

THE NATURE OF NIGHT EATING SYNDROME: USING NETWORK ANALYSIS TO  
UNDERSTAND CAUSAL SYMPTOMOLOGICAL RELATIONSHIPS

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# THE NATURE OF NIGHT EATING SYNDROME: USING NETWORK ANALYSIS TO UNDERSTAND CAUSAL SYMPTOMOLOGICAL RELATIONSHIPS

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University of Missouri-Kansas City, 2019

## ABSTRACT

The purpose of this study was to examine and characterize the core symptoms in a psychopathology network for night eating syndrome (NES) among persons with and without overweight/obesity, and to compare the network density between samples with and without NES. The current conceptualization of NES, and interventions based on that conceptualization, are grounded in a latent variable, medical model nosology and theory of etiology. A recently developed advanced statistical technique, network analysis, allows for better understanding of the functional relationship among symptoms of behavioral health disorders. Network analysis has been applied to other eating disorders, but no studies to date have used network analysis to understand the functional relationship among proposed core symptoms of NES. Data from 148 individuals with NES and 69 individuals without NES were used to evaluate the unique variance among core symptoms and identify key symptom relationships. It was hypothesized that nocturnal ingestion of food and evening hyperphagia would present as the most central symptoms within the psychopathology network of NES compared to other symptoms. A Gaussian graphical model (GGM), utilizing a graphical least absolute shrinkage and selection operator (GLASSO) method, was used to estimate network. Symptoms with high centrality indices were assessed via bootstrapped difference tests.

Results indicated that depressed mood and a strong urge to eat upon awakening at night were highly central to the psychopathology network for NES and were significantly more central than most other NES symptoms. These symptoms represent key elements of the core psychopathology of NES and should represent primary treatment targets for intervention.

## APPROVAL PAGE

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## CHAPTER 1

### INTRODUCTION

Psychiatric nosology has been dominated by the medical model paradigm of disease, which conceptualizes mental health disorders as causal entities that result in myriad dysfunctional symptoms (Borsboom & Cramer, 2013). For centuries, this has been the prevailing model for conceptualizing psychopathology (Lilienfeld & Treadway, 2016), although it is not without limitations. Whereas an organic medical condition, such as a tumor, may present asymptotically, such a disease is detectable and thought to exist regardless of symptom presentation. The same cannot be said about psychiatric disorders; one cannot present with depression without experiencing symptomatology related to depression (Borsboom & Cramer, 2013). This medical model, however, assumes the existence of an underlying cause of a psychiatric disorder that both leads to the expression of symptoms and explains why certain symptoms tend to appear in related clusters (e.g., depressed mood, fatigue, concentration difficulties, etc.; Guze, 1992). The network theory of psychopathology, an alternative approach to the conceptualization of psychiatric disorders, reverses the assumptions of the medical model paradigm, and posits that psychiatric disorders are the result of a complex functional network of symptoms that cause each other (Borsboom & Cramer, 2013). This theory can be both modeled and tested using a statistical approach known as network analysis. Network analysis can be used to identify core symptoms that are likely to propagate additional symptoms, which in turn leads to the manifestation of a psychiatric disorder (Borsboom & Cramer, 2013; Epskamp, Borsboom, & Fried, 2018).

This network theory approach to psychopathology has been applied to a wide variety of psychiatric disorders (For a review, see Borsboom, 2017), including eating disorders

(Levinson, Vanzhula, Brosos, & Forbush, 2018). A review of the literature on the application of network theory in this population, however, reveals a dearth of research on the use of network analysis among individuals with night eating syndrome (NES), an eating disorder currently conceptualized through the lens of the medical model paradigm. NES is also an eating disorder that is more prevalent among persons with overweight/obesity (Adami, Campostano, Marinari, Ravera, & Scopinaro, 2002; Cerú-Björk, Andersson, & Rössner, 2001), although not all individuals with NES are persons with overweight/obesity (Birketvedt et al., 1999, de Zwaan, Roerig, Crosby, Karaz, & Mitchell, 2006, Lundgren, Allison, O'Reardon, & Stunkard, 2008; Marshall, Allison, O'Reardon, Birketvedt, & Stunkard, 2004; Striegel-Moore, Franko, Thompson, Affenito, & Kraemer, 2006). As such, this study aimed to examine and characterize the core symptoms in the psychopathology network of NES among persons with and without overweight/obesity, and to compare psychopathology networks between samples with and without NES.

This study, the first of its kind among the eating disorder literature, used data from a previous National Institutes of Health (NIH) funded outpatient study that examined the characteristics of NES to present an alternative conceptualization of the core psychopathology of NES as a causally related network of symptoms, rather than the result of an underlying latent cause. As the medical model paradigm dictates not only the current nosological and etiological conceptualization of NES, but its current intervention strategies as well, a re-examination of the complex network of symptoms within NES may lead to improvements in future research and the refinement of treatment options that target core symptoms within this causal network.



This dissertation is organized as follows: chapter 2 presents a review of the literature on psychiatric nosology, from its early roots to modern psychopathology conceptualization; a thorough examination of the network theory of psychopathology, including the various approaches to model this theory quantifiably and graphically using network analysis; an outline of the literature on NES, including its history from first description to current diagnostic criteria; and a review of the use of network analysis with other eating disorders, culminating in the specific aims for this study. Chapter 3 presents the methodology of this study, including participant characteristics, recruitment and data collection procedures, and an overview of the measures used. This chapter concludes with an explanation of the specific hypotheses generated by the aims of this study and how the proposed statistical methods would test these hypotheses. Chapter 4 presents the results of the study, broken down by aim and hypothesis. Lastly, chapter 5 presents a discussion of this study, including a summary and interpretation of the findings, an examination of the clinical implications of this study, the unique limitations and strengths of the present study, and proposed future directions.

## CHAPTER 2

### REVIEW OF THE LITERATURE

#### **What is a Psychiatric Disorder?**

The term “mental or psychiatric disorder” has been variably defined in the psychopathology literature (Stein, Phillips, Bolton, Fulford, Sadler, & Kendler, 2010). The most recent edition of the Diagnostic and Statistical Manual of Mental Disorders (5<sup>th</sup> ed.; DSM-5; American Psychiatric Association, 2013) lists several factors that characterize a psychiatric disorder, including that a disorder is (a) a syndrome (either behavioral or psychological) that reflects some form of clinically significant disturbance or dysfunction, (b) is associated with significant distress or impairment in some portion of an individual’s life, (c) is not an expected or culturally appropriate response to a stressor or event (i.e., death of a loved one, religious trance state), and (d) does not include socially deviant behavior not accounted for by the above.

Since its third edition, the DSM has claimed to be an atheoretical description and classification tool, such that it does not subscribe to a particular stance on the etiology of psychiatric disorders (Castiglioni & Laudisa, 2015). At the same time, an examination of the DSM system reveals that this psychiatric nosology has been heavily influenced by a medical model, not only in that it organizes psychiatric disorders into discrete categories as medicine does with disease, but that it assumes that these symptoms arise due to a shared underlying cause: the “disorder itself”. This medical model, the dominant paradigm of Western medicine (Hyland, 2011), is not new to the DSM, and has been the prevailing mindset for psychiatric nosology for the past century (Lilienfeld & Treadway, 2016).

## **Early Psychiatric Nosology**

In the early nineteenth century, physicians developed rough categories of psychiatric nosology (e.g., diagnoses based on neuropathology and symptom patterns; Kahlbaum, 1863/1996). In the late nineteenth to early twentieth century, these rudimentary classification systems became much more refined (Shorter, 2013). Specifically, psychiatric nosology was dominated by the paradigm of “the great professor principle”, wherein prominent individuals developed their own nosologic systems. For example, Emil Kraepelin proposed that several clinical syndromes could be grouped into major psychoses (i.e., bipolar and schizophrenia). Indeed, his nosological system of symptoms, signs, and natural history became adopted by practitioners worldwide (Kendler, 1990; Lilienfeld & Treadway, 2016; Mullen, 2006).

As the twentieth century progressed, there was a paradigmatic shift away from personal authority to “the consensus of experts.” From this shift emerged a psychiatric nosology that was dictated by committees appointed by national or international organizations. One result of this was the formation of the first and second edition of the DSM (Kendler, 1990). These versions of the DSM consisted of vague descriptions of pathology, with little in the way of inter-rater reliability (Lilienfeld & Treadway, 2016). Moreover, early editions of the DSM were the result of a compromise of conflicted standpoints based on historical and current diagnostic usage, personal opinions, and clinical experiences. For example, the conflicting influences of Kraepelinian perceptions for psychoses and Freudian perspectives for neuroses were the foundation of DSM-II (Kendler, 1990).

Another paradigmatic shift occurred during the development of the DSM-III. The appointed committee of experts for this edition (St. Louis psychiatric group at Washington University) took a conscientious effort to utilize the scientific knowledge of the time to

inform nosologic changes. As such, the addition of explicit diagnostic criteria for 14 major mental disorders was made to facilitate diagnostic reliability and validity, improve communication between providers/researchers, and to improve accurate diagnosis and treatment through a defined clinical structure (Kendler, 1990; Lilienfeld & Treadway, 2016; Mullen, 2006). This approach has pervaded the DSM to this day, although debate still exists between diagnostic criteria, the use of categorization versus dimensionality, and the clinical utility of such nosology. Indeed, researchers have stated that “the increasingly obvious gaps between research findings and the definitions of mental disorders in today’s diagnostic manuals is beginning to raise questions about whether validity has been sacrificed on the altar of reliability” (Mullen, 2006, p. 113). In addition, previous attempts to identify sets of genes that cause psychiatric disorders have revealed that “genes do not read DSM” (Stefanis, 2008), and despite some heritability, genes’ effects on the risk of developing a psychiatric disorder appear rather small and non-specific to such disorders (Kendler, 2005).

### **Biopsychosocial Model and RDoC**

Several alternative approaches have been presented in an attempt to overcome the shortcomings of the DSM, or to provide an entirely new conceptualization of psychiatric nosology. Engel (1977) described the deficiencies of the biomedical model and biological reductionism (i.e., that psychiatric disorders and behaviors are biological in nature) and proposed a system that eventually developed into the biopsychosocial model.

Several decades later, Insel et al. (2010) developed the Research Domain Criteria (RDoC) as a new framework to “conceptualize mental illnesses as brain disorders” (p. 749). RDoC was designed to identify various fundamental behavioral functions and their underlying neural systems, and posited that psychopathology is the result of dysfunction that

occurs among these systems, and that this may span multiple disorders (Cuthbert & Insel, 2013). Indeed, the goal of RDoC has been to “facilitate the establishment of psychiatric nosology grounded in the neuroscience of human behavior” (Wildes & Marcus, 2015, p. 2). In 2013, Insel wrote a blog post reporting that the National Institute of Mental Health (NIMH) would be “re-orienting its research away from DSM categories” and focusing instead on RDoC (Insel, 2013).

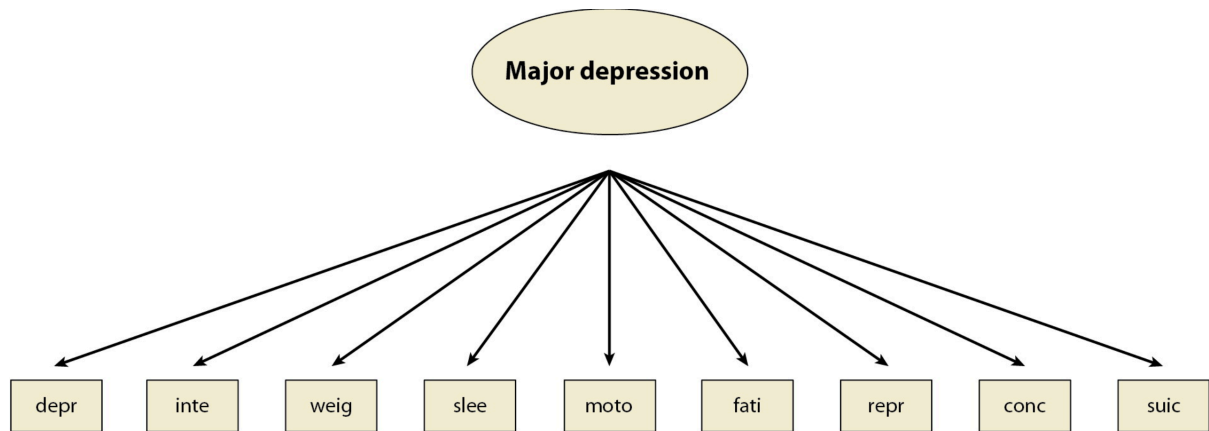
### **DSM Conceptualization Approach: Categories or Dimensions**

McNally (2016) described the categorical versus dimensional conceptualization of psychopathology debate that occurred during the development of DSM-5, which has been a large point of contention among experts. He stated that although both conceptualizations aim to explain what precipitates the co-occurrence of psychiatric symptoms differently, they both postulate the existence of an underlying common cause or factor; a “latent entity that causes [symptom] emergence and covariance” (McNally, 2016, p. 95).

### **Network Theory of Psychopathology**

One of the major weaknesses of the current conceptualization of psychiatric disorders is the reliance on a latent variable theory, or the belief that a common cause precipitates dysfunctional mental health symptoms. For example, major depression is posited as the manifestation of specific observable symptoms (e.g., depressed mood, loss of interest, fatigue, difficulty concentrating, etc.) that are caused by an underlying unobserved factor (see Figure 1). This is a carry-over from the previously described medical model nosology, wherein an individual may develop observable symptoms (e.g., bloody cough, shortness of breath, and angina), due to the presence of a tumor in the lungs; the underlying common cause of the overt symptoms (Borsboom & Cramer, 2013). While these are distinct

symptoms, they are causally homogeneous, and the removal of the tumor (the common cause) will result in symptom alleviation. Furthermore, in the medical model, symptoms are distinct from the underlying condition (i.e., conditions can occur without the presence of symptoms).



*Figure 1.* A latent variable theory model of major depressive disorder. Symptom label descriptions: depr = depressed mood; inte = loss of interest; weig = weight problems; slee = sleep problems; moto = psychomotor disturbances; fati = fatigue; repr = self-reproach; conc = concentration problems; suic = suicidal ideation. Adapted from “Network Analysis: An Integrative Approach to the Structure of Psychopathology,” by D. Borsboom and A. O. J. Cramer, 2013, *Annual Review of Clinical Psychology*, 9, p. 94. Copyright 2013 by Annual Reviews.

Borsboom and Cramer (2013) explored the medical model approach using an analogy of headaches and brain tumors. They state that one can experience a headache that is not caused by a tumor, or conversely, can present with a tumor without any observable headache symptoms (i.e., and asymptomatic tumor). Should a tumor be present, however, the suggested approach is to remove the tumor (the cause) rather than the headache (the symptom); treatment of the headache will not affect the tumor whatsoever.

Borsboom and Cramer (2013) argue that, applying this medical model approach to psychopathology, it stands to reason that a psychiatric disorder like depression should both (a) have an underlying common cause that is independent from the symptoms, and (b) this underlying common cause should cause all related depressive symptoms. Therefore, such a theory posits that an individual could have depression without any symptoms (e.g., feeling blue or loss of interest), or panic disorder without panic attacks. It is highly unlikely that these scenarios exist, and it remains unlikely that any future measurement techniques will be developed to detect disorders independent of their symptoms. Indeed, the medical model of disease as the cause of symptoms does not appear to hold true with psychiatric disorders, and the relationship between symptoms and disorders needs to be conceptualized differently (Borsboom & Cramer, 2013).

An additional assumption in the latent variable conceptualization of psychopathology is that symptoms do not appear to co-occur randomly, and certain symptoms appear in select syndrome clusters more often than not (i.e., symptoms of depression are more likely to occur together than with random symptoms from other diagnoses; Borsboom & Cramer, 2013). This is again assumed due to the presence of an underlying latent cause and supports the delineation of separate disorders into separate categories. This model again fails because symptoms may appear to be causally related to one another, such as those that arise from adverse life events (e.g., death of a loved one causing rumination, which leads to insomnia, further causing fatigue, which in turn leads to concentration difficulties, leading to irritability, and finally anhedonia and sadness). Symptoms can also present with a different causal path depending on the life event (e.g., an individual experiences a myocardial infarction and develops somatic symptoms; de Jonge et al., 2006). In addition, the covariance

among clusters of symptoms does not appear to be explained by a common cause, which violates the axiom of independence assumption inherent to the latent variable theory (that “correlations among symptoms must disappear once one conditionalizes on the latent variable”; McNally, 2016, p. 96).

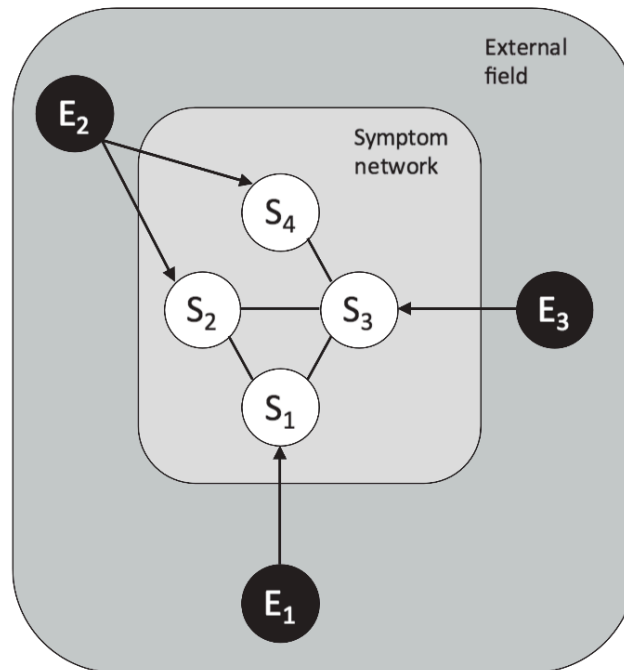
The dominance of the latent variable theory in psychiatric nosology is no accident; it is reinforced as the dominant theory due to the methodological constraints presented by traditional psychometric perspectives that mirror this theory (Borsboom & Cramer, 2013). In these psychometric designs, observed variables (i.e., symptoms) are thought to be caused by unobserved, latent variables (i.e., disorders), and such symptoms are combined to represent an overall score on the latent variable (Borsboom 2008a; Borsboom 2008b; Borsboom, Mellenbergh, & van Heerden, 2003).

Network theory presents an alternative approach to this conceptualization. It posits that symptoms are not the result of an underlying latent cause, but that psychiatric disorders result from a functional, causal interaction among symptoms (i.e., symptoms cause each other; Borsboom & Cramer, 2013; Borsboom, 2017). This interaction is conceptualized as connections between, and dynamic relationships among, a network of symptoms. This has been referred to as the “network approach to psychopathology (Borsboom, 2017). Indeed, Borsboom and Cramer (2013) state that

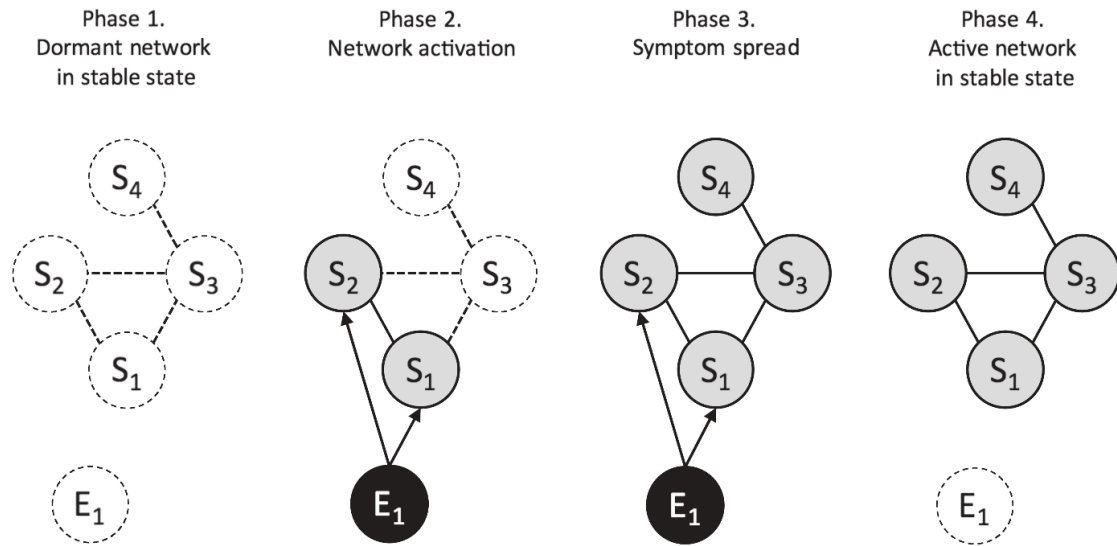
The heart of this approach lies precisely in what separates medical conditions from mental disorders: the general idea that causal, meaningful relations between symptoms not only exist and should be acknowledged, but in fact are the very stuff which mental disorders are made (p. 96).



Figure 2 illustrates this approach through a visual representation of a proposed network structure. The external field represents circumstances from outside the network that have direct influences on symptoms (i.e., adverse life events such as death of a loved one or a traumatic event). These circumstances can “activate” symptoms within the network, which in turn may activate neighboring symptoms, leading to the presentation of depression, post-traumatic stress, or so on (Borsboom & Cramer, 2013). Some symptoms may be more strongly connected to others, or may be clustered near select symptoms, as these symptoms are likely to impact each other to a greater degree. Indeed, such symptom connections can lead to the point where symptoms become self-sustaining and the disorder persists even with the removal of the original influence (see Figure 3). Conversely, symptoms that are weakly connected or are further apart from each other are unlikely to propagate the initial activation of symptoms and will likely not be self-sustaining once the external influence is removed. These networks are referred to as vulnerable and resilient networks, respectively (Borsboom, 2017).



*Figure 2.* A symptom network comprised of four symptoms. External factors (e.g., adverse events such as loss of loved one) exist in the external field and can directly affect one or multiple symptoms. Symptoms that tend to directly activate one another are connected by a line. Adapted from “A Network Theory of Mental Disorders,” by D. Borsboom, 2017, *World Psychiatry*, 16, p. 6. Copyright 2017 by World Psychiatric Association.



*Figure 3.* The development of psychiatric disorders as posited by network theory. Phase 1 represents a dormant network, wherein there are no symptoms present. An external event occurs that triggers the activation of select symptoms (phase 2), which spreads as it activates adjacent connected symptoms (phase 3). Strongly connected symptoms may maintain the network long after the external event is removed (phase 4), thus perpetuating a self-sustaining active network of symptoms. Adapted from “A Network Theory of Mental Disorders,” by D. Borsboom, 2017, *World Psychiatry*, 16, p. 9. Copyright 2017 by World Psychiatric Association.

### **Network Theory and Comorbidity**

As mentioned previously, one approach to diagnostic conceptualization is a categorical approach, which emphasizes those symptoms that represent the core attributes of a diagnosis and that are rarely associated with other diagnoses. Given the number of nonspecific symptoms (i.e., associated with a disorder, but not among its core symptoms) present in each disorder, one issue with this approach is the high levels of comorbidity among diagnoses (McNally, 2016). Spitzer, First, and Wakefield (2007) proposed that these

nonspecific symptoms be removed, so as to allow only core symptoms for each diagnosis. Network theory, however, conceptualizes comorbidity differently, such that “comorbidity is the result of a direct relationship between the symptoms of multiple disorders” (Levinson et al., 2018, p. 67), and views nonspecific symptoms as equally important to these core symptoms (Cramer, Waldorp, van der Maas, & Borsboom, 2010). These nonspecific symptoms are represented as *bridge symptoms*, which causally link two or more diagnoses. Although symptom-symptom interactions may be most active among core symptoms, this pattern of activation is not limited to traditional DSM diagnostic boundaries and it is likely that bridge symptoms will be activated at some point in the network that would establish and maintain comorbidity (Borsboom, 2017). Figure 4 provides an example of the comorbidity between major depressive disorder and generalized anxiety disorder and identifies specific bridge symptoms within the network.

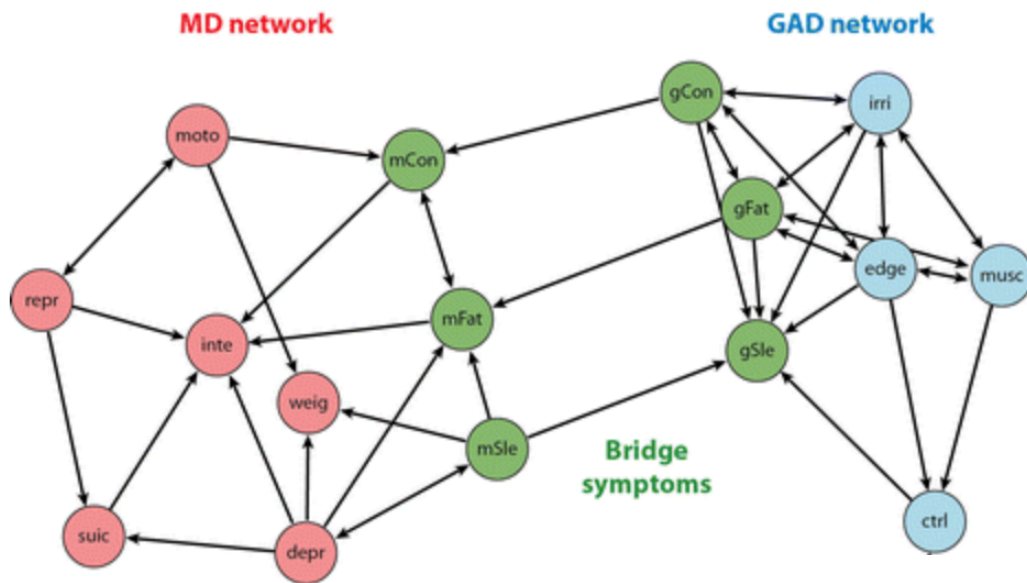


Figure 4. A symptom network of major depressive disorder (MD) and generalized anxiety disorder (GAD). Nonspecific symptoms that overlap/occur within the two disorders (e.g., difficulty sleeping, fatigue, concentration difficulties) are represented as bridge symptoms. Symptom label descriptions: depr = depressed mood; inte = loss of interest; weig = weight problems; slee = sleep problems; moto = psychomotor disturbances; fati = fatigue; repr = self-reproach; conc = concentration problems; suic = suicidal ideation; irri = irritable; edge = feeling on edge; musc = muscle tension; ctrl = no control over anxiety; g(symptom) = bridge symptom belonging to GAD; m(symptom) = bridge symptom belonging to MD. Adapted from “Network Analysis: An Integrative Approach to the Structure of Psychopathology,” by D. Borsboom and A. O. J. Cramer, 2013, *Annual Review of Clinical Psychology*, 9, p. 109. Copyright 2013 by Annual Reviews.

