia severity (*SD* = 1.10, range: 0.00-4.00), and hypersomnia severity (*SD* = 1.10, range: 0.00-4.00) variables all have good variability (possible range for all three variables: 0.00-4.00). Similarly, high calorie direction bias (*SD* = 0.10, range: 0.08-0.70) and duration bias (*SD* = 0.07, range: 0.21-0.67) have good variability (possible range for these variables: 0.00-1.00). Thus, limited variability in key variables does not appear to explain my null findings. Third, the present study may have insufficient statistical power to detect true associations. Because my regression models were powered to detect small-to-medium effects or larger, any effects of smaller magnitude would have been missed. However, examination of the correlation coefficients (all rs $\leq .17$) and standardized regression coefficients (all βs ≤ 0.16) for the nonsignificant associations between the depressive symptom measures and food attentional bias measures suggests that I did not fail to detect meaningful associations.

Emotional Eating and External Eating

The findings that total depressive symptom severity is positively associated with both emotional eating and external eating are consistent with the existing literatures (Antoniou, Bongers, & Jansen, 2017; Lazarevich, Irigoyen Camacho, Velazquez-Alva, & Zepeda Zepeda, 2016; Masheb & Grilo, 2006; Ouwens, van Strien, & van Leeuwe, 2009; Paans et al., 2018a; Paans et al., 2018b; Paans et al., 2019; Sevinçer et al., 2017). Furthermore, the present study extends previous findings by being among the first to examine the individual symptoms of hyperphagia and hypersomnia. In the only other studies in these literatures to examine these atypical symptoms, Paans and colleagues found that increased appetite and increased weight are more strongly associated with both emotional (Paans et al., 2018a) and external eating (Paans et al., 2018b) than are other depressive symptoms. These authors also found that hypersomnia is not significantly associated with emotional eating nor external eating (Paans et al., 2018b). Supporting these findings, the present study revealed that hyperphagia may be driving the observed relationships of total depressive symptom severity with emotional eating and external eating and that hyperphagia has its own independent associaitons with these self-reported eating patterns. Also in line with Paans and colleagues' (2018b) findings, hypersomnia did not appear to have important associations with emotional eating or external eating in the present study.

There are plausible reasons for why hyperphagia is associated with emotional and external eating. A first explanation is that hyperphagia may predict future emotional eating and external eating. Specifically, higher self-reported "overeating" in general may increase one's likelihood of also eating in response to negative emotions or environmental food cues. A second explanation is the possibility of reverse causality: if a person reports higher emotional eating and/or external eating, he or she may be more likely to report more perceived overeating in

general. Paans and colleagues (2018a) support this rationale in their discussion of their findings, as they suggest that intervening on unhealthy eating styles may help to reduce the atypical depressive symptom of increased appetite. Nevertheless, because the present evidence is cross-sectional, it is unclear whether hyperphagia predicts emotional and external eating, these eating patterns predict hyperphagia, or both directions. However, a third noteworthy explanation for this relationship is construct and measurement overlap. To illustrate, rating one's "overeating" may be similar to rating one's desire to eat when "anxious, worried, or tense," or rating how often one "eats more than usual if food smells and looks good." This overlap calls into question the relationships of hyperphagia with emotional eating and external eating. These questionnaires may be assessing very similar or the same constructs using similar assessment methods (i.e., self-report questionnaires), which could explain the positive associations detected in my study and others.

Strengths, Limitations and Future Directions

The present study has several strengths and limitations to consider. First, the sample of college students is beneficial for studying the role of depressive symptoms in eating behaviors that could lead to obesity development due to (a) young adulthood being a time of increasing autonomy regarding food choices and eating behaviors (Han & Lee, 2017), initiating a trajectory for future eating habits, and (b) minimal potential confounding by other medical conditions. However, a college student sample also has limitations. This sample is not representative of the general community in terms of predispositions for obesity, including low socioeconomic status and limited access to healthy foods (APA, 2013; Benjamin et al., 2017; Hales, Carroll, Fryar, & Ogden, 2017; McLaren, 2007; USDA & USDHHS, 2010). In addition to a community-based sample, another sample of interest could be people living in food deserts (urban areas where

accessing healthy foods is difficult; White, 2007). In this population, people who are overweight or obese may have differential liking toward inexpensive, unhealthy foods and higher rates of emotional eating (Dressler & Smith, 2013). It could be valuable to study food attentional bias and external eating in this context. Despite the limitations of this study's sample, it is unlikely that the sample alone accounts for the null findings; it seems more likely that the food attentional bias assessment did not adequately measure true individual differences in food attentional bias.

Second, the assessment approaches for the outcome variables have limitations. While the eye tracking paradigm is innovative, the food images may not have had sufficient ecological validity to detect true individual differences in food attentional bias. Using eye tracking glasses to measure food attentional bias in a natural environment (e.g., a food court) and decreasing the potential for habituation to the task (e.g., a stimulating natural environment or interactive virtual reality rather than a seated computer task) may strengthen the ecological validity of the task. In addition, self-report assessments of eating behaviors have limited predictive validity for actual eating behaviors and food intake (Jansen et al., 2011). Future studies may consider using actual food consumption in response to negative emotions or environmental food cues. Alternative assessment approaches for emotional eating include (a) laboratory mood induction and measurement of subsequent food consumption and (b) using ecological momentary assessment to assess mood and subsequent eating behavior, testing whether increases in negative emotions are associated with increases in eating behavior. Similarly, alternative assessment approaches for external eating include (a) using virtual reality to expose participants to food cues and measuring subsequent food consumption and (b) using ecological momentary assessment to assess environmental food cues (e.g., with photos or video) and subsequent eating behavior, testing whether increases in environmental food cues are associated with increases in eating behavior.

Third, a limitation of the present study is that it examined cross-sectional associations of depressive symptom severity with food attentional bias and eating behaviors that may lead to obesity development, rather than assessing these relationships across time. This is problematic because the tested relationships have plausible bidirectional associations. To illustrate, the depression-and-obesity relationship is bidirectional (Luppino et al., 2010), and it is plausible that emotional and external eating could promote the development of overweight and obesity, which may lead to depression. A prospective study with multiple time points would allow for further clarification of the temporal order of depressive symptom severity, food attentional bias, emotional eating, and external eating.

In sum, future studies should consider more representative samples of the general community, alternative approaches for measuring food attentional bias and eating behaviors, and longitudinal designs to determine the directionality of associations among depressive symptoms, food attentional bias, and eating behaviors.

Conclusion

Total depressive symptom severity and hypersomnia severity were not associated with measures of food attentional bias in this sample of generally healthy, young adults. However, hyperphagia severity was negatively associated with direction bias, but not associated with duration bias, for high and low calorie food images. Total depressive symptom severity and hyperphagia severity were positively associated with both emotional eating and external eating. Furthermore, the pattern of results suggests that hyperphagia may be driving relationships between total depressive symptom severity and emotional and external eating. Hypersomnia severity was not associated with emotional eating and external eating, suggesting that this symptom does not play an important role in the relationships between depressive symptoms and

these eating behaviors. Several factors may have contributed to the present null results, including inadequate measurement of key variables, limited variability in key variables, and insufficient statistical power. Although the novel hypotheses of the present study were not supported, it may still be important to investigate food attentional bias as a potential mechanism in the depression-to-obesity relationship. Future studies should examine prospective associations of hyperphagia severity with food attentional bias, emotional eating, and external eating in larger, more representative samples.

REFERENCES

Antoniou, E. E., Bongers, P., & Jansen, A. (2017). The mediating role of dichotomous thinking and emotional eating in the relationship between depression and BMI. *Eat Behav, 26*, 55-60. doi:10.1016/j.eatbeh.2017.01.007

Aronne, L. J., Nelinson, D. S., & Lillo, J. L. (2009). Obesity as a disease state: A new paradigm for diagnosis and treatment. *Clin Cornerstone, 9*(4), 9-29.

American Medical Association. (2013). *Is obesity a disease?* (CSAPH Report 3-A-13). Retrieved from https://www.ama-assn.org/sites/default/files/media-browser/public/about-ama/councils/Council%20Reports/council-on-science-public-health/a13csaph3.pdf

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5 ed.). Arlington, VA: American Psychiatric Association.

Babor, T. F., de la Fuente, J. R., Saunders, J., & Grant, M. (1989). *AUDIT The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care. WHO/MNH/DAT 89.4*. Geneva: World Health Organization.

