SYMPTOM DECOUPLING AS A PROCESS OF CHANGE IN ACCEPTANCE AND MINDFULNESS-BASED THERAPY FOR EATING DISORDERS: PRELIMINARY INSIGHTS FROM A NETWORK PERSPECTIVE

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Russell DuBois

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CHAPTER 1
INTRODUCTION

Background and Context

Despite their relative infrequency in the general population (Swanson, 2011), eating disorders hold the highest incidence of mortality of any psychiatric disorder and can be life-long conditions (Arcelus, Mitchell, Wales, & Nielsen, 2011; Franko et al., 2013; Touyz & Hay, 2015). Although the core psychopathology of an eating disorder is often the overvaluation of weight and shape (Fairburn, Cooper, & Shafran, 2003; Murphy, Straebler, Cooper, & Fairburn, 2010), the symptoms that constitute the disorders are multiple and include a fear of gaining weight, binge eating and purging, and dietary restriction, among others. Contemporary clinical strategies for the treatment of eating disorders have been largely drawn from cognitive psychology and behaviorism in an attempt to combat the maladaptive cognitions and behaviors central to the disorders. Recently, a sub-group of cognitive and behavioral treatments for eating disorders has emerged that utilize traditional behavioral principles (Hayes, 2004; Skinner, 1953) together with acceptance and mindfulness, which are components of psychological and spiritual processes derived from ancient Eastern practices (hereby referred to solely as mindfulness for simplicity). This sub-group of psychotherapies has since been referred to as Acceptance and Mindfulness Based Therapies (AMTs).

AMTs are in a critical developmental period. The origin of AMTs lies in ancient Eastern practices; however, only recently has the inquiry into the effectiveness of these practices been a focus of Western medicine. Such inquiry is framed in the context of the Western medical model, whereby the primary focus of psychological treatment outcome is the reduction of symptomatology, analogous to reductions of headaches as an indicator of successful treatment of
a brain tumor. According to this model, psychopathology is manifested as the existence of unpleasant or otherwise negative internal experiences, and alleviation of these experiences is the cornerstone of health restoration.

In direct opposition to this model are the basic tenets of acceptance and mindfulness, which are held in all AMT-based interventions (albeit to various degrees). AMTs emphasize the normality of negative experiences in everyday life as a human being and postulate that psychopathology is not simply the existence of these experiences per se. Rather, psychopathology arises from the engagement in behaviors aimed to alter, control, or otherwise avoid these experiences. Such behaviors may be effective in the short term, yet inevitably lead to long-term suffering. Thus, the essence of therapeutic growth in AMTs rests in successfully changing one’s relationship to negative experiences through the process of acceptance and mindfulness, and health is restored when an individual is able to make room for all experiences regardless of their perceived valence. Notwithstanding this difference, AMTs do, in fact, lead to a reduction in symptomatology across a number of mental health conditions, including eating disorders (Baer, 2003; Baer, Fischer, & Huss, 2005). Of course, this is indeed a desirable goal in the context of psychotherapy from the perspective of both client and the mental health professional. However, we do not yet know the mechanism for this effect. In other words, we have evidence that AMTs are helpful but we do not yet fully know the how.

A transformational approach to understanding the structure of psychopathology has emerged in recent years. This approach, referred to as the network approach, posits that disorders arise due to complex interactions among their symptoms (Borsboom & Cramer, 2013; McNally, 2016). Diverging drastically from the commonly held latent trait approach whereby symptoms are independent indicators of an underlying disease entity, the network approach
holds symptoms as constituents of a disorder such that no underlying disorder actually exists beyond its symptoms. Although a full description of the network approach is beyond the scope of this chapter, a critically important contribution of the network approach is that it allows for the quantification of a dimension of psychopathology that has not been accessible to date. Specifically, the network approach allows for the measurement of symptom network connectivity, which, in the context of the proposed research project, encompasses a separate dimension of psychopathology above and beyond disorder severity measured by aggregated symptom totals.

Symptom network connectivity refers to the magnitude of functional relationships among symptoms within a certain disorder. In the context of eating disorders, let us consider fear of gaining weight, dietary restriction, and binge eating as three examples of eating disorder symptoms. The network approach posits that these symptoms may be functionally related to one another such that a fear of gaining weight leads to dietary restriction, which then leads to an episode of binge eating. Subsequently, a heightened fear of gaining weight follows this binge eating episode, thus perpetuating the cycle. In this way, the stronger the network connectivity (i.e., the higher the likelihood that one symptom will lead to the next), the harder it would be to break the cycle. Furthermore, even without directly experiencing high levels of symptoms at a given time, a highly connected network would put an individual at risk such that a relatively small experience of one of the symptoms would quickly trigger this cycle. In fact, research has shown that individuals with high symptom network connectivity have higher global psychopathology and have a poorer disorder prognosis when compared to individuals with low symptom network connectivity (Pe et al., 2015; van Borkulo et al., 2015)
Consequentially, symptom connectivity is an ideal candidate for capturing the process of therapeutic change as conceptualized by AMTs. This is because the dimension of psychopathology considered in this case is not how much or little one is experiencing symptoms, but rather the magnitude to which these experiences influence each other across time and in maladaptive ways. According to the medical model, the goal of psychotherapy would be to reduce symptoms, a process which, according to AMTs, is (paradoxically) at the heart of the problem. Conversely, considering eating disorders from a network perspective enables the process of psychotherapy to align with basic tenets of AMTs. Specifically, the process of therapeutic change in AMTs can be conceptualized as decoupling maladaptive behavioral patterns rather than simply reducing the severity of these experiences, and this distinction will be the focus of discussion in subsequent chapters. However, no research to date has investigated the role of symptom connectivity in relation to eating disorders or has framed symptom networks as a suitable model for understanding the process of change underlying AMTs.

