Associations of Somatic Depressive Symptoms with Food Attentional Bias and Eating Behaviors

Aubrey L. Shell, M.S., Rachel A. Jackson, B.A., Jay S. Patel, M.S., Adam T. Hirsh, Ph.D., Melissa A. Cyders, Ph.D., and Jesse C. Stewart, Ph.D.

Department of Psychology, Indiana University-Purdue University Indianapolis (IUPUI)

Author Note

All authors have the same affiliation: Department of Psychology, IUPUI. The authors have no conflicts of interest to disclose.

Correspondence concerning this article should be addressed to Jesse C. Stewart, Ph.D., Department of Psychology, Indiana University-Purdue University Indianapolis (IUPUI), 402 North Blackford Street, LD 100E, Indianapolis, IN 46202. Telephone: (317) 274-6761. Fax: (317) 274-6756. Email: jstew@iupui.edu

---

This is the author's manuscript of the article published in final edited form as:

Shell, A. L., Jackson, R. A., Patel, J. S., Hirsh, A. T., Cyders, M. A., & Stewart, J. C. (2021). Associations of somatic depressive symptoms with food attentional bias and eating behaviors. Appetite, 167, 105593. https://doi.org/10.1016/j.appet.2021.105593

Associations of Somatic Depressive Symptoms with Food Attentional Bias and Eating Behaviors

1. Introduction

Depression is an emerging risk factor for obesity: people with versus without depression have 58% greater odds of developing obesity over time (Luppino, de Wit, Bouvy, & et al., 2010). Recent evidence suggests obesity risk may vary across depressive disorder subtypes, with the highest risk among people with atypical major depressive disorder (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). Atypical MDD is more strongly associated with obesity incidence and BMI increases over time than other MDD subtypes (Lasserre et al., 2014; Polanka, Vrany, Patel, & Stewart, 2017). Atypical MDD has also been associated with biological pathways thought to contribute to obesity development, including leptin dysregulation and systemic inflammation (Hickman, Khambaty, & Stewart, 2014; Milaneschi, Lamers, Bot, Drent, & Penninx, 2017; Penninx, Milaneschi, Lamers, & Vogelzangs, 2013; Scarpace & Zhang, 2009).

Hyperphagia and hypersomnia are of particular interest 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). Hyperphagia and hypersomnia have been associated with adiposity measures, such as BMI and waist circumference (Lamers, Milaneschi, de Jonge, Giltay, & Penninx, 2017; Murru et al., 2019), and are plausible candidates for increasing obesity risk over time, as they could lead to increased energy intake (food consumption) and decreased energy expenditure (physical activity). In addition, these symptoms may reflect circadian rhythm

dysregulation, which has demonstrated associations with obesity (Zelinski, Deibel, & McDonald, 2014).

Although the precise mechanisms underlying the depression-to-obesity relationship have yet to be elucidated, candidate mechanisms have been proposed. Traditional candidate mechanisms fall into two broad domains: biological pathways and behavioral pathways. Biological pathways include hypothalamic-pituitary-adrenal (HPA) dysregulation, leptin dysregulation, neurotransmitter imbalance, neuropeptide imbalance, and systemic inflammation (Bornstein, Schuppenies, Wong, & Licinio, 2006; Howren, Lamkin, & Suls, 2009; Majd, Saunders, & Engeland, 2020; Markowitz, Friedman, & Arent, 2008; Milaneschi, Lamers, Berk, & Penninx, 2020; Milaneschi et al., 2017; Milaneschi, Simmons, van Rossum, & Penninx, 2019; Penninx et al., 2013). Behavioral pathways include increased food consumption, decreased physical activity, smoking, alcohol use, and psychotropic medication use (Markowitz et al., 2008; McCarty et al., 2009; Shi, An, & Meijgaard, 2013). More novel and upstream candidate mechanisms that may promote increased food consumption and that have been associated with obesity-related factors include food attentional bias (a cognitive tendency to notice food cues over other environmental cues; Berridge, Ho, Richard, & DiFeliceantonio, 2010; Castellanos et al., 2009; Faunce, 2002; Hendrikse et al., 2015; Nijs & Franken, 2012), emotional eating (eating in response to negative emotions; Konttinen, van Strien, Männistö, Jousilahti, & Haukkala, 2019; van Strien, Herman, & Verheijden, 2009; van Strien, Konttinen, Homberg, Engels, & Winkens, 2016), external eating (eating in response to external food cues; Hou et al., 2011; Vainik, Dagher, Dubé, & Fellows, 2013), and restrained eating (controlling food intake to prevent weight gain or promote weight loss; Baños et al., 2014; Tuschl, 1990). These candidate mechanisms are the outcome variables of the present study.