Babor, T. F., Higgins-Biddle, J. C., Saunders, J., & Monteiro, M. G. (2001). *The Alcohol Use Disorders Identification Test: Guidelines for use in primary care*. Geneva: World Health Organization.

Barness, L. A., Opitz, J. M., & Gilbert-Barness, E. (2007). Obesity: Genetic, molecular, and environmental aspects. *American Journal of Medical Genetics Part A, 143A*(24), 3016-3034. doi:10.1002/ajmg.a.32035

Belzer, L. M., Smulian, J. C., Lu, S. E., & Tepper, B. J. (2010). Food cravings and intake of sweet foods in healthy pregnancy and mild gestational diabetes mellitus. A prospective study. *Appetite, 55*(3), 609-615. doi:10.1016/j.appet.2010.09.014

Benazzi, F. (2002). Can only reversed vegetative symptoms define atypical depression? *Eur Arch Psychiatry Clin Neurosci, 252*(6), 288-293. doi:10.1007/s00406-002-0395-0

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., . . . & Muntner, P. (2017). Heart disease and stroke statistics—2017 update: A report from the American Heart Association. *Circulation*. doi:10.1161/cir.0000000000000485

Berridge, K. C. (2009). 'Liking' and 'wanting' food rewards: Brain substrates and roles in eating disorders. *Physiol Behav, 97*(5), 537-550. doi:10.1016/j.physbeh.2009.02.044

Berridge, K. C., Ho, C. Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Res, 1350*, 43-64. doi:10.1016/j.brainres.2010.04.003

Berridge, K. C., & Robinson, T. E. (2016). Liking, wanting, and the incentive-sensitization theory of addiction. *The American Psychologist, 71*(8), 670-679. doi:10.1037/amp0000059

Bongers, P., & Jansen, A. (2016). Emotional eating is not what you think it is and emotional eating scales do not measure what you think they measure. *Frontiers in Psychology, 7*, 1932-1932. doi:10.3389/fpsyg.2016.01932

Bornstein, S. R., Schuppenies, A., Wong, M. L., & Licinio, J. (2006). Approaching the shared biology of obesity and depression: The stress axis as the locus of gene-environment interactions. *Mol Psychiatry, 11*(10), 892-902. doi:10.1038/sj.mp.4001873

Braden, A., Flatt, S. W., Boutelle, K. N., Strong, D., Sherwood, N. E., & Rock, C. L. (2016). Emotional eating is associated with weight loss success among adults enrolled in a weight loss program. *J Behav Med, 39*(4), 727-732. doi:10.1007/s10865-016-9728-8

Bradley, B. P., Mogg, K., Wright, T., & Field, M. (2003). Attentional bias in drug dependence: Vigilance for cigarette-related cues in smokers. *Psychol Addict Behav, 17*(1), 66-72.

Caro, J. F., Sinha, M. K., Kolaczynski, J. W., Zhang, P. L., & Considine, R. V. (1996). Leptin: The tale of an obesity gene. *Diabetes, 45*(11), 1455-1462.

Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *Int J Obes (Lond), 33*(9), 1063-1073. doi:10.1038/ijo.2009.138

Center for Collegiate Mental Health. (2017). *2016 Annual Report* (STA 17-74). Retrieved from https://sites.psu.edu/ccmh/files/2017/01/2016-Annual-Report-FINAL_2016_01_09-1gc2hj6.pdf

Chou, K. L., & Yu, K. M. (2013). Atypical depressive symptoms and obesity in a national sample of older adults with major depressive disorder. *Depress Anxiety, 30*(6), 574-579. doi:10.1002/da.22098

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

Copersino, M. L., Serper, M. R., Vadhan, N., Goldberg, B. R., Richarme, D., Chou, J. C. Y., . . . & Cancro, R. (2004). Cocaine craving and attentional bias in cocaine-dependent schizophrenic patients. *Psychiatry Research, 128*(3), 209-218. doi:10.1016/j.psychres.2004.07.006

Craig, C. L., Marshall, A., Sjostrom, M., Bauman, A., Booth, M. L., Ainsworth, B., . . . & Oja, P. (2003). *International Physical Activity Questionnaire: 12-Country Reliability and Validity* (Vol. 35).

de Ruiter, C., & Brosschot, J. F. (1994). The emotional Stroop interference effect in anxiety: Attentional bias or cognitive avoidance? *Behav Res Ther, 32*(3), 315-319.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL). A measure of primary symptom dimensions. *Mod Probl Pharmacopsychiatry, 7*(0), 79-110.

Dobson, K. S., & Dozois, D. J. (2004). Attentional biases in eating disorders: A meta-analytic review of Stroop performance. *Clin Psychol Rev, 23*(8), 1001-1022.

Domoff, S. E., Meers, M. R., Koball, A. M., & Musher-Eizenman, D. R. (2014). The validity of the Dutch Eating Behavior Questionnaire: Some critical remarks. *Eat Weight Disord, 19*(2), 137-144. doi:10.1007/s40519-013-0087-y

Dressler, H., & Smith, C. (2013). Food choice, eating behavior, and food liking differs between lean/normal and overweight/obese, low-income women. *Appetite, 65*, 145-152. doi:https://doi.org/10.1016/j.appet.2013.01.013

Epstein, L. H., Carr, K. A., Cavanaugh, M. D., Paluch, R. A., & Bouton, M. E. (2011). Long-term habituation to food in obese and nonobese women. *Am J Clin Nutr, 94*(2), 371-376. doi:10.3945/ajcn.110.009035

Faunce, G. J. (2002). Eating disorders and attentional bias: A review. *Eat Disord, 10*(2), 125-139. doi:10.1080/10640260290081696

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug Alcohol Depend, 97*(1-2), 1-20. doi:10.1016/j.drugalcdep.2008.03.030

Field, M., Werthmann, J., Franken, I., Hofmann, W., Hogarth, L., & Roefs, A. (2016). The role of attentional bias in obesity and addiction. *Health Psychol, 35*(8), 767-780. doi:10.1037/hea0000405

Flegal, K. M., Carroll, M. D., Kit, B. K., & Ogden, C. L. (2012). Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *JAMA, 307*(5), 491-497. doi:10.1001/jama.2012.39

Fleming, M. F., Barry, K. L., & MacDonald, R. (1991). The Alcohol Use Disorders Identification Test (AUDIT) in a college sample. *Int J Addict, 26*(11), 1173-1185.

Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord, 24*(1), 38-48.

Foulds, J. A., Adamson, S. J., Boden, J. M., Williman, J. A., & Mulder, R. T. (2015). Depression in patients with alcohol use disorders: Systematic review and meta-analysis of outcomes for independent and substance-induced disorders. *J Affect Disord, 185*, 47-59. doi:10.1016/j.jad.2015.06.024

Frayn, M., Sears, C. R., & von Ranson, K. M. (2016). A sad mood increases attention to unhealthy food images in women with food addiction. *Appetite, 100,* 55-63. 10.1016/j.appet.2016.02.008

Hales, C. M., Carroll, M. D., Fryar, C. D., & Ogden, C. L. (2017). Prevalence of obesity among adults and youth: United States, 2015-2016. *NCHS Data Brief*(288), 1-8.

Han, D. H., Lyool, I. K., Sung, Y. H., Lee, S. H., & Renshaw, P. F. (2008). The effect of acamprosate on alcohol and food craving in patients with alcohol dependence. *Drug Alcohol Depend, 93*(3), 279-283. doi:10.1016/j.drugalcdep.2007.09.014

Han, S., Lee, S. (2017). College student binge eating: Attachment, psychological needs satisfaction, and emotion regulation. *Journal of College Student Development, 58*(7), 1074-1086.