**Research Questions**

The current study attempted to answer a number of important questions regarding the role of eating disorder network parameters in general, and network *connectivity* in particular, in eating disorder psychopathology, and the potential process by which AMTs exert their influence on this network of symptoms. Specifically, the proposed study attempted to answer the following questions:

1) Is symptom connectivity an indicator of eating disorder severity?

2) Is symptom connectivity a construct that accounts for the relationship between mindfulness and symptom severity?
Aims and Hypotheses

To answer these research questions, the proposed study had a number of aims and related hypotheses regarding the role of symptom network connectivity in eating disorder psychopathology and its relation to AMT intervention. The first aim was to examine the relationship between symptom network connectivity and global levels of eating disorder symptom severity. It was hypothesized that greater levels of symptom network connectivity would be related to greater levels of eating disorder symptom severity. The second aim was to investigate whether symptom connectivity accounted for the relationship between mindfulness and eating disorder symptom severity. It was hypothesized that symptom network connectivity would mediate the relationship between mindfulness and eating disorder symptom severity. Given that aim 1 and aim 2 of the study rest on within-subject network models of eating disorder psychopathology, an exploratory aim of the study was to investigate the structure of the network. Specifically, the study aimed to investigate symptom feedback loops (i.e., specific patterns of relationships among two to three symptoms that mutually reinforce one another) and symptom centrality (i.e., the relative associative strength of each symptom on all other symptoms), as these constructs have been identified as important components in both psychopathology network theory (Borsboom & Cramer, 2013) and cognitive-behavioral formulations of eating disorders (Fairburn, Cooper, & Shafran, 2003; Murphy, Straebler, Cooper, & Fairburn, 2010).
CHAPTER 2

LITERATURE REVIEW

In the context of eating disorders and sub-clinical disordered eating, contemporary research and clinical practice has identified acceptance and mindfulness based therapies (AMTs) as promising new approaches to treating the cognitive, affective, and behavioral symptoms that constitute the disorders (Baer et al., 2005; Juarascio et al., 2013; Masuda, Price, & Latzman, 2012). The specific treatment protocols that encompass AMTs as a category are numerous; however, they all share core tenets and practices, which act as the bedrock for the specific skills, techniques, and lessons that are unique to each therapy. These basic tenets include (not surprisingly) mindfulness and acceptance.

Recent developments in the field of psychopathology have led to the identification of a transformational model for understanding mental disorders, which is in opposition with the widely held view of mental disorders as latent entities. Specifically, a contemporary approach to conceptualizing mental disorders, referred to as the network approach, has been developed that allows for study of psychopathology using unique and clinically-relevant methods and analyses. Importantly, these methods and analyses are highly congruent with the therapeutic processes inherent in AMTs. However, despite the dramatic rise in AMTs in recent years (Herbert & Forman, 2011b) and the fact that basic mindfulness and acceptance practices have been around for thousands of years, the integration of their core tenets with network models of psychopathology in general, and of eating disorders in particular, is in its youth. As such, the remainder of this chapter is intended to present a comprehensive conceptualization of the mechanism of change in AMTs that integrates the recent network model of eating disorder psychopathology with the clinical theory and practice of mindfulness and acceptance.
Acceptance and Mindfulness Based Therapy in Context

Acceptance and mindfulness-based therapies, hereby referred to as AMTs, represent a coalition of recent developments in mental health treatment that encompasses a wide range of empirically-based treatment programs including Mindfulness-Based Stress Reduction (MBSR; Kabat-Zinn, 2003), Mindfulness-Based Cognitive Therapy (MBCT; Teasdale et al., 2000), Metacognitive Therapy (Wells, 2008), Acceptance and Commitment Therapy (ACT; Hayes et al., 2006), and Dialectical Behavioral Therapy (Linehan, 1987). Often referred to as the third wave of the traditional behavioral and, later, cognitive-behavioral approach to mental health treatment (Hayes, 2004), AMTs integrate practices from many different disciplines including cognitive therapy (Beck, 1976), behavioral theory (Skinner, 1953), metacognitive therapy (Wells, 2008), relational frame theory (Hayes, Barnes-Holmes, & Roche, 2001), and ancient eastern practices (Kabat-Zinn, 1994). Although AMTs represent an eclectic and heterogeneous group of treatments, they share two underlying principles: the teaching and practice of mindfulness and acceptance.

Although definitions of the construct abound, the most commonly cited operational definition of mindfulness comes from Kabat-Zinn (1994), who states that mindfulness means “paying attention in a particular way: on purpose, in the present moment, and non-judgmentally” (Kabat-Zinn, 1994). Others have emphasized similar conceptualizations, including an enhanced awareness of the full range of physiological, emotional, and cognitive experiences, and a curious and open sustained attention to the present moment (Bishop, 2004; Herbert & Cardaciotto, 2005). From a cognitive perspective, mindfulness encompasses a mental state whereby every thought and feeling is processed in a present-centered and nonjudgmental way (Teasdale et al., 2000). In the context of experiences related to disordered eating and body image concerns, being
mindful may manifest as an increased awareness of, and openness toward the experience of, upward appearance comparisons (i.e., negatively comparing one’s appearance to other individuals of greater perceived attractiveness), negative affect, or bodily sensations such as hunger and satiety cues. In fact, this latter process, referred to as interoceptive awareness, has been found to be a critical deficit for individuals with eating disorders (Brown, 2010; Fassino, Pier, Gramaglia, & Abbate-Daga, 2004).

Acceptance, on the other hand, is a willingness to experience physical and psychological events without attempts to avoid them and without them exerting an undue influence on one’s behavior (Butler & Ciarrochi, 2007). Acceptance denotes a process of allowing, tolerating, and embracing all events regardless of their perceived valence. From a conceptual standpoint, a lack of acceptance of experiences (i.e., experiential avoidance) is hypothesized to be at the core of psychological distress (Hayes, 2004). In contrast, fostering an orientation of acceptance ends the struggle with unwanted thoughts and feelings by changing how we relate to them (e.g., relating to unwanted thoughts simply as thoughts as opposed to relating to unwanted thoughts as truth). Attempts to control internal and external experiences by eradicating their presence or changing their form is, paradoxically, part of the problem, not the solution.