Prior research has demonstrated positive associations between total depressive symptom severity and emotional eating (Antoniou, Bongers, & Jansen, 2017; Konttinen et al., 2019; Lazarevich, Irigoyen Camacho, Velazquez-Alva, & Zepeda Zepeda, 2016; Ouwens, van Strien, & van Leeuwe, 2009; Paans, Bot, Brouwer, et al., 2018), external eating (Paans, Bot, Brouwer, et al., 2018), and restrained eating (Brechan & Kvalem, 2015; van Strien et al., 2016; Werrij, Mulkens, Hospers, & Jansen, 2006). In addition, recent studies have begun to assess whether these associations are stronger for particular depressive symptoms. In the only study examining the relationship between depression and food attentional bias, Hawkins et al. (2018) found that the somatic depressive symptom cluster (which includes appetite and sleep disturbance), but not total depressive symptoms, was associated with greater food attentional bias as measured by reaction time during a dot-probe task. Regarding eating behaviors, a European depression prevention study found that increased appetite and weight gain are more strongly associated with emotional eating than other depressive symptoms (Paans, Bot, Brouwer, et al., 2018). Similarly, a recent European study of depressed patients and healthy controls found that increased appetite and weight gain are more strongly associated with external eating than other depressive symptoms, while weight loss is more strongly associated with restrained eating (Paans, Bot, van Strien, et al., 2018). While results of these initial studies are intriguing and potentially significant, there is a need for additional research to establish the reproducibility of these findings and extend them to other measures and populations.

Accordingly, the present study's objective is to examine associations of total depressive symptom severity, hyperphagia severity, and hypersomnia severity with food attentional bias (Food Stroop task), emotional eating, external eating, and restrained eating (self-reported on the Dutch Eating Behavior Questionnaire) in a sample of young adults without obesity. We

hypothesized positive relationships between these three depressive symptom variables and the food attentional bias and eating behavior variables. For comparison, we also assessed associations of poor appetite and disturbed sleep with food attentional bias and eating behaviors.

2. Methods

2.1 Participants

Participants were 103 young adults enrolled in a Midwestern university who were compensated with psychology course credit for their time and effort. Eligibility criteria were: age ≥ 18 years, BMI in the healthy weight (18.5-24.9 kg/m^2) or overweight (25.0-29.9 kg/m^2) categories, and not pregnant at the time of their study visit. A young adult sample is appropriate for testing our hypotheses, as young adulthood is a time of increasing autonomy regarding food choices and eating behaviors and a time when disordered eating may develop in response to the stress of separation from relatives and adjustment to their new autonomy (Han & Lee, 2017). Supporting this point, eating behaviors are a common concern for young adults as indicated by them being 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). Only individuals with BMIs in the healthy or overweight categories (18.5-29.9 kg/m^2 BMI range) were included. Individuals outside of these BMI categories were not included because underweight or obesity may lead to food-avoidant behaviors and influence responses on the outcome measures (Hudson, Hiripi, Pope, & Kessler, 2007; Kenardy, Arnow, & Agras, 1996; Werthmann et al., 2011). In addition, our objective is to advance understanding of depression and its facets (e.g., hyperphagia and hypersomnia) as emerging risk factors for obesity, so it is important to examine the relationships of interest in people who have not yet developed obesity. This study was approved

by the university's Institutional Review Board and all participants provided written informed consent to all study procedures.

2.2 Measures

2.2.1 Depressive Symptom Variables

Total depressive symptom severity was measured by the Hopkins Symptom Checklist-20 (SCL-20; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). On a 0-4 scale, participants indicated their level of distress regarding 20 depressive symptoms over the past week. The SCL-20 total score, computed as the mean of item responses, ranges from 0-4, with higher scores indicating greater depressive symptom severity (Derogatis et al., 1974). Hyperphagia and poor appetite were computed using responses to the "overeating" and "poor appetite" items on the SCL-20, respectively. Hypersomnia and disturbed sleep were computed using responses to the supplemental "sleeping too much" item—which we added using the same 0-4 response set because the SCL-20 does not otherwise assess hypersomnia—and the "sleep that is restless or disturbed" item, respectively. We chose this item over the other two hyposomnia items assessed by the SCL-20 ("trouble falling asleep" and "awakening in the early morning") because it is the most global single item of hyposomnia. Finally, the SCL-15 is the mean of all depressive symptoms other than those pertaining to appetite or sleep.

2.2.2 Food Attentional Bias

Food attentional bias was measured using a modified computer-based Stroop task (i.e., Food Stroop) through the Psychology Experiment Building Language (Mueller & Piper, 2014). The Food Stroop involves 24 trials of food words and 24 trials of neutral words appearing one at a time in the center of the screen. Participants were instructed to respond quickly and accurately to the color of the words (blue, green, or red) by pressing specific keys on the keyboard (J, K, or F), while paying attention to the meaning of the words. We compared response latencies in color

naming for food-related (e.g., “cake”) versus neutral (e.g., “pencil”) words (Williams, Mathews, & MacLeod, 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 greater food attentional bias (Phelan et al., 2011).