Hawkins, M. A. W., Miller, D. K., & Stewart, J. C. (2015). A 9-year, bidirectional prospective analysis of depressive symptoms and adiposity: The African American Health Study. *Obesity, 23*(1), 192-199. doi:doi:10.1002/oby.20893

Hawkins, M. A. W., Vrany, E. A., Cyders, M. A., Ciciolla, L., Wells, T. T., & Stewart, J. C. (2018). Association between depressive symptom clusters and food attentional bias. *Eat Behav, 31*, 24-27. doi:10.1016/j.eatbeh.2018.07.002

Hendrikse, J. J., Cachia, R. L., Kothe, E. J., McPhie, S., Skouteris, H., & Hayden, M. J. (2015). Attentional biases for food cues in overweight and individuals with obesity: A systematic review of the literature. *Obesity Reviews, 16*(5), 424-432. doi:doi:10.1111/obr.12265

Hermans, R. C., Larsen, J. K., Lochbuehler, K., Nederkoorn, C., Herman, C. P., & Engels, R. C. (2013). The power of social influence over food intake: Examining the effects of attentional bias and impulsivity. *Br J Nutr, 109*(3), 572-580. doi:10.1017/s0007114512001390

Hickman, R. J., Khambaty, T., & Stewart, J. C. (2014). C-reactive protein is elevated in atypical but not nonatypical depression: Data from the National Health and Nutrition Examination survey (NHANES) 1999-2004. *J Behav Med, 37*(4), 621-629. doi:10.1007/s10865-013-9510-0

Hou, R., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite, 56*(2), 424-427. doi:10.1016/j.appet.2011.01.019

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological psychiatry, 61*(3), 348-358. doi:10.1016/j.biopsych.2006.03.040

Hutchison, K. E., McGeary, J., Smolen, A., & Wooden, A. (2001). Craving after alcohol consumption: Olanzapine and the DRD4 VNTR polymorphism. *Alcoholism: Clinical and Experimental Research, 25*(5).

Jansen, A., Nederkoorn, C., Roefs, A., Bongers, P., Teugels, T., & Havermans, R. (2011). The proof of the pudding is in the eating: Is the DEBQ-external eating scale a valid measure of external eating? *Int J Eat Disord, 44*(2), 164-168. doi:10.1002/eat.20799

Jensen, M. D., Ryan, D. H., Apovian, C. M., Ard, J. D., Comuzzie, A. G., Donato, K. A., … & Yanovski, S. Z. (2013). 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association task force on practice guidelines and the Obesity Society. *Circulation, 129*(25), S102-S138. doi:10.1161/01.cir.0000437739.71477.ee

Johns, S. A., Kroenke, K., Krebs, E. E., Theobald, D. E., Wu, J., & Tu, W. (2013). Longitudinal comparison of three depression measures in adult cancer patients. *J Pain Symptom Manage, 45*(1), 71-82. doi:10.1016/j.jpainsymman.2011.12.284

Katon, W., Von Korff, M., Lin, E., Walker, E., Simon, G. E., Bush, T., . . . & Russo, J. (1995). Collaborative management to achieve treatment guidelines: Impact on depression in primary care. *JAMA, 273*(13), 1026-1031. doi:10.1001/jama.1995.03520370068039

Kazdin, A. E. (2003). *Research design in clinical psychology, 4th ed.* Boston, MA: Allyn & Bacon.

Kemps, E., & Tiggemann, M. (2009). Attentional bias for craving-related (chocolate) food cues. *Experimental and Clinical Psychopharmacology, 17*(6), 425-433. doi:10.1037/a0017796

Kenardy, J., Arnow, B., & Agras, W. S. (1996). The aversiveness of specific emotional states associated with binge-eating in obese subjects. *Aust N Z J Psychiatry, 30*(6), 839-844. doi:10.3109/00048679609065053

Kline, R. B. (2005). *Principles and practice of structural equation modeling, 2nd ed.* New York, NY: Guilford Press.

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *J Gen Intern Med, 16*(9), 606-613.

Labarthe, D. R. (1998). *Epidemiology and prevention of cardiovascular diseases: A global challenge*. Gaithersburg, MD: Aspen Publishers.

Lamers, F., Milaneschi, Y., de Jonge, P., Giltay, E. J., & Penninx, B. W. J. H. (2017). Metabolic and inflammatory markers: Associations with individual depressive symptoms. *Psychological Medicine, 48*(7), 1102-1110. doi:10.1017/S0033291717002483

Lasserre, A. M., Glaus, J., Vandeleur, C. L., Marques-Vidal, P., Vaucher, J., Bastardot, F., . . . & Preisig, M. (2014). Depression with atypical features and increase in obesity, body mass index, waist circumference, and fat mass: A prospective, population-based study. *JAMA Psychiatry, 71*(8), 880-888. doi:10.1001/jamapsychiatry.2014.411

Lazarevich, I., Irigoyen Camacho, M. E., Velazquez-Alva, M. D. C., & Zepeda Zepeda, M. (2016). Relationship among obesity, depression, and emotional eating in young adults. *Appetite, 107*, 639-644. doi:10.1016/j.appet.2016.09.011

Lev-Ran, S., Roerecke, M., Le Foll, B., George, T. P., McKenzie, K., & Rehm, J. (2014). The association between cannabis use and depression: A systematic review and meta-analysis of longitudinal studies. *Psychol Med, 44*(4), 797-810. doi:10.1017/s0033291713001438

Luecken, L. J., Gallo, L. C. (2007). *Handbook of physiological research methods in health psychology*. Thousand Oaks, CA: Sage Publications.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W., & Zitman, F. G. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry, 67*(3), 220-229. doi:10.1001/archgenpsychiatry.2010.2

Markowitz, S., Friedman, M. A., & Arent, S. M. (2008). Understanding the relation between obesity and depression: Causal mechanisms and implications for treatment. *Clinical Psychology-Science and Practice, 15*(1), 1-20. doi:10.1111/j.1468-2850.2008.00106.x

Masheb, R. M., & Grilo, C. M. (2006). Emotional overeating and its associations with eating disorder psychopathology among overweight patients with binge eating disorder. *Int J Eat Disord, 39*(2), 141-146. doi:10.1002/eat.20221

Matza, L. S., Revicki, D. A., Davidson, J. R., & Stewart, J. W. (2003). Depression with atypical features in the National Comorbidity Survey: Classification, description, and consequences. *Arch Gen Psychiatry, 60*(8), 817-826. doi:10.1001/archpsyc.60.8.817

McCarty, C. A., Kosterman, R., Mason, W. A., McCauley, E., Hawkins, J. D., Herrenkohl, T. I., & Lengua, L. J. (2009). Longitudinal associations among depression, obesity and alcohol use disorders in young adulthood. *Gen Hosp Psychiatry, 31*(5), 442-450. doi:10.1016/j.genhosppsych.2009.05.013

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiol Rev, 29*, 29-48. doi:10.1093/epirev/mxm001

McMahon, A. B., Arms-Chavez, C. J., Harper, B. D., & LoBello, S. G. (2017). PHQ-8 minor depression among pregnant women: Association with somatic symptoms of depression. *Archives of women's mental health, 20*(3), 405-409. doi:10.1007/s00737-017-0715-z

Milaneschi, Y., Lamers, F., Bot, M., Drent, M. L., & Penninx, B. W. (2017). Leptin dysregulation is specifically associated with major depression with atypical features: Evidence for a mechanism connecting obesity and depression. *Biol Psychiatry, 81*(9), 807-814. doi:10.1016/j.biopsych.2015.10.023

Milaneschi, Y., Simmons, W. K., van Rossum, E. F. C., & Penninx, B. W. J. H. (2018). Depression and obesity: Evidence of shared biological mechanisms. *Mol Psychiatry*. doi:10.1038/s41380-018-0017-5

Mogg, K., Bradley, B. P., Hyare, H., & Lee, S. (1998). Selective attention to food-related stimuli in hunger: Are attentional biases specific to emotional and psychopathological states, or are they also found in normal drive states? *Behav Res Ther, 36*(2), 227-237.