Although there are sizeable similarities between acceptance and mindfulness (e.g., a nonjudgmental stance), research suggests that acceptance and mindfulness are, in fact, distinct constructs (Cardaciotto, Herbert, Forman, Moitra, & Farrow, 2008), such that an individual may experience one without the other. For example, generalized anxiety disorder is conceptualized as a disorder with a high degree of attention allocated to immediate physiological sensations (i.e., mindful awareness) coupled with a low degree of acceptance of these sensations. This idea has since been validated by studies showing that items on a measure that assess mindfulness (e.g., “I
notice how my emotions express themselves through my body”) and acceptance (e.g., “I [do not]
criticize myself for having irrational or inappropriate emotions”) do, in fact, load onto separate factors (Baer, 2006).

Taken together, mindfulness is the ‘what’ of experience (noticing what is occurring at a
given moment in time) and acceptance is the ‘how’ (cultivating an open and accepting stance
toward personal experiences, both good and bad, at a given moment in time; Linehan, 1987). In
the context of eating disorders, being mindful of personal experiences refers to noticing typical
cognitions, behaviors, emotions, and physiological sensations that are specific to the disorder.
These may include self-deprecating thoughts such “I am ugly” and “I am imperfect,” behaviors
such as restricting food intake and engaging in compensatory behaviors, emotions such as guilt
and sadness, and physiological sensations such as hunger cues and overall emotional awareness
(Garner, Olmstead, & Polivy, 1983). Given that these experiences are commonplace in
individuals with an eating disorder, simply bringing them to conscious awareness is the first step
of therapeutic growth with AMTs.

The aim of cultivating an open and accepting stance toward how these disordered
experiences are understood and evaluated represents the component of AMTs that has the
clearest divergence from traditional forms of CBT. In the context of eating disorders, one may,
for example, experience the thought “I am ugly” as an accurate and truthful reflection of reality
and subsequently engage in maladaptive control behaviors such as extreme caloric restriction and
excessive exercise. Inability to maintain such extreme behavior may lead to maladaptive
behaviors such as binge eating, which may exacerbate the original thought and perpetuate the
cycle. In contrast, AMTs aim to change the context in which these events are experienced (see,
for example, Hayes et al., 2001). In this way, rather than attempting to control unpleasant
experiences by changing the form or severity, AMTs aim to change one’s relationship to internal experiences such that the thought “I am ugly,” for example, may be experienced simply as a thought and not as a self-referent truth. Similarly, the behavior of binge eating may be experienced with a sense of self-compassion and understanding that it is acceptable to make mistakes from time to time. In doing so, AMTs are hypothesized to disrupt the maladaptive and pathological behavioral patterns that are frequently seen in individuals with eating disorders (Baer et al., 2005).

Understanding when and how AMTs differ from the previous therapeutic approaches is an important step in elucidating the unique and powerful contribution of mindfulness and acceptance in therapeutic growth. Despite the differences between AMTs and early behaviorally-based treatments, these therapeutic orientations have remarkable overlap in certain domains. For example, although a first means of contrasting AMTs and behavioral and cognitive-behavioral treatments may lie in the comparison of their specific techniques, highlighting these discrepancies may be insufficient to capture the bifurcation of the clinical treatments. In fact, recent theories suggest that the specific skills and techniques of both AMTs and CBT have much more considerable overlap than first thought (Herbert & Forman, 2011a). Similarly, Beck (1976) stated that differences in therapeutic approaches should not be understood simply by differences in their specific therapeutic techniques. Instead, he suggested that differences in therapeutic approaches should be understood on a wider level, considering the model of psychopathology and health, as well as the mechanisms of change. (Levin & Hayes, 2011). In the context of AMTs, consideration of these global differences adds clarity to how they, as a group, differ from their predecessors.
From this perspective, AMTs and the behavioral and cognitive-behavioral treatments that they originated from reveal considerable differences both in their model of psychopathology and health as well as in their mechanisms of change. For example, traditional cognitive therapy models view psychopathology as a combination of discrete and identifiable behaviors and cognitions (i.e., symptoms) that vary in their respective levels of accuracy and utility (Beck, 1976). Changing the form of these symptoms from inaccurate/unhelpful to accurate/helpful leads to positive clinical gains (a process referred to as cognitive causality; Beck, 1976). In this way, mental health disorders exist via their symptoms and eliminating symptoms by challenging their form or utility restores health.

In contrast, AMTs view psychopathology from a fundamentally different perspective in that distressing and uncomfortable thoughts and behaviors are not in themselves the problem. Rather, distress arises from non-acceptance of these experiences and the subsequent behaviors that emerge in an attempt to control, suppress, or otherwise push them away. Health, in the context of AMTs, encompasses a larger dimension than that of Behavior Therapy (BT) and CBT, is inclusive of uncomfortable and unpleasant experiences as well as desirable and positive ones, and emphasizes an acceptance of negative (and positive) experiences, a mindful awareness of present-moment existence, and a commitment to values, goals, and meaningful experiences. In short, health is not simply a lack of disease but a capacity to embrace experiences of any valence while pursuing a rich and meaningful life.

In summary, in contrast to traditional cognitive behavioral models of treatment, AMTs reduce the focus of cognitive reappraisal via evaluating the literal content of cognitions. Rather, fundamental goals in AMTs include mindfully contacting the present-moment experience in a non-judgmental way, promoting a sense of distance from internal events, and fostering a deeper
sense of self. An implicit emphasis is placed on empowering clients to actively notice these private events in the present moment, understand them as distinct from who they are as a person, and stop attempts to reduce, control, or change them in any way. However, despite the fact that AMTs do not necessarily focus on reducing symptomatology, this outcome is, indeed, an important goal in psychological treatment. In this regard, AMTs have been shown to be effective in reducing symptoms for a wide range of mental health disorders (Khoury et al., 2013) and specifically for individuals with eating disorders (Baer, 2003; Baer et al., 2005).