## **Advantages of Network Theory**

Network theory has an advantage over the more traditional latent variable theory in that it more closely models how clinicians instinctively conceptualize and diagnose psychopathology (Borsboom, 2017). An examination of a disorder through network theory involves the conceptualization of a network where one identifies which symptoms are present and which interactions are sustaining each other. In other words, one would ideally identify the most important (i.e., the most influential to others or the most likely to maintain the activation of others) symptom as a target for treatment. In addition, this model is well-aligned with contemporary cognitive-behavioral models of psychopathology, in that one acknowledges how patterns of thoughts, behaviors, and emotions might be self-reinforcing and thus perpetuating a disorder (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017). Therefore, interventions would be geared more toward manipulating or interrupting a network of symptoms, rather than identifying and addressing an underlying cause (Borsboom, 2017).

## **Use of Network Analysis to Explore Network Structure**

Network theory can be both graphically and quantitatively modeled using a method known as network analysis (Borsboom & Cramer, 2013; Epskamp, Borsboom, et al., 2018). This method is a data-driven approach used to determine the network structure of a particular disorder. Within a given network, symptoms are represented as *nodes*, and the connection between symptoms is referred to as an *edge*. These edges can either represent the presence of a correlation (unweighted) or can be used to indicate both the presence and the magnitude of the correlation (weighted). In a weighted network, the size of an edge denotes the strength of the correlation (thicker lines indicate stronger correlations, and vice versa), and the color

reflects the sign of the correlation (typically green or blue lines indicate positive correlations, and red lines indicate negative correlations). In addition, in some network models, edges can also be used to determine the directionality of relationships between symptoms, with directed models using arrows on the edges to represent the direction of the association, and undirected models using only lines.

Network analysis can be both used to model the network structure of a disorder and to identify the core symptoms that are most important to maintaining the structure of the network (Borsboom & Cramer, 2013). These core symptoms are identified by assessing centrality indices, which work by determining the relative influence of specific nodes within the network. It should be noted, however, that centrality indices identify nodes that are of the greatest importance to the overall network structure (i.e., they are considered the driving or maintaining force in the network), and that these nodes do not necessarily have to represent hallmark symptoms of a disorder (i.e., depressed mood in major depressive disorder; Borsboom, 2017; McNally, 2016). Indeed, there are several indices used to determine centrality, all of which differ slightly in their approach to determining relative influence, but they all represent how closely related a specific symptom is to other symptoms within the network. Five such indices include *strength*, *betweenness*, *closeness*, *expected influence*, and *degree* (a new set of centrality indices exist to determine bridge symptoms empirically [i.e., nodes high on *bridge* centrality], but these will not be included in this section, as they are extensions of previously listed indices; Jones, 2018; Jones, Ma, & McNally, 2019). *Degree* is used primarily in unweighted networks, which are scarcely used thus far in the eating disorder literature, and only the remaining four will be outlined below.

*Strength* centrality represents how strongly a node is connected directly to other nodes in the network. This is measured by the sum of the edge weights between a particular node and all other nodes directly connected to it. A metaphor for this was made by Fried (2018), such that centrality is akin to a railway, with high strength centrality represented by a central station with the largest number of direct connections to it. This centrality index is considered highly important to psychopathology networks because it is likely that activation of symptoms (nodes) with high strength centrality will trigger the activation of other symptoms (McNally, 2016). This index tends to be the most used in interpretations of central symptoms of network structures, primarily due to it being the most stable centrality index (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012).

*Betweenness* centrality represents the degree to which a node connects other nodes. This is measured by assessing the shortest path between two nodes in a network and determining the number of times a particular node ends up on the shortest path between two other nodes. Using the railway metaphor, high betweenness centrality is represented by a central station that is considered an important transit station for all other stops (Fried, 2018). Nodes with high betweenness centrality tend to be bridge symptoms between comorbid disorders, and activation of these symptoms tends to spread to both disorders, leading to a comorbid presentation (McNally, 2016).

*Closeness* centrality represents how strongly a node is connected indirectly, or the average distance from one node to all other remaining nodes. In the railway metaphor, high closeness centrality is represented by a central station in the center of a country close to all other destinations (Fried, 2018). In psychopathology networks, symptoms with high



closeness centrality may have a greater impact on more symptoms directly, rather than through mediator symptoms (McNally, 2016).

*Expected influence* centrality arose as a response to limitations with the strength centrality index. While strength centrality is measured by the sum of the edge weights between a particular node and all other nodes directly connected to it, this method uses the absolute value of the edge weight, and issues can arise if there are negative edges present in a network. Expected influence takes the sign of the edge weights into account. When all edges in a network are positive, expected influence matches the strength centrality index, but it provides a more accurate measure of strength centrality as the number of negative edges in a network increases (McNally, 2016).

One important aspect regarding centrality indices is that, in order to be meaningfully interpreted, any high centrality values on a specific symptom must be statistically significantly different from centrality values on all other symptoms in the network (Epskamp, Borsboom, et al., 2018). This is assessed using bootstrapped difference tests, and if there are no significant differences among node centrality indices, these indices should not be interpreted (Epskamp, Borsboom, et al., 2018).

### **Approaches to Modeling Network Structure in Network Analysis**

There exist various approaches to modeling network structure using cross-sectional data (a brief synopsis of approaches with other types of data will follow below), with several approaches designed to allow researchers to model causal systems in order to characterize mental disorders (McNally, 2016). These approaches differ on their use of undirected versus directed models, and whether their edges represent zero-order correlations or partial correlations (Borsboom & Cramer, 2013).

**Association (zero order).** Association networks represent the most basic network analysis model, as they model zero-order correlations between nodes. These networks can be useful to determine the strength of correlations between symptoms, but since these correlations may be influenced by bi-directional relationships or third variable effects, these models are insufficient to determine causal inference (Borsboom & Cramer, 2013; McNally, 2016). These models also often include many small and possibly spurious correlations (Borsboom & Cramer, 2013).

**Concentration (partial correlation).** Concentration networks are far more commonly used models that utilize a matrix of partial correlations as a means to determine correlations that are representative of direct or indirect relationships, thus attempting to remedy the main limitation of association networks (Borsboom & Cramer, 2013). Networks that use partial correlations are also known as Gaussian graphical models (GGM; Lauritzen, 1996), and are part of a more general class of models called pairwise Markov random fields (Epskamp & Fried, 2018). As partial correlations are correlations between nodes that remain after all other nodes are controlled for, this approach may assist in determining the causal structure of a network. Indeed, any edges from an association network that remain in a concentration network are possibly part of such a causal structure (McNally, 2016). These networks, however, are vulnerable to sampling error, which may result in artificially inflated or spurious edge estimates (Epskamp & Fried, 2018).

**GLASSO (regularized partial correlation).** Thus far, both association and concentration networks are limited by the presence of many small and potentially spurious correlations. One strategy to solve these issues is the use of regularization (Epskamp & Fried, 2018). One such method is the least absolute shrinkage and selection operator technique

(LASSO; Tibshirani, 1996). This method reduces the overall number of edges by fixing a regularization penalty on small (and likely spurious) values, shrinking them to exactly zero and dropping these edges from the model. This leads to the estimation of a sparse model with relatively few edges used to explain the covariance in the model. Such parsimonious models are considered more interpretable (Epskamp, Borsboom, et al., 2018). This regularization penalty is set by a tuning parameter ( $\lambda$ ), although tuning parameters set too low or too high can both lead to inaccurate networks. Therefore, multiple networks are estimated with different tuning parameters. Among these networks, the best is selected through maximizing fit of the network to the data by minimizing the Extended Bayesian Information Criterion (EBIC; Chen & Chen, 2008; Epskamp & Fried, 2018). Regularization also accounts for small sample sizes commonly present in psychopathological literature (see Power section in Chapter 3), which are likely to produce more spurious edges (Epskamp, Borsboom, et al., 2018). These models can also be displayed graphically using a graphical LASSO (GLASSO) model (Friedman, Hastie, & Tibshirani, 2008).

**Relative importance networks.** The above models all utilize weighted, undirected approaches, which can describe the magnitude, but not the direction of the correlations within the network. As such, these models cannot determine if relationships modeled between nodes indicate that symptom A influences symptom B, symptom B influences symptom A, or both influence each other bi-directionally (McNally, 2016). Relative importance networks take the benefits of concentration networks (i.e., partial correlations), but employ directed edges to determine both the strength and direction of the correlations, as well as to represent the proportion of variance explained in a node after controlling for all other nodes (Epskamp, Borsboom, et al., 2018).

**Bayesian networks.** This last type of model differs from the rest in that it contains directed, but unweighted edges as part of its network, and also utilizes Bayesian theory to establish the direction of probabilistic correlations (McNally, 2016). This method produces a graphical model called a directed acyclic graph (DAG), which contains edges that demonstrate the direction of the correlation and represent node activation that does not cycle back through other nodes (i.e., node activation travels outward from the original node, and does not reverse back toward the original node; McNally, 2016). For more about Bayesian networks, see Pearl (2010).

### **Network Analysis Complimentary to Latent Variable Theory**

While network theory provides an alternative approach to psychopathology characterization, there exists literature to support the idea that network theory and more traditional latent variable approaches can be complementary (Bringmann & Eronen, 2018; Epskamp, Rhemtulla, & Borsboom, 2017). Researchers have argued that network theory helps characterize dynamic processes of the parts (i.e., symptoms) of the whole (i.e., syndrome), and that a “hybrid model” of the two theories may posit a common cause that precipitates select symptoms, which then in turn perpetuates the activation of other symptoms via a network process (Epskamp et al., 2017). Levinson et al. (2018) provided an example for this hybrid model in an eating disorder conceptualization, wherein a common cause of overvaluation of shape and weight could precipitate symptoms of binge eating, purging, and restriction, and that this restriction may perpetuate further binge eating and purging symptoms as a response to starvation. Levinson and colleagues (2018) have further stated that this approach may hold promise, as interventions could be efficiently tailored to target the symptoms that arise from the latent cause and perpetuate further symptoms

## New Methods of Network Analysis

The emergence of network analysis as a technique in the field of psychopathology within the past decade has resulted in numerous novel methods and tools to assess the network structure of various psychological disorders. More recent research questions have necessitated new methods for network analysis, and while a comprehensive review of these methods is outside of the scope of this project, several methods are briefly described below.

**Intra-individual and longitudinal network analyses.** Network analysis models developed from cross-sectional data have been limited in their ability to model within-subject variance or to account for changes across time. Newer methods have been developed to capture this within-subject variation to identify more accurately the network of symptoms that maintain disorders at an individual level (i.e., what specific symptoms within an individual are maintaining the disorder; Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). Models can also be established that assess for intra-individual differences across time, between-subjects similarity, and differences between the within- and between-subjects networks (Temporal group-level models, or mlVAR; For a review, see Epskamp, van Borkulo, et al., 2018).

**Pre-post treatment.** Currently, different network models can be directly compared based on differences in node connections, edge strength, or global edge strength (i.e., the sum of the strength of all the edges in a network) using the Network Comparison Test (NCT; van Borkulo, Boschloo, Borsboom, Penninx, Waldorp, and Schoevers, 2015). Levinson and colleagues (2018) have indicated that this method is especially relevant for research assessing treatment outcomes, and have stated that “[t]heoretically, if the treatment is effective, networks should be less dense posttreatment. Furthermore, we may mischaracterize treatment

efficacy if we focus on summed scores of outcomes, instead of on the specific maintaining symptoms” (p. 11). Researchers have used the NCT and found differences in the network structures between groups that did and did not respond to treatment, suggesting that this method can help determine differences between treatment responders and non-responders, or even to compare between individuals in treatment and healthy controls (van Borkulo et al., 2015).

### **Summary**

Indeed, the latent variable theory/medical model disease paradigm does not appear to adequately explain the causal structure of psychiatric disorders, and the belief that some common cause precipitates mental health symptoms, as viewed in medical conditions, is conceptually flawed. Issues that arise when applying this paradigm include: (a) symptoms of psychiatric disorders do not appear to be distinct from their respective conditions, (b) symptoms may appear to be causally related to one another, (c) the covariance between symptom clusters is not explained by a common cause, and (d) the high level of comorbidity presents as an issue to the delineation of separate disorders (i.e., the ability to carve nature at its joints properly). Network theory provides an alternative model that proposes that symptoms are not the result of a psychiatric disorder, but rather that such disorders are the result of a functional, causal network of symptoms. This focus on the presence of symptoms and their respective interactions more closely aligns with how clinicians conceptualize and ultimately treat psychopathology. As such, network theory has been used to examine the symptom structure of a wide variety of psychiatric disorders, including depression (Fried et al., 2015), posttraumatic stress disorder (McNally, Robinaugh, Wu, Wang, Deserno, & Borsboom, 2015), schizophrenia (van Kampen, 2014), obsessive-compulsive disorder

(McNally, Mair, Mugno, & Riemann, 2017), social anxiety disorder (Heeren & McNally, 2016), substance use disorders (Rhemtulla, Fried, Aggen, Tverlinckx, Kendler, & Borsboom, 2016), and, as described below, eating disorders (Levinson et al., 2018). The focus of this project, however, will be applied to the conceptualization of Night Eating Syndrome.

## **Night Eating Syndrome**

### **History of Night Eating Syndrome**

Night Eating Syndrome (NES) represents an instance of a disorder that has been characterized and defined in the context of the latent variable theory/medical model paradigm. NES was first described in the literature in 1955 by Dr. Albert Stunkard (Stunkard, Grace, & Wolff, 1955). At that time, Stunkard described NES as a cluster of non-normative eating behaviors that would arise during periods of stress. Such symptoms included morning anorexia (lack of morning hunger or eating behavior), evening hyperphagia (consumption of more than 25% of total daily calories after the evening meal), and sleep-onset insomnia (difficulty getting to sleep more than 50% of the time; Stunkard et al., 1955). There was little research on NES between 1955 and the late 1990s. In 1996, these symptoms were revised by Stunkard and his colleagues, and NES was defined as follows: absence of appetite for breakfast, consumption of 50% or more of total daily calories after 7:00 p.m., and difficulty with sleep onset or maintenance (Stunkard, Berkowitz, Wadden, Tanrikut, Reiss, & Young, 1996).

These criteria were included in a number of studies (Adami, Meneghelli, & Scopinaro, 1999; Birketvedt et al., 1999), but there were some proposed additions as a result of emerging literature. Rand, Macgregor, and Stunkard (1997) suggested that symptoms should include (among those previously discussed) delay of eating after awakening for

several hours, and “evening tension and/or feeling upset” (an early allusion toward negative mood, which would be added later). Spaggiari and colleagues (1994) examined NES as a sleep disorder, and their criteria were more focused on those occurring during sleep. They did, however, propose the addition of several criteria in that domain, and suggested the following be added: a drive to eat upon awakening in the night, the inability to return to sleep without eating, becoming fully awake during nocturnal eating episodes and full recall of such episodes in the morning, and a quick return to sleep after eating (Spaggiari et al., 1994). Nocturnal ingestions (i.e., awakening in the middle of the night to eat) was again referenced in 1999, as was worsening of mood in the evening, contrary to the circadian pattern typically seen in depression (Birketvedt et al., 1999; Greeno, Wing, & Marcus, 1995, Vinai et al., 2008).

While extant research has demonstrated researchers’ desires to conceptualize the diagnostic criteria for NES more comprehensively, NES itself has had a wide range of diagnostic definitions since its first description in 1955 (Striegel-Moore, Franko, May, Ach, Thompson, & Hook, 2006). Most all definitions of NES have included some form of the three original symptoms (i.e., morning anorexia, evening hyperphagia, and sleep disturbance), but amendments to these symptoms (particularly with evening hyperphagia) have led to definitional inconsistencies across studies (For a review, see Striegel-Moore, Franko, May, et al., 2006). Few studies have specifically operationalized morning anorexia in terms of an amount of food and cutoff times (i.e., “skipping breakfast” or “delay of eating for several hours after awakening; Gluck, Geliebter, & Satov, 2001, Geliebter, 2002, Rand et al., 1997). Evening hyperphagia has also differed considerably in its operational definition. For example, Rand and colleagues (1997) amended evening hyperphagia as “excessive evening



eating”, as they proposed dropping the specified percentage of caloric intake after the evening meal due to claims that persons with obesity were inaccurate at reporting caloric intake. In addition, studies have varied widely on the percentage of food consumed in the evening (i.e., greater than 25%-greater than 50%) and the operationalization of “evening” (i.e., after evening meal, after evening meal but before bed, after 6:00 pm-after 8:00 pm; Streigel-Moore, Franko, May, et al., 2006). Lastly, several studies fail to define sleep disturbance beyond “insomnia”, and there exist wide ranges of frequencies for number of sleep disturbances (i.e., three times per week, four times per week, more than half the time, or nightly; Striegel-Moore, Franko, May, et al., 2006). Indeed, such variation in operationalized diagnostic criteria for NES have presented a challenge in conceptualizing NES as a separate eating disorder.

### **Development of the Research Diagnostic Criteria**

In order to provide a much-needed standardized definition for NES, Allison and colleagues proposed a set of research diagnostic criteria, presented in Table 1 (Allison, Lundgren, O'Reardon, et al., 2010). Within these criteria, evening hyperphagia, nocturnal ingestion of food, or both must be present for a diagnosis of NES (Criterion A), along with awareness of these eating episodes (Criterion B) and at least three of five additional symptoms (Criterion C).

Table 1.

*Research diagnostic criteria for Night Eating Syndrome (NES)*

Criterion	Definition
A	<p>The daily pattern of eating demonstrates a significantly increased intake in the evening and/or nighttime, as manifested by one or both of the following:</p> <ol style="list-style-type: none"> <li>1. At least 25% of food intake is consumed after the evening meal</li> <li>2. At least two episodes of nocturnal eating per week</li> </ol>
B	Awareness and recall of evening and nocturnal eating episodes are present.
C	<p>The clinical picture is characterized by at least three of the following features:</p> <ol style="list-style-type: none"> <li>1. Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week</li> <li>2. Presence of a strong urge to eat between dinner and sleep onset and/or during the night</li> <li>3. Sleep onset and/or sleep maintenance insomnia are present four or more nights per week</li> <li>4. Presence of a belief that one must eat in order to initiate or return to sleep</li> <li>5. Mood is frequently depressed and/or mood worsens in the evening</li> </ol>
D	The disorder is associated with significant distress and/or impairment in functioning.
E	The disordered pattern of eating has been maintained for at least 3 months.
F	The disorder is not secondary to substance abuse or dependence, medical disorder, medication, or another psychiatric disorder.

**Evening hyperphagia.** Evening hyperphagia has been considered a core feature of NES and is the manifestation of a delay in the circadian rhythm of food intake such that few calories are consumed during the first half of the day, followed by a marked increase in calorie consumption during the second half of the day, which often disrupts the sleep cycle (O'Reardon, Peshek, & Allison, 2005). This symptom is operationalized as the consumption of at least 25% of daily caloric intake after the evening meal, and is typically assessed through dietary recall/food records or other monitoring, and compared to cumulative caloric intake of control groups (Boston, Moate, Allison, Lundgren, & Stunkard, 2008). The consumption of calories after the evening meal has been shown to be three times higher in persons with obesity (O'Reardon et al., 2004) and without obesity (Lundgren et al., 2008), compared to control groups. Cultural differences in the timing of the evening meal are also accounted for, as some cultures (e.g., Mediterranean) eat meals at later times compared to others (Bellisle, 2009). In these cultures, the presence of other diagnostic criteria are necessary to consider a diagnosis of NES (Allison, Lundgren, Moore, O'Reardon, & Stunkard, 2010). Evening hyperphagia can often co-occur with nocturnal ingestions of food (described below), but it must be present for an NES diagnosis if nocturnal ingestions of food are absent (Allison, Lundgren, O'Reardon, et al., 2010).

**Nocturnal ingestions of food.** Nocturnal ingestions of food are specific instances where an individual awakens from a period of sleep to consume food, usually in small amounts (O'Reardon et al., 2004). Studies have shown that nocturnal awakenings have been associated with food consumption in both persons with obesity (Birkevedt et al., 1999) and without obesity (Lundgren et al., 2008), with little to no instances of food consumption after nocturnal awakenings in control groups. Nocturnal ingestions are assessed through sleep

diaries and food records (these episodes are typically recalled clearly in the morning; see below). This symptom often co-occurs with evening hyperphagia, as noted above, but two or more episodes of nocturnal ingestions of food per week must occur if evening hyperphagia is absent (Allison, Lundgren, O'Reardon, et al., 2010).

**Awareness of eating behavior.** In order to differentiate NES from a sleep-related eating disorder (SRED; a parasomnia; Schenck & Mahowald, 2000), an individual must demonstrate awareness of their evening and nocturnal eating behavior through clear recall of these symptoms the day after they occur (Allison, Lundgren, O'Reardon, et al., 2010; Howell, Schenck, & Crow, 2009). In SRED, individuals are often sleepwalking and are therefore unable to recall or otherwise demonstrate any awareness of nocturnal eating, and these individuals can often consume non-foods or strange foods (Howell et al., 2009; Vinai et al., 2012). Symptoms of NES and SRED have been shown to overlap (Vinai et al., 2012), and more recent definitions of SRED stipulate either complete or partial amnesia of the nocturnal eating episodes (Chiaro, Caletti, & Provini, 2015). Awareness can be assessed via sleep diaries and food records, or other self-report. Lastly, those with NES typically report consumption of foods during nocturnal ingestions that were restricted earlier in the day or were desired by the individual (Allison, Stunkard, & Thier, 2004).

**Morning anorexia.** Morning anorexia, as reported above, is described as a lack of appetite or eating behavior upon awaking in the morning. While this criterion was included as part of the initial description of NES (Stunkard et al., 1955), Allison and colleagues (2010) specified that this symptom must be present for at least four days per week. Assessment of this symptom can include self-reported hunger levels or review of food records demonstrating a lack of caloric intake during the first half of the day. Individuals with NES

have reported reduced levels of morning and daytime hunger compared with control groups (Boston et al., 2008, Gluck et al., 2001, Lundgren et al., 2008). Morning anorexia, however, has been shown to be a common experience among the general population, particularly among persons with obesity, and a study by Allison, Engel, and colleagues (2008) found that morning anorexia was not useful in discriminating individuals with NES from other individuals.

**Strong urge to eat between dinner and sleep onset and/or upon waking at night.**

Allison, Lundgren, and colleagues (2008) have reported that some individuals with NES tend to experience a strong desire to eat in the evening and/or after a nocturnal awakening. This can be related to the belief that one needs to eat to resume sleep after sleep has been initiated (see below), but can sometimes be limited to just the evening hours (Allison, Lundgren, O'Reardon, et al., 2010). Food consumption is not typically objectively large (individuals with NES on average eat about 400 more calories per day and tend to consume significantly more calories between 10:00pm and 6:00am compared to control groups; Lundgren et al., 2008; O'Reardon et al., 2004), and there may be cravings for specific foods. In addition, individuals with this symptom may experience intense anxiety or distress regarding their ability to sleep without disruption, and they can often believe that sleep is dependent on late-night eating, thus perpetuating or sustaining evening hyperphagia (Allison, Lundgren, O'Reardon, et al., 2010). This symptom has commonly been assessed via self-report (e.g., via the Night Eating Questionnaire [NEQ]; Allison, Lundgren, et al., 2008).

**Insomnia.** Insomnia is another criterion that has been present since the initial description of NES (Stunkard et al., 1955). Originally, Stunkard described insomnia in this population to mean sleep-onset insomnia (i.e., difficulty initiating sleep), but he and his

colleagues later expanded on this to include both sleep-onset and sleep-maintenance insomnia (i.e., waking up in the middle of the night with or without difficulty getting back to sleep, or waking up earlier than desired; Stunkard et al., 1996). Allison, Engel, and colleagues (2008) examined NES symptoms, as assessed by the NEQ, using item response theory, and found that sleep-onset insomnia was able to discriminate between individuals with NES from control groups. In addition, other studies have shown that individuals with NES have significantly more awakenings per night than control groups (Birkevedt et al., 1999) and tend to have awakenings earlier in their sleep cycle (O'Reardon et al., 2004). This symptom can be assessed via self-report measures, through the use of a sleep diary, or with polysomnography.

**Belief that one must eat in order to initiate or return to sleep.** This criterion, proposed by Allison and colleagues (2010), is the result of reports by individuals with NES who claim a need to eat (usually small amounts of food) in order to initiate or resume sleep. These individuals have also reported that any attempts to avoid or resist eating ultimately fail, as they will be unable to resume sleep unless they eat, and that sleep comes easily once they consume food. They also have indicated a lack of control over their eating and have expressed heightened levels of anxiety/distress if they perceive any barriers that may limit their ability to eat (Allison et al., 2004). This symptom is assessed through self-report via the NEQ.