2.2.3 Eating Behaviors

Emotional eating, external eating, and restrained eating were measured by the English version of the Dutch Eating Behavior Questionnaire (DEBQ; Wardle, 1987; van Strien et al., 1986). The DEBQ is a 33-item self-report questionnaire which asks respondents to rate their general frequency of particular eating behaviors (e.g., “Do you have a desire to eat when somebody lets you down?”) on a 1-5 scale, with response options ranging from “never” to “very often.” Mean scores for emotional eating (13 items), external eating (10 items), and restrained eating (10 items) were computed, with higher scores indicating greater levels of each eating behavior.

2.2.4 Covariates

We assessed several *a priori* covariates that are potential confounders of associations of depressive symptom severity with food attentional bias and/or eating behaviors. These covariates were age, sex, race/ethnicity, residential status, body mass index, state hunger, past-year alcohol use, and past-year cannabis use. Data regarding age (years), sex (0 = male, 1 = female), race/ethnicity, and residential status were collected using standard questions. Race was assessed using seven options (White/Caucasian, Black/African American, Asian, Native Hawaiian or Other Pacific Islander, American Indian/Alaskan Native, Other/Biracial, and Don’t Know) and ethnicity was assessed as Hispanic or non-Hispanic. Due to low frequency counts for individual

racial and ethnic minority groups, race/ethnicity was coded as 0 = non-Hispanic White and 1 = non-White. We assessed residential status (0 = living at home with parents/family, 1 = not living at home with parents/family) due to the possibility that this factor could affect participants' access to healthy foods and their autonomy in making food choices. Body mass index (kg/m^2) was computed from weight and height measured by a standard medical scale (Tanita WB-3000 440lb capacity). We assessed state hunger because evidence supports a positive relationship between state hunger and food attentional bias (Castellanos et al., 2009). Participants responded to two 0-100 visual analogue scales ("How hungry do you feel?" and "How strong is your urge to eat?"). We also measured alcohol use and cannabis use because they have been associated with depression (Foulds, Adamson, Boden, Williman, & Mulder, 2015; Lev-Ran et al., 2014), food craving (Han, Lyool, Sung, Lee, & Renshaw, 2008; Hutchison, 2001; Sobik, Hutchison, & Craighead, 2005), and disordered eating (Sinha & O'Malley, 2000). Past-year alcohol use was assessed by the Alcohol Use Disorder Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1989). Past-year cannabis use was assessed by the cannabis item from the National Institute on Drug Abuse Quick Screen (NIDA, 2012). Of note, we changed the timeframe from "in your lifetime" to "in the past year" to align with that of the AUDIT.

2.3 Procedure

Participants attended a one-hour laboratory visit, during which they provided written informed consent and then completed state hunger assessments, a computerized eye tracking food attentional bias task, anthropometric measurements, the Food Stroop, and computerized questionnaires.[1]

[1] Only the procedures directly related to this study's objective are described in detail. Participants completed a computerized eye tracking food attentional bias task which assessed direction bias and duration bias. Data from this task are not included in this report due to concerns about the validity of the task. First, the food images lacked ecological validity (e.g., cupcakes next to colorful yarn), and participants likely habituated to the task. Second, the images were small and close together, allowing for peripheral and undetectable gazing at food images. Two additional Stroop tasks, an Exercise Stroop and a Rest Stroop, were also given; however, these data have yet to be analyzed and are not included in this report because they pertain to physical activity rather than food attentional bias.

Participants were instructed to eat a full meal within three hours of their visit start time to minimize variability in state hunger and its influence on the assessments. Participants completed two 0-100 visual analogue scales to assess state hunger, then had theirheight and weight measured using a Tanita WB-3000 440lb capacity scale. Next, they completed the Stroop task, which took approximately five minutes and included a practice set followed by the Food Stroop containing 24 food words and 24 neutral words appearing in alternating order. After completing the Stroop task, participants were administered a battery of self-report questionnaires, including the eating behavior and covariate assessments, on a password-protected web-based research tool. To end the visit, participants were provided with a debriefing form clearly describing the study purpose and instructing them not to discuss the study with other students.

2.4 Data Analysis

To achieve our study's objective, we conducted a set of linear regression models with the depressive symptom variables (hyperphagia, hypersomnia, poor appetite, disturbed sleep, and SCL-15) as the predictor variables and the food attentional bias and eating behavior variables (Food Stroop, emotional eating, external eating, and restrained eating) as the outcome variables. These models were adjusted for age, sex, race/ethnicity, residential status, BMI, state hunger, past-year alcohol use, and past-year cannabis use, which are potential confounders of the associations of depressive symptom severity with food attentional bias and/or eating behaviors. The models with individual depressive symptoms as the predictor variables were further adjusted for the remaining depressive symptoms (SCL-15). Separate models were run for each depressive symptom variable and each outcome variable. All models were conducted in Mplus version 8 (Muthén & Muthén, 2017), and FIML was used to handle missing data. To adjust for multiple

comparisons, we applied the Benjamini-Hochberg (1995) correction to each set of five analyses per outcome variable (e.g., five analyses for Food Stroop, five analyses for emotional eating).