**AMTs in the Treatment of Eating Disorders**

A number of AMTs have been developed specifically for eating disorders and sub-clinical disordered eating (Baer, 2003; Baer et al., 2005; Telch, 2000; Kristeller, 1999; Kristeller & Wolever, 2010; Heffner, 2002). The majority of the existing research on developing or modifying AMTs for eating disorder treatment has focused on binge eating disorder. In particular, MBCT has been modified for binge eating disorder (Baer, 2003; Baer et al., 2005) and preliminary evidence supporting its efficacy in treating symptoms related to binge eating disorder has emerged (Baer et al., 2005). Mindfulness-based eating awareness training (MB-EAT; Kristeller & Wolever, 2010) has also been developed as a specific treatment protocol for binge eating and represents the first AMT that was developed specifically for eating disorders. MB-EAT is a 9-week group therapy protocol aimed at decreasing binge eating through daily meditative practices and related mindfulness and acceptance-based teachings. In a preliminary un-controlled trial using a single group extended baseline design, a sample of 18 women who met the criteria for binge eating disorder participated in a 6-week version of MB-EAT (Kristeller, 1999). The intervention was found to be effective in decreasing binge eating episodes. Most notably, the MB-EAT intervention reduced the overall amount of binges rated as
“large” from 70.3% pre-intervention to 18.1% post-intervention. The intervention also significantly increased self-report levels of mindfulness, awareness of hunger and satiety cues, and control over eating. More recently, a two-site randomized controlled trial (RCT) compared MB-EAT to psychoeducation/cognitive behavioral therapy (PECB) and a wait list control among a sample of 150 individuals (Kristeller, Wolever, & Sheets, 2013). The intervention consisted of a 12-week version of MB-EAT and assessed pre and post binge eating psychopathology as well as 1 month and 4 month follow-ups. Results showed that both interventions were superior to the control group and had generally comparable symptom improvements 1 month following treatment. However, at 4-months post-intervention, 95% of individuals in the MB-EAT group no longer met diagnostic criteria for binge eating disorder versus 76% for the PECB group. In sum, there is preliminary evidence for the efficacy of AMTs in treating binge eating disorder.

Bulimia Nervosa has also been subject to clinical research using AMTs. Specifically, preliminary evidence has been found for a modified version of DBT for bulimia nervosa and related binge eating behaviors (Telch, 2000; Safer, Telch, & Agras, 2001). Using a 20-week group therapy format, DBT for bulimia nervosa typically targets deficits in emotion regulation and distress tolerance skills underlying the disorder. Specifically, this approach is aimed at increasing an individual’s ability to manage negative affect in healthy and adaptive ways with the ultimate goal of replacing binge eating as an avoidance strategy with a stance of nonjudgmental acceptance of negative affect and distressing life events. Although no studies to date have assessed MBCT or MB-EAT for bulimia nervosa, these two treatments may also show promise for this population when considering that binge eating is a primary component of bulimia nervosa.
Likewise, AMTs specific for anorexia nervosa are limited due to a number of factors including the low base-rate of the disorder and the high degree of debilitation inherent in the illness. However, a modified version of ACT has been hypothesized to be a suitable treatment for the disorder. Heffner, Eifner, & Hayes (2004) created an ACT-based self-help book for anorexia nervosa that advocates for decreasing attempts to avoid adverse internal experiences while changing overt behavior to strive for long-term goals and values. A detailed case study of the use of ACT for anorexia nervosa has also been published (Heffner, 2002). Furthermore, a recent uncontrolled study compared the efficacy of ACT to treatment-as-usual among a residential sample of 140 individuals with anorexia nervosa (Juarascio et al., 2013). The treatment-as-usual group received intensive residential treatment, and the ACT group received the same treatment along with additional weekly ACT-based group therapy sessions. Both groups were found to have significant decreases in symptomatology. Although there were no significant group differences, there were trends toward larger decreases among the ACT group when compared to the treatment-as-usual group, and the ACT group had a decrease in re-hospitalization at 6-month follow up. In light of this study, AMTs thus show some promise in the treatment of anorexia nervosa and may represent a contemporary approach to conceptualizing treatment and outcomes for a notoriously difficult condition to treat. However, this is only one study, and it was uncontrolled.

Despite the number of unique treatments protocols within the AMT category, each with their own set of therapeutic goals and practices, there are, in fact, a number of clinical tools which cut across most, if not all, of the approaches. For example, meditation is a basic practice of most AMTs that aims to bolster present-moment awareness and regulation of attention (Walsh & Shapiro, 2006). Similarly, the process that differentiates AMTs from traditional forms of
Eastern meditative practice is a relative focus on cognition and behavior. For example, many treatments within the AMT category advocate for cognitive-based practices such as experiencing thought as thought (Teasdale et al., 2000), cognitive flexibility (Hayes, 2004) and thought-action fusion (Hayes, 2004; Teasdale et al., 2000; Wells, 2008). These aspects of treatment are at the core of the therapeutic practices that bond together the various AMT approaches to treatment, and allow for a flexible delivery of therapy that can draw from different acceptance and mindfulness practices in order to tailor treatment to a specific individual and her therapeutic needs.

In sum, there is emerging but limited evidence that suggests that AMTs can, in fact, decrease problematic and pathological symptoms among certain eating disorder diagnoses. However, the implications of these findings are limited due to relatively small sample sizes and relatively weak effects when compared to other forms of cognitive therapy. Despite these limitations, a preliminary clinical trial has indicated that AMTs perform equivalently or better than other forms of psychological treatment among certain clinical diagnoses such as binge eating disorder, and represents an important early-stage milestone in the development of psychological treatment for eating disorders. Specific AMT protocols catering to certain diagnostic groups have been developed, each drawing from core AMT practices and extending their reach to influence the unique aspects of the various eating disorders. Understanding these core practices and their relative functions may thus shed light on the specific mechanism through which AMTs are successful in reducing eating disorder symptomatology.