**Depressed mood.** While depressed mood was not originally a part of Stunkard's description of NES, myriad studies have observed a low or depressed mood among individuals with NES (Allison, Grilo, Masheb, & Stunkard, 2005; Birkevedt et al., 1999, Calugi, Dalle Grave, & Marchesini, 2009; Geliebter et al., 2016; Gluck et al., 2001; Greeno

et al., 1995; Fischer, Meyer, Hermann, Tuch, & Munsch, 2012; Rogers et al., 2006). In each of these studies, individuals with NES have been shown to have significantly lower mood or higher rates of depression compared to controls, regardless of weight status. Several studies have shown a pattern of worsening mood as the day progressed (Boseck, Engel, Allison, Crosby, Mitchell, & de Zwaan, 2007; Marshall et al., 2004; Striegel-Moore, Dohm, Hook, Schreiber, Crawford, & Daniels, 2005). Birkevedt and colleagues (1999) noted that mood decreased after 4:00pm, which was not seen among controls (i.e., there was no change in mood). This pattern is also not typical of the pattern of mood change seen in individuals with major depression (i.e., mood improves throughout day and is worse in the morning; Vinai et al., 2008). Depressed mood, while a significant predictor of NES (i.e., related to lack of control or helplessness regarding eating behavior), has not been seen in all cases of NES (Allison, Lundgren, O'Reardon, et al., 2010). In one study, 56% of participants with NES had a history of major depressive disorder (de Zwaan et al., 2006). Another study demonstrated that individuals with NES who were not obese were more likely to meet criteria for major depressive disorder in their lifetime compared to a control group (52.6% to 9.1%; Lundgren et al., 2008). In addition, no significant mood differences were found in a study where participants were grouped by body mass index (BMI) status, suggesting that low mood is more related to NES than BMI (Marshall et al., 2004). It is currently unknown, however, whether depressed mood is a cause or consequence of NES and the specific mechanism behind worsening mood during the day. Also, given the relationship between sleep disturbance and depressed mood (Sbarra & Allen, 2009), there is the likelihood that sleep may mediate this NES-mood relationship, as Colles and colleagues (2007) found greater levels of depression only in those with NES who woke up to eat (Colles, Dixon, &

O'Brian, 2007). This symptom is typically assessed through self-report (e.g., NEQ, Beck Depression Inventory, etc.)

### **Specific Differences Between Persons With and Without Overweight/Obesity**

NES was first described through the clinical observation of individuals with BMIs in the obese range, and was thought to be a behavioral expression of this weight status (Stunkard et al., 1955). While the prevalence of NES occurs in roughly 1.5% the general population (Rand et al., 1997; Striegel-Moore et al., 2005), NES appears to be more common in populations with BMIs in the obese range (6-16%; Adami et al., 2002; Cerú-Björk et al., 2001) and studies have shown that individuals in a psychiatric outpatient setting with obesity were five times more likely to meet criteria for NES compared to individuals without obesity (Lundgren, Allison, Crow, et al., 2006) and NES was 2.5 times more common in men with obesity, and 2.8 times more common in women with obesity compared to individuals without obesity (Tholin et al., 2009).

Despite this focus on individuals with obesity, extant research revealed that not all individuals with NES are persons with overweight/obesity (Birketvedt et al., 1999, de Zwaan et al., 2006, Lundgren et al., 2008; Marshall et al., 2004; Striegel-Moore, Franko, Thompson, et al., 2006). Marshall and colleagues (2004) found that individuals with NES and without obesity responded similarly to their counterparts with obesity on the NEQ, and reported only two differences between samples: individuals without obesity were younger and experienced more nocturnal awakenings and food consumption in the middle of the night. Birketvedt and colleagues (1999) also found no differences in neuroendocrine hormones (e.g., melatonin, cortisol), as well as blood glucose and plasma insulin, between individuals with NES who were persons with or without obesity. Lastly, Lundgren and colleagues (2008) examined



individuals with NES and without obesity and found that NES symptoms were independent of weight status. They did note, however, that the discrepancy in caloric intake between individuals with and without NES who were persons without obesity (i.e., about 400 calories) suggested that individuals with NES and without obesity must be engaging in some compensatory behaviors (e.g., food restriction during the day beyond morning anorexia) that explain weight maintenance over time (Lundgren et al., 2008). In addition, Vander Wal (2012) has stated that obesity may be associated with NES, but the directionality of this relationship is not known (e.g., does NES lead to weight gain, or does weight gain from another cause lead to disrupted circadian eating pattern), and key aspects of NES may balance out the likelihood of potential weight gain (i.e., morning anorexia compensating for evening hyperphagia and night eating, as described above).

### **Etiology of Night Eating Syndrome**

As stated previously, the conceptualization and etiology of NES to date has been heavily influenced by latent variable theory/medical model paradigm. NES has been hypothesized as a disorder manifested by a phase delayed circadian rhythm of food intake, and caused by genetics (Root et al., 2010; Lundgren, Allison, & Stunkard, 2006; Rasmussen, Tholin, & Tynelius, 2007), increased stress (Allison et al., 2004; Takeda et al., 2004; Vander Wal, 2012), and biobehavioral mechanisms (Stunkard, Allison, Lundgren, & O'Reardon, 2009). Indeed, several studies have examined the role of neuroendocrine hormone (e.g., plasma melatonin, leptin, ghrelin, blood glucose, insulin, plasma cortisol, etc.) functioning in individuals with NES (Allison et al., 2005; Birkedvedt et al., 1999; Birkedvedt, Geliebter, Florholmen, & Gluck, 2014; Goel et al., 2009), with some studies recognizing the unknown directionality of circadian phase shift among those with NES (i.e., does the shift cause the

altered eating, or does altered eating cause the shift; Vander Wal, 2012). This focus on an etiology of food intake driven by biological processes associated with circadian rhythm suggests that NES has been conceptualized as having an underlying common cause (i.e., latent variable), and research to date has been focused on identifying this exact causal mechanism of NES.

### **Night Eating Syndrome Status in the DSM-5**

As of the most recent version of the DSM (DSM-5; American Psychiatric Association, 2013), NES has not been recognized as a separate diagnosis, and is instead classified under the “Other Specified Feeding and Eating Disorder” (OSFED) category. This represents a revised version of the “Not Otherwise Specified” (NOS) classification used in previous DSM editions, which was used as a “catch-all” diagnosis to denote clinically significant symptomatology that did not fit with other defined eating disorder psychopathology (e.g., anorexia nervosa or bulimia nervosa). Criteria for NES in the OSFED section does not include any mention of morning anorexia, and conceptualizes evening hyperphagia and nocturnal eating as “[r]ecurrent episodes of night eating, as manifested by eating after awakening from sleep, or by excessive food consumption after the evening meal. There is awareness and recall of the eating” (American Psychiatric Association, 2013, p. 354).

The attitude of excluding NES as a distinct diagnosis reflects an historical paucity of research since the first description of NES. Striegel-Moore and colleagues (2006) have reported that, at the time of publication, NES did not meet any of the five criteria outlined by Blashfield, Sprock, and Fuller (1990) needed to recognize it as a discrete disorder. While interest in NES has steadily increased, a significant roadblock outlined in Striegel-Moore’s

review has been the presence of several varied definitions of NES, which have contributed to challenges in both research and clinical examinations of the syndrome (Vander Wal, 2012).

While the addition of the research diagnostic criteria by Allison and colleagues (2010) has helped to standardize the definition of NES, much research has been focused on identifying the underlying cause of NES, which is still unknown. In addition, the presence of overlapping features with other eating disorders, such as binge eating disorder (Greeno et al., 1995, Napolitano, Head, Babyak, & Blumenthal, 2001, Stunkard et al., 1996), has contributed concern as to whether NES is a discrete diagnosis, or merely a unique presentation of binge eating disorder (Adami et al., 1999; Allison et al., 2005; Stunkard, Allison, Geliebter, Lundgren, Gluck, & O'Reardon, 2009). I propose that efforts to conceptualize NES should shift away from reliance on the latent variable theory/medical model paradigm, and would be better suited for examination through the network approach to psychopathology. It may very well be possible that there exists a functional, causal interaction among NES symptoms proposed by Allison and colleagues (2010), which appears quite simply when one considers the symptoms from the perspective of a network theory model (e.g., morning anorexia leads to evening hyperphagia, belief that one must eat in order to sleep leads to insomnia, etc.). Indeed, network analysis has appeared to have already gained momentum in the re-conceptualization of other eating disorders, as described below.

### **Network Theory of Psychopathology in Eating Disorders**

#### **Outline of Emerging Research**

Levinson and colleagues (2018) have stated that network analysis “presents an alternative approach to conceptualizing eating disorders and holds significant promise for the development of novel treatment for [eating disorders]” (p. 1). To date, there exists a limited

but growing literature applying network theory to eating disorders. As of August 2018, 11 articles have been published in this field, two of which are review papers (Levinson et al., 2018; Smith, Crosby, Wonderlich, Forbush, Mason, & Moessner, 2018). Specific populations/disorders addressed include anorexia nervosa/bulimia nervosa/binge eating disorder (Solmi, Collantoni, Meneguzzo, Degortes, Tenconi, & Favaro, 2018), anorexia nervosa/bulimia nervosa (Forrest, Jones, Ortiz, & Smith, 2018), bulimia nervosa only (Levinson et al., 2017), and mixed eating pathology in general (DuBois et al., 2017; Forbush, Siew, & Vitevitch, 2016; Goldschmidt et al., 2018; Olatunji, Levinson, & Calebs, 2018; Smith, Mason, et al., 2018; Vanzhula, Calebs, Fewell, & Levinson, 2018). Articles are presented below in order of network approach and time of publication.

**Association networks.** Forbush and colleagues (2016) modeled an association network to examine the core symptoms of eating pathology among adults with various eating disorders. They employed the Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013) and found that overvaluation of shape and weight (via body dissatisfaction and body checking behaviors) were the most central symptoms in their network. The authors have indicated that their findings match onto the transdiagnostic cognitive-behavioral theory of eating disorders, which states that overvaluation of shape, weight, and control represent the core psychopathology common across all eating disorders, and that these core symptoms lead to, and maintain, further symptomatic expressions (Fairburn, Cooper, & Shafran, 2003).

**GLASSO networks.** The first study to employ a regularized partial correlation (GLASSO) network was Levinson et al. (2017). Levinson and colleagues used the Eating Disorder Examination (EDE; Fairburn, Cooper, & O'Connor, 2008) to create a network of eating disorder symptoms among a sample of individuals with bulimia nervosa. They found

that fear of weight gain was the most central symptom in this network, while controlling for all others, and that binge eating and purging (typically seen as the traditional central symptoms of bulimia nervosa) were at the fringe of the network. The authors noted that their results were particularly interesting, as fear of weight gain does not present as a symptom for bulimia nervosa in the DSM-5, but yet this symptom was more central than binge eating or purging (Levinson et al., 2017).

DuBois and colleagues (2017) sought to expand on the findings from Forbush et al. (2016), specifically to investigate if overvaluation of shape and weight would emerge as central symptoms (as claimed by the transdiagnostic model), in a sample of treatment-seeking individuals with a mix of eating disorders. They used the EPSI, but utilized the eight subscales of this measure as nodes in their network, and they also included a variable for overvaluation of shape and weight from the sum of two items from the Eating Disorder Examination – Questionnaire (EDE-Q; Fairburn & Beglin, 1994). They estimated several networks: two networks of the eight subscales based on those with high and low levels of overvaluation, and three networks of the eight subscales and the composite overvaluation scale; one each for individuals with either anorexia nervosa, bulimia nervosa, or binge eating disorder. Results from the NCT revealed that the nodes within the network of those with high overvaluation were more strongly connected compared to the network of the low overvaluation group. They also found that overvaluation was the most central symptom across all three diagnostic networks (DuBois et al., 2017).

The above methodology was mimicked in a study by Goldschmidt and colleagues (2018), although their sample was made up of youths with eating disorders. The authors created separate networks for youths with anorexia nervosa, bulimia nervosa, and OSFED,

and found no differences between the connectivity within each network. They used 28 items from the EDE to estimate their networks and similarly found that overvaluation of shape and weight, as well as dietary restraint, were central symptoms in all three diagnostic networks (Goldschmidt et al., 2018).

Olatunji et al. (2018) was the first study to use a rather large sample ( $n = 5193$ ) of individuals in inpatient treatment for eating disorders in a network analysis, and was the first to assess whether central symptoms within a network affected treatment outcomes. They used the 11 subscales from the Eating Disorder Inventory-2 (EDI-2; Garner, 1991) as nodes and estimated a GLASSO network for individuals both before and after treatment. They found that interoceptive awareness and ineffectiveness (i.e., feelings of inadequacy or worthlessness) were central to both eating disorder networks, and that ineffectiveness pre-treatment predicted post-treatment BMI and depression, over and above interoceptive awareness, BMI, and depression at admission. Interestingly, they did not find that overvaluation of shape or weight, body dissatisfaction, or drive for thinness/fear of weight gain to be central symptoms, as was found in emerging literature to date (Olatunji et al., 2018).

In another study that examined networks at both admission and discharge, Smith, Mason, et al. (2018) assessed individuals with mixed eating disorder presentations from residential and partial hospital eating disorder treatment programs using the EDE-Q, along with measures of depression (Quick Inventory of Depressive Symptomatology-Self-Report [QIDS-SR]; Rush et al., 2003) and anxiety (State-Trait Anxiety Inventory-Trait Subscale [STAI-T]; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Along with the characterization of the network structure, they also aimed to assess global network strength at

both admission and discharge to evaluate if changes would be associated with a decrease in symptom severity, and they aimed to examine treatment outcomes by seeing if individuals with greater network strength at admission experienced poorer outcomes. Results demonstrated that shape and weight concerns, along with guilt about eating, were central eating disorder symptoms, and bridge symptoms between anxiety, depression, and eating disorders included restlessness, feeling overwhelmed, lack of energy, and self-esteem. The authors also found that global network strength did not significantly change over time, but those who were less improved had greater network strength at admission compared to those who were more improved. They suggested that these findings could reflect either a need for a longer period of time or greater reduction in symptoms in order to see changes in network strength, or that the treatments used in these programs, while having ultimately reduced symptom severity, do not target key symptom relationships (i.e., network edges) that maintain eating disorder psychopathology (Smith, Mason, et al., 2018).

Forrest and colleagues (2018) recognized a lack of research on network analysis and core psychopathology of individuals with anorexia nervosa alone, and estimated networks for individuals seeking residential treatment diagnosed with either anorexia nervosa or bulimia nervosa, as well as a network of both. The authors used 27 items from the EDE-Q as nodes in their GLASSO networks and found that among all three networks, central symptoms included shape overvaluation, desiring weight loss, shape and weight preoccupation, and restraint. They also found central symptoms that were unique to the diagnostic networks, such that in the anorexia nervosa and combined network, fearing weight gain had high importance; and in the bulimia nervosa network, weight overvaluation had high importance.

In the first non-American sample, Solmi et al. (2018) examined the network structure of individuals with anorexia nervosa, bulimia nervosa, and binge eating disorder seeking outpatient treatment in Italy. They assessed eating disorder symptoms with subscales from the Eating Disorder Inventory (EDI; Garner & Olmstead, 1984), along with general psychiatric symptoms (Symptom Checklist-90, revised [SCL-90-R]; Derogatis, 1994), and personality traits (Tridimensional Personality Questionnaire [TPQ]; Cloninger, Przybeck, & Svrakic, 1991). Four networks were estimated (one for eating disorders combined, and one for each diagnosis) and results indicated that ineffectiveness, interoceptive awareness, interpersonal distrust, and drive for thinness were central symptoms in all networks (along with depression, anxiety, and interpersonal sensitivity symptoms from the SCL-90-R). They also found that BMI and EDI bulimia were central nodes when the eating disorders were combined, but not when examining each network individually. The authors attributed this finding to differences in intensity of body dissatisfaction compared to weight status within the diagnostic groups (i.e., individuals with anorexia nervosa who are underweight may experience less body dissatisfaction due to weight status compared to individuals with bulimia nervosa or binge eating disorder who are or are not overweight, who may experience larger differences between weight status and desired weight; Solmi et al., 2018).

In the most recent study to date, Vanzhula et al. (2018) aimed to examine the specific mechanisms that maintain comorbidity between eating disorders and post-traumatic stress disorder (PTSD). They estimated two GLASSO networks: one with a sample of individuals with an eating disorder diagnosis, and a nonclinical sample of college-aged adults. Items from the EDE-Q and the PTSD Checklist – Civilian Version (PCL-C; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996) were used as nodes in both networks. Although the



primary focus of this study was to examine comorbidity pathways via bridge symptoms, results from the eating disorder-PTSD network demonstrated that body dissatisfaction (desire for a flat stomach), fear of weight gain, and binge eating were central symptoms and linked as bridge symptoms with PTSD. Interestingly, their results of body dissatisfaction/shape and weight overvaluation were similar to emerging literature in this field, but theirs was the first to find binge eating as a central symptom, despite research suggesting that binge eating was on the periphery of these networks (Levinson et al., 2017). The authors suggested that the presence of binge eating as a central symptom is explained through extant literature demonstrating a link between the experience of traumatic events and disorders characterized by bulimic symptoms, and that this is only seen in an eating disorder-PTSD network, rather than in an eating disorder network alone (Vanzhula et al., 2018).

### **Lack of Literature on Network Analysis and Night Eating Syndrome**

Despite this growing literature on network analysis, no studies to date have specifically examined this theory with relation to NES, and only one sample with mixed eating disorders specifically noted that they had two individuals with NES among their participants. All other mixed eating disorder samples only reported OSFED or EDNOS, which can accompany myriad eating disorder classifications outside of anorexia nervosa, bulimia nervosa, or binge eating disorder (e.g., subthreshold anorexia nervosa or bulimia nervosa, atypical anorexia nervosa, purging disorder, etc.). Indeed, among the 9,479 participants sampled across the entirety of this literature to date, 1,734 (18.3%) were classified as OSFED or EDNOS.

## **Sparsity of the Inclusion of a Control Group**

As noted above, only one study (Vanzhula et al., 2018) utilized a control group to compare the network structure and overall network strength between a clinical and non-clinical sample using the Network Comparison Test (NCT; van Borkulo et al., 2015). This technique has been used recently to compare individuals with social anxiety disorder to a non-clinical sample (Heeran & McNally, 2018). Results from both studies indicated that the network structures between both clinical and non-clinical samples are the same, but that there was greater network connectivity and density among the clinical samples (i.e., the symptoms were more strongly connected). The authors of these studies have stated that the lack of differences in network structure suggest that symptoms interact with each other in similar ways, regardless of symptom severity, and that stronger network connectivity leads to stronger symptom severity (Heeran & McNally, 2018; Vanzhula et al., 2018).

This suggestion mirrors the resilient and vulnerable networks described by Borsboom (2017), such that psychopathology is the result of the self-sustaining activity seen in a densely connected (i.e., vulnerable) network, and that symptom activation in a weakly connected (i.e., resilient) network may not lead to the development of psychopathology. Indeed, it is not how the symptom network is structured, but rather how strongly connected symptoms are (e.g., sleep disturbance would be unlikely to trigger other NES symptoms in a healthy sample, but it may be more likely to do so in an NES sample because of how strongly the symptoms are connected in this clinical sample). Vanzhula et al. (2018) stated that “this suggests that treatments should focus on weakening connections as a whole within psychopathology networks and that this reduction in symptom connections may be representative of healthy functioning” (p. 11).

### **Aims of the Current Study**

The purpose of this study was to examine and characterize the core symptoms in a psychopathology network for NES among persons with and without overweight/obesity, and to compare the network density between an NES sample and a non-NES control group. The current conceptualization of NES, and interventions based on that conceptualization, are grounded in a latent variable, medical model nosology and theory of etiology. It is imperative that we accurately characterize the complex network of psychopathology to further our understanding of the causal chain of symptoms therein, which can lead to improvement in research and better refined treatment options. In addition, while NES is more prevalent in persons with overweight/obesity compared to the general population, not all persons with NES are persons with overweight/obesity. While some studies have found no differences in symptomatology between these two groups, others have found (a) higher incidences of nocturnal awakenings and food consumption, (b) that populations with NES who were persons without overweight/obesity were younger, and (c) that NES symptoms preceded obesity. Indeed, evening hyperphagia and conscious nocturnal eating may still be the most central symptoms in NES psychopathology among these two groups, as proposed by the research diagnostic criteria. Lastly, while the presence of at least one of these symptoms is necessary for an NES diagnosis according to these criteria, only three of the five symptoms in criterion C are necessary. This suggests potential differences in network density, such that the expression of certain chains of core and distal symptoms lead to differences in presentation and clinically impairing distress experienced by an individual. Indeed, the following aims were addressed:

- (1) To characterize the psychopathology network of core symptoms of NES (food records/NESHI/NES), including symptoms of mood (BDI-II), stress (PSS-10), sleep (PSQI/ESS) and circadian rhythm (MEQ), to identify which symptoms are most central to this disorder and are most likely to impact other NES symptoms.
  - a. Hypothesis 1: Nocturnal ingestion of food and evening hyperphagia symptoms (Research diagnostic criterion A; Food records) will present as the most central nodes within the psychopathology network of NES compared to other NES symptoms (Criterion B and C) and associated symptoms (i.e., mood, stress, sleep, and circadian rhythm).
- (2) To identify key differences in the psychopathology networks of NES symptoms between persons with overweight/obesity compared to persons without overweight/obesity.
  - a. Hypothesis 1: Nocturnal ingestion of food and evening hyperphagia symptoms (Food records) will present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.
  - b. Hypothesis 2: Overall network structure will be dissimilar between populations of persons with and without overweight/obesity.
- (3) To determine differences in the psychopathology network structure and density of NES symptoms between individuals with NES and a non-NES control group.
  - a. Hypothesis 1: Overall network structure will be similar between individuals with NES and non-NES controls.
  - b. Hypothesis 2: Network density will be greater in the NES group compared to the non-NES controls.

- (4) To assess the validity of the transdiagnostic cognitive-behavioral model of eating disorders within an NES network by examining if overvaluation of shape and weight (EDE) are most central when added to the model. As emerging network analysis literature of eating disorders demonstrated these symptoms as central regardless of diagnosis, if NES were to fit into the transdiagnostic model of EDs, then this sample should exhibit a similar pattern. Otherwise, NES may be a conceptually different kind of feeding and eating disorder (i.e., one without shape/weight concerns).
- a. Hypothesis 1: Overvaluation of shape and weight will present as the most central nodes within individuals' psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.
  - b. Hypothesis 2: Overall network structure will be dissimilar between populations of persons with and without overweight/obesity.

## CHAPTER 3

### METHODOLOGY

#### **Procedure and Participants**

This project was a secondary data analysis from a previous six-year outpatient study (NIH RO1 DK056735-05) that examined the characteristics of NES. 148 individuals with NES and 69 controls (i.e., individuals without NES) were recruited from the community at the University of Pennsylvania from April 2001 to June 2006. The previous study was reviewed and approved by the University of Pennsylvania Institutional Review Board (IRB).

The age of participants ranged from 20 to 85 ( $M_{\text{age}} = 42.13$ ,  $SD = 12.13$ ), and 150 participants (69.1%) were female. Participant ethnicity was as follows: 62.7% Caucasian, 32.5% African American, 2.3% Hispanic/Latinx, 1.4% Asian/Pacific Islander, 0.9% other, and 0.5% Native American. 43.3% of the sample identified as single, 37.3% were married, 16.1% were separated or divorced, and 3.2% were widowed. The sample appeared well-educated; almost half (47.5%) of the sample reported completing college or advanced degrees, 27.6% reported some college education, 19.8% completed high school, 3.2% reported less than high school education, and 1.8% were missing educational demographic data. Lastly, objective height and weight data were collected to assess body mass index (BMI) using the formula provided by the Center for Disease Control ( $\text{kg/m}^2$ ) to categorize participants as those without overweight/obesity ( $\text{BMI} < 25$ ; 18.9% of sample) or those with overweight/obesity ( $\text{BMI} > 25$ ; 81.1%). Average BMI for this sample was 32.36 ( $SD = 8.23$ ); 41 (18.9%) were classified as persons without overweight/obesity; 176 (81.1%) were classified as persons with overweight/obesity. Among NES diagnosis, 128 (86.5%)

individuals in the NES group were classified as persons with overweight/obesity, and 47 (68.1%) individuals in the control group were classified as persons with overweight/obesity.

## **Recruitment**

Individuals were recruited from the community via television and newspaper advertisements targeting individuals experiencing symptoms related to NES. Sample inclusion criteria were individuals (men and women) 18 years of age or older. Exclusion criteria included individuals who were pregnant or planning to become pregnant, engaging in night shift work during the time of the study, participation in a weight reduction program or active attempts at weight loss, current diagnoses of diabetes mellitus, thyroid disease or other endocrine or metabolic disorders, sleep apnea, anorexia nervosa or bulimia nervosa, lifetime history of bipolar disorder or psychotic disorder, abuse of substances or dependency within the previous three months, use of any psychotropic medication, steroids, diuretics or hypnotics within the previous six months, and current major depression greater than moderate severity as measured by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID I; First, 1997).

Individuals kept food and sleep diaries (see Data Collection section) for a total of ten days and were confirmed to have NES if they reported (a) evening hyperphagia (consumption of 25% or more of total daily calories after the evening meal until the final awakening the following morning) and/or nocturnal awakening with ingestion of food three or more times per week. Within the sample with NES, 99 individuals met criteria for both evening hyperphagia and nocturnal awakening and ingestion of food, 25 had evening hyperphagia only, and 24 had nocturnal awakening and ingestion of food only.

A control sample (i.e., individuals with no NES symptoms) was also recruited concurrently with similar inclusion/exclusion criteria, and included a total of 69 participants. The first three years of the study focused on recruiting persons with overweight/obesity, and the last three years focused on recruiting persons without overweight/obesity. Indeed, participants were roughly matched on BMI categorization and age.

### **Data Collection**

Following recruitment and a brief telephone screen, participants were interviewed using the Night Eating Syndrome History and Inventory (NESHI; Lundgren, Allison, Vinai, & Gluck, 2012) to confirm NES diagnosis, and those meeting criteria were instructed to complete ten-day, 24-hour food and sleep diaries. The first two days were deemed practice days, and the final day contained incomplete/missing data; therefore, only data from the middle seven days were considered for analysis. Food records included all food and beverages consumed by the participant from 6:00am to 5:59am the following morning, as well as a rating of mood and hunger before each meal, and an additional hunger rating at 10:00am to assess morning anorexia. Records were examined for nocturnal awakening and ingestions of food (compared with sleep diaries to determine when the individual went to sleep) and evening hyperphagia (percent of caloric intake after the evening meal compared to total daily intake). Nocturnal awakenings were also assessed through number of reported awakenings recorded in sleep diary. In addition to the NESHI and food/sleep diaries, participants completed a battery of measures (see Measures section) administered by trained project coordinators/research assistants. Participants were compensated \$100 as part of the study. Additional data collected as part of the larger study (e.g., cortisol, macronutrient data, etc.) were not included in the following analyses.



## **Measures**

### **Night Eating Syndrome History and Inventory**

The Night Eating Syndrome History and Inventory (NESHI; Lundgren et al., 2012) is a semi-structured interview used to help establish a diagnosis of NES based on Allison and colleagues' research diagnostic criteria (Allison, Lundgren, O'Reardon, et al., 2010). Items in this interview are specific to daily food intake, previous NES symptoms, sleep, mood, body weight, any previous dieting behavior, medications and supplements, medical conditions, and any previously used means to alleviate symptoms of NES (Allison, Lundgren, et al., 2008).