Murru, A., Guiso, G., Barbuti, M., Anmella, G., Verdolini, N., Samalin, L., . . . & Pacchiarotti, I. (2019). The implications of hypersomnia in the context of major depression: Results from a large, international, observational study. *European Neuropsychopharmacology*. doi:https://doi.org/10.1016/j.euroneuro.2019.02.011

Nathan, P. J., O'Neill, B. V., Mogg, K., Bradley, B. P., Beaver, J., Bani, M., . . . & Bullmore, E. T. (2012). The effects of the dopamine D(3) receptor antagonist GSK598809 on attentional bias to palatable food cues in overweight and obese subjects. *Int J Neuropsychopharmacol, 15*(2), 149-161. doi:10.1017/s1461145711001052

Neeland, I. J., Poirier, P., Després, J. P. (2018). Cardiovascular and metabolic heterogeneity of obesity: Clinical challenges and implications for management. *Circulation, 137*, 1391-1406. doi:10.1161/CIRCULATIONAHA.117.029617

National Institude on Drug Abuse. (2012). *The NIDA Quick Screen*. Retrieved from https://www.drugabuse.gov/publications/resource-guide-screening-drug-use-in-general-medical-settings/nida-quick-screen

Nijs, I. M., Muris, P., Euser, A. S., & Franken, I. H. (2010). Differences in attention to food and food intake between overweight/obese and normal-weight females under conditions of hunger and satiety. *Appetite, 54*(2), 243-254. doi:10.1016/j.appet.2009.11.004

Nijs, I. M. T., & Franken, I. H. A. (2012). Attentional processing of food cues in overweight and obese individuals. *Current Obesity Reports, 1*(2), 106-113. doi:10.1007/s13679-012-0011-1

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA, 311*(8), 806-814. doi:10.1001/jama.2014.732

Ouwens, M. A., van Strien, T., & van Leeuwe, J. F. (2009). Possible pathways between depression, emotional and external eating. A structural equation model. *Appetite, 53*(2), 245-248. doi:10.1016/j.appet.2009.06.001

Paans, N. P. G., Bot, M., Brouwer, I. A., Visser, M., Roca, M., Kohls, E., . . . & Penninx, B. (2018). The association between depression and eating styles in four European countries: The MooDFOOD prevention study. *J Psychosom Res, 108*, 85-92. doi:10.1016/j.jpsychores.2018.03.003

Paans, N. P. G., Bot, M., van Strien, T., Brouwer, I. A., Visser, M., & Penninx, B. W. J. H. (2018). Eating styles in major depressive disorder: Results from a large-scale study. *Journal of Psychiatric Research, 97*, 38-46. doi:10.1016/j.jpsychires.2017.11.003

Paans, N. P. G., Gibson-Smith, D., Bot, M., van Strien, T., Brouwer, I. A., Visser, M., & Penninx, B. W. J. H. (2019). Depression and eating styles are independently associated with dietary intake. *Appetite, 134,* 103-110. doi:10.1016/j.appet.2018.12.030

Penninx, B. W., Milaneschi, Y., Lamers, F., & Vogelzangs, N. (2013). Understanding the somatic consequences of depression: Biological mechanisms and the role of depression symptom profile. *BMC Med, 11*, 129. doi:10.1186/1741-7015-11-129

Phelan, S., Hassenstab, J., McCaffery, J. M., Sweet, L., Raynor, H. A., Cohen, R. A., & Wing, R. R. (2011). Cognitive interference from food cues in weight loss maintainers, normal weight, and obese individuals. *Obesity (Silver Spring), 19*(1), 69-73. doi:10.1038/oby.2010.138

Placanica, J. L., Faunce, G. J., & Soames Job, R. F. (2002). The effect of fasting on attentional biases for food and body shape/weight words in high and low Eating Disorder Inventory scorers. *Int J Eat Disord, 32*(1), 79-90. doi:10.1002/eat.10066

Polanka, B. M., Vrany, E. A., Patel, J., & Stewart, J. C. (2017). Depressive disorder subtypes as predictors of incident obesity in US adults: Moderation by race/ethnicity. *Am J Epidemiol, 185*(9), 734-742. doi:10.1093/aje/kwx030

Pope, J. F., Skinner, J. D., & Carruth, B. R. (1992). Cravings and aversions of pregnant adolescents. *Journal of the American Dietetic Association, 92*(12), 1479-1482.

Racette, S. B., Deusinger, S. S., & Deusinger, R. H. (2003). Obesity: Overview of prevalence, etiology, and treatment. *Phys Ther, 83*(3), 276-288.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*(3), 385-401. doi:10.1177/014662167700100306

Rahe, C., Baune, B. T., Unrath, M., Arolt, V., Wellmann, J., Wersching, H., & Berger, K. (2015). Associations between depression subtypes, depression severity and diet quality: Cross-sectional findings from the BiDirect Study. *BMC Psychiatry, 15*, 38. doi:10.1186/s12888-015-0426-9

Ravussin, E., & Bouchard, C. (2000). Human genomics and obesity: Finding appropriate drug targets. *European Journal of Pharmacology, 410*(2), 131-145. doi:10.1016/S0014-2999(00)00811-6

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Res Brain Res Rev, 18*(3), 247-291.

Roche, A. F., Sievogel, R. M., Chumlea, W. C., & Webb, P. (1981). Grading body fatness from limited anthropometric data. *Am J Clin Nutr, 34*(12), 2831-2838. doi:10.1093/ajcn/34.12.2831

Rosenbaum, M., Leibel, R. L., & Hirsch, J. (1997). Obesity. *N Engl J Med, 337*(6), 396-407. doi:10.1056/nejm199708073370606

Scarpace, P. J., & Zhang, Y. (2009). Leptin resistance: A prediposing factor for diet-induced obesity. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 296*(3), R493-R500. doi:10.1152/ajpregu.90669.2008

Sevinçer, G. M., Konuk, N., İpekçioğlu, D., Crosby, R. D., Cao, L., Coskun, H., & Mitchell, J. E. (2017). Association between depression and eating behaviors among bariatric surgery candidates in a Turkish sample. *Eat Weight Disord, 22*(1), 117-123. doi:10.1007/s40519-016-0296-2

Shi, L., An, R., & Meijgaard, J. V. (2013). Cigarette smoking and abdominal obesity: A meta-analysis of observational studies. *Journal of Substance Use, 18*(6), 440-449. doi:10.3109/14659891.2012.715227

Sinha, R., & O'Malley, S. S. (2000). Alcohol and eating disorders: Implications for alcohol treatment and health services research. *Alcoholism, clinical and experimental research, 24*(8), 1312-1319. doi:10.1111/j.1530-0277.2000.tb02097.x

Sobal, J., & Stunkard, A. J. (1989). Socioeconomic status and obesity: A review of the literature. *Psychol Bull, 105*(2), 260-275.

Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: A fresh approach to the study of binge eating. *Appetite, 44*(3), 253-261. doi:10.1016/j.appet.2004.12.001

Song, Y. M., Lee, K., & Sung, J. (2017). Genetic and environmental relationships between eating behavior and symptoms of anxiety and depression. *Eat Weight Disord*. doi:10.1007/s40519-017-0445-2

Stroop, J. R. (1992). *Studies of Interference in Serial Verbal Reactions* (Vol. 121).

Tobii StudioTM 3.X software release 3.2 product description. (2012). Tobii Technology.

U.S. Department of Agriculture & U. S. Department of Health and Human Services. (2010). *Dietary guidelines for Americans*. Washington, DC: U.S. Government Printing Office Retrieved from https://health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf

Vainik, U., Dagher, A., Dubé, L., & Fellows, L. K. (2013). Neurobehavioural correlates of body mass index and eating behaviours in adults: A systematic review. *Neuroscience and biobehavioral reviews, 37*(3), 279-299. doi:10.1016/j.neubiorev.2012.11.008

van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behavior. *International Journal Eating Disorders, 5*, 295-315.

van Strien, T., Herman, C., & Anschutz, D. (2012). The predictive validity of the DEBQ-external eating scale for eating in response to food commercials while watching television. *Int J Eat Disord, 45*(2), 257-262. doi:10.1002/eat.20940

van Strien, T., Herman, C. P., Anschutz, D. J., Engels, R. C., & de Weerth, C. (2012). Moderation of distress-induced eating by emotional eating scores. *Appetite, 58*(1), 277-284. doi:10.1016/j.appet.2011.10.005

van Strien, T., Herman, C. P., & Verheijden, M. W. (2009). Eating style, overeating, and overweight in a representative Dutch sample. Does external eating play a role? *Appetite, 52*(2), 380-387. doi:10.1016/j.appet.2008.11.010

van Strien, T., Konttinen, H., Homberg, J. R., Engels, R. C., & Winkens, L. H. (2016). Emotional eating as a mediator between depression and weight gain. *Appetite, 100*, 216-224. doi:10.1016/j.appet.2016.02.034

van Strien, T., Schippers, G. M., & Cox, W. M. (1995). On the relationship between emotional and external eating behavior. *Addictive behaviors, 20*(5), 585-594. doi:https://doi.org/10.1016/0306-4603(95)00018-8

Walker, E. A., Katon, W. J., Russo, J., Von Korff, M., Lin, E., Simon, G., . . . & Unützer, J. (2000). Predictors of outcome in a primary care depression trial. *J Gen Intern Med, 15*(12), 859-867. doi:10.1046/j.1525-1497.2000.91142.x

Wardle, J. (1987). Eating style: A validation study of the Dutch Eating Behaviour Questionnaire in normal subjects and women with eating disorders. *J Psychosom Res, 31*(2), 161-169.

Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can(not) take my eyes off it: Attention bias for food in overweight participants. *Health Psychol, 30*(5), 561-569. doi:10.1037/a0024291

Westerterp, K. R., & Speakman, J. R. (2008). Physical activity energy expenditure has not declined since the 1980s and matches energy expenditures of wild mammals. *Int J Obes (Lond), 32*(8), 1256-1263. doi:10.1038/ijo.2008.74

Williams, J. M., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychol Bull, 120*(1), 3-24.

White, M. (2007). Food access and obesity. *Obesity Reviews, 8*(s1), 99-107. doi: 10.1111/j.1467-789X.2007.00327.x

World Health Organization. (2000). *Obesity: Preventing and managing the global epidemic* (Report of a WHO Consultation). Retrieved from Geneva, Switzerland: http://whqlibdoc.who.int/trs/WHO_TRS_894.pdf

TABLES

Table 1. Characteristics of Participants ($N = 95$)

Age, years	20.4 (3.8)
Female, %	81.1
Non-Hispanic White, %	73.7
BMI, kg/m2	23.4 (2.8)
Subjective Hunger (possible range: 0.0-100.0)	15.4 (14.9)
Living Independent of Family, %	73.7
IPAQ Overall MET Value†	3493 (2926)
Past-Year Alcohol Use, AUDIT Total (possible range: 0-40)	2.8 (3.9)
Past-Year Cannabis Use, NIDA Quick Screen %	25.3
Antidepressant Use, %	13.7
Food Condition, % †	9.6
SCL-20 Total Score (possible range: 0.0-4.0)	0.85 (0.70)
SCL-20 Hyperphagia Item Score (possible range: 0.00-4.00)	0.68 (1.10)
SCL-20 Hypersomnia Item Score (possible range: 0.00-4.00)	0.83 (1.10)
High Calorie Direction Bias Score (possible range: 0.00-4.00)	0.41 (0.10)
High Calorie Duration Bias Score (possible range: 0.00-4.00)	0.49 (0.07)
Low Calorie Direction Bias Score (possible range: 0.00-4.00)	0.40 (0.11)
Low Calorie Duration Bias Score (possible range: 0.00-4.00)	0.47 (0.07)
DEBQ Emotional Eating Score (possible range: 1.00-5.00)	2.08 (0.90)
DEBQ External Eating Score (possible range: 1.00-5.00)	3.05 (0.76)

Note: Continuous variables are presented as mean (standard deviation), and categorical variables are presented as percentage. BMI = body mass index; IPAQ = International Physical Activity Questionnaire; MET = metabolic equivalent of task; AUDIT = Alcohol Use Disorder Identification Test; NIDA = National Institute on Drug Abuse; SCL-20 = Hopkins Symptom Checklist; DEBQ = Dutch Eating Behavior Questionnaire

† Reported on a reduced sample. Physical activity is reported on $n = 92$. Food condition is reported on $n = 94$.

Table 2. Correlations Among Measures of Food Attentional Bias, Eating Behaviors, and Depressive Symptom Severity

	High Calorie Direction Bias	High Calorie Duration Bias	Low Calorie Direction Bias	Low Calorie Duration Bias	Emotional Eating	External Eating	SCL-19	Hyperphagia	Hypersomnia
High Calorie Direction Bias	1.00								
High Calorie Duration Bias	.01	1.00							
Low Calorie Direction Bias	.72**	.02	1.00						
Low Calorie Duration Bias	.09	.63**	.14	1.00					
Emotional Eating	.08	.08	.08	.14	1.00				
External Eating	-.06	.31**	-.06	.17	.47**	1.00			
SCL-19	-.12	.17	.00	.00	.30**	.23*	1.00		
Hyperphagia	-.17	.22*	-.12	.17	.54**	.36**	.55**	1.00	
Hypersomnia	-.09	.06	.02	-.16	.03	.03	.47**	.13	1.00

Note: N = 95. SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item. SCL-20 = Hopkins Symptom Checklist-20 item.

** = $p < .01$

* = $p < .05$

Table 3. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with High Calorie Direction and Duration Bias

	High Calorie Direction Bias					**High Calorie Duration Bias**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-19	0.01	0.02	0.06	0.40	0.69	0.01	0.01	0.11	0.79	0.43
Hyperphagia	-0.02	0.01	-0.26	-2.00	0.05	0.01	0.01	0.10	0.78	0.44
Hypersomnia	-0.01	0.01	-0.08	-0.62	0.54	0.00	0.01	0.02	0.18	0.86

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger.
SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item.
SCL-20 = Hopkins Symptom Checklist-20.

Table 4. Individual Linear Regression Models Examining the Associations of Depressive Symptoms with High Calorie Direction and Duration Bias

	High Calorie Direction Bias					**High Calorie Duration Bias**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-20	-0.02	0.01	-0.13	-1.26	0.21	0.02	0.01	0.18	1.78	0.08
Hyperphagia	-0.02	0.01	-0.26	-1.97	0.05	0.01	0.01	0.10	0.75	0.46
Hypersomnia	0.00	0.01	-0.03	-0.23	0.82	0.00	0.01	0.02	0.13	0.90

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger. Models where hyperphagia is a predictor are adjusted for the SCL-20 minus appetite. Models where hypersomnia is a predictor are adjusted for the SCL-20 minus sleep. SCL-20 = Hopkins Symptom Checklist-20.

Table 5. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with High Calorie Direction and Duration Bias Adjusting for Additional Covariates

	High Calorie Direction Bias			High Calorie Duration Bias		
	β	***t***	***p***	***β***	***t***	***p***
Residential Status (*N* = 95)						
SCL-19	0.04	0.29	0.77	0.11	0.78	0.44
Hyperphagia	-0.25	-1.88	0.06	0.10	0.75	0.46
Hypersomnia	-0.07	-0.55	0.59	0.02	0.17	0.87
Physical Activity (*n* = 92)†						
SCL-19	0.10	0.70	0.49	0.11	0.78	0.44
Hyperphagia	-0.22	-1.59	0.12	0.08	0.62	0.54
Hypersomnia	-0.09	-0.68	0.50	0.03	0.21	0.83
Alcohol Use (*N* = 95)						
SCL-19	0.06	0.45	0.66	0.10	0.73	0.47
Hyperphagia	-0.26	-1.91	0.06	0.09	0.67	0.50
Hypersomnia	-0.07	-0.59	0.56	0.02	0.14	0.89
Cannabis Use (*N* = 95)						
SCL-19	0.07	0.46	0.65	0.11	0.74	0.46
Hyperphagia	-0.26	-1.99	0.05	0.10	0.77	0.44
Hypersomnia	-0.06	-0.45	0.66	0.01	0.05	0.96
Antidepressant Use (*N* = 95)						
SCL-19	0.03	0.23	0.82	0.15	1.08	0.29
Hyperphagia	-0.26	-1.99	0.05	0.10	0.77	0.44
Hypersomnia	-0.08	-0.64	0.52	0.03	0.23	0.82
Food Condition (*n* = 94)†						
SCL-19	0.05	0.37	0.71	0.11	0.77	0.45
Hyperphagia	-0.26	-1.92	0.06	0.11	0.84	0.40
Hypersomnia	-0.05	-0.42	0.68	0.04	0.32	0.75

Note: All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger.
SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item.
SCL-20 = Hopkins Symptom Checklist-20.
† Reported on a reduced sample. Physical activity is reported on $n = 92$. Food condition is reported on
$n = 94$.

Table 6. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with Low Calorie Direction and Duration Bias

	Low Calorie Direction Bias					**Low Calorie Duration Bias**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-19	0.02	0.02	0.16	1.14	0.26	0.00	0.01	-0.03	-0.21	0.83
Hyperphagia	-0.03	0.01	-0.30	-2.29	0.02	0.01	0.01	0.15	1.15	0.26
Hypersomnia	0.00	0.01	-0.02	-0.18	0.86	-0.01	0.01	-0.15	-1.22	0.22

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger.
SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item.
SCL-20 = Hopkins Symptom Checklist-20.

Table 7. Individual Linear Regression Models Examining the Associations of Depressive Symptoms with Low Calorie Direction and Duration Bias

	Low Calorie Direction Bias					**Low Calorie Duration Bias**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-20	0.00	0.02	-0.03	-0.24	0.81	0.00	0.01	-0.01	-0.10	0.92
Hyperphagia	-0.03	0.01	-0.30	-2.36	0.02	0.01	0.01	0.18	1.35	0.18
Hypersomnia	0.00	0.01	0.03	0.27	0.79	-0.01	0.01	-0.17	-1.47	0.15

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger. Models where hyperphagia is a predictor are adjusted for the SCL-20 minus appetite. Models where hypersomnia is a predictor are adjusted for the SCL-20 minus sleep.
SCL-20 = Hopkins Symptom Checklist-20.