**Mechanisms of Change in Acceptance and Mindfulness Based Therapies**

AMTs encompass a group of effective treatments for mental health disorders in general (e.g., Hayes et al., 2006, Kabat-Zinn, 2003) and eating disorders in particular (e.g., Baer, 2003;
Baer, Fischer, & Huss, 2005), which focus on fostering a state of mindfulness and acceptance. Based on the RCT (Kristeller, Wolever, & Seets, 2013) and related treatment outcome studies reviewed above (Kristeller, 1999; Juarascio et al., 2013; Safer, Telch, & Agras, 2001; Telch, 2000), we know that teaching skills fundamental to AMTs do, in fact, result in decreases in eating disorder symptoms among certain populations. What is much less known, however, are the mechanisms and processes through which these therapies work.

The medical model and treatment outcome. Virtually all the treatment efficacy studies mentioned above use decreases in symptomatology as the clinical measure for successful treatment outcome. This is, of course, for good reason: lower experiences of symptoms related to a certain mental disorder is, indeed, a meaningful outcome. In the context of the predominant model of psychopathology, this conceptualization of clinical change is largely influenced by the medical model of disease in which the presence of a disease is indicated by the presence of its observable symptoms (Borsboom & Cramer, 2013; McNally et al., 2015). Thus, eradicating the observable symptoms via some form of treatment indicates that the disorder has been successfully treated and health has been restored.

In the context of psychopathology, this disease model is the predominant means of identifying mental disorders. This is primarily done by measuring the presence or absence of discrete psychological symptoms and considering the frequency and severity of these symptoms as indicators of the presence and severity of the underlying disorder. For example, a person who is experiencing a fear of weight gain, negative affect, a strong desire to be thin, and who is engaging in restrictive and compensatory behaviors is doing so because the anorexia nervosa is causing these symptoms. In this way, the symptoms (e.g., restriction, fear of weight gain) are
viewed as independent indicators of a latent disorder (e.g., anorexia nervosa) and reductions in symptoms act as the primary clinical measure for successful interventions.

In contrast, the basic practices of AMTs do not focus on decreasing the symptoms associated with a given disorder. In fact, most even advocate against such a practice, stating that efforts to decrease negative experiences are an attempt to control the inner world and a form of experiential avoidance (both of which, according to AMTs, are core aspects underlying psychological distress in general). As such, the core tenets of AMTs seem to be fundamentally divergent from the disease approach to measuring health and wellness. The question thus arises as to how AMTs decrease symptomatology despite having no explicit aim of doing so.

**Mechanisms of change.** To evaluate the how, AMT researchers have aimed to identify mechanisms of change associated with acceptance and mindfulness. A mechanism of change (in psychological treatment) is defined as the process or event that is responsible for the change in symptomatology. In short, a mechanism of change is the basis for the effect (Kazdin, 2007). Conceptualizing and investigating mechanisms of change in psychological treatment is often carried out by assessing core processes associated with a specific theoretical model (Gu, Strauss, Bond, & Cavanagh, 2015). In the context of AMTs, the core theoretical processes that were initially hypothesized to account for positive therapeutic change were a non-judgmental stance and non-reactive acceptance of all experiences on the part of the patient (Kabat-Zinn, 1982, 1994). However, no studies at the time had empirically tested this claim using formal mediation models that are considered to be the most appropriate means of testing these hypotheses.

Since this time, a number of empirically-derived theoretical models have been postulated with regard to the mechanisms of change in AMTs (Baer, 2003; Brown, Ryan, & Creswell, 2007; Grabovac, Lau, & Willett, 2011; Holzel et al., 2011; Shapiro, Carlson, Astin, & Freedman,
2006; Vago & Silbersweig, 2012). For example, Brown et al., (2007) hypothesized that the constructs underlying the positive effects of AMTs are insight, exposure, nonattachment, enhanced mind-body functioning, and integrated functioning. Similarly, Baer (2003) posited that exposure to internal experiences, cognitive change, self-management, relaxation, and acceptance are the components that account for the positive therapeutic effects of AMTs. Integrating findings from neural/biological research on AMTs, Holzel (2011) argued that attention regulation, body awareness, emotion regulation (including reappraisal, exposure, extinction, and reconsolidation), and change in perspective on the self were all constructs that accounted for the effectiveness of AMTs. Needless to say, the theoretical models and related mechanisms of change associated with AMTs that have been included in empirical investigations encompass a great variety of discrete yet overlapping constructs and suffer from a lack of homogeneity in the terminology used.

Shapiro et al. (2006) proposed a formal model of the mechanisms underlying the effectiveness of AMTs that has since been used as the benchmark for psychological research of acceptance and mindfulness. Based on the original definition of Kabat-Zinn, their model postulates that intention (i.e., "on purpose"), awareness (i.e., "paying attention"), and acceptance (i.e., "in a particular way") are the underlying mechanisms accounting for the positive treatment effects seen in clinical trials with AMTs. These constructs are, in turn, part of a larger meta-mechanism called repercieving. Analogous to the concept of decentering and deautomatization in cognitive therapy (Safran & Segal, 1996) and similar to that of cognitive defusion and deliteralization in Dialectical Behavioral Therapy (Hayes et al., 2006; Hayes, Strosahl, & Wilson, 1999), repercieving involves a fundamental shift in perspective such that personal experiences are witnessed as an objective phenomenon rather than as a subjective experience. In
other words, reperceiving is the ability to step outside of personal experience such that an individual’s relationship to an event shifts from a passive recipient to an active witness. In this way, reperceiving is conceptualized as a meta-mechanism of mindfulness and acceptance whereby one gains an increased capacity for psychological objectivity through fostering experiential intention, awareness, and acceptance (Shapiro et al., 2006).