### **Night Eating Questionnaire**

The Night Eating Questionnaire (NEQ), which is imbedded in the NESHI, is a 14-item questionnaire used as a screening tool and symptom severity assessment for NES (Allison, Lundgren, et al., 2008). Responses are based on a five-point Likert-type scale (0-4), and assess NES symptoms during the past week, which include morning anorexia, timing of initial food consumption, food cravings and perceptions of control over eating over the course of the evening and during the night, percentage of food consumption post evening meal, insomnia, number of awakenings during the night, any food consumption during these awakenings along with awareness of food consumption, and mood (Allison, Lundgren, et al., 2008). Internal consistency, measured via Cronbach's alpha ( $\alpha$ ), has been reported as .70 (Allison, Lundgren, et al., 2008), and was .73 in the present sample

### **Beck Depression Inventory**

The Beck Depression Inventory-II (BDI-II) is a 21-item self-report questionnaire used to assess severity of depressive attitudes and symptoms (Beck, Steer, & Brown, 1996). Responses are based on a four-point Likert-type scale, with higher scores representing higher

levels of severity. Scores are summed to create a single composite score of depression and range from 0-63. Cutoff values are assigned to the overall total score to categorize levels of depression as minimal (0-9), mild (10-18), moderate (19-29), and severe (30-63). Internal consistency ranged from .73 to .92 (Beck et al, 1996), and was .94 in the present sample.

### **Morningness-Eveningness Questionnaire**

The Morningness-Eveningness Questionnaire (MEQ) is a 19-item self-report questionnaire used to assess circadian rhythm via the timing of an individual's subjective levels of peak alertness (Horne & Östberg, 1976). Its assessment of circadian rhythm has been validated against variation in peak body temperature (Horne & Östberg, 1976). A modified 13-item questionnaire was used with this sample. Internal consistency has been reported as .82, and was .91 in current sample.

### **Pittsburgh Sleep Quality Index**

The Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-report questionnaire used to assess quality of sleep over a previous one-month period (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI provides seven component scores representing different aspects of sleep quality, which combine to create a single composite score ranging from 0-21, with higher scores representing poorer sleep quality. This questionnaire demonstrates good construct validity and internal consistency between .70 and .83 (Mollayeva, Thurairajah, Burton, Mollayeva, Shaprio, & Colantonio, 2016). Cronbach's alpha for the current sample was .82.

### **Epworth Sleepiness Scale**

The Epworth Sleepiness Scale (ESS) is an eight-item self-report questionnaire used to measure levels of daytime sleepiness (Johns, 1991). Individuals are asked to report their

likelihood of falling asleep during eight different situations on a scale from 0-3, with higher scores indicating a higher probability of falling asleep. Scores are summed to create a total score ranging from 0-24, with higher scores representing a higher average sleep propensity in daily life, and scores between 11-24 representing “excessive daytime sleepiness” (Johns & Hocking, 1997). Internal consistency has been reported between .73 and .90 (Hagell & Broman, 2007), and was .81 in the current sample.

### **Eating Disorder Examination**

The Eating Disorder Examination (EDE) is a semi-structured interview used to assess the range and severity of various features of eating disorders, and is viewed as the “gold standard” assessment for eating disorder psychopathology (Fairburn, Cooper, & O’Connor, 2008). Item responses on the EDE can be used to derive subscale scores representing the degree of psychopathology among four aspects of eating disorders (e.g., restraint, eating concern, shape concern, and weight concern). These four subscales can also be averaged to obtain an overall global score. Twenty-eight items in total are used to create these subscales. This measure was also normed on a community-based sample, thus providing normative data. This questionnaire has demonstrated adequate criterion and construct validity, and ranges for internal consistencies among the four subscales, in the order listed above, have been reported as .58-.78, .44-.78, .68-.85, and .51-.76 (Berg, Peterson, Frazier, and Crow, 2012). Internal consistencies in the present sample were .69, .75, .88, and .77 for restraint, eating concern, shape concern, and weight concern, respectively.

### **Perceived Stress Scale**

The Perceived Stress Scale-10 (PSS-10) is a 10-item self-report questionnaire used to assess the degree to which an individual considers life situations to be markedly stressful

relative to their coping ability (Cohen & Williamson, 1988). Individuals are asked to rate themselves over the past month on a five-point Likert-type scale (0-4) on different aspects of stress, with higher scores representing higher levels of perceived stress. Internal consistency has been reported between .78 and .90 (Taylor, 2015), and was .84 in the present sample.

## **Statistical Analyses**

### **Power**

Power analysis, in the traditional sense as described by Cohen (1988), is not possible in network analysis because the effect size is unknown (Epskamp, Borsboom, et al, 2018). Instead, best practices to date specify that network structures should be assessed for edge weight accuracy, centrality stability, and network size (Levinson et al., 2018). As sample sizes increase, edge weights within networks are estimated more accurately, which increases the likelihood that true differences between centrality estimates are detected, and the order of the centrality estimates are deemed more stable. Indeed, the question remains as to the sample size required to estimate accuracy and stability. The R package *bootnet* was used to assess for edge weight accuracy and centrality stability. Assessment of stability (akin to reliability) has been considered an essential step in network analysis (Epskamp, Borsboom, et al., 2018).

Centrality stability was assessed using the case-dropping subset bootstrap framework (Epskamp, Borsboom, et al., 2018). This method determines if the order of centrality estimates remains the same (or similar) if an increasing number of cases are dropped and the network re-estimated. The centrality indices in these new networks are then correlated with those from in the original network. Epskamp and colleagues have stated that “if this correlation completely changes after dropping, say, 10% of the cases, then interpretations of

centralities are prone to error” (p. 200). This is quantified by the correlation stability coefficient (CS-coefficient), which is defined as “the maximum proportion of cases that can be dropped, such that with 95% probability the correlation between original centrality indices and centrality of networks based on subsets is 0.7 or higher” (Epskamp, Borsboom, et al., 2018, p. 200). Epskamp and colleagues have recommended a CS-coefficient no lower than 0.25, with preferred values above 0.5, but have also indicated that these are arbitrary cutoffs determined by a simulation study and are not definitive guidelines.

Network size is determined by the number of parameters estimated based on the number of nodes present. For example, in a network with 10 nodes, 55 parameters are estimated (10 threshold and 45 pairwise association). If the number of nodes increases to 20, then the number of estimated parameters grows to 210. Akin to other latent variable analyses such as structural equation modeling, a relatively large number of observations are needed to estimate each parameter reliably. One method used to solve for the need for large sample sizes is to utilize the LASSO technique (Tibshirani, 1996). This method limits the overall number of parameter values by fixing a regularization penalty on small values (both positive and negative edge weights), shrinking them to exactly zero and dropping these edge estimates from the model. This leads to the estimation of a sparse model with relatively few edges used to explain the covariance in the model, with such models being more interpretable (Epskamp, Borsboom, et al., 2018). In addition, the *goldbricker* function in the R package *networktools* was used to assess for multicollinearity among nodes in the network to ensure that items were not too similar in content (Jones, 2018).

As noted above, nine articles exist examining eating pathology using network analysis. Four of these studies include sample sizes between 143-196 participants. The

current sample includes 148 individuals with NES and 69 controls without NES. Therefore, this sample is representative of the size employed in other studies, and was deemed likely to be suitable to estimate replicable and valid networks.

### **Specific Aim One**

- (1) To characterize the psychopathology network of core symptoms of NES, including symptoms of mood, stress, sleep, and circadian rhythm, to identify which symptoms are most central to this disorder and are most likely to impact other NES symptoms.
  - a. Hypothesis 1: Nocturnal ingestion of food and evening hyperphagia symptoms will present as the most central nodes within the psychopathology network of NES compared to other NES symptoms.
    - i. A GGM using GLASSO will be used to estimate this network using the *qgraph* R package. Specific items will be pulled from food records, NESHI/NEQ, and EDE that map onto NES symptoms from the research diagnostic criteria (see Table 2).
    - ii. In addition, total scores from the BDI-II, PSQI/ESS, MEQ, and PSS-10 will be used as nodes for mood, sleep, circadian rhythm, and stress, respectively (see Table 3).
    - iii. Symptoms with high centrality indices (i.e., strength and/or expected influence) will be assessed via bootstrapped difference tests to test this hypothesis.
    - iv. The *goldbricker* function in the R package *networktools* will be used to assess for multicollinearity among nodes in the network to ensure

that items are not too similar in content. Nodes demonstrating multicollinearity will be reviewed to determine item removal.

### **Specific Aim Two**

(2) To identify key differences in the psychopathology networks of NES symptoms between persons with overweight/obesity compared to persons without overweight/obesity.

- a. Hypothesis 1: Nocturnal ingestion of food and evening hyperphagia symptoms will present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.
  - i. A GGM using GLASSO will be used to estimate both networks, using items from network estimated in the first aim using the *qgraph* R package.
  - ii. Symptoms with high centrality indices (i.e., strength and/or expected influence) will be assessed via bootstrapped difference tests to test this hypothesis.
  - iii. The *goldbricker* function in the R package *networktools* will be used to assess for multicollinearity among nodes in the network to ensure that items are not too similar in content. Nodes demonstrating multicollinearity will be reviewed to determine item removal.
- b. Hypothesis 2: Overall network structure will be dissimilar between populations of persons with and without overweight/obesity.

- i. A Network Comparison Test (NCT) will be used to assess for differences in network structure, edge strength, and global strength to test this hypothesis.

### **Specific Aim Three**

- (3) To determine differences in the psychopathology network structure and density of NES symptoms between individuals with NES and a non-NES control group.
  - c. Hypothesis 1: Overall network structure will be similar between individuals with NES and non-NES controls.
    - i. A GGM using GLASSO will be used to estimate both networks, using items from network estimated in the first aim using the *qgraph* R package.
    - ii. A Network Comparison Test (NCT) will be used to assess for differences in network structure, edge strength, and global strength to test this hypothesis.
  - d. Hypothesis 2: Network density will be greater in the NES group compared to the non-NES controls.
    - i. A Network Comparison Test (NCT) will be used to assess for differences in network structure, edge strength, and global strength to test this hypothesis.

### **Specific Aim Four**

- (4) To assess the validity of the transdiagnostic cognitive-behavioral model of eating disorders within an NES network by examining if overvaluation of shape and weight are most central when added to the model. As emerging network analysis literature of eating disorders demonstrated these symptoms as central regardless of diagnosis, if



NES were to fit into the transdiagnostic model of EDs, then this sample should exhibit a similar pattern. Otherwise, NES may be a conceptually different kind of feed and eating disorder (i.e., one without shape/weight concerns).

- a. Hypothesis 1: Overvaluation of shape and weight will present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.
  - i. A GGM using GLASSO will be used to estimate the network, using items from the network estimated in the first aim using the *qgraph* R package, along with relevant items from the EDE (see Table 4).
  - ii. Symptoms with high centrality indices (i.e., strength and/or expected influence) will be assessed via bootstrapped difference tests to test this hypothesis.
  - iii. The *goldbricker* function in the R package *networktools* will be used to assess for multicollinearity among nodes in the network to ensure that items are not too similar in content. Nodes demonstrating multicollinearity will be reviewed to determine item removal.
- b. Hypothesis 2: Overall network structure will be dissimilar between populations of persons with and without overweight/obesity.
  - i. A Network Comparison Test (NCT) will be used to assess for differences in network structure, edge strength, and global strength to test this hypothesis.

## CHAPTER 4

### RESULTS

#### **Data Analysis**

##### **Data Screening and Missing Data**

Data were screened for accuracy, missing data, outliers, and assumptions for multivariate analyses. Data were examined using R version 3.5.2. Missing data were inspected using the *mice* package in R, with five cases presenting with greater than 50% missing data (4 participants with NES, 1 control). These cases were deleted. The remaining cases had less than 10% missing data, with one participant having 17.78% missing data. Among variables, the item capturing criterion C.4, “Must Eat to Sleep” (NESHI item “needeat”) had the highest percentage of missing data at 15.57%. To determine the pattern of missing data, I used Little’s MCAR (Missing Completely At Random) test in SPSS version 25 (IBM Corporation, 2017), which was significant ( $\chi^2(872) = 988.24, p = 0.004$ ). This suggests that the data are not missing completely at random, and that deletion of these cases may introduce bias into the sample (Kang, 2013). Indeed, missing data were handled using a multiple imputation method consistent with best practices for such data in network analyses (van Buuren & Groothuis-Oudshoorn, 2011). Predictive mean matching was used as the imputation method, resulting in five imputed datasets in total with 100 maximum iterations. As pooled data was unavailable, accuracy of imputed data was explored through re-running all subsequent analyses with three randomly selected imputed datasets, consistent with practices found in previous literature (Azur, Stuart, Frangakis, & Leaf, 2011; Forrest et al., 2018). Results were similar across these selected imputed datasets.

Data were examined for outliers using Mahalanobis multivariate distances with  $p < 0.001$ . Thirteen outliers (all participants with NES; NES and control samples were screened separately) were detected. However, as all outliers were from participants with NES, it is feasible that these outliers represented extreme, but possible, clinical data (e.g., participants who scored in the extremes on measures of NES), and thus, outliers were not removed. This follows recommendations by Bakker and Wicherts (2014) to limit false positives. I also ran all analyses with and without outliers (consistent with recommendations by Bakker and Wicherts, 2014) and found no major differences in any of the models reported below (no differences in the central nodes; only in the magnitude of reported centrality values). Data were also screened for assumptions of multivariate normality, linearity, and homogeneity of variance, with no issues detected. Final participant count after data screening were 144 participants with NES and 68 non-NES controls.

## **Aims and Hypotheses Results**

### **Specific Aim One**

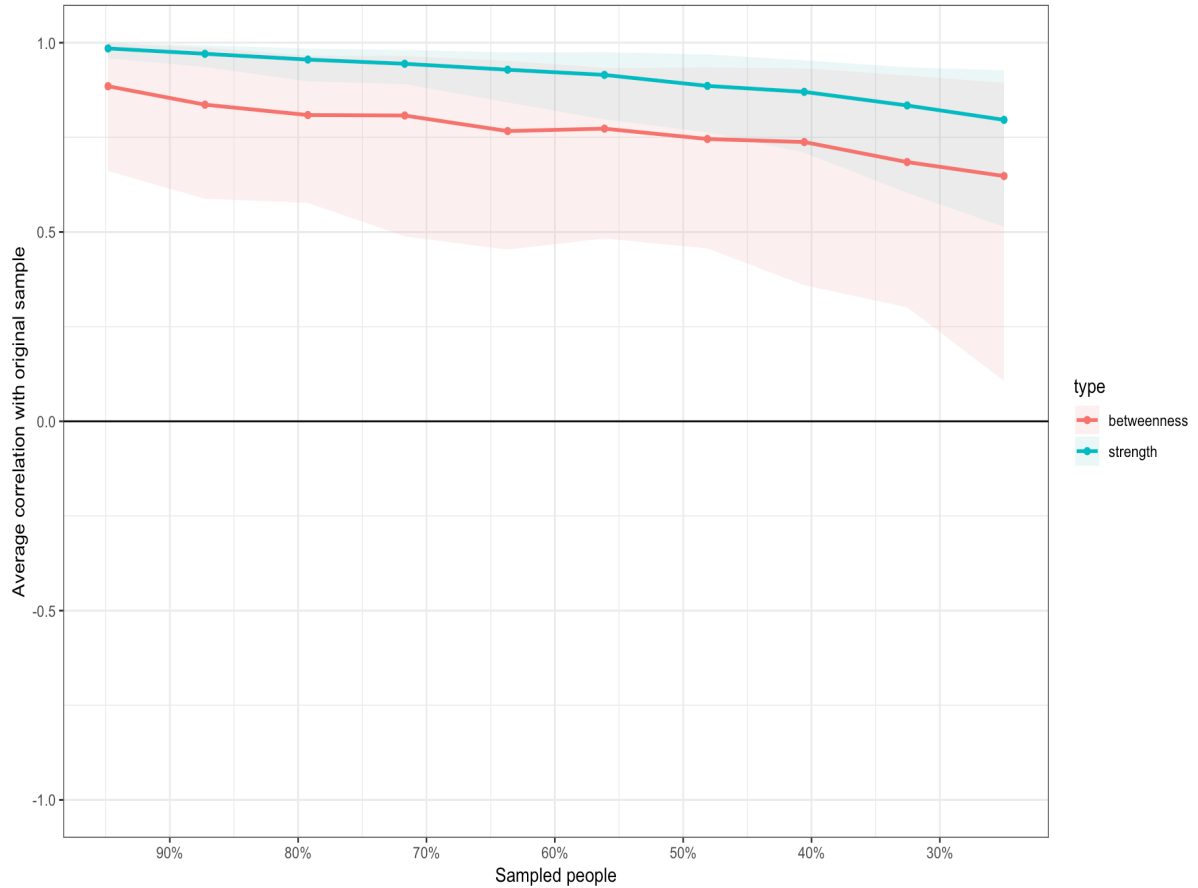
The first specific aim of this study was to characterize the psychopathology network of core symptoms of NES, including symptoms of mood, stress, sleep, and circadian rhythm, to identify which symptoms are most central to this disorder and are most likely to impact other NES symptoms.

**Hypothesis 1.** It was hypothesized that nocturnal ingestion of food and evening hyperphagia symptoms would present as the most central nodes within the psychopathology network of NES compared to other NES symptoms.

*Item selection.* The *goldbricker* function, which examined multicollinearity among nodes in the network, revealed two pairs of overlapping items (total score for the PSQI and

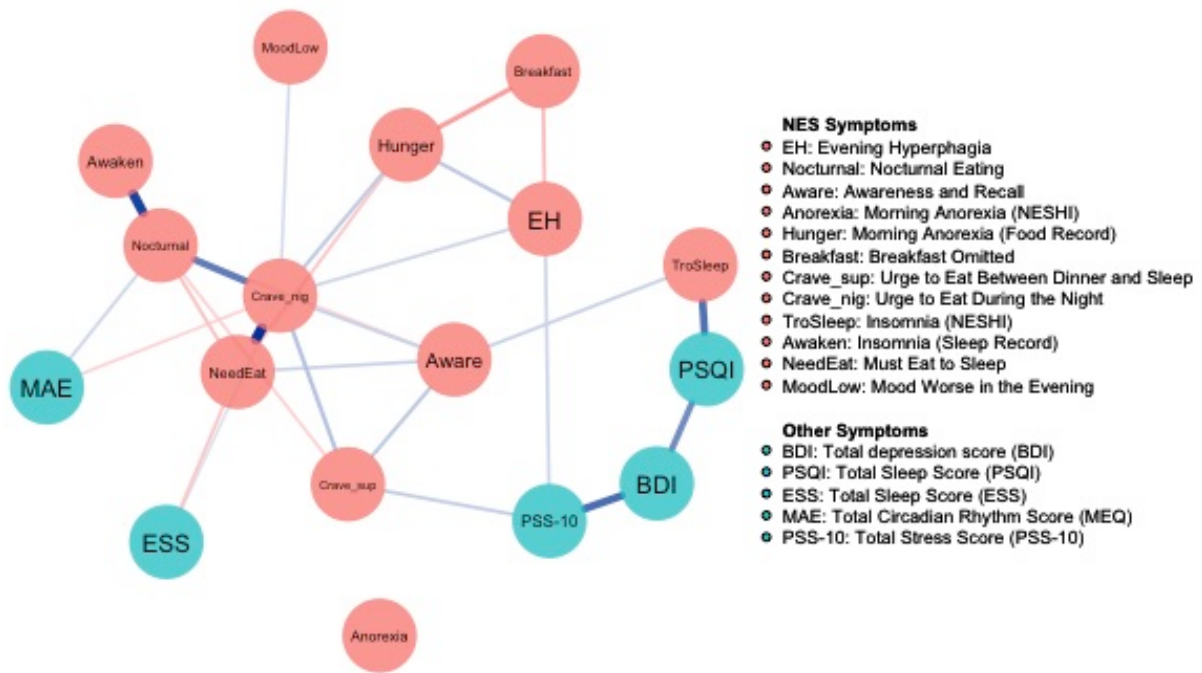
the current mood score on the NESHI; total score on the PSS and the current mood score on the NESHI), suggesting that these overlapping items were similar constructs. As the current mood score on the NESHI is theoretically captured by the total score of the BDI-II, I decided to remove the item capturing current mood score on the NESHI.

*Network stability.* Centrality stability (CS) coefficients in this network were: strength = 0.59, closeness = 0.00, and betweenness = 0.05 (See figure 5). This indicates that at least 59% of the sample can be dropped to retain a strength correlation of above 0.7 between the original index and the indices among the subsets. As stated above, Epskamp and colleagues (2018) have recommended a CS-coefficient no lower than 0.25, with preferred values above 0.5, but have also indicated that these are arbitrary cutoffs determined by a simulation study and are not definitive guidelines. As strength had the highest stability coefficient, and as prior literature has demonstrated that this index tends to be the most used in interpretations of central symptoms of network structures, I focused on this centrality index for interpretation. Given the low values for the other indices, it is likely that interpretations of these indices would be error-prone. Also, while it does not provide a CS-coefficient value, I chose to examine the expected influence centrality index as well, as it provides a more accurate measure of the strength centrality as the number of negative edges in a network increase.

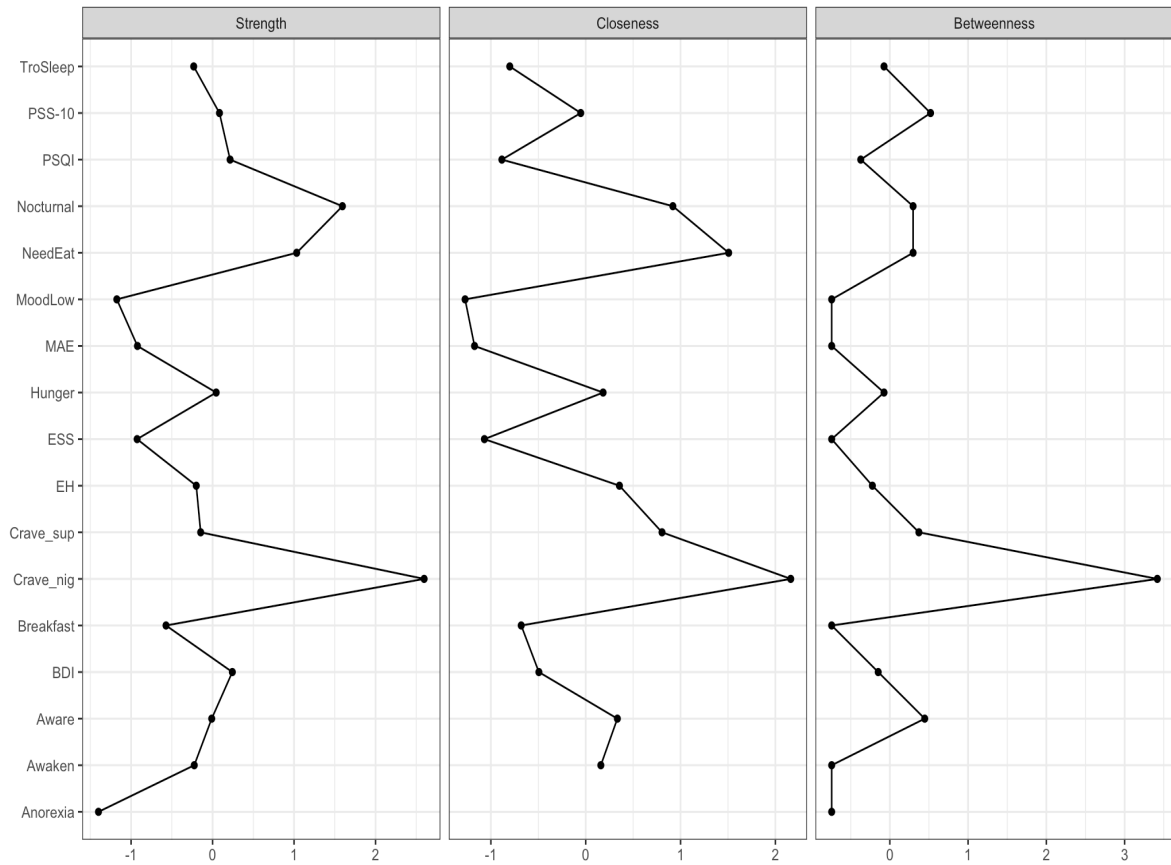


*Figure 5.* Bootstrapped centrality stability plot of the psychopathology network of core symptoms of NES and other symptoms. Closeness is not shown due to lack of variability in this index. Solid line indicates centrality correlation, and shaded area represents 95% confidence interval for the correlation.

*Network estimation.* The psychopathology network of core symptoms of NES, as well as the centrality plot, are presented in Figures 6 and 7. Urge to eat during the night had the highest strength ( $S = 2.60$ ) and expected influence ( $EI = 2.70$ ) centrality.