Table 8. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with Low Calorie Direction and Duration Bias Adjusting for Additional Covariates

	Low Calorie Direction Bias			Low Calorie Duration Bias		
	β	***t***	***p***	***β***	***t***	***p***
Residential Status (*N* = 95)						
SCL-19	0.15	1.06	0.29	-0.03	-0.22	0.83
Hyperphagia	-0.29	-2.20	0.03	0.15	1.13	0.26
Hypersomnia	-0.02	-0.15	0.88	-0.15	-1.19	0.24
Physical Activity (*n* = 92)†						
SCL-19	0.19	1.35	0.18	-0.03	-0.21	0.84
Hyperphagia	-0.25	-1.90	0.06	0.15	1.11	0.27
Hypersomnia	-0.03	-0.25	0.80	-0.15	-1.17	0.25
Alcohol Use (*N* = 95)						
SCL-19	0.18	1.28	0.20	-0.05	-0.32	0.75
Hyperphagia	-0.27	-2.13	0.04	0.13	0.99	0.32
Hypersomnia	-0.01	-0.12	0.91	-0.16	-1.29	0.20
Cannabis Use (*N* = 95)						
SCL-19	0.17	1.19	0.24	-0.04	-0.27	0.79
Hyperphagia	-0.30	-2.29	0.03	0.15	1.14	0.26
Hypersomnia	0.00	-0.03	0.98	-0.17	-1.36	0.18
Antidepressant Use (*N* = 95)						
SCL-19	0.16	1.08	0.28	0.03	0.21	0.83
Hyperphagia	-0.30	-2.28	0.03	0.15	1.15	0.25
Hypersomnia	-0.02	-0.19	0.85	-0.14	-1.18	0.24
Food Condition (*n* = 94)†						
SCL-19	0.16	1.11	0.27	-0.03	-0.21	0.83
Hyperphagia	-0.29	-2.22	0.03	0.15	1.14	0.26
Hypersomnia	0.00	-0.01	0.99	-0.15	-1.17	0.25

Note: All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger.
SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item.
SCL-20 = Hopkins Symptom Checklist-20.
† Reported on a reduced sample. Physical activity is reported on *n* = 92. Food condition is reported on
n = 94.

Table 9. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with Emotional Eating and External Eating

	DEBQ Emotional Eating					**DEBQ External Eating**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-19	0.10	0.16	0.08	0.64	0.53	0.15	0.14	0.14	1.05	0.30
Hyperphagia	0.38	0.09	0.47	4.07	<0.001	0.15	0.08	0.21	1.73	0.09
Hypersomnia	-0.05	0.09	-0.06	-0.57	0.57	-0.02	0.08	-0.03	-0.30	0.77

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger. SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item. SCL-20 = Hopkins Symptom Checklist-20. DEBQ = Dutch Eating Behavior Questionnaire.

Table 10. Individual Linear Regression Models Examining the Associations of Depressive Symptoms with Emotional Eating and External Eating

	DEBQ Emotional Eating					**DEBQ External Eating**				
	B	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
SCL-20	0.41	0.12	0.32	3.33	0.001	0.27	0.10	0.25	2.55	0.01
Hyperphagia	0.38	0.09	0.46	4.07	<0.001	0.14	0.08	0.21	1.70	0.09
Hypersomnia	-0.10	0.09	-0.12	-1.12	0.27	-0.04	0.08	-0.06	-0.50	0.62

Note: N = 95. All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger. Models where hyperphagia is a predictor are adjusted for the SCL-20 minus appetite. Models where hypersomnia is a predictor are adjusted for the SCL-20 minus sleep.
SCL-20 = Hopkins Symptom Checklist-20. DEBQ = Dutch Eating Behavior Questionnaire.

Table 11. Simultaneous Linear Regression Models Examining the Associations of Depressive Symptoms with Emotional Eating and External Eating Adjusting for Additional Covariates

	DEBQ Emotional Eating			DEBQ External Eating		
	β	***t***	***p***	***β***	***t***	***p***
Residential Status (*N* = 95)						
SCL-19	0.09	0.67	0.50	0.14	1.02	0.31
Hyperphagia	0.46	3.93	<0.001	0.21	1.68	0.10
Hypersomnia	-0.06	-0.60	0.55	-0.03	-0.30	0.77
Physical Activity (*n* = 92)†						
SCL-19	0.10	0.83	0.41	0.15	1.16	0.25
Hyperphagia	0.52	4.68	<0.001	0.23	1.86	0.07
Hypersomnia	-0.07	-0.68	0.50	-0.03	-0.28	0.78
Alcohol Use (*N* = 95)						
SCL-19	0.08	0.62	0.54	0.12	0.94	0.35
Hyperphagia	0.46	4.01	<0.001	0.19	1.57	0.12
Hypersomnia	-0.06	-0.57	0.57	-0.04	-0.37	0.72
Cannabis Use (*N* = 95)						
SCL-19	0.06	0.47	0.64	0.12	0.89	0.38
Hyperphagia	0.46	4.14	<0.001	0.21	1.75	0.08
Hypersomnia	-0.11	-1.01	0.32	-0.09	-0.75	0.46
Antidepressant Use (*N* = 95)						
SCL-19	0.08	0.64	0.53	0.15	1.09	0.28
Hyperphagia	0.47	4.05	<0.001	0.21	1.71	0.09
Hypersomnia	-0.06	-0.56	0.58	-0.03	-0.29	0.78
Food Condition (*n* = 94)†						
SCL-19	0.08	0.67	0.51	0.14	1.06	0.30
Hyperphagia	0.46	3.99	<0.001	0.21	1.67	0.10
Hypersomnia	-0.09	-0.84	0.40	-0.04	-0.39	0.70

Note: All models are adjusted for age, sex, race/ethnicity, BMI, and subjective hunger. SCL-19 = the average of all SCL-20 items excluding the hyperphagia ("overeating") item. SCL-20 = Hopkins Symptom Checklist-20. DEBQ = Dutch Eating Behavior Questionnaire. † Reported on a reduced sample. Physical activity is reported on $n = 92$. Food condition is reported on $n = 94$.

FIGURES

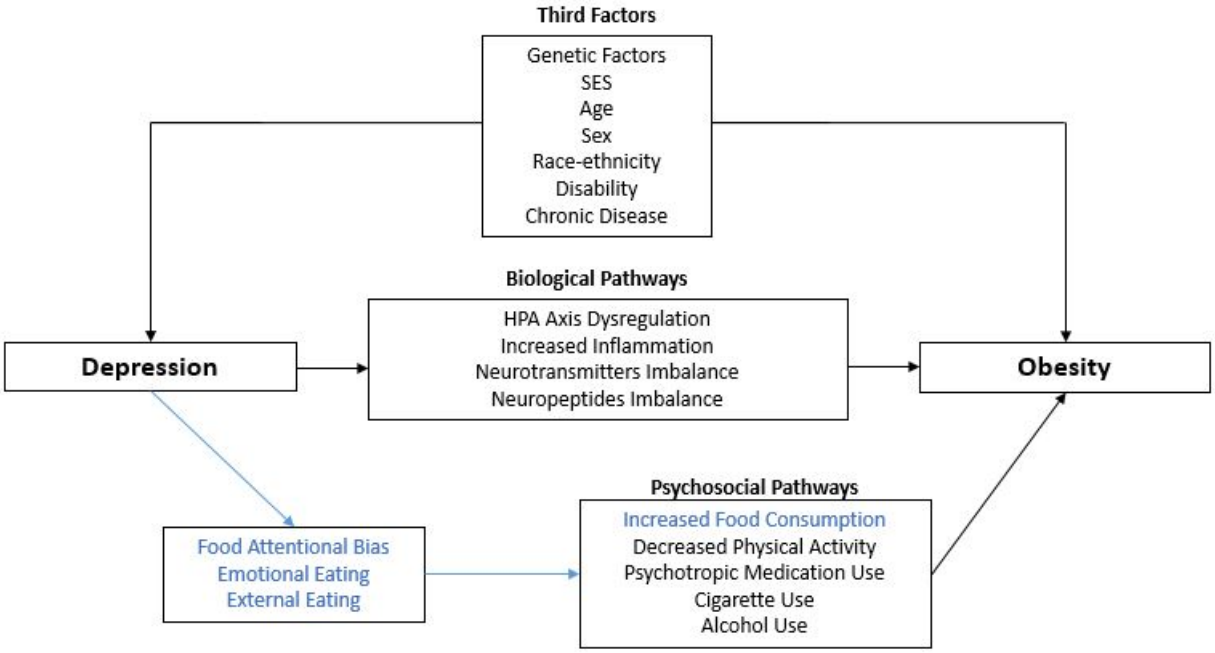

Figure 1. Depression as an Emerging Risk Factor for Obesity and Underlying Candidate Mechanisms.