Furthermore, Shapiro et al. (2006) put forth a number of additional mechanisms, which are hypothesized to account for the relationship between reperceiving and actual clinical change. These mechanisms include (among others) increased self-regulation, psychological flexibility, and exposure. Self-regulation is defined as the process by which an individual influences his or her psychological experiences and how these experiences are understood and expressed (Gross, 1998; Hayes, Villatte, Levin, & Hildebrandt, 2011). In the context of eating disorders, improvement in this domain is especially important because control is a critical dimension in eating disorders, with both deficits in control and a tendency to over-control underlying the behaviors, depending on the presentation (Sim, 2002). As such, the process of reperceiving fosters increased self-regulation by elucidating and objectifying behavioral patterns that were once automatic and out of immediate behavioral control. Secondly, psychological flexibility refers to the ability to respond to cognitive, emotional, and behavioral events with a greater freedom of choice. Given that many eating disorders are maintained by a high degree of cognitive rigidity, psychological flexibility is likely a particularly salient treatment goal for eating disorder treatment and prevention (Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007). Similar to self-regulation, reperceiving is hypothesized to foster psychological flexibility through the process of stepping back from personal experiences, allowing a greater view of all possible responses. This may take the form, for example, of a patient with anorexia nervosa.
being able to understand that gaining weight means that he or she will be slim, not fat (a perception that gaining any weight equates to ‘fatness’ regardless of one’s weight comparative to social norms is common in eating disorders). Lastly, exposure refers to the ability to openly attend to affective experiences in the body. Although similar to the common use of the word in traditional CBT, exposure in the context of Shapiro’s (2006) model refers specifically to the process of allocating one’s attention inward toward his or her experiences (most notably in the presence of unwanted or uncomfortable thoughts, sensations, and emotions), rather than simply referring to withholding avoidance in the presence of a feared stimuli. The process of exposure has been associated with a number of positive psychological outcomes (Ougrin, 2011). The excessive preoccupation with eating and weight often seen in eating disorders has been hypothesized to develop, in part, as an emotional avoidance strategy (Merwin & Wilson, 2009). Thus, turning one’s attention inward and experiencing the full range of emotions (i.e., exposure) may represent another important step in eating disorder treatment. In this way, reperceiving is hypothesized to lead to an increase in exposure by focusing one’s attention on internal, present-moment experiences.

**Empirical evidence for mechanisms of change.** Research using mediational designs (Baron & Kenny, 1986; Shrout & Bolger, 2002) has evaluated the clinical processes mentioned above as potential mechanisms of change. It should be noted that something that mediates change may not necessarily be a mechanism explaining how the effect occurred. This is due to a number of factors including the overarching psychological theory, the possibility that the mediator is a proxy for one or more other variables, and the fact that the mediator may be a general construct not intended to explain the effect in the first place (Bullock, Green, & Ha, 2010; Kazdin, 2007). Despite these factors, mediation analysis is the most widely used method
of analysis for identifying the mechanism of change in psychological research and is a viable method when psychological theory guides the process of identifying mediators (Kazdin, 2007).

The majority of the research on the mechanisms hypothesized by Shapiro et al. (2006) has assessed the broad constructs (e.g., mindfulness, awareness, acceptance) related to reperceiving. Overall, support has been found for the specific components of reperceiving as a mechanisms of change in AMTs (Batink, Peeters, Geschwind, van Os, & Wichers, 2013; Bieling et al., 2012; Kuyken et al., 2010; Michalak, Heidenreich, Meibert, & Schulte, 2008; Teasdale et al., 2002). All but one study (Michalak et al., 2008) utilized an RCT research design. Regarding the additional mechanisms of change, overall support has been found for psychological flexibility and self-regulation using self-report measures (see Baer, 2003 for a review). Exposure as a general construct has the largest pool of empirical support for its efficacy in decreasing mental health symptomatology (Ougrin, 2011). In total, reperceiving and its constituents seem to be important mechanisms underlying the effectiveness of AMTs in reducing symptom severity.

In relation to the mechanisms of change specific to the treatment of eating disorder treatment, only one study to date directly assessed processes through which changes in mindfulness and acceptance are associated with symptom severity and treatment outcome in eating disorders (Butryn et al., 2013). The aim of the study was to assess: a) the relationship between various constructs related to mindfulness and eating disorder symptom severity at baseline; and b) the association between changes in mindfulness following a residential treatment program and changes in symptom severity. The study utilized a sample of 88 participants admitted to a residential treatment who were diagnosed with anorexia nervosa (N = 35), bulimia nervosa (N = 29) and eating disorder not otherwise specified (N = 24). Treatment was uncontrolled and consisted of a comprehensive program of individual, group, and family therapy.
provided by an interdisciplinary team. Facets of mindfulness were measured by the Body Image Acceptance and Awareness Questionnaire (Sandoz, Wilson, Merwin, & Kellum, 2013) and the Philadelphia Mindfulness Scale (Cardaciotto et al., 2008). The findings revealed that lower levels of body image acceptance, general psychological acceptance, present-moment awareness, and cognitive defusion, as well as greater levels of emotional avoidance, were associated with greater levels of specific and general measures of eating disorder symptomatology. Similarly, increases in body image acceptance and present-moment awareness were associated with decreases in eating disorder symptomatology post intervention. Likewise, decreases in avoidance of positive emotions, negative beliefs about emotions, and social concerns about displaying emotions were associated with decreases in eating disorder symptomatology post intervention. Although a formal mediation analysis was not conducted, these findings provide preliminary evidence for the importance of these constructs in the process of eating disorder treatment with AMTs. However, the study was uncontrolled, treatment was unstandardized across participants, and the sample size was small.