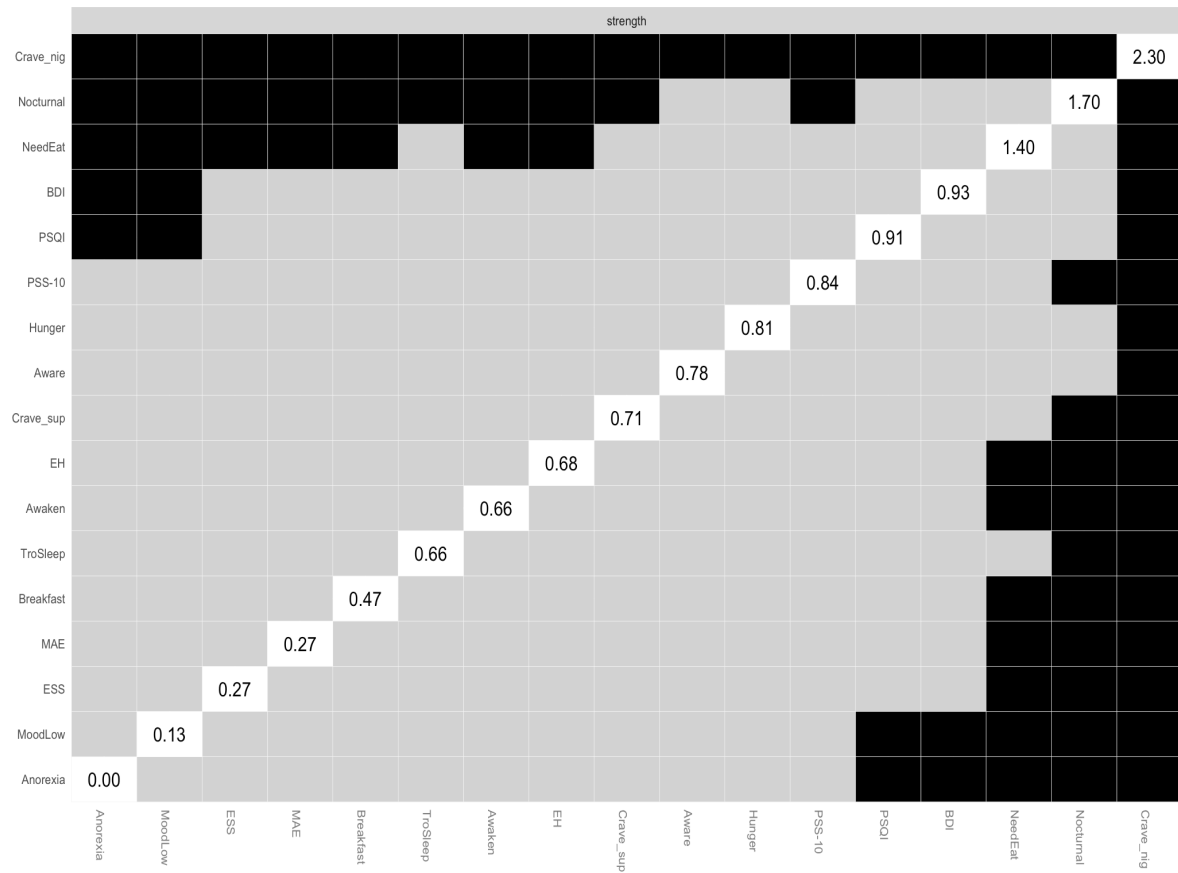
*Figure 6.* A psychopathology network of core symptoms of NES and other symptoms. Blue lines indicate positive associations, red lines indicate negative associations, and line thickness indicates strength of association (e.g., thicker line equals stronger association).



*Figure 7.* Centrality plot of the psychopathology network of core symptoms of NES and other symptoms. This plot is standardized on z-scores ( $M = 0$ ,  $SD = 1$ ) and higher numbers equate to greater centrality. Anorexia = morning anorexia (NESHI); awaken = insomnia (sleep record); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); nocturnal = nocturnal eating (food record); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESHI)

*Centrality difference test.* As stated previously, in order to be meaningfully interpreted, any high centrality values must be statistically significantly different from centrality values on all other symptoms in the network (Epskamp, Borsboom, et al., 2018). This is assessed using bootstrapped difference tests, and if there are no significant differences among node centrality indices, these indices should not be interpreted (Epskamp, Borsboom, et al., 2018). Using the R package *bootnet*, I conducted a centrality difference test on strength, calculating 2000 non-parametric bootstraps on the differences between all node strength values to determine if the bootstrapped difference confidence interval does not cross 0 (difference tests for expected influence are not currently available; Forrest et al., 2018). Figure 8 revealed that the strength centrality for urge to eat during the night was significantly different from all other nodes, suggesting that this symptom is not only highly central to this network, but is also significantly more central than other symptoms.





*Figure 8.* Bootstrapped strength difference results. Symptoms presented in descending order of strength, with values on the diagonal indicating unstandardized strength centrality values for the corresponding symptom. Black boxes represent significant differences in strength between symptoms, such that the bootstrapped confidence interval does not cross 0, and gray boxes represent nonsignificant differences, such that the bootstrapped confidence interval did cross 0. Anorexia = morning anorexia (NESHI); awaken = insomnia (sleep record); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); nocturnal =

nocturnal eating (food record); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESH1)

**Specific Aim One Summary.** Results from the network analysis did not support the hypothesis that nocturnal ingestion of food and evening hyperphagia symptoms would present as the most central nodes within the psychopathology network of NES compared to other symptoms. Urge to eat during the night was found to be highly central to this network and significantly more central than other NES symptoms, including symptoms of mood, stress, sleep, and circadian rhythm.

### **Specific Aim Two**

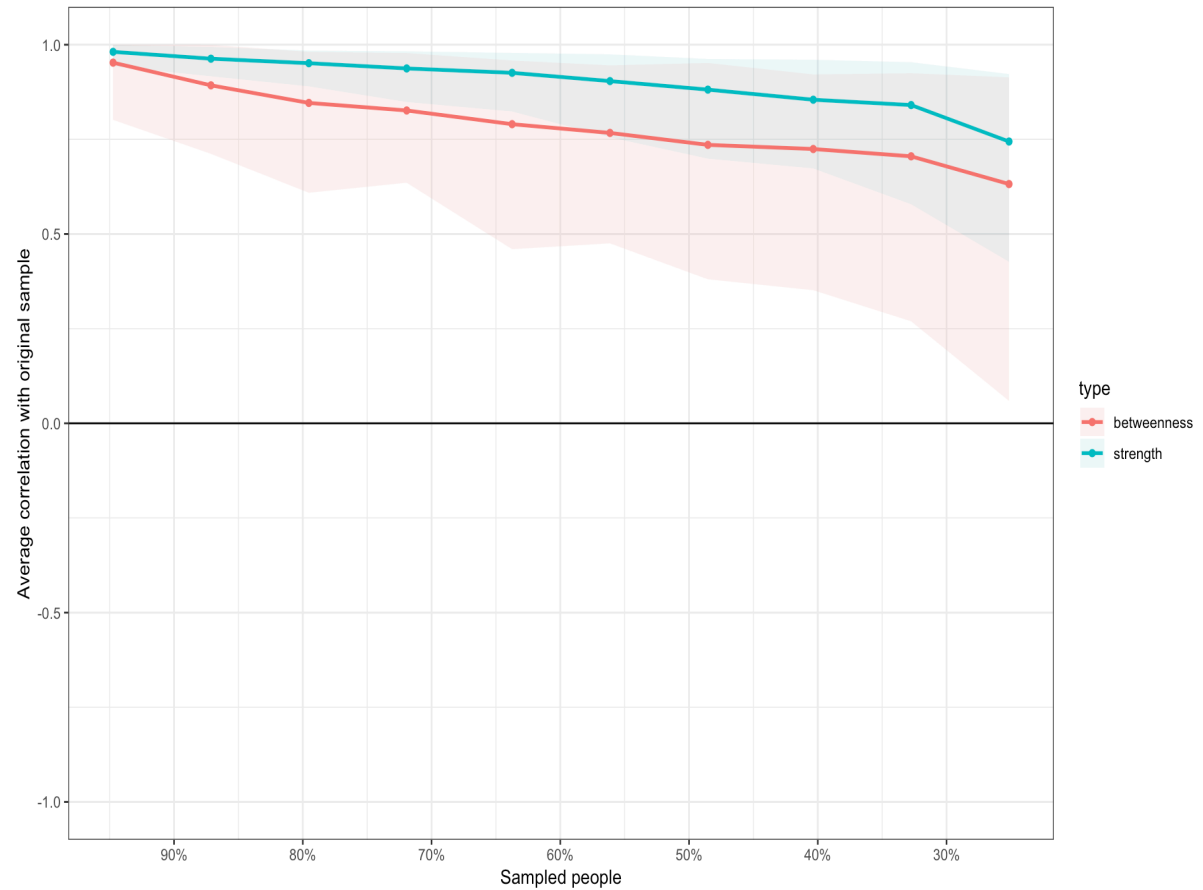
The second specific aim of this study was to identify key differences in the psychopathology networks of NES symptoms between persons with overweight/obesity and persons without overweight/obesity.

**Hypothesis 1.** It was hypothesized that nocturnal ingestion of food and evening hyperphagia symptoms would present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.

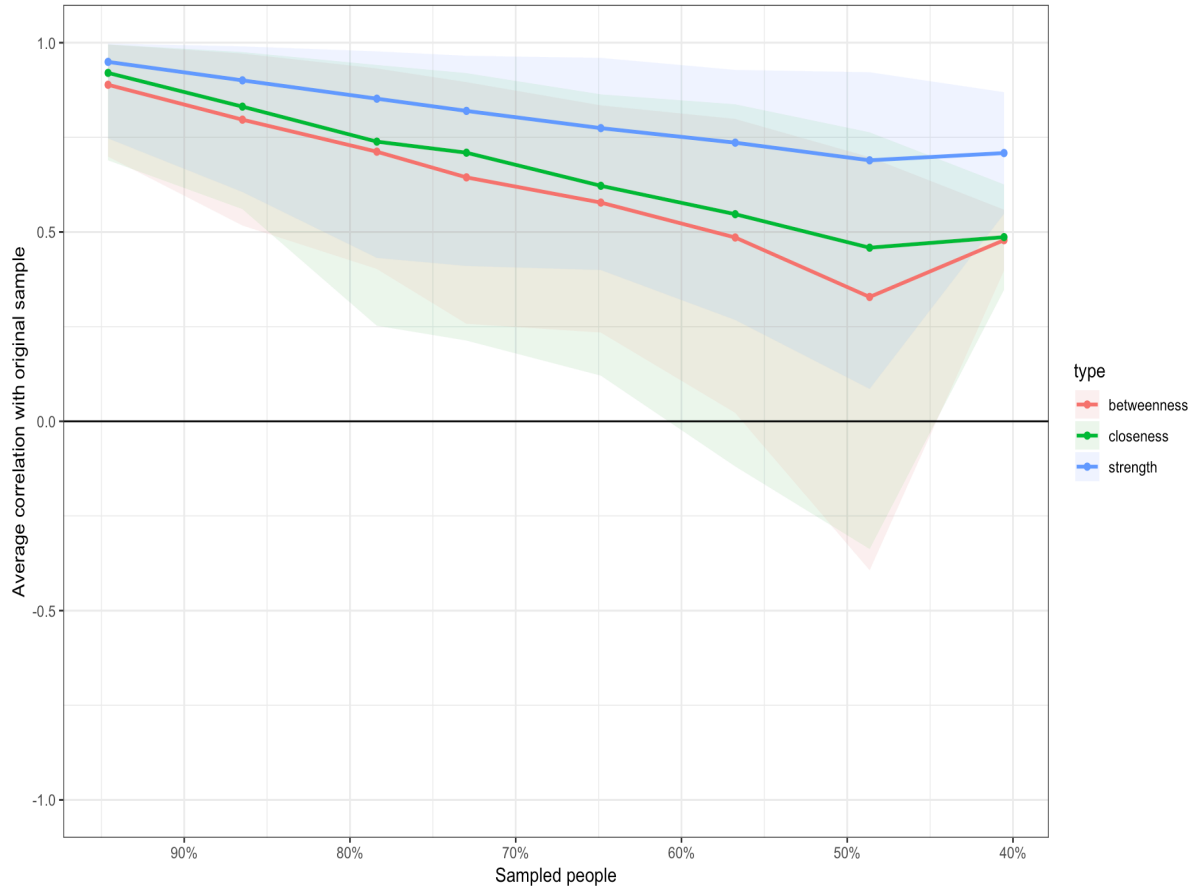
*Item selection.* Data were organized into two separate networks: those who were classified as persons with overweight/obesity ( $n = 171$ ) and persons without overweight/obesity ( $n = 41$ ). The *goldbricker* function revealed four pairs of overlapping items in the with overweight/obesity data (total score on the PSS and the current mood score on the NESH1; total score for the PSQI and total score for the BDI-II; total score on the PSS and total score on the BDI-II; and total score on the PSS and total score on the PSQI), suggesting that these overlapping items were similar constructs. Similar to the first network, I

decided to remove the item capturing current mood score on the NESHI. However, I chose not to remove the total scores for the BDI-II, PSQI, or PSS, as they theoretically and conceptually are different constructs (depressed mood, sleep quality, and stress level, respectively). In addition, previous literature has stated that *goldbricker* involves “research-guided judgement” in terms of choosing cutoffs (similar to interpreting a scree plot in principal components analysis) and can “output pairs that theoretically seem meaningless” (Levinson, Brosiof, et al., 2019, p. 699). No overlapping pairs were detected in the without overweight/obesity data, but current mood score on the NESHI was removed to preserve consistency between networks.

*Network stability.* CS coefficients for both networks were: With overweight/obesity network - strength = 0.60, closeness = 0.00, and betweenness = 0.13; Without overweight/obesity network – strength = 0.05, closeness = 0.00, and betweenness = 0.05 (See figure 9 and 10, respectively). This indicates that at least 60% of the sample of those with overweight/obesity and only 5% of the sample of those without overweight/obesity can be dropped to retain a strength correlation of above 0.7 between the original index and the indices among the subsets. As the centrality indices of the sample of those without overweight/obesity were well below Epskamp and colleagues (2018) recommended CS-coefficient of no lower than 0.25, it is improper to interpret these indices due to the likelihood of error.

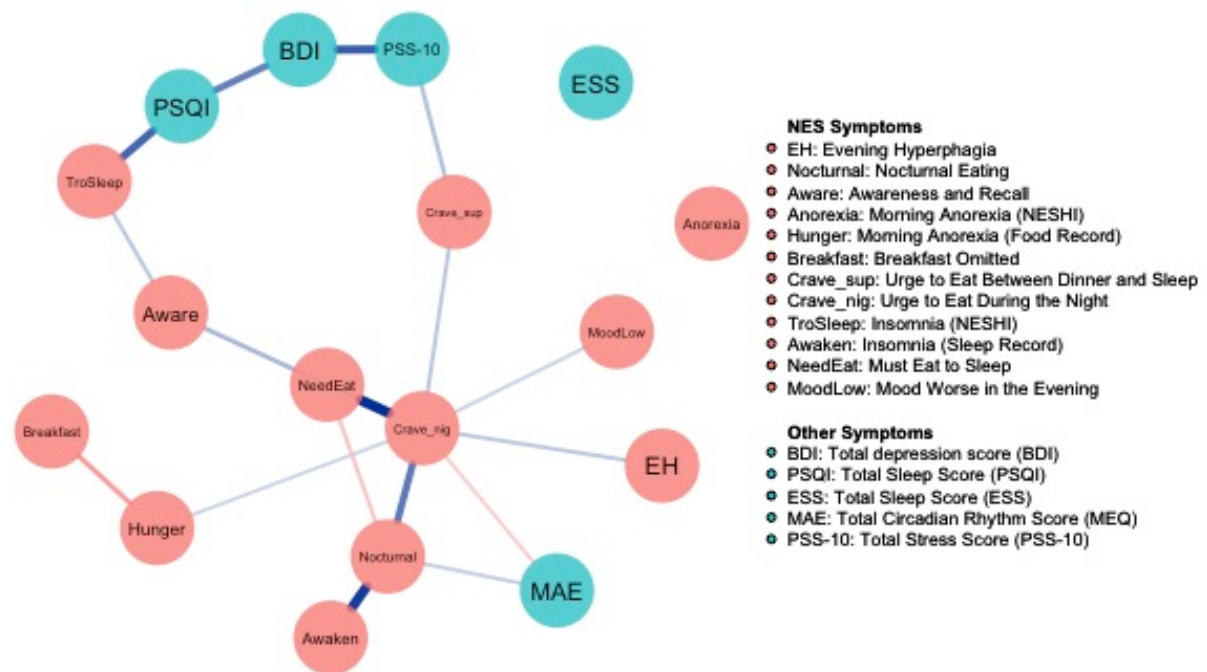


*Figure 9.* Bootstrapped centrality stability plot of the psychopathology network of core symptoms of NES and other symptoms in persons with overweight/obesity. Closeness is not shown due to lack of variability in this index.

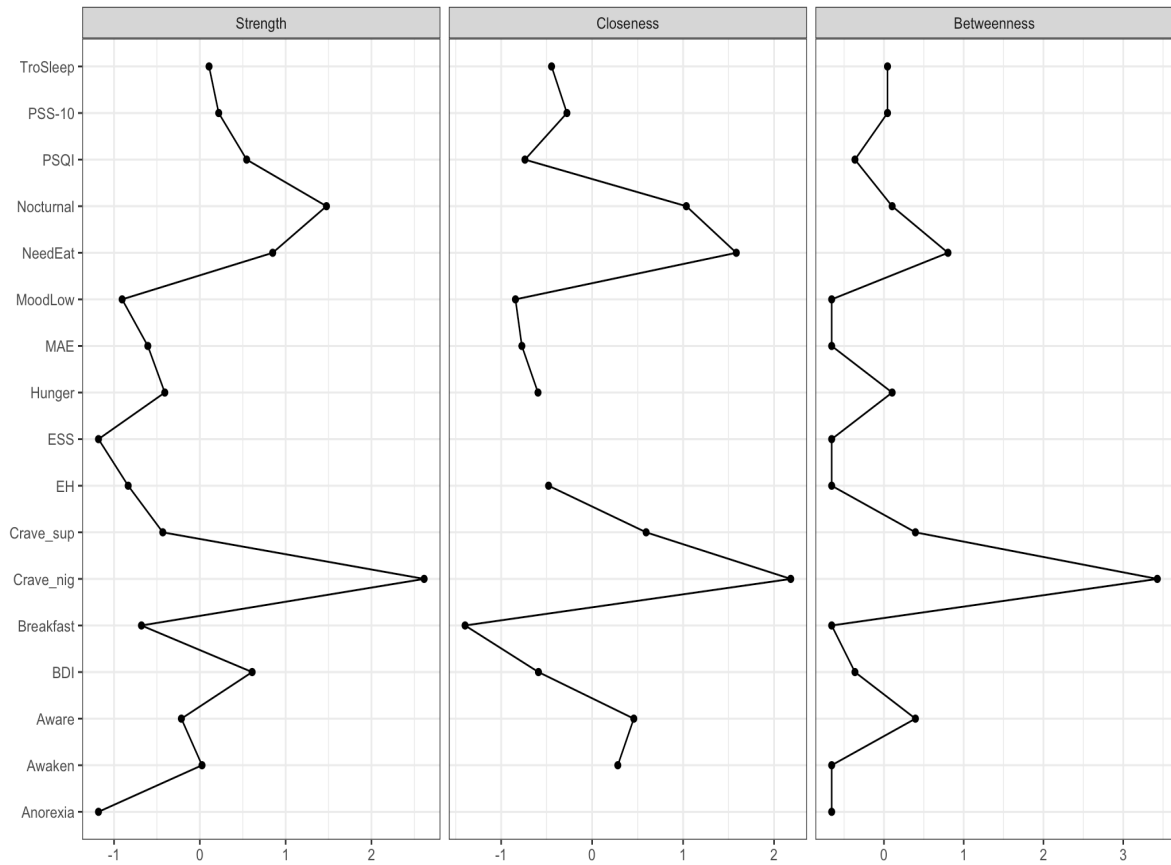


*Figure 10.* Bootstrapped centrality stability plot of the psychopathology network of core symptoms of NES and other symptoms in persons without overweight/obesity.

*Network estimation.* The psychopathology network of core symptoms of NES among persons with overweight/obesity, as well as the centrality plot, are presented in Figures 11 and 12. Urge to eat during the night had the highest strength ( $S = 2.61$ ) and expected influence ( $EI = 2.38$ ) centrality.



*Figure 11.* A psychopathology network of core symptoms of NES and other symptoms in persons with overweight/obesity. Blue lines indicate positive associations, red lines indicate negative associations, and line thickness indicates strength of association (e.g., thicker line equals stronger association).

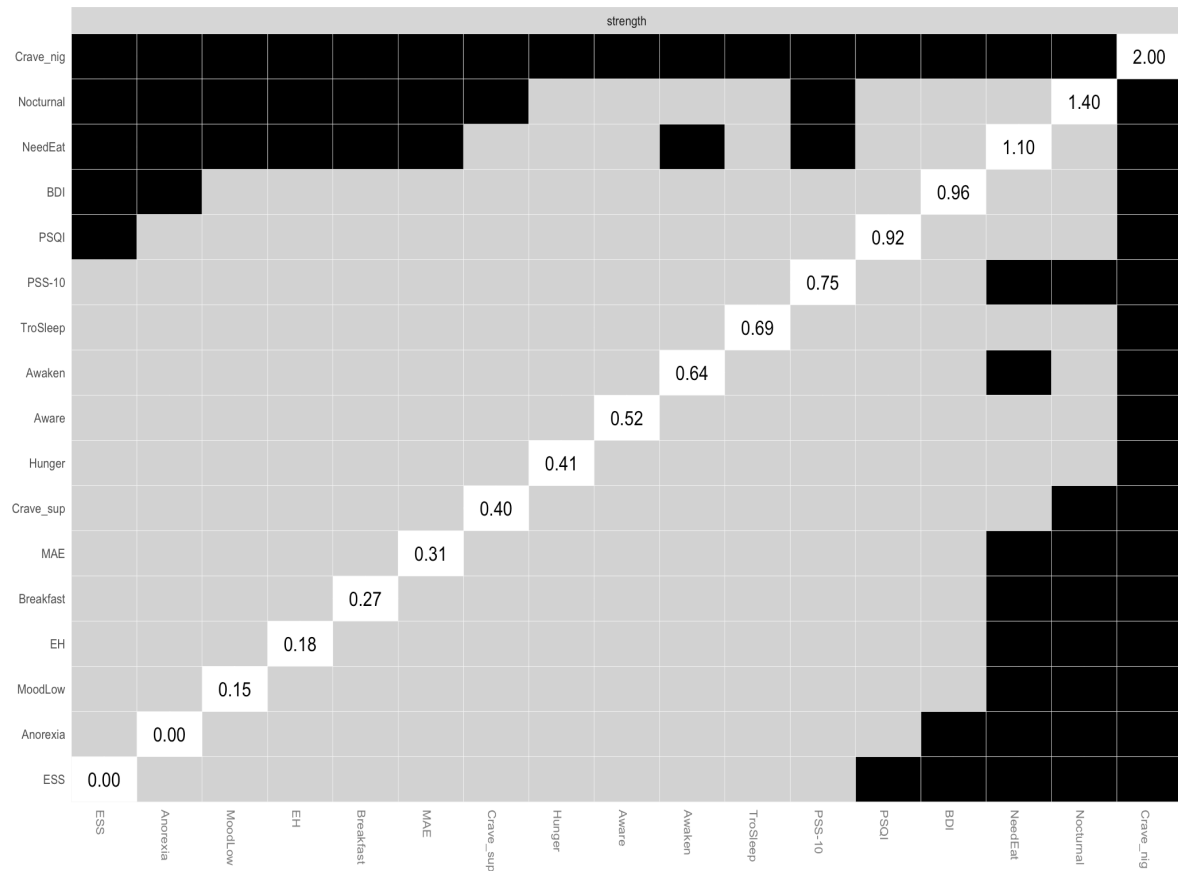


*Figure 12.* Centrality plot of the psychopathology network of core symptoms of NES and other symptoms in persons with overweight/obesity. This plot is standardized on z-scores ( $M = 0$ ,  $SD = 1$ ) and higher numbers equate to greater centrality. Anorexia = morning anorexia (NESHI); awaken = insomnia (sleep record); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must eat to sleep (NESHI); nocturnal = nocturnal eating (food record); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESHI)

Estimation of the psychopathology network of core symptoms of NES among persons without overweight/obesity resulted in a correlation matrix that was non-positive definite. It is likely that this is due to the small sample size of this subset of data ( $n = 41$ ) relative to the number of parameters being estimated, despite the use of the LASSO technique. However, the *bootnet* package in R was able to determine the nearest possible positive definite matrix. Within this estimated network, urge to eat during the night also had the highest strength ( $S = 2.30$ ) and expected influence ( $EI = 1.85$ ) centrality. Nevertheless, while these results were similar to the network of persons with overweight/obesity, these indices should not be interpreted due to poor stability.

*Centrality difference test.* Similar to aim one, I conducted a centrality difference test on the strength centrality for the network of persons with overweight/obesity. Figure 13 reveals that the strength centrality for urge to eat during the night was significantly different from all other nodes, suggesting that this symptom is not only highly central to this network, but is also significantly more central than other symptoms.





*Figure 13.* Bootstrapped strength difference results. Symptoms presented in descending order of strength, with values on the diagonal indicating unstandardized strength centrality values for the corresponding symptom. Black boxes represent significant differences in strength between symptoms, such that the bootstrapped confidence interval does not cross 0, and gray boxes represent nonsignificant differences, such that the bootstrapped confidence interval did cross 0. Anorexia = morning anorexia (NESHI); awaken = insomnia (sleep record); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must

eat to sleep (NESH1); nocturnal = nocturnal eating (food record); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESH1)

**Hypothesis 2:** It was hypothesized that the overall network structure would be dissimilar between populations of persons with and without overweight/obesity.

*Network comparison test.* Using the *NetworkComparisonTest* package in R (van Borkulo et al., 2015), I attempted to conduct a Network Comparison Test (NCT) between the networks of populations of persons with and without overweight/obesity to determine if these networks were significantly different from one another. I aimed to assess invariance in network structure and global strength, which tests whether any edges in the networks are significantly different, and to compare the weighted absolute sum of all the edges across networks, respectively. Indeed, NCT can determine if the exact same symptoms are connected (i.e., network structure) and how strongly these symptoms are connected (i.e., network density/global strength) between these two networks. Results of the NCT revealed no significant network structure or global strength invariance between these two networks (network invariance  $[M] = 0.32, p = .86$ ; global strength invariance  $[S] = 0.75$ , with overweight/obesity network = 6.83, without overweight/obesity network = 7.58,  $p = .86$ ). This suggests no differences in the central symptoms in the networks between persons with or without overweight/obesity, nor differences in connectivity between symptoms in both networks. However, due to the instability of the network of persons without overweight/obesity, these tests for potential invariance between these two networks should be interpreted with extreme caution.

**Specific Aim Two Summary.** Results from the network analysis was inconclusive regarding the hypothesis that nocturnal ingestion of food and evening hyperphagia symptoms

would present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity. Urge to eat during the night was found to be highly central to both networks and significantly more central than other NES symptoms, including symptoms of mood, stress, sleep, and circadian rhythm. However, the network of symptoms among persons without overweight/obesity had very poor stability among the three centrality indices and results from this network should not be interpreted. In addition, the hypothesis that the overall network structure would be dissimilar between populations of persons with and without overweight/obesity was inconclusive, as the lack of adequate stability among the centrality indices limits the interpretability of this analysis.