3. Results

3.1 Participant Characteristics

Our sample consisted of 103 young adults (mean age: 20 years, 79% women, 26% non-White; see Table 1). Nearly three quarters (72%) lived independent of their family. Mean BMI (23.4 kg/m^2) fell in the normal weight category, and participants, on average, rated their state hunger at the laboratory session as 15.6 on a 0-100 scale. Mean AUDIT score for past-year alcohol use was 2.8 (low level of alcohol problems; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001), and 24% of participants reported past-year cannabis use. While participants, on average, reported mild depressive symptom severity (mean SCL-20 total score: 0.9), 8% had scores falling above the cut point (2.0) indicating severe depressive symptoms (Katon et al., 1995; Walker et al., 2000). The mean Food Stroop interference score (37 ms) was greater than zero, suggesting slight interference of food cues. Emotional eating, external eating, and restrained eating were all reported as occurring moderately frequently.

Table 1

Characteristics of Participants (N = 103)

Background Factors	
Age, years	19.9 (2.2)
Sex, % female	79
Race/Ethnicity, % non-White	26
Living Independent of Family, %	72
BMI, kg/m^2	23.4 (2.8)
Subjective Hunger (possible range: 0-100)	15.6 (15.2)
Past-Year Alcohol Use, AUDIT Total Score (possible range: 0-40)	2.8 (3.8)
Past-Year Cannabis Use, %	24
Depressive Symptom Variables	
SCL-20 Total Score (possible range: 0.0-4.0)	0.9 (0.7)
SCL-15 Total Score (possible range: 0.0-4.0)	0.8 (0.7)
SCL-20 Hyperphagia Item Score (possible range: 0.0-4.0)	0.7 (1.1)
SCL-20 Hypersomnia Item Score (possible range: 0.0-4.0)	0.8 (1.1)
SCL-20 Poor Appetite Item Score (possible range: 0.0-4.0)	0.8 (1.0)
SCL-20 Disturbed Sleep Item Score (possible range: 0.0-4.0)	1.1 (1.2)
Food Attentional Bias and Eating Behaviors	
Food Stroop Interference Score, ms	37 (167)
DEBQ Emotional Eating Score (possible range: 1.0-5.0)	2.1 (0.9)
DEBQ External Eating Score (possible range: 1.0-5.0)	3.1 (0.7)
DEBQ Restrained Eating Score (possible range: 1.0-5.0)	2.0 (0.7)

Note. Continuous variables are presented as mean (standard deviation), and categorical variables are presented as percentage. BMI = body mass index; AUDIT = Alcohol Use Disorder Identification Test; SCL-20 = Hopkins Symptom Checklist-20; SCL-15 = Hopkins Symptom Checklist excluding the five sleep and appetite symptoms; DEBQ = Dutch Eating Behavior Questionnaire.

There was missing data on the following variables: race/ethnicity (n = 1), SCL-20 total score (n = 1), SCL-15 total score (n = 1), SCL-20 hypersomnia item score (n = 1), Food Stroop interference score (n = 6), and DEBQ emotional eating score (n = 2). Missing data was handled using full information maximum likelihood (FIML) in Mplus version 8 (Muthén & Muthén, 2017).

3.2 Food Attentional Bias

In separate regression models (adjusted for age, sex, race/ethnicity, residential status, BMI, state hunger, past-year alcohol use, past-year cannabis use, and SCL-15 total score) for each depressive symptom variable (see Figure 1), we found that hyperphagia was negatively associated with food attentional bias, as indicated by Food Stroop interference score (β = -0.31, p = .01). We also found that disturbed sleep was positively associated with food attentional bias (β = 0.34, p = .01). Poor appetite, hypersomnia, and the SCL-15 total score were not associated with food attentional bias (all ps ≥ .44).

3.3 Emotional Eating

Separate adjusted regression models (see Figure 1) revealed that hyperphagia was positively associated with emotional eating as measured by the DEBQ (β = 0.48, p < .001), whereas poor appetite was negatively associated (β = -0.48, p < .001). Conversely, hypersomnia was negatively associated with emotional eating (β = -0.19, p = .04), while disturbed sleep was positively associated (β = 0.21, p = .03). In addition, the SCL-15 total score was positively associated with emotional eating (β = 0.29, p = .006).