In sum, a small group of previous research studies have explored which facets of mindfulness need to change in order for AMTs to be effective, but have not explored how changes in these constructs actually lead to reductions in symptom severity. In other words, these studies have explored the constructs related to symptom improvement, not the process by which symptoms are reduced. This distinction is important because practices related to mindfulness and its various facets do not advocate for decreasing symptoms, and many even advocate against such efforts, thus creating a gap between the aim of the therapeutic skill and the desired outcome. In this way, identifying a clinical process that may account for therapeutic change in AMTs is a critical step in aligning research and practice. For example, CBT is hypothesized to operate via
the process of cognitive causality. In this view, cognitions play a direct causal role in subsequent emotions and behaviors. As such, adaptive cognitions lead to adaptive emotions and behaviors, and maladaptive cognitions lead to maladaptive emotions and behaviors. A primary focus of treatment is, thus, to change the content of maladaptive cognitions to a more adaptive form in order to cause positive changes in subsequent emotions and behaviors. Although empirical research on the cognitive causality hypothesis to date is relatively weak, this process still represents (at minimum) a fundamental conceptualization regarding how cognitive therapy effectively reduces psychological symptoms.

It is necessary to capture such an overarching process related to AMTs in order to provide a framework for understanding therapeutic change following improvements in acceptance and mindfulness. Identifying such a process can aid in unifying the specific theoretical models and proposed mechanisms of change in AMTs mentioned above and can elucidate the way in which these mechanisms relate to one another. However, research and theory related to AMTs is only beginning to identify a process such as this that is discrete and quantifiable.

**Decoupling as a Process of Change Related to AMTs**

Decoupling has recently been introduced as a mechanism of change associated with AMTs that advances the understanding of how AMTs lead to symptom reductions by identifying a single hypothesized process accounting for therapeutic change. Decoupling is defined as a process by which the relationships between two internal experiences (e.g., body dissatisfaction, fear of weight gain) or between an internal experience and overt behavior (e.g., desire to be thin and restricted eating) are reduced, eliminated, or altered (Levin, Luoma, & Haeger, 2015). In other words, decoupling weakens the relationship between symptoms without necessarily
changing their form or severity. In this way, for example, desire to be thin may be experienced with equal intensity but would no longer be associated with restriction.

Importantly, a distinction should be made between the terms decoupling and uncoupled. Decoupling refers to the active process of decreasing the association between two things (as defined above), whereas the term uncoupled refers to the level of an already known association relative to something else. For example, two symptoms may be uncoupled in a control group relative to a clinical sample, but that information alone does not provide insight into the mechanism that contributes to decoupling the two symptoms. Likewise, symptoms are decoupled if, for example, an intervention has been shown to decrease the association between negative affect and binge eating across time or experimental conditions. In other words, the term uncoupled describes a current state between two symptoms and the term decoupling describes a dynamic process between two symptoms unfolding over time. The former is measured through between-subject research designs and the latter is measured through within-subject research designs.

In the context of eating disorders, between-subject research has indicated that symptoms are uncoupled among groups of individuals with high levels of acceptance and mindfulness. For example, state mindfulness was found to moderate the association between disordered eating cognitions (e.g., “If my weight goes up, my self-esteem goes down” and “No one likes fat people; therefore, I must remain thin to be liked by others”) and endorsement of disordered eating behaviors (e.g., “I avoid eating when I am hungry” and “I vomit after I have eaten”; Masuda, Price, & Latzman, 2012). Similarly, body dissatisfaction was associated with greater drive for thinness and pathological eating behaviors only among individuals reporting low body image acceptance (Ferreira, Pinto-Gouveia, & Duarte, 2011). Together, both studies point to
cross-sectional evidence of the association between AMT-based constructs and symptom connectivity among disordered-eating individuals.

Preliminary evidence for a decoupling effect has been found among a broad range of psychosocial and behavioral disorders such as substance abuse, depression, anxiety, chronic pain, anger, and self-harm (see Levin et al., 2015 for a review). Three studies experimentally assessed the process of decoupling in the context of eating behaviors by examining the effect of mindfulness interventions on the relationship between internal cues and unhealthy eating behaviors (Marchiori & Papis, 2014; Papis, Barsalou, & Custers, 2012a; Papis, Pronk, Keesman, & Barsalou, 2015). For example, one study assessed the role of mindfulness in the relationship between hunger and eating behaviors by comparing a control group to an intervention group exposed to a brief mindfulness training (Marchiori & Papis, 2014). Using a sample of 110 undergraduate students, the study was designed such that participants were presented with a small or large portion of chocolate chip cookies (defined as unhealthy food) after listening to an audio book or performing a mindfulness exercise. Results of the study indicated that a significant relationship between hunger and unhealthy food consumption was present only for the control group, such that the amount of calories consumed in the control condition was a function of their state level of hunger (being hungry was associated with consuming more unhealthy foods). Individuals in the mindfulness condition did not show this effect. Thus, mindfulness training decoupled the relationship between feeling hungry and consuming unhealthy foods.

A similar study found that hunger was significantly predictive of subjective attractiveness of unhealthy foods for a control group but not for a mindfulness intervention group, suggesting that the mindfulness-based intervention decoupled the relationship between hunger and
desirability of unhealthy food choices (Papies et al., 2015). A similar effect has been found for
general approach and avoidance behaviors related to unhealthy food choices (Papies, Barsalou,
& Custers, 2012b). Approach responses to food were measured by an approach-avoidance task
whereby pictures of healthy and unhealthy were presented in blue or purple frame on a computer
screen and participants were told to press on one arrow key to “move toward” the picture if it
appeared in the blue frame, or press a separate arrow key to “move away” from the picture if it
appeared in the purple frame. Response latencies were calculated for each condition x stimuli
pairing (Bamford & Ward, 2008). Results indicated that approach responses to unhealthy foods
were fully eliminated among a mindfulness intervention group when compared to a control
group (Papies et al., 2012a). Taken together, these studies preliminarily suggest that AMTs may
play a role in the regulation of eating behaviors by decoupling the relationship between internal
cues such as hunger and unhealthy eating behaviors. However, given that none of the studies
formally assessed the level of eating disorder symptom severity, it remains unclear whether these
findings will generalize to eating disorder diagnostic symptoms or to a clinical sample of
individuals diagnosed with an eating disorder.