### **Specific Aim Three**

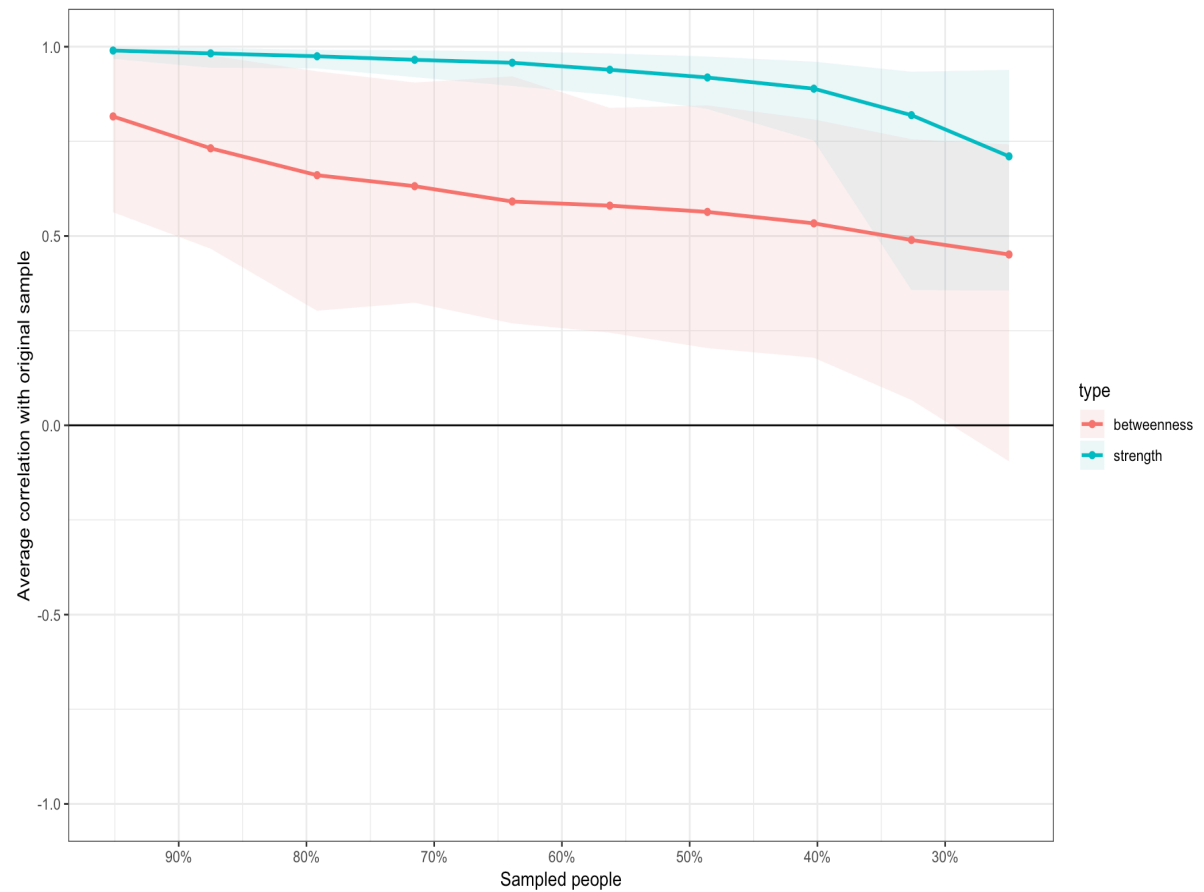
The third specific aim of this study was to determine differences in the psychopathology network structure and density of NES symptoms between individuals with NES and non-NES control group.

**Hypothesis 1.** It was hypothesized that the overall network structure would be similar between individuals with NES and non-NES controls.

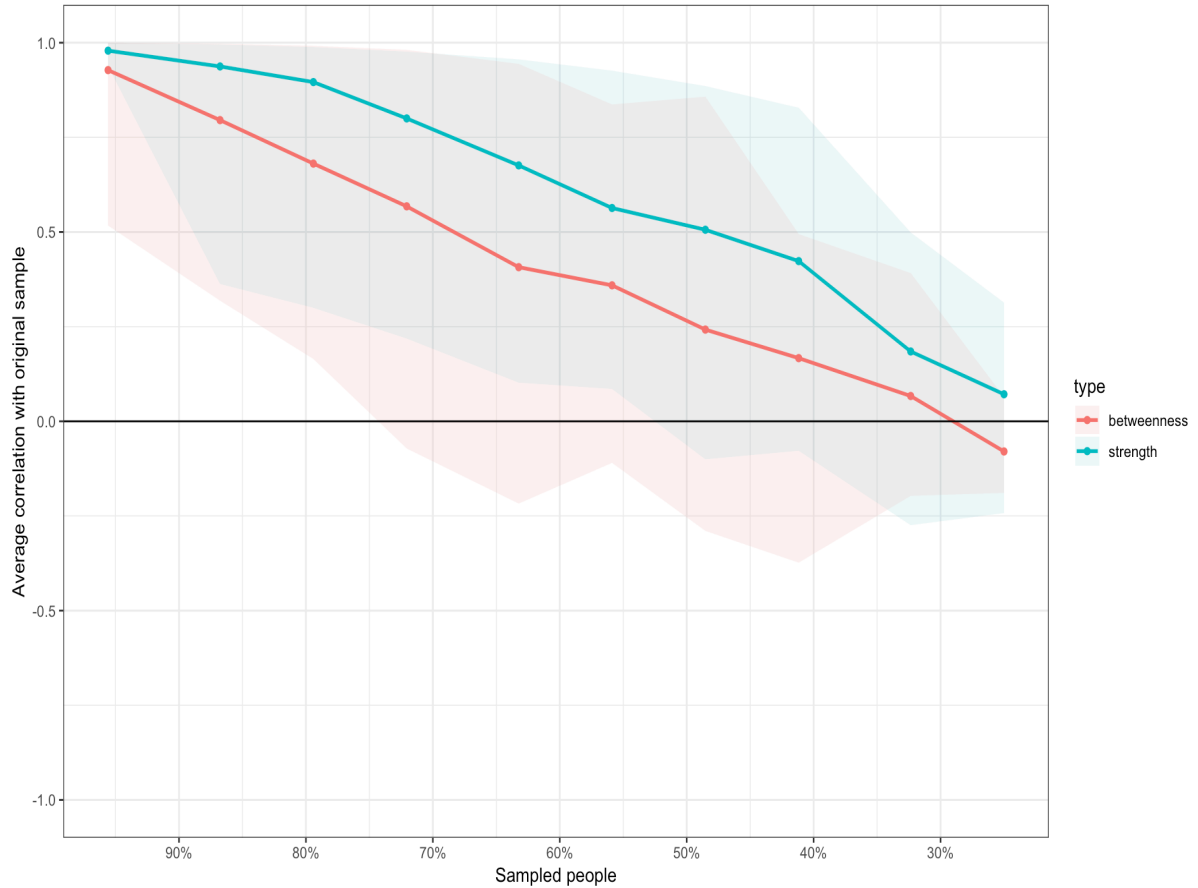
*Item selection.* Data were organized into two separate networks: those who were classified as individual with NES ( $n = 144$ ) and non-NES controls ( $n = 68$ ). The *goldbricker* function revealed one pair of overlapping items in the NES data (total number of awakenings over seven days according to sleep records and the total number of nocturnal ingestions over seven days according to food records), suggesting that these overlapping items were similar constructs. I decided to remove the item capturing the total number of awakenings over the past seven days. In the non-NES control data, there was one pair of overlapping items

detected (total score on the PSS and total score on the BDI-II), but only the total number of awakenings over the past seven days was removed to preserve consistency between this network and the NES network.

*Network stability.* Centrality stability (CS) coefficients for both networks were: Individuals with NES network - strength = 0.60, closeness = 0.00, and betweenness = 0.00; non-NES control network - strength = 0.04, closeness = 0.00, and betweenness = 0.00 (See figure 14 and 15, respectively). This indicates that at least 60% of the sample of individuals with NES and only 4% of the non-NES control sample can be dropped to retain a strength correlation of above 0.7 between the original index and the indices among the subsets. As the centrality indices of the non-NES control sample were well below Epskamp and colleagues (2018) recommended CS-coefficient of no lower than 0.25, it is improper to interpret these indices due to the likelihood of error.

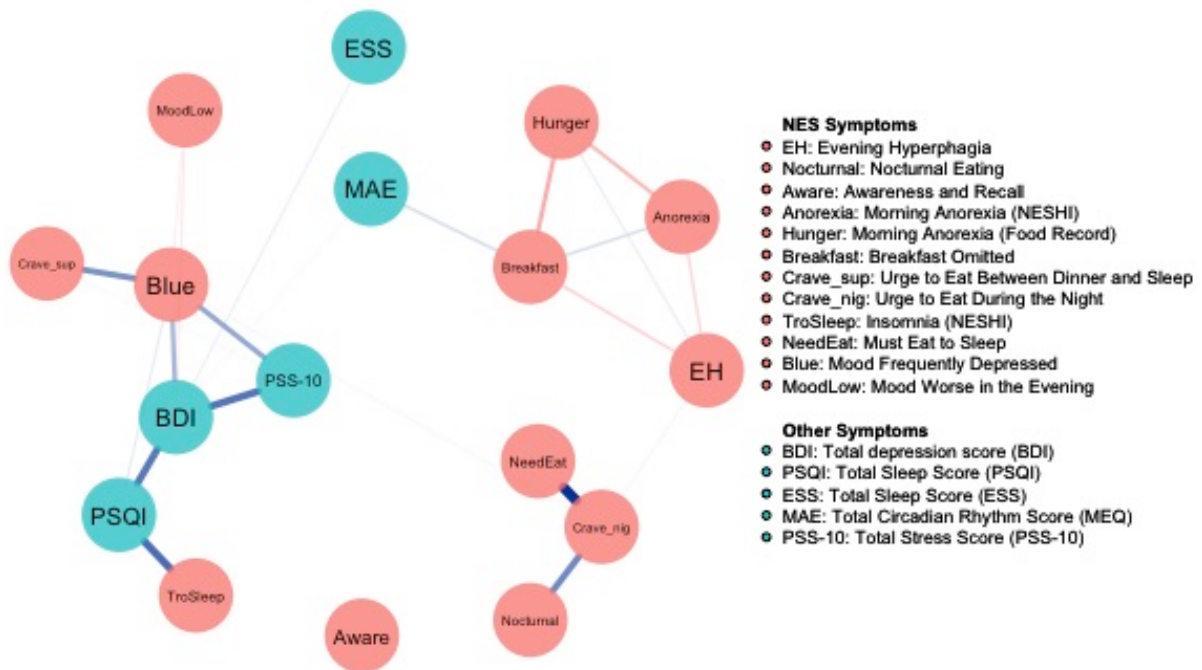


*Figure 14.* Bootstrapped centrality stability plot of the psychopathology network of core symptoms of NES and other symptoms in individuals with NES.

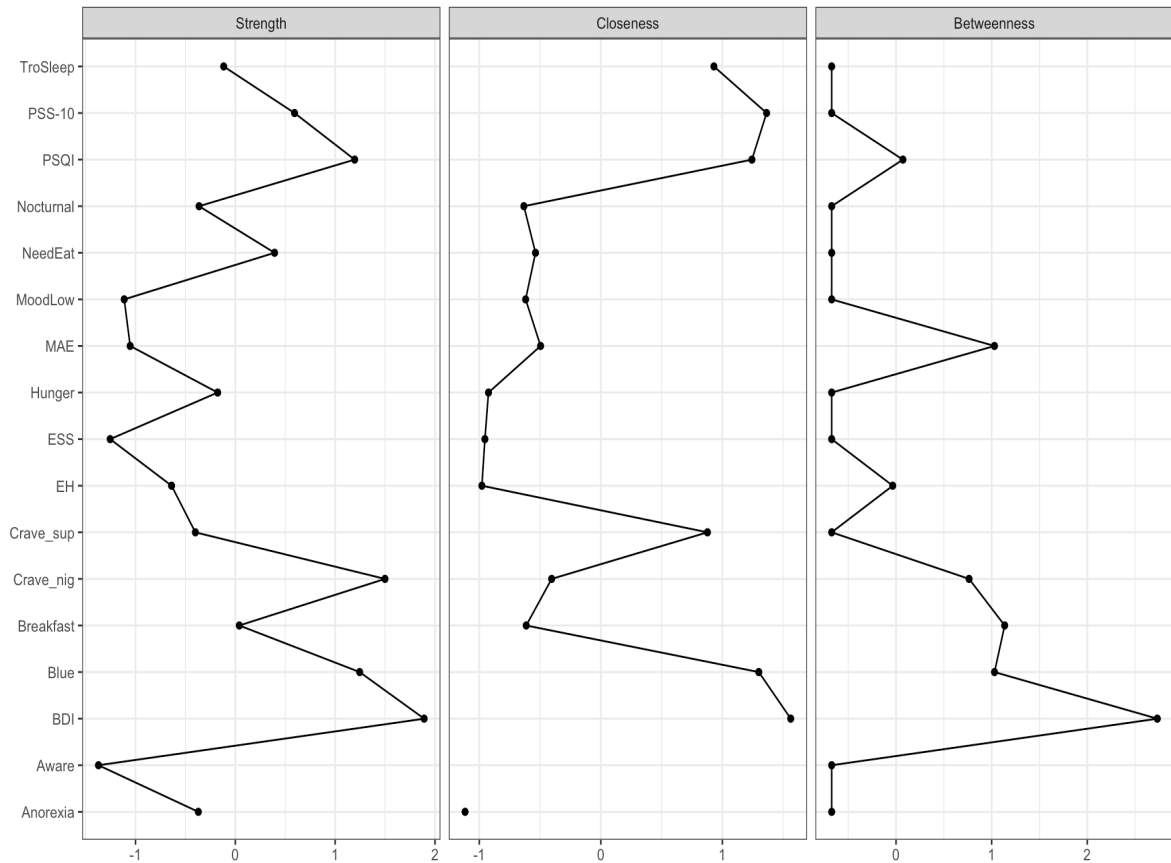


*Figure 15.* Bootstrapped centrality stability plot of the psychopathology network of core symptoms of NES and other symptoms in a non-NES control sample.

*Network estimation.* The psychopathology network of core symptoms of NES in a sample of individuals with NES, as well as the centrality plot, are presented in Figures 16 and 17. Depressed mood and urge to eat in the middle of the night had the highest strength ( $S = 1.89$ ;  $S = 1.50$ , respectively) and expected influence ( $EI = 1.53$ ;  $EI = 1.52$ , respectively) centrality.



*Figure 16.* A psychopathology network of core symptoms of NES and other symptoms in individuals with NES. Blue lines indicate positive associations, red lines indicate negative associations, and line thickness indicates strength of association (e.g., thicker line equals stronger association).

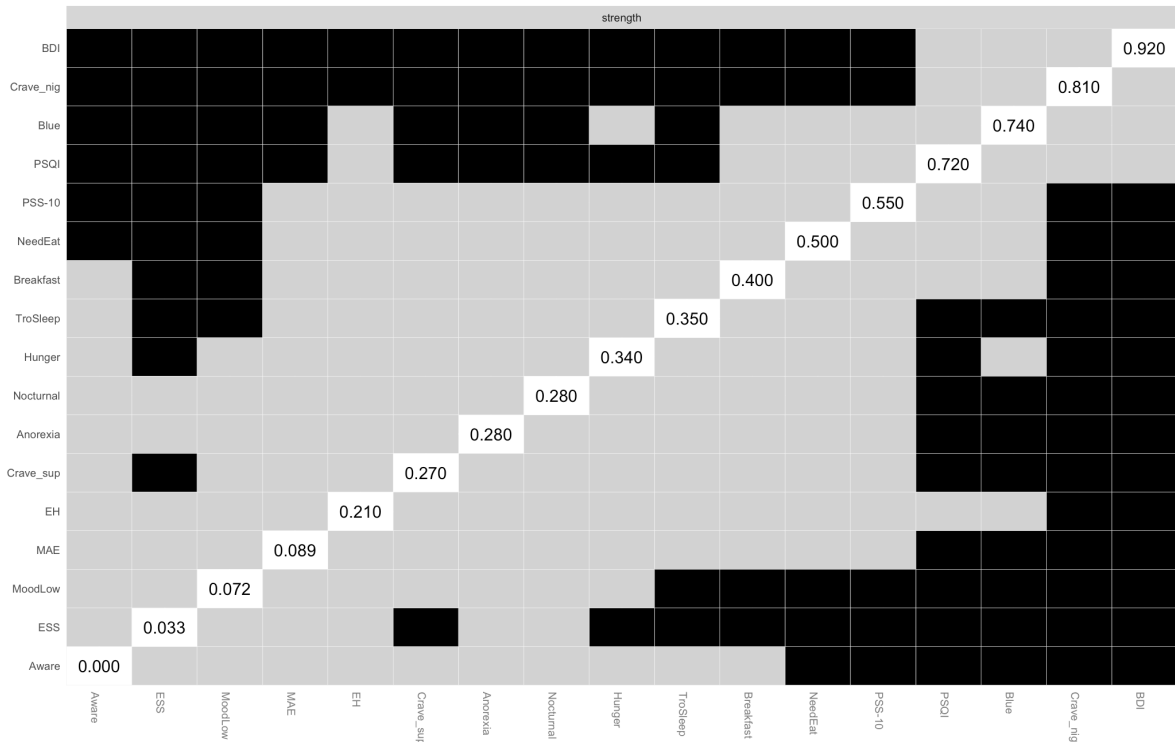


*Figure 17.* Centrality plot of the psychopathology network of core symptoms of NES and other symptoms in individuals with NES. This plot is standardized on z-scores ( $M = 0$ ,  $SD = 1$ ) and higher numbers equate to greater centrality. Anorexia = morning anorexia (NESHI); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; blue = mood frequently depressed (NESHI); breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must eat to sleep (NESHI); nocturnal = nocturnal eating (food record); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESHI).



Similar to aim two, estimation of the psychopathology network of core symptoms of NES among non-NES controls resulted in a correlation matrix that was non-positive definite. It is likely that this is due to the small sample size of this subset of data ( $n = 68$ ) relative to the number of parameters being estimated, despite the use of the LASSO technique. However, the *bootnet* package in R was able to determine the nearest possible positive definite matrix. Within this estimated network, total number of nocturnal ingestions over seven days, urge to eat during the night, and depressed mood had the highest strength centrality ( $S = 1.82$ ;  $S = 1.75$ ;  $S = 1.49$ , respectively), and depressed mood and urge to eat during the night had the highest expected influence ( $EI = 2.07$  and  $1.32$ , respectively). Nevertheless, these indices should not be interpreted due to poor stability.

*Centrality difference test.* Similar to the previous aims, I conducted a centrality difference test on the strength centrality for the network of individuals with NES. Figure 18 reveals that the strength centrality for depressed mood was significantly different from 81% of all other nodes (excluding sleep quality, depressed mood on the NESHI, and urge to eat during the night), and urge to eat during the night was also significantly different from 81% of all other nodes. This suggests that depressed mood and urge to eat during the night are not only highly central to this network, but are also significantly more central than most other symptoms.



*Figure 18.* Bootstrapped strength difference results. Symptoms presented in descending order of strength, with values on the diagonal indicating unstandardized strength centrality values for the corresponding symptom. Black boxes represent significant differences in strength between symptoms, such that the bootstrapped confidence interval does not cross 0, and gray boxes represent nonsignificant differences, such that the bootstrapped confidence interval did cross 0. Anorexia = morning anorexia (NESHI); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; blue = mood frequently depressed (NESHI); breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); EH = evening hyperphagia (food record); ESS = total sleep score on the ESS; hunger = morning anorexia (food record); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must eat to sleep (NESHI); nocturnal = nocturnal eating (food record); PSQI =

total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; trosleep = insomnia (NESHI).

*Network comparison test.* Results of the NCT revealed significant network structure invariance between these two networks (network invariance  $[M] = 0.47, p = .01$ ). This suggests that the central symptoms in the networks between individuals with and without NES differ. However, due to the instability of the network of individuals without NES, these tests for potential invariance between these two networks should be interpreted with extreme caution.

**Hypothesis 2.** It was hypothesized that the network density would be greater in the group with NES compared to non-NES controls.

*Network comparison test.* Results of the NCT revealed no significant network global strength invariance between these two networks (global strength invariance  $[S] = 1.27$ , with NES = 2.97, non-NES controls = 4.24,  $p = .13$ ). This suggests that there are no differences in connectivity between symptoms in both networks. However, as stated above, due to the instability of the network of individuals without NES, these tests for potential invariance between these two networks should be interpreted with extreme caution.

**Specific Aim Three Summary.** Results from the network analysis were inconclusive regarding the hypothesis that the overall network structure would be similar between individuals with NES and non-NES controls. Depressed mood and urge to eat in the middle of the night were found to be highly central to the network of individuals with NES and significantly more central than most other NES symptoms, including symptoms of mood, stress, sleep, and circadian rhythm. However, while the total number of nocturnal ingestions over seven days, urge to eat during the night, and depressed mood were most central to the

network of symptoms among the non-NES control individuals, this network had very poor stability among the three centrality indices and thus results should not be interpreted. In addition, the hypothesis that the network density would be greater in the group with NES compared to non-NES controls was also not supported, though again the lack of adequate stability among the centrality indices limits the interpretability of this analysis.

#### **Specific Aim Four**

The fourth specific aim of this study was to assess the validity of the transdiagnostic cognitive-behavioral model of eating disorders within an NES network by examining if overvaluation of shape and weight would be most central when added to the model. As stated above, emerging network analysis literature of eating disorders demonstrated these symptoms as central regardless of diagnosis, and if NES were to fit into the transdiagnostic model of EDs, then this sample should exhibit a similar pattern. Otherwise, NES may be a conceptually different kind of feeding and eating disorder (i.e., one without shape/weight concerns).

**Hypothesis 1.** It was hypothesized that overvaluation of shape and weight would present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity.

*Item selection.* Three items (episodes of self-induced vomiting, episodes of laxative misuse, and episodes of diuretics misuse) were removed due to limited endorsement (e.g., only one individual reported at least one episode of self-induced vomiting, six individuals reported laxative misuse, and one individual reported diuretic misuse). The item assessing episodes of subjective bingeing was removed as well, with episodes of objective bingeing representing “binge eating” in this population. The *goldbricker* function revealed 36 pairs of

overlapping items, most being combinations of items from the EDE, and were to be expected given that the EDE has four subscales made up of similar items within each subscale.

However, there remained six pairs of non-EDE items (total score for the PSQI and the current mood score on the NESHI; total score on the PSS and the current mood score on the NESHI; total number of awakenings over seven days according to sleep records and the total number of nocturnal ingestions over seven days according to food records; total score for the PSQI and urge to eat during the night; total score on the PSS and urge to eat during the night; and total score on the PSS and the total mood score on the BDI-II), suggesting that these overlapping items were similar constructs. Similar to previous aims, I decided to remove the item capturing current mood score on the NESHI and the total number of nocturnal awakenings over the past seven days.

*Network stability.* CS coefficients in this network were: strength = 0.44, closeness = 0.13, and betweenness = 0.13 (See figure 19). This indicates that at least 44% of the sample can be dropped to retain a strength correlation of above 0.7 between the original index and the indices among the subsets. This value for the strength centrality index was above Epskamp and colleagues (2018) recommendation of 0.25, though below the preferred value of above 0.5. As such, I focused on this centrality index for interpretation, as well as the expected influence centrality index.