3.4 External Eating

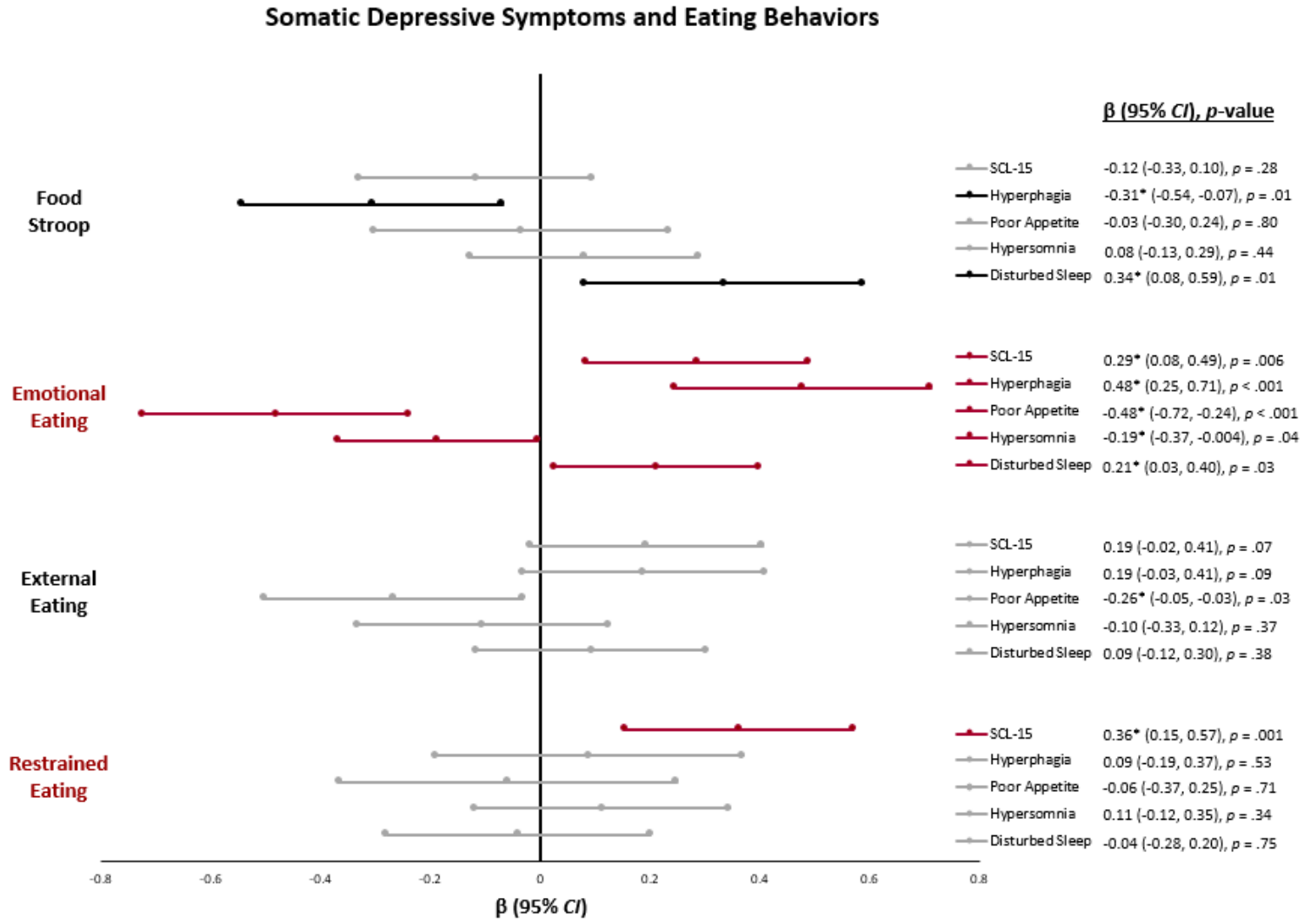
Separate adjusted regression models (see Figure 1) indicated that the association between poor appetite and external eating as measured by the DEBQ fell short of significance after the Benjamini-Hochberg (1995) correction (β = -0.26, p = .03). Hyperphagia, hypersomnia, disturbed sleep, and the SCL-15 total score were not associated with external eating (all ps ≥ .07).

3.5 Restrained Eating

Separate adjusted regression models (see Figure 1) showed that only the SCL-15 total score was positively associated with restrained eating as measured by the DEBQ ($\beta = 0.36$, $p = .001$). None of the individual depressive symptoms (hyperphagia, hypersomnia, poor appetite, and disturbed sleep) were associated with restrained eating (all ps $\geq .34$).

Figure 1

Associations of Somatic Depressive Symptoms with Food Stroop and Eating Behaviors



Note. Forest plot summarizing the results of linear regression models with the depressive symptom variables as the predictor variables and the food attentional bias and eating behavior variables as the outcome variables. All models were adjusted for age, sex, race/ethnicity, residential status, BMI, state hunger, past-year alcohol use, and past-year cannabis use. The models with individual depressive symptoms as the predictor variables were further adjusted for the remaining depressive symptoms (SCL-15). *$p < .05$. SCL-15 = Hopkins Symptom Checklist excluding the five sleep and appetite symptoms; β = standardized regression coefficient; *CI* = confidence interval.