This model is adapted from Hawkins et al. (2015). It portrays the depression-to-obesity relationship, traditional pathways in the relationship, and candidate mechanisms in the depression-to-increased food consumption relationship (blue text), which I examine in the present study. SES: socioeconomic status; HPA axis: hypothalamic-pituitary-adrenal axis.

Figure 2. Examples of High Calorie Image Pairs in the Food Attentional Bias Assessment

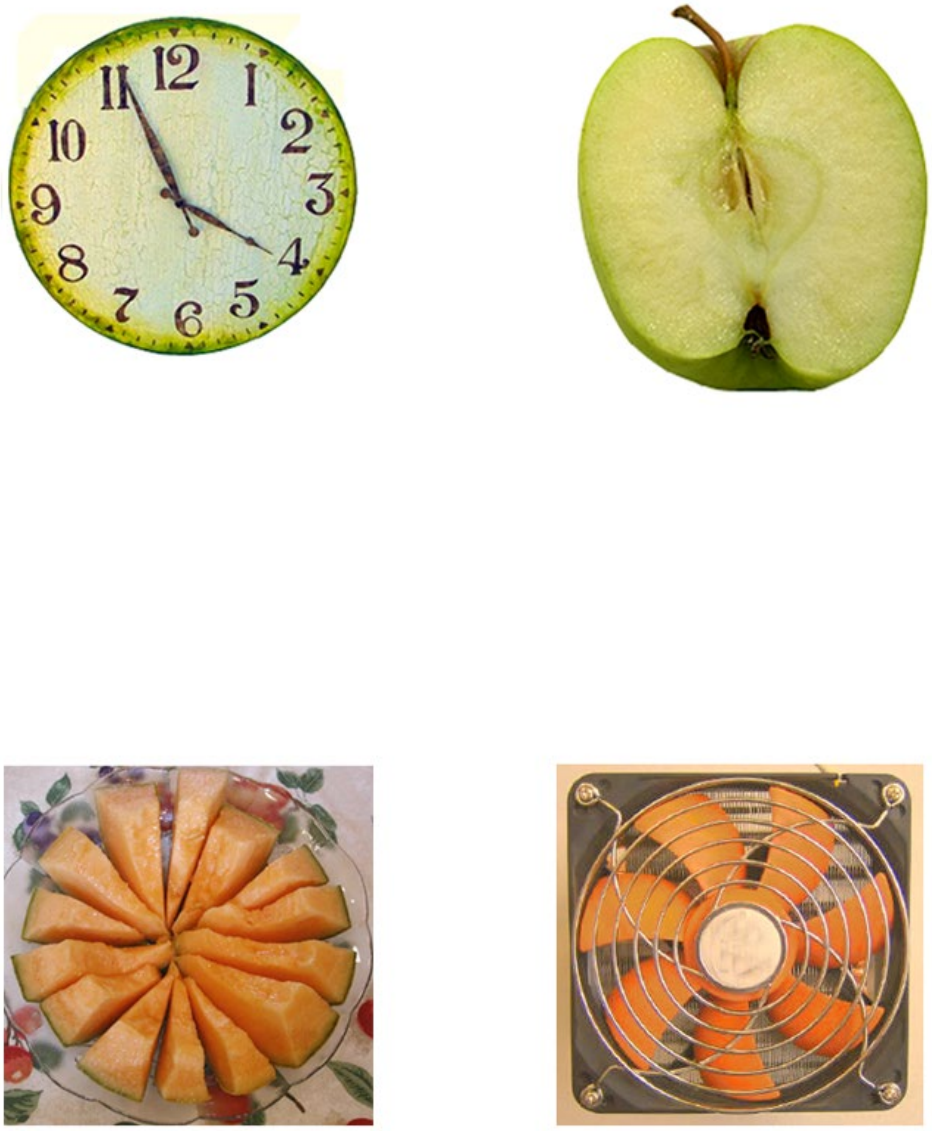

Figure 3. Examples of Low Calorie Image Pairs in the Food Attentional Bias Assessment




Figure 4. Examples of Control Image Pairs in the Food Attentional Bias Assessment

APPENDIX

State VAS Items

Please read each question carefully and rate how much you **feel that way at the present moment**. You may mark anywhere on each line with a perpendicular line (I). Be sure to mark each line *only once*.

How tense do you feel?

Not very _____ Very much

How sad do you feel?

Not very _____ Very much

How hungry do you feel?

Not very _____ Very much

How much of an effort is it to do anything?

Not very _____ Very much

How full (not hungry) do you feel?

Not very _____ Very much

How happy do you feel?

Not very _____ Very much

How calm do you feel?

Not very _____ Very much

How strong is your urge to eat?

Not very _____ Very much

How sleepy do you feel?

Not at all _____ Extremely

How weary do you feel?

Not at all _____ Extremely

Demographic Questionnaire

What is your date of birth? _____/_____/_____

What is your biological sex?

1. Female
2. Male
3. Other/Intersex

What is your gender?

1. Male
2. Female
3. Transgender
4. Other

Which race do you most identify with or consider yourself to be?

1. White/Caucasian
2. Black/African American
3. Asian
4. Native Hawaiian or Other Pacific Islander
5. American Indian/Alaskan Native
6. Biracial (Specify:_____)
7. Other (Specify:_____)
8. Don't know

Are you Hispanic or Latino?

1. Yes
2. No

What is your residential status?

☐ Live in on-campus housing
☐ Live in off-campus housing independent from family
☐ Live off campus with family (parents, guardians, etc.)
☐ Other, please specify _____

Do you have diabetes mellitus (Type 1 or Type 2)?

☐ Yes, Type 1

☐ Yes, Type 2
☐ I do not have diabetes

Do you have any food allergies or food-related conditions?

☐ Yes
☐ No
☐ If yes, please describe: _____

Have you gained weight in the past 6 months?

☐ Yes
☐ No

If yes, how many pounds have you gained? _____

Have you lost weight in the past 6 months?

☐ Yes
☐ No

If yes, how many pounds have you lost?

Are you currently trying to lose weight?

☐ Yes
☐ No

If yes, how are you trying to lose weight? Check all that apply.

☐ Skipping meals
☐ Diet foods or diet products
☐ Eating less food
☐ Drinking a lot of water
☐ Liquid diet
☐ Eating more fruits/vegetables/salads
☐ Eating less fat
☐ Switching to low calorie foods
☐ Eating fewer carbohydrates
☐ Eating less sugar, candy, sweets
☐ Changing eating habits
☐ Following a special diet
☐ Exercising

☐ Taking non-prescription supplements
☐ Taking prescription diet pills
☐ Joining a weight loss program
☐ Seeking help from a health professional
☐ Taking laxatives or vomiting
☐ Smoking
☐ N/A (I am not currently trying to lose weight)
☐ Other, please explain:

_____

_____

Do you take any prescription medications?

☐ Yes
☐ No

If yes, please list the name of your prescription medications, as well as what you are taking each medication for.

_____

_____

_____

NIDA Quick Screen

In the past year, how often have you used the following?

Alcohol

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Tobacco Products (cigarettes, chewing tobacco, snuff, dip, snus, etc.)

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Cannabis (marijuana, pot, grass, hash, etc.)

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Prescription Drugs for Non-Medical Reasons

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Illegal Drugs

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Synthetic Cannabinoids (Spice, K2/herbal incense, sometimes called “fake weed” or “synthetic marijuana”

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Synthetic Cathinones (“bath salts,” mephedrone, pentedrone, alpha-PVP)

Never	Once or Twice	Monthly	Weekly	Daily or Almost Daily

Symptom Checklist-20 (SCL-20)

Below is a list of problems and complaints that people sometimes have. Please read each one carefully. After you have done so, please check one of the spaces to the right that best describes HOW MUCH THAT PROBLEM HAS BOTHERED OR DISTRESSED YOU *DURING THE PAST WEEK*, INCLUDING TODAY. Mark only one space for each problem and do not skip any.