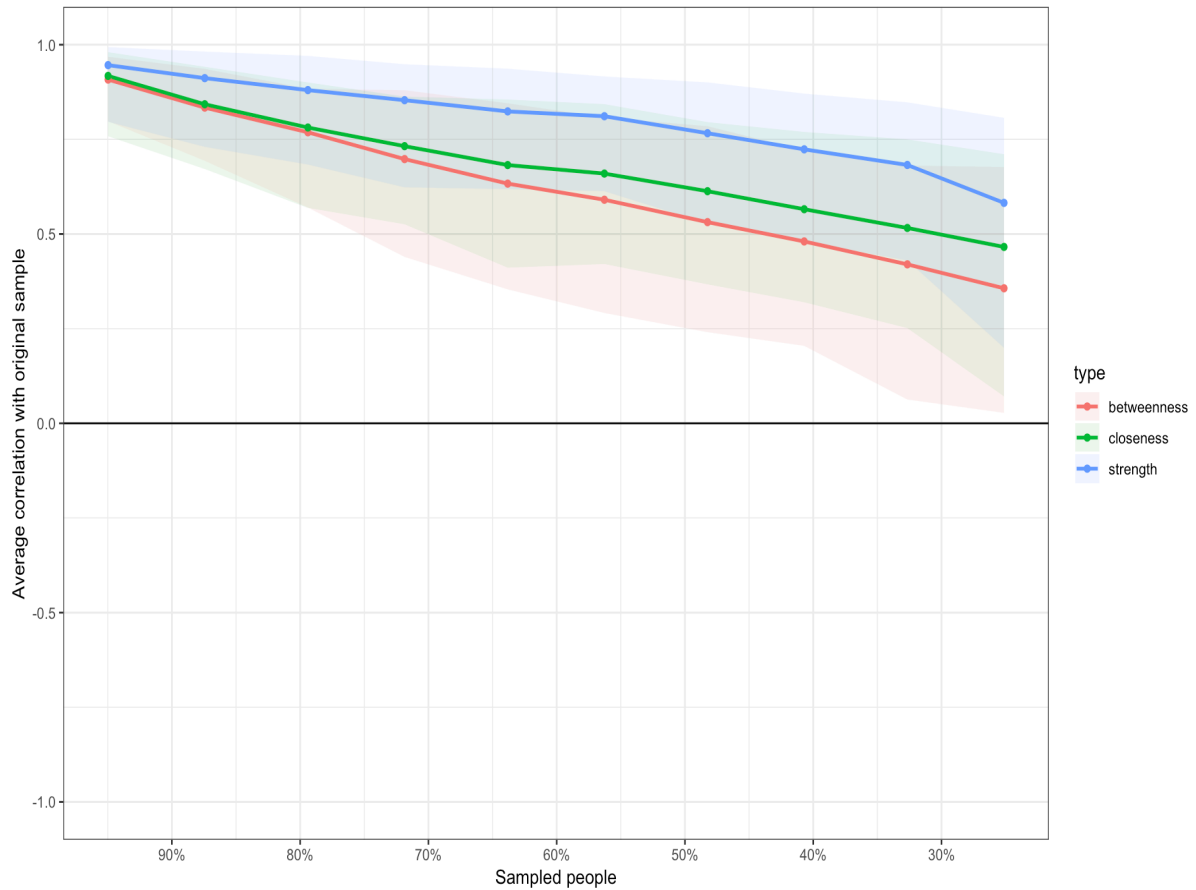
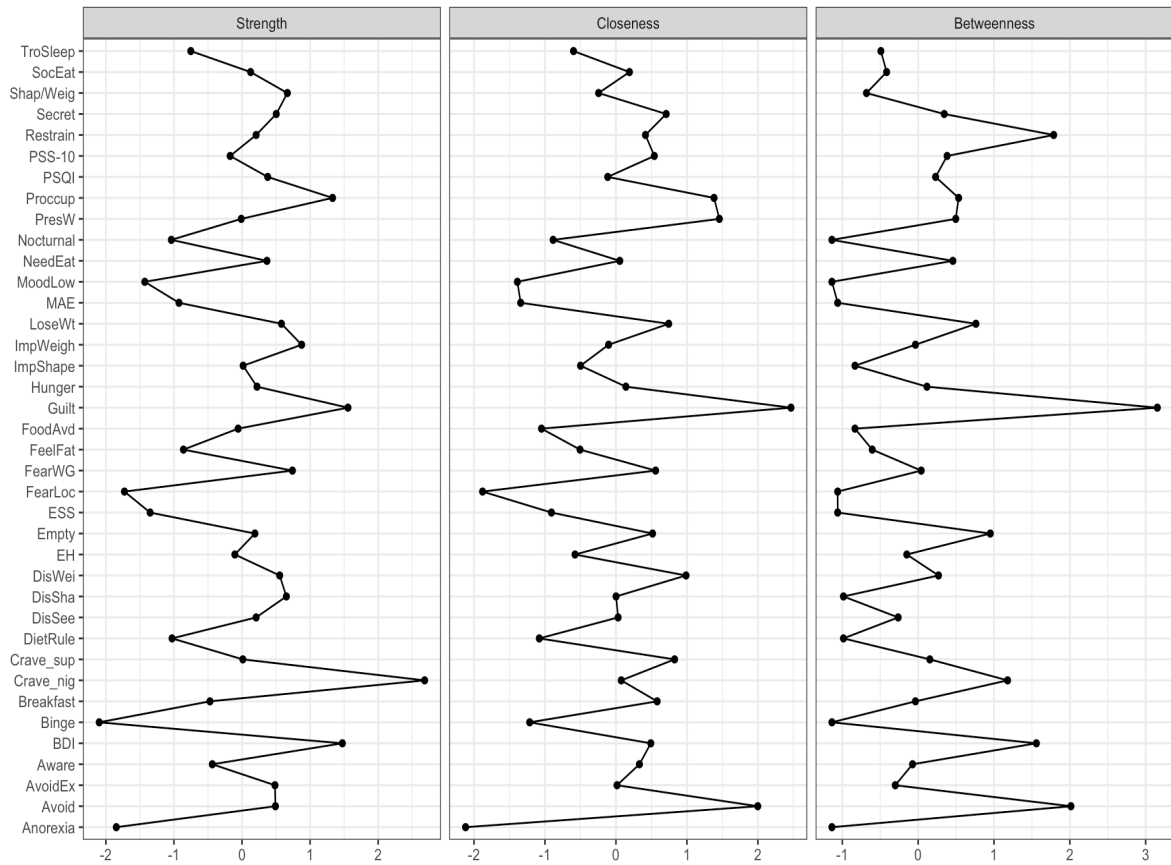


Figure 19. Bootstrapped centrality stability plot of the transdiagnostic cognitive-behavioral model network including core symptoms of NES and other symptoms.

*Network estimation.* The transdiagnostic cognitive-behavioral model network that includes core symptoms of NES, as well as the centrality plot, are presented in Figures 20 and 21. Urge to eat during the night had the highest strength ( $S = 2.68$ ) and expected influence ( $EI = 2.20$ ) centrality. While guilt over eating and preoccupation with food, eating, and calories had the next highest strength values ( $S = 1.56$  and  $1.33$ , respectively), total mood score on the BDI-II and importance of weight had the next highest expected influence ( $EI = 1.12$  and  $1.11$ , respectively).





*Figure 21.* Centrality plot of the transdiagnostic cognitive-behavioral model network

including core symptoms of NES and other symptoms. This plot is standardized on z-scores

( $M = 0$ ,  $SD = 1$ ) and higher numbers equate to greater centrality. Anorexia = morning

anorexia (NESHI); avoid = avoidance of eating (EDE); avoidex = avoidance of exposure

(EDE); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; binge =

binge episodes; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night

(NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); dietrule = dietary rules

(EDE); dissee = discomfort with seeing body (EDE); dissha = dissatisfaction with shape

(EDE); diswei = dissatisfaction with weight (EDE); EH = evening hyperphagia (food

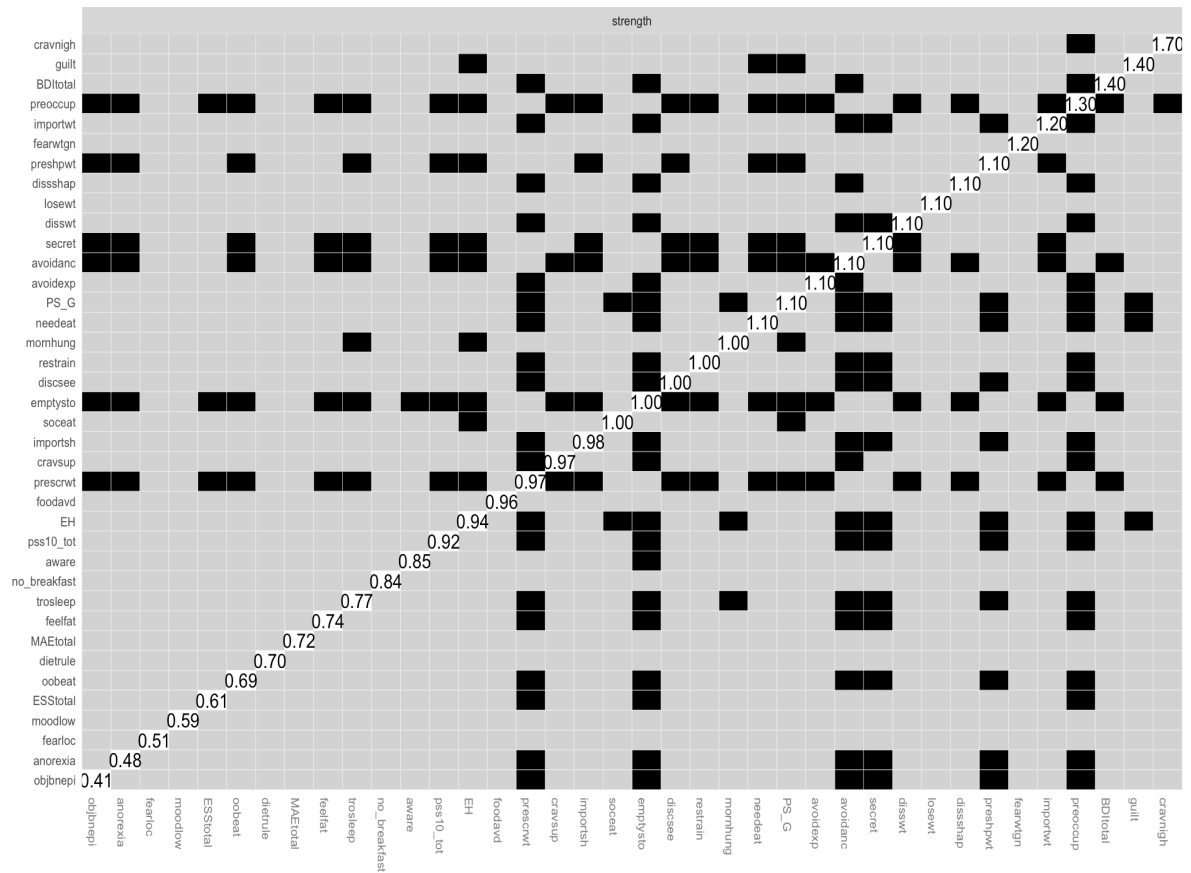
record); empty = empty stomach (EDE); ESS = total sleep score on the ESS; fearloc = fear of

losing control over eating (EDE); fearwg = fear of weight gain (EDE); feelfat = feeling fat



(EDE); foodavd = food avoidance (EDE); guilt = guilt about eating (EDE); hunger = morning anorexia (food record); impshape = importance of shape (EDE); impweigh = importance of weight (EDE); losewt = desire to lose weight (EDE); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must eat to sleep (NESHI); nocturnal = nocturnal eating (food record); presw = reaction to weighing (EDE); proccup = preoccupation with food (EDE); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; Restrain = restraint over eating (EDE); secret = eating in secret (EDE); shap/weig = preoccupation with shape and weight (EDE); soceat = social eating (EDE); trosleep = insomnia (NESHI).

*Centrality difference test.* I conducted a centrality difference test on strength. Figure 22 revealed that the strength centrality values for the above listed nodes were not significantly different from all other nodes, and that having an empty stomach and avoidance of exposure were significantly different from 59% and 49% of all other nodes.



*Figure 22.* Bootstrapped strength difference results. Symptoms presented in descending order of strength, with values on the diagonal indicating unstandardized strength centrality values for the corresponding symptom. Black boxes represent significant differences in strength between symptoms, such that the bootstrapped confidence interval does not cross 0, and gray boxes represent nonsignificant differences, such that the bootstrapped confidence interval did cross 0. Anorexia = morning anorexia (NESHI); avoid = avoidance of eating (EDE); avoidexp = avoidance of exposure (EDE); aware = awareness and recall (NESHI); BDI = total mood score on BDI-II; binge = binge episodes; breakfast = breakfast omitted (EDE); crave\_nig = urge to eat during the night (NESHI); crave\_sup = urge to eat between dinner and sleep (NESHI); dietrule = dietary rules (EDE); dissee = discomfort with seeing body (EDE); dissha = dissatisfaction with shape (EDE); diswei = dissatisfaction with weight (EDE); EH =

evening hyperphagia (food record); empty = empty stomach (EDE); ESS = total sleep score on the ESS; fearloc = fear of losing control over eating (EDE); fearwg = fear of weight gain (EDE); feelfat = feeling fat (EDE); foodavd = food avoidance (EDE); guilt = guilt about eating (EDE); hunger = morning anorexia (food record); impshape = importance of shape (EDE); impweigh = importance of weight (EDE); losewt = desire to lose weight (EDE); MAE = total circadian rhythm score on the MAE; moodlow = mood worse in the evening (NESHI); needeat = must eat to sleep (NESHI); nocturnal = nocturnal eating (food record); presw = reaction to weighing (EDE); proccup = preoccupation with food (EDE); PSQI = total sleep score on the PSQI; PSS-10 = total stress score on the PSS-10; Restrained = restraint over eating (EDE); secret = eating in secret (EDE); shap/weig = preoccupation with shape and weight (EDE); soceat = social eating (EDE); trosleep = insomnia (NESHI).

**Hypothesis 2:** It was hypothesized that the overall network structure would be dissimilar between populations of persons with and without overweight/obesity.

*Network comparison test.* Due to the instability of the network of persons with overweight/obesity in aim two, it was unlikely that the addition of extra estimated parameters from the EDE would lead to a more stable network, and this was confirmed when I examined CS coefficients for this network of persons without overweight/obesity (strength = 0.04). Thus, I chose not to test any potential invariance between networks.

**Specific Aim Four Summary.** Results from the network analysis generally did not support the hypothesis that overvaluation of shape and weight would present as the most central nodes within psychopathology networks of NES, compared to other symptoms, in both persons with and without overweight/obesity. While results were mixed regarding which symptoms were highly central to the network of persons with overweight/obesity,

neither overvaluation of shape nor weight were found to be central symptoms. Also, the network of persons without overweight/obesity had very poor stability among the three centrality indices and thus results should not be interpreted. In addition, the hypothesis that the overall network structure would be dissimilar between populations of persons with and without overweight/obesity was unable to be tested due to the lack of adequate stability among the centrality indices.

## CHAPTER 5

### DISCUSSION

This study is the first to examine and characterize the core symptoms in a psychopathology network for NES, as well as to do so among populations of persons with and without overweight/obesity and between an NES sample and a non-NES control group. I estimated psychopathology networks of NES with all participants combined to establish the network first, and then among each of the above listed subgroups to assess for differences in the most central symptoms between each respective network. I also assessed the validity of the transdiagnostic cognitive-behavioral model of eating disorders within an NES network by examining if overvaluation of shape and weight would be most central when added to the model, as emerging literature of eating disorders and network analysis demonstrated these constructs as central regardless of diagnosis. According to this literature, if NES were to fit into the transdiagnostic model of EDs, then this sample should exhibit a similar pattern. Otherwise, NES may be a conceptually different kind of feeding and eating disorder (i.e., one without shape/weight concerns).

Overall, results from this study did not support the hypothesis that nocturnal ingestion of food and evening hyperphagia symptoms would present as the most central nodes within the psychopathology network of NES compared to other symptoms among all participants combined, nor among any of the examined subgroups, although the interpretation of some of the smaller subgroups (e.g., persons without overweight/obesity; those in the non-NES control group) was limited due to poor stability of their respective centrality indices. In the psychopathology network of NES that included all participants, as well as the sample that included those persons with overweight/obesity, urge to eat during the night was found to be

highly central to these networks and significantly more central than other NES symptoms, including symptoms of mood, stress, sleep, and circadian rhythm. In the psychopathology network of NES that included only participants who were diagnosed with NES, both depressed mood and urge to eat during the night were found to be highly central to this network and significantly more central than most other NES symptoms, including symptoms of stress, sleep, and circadian rhythm. Network structure and density comparisons between subgroups (e.g., differences in networks between persons with and without overweight/obesity, or between those in the NES group and those in the non-NES control group) were explored, but due to the limitations of the small sample sizes and poor stability of centrality indices among the smaller subgroups, it would be improper to interpret the results of these comparisons.

These results do not support the current outline of symptoms proposed by the research diagnostic criteria (Allison, Lundgren, O'Reardon, et al., 2010). Urge to eat during the night and depressed mood, which are part of criterion C in the research diagnostic criteria, appear to be the most central symptoms of NES, rather than evening hyperphagia or nocturnal ingestion of food. However, it does appear that in all of the psychopathology network models established in this study, nocturnal ingestion of food is associated with urge to eat during the night, suggesting that nocturnal ingestion of food likely arises as a result of this urge to eat during the night. Indeed, under the research diagnostic criteria, it is possible for an individual to meet criteria for NES having experienced nocturnal ingestions of food, without evening hyperphagia, and results from this study may elucidate the specific symptom interaction that leads to nocturnal ingestion of food (it is important to note, however, that these network models are non-directional, and thus while these most central symptoms can

be seen as the driving or maintaining force in the network, this does not provide the necessary criteria to establish temporal causality between symptoms without further investigation; Cook, Campbell, & Shadish, 2002; Hume 1748). In addition, all of the psychopathology network models in this study suggest that depressed mood is associated with sleep disturbances (e.g., insomnia, sleep quality) and other non-NES-specific symptoms (e.g., stress), with minimal direct association with other NES symptoms outside of urge to eat between dinner and sleep.

With regard to the transdiagnostic cognitive-behavioral model of eating disorders within an NES network, results of this study did not support the hypothesis that overvaluation of shape and weight would be most central when added to the model. Indeed, results were mixed and indicated several potential symptoms that were most central to this network, mostly coming from the restraint and eating concern factors on the EDE (i.e., urge to eat during the night, guilt over eating, preoccupation with food, eating, and calories, depressed mood, importance of weight, having an empty stomach, reaction to prescribed weighing). This finding is in contrast with the existing ED network analysis literature, although Forrest and colleagues (2018) stated that other symptoms beyond overvaluation of shape and weight were highly central, though those that were reported appeared to come from the shape and weight concern factors. While it is possible that this could be due to the use of the EDE to assess overvaluation of shape and weight, as other studies have used other measures such as the EDE-Q (DuBois et al., 2017; Forrest et al., 2018; Smith, Mason, et al., 2018; Vanzhula et al., 2018), the EDI/EDI-2 (Olatunji et al., 2018; Solmi et al., 2018), or the EPSI (DuBois et al., 2017; Forbush et al., 2016), these other studies have consistently found that overvaluation of shape and weight are central symptoms. In addition, at least two other

studies (Goldschmidt et al., 2018; Levinson et al., 2017) have also used the EDE and found results consistent with the rest of the stated literature. It is also possible, however, that this finding represents the unique ways that weight and shape concerns present in NES, as importance of weight, empty stomach, restraint are all functionally and conceptually related to weight and shape concerns, even if they load on different factors of the EDE.

Furthermore, these results regarding the inconsistency with the rest of the ED network analysis literature that overvaluation of shape and weight should present as central symptoms might suggest that NES is unique among other EDs (i.e., functionally different than anorexia nervosa, bulimia nervosa, and potentially binge eating disorder) or that it might be inappropriate to categorize NES alongside other EDs with a strong weight/shape control function (i.e., classified alongside pica or avoidant restrictive food intake disorder [ARFID] rather than as an OSFED or EDNOS). However, it is also likely that these results are due to the unique nature of the present sample, as the centrality stability was the lowest when estimating this network compared with those found in the rest of the study. Future research with a larger and more diverse sample of individuals with NES with replicated findings would be necessary to sufficiently support the claim that NES is truly distinct from other EDs.

Also, it is possible that the most central symptoms would have been made clearer had the sample been comprised entirely of those with NES rather than a mix of those with and without NES. While emergent research has shown that the network structures between both clinical and non-clinical samples are the same, clinical samples have greater network connectivity and density and thus stronger symptom severity (Heeran & McNally, 2018; Vanzhula et al., 2018). This also explains why EDs are maintained in certain individuals, as



the central symptoms are *more important* than other symptoms for these individuals compared to others. As such, using a clinical sample, the most central symptoms would have likely been significantly different from more of the remaining symptoms compared to the presentation of differences among symptoms in a non-clinical sample. Indeed, it remains possible that overvaluation of shape and weight are central symptoms for NES, but the inclusion of non-NES controls made other connections appear stronger and thus minimized the overall association of these symptoms on others.

### **Clinical Implications**

This study demonstrated how to use network analysis to characterize the core symptoms of NES, and tried to compare networks of populations of persons with and without overweight/obesity and those between an NES sample and a non-NES control group. Clinically, this study took the first steps toward furthering our understanding of the causal chain of symptoms in NES. To be sure, further research is needed to determine the temporal precedence of these symptoms and their development. Even so, this study identified depressed mood and urge to eat during the night as key elements of the core psychopathology of NES that might be considered primary treatment targets for intervention. Indeed, interventions on these symptoms may disrupt the connections among other symptoms down the chain that maintain NES. In fact, prior research has found that changes to the most central symptoms seen in psychopathology networks has been strongly associated with changes in overall symptom severity compared to changes in less central symptoms (Robinaugh, Millner, & McNally, 2016). In addition, though this study was unable to make any confident determination in the differences in network structure or density between subgroups (e.g., overweight/obesity classification, clinical versus non-clinical samples) due to limited sample

size/poor stability of these networks, it has laid the foundation for future studies to assess such network comparisons by identifying a need for larger sample sizes.

### **Limitations**

There were several limitations of note in this study. First, while the sample size for this study appeared sufficient to confidently conduct network analysis and contributed to adequate stability of centrality indices, select subgroups (e.g., persons without overweight/obesity, individuals in the non-NES control group) had small sample sizes that prevented some network models from being interpreted due to poor stability of centrality indices or prevented a statistical comparison of the network structure and density of models that included just these individuals. Second, this sample was predominantly female, Caucasian, and classified with overweight/obesity, and it is unclear whether these results would generalize to those outside of these demographic groupings. Third, participants kept food records as part of the protocol, which may have had an impact on their food intake. Prior research has indicated that eating behavior can be altered via self-monitoring due to changes in an individual's awareness of their own eating behavior (Waden, Crerand, & Brock, 2005). Such self-monitoring is also strongly correlated with weight loss and weight maintenance (Burke, Wang, & Sevvick, 2011; Butryn, Phelan, Hill, & Wing, 2007). Indeed, participants may have changed their eating behaviors as a result of self-monitoring. Fourth, missing data were replaced using multiple imputed datasets and comparing the similarity of results across a random selection of three of five imputed datasets, which, while consistent with other studies using network analysis, is not considered full multiple imputation (e.g., imputing multiple datasets and combining results obtained from each dataset; Rubin, 1996). Fifth, the confidence intervals of node strength in each of the networks, which was used to

conduct the centrality difference test of strength between symptoms and was determined by drawing a large number of bootstrap samples of the data, may suffer from narrowness bias due to small sample size (Hesterberg, 2015). However, this is likely the most impactful on the subgroups with particularly small sample size (e.g., persons without overweight/obesity, those in the non-NES control group), of which the centrality difference test on node strength was not conducted. Lastly, these data are cross-sectional in nature, and thus results from this study represent psychopathology networks of core symptoms of NES for individuals at only one time point. Longitudinal network analyses are necessary to assess if these results generalize across time and within persons.

### **Strengths**

While this study demonstrated several limitations, it was certainly not without strengths. First, this study represents the first use of network analysis to date on a sample of individuals with NES. As stated previously, literature on the use of network analysis in EDs have not specifically examined this theory with relation to NES, and only one study specifically noted that they had two individuals with NES among their participants (DuBois et al., 2017). All other studies using network analysis to examine EDs only reported OSFED or EDNOS, of which these those meeting these criteria only made up approximately one-fifth of all participants in this literature.

Second, this study included a number of symptoms that are related to, but not specific to, NES (e.g., mood, sleep, circadian rhythm, and stress). Much of the emergent literature on the network approach to psychopathology in EDs include items that are directly related to the specific disorders under examination, and several of these studies have stated that the results of their networks can only reveal information about items included; these networks cannot

reveal anything about items not included. As such, numerous studies have requested that researchers begin to include items that span multiple forms of psychopathology that may present as important risk or maintenance factors (Forrest et al., 2018; Levinson et al., 2017; Olatunji et al., 2018; Smith, Mason, et al., 2018). The addition of items assessing these extra symptoms was especially important given the relationship between NES and mood (Allison et al., 2005; Geliebter et al., 2016), sleep quality (Allison, Engel, et al., 2008), circadian rhythm (Root et al., 2010; Lundgren, Allison, & Stunkard, 2006; Rasmussen et al., 2007), and stress (Allison et al., 2004; Takeda et al., 2004; Vander Wal, 2012).

Third, this study was comprised of a community-recruited sample of participants, rather than from individuals seeking ED treatment. Extant research has demonstrated that individuals who do not seek treatment for EDs often have differing symptom presentations that that of those who are treatment-seeking (e.g., those who are not treatment-seeking appear as having “counter-stereotypic” presentations, or presentations that do not fit the stereotype of one who is suffering from an ED, such as being underweight or purging after a binge; Forrest, Smith, & Swanson, 2017). The vast majority of the literature regarding the network approach to psychopathology in EDs have sampled from treatment-seeking populations, with only one other study using a community-recruited sample (Forbush et al., 2016).

Lastly, the present study included a control group to compare the network structure and overall network density between a clinical and non-clinical sample. To date, only one other study in this literature has included a control group for similar reasons (Vanzhula et al., 2018). In addition, this study is also the first to compare network structure and density between a sample of persons with and without overweight/obesity.

## Future Research

Although this study achieved the aim of characterizing the core symptoms in a psychopathology network for NES, further research is needed to investigate this psychopathology network in more depth. Indeed, one of the challenges in this study was the relatively small sample sizes among several subgroups (e.g., persons without overweight/obesity, those in the non-NES control group), and while it is certainly recommended that future studies include larger sample sizes, it might also be of great benefit to examine group differences with a somewhat different network analysis technique. Recently, Costantini and colleagues (2019) outlined a new method for estimating and analyzing psychopathology networks, particularly when trying to estimate networks in different groups (e.g., clinical versus non-clinical samples, males versus females, etc.), called the *Fused Graphical Lasso* (FGL) method. These researchers state that estimating a single network that includes all groups will fail to illustrate inter-group differences, but also that estimating a separate network for each group would fail to capture inter-group similarities. In addition, they report that differences observed are likely to arise as much from sampling fluctuations as from true differences. This FGL method more accurately estimates different networks for each group by including group similarities as they occur among the different groups. So far, this technique has not been applied to the ED network analysis literature, although it has been used in at least one other psychopathology network study (Fried, 2017).

Future studies should also include some of the newer methods of network analysis described above. For example, a longitudinal analysis of individuals with NES using temporal group-level models (mlVAR) can help to establish the causal relationship among symptoms within the psychopathology network of NES by exploring within-subject variance

and change across time. This can be useful to determine which symptoms maintain disorders at an individual level. In addition, studies that include treatment-seeking samples of individuals with NES can also explore pre-post treatment differences to determine specific differences in the networks of those who respond to treatment versus non-responders, or even to predict treatment outcomes (Olatunji et al., 2018).

Finally, the current study attempted to explore differences between persons with and without overweight/obesity. These classifications were based on a BMI cutoff of 25 kg/m<sup>2</sup>. However, it is possible that there exist differences in either network structure or density between these two groups, but that this BMI value does not represent the appropriate cutoff that differentiates such network differences. Future studies can explore this by identifying where networks differ on a BMI continuum through the use of model-based recursive partitioning. This technique uses decision trees to detect optimal splits on selected variables by assessing parameter instability, and is seen as similar to a moderation analysis (e.g., examining how parameters differ based on subgroups; Jones, Simon, Zeileis, & Mair, 2018). This technique could more accurately explore exactly where network differences are on a continuum of BMI.