4. Discussion

Results of the present study suggest that individual depressive symptoms have differential associations with food attentional bias, emotional eating, external eating, and restrained eating – candidate mechanisms underlying the depression-to-obesity relationship. Some of the observed associations were expected, while others were not. Hyperphagia was associated with greater emotional eating (expected) but not with the other eating behaviors; it was also associated with lower food attentional bias (unexpected). In general, hypersomnia was not associated with the outcomes, but it did have a negative relationship with emotional eating (unexpected). Poor appetite was associated with lower emotional eating (expected) and was not related to food attentional bias, external eating, or restrained eating (unexpected). In contrast, disturbed sleep was associated with greater food attentional bias and emotional eating (unexpected). Finally, other depressive symptoms (captured by the SCL-15) were positively associated with emotional eating and restrained eating (expected); however, they were not associated with food attentional bias or external eating (unexpected). Altogether, our findings raise the possibility that certain subgroups of people with elevated depressive symptoms – i.e., those experiencing hyperphagia and/or disturbed sleep – may be at higher risk of developing obesity over time than other subgroups – i.e., those experiencing hypersomnia and/or poor appetite.

The present study is among the first to examine associations of individual depressive symptoms – namely, hyperphagia, hypersomnia, poor appetite, and disturbed sleep – with food attentional bias and eating behaviors. The observed associations between individual depressive symptoms and performance on the Food Stroop task conflict with the limited available literature on depression and food attentional bias. Hawkins and colleagues (2018) detected a positive

association between somatic depressive symptoms and food attentional bias as measured by reaction time on a dot-probe task. Thus, it is surprising that we found a negative association between hyperphagia and food attentional bias and no associations for hypersomnia, poor appetite, or disturbed sleep. However, Hawkins et al. (2018) were not able to examine the direction of somatic symptoms due to their use of the Patient Health Questionnaire-8. Even so, our finding that the remaining depressive symptoms (SCL-15) were not associated with Food Stroop interference scores is consistent with Hawkins et al.'s (2018) null association between total depressive symptom severity and food attentional bias. In sum, there is limited available research on depressive symptoms and food attentional bias. More research is warranted to determine which findings are reproducible.

Our findings regarding associations of appetite and sleep-related depressive symptoms with eating behaviors demonstrate mixed consistency with the small extant literature. First, hyperphagia's association with emotional eating and lack of association with restrained eating and external eating demonstrate mixed consistency with studies conducted by Paans and colleagues (Paans, Bot, Brouwer, et al., 2018; Paans, Bot, van Strien, et al., 2018). There are at least two plausible reasons why hyperphagia is positively associated with emotional eating. First, the presence of hyperphagia may increase one's likelihood of subsequently eating in response to negative mood. Second, there may be construct and/or measurement overlap between hyperphagia and emotional eating (e.g., eating in response to negative mood may increase one's likelihood of self-reporting "overeating" [hyperphagia]). Next, poor appetite's negative association with emotional eating is consistent with Paans, Bot, van Strien, et al.'s (2018) results. Thus, the presence of poor appetite may protect against eating in response to negative mood and, perhaps, future obesity.

Third, hypersomnia's null associations with external eating and restrained eating are consistent with Paans, Bot, van Strien, et al.'s (2018) results. In contrast, disturbed sleep's positive association with emotional eating conflicts with Paans and colleagues' (2018a and 2018b) null findings. However, sleep disturbance may be an important factor to consider in the relationship between depression, emotional eating, and obesity. Konttinen (2019) found that emotional eating mediated depression's relationships with BMI and waist circumference. Furthermore, people who engaged in emotional eating and had short sleep duration were at higher risk of increasing BMI and waist circumference compared with those who had longer sleep duration. Short sleep duration may lead to increased appetite and obesity development through decreased leptin and increased ghrelin (Tobaldini et al., 2019).

Finally, our findings regarding total depressive symptom severity (SCL-15) are consistent with most of the extant literature (Antoniou et al., 2017; Hawkins et al., 2018; Lazarevich et al., 2016; Ouwens et al., 2009; Paans, Bot, Brouwer, et al., 2018; Brechan & Kvalem, 2015; van Strien et al., 2016; Werrij et al., 2006), although our findings conflict with a few restrained eating reports (Clum, Rice, Broussard, Johnson, & Webber, 2014; Paans, Bot, Brouwer, et al., 2018; Paans, Bot, van Strien, et al., 2018). In sum, our work adds to a growing literature that highlights the importance of examining the direction of somatic depressive symptoms (e.g., increased versus decreased appetite) in investigations that seek to understand relationships between depression and obesity-promoting eating behaviors. Certain symptoms (e.g., hyperphagia and disturbed sleep) may be key predictors in future prospective studies and key intervention targets in future clinical trials, while other symptoms (e.g., hypersomnia) do not appear as important from an obesity risk and prevention perspective.