How much were you bothered by:

1. Loss of sexual interest or pleasure

Not at all	A little bit	Moderately	Quite a bit	Extremely

2. Feeling low in energy or slowed down

Not at all	A little bit	Moderately	Quite a bit	Extremely

3. Thoughts of ending your life

Not at all	A little bit	Moderately	Quite a bit	Extremely

4. Poor appetite

Not at all	A little bit	Moderately	Quite a bit	Extremely

5. Crying easily

Not at all	A little bit	Moderately	Quite a bit	Extremely

6. Feeling of being trapped or caught

Not at all	A little bit	Moderately	Quite a bit	Extremely

7. Blaming yourself for things

Not at all	A little bit	Moderately	Quite a bit	Extremely

8. Feeling lonely

Not at all	A little bit	Moderately	Quite a bit	Extremely

9. Feeling blue

Not at all	A little bit	Moderately	Quite a bit	Extremely

10. Worrying too much about things

Not at all	A little bit	Moderately	Quite a bit	Extremely

11. Feeling no interest in things

Not at all	A little bit	Moderately	Quite a bit	Extremely

12. Trouble falling asleep

Not at all	A little bit	Moderately	Quite a bit	Extremely

13. Feeling hopeless about the future

Not at all	A little bit	Moderately	Quite a bit	Extremely

14. Thoughts of death or dying

Not at all	A little bit	Moderately	Quite a bit	Extremely

15. Overeating

Not at all	A little bit	Moderately	Quite a bit	Extremely

16. Awakening in the early morning

Not at all	A little bit	Moderately	Quite a bit	Extremely

17. Sleep that is restless or disturbed

Not at all	A little bit	Moderately	Quite a bit	Extremely

18. Feeling everything is an effort

Not at all	A little bit	Moderately	Quite a bit	Extremely

19. Feelings of worthlessness

Not at all	A little bit	Moderately	Quite a bit	Extremely

20. Feelings of guilt

Not at all	A little bit	Moderately	Quite a bit	Extremely

Supplemental SCL-20 Sleep Question

Below is a list of problems and complaints that people sometimes have. Please read each one carefully. After you have done so, please check one of the spaces to the right that best describes HOW MUCH THAT PROBLEM HAS BOTHERED OR DISTRESSED YOU *DURING THE PAST WEEK*, INCLUDING TODAY. Mark only one space for each problem and do not skip any.

How much were you bothered by:

1. Sleeping too much

Not at all	A little bit	Moderately	Quite a bit	Extremely

Dutch Eating Behavior Questionnaire

Emotional Eating

1. Do you have a desire to eat when you are irritated?

Never	Seldom	Sometimes	Often	Very Often

2. Do you have a desire to eat when you have nothing to do?

Never	Seldom	Sometimes	Often	Very Often

3. Do you have a desire to eat when you are depressed or discouraged?

Never	Seldom	Sometimes	Often	Very Often

4. Do you have a desire to eat when you are feeling lonely?

Never	Seldom	Sometimes	Often	Very Often

5. Do you have a desire to eat when somebody lets you down?

Never	Seldom	Sometimes	Often	Very Often

6. Do you have a desire to eat when you are cross?

Never	Seldom	Sometimes	Often	Very Often

7. Do you have a desire to eat when something unpleasant is about to happen?

Never	Seldom	Sometimes	Often	Very Often

8. Do you get the desire to eat when you are anxious, worried or tense?

Never	Seldom	Sometimes	Often	Very Often

9. Do you have a desire to eat when things are going against you or have gone wrong?

Never	Seldom	Sometimes	Often	Very Often

10. Do you have a desire to eat when you are frightened?

Never	Seldom	Sometimes	Often	Very Often

11. Do you have a desire to eat when you are disappointed?

Never	Seldom	Sometimes	Often	Very Often

12. Do you have a desire to eat when you are emotionally upset?

Never	Seldom	Sometimes	Often	Very Often

13. Do you have a desire to eat when you are bored or restless?

Never	Seldom	Sometimes	Often	Very Often

External Eating

14. If food tastes good to you do you eat more than usual?

Never	Seldom	Sometimes	Often	Very Often

15. If food smells and looks good do you eat more than usual?

Never	Seldom	Sometimes	Often	Very Often

16. If you see or smell something delicious do you have a desire to eat it?

Never	Seldom	Sometimes	Often	Very Often

17. If you have something delicious to eat do you eat it straight away?

Never	Seldom	Sometimes	Often	Very Often

18. If you see others eating do you also want to eat?

Never	Seldom	Sometimes	Often	Very Often

19. Do you eat more than usual when you see others eating?

Never	Seldom	Sometimes	Often	Very Often

20. When preparing a meal are you inclined to eat something?

Never	Seldom	Sometimes	Often	Very Often

21. If you walk past the baker do you have a desire to buy something delicious?

Never	Seldom	Sometimes	Often	Very Often

22. If you walk past a snack bar or a café do you have the desire to buy something delicious?

Never	Seldom	Sometimes	Often	Very Often

23. Can you resist eating delicious food?

Never	Seldom	Sometimes	Often	Very Often

International Physical Activity Questionnaire

This survey asks you about the time you spent being physically active in the *last 7 days*. Please answer each question even if you do not consider yourself to be an active person. Think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise, or sport.

1. Now, think about all the vigorous activities which take hard physical effort that you did in the last 7 days. Vigorous activities make you breathe much harder than normal and may include heavy lifting, digging, aerobics, or fast bicycling. Think only about those physical activities that you did for at least 10 minutes at a time.

A. During the *last 7 days*, on how many days did you do *vigorous* physical activities?

☐ None
☐ 1 day
☐ 2 days
☐ 3 days
☐ 4 days
☐ 5 days
☐ 6 days
☐ 7 days
☐ Don't Know/Not sure
☐ Refused

B. How much time did you usually spend doing *vigorous* physical activities *on one of those days*?

0 minutes

15 minutes

…

16 hours

Don’t know/Not sure

Refused

2. Now think about activities which take moderate physical effort that you did in the last 7 days. Moderate physical activities make you breathe somewhat harder than normal and may include carrying light loads, bicycling at a regular pace, or doubles tennis. Do not include walking. Again, think about only those physical activities that you did for at least 10 minutes at a time.

A. During the *last 7 days*, on how many days did you do *moderate* physical activities?

☐ None
☐ 1 day
☐ 2 days
☐ 3 days
☐ 4 days
☐ 5 days
☐ 6 days
☐ 7 days
☐ Don't Know/Not sure
☐ Refused

B. How much time did you usually spend doing *moderate* physical activities *on one of those days*?

0 minutes

15 minutes

…

16 hours

☐ Don't Know/Not sure
☐ Refused

3. Now think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

A. During the *last 7 days*, on how many days did you *walk* for at least 10 minutes at a time?

☐ None
☐ 1 day
☐ 2 days
☐ 3 days

☐ 4 days
☐ 5 days
☐ 6 days
☐ 7 days
☐ Don't Know/Not sure
☐ Refused

B. How much time did you usually spend walking on one of those days*?*

0 minutes

15 minutes

…

16 hours

☐ Don't Know/Not sure
☐ Refused

4. Now think about the time you spent sitting on week days during the last 7 days. Include time spent at work, at home, while doing course work, and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

A. During the last 7 days, how much time did you usually spend ***sitting*** on a week day?

0 minutes

15 minutes

…

16 hours

☐ Don't Know/Not sure
☐ Refused

The Alcohol Use Disorders Identification Test (AUDIT)

1. How often do you have a drink containing alcohol?

Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week

2. How many drinks containing alcohol do you have on a typical day when you are drinking?

1 or 2	3 or 4	5 or 6	7 to 9	10 or more

3. How often do you have six or more drinks on one occasion?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

4. How often during the last year have you found that you were not able to stop drinking once you had started?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

5. How often during the last year have you failed to do what was normally expected of you because of drinking?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

7. How often during the last year have you had a feeling of guilt or remorse after drinking?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

8. How often during the last year have you been unable to remember what happened the night before because of your drinking?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

9. Have you or someone else been injured because of your drinking?

No		Yes, but not in the last year		Yes, during the last year

10. Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down?

No		Yes, but not in the last year		Yes, during the last year