## **Conclusion**

In sum, this study represents an important step in advancing the literature on both NES and on network analysis on EDs. Until now, the current conceptualization of NES, and interventions based on that conceptualization, have been based on a latent variable, medical model nosology and theory of etiology. This is the first study to examine and characterize the core symptoms of NES using the network approach to psychopathology, and used both populations of persons with and without overweight/obesity as well as those classified with

NES and those in a non-NES control group. Depressed mood and urge to eat during the night were found to be key elements of the core psychopathology of NES, rather than nocturnal ingestions of food and evening hyperphagia, and might represent primary treatment targets for intervention. Differences between subgroups were unable to be confidently interpreted, and further research is needed to assess for the differences in the relationship among symptoms and symptom severity between these subgroups.

Table 2.

*Research diagnostic criteria and associated items for network analysis*

Criterion	Dependent variable	Measure
A.1 Evening Hyperphagia	% Total daily calories after evening meal, based on seven-day average	Food record
A.2 Nocturnal Eating	Total number of nocturnal ingestions over seven days	Food record
B. Awareness and Recall	Level of awareness (0-4 scale)	NESHI/NEQ
C.1 Morning Anorexia	Average 10:00am hunger rating based on seven-day average	Food record
	How hungry are you in the morning/when do you usually eat (0-4)	NESHI/NEQ
C.1 Breakfast Omitted $\geq 4$ x/week	How often one eats breakfast (0-6)	EDE
C.2 Urge to Eat Between Dinner and Sleep	Craving after supper score (0-4)	NESHI/NEQ
C.2 Urge to Eat During the Night	Craving during night score (0-4)	NESHI/NEQ
C.3 Insomnia	Trouble sleeping score (0-4)	NESHI/NEQ
	Total number of awakenings over seven days	Sleep record
C.4 Must Eat to Sleep	Need to eat score (0-4)	NESHI/NEQ
C.5 Mood Frequently Depressed	Current mood score (0-4)	NESHI/NEQ
C.5 Mood Worse in the Evening	Mood score during evening (0-4)	NESHI/NEQ



Table 3.

*Associated symptom domain and associated items for network analysis*

Domain	Dependent Variable	Measure
Mood	Total depression score (0-63)	BDI
Sleep	Total Composite Score (0-21)	PSQI
	Total score (0-24)	ESS
Circadian Rhythm	Total Score	MEQ
Stress	Total Stress Score (0-40)	PSS-10

Table 4.

*Eating Disorder Examination (EDE) subscales and associated items for network analysis*

Subscale	Item
Restraint	Restraint over eating
	Avoidance of eating
	Food avoidance
	Dietary rules
	Empty stomach
Eating Concern	Preoccupation with food, eating, or calories
	Fear of losing control over eating
	Eating in secret
	Social eating
	Guilt about eating
Shape Concern	Flat stomach
	Preoccupation with shape or weight
	Importance of shape
	Fear of weight gain
	Dissatisfaction with shape
	Discomfort seeing body
	Avoidance of exposure
	Feelings of fatness
Weight Concern	Importance of weight
	Reaction to prescribed weighing
	Preoccupation with shape or weight
	Dissatisfaction with weight
	Desire to lose weight

## APPENDIX A

### Food Record

Date: \_\_\_\_\_

Wake time: \_\_\_\_\_

Bed time: \_\_\_\_\_

Time	Food or Beverage	Amount	Check if NE episode

Observations/Notes:

## APPENDIX B

### NIGHT EATING SYNDROME HISTORY AND INVENTORY

**Instructions:** The NESHI is a comprehensive semi-structured clinical interview developed by Allison and colleagues to assess the degree of and severity of night eating syndrome (NES) symptoms and to establish a diagnosis of NES based on research diagnostic criteria (see Allison, K.C., Lundgren, J.D., O'Reardon, J.P., Geliebter, A., Gluck, M., Vanai, P., et al. (2010). Proposed Diagnostic Criteria for Night Eating Syndrome. *International Journal of Eating Disorders*, 43, 2414-247).

The Night Eating Questionnaire (NEQ; Allison, Lundgren, et al., 2008) is embedded in the NESHI (numbered items) and instructions for calculating the NEQ score are included in the interview. In addition, the NESHI includes a checklist to determine the patient's NEQ diagnosis (none, sub-threshold, full threshold).

Patient Name: \_\_\_\_\_ Date of Interview: \_\_\_\_\_  
Male \_\_\_\_\_ Female \_\_\_\_\_ Ethnicity \_\_\_\_\_ Age \_\_\_\_\_  
Height \_\_\_\_\_ Current Weight \_\_\_\_\_ Current BMI \_\_\_\_\_

#### Beginning of Interview:

**“Please answer the following questions according to your behavior during the past 4 weeks (28 days).”**

- What time do you typically get up each day?

**1. How hungry are you usually in the morning?**

0            1            2            3            4  
Not at all    A little    Somewhat    Moderately    Very

**2. When do you usually eat for the first time?**

0            1            2            3            4  
Before 9am   9:01 to 12pm   12:01 to 3pm   3:01 to 6pm   6:01 or later

**Estimate with the patient how many days per week he/she has experienced a lack of desire to eat in the morning or has omitted breakfast: \_\_\_\_\_**

- (If breakfast is eaten...) What do you typically eat for breakfast?
- Do you snack between breakfast and lunch? If yes, how much?
- When do you eat lunch? What do you typically eat for lunch?
- Do you snack between lunch and your evening meal? If yes, how much?

- When do you eat your dinner/evening meal? What do you typically eat?
- Do you snack after dinner, but before you go to bed? If yes, what do you typically eat? How much of that do you eat?

**3. Do you have cravings or urges to eat snacks after supper, but before bedtime?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Not at all	A little	Somewhat	Very much so	Extremely so

**4. How much control do you have over your eating between supper and bedtime?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
None at all	A little	Some	Very much	Complete

- When do you usually go to bed?
- Do you have any problems falling asleep? How long does it usually take (as long as 30 minutes)?

**5. How often do you have trouble getting to sleep?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Never	Sometimes	About half the time	Usually	Always

**6. How often do you get up at least once in the middle of the night?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Never	Less than once a week	About once a week	More than once a week	Every night

**Estimate with the patient how many nights per week he/she has difficulty falling asleep or maintaining sleep: \_\_\_\_\_**

**\*\*\*\*\* IF ANSWERED NEVER, SKIP DOWN TO CALCULATE PERCENTAGE OF FOOD EATEN AFTER THE EVENING MEAL AND COMPLETE REMAINING INTERVIEW QUESTIONS \*\*\*\*\***

**7. Do you have cravings or urges to eat snacks when you wake up at night?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Not at all	A little	Somewhat	Very much so	Extremely so

**8. Do you need to eat in order to get back to sleep when you awake at night?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Not at all	A little	Somewhat	Very much so	Extremely so

**9. When you get up in the middle of the night, how often do you snack?**

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
----------	----------	----------	----------	----------

Never      Sometimes      About half the time      Usually      Always

- If > “never”, how many episodes of nocturnal eating do you experience per week?
- What and how much do you typically eat when you are snacking during the night?

- When you snack in the middle of the night, how aware are you of your eating?  
(Circle one – if “Not at all” or “A little” – may be Nocturnal Sleep Related Eating Disorder)

Not at all      A little      Somewhat      Very much so      Completely

**10. How much control do you have over your eating while you are up at night?**

0      1      2      3      4  
None at all      A little      Some      Very much      Complete

**BASED ON THE AMOUNT EATEN DURING MEALS AND SNACKS DURING THE DAY, EVENING, AND NIGHT, ESTIMATE WITH THE PATIENT WHAT PROPORTION OF HIS/HER TOTAL DAILY INTAKE HE/SHE CONSUMES AFTER THE EVENING MEAL (THIS INCLUDES SNACKS BEFORE BEDTIME AND NOCTURNAL INGESTIONS) : \_\_\_\_\_%**

**11. How much of your daily food intake do you consume after suppertime?**

0      1      2      3      4  
0%      1-25%      26-50%      51-75%      76-100%

**12. Are you currently feeling blue or down in the dumps?**

0      1      2      3      4  
Not at all      A little      Somewhat      Very much so      Extremely

**13. When you are feeling blue, is your mood lower in the (circle one):**

0      1      2      3      4      0  
Early Morning      Late Morning      Afternoon      Early Evening      Late Evening/ Nighttime      Mood does not change during the day

**If the core features of NES (eating  $\geq$  25% of intake after evening meal and/or nocturnal ingestions  $\geq$  2 times per week) are present, rate the following:**

**Distress/Impairment in Functioning:**

A. How upsetting is your night eating to you?

0      1      2      3      4  
Not at all      A little      Somewhat      Very much so      Extremely

B. How much has your night eating affected your life?

<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>
Not at all	A little	Somewhat	Very much so	Extremely

**Assessment of Compensatory Behaviors:**

A. Have you ever done or used the following during a time when (or shortly after) you were night eating?

- |   |     |    |
|---|-----|----|
| a) Make yourself vomit:                           | Yes | No |
| b) Laxatives:                                     | Yes | No |
| c) Diuretics (water pills):                       | Yes | No |
| d) Diet pills (over the counter or prescription): | Yes | No |
| e) Exercise more than 2 hours per day:            | Yes | No |
| f) Fast or not eat (for 24 hours or more):        | Yes | No |
| g) Other methods – please indicate below:         | Yes | No |

-

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B. If yes to any purgative method, how often? \_\_\_\_\_

**Background:**

A. At what age did you begin night eating? \_\_\_\_\_

B. How much did you weigh when you began night eating (to assess if normal weight or overweight at onset)? \_\_\_\_\_

C. How often did you diet before your night eating began?

- a. never
- b. once every couple of years
- c. once or twice a year
- d. once every 3 months or more

D. Before you began night eating, did you (circle all that apply):

- a. stay up late at night regularly
- b. work a night shift
- c. endure a stressful event; if yes, identify \_\_\_\_\_
- d. experience a medical condition which would account for night eating behavior
- e. begin medication/substance which would account for night eating behavior

E. Does anyone else in your family have symptoms of night eating?

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F. What strategies, medications, or supplements have you tried to stop night eating, and were they successful (for example, sleeping pills, melatonin, SSRIs)?

- 1. \_\_\_\_\_
- 2. \_\_\_\_\_
- 3. \_\_\_\_\_
- 4. \_\_\_\_\_



**NES Diagnosis (Check criteria based on NESHI interview and other corroborating evidence [e.g., food record]):**

- \_\_\_\_\_ I. The daily pattern of eating demonstrates a significantly increased intake in the evening and/or nighttime, as manifested by *one or both* of the following:
- \_\_\_\_\_ A. At least 25% of food intake is consumed after the evening meal
  - \_\_\_\_\_ B. At least two episodes of nocturnal eating per week
- \_\_\_\_\_ II. Awareness and recall of evening and nocturnal eating episodes are present.
- \_\_\_\_\_ III. The clinical picture is characterized by *at least three* of the following features:
- \_\_\_\_\_ A. Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week
  - \_\_\_\_\_ B. Presence of a strong urge to eat between dinner and sleep onset and/or during the night
  - \_\_\_\_\_ C. Sleep onset and/or sleep maintenance insomnia are present four or more nights per week
  - \_\_\_\_\_ D. Presence of a belief that one must eat in order to initiate or return to sleep
  - \_\_\_\_\_ E.. Mood is frequently depressed and/or mood worsens in the evening
- \_\_\_\_\_ IV. The disorder is associated with significant distress and/or impairment in functioning.
- \_\_\_\_\_ V. The disordered pattern of eating has been maintained for at least 3 months.
- \_\_\_\_\_ VI.. The disorder is not secondary to substance abuse or dependence, medical disorder, medication, or another psychiatric disorder.

*Directions for scoring the NEQ:* 1) reverse score items 1, 4, and 10. 2) Sum all numbered items.

NEQ Score After Interview: \_\_\_\_\_

NES Diagnosis (circle): None              Sub-threshold              Full Threshold

## APPENDIX C

### BECK DEPRESSION INVENTORY-II

Name: \_\_\_\_\_ Marital Status: \_\_\_\_\_ Age: \_\_\_\_\_ Sex: \_\_\_\_\_

Occupation: \_\_\_\_\_ Education: \_\_\_\_\_

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

<p><b>1. Sadness</b></p> <p>0 I do not feel sad.</p> <p>1 I feel sad much of the time.</p> <p>2 I am sad all the time.</p> <p>3 I am so sad or unhappy that I can't stand it.</p> <p><b>2. Pessimism</b></p> <p>0 I am not discouraged about my future.</p> <p>1 I feel more discouraged about my future than I used to be.</p> <p>2 I do not expect things to work out for me.</p> <p>3 I feel my future is hopeless and will only get worse.</p> <p><b>3. Past Failure</b></p> <p>0 I do not feel like a failure.</p> <p>1 I have failed more than I should have.</p> <p>2 As I look back I see a lot of failures.</p> <p>3 I feel I am a total failure as a person.</p> <p><b>4. Loss of Pleasure</b></p> <p>0 I get as much pleasure as I ever did from the things I enjoy.</p> <p>1 I don't enjoy things as much as I used to.</p> <p>2 I get very little pleasure from the things I used to enjoy.</p> <p>3 I can't get any pleasure from the things I used to enjoy.</p> <p><b>5. Guilty Feelings</b></p> <p>0 I don't feel particularly guilty.</p> <p>1 I feel guilty over many things I have done or should have done.</p> <p>2 I feel quite guilty most of the time.</p> <p>3 I feel guilty all the time.</p>	<p><b>6. Punishment Feelings</b></p> <p>0 I don't feel I am being punished.</p> <p>1 I feel I may be punished.</p> <p>2 I expect to be punished.</p> <p>3 I feel I am being punished.</p> <p><b>7. Self-Dislike</b></p> <p>0 I feel the same about myself as ever.</p> <p>1 I have lost confidence in myself.</p> <p>2 I am disappointed in myself.</p> <p>3 I dislike myself.</p> <p><b>8. Self-Criticalness</b></p> <p>0 I don't criticize or blame myself more than usual.</p> <p>1 I am more critical of myself than I used to be.</p> <p>2 I criticize myself for all of my faults.</p> <p>3 I blame myself for everything bad that happens.</p> <p><b>9. Suicidal Thoughts or Wishes</b></p> <p>0 I don't have any thoughts of killing myself.</p> <p>1 I have thoughts of killing myself, but I would not carry them out.</p> <p>2 I would like to kill myself.</p> <p>3 I would kill myself if I had the chance.</p> <p><b>10. Crying</b></p> <p>0 I don't cry anymore than I used to.</p> <p>1 I cry more than I used to.</p> <p>2 I cry over every little thing.</p> <p>3 I feel like crying, but I can't.</p>
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<p><b>11. Agitation</b></p> <p>0 I am no more restless or wound up than usual.</p> <p>1 I feel more restless or wound up than usual.</p> <p>2 I am so restless or agitated that it's hard to stay still.</p> <p>3 I am so restless or agitated that I have to keep moving or doing something.</p> <p><b>12. Loss of Interest</b></p> <p>0 I have not lost interest in other people or activities.</p> <p>1 I am less interested in other people or things than before.</p> <p>2 I have lost most of my interest in other people or things.</p> <p>3 It's hard to get interested in anything.</p> <p><b>13. Indecisiveness</b></p> <p>0 I make decisions about as well as ever.</p> <p>1 I find it more difficult to make decisions than usual</p> <p>2 I have much greater difficulty in making decisions than I used to.</p> <p>3 I have trouble making any decisions.</p> <p><b>14. Worthlessness</b></p> <p>0 I do not feel I am worthless.</p> <p>1 I don't consider myself as worthwhile and useful as I used to.</p> <p>I feel more worthless as compared to other people.</p> <p>I feel utterly worthless.</p>	<p><b>15. Loss of Energy</b></p> <p>0 I have as much energy as ever.</p> <p>1 I have less energy than I used to have.</p> <p>2 I don't have enough energy to do very much.</p> <p>3 I don't have enough energy to do anything.</p> <p><b>16. Changes in Sleeping Pattern</b></p> <p>0 I have not experienced any change in my sleeping patterns.</p> <p>-----</p> <p>--</p> <p>1a I sleep somewhat more than usual.</p> <p>1b I sleep somewhat less than usual.</p> <p>-----</p> <p>--</p> <p>2a I sleep a lot more than usual.</p> <p>2b I sleep a lot less than usual.</p> <p>-----</p> <p>--</p> <p>3a I sleep most of the day.</p> <p>3b I wake up 1-2 hours early and can't get back to sleep.</p> <p>-----</p> <p>-</p> <p><b>17. Irritability</b></p> <p>0 I am no more irritable than usual.</p> <p>1 I am more irritable than usual.</p> <p>2 I am much more irritable than usual.</p> <p>3 I am irritable all the time.</p> <p><b>18. Changes in Appetite</b></p> <p>0 I have not experienced any change in my appetite.</p> <p>-----</p> <p>--</p> <p>1a My appetite is somewhat less than usual.</p> <p>1b My appetite is somewhat greater than usual.</p> <p>-----</p> <p>--</p> <p>2a My appetite is much less than before.</p> <p>2b My appetite is much greater than usual.</p> <p>-----</p> <p>--</p> <p>3a I have no appetite at all.</p> <p>3b I crave food all of the time.</p>
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<p><b>19. Concentration Difficulty</b></p> <ul style="list-style-type: none"> <li>0 I can concentrate as well as ever.</li> <li>1 I can't concentrate as well as usual.</li> <li>2 It's hard to keep my mind on anything for very long.</li> <li>3 I find I can't concentrate on anything.</li> </ul> <p><b>20. Tiredness or Fatigue</b></p> <ul style="list-style-type: none"> <li>0 I am no more tired or fatigued than usual.</li> <li>1 I get more tired or fatigued more easily than usual.</li> <li>2 I am too tired or fatigued to do a lot of the things I used to do.</li> <li>3 I am too tired or fatigued to do most of the things I used to do.</li> </ul> <p><b>21. Loss of Interest in Sex</b></p> <ul style="list-style-type: none"> <li>0 I have not noticed any recent change in my interest in sex.</li> <li>1 I am less interested in sex than I used to be.</li> <li>2 I am much less interested in sex now.</li> <li>3 I have lost interest in sex completely.</li> </ul>	
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## APPENDIX D

### MORNINGNESS-EVENINGNESS QUESTIONNAIRE

#### *Morningness and Eveningness*

**DIRECTIONS:** Please *check* the response for *each* item that best describes you.

1. Considering only your own "feeling best" rhythm, at what time would you get up if you were entirely free to plan your day?

5:00-6:30 AM	_____
6:30-7:45 AM	_____
7:45-9:45 AM	_____
9:45-11:00 AM	_____
11:00 AM-12:00 (noon)	_____

2. Considering your own "feeling best" rhythm, at what time would you go to bed if you were entirely free to plan your evening?

8:00-9:00 PM	_____
9:00-10:15 PM	_____
10:15-12:30 AM	_____
12:30-1:45 AM	_____
1:45-3:00 AM	_____

3. Assuming normal circumstance, how easy do you find getting up in the morning?

(Check one.)

Not at all easy	_____
Slightly easy	_____
Fairly easy	_____
Very easy	_____

4. How alert do you feel during the first half hour after having awakened in the morning?

(Check one.)

Not at all alert	_____
Slightly alert	_____
Fairly alert	_____
Very alert	_____

5. During the first half hour after having awakened in the morning, how tired do you feel?

(Check one.)

Very tired	_____
Fairly tired	_____
Fairly refreshed	_____
Very refreshed	_____

*Morningness and Eveningness*

6. You have decided to engage in some physical exercise. A friend suggests that you do this one hour twice a week and the best time for him is 7:00-8:00 AM. Bearing in mind nothing else but your own "feeling best" rhythm, how do you think you would perform?

- Would be in good form \_\_\_\_\_
- Would be in reasonable form \_\_\_\_\_
- Would find it difficult \_\_\_\_\_
- Would find it very difficult \_\_\_\_\_

7. At what time in the evening do you feel tired and, as a result, in need of sleep?

- 8:00-9:00 PM \_\_\_\_\_
- 9:00-10:15 PM \_\_\_\_\_
- 10:15 PM-12:30 AM \_\_\_\_\_
- 12:30-1:45 AM \_\_\_\_\_
- 1:45-3:00 AM \_\_\_\_\_

8. You wish to be at your peak performance for a test, which you know is going to be mentally exhausting and lasting for two hours. You are entirely free to plan your day, and considering only your own "feeling best" rhythm, which ONE of the four testing times would you choose?

- 8:00-10:00 AM \_\_\_\_\_
- 11:00 AM-1:00 PM \_\_\_\_\_
- 3:00-5:00 PM \_\_\_\_\_
- 7:00-9:00 PM \_\_\_\_\_

9. One hears about "morning" and "evening" types of people. Which ONE of these types do you consider yourself to be?

- Definitely a morning type \_\_\_\_\_
- More a morning than an evening type \_\_\_\_\_
- More an evening than a morning type \_\_\_\_\_
- Definitely an evening type \_\_\_\_\_

10. When would you prefer to rise (provided you have a full day's work- 8 hours) if you were totally free to arrange your time?

- Before 6:30 AM \_\_\_\_\_
- 6:30-7:30 AM \_\_\_\_\_
- 7:30-8:30 AM \_\_\_\_\_
- 8:30 AM or later \_\_\_\_\_

*Morningness and Eveningness*

11. If you always had to rise at 6.00 AM, what do you think it would be like?

- Very difficult and unpleasant \_\_\_\_\_
- Rather difficult and unpleasant \_\_\_\_\_
- A little unpleasant but no great problem \_\_\_\_\_
- Easy and not unpleasant \_\_\_\_\_

12. How long a time does it usually take before you "recover your senses" in the morning after rising from a night's sleep?

- 0-10 minutes \_\_\_\_\_
- 11-20 minutes \_\_\_\_\_
- 21-40 minutes \_\_\_\_\_
- More than 40 minutes \_\_\_\_\_

13. Please indicate to what extent you are a morning or evening active individual.

- Pronounced morning active (morning alert and evening tired) \_\_\_\_\_
- To some extent, morning active \_\_\_\_\_
- To some extent, evening active \_\_\_\_\_
- Pronounced evening active (morning tired and evening alert) \_\_\_\_\_

## APPENDIX E

### PITTSBURGH SLEEP QUALITY INDEX

#### Instructions:

The following questions relate to your usual sleep habits during the past month ONLY. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?

USUAL BED TIME \_\_\_\_\_

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

NUMBER OF MINUTES \_\_\_\_\_

3. During the past month, when have you usually gotten up in the morning?

USUAL GETTING UP TIME \_\_\_\_\_

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)

HOURS OF SLEEP PER NIGHT \_\_\_\_\_

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you.....

(a) cannot get to sleep within 30 minutes

Not during the	Less than	Once or	Three or more
past month _____	once a week _____	twice a week _____	times a week _____

(b) Wake up in the middle of the night or early morning

Not during the	Less than	Once or	Three or more
past month _____	once a week _____	twice a week _____	times a week _____

(c) Have to get up to use the bathroom.

Not during the	Less than	Once or	Three or more
past month _____	once a week _____	twice a week _____	times a week _____

(d) Cannot breathe comfortably.

Not during the	Less than	Once or	Three or more
past month _____	once a week _____	twice a week _____	times a week _____

(e) Cough or snore loudly.

Not during the	Less than	Once or	Three or more
past month _____	once a week _____	twice a week _____	times a week _____



(f) Feel too cold.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(g) Feel too hot.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(h) Had bad dreams.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(i) Have pain.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(j) Other reason(s), please describe \_\_\_\_\_

---

How often during the past month have you had trouble sleeping because of this?

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

6. During the past month, how would you rate your sleep quality overall?

Very good \_\_\_\_\_

Fairly good \_\_\_\_\_

Fairly bad \_\_\_\_\_

Very bad \_\_\_\_\_

7. During the past month, how often have you taken medicine (Prescribed or "over the counter") to help you sleep?

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all \_\_\_\_\_

Only a very slight problem \_\_\_\_\_

Somewhat of a problem \_\_\_\_\_

A very big problem \_\_\_\_\_

10. Do you have a bed partner or share a room?

No bed partner or do not share a room \_\_\_\_\_

Partner/ flatmate in other room \_\_\_\_\_

Partner in same room, but not same bed \_\_\_\_\_

Partner in same bed \_\_\_\_\_

11. If you have a bed partner or share a room, ask him/her how often in the past month you have had.....

(a) Loud snoring.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(b) Long pauses between breaths while asleep.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(c) Legs twitching or jerking while you sleep.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(d) Episodes of disorientation or confusion during sleep.

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

(e) Other restlessness while you sleep: please describe \_\_\_\_\_

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Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

## APPENDIX F

### EPWORTH SLEEPINESS SCALE

Name:

Date:

Your age: (Yr)

Your sex: Male Female

How likely are you to doze off or fall asleep in the situations described below, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the most appropriate number for each situation:-

0 = would never doze

1 = Slight chance of dozing

2 = Moderate chance of dozing

3 = High chance of dozing

Situation	Chance of dozing
Sitting and reading .....	
Watching TV .....	
Sitting, inactive in a public place (e.g. a theatre or a meeting) .....	
As a passenger in a car for an hour without a break .....	
Lying down to rest in the afternoon when circumstances permit .....	
Sitting and talking to someone .....	
Sitting quietly after a lunch without alcohol .....	
In a car, while stopped for a few minutes in the traffic .....	

Total .....

Score:

0-10 Normal range

10-12 Borderline

12-24 Abnormal

## APPENDIX G

### PERCEIVED STRESS SCALE

The questions in this scale will ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate *how often* you felt or thought a certain way.

Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems to be a reasonable estimate.

1. In the last month, how often have you been upset because of something that happened unexpectedly?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

2. In the last month, how often have you felt unable to control the important things in your life?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

3. In the last month, how often have you felt nervous and 'stressed'?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

4. In the last month, how often have you felt confident about your ability to handle your personal problems

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

5. In the last month, how often have you felt that things were going your way?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

6. In the last month, how often have you felt that you could not cope with all the things that you had to do?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

7. In the last month, how often have you been able to control irritations in your life?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

8. In the last month, how often have you felt that you were on top of things?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

9. In the last month, how often have you been angered because of things that happened that were outside of your control?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

10. In the last month, how often have you felt that difficulties were piling up so high that you could not overcome them?

0	1	2	3	4
never	almost never	sometimes	fairly often	very often

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