While the present study has several strengths (laboratory study, examination of individual depressive symptoms, and assessment of multiple candidate mechanisms of the depression-to-obesity relationship), it also has limitations. First, because the study design is cross-sectional, we cannot determine the directionality of the observed associations. Reverse causality is plausible for some of the relationships of interest, as emotional eating, external eating, and restrained eating could lead to increases in depressive symptoms. Supporting this possibility is meta-analytic evidence indicating that eating pathology – such as bulimia nervosa, binge eating disorder, and anorexia nervosa – has a bi-directional relationship with depression (Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016). Second, although a young adult sample is appropriate for achieving our study objective (due to an increase in prevalence of depressive symptoms, autonomy over food choices, and disordered eating), it is not representative of the U.S. population with respect to predisposing factors for obesity, such as low socioeconomic status or limited access to healthy foods (Benjamin et al., 2017; Hales, Carroll, Fryar, & Ogden, 2017; McLaren, 2007; USDA, 2010). Third, although we utilized valid assessments to measure food attentional bias and eating behaviors, some have questioned their predictive validity for food consumption in daily life (Jansen et al., 2011). Future studies should consider using more ecologically valid food attentional bias tasks, such as virtual reality with eye tracking, and/or laboratory food consumption tasks in response to negative emotions or environmental food cues, which may have greater predictive validity.

4.1 Conclusion

The present study sought to elucidate whether the individual symptoms of hyperphagia, hypersomnia, poor appetite, and disturbed sleep have differential relationships with food attentional bias and eating behaviors. Our results suggest that young adults who experience

hyperphagia and/or disturbed sleep may engage in more emotional eating, which has been associated with obesity development (van Strien et al., 2016). This work highlights the importance of future studies examining the direction of somatic depressive symptoms in an effort to identify subgroups of people with depression at greatest obesity risk (e.g., those with hyperphagia and/or disturbed sleep), the mechanisms responsible for this elevated risk (e.g., emotional eating), and effective interventions to prevent obesity in these at-risk subgroups.

References

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

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. 10.1016/j.eatbeh.2017.01.007

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*. 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*. World Health Organization.

Baños, R. M., Cebolla, A., Moragrega, I., Van Strien, T., Fernández-Aranda, F., Aguera, Z., de la Torre, R., Casanueva, F. F., Fernández-Real, J. M., Fernández-Garcia, J. C., Frühbeck, G., Gómez-Ambrosi, J., Jiménez-Murcia, S., Rodríguez, R., Tinahones, F. J., & Botella, C. (2014). Relationship between eating styles and temperament in an anorexia nervosa, healthy control, and morbid obesity female sample. *Appetite, 76*, 76-83. 10.1016/j.appet.2014.01.012

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

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., de Ferranti, S. D., Floyd, J., Fornage, M., Gillespie, C., Isasi, C. R., Jiménez, M. C., Jordan, L. C., Judd, S. E., Lackland, D., Lichtman, J. H., Lisabeth, L., Liu, S., Longenecker, C. T., Mackey, R.

H., . . . Muntner, P. (2017). Heart disease and stroke statistics—2017 update: a report from the American Heart Association. *Circulation*. 10.1161/cir.0000000000000485

Benjamini, Y. & Hochberg, Y. (1995). Controlling the false discovery rate: a practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B (Methodological), 57*(1), 289-300. 10.1111/j.2517-6161.1995.tb02031.x

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. 10.1016/j.brainres.2010.04.003

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. 10.1038/sj.mp.4001873

Brechan, I., & Kvalem, I. L. (2015). Relationship between body dissatisfaction and disordered eating: Mediating role of self-esteem and depression. *Eat Behav, 17*, 49-58. 10.1016/j.eatbeh.2014.12.008

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. 10.1038/ijo.2009.138

Center for Collegiate Mental Health. (2017). *2016 Annual Report* (STA 17-74).

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. 10.1002/da.22098

Clum, G. A., Rice, J. C., Broussard, M., Johnson, C. C., & Webber, L. S. (2014). Associations between depressive symptoms, self-efficacy, eating styles, exercise and body mass index in women. *J Behav Med, 37*(4), 577-586. 10.1007/s10865-013-9526-5

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. 10.1016/0005-7967(94)90128-7

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. 10.1159/000395070

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

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. 10.1016/j.jad.2015.06.024

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. 10.1016/j.drugalcdep.2007.09.014

Han, S., Lee, S. (2017). College student binge eating: Attachment, psychological needs satisfaction, and emotion regulation. *J Coll Stud Dev, 58*(7), 1074-1086. 10.1353/csd.2017.0084

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. 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. *Obes Rev, 16*(5), 424-432. 10.1111/obr.12265

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. 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. 10.1016/j.appet.2011.01.019

Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with c-reactive protein, IL-1, and IL-6: a meta-analysis. *Psychosom Med, 71*(2), 171-186. 10.1097/PSY.0b013e3181907c1b

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. *Biol Psychiatry, 61*(3), 348-358. 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. *Alcohol Clin Exp Res, 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. 10.1002/eat.20799

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

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. 10.3109/00048679609065053

Konttinen, H., van Strien, T., Männistö, S., Jousilahti, P., & Haukkala, A. (2019). Depression, emotional eating and long-term weight changes: a population-based prospective study. *Int J Behav Nutr Phys Act, 16*(1), 28. 10.1186/s12966-019-0791-8

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. *Psychol Med, 48*(7), 1102-1110. 10.1017/S0033291717002483

Lasserre, A. M., Glaus, J., Vandeleur, C. L., Marques-Vidal, P., Vaucher, J., Bastardot, F., Waeber, G., Vollenweider, P., & 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. 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. 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. 10.1017/s0033291713001438

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

Majd, M., Saunders, E. F. H., & Engeland, C. G. (2020). Inflammation and the dimensions of depression: a review. *Front Neuroendocrinol, 56*, 100800. 10.1016/j.yfrne.2019.100800

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

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. 10.1016/j.genhosppsych.2009.05.013

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

Milaneschi, Y., Lamers, F., Berk, M., & Penninx, B. W. J. H. (2020). Depression heterogeneity and its biological underpinnings: Toward immunometabolic depression. *Biol Psychiatry*. 10.1016/j.biopsych.2020.01.014

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. 10.1016/j.biopsych.2015.10.023

Milaneschi, Y., Simmons, W. K., van Rossum, E. F. C., & Penninx, B. W. (2019). Depression and obesity: Evidence of shared biological mechanisms. *Mol Psychiatry, 24*(1), 18-33. 10.1038/s41380-018-0017-5

Mueller, S. T., & Piper, B. J. (2014). The Psychology Experiment Building Language (PEBL) and PEBL Test Battery. *J Neurosci Methods, 222*, 250-259. 10.1016/j.jneumeth.2013.10.024

Murru, A., Guiso, G., Barbuti, M., Anmella, G., Verdolini, N., Samalin, L., Azorin, J. M., Angst, J. J., Bowden, C. L., Mosolov, S., Young, A. H., Popovic, D., Valdes, M., Perugi, G., Vieta, E., & Pacchiarotti, I. (2019). The implications of hypersomnia in the context of major depression: Results from a large, international, observational study. *Eur Neuropsychopharmacol*. 10.1016/j.euroneuro.2019.02.011

Muthén, L. K., & Muthén, B. O. (2017). *Mplus User's Guide* (8 edition). Muthén & Muthén.

National Institute 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. T., & Franken, I. H. A. (2012). Attentional processing of food cues in overweight and obese individuals. *Curr Obes Rep, 1*(2), 106-113. 10.1007/s13679-012-0011-1

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. 10.1016/j.appet.2009.06.001

Paans, N. P. G., Bot, M., Brouwer, I. A., Visser, M., Roca, M., Kohls, E., Watkins, E., & Penninx, B. W. J. H. (2018). The association between depression and eating styles in four European countries: The MooDFOOD prevention study. *J Psychosom Res, 108*, 85-92. 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. *J Psychiatr Res, 97*, 38-46. 10.1016/j.jpsychires.2017.11.003

Penninx, B. W. J. H., 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. 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. 10.1038/oby.2010.138

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. 10.1093/aje/kwx030

Puccio, F., Fuller-Tyszkiewicz, M., Ong, D., & Krug, I. (2016). A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *Int J Eat Disord, 49*(5), 439-454. 10.1002/eat.22506

Scarpace, P. J., & Zhang, Y. (2009). Leptin resistance: a prediposing factor for diet-induced obesity. *Am J Physiol Regul, 296*(3), R493-R500. 10.1152/ajpregu.90669.2008

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

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

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. 10.1016/j.appet.2004.12.001

Tobaldini, E., Fiorelli, E. M., Solbiati, M., Costantino, G., Nobili, L., & Montano, N. (2019). Short sleep duration and cardiometabolic risk: From pathophysiology to clinical evidence. *Nat Rev Cardiol, 16*(4), 213-224. 10.1038/s41569-018-0109-6

Tuschl, R. J. (1990). From dietary restraint to binge eating: Some theoretical considerations. *Appetite, 14*(2), 105-109. 10.1016/0195-6663(90)90004-R

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. *Neurosci Biobehav Rev, 37*(3), 279-299. 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. *Int J Eat Disord, 5*, 295-315.

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. 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. 10.1016/j.appet.2016.02.034

Walker, E. A., Katon, W. J., Russo, J., Von Korff, M., Lin, E., Simon, G., Bush, T., Ludman, E., & Unützer, J. (2000). Predictors of outcome in a primary care depression trial. *J Gen Intern Med, 15*(12), 859-867. 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. 10.1016/0022-3999(87)90072-9

Werrij, M. Q., Mulkens, S., Hospers, H. J., & Jansen, A. (2006). Overweight and obesity: the significance of a depressed mood. *Patient Educ Couns, 62*(1), 126-131. 10.1016/j.pec.2005.06.016

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. 10.1037/a0024291

Williams, J. M., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychol Bull, 120*(1), 3-24. 10.1037/0033-2909.120.1.3

Zelinski, E. L., Deibel, S. H., & McDonald, R. J. (2014). The trouble with circadian clock dysfunction: multiple deleterious effects on the brain and body. *Neuroscience & Biobehavioral Reviews, 40*(2014), 80-101. 10.1016/j.neubiorev.2014.01.007