Although the evidence for decoupling as a mechanism of change associated with AMTs
has increased, the research studies examining this effect thus far have suffered from a number of
limitations. For example, only discrete pairs of symptoms and associated features of eating
behavior have been assessed. Consequently, it is unclear how the process of decoupling fits in
with eating disorder psychopathology as a whole and how other symptoms associated with the
disorders may affect this process. It may be that although AMTs impact the relationship between
hunger and unhealthy eating behaviors, this relationship may only play a minor role due to
greater contributory effects of other symptoms associated with unhealthy eating. For example,
other constructs such as negative affect may predict unhealthy eating behavior regardless of the strength of the relationship between hunger and unhealthy eating. Also, the process of decoupling was only assessed through cross-sectional research designs and it is not known whether a relationship exists between mindfulness and within-subject symptom connectivity. This limitation is critically important given the fact that the mechanism of change (i.e., decoupling) is, by definition, a temporal process unfolding across time and within an individual. Lastly, the process of decoupling has not been integrated into models of psychopathology and health. Given that the distinguishing features of different treatment models are derived in part from their overall model of psychopathology and health (Levin & Hayes, 2011), an integral step in progressing decoupling as an important theoretical mechanism in eating disorders is to directly integrate the process of decoupling with an overarching model of eating disorder psychopathology.

Considering the limitations above, the preliminary steps to broaden our understanding of the process of decoupling as a mechanism of change in AMTs for eating disorders is to: a) identify a method for analyzing symptom connectivity of multiple symptoms; b) assess symptom connectivity across time and within individuals; c) relate symptom connectivity to clinical outcome measures; and d) integrate the process of decoupling into a conceptual model of eating disorder psychopathology.

**The Network Approach to Eating Disorders and the Role of Connectivity**

The network approach to psychopathology (Borsboom & Cramer, 2013) provides a framework for empirically examining the process of decoupling. Although originating as a model for depression and anxiety, the network approach has since been applied to a number of mental health disorders such as persistent complex bereavement disorder (Robinaugh, LeBlanc,
Vuletich, & McNally, 2014), post-traumatic stress disorder (McNally et al., 2015), depression (van Borkulo et al., 2015), obsessive-compulsive disorder (Ruzzano, Borsboom, & Geurts, 2015), and eating disorders (Forbush, Siew, & Vitevitch, 2016). This approach rests on the premise that psychological symptoms are constitutive, rather than reflective, of a particular mental disorder.

The Latent Trait Approach to Psychopathology. A helpful way to describe this approach is first to differentiate it from the prevailing model of psychopathology. The mid-twentieth century represents a period of enormous growth for empirically-based psychological research in America and, subsequently, the beginning of the modern era of psychological classification. Driven by the prevailing emphasis on empiricism and biology, and by the growing medicalization of the mental health field, eating disorders at this time were conceptualized from a disease model. This model, referred to as the latent trait approach, conceptualized eating disorders as latent entities. Eating disorder symptoms were construed as reflective indicators of the underlying disorder, the presence of which denoted the presence of the disorder. According to this view, the specific disorder (e.g., bulimia nervosa) acted as a common cause of each of its symptoms (e.g., fear of weight gain, restriction, binge eating) and, in this way, symptoms occurred together because of a common determinant (Edwards & Bagozzi, 2000).

Although this approach has remained the predominant model of eating disorders (and psychopathology as a whole), a number of problematic assumptions have recently called its utility into question. For example, one problematic aspect of the latent trait approach lies in the psychometric assumptions of the model. Most notably, the symptoms of eating disorders often violate the axiom of local independence. Specifically, the latent trait model assumes that symptoms (e.g., fear of weight gain, restriction, binge eating) are reflective indicators of a
common cause (e.g., bulimia nervosa). In this way, symptoms are independent and interchangeable indicators of the latent disorder and covariation among them is due to their shared cause, not from their shared influence on one another. In other words, the symptoms must be locally independent (McNally et al., 2015). However, a great majority of research has shown that symptoms of eating disorders do, in fact, interact with one another (Fairburn et al., 2003; Lampard, Byrne, McLean, & Fursland, 2011). Congruent with the network approach, this local dependence is the cornerstone of the leading transdiagnostic eating disorder treatment (Fairburn et al., 2003), whereby targeting certain eating disorder symptoms will cause positive changes in other symptoms down the causal chain. In this way, it is a fair assumption that people who worry about their weight might likely diet in an attempt to control or lose weight, and that this dieting may inevitably lead to instances of eating more food than usual.

One may also question whether mental disorders consist of an underlying latent entity on the grounds that little to no evidence has supported the existence of such an underlying entity. Despite an enormous amount of research, strikingly little evidence has been found for a common cause related to any of the mental disorders, with most research pointing to highly multifactorial etiologies (Kendler, 2005; Nolen-Hoeksema & Watkins, 2011). A few exceptions exist. For example, McNally et al. (2015) described the case study of general paresis of the insane (GPI), one of the most serious mental health syndromes of the 19th century. The main symptoms of GPI included delusions, fatigue, and mood disturbances and over time was found to progress to dementia, paralysis, and death (Noguchi & Moore, 1913). Following decades of research, *treponema pallidum*, a type of bacteria that causes syphilis in the brain, was identified as a common cause of the disorder and confirmed the causal link between syphilis and GPI. However, considering the complexity of their etiology and their vastly heterogeneous
presentations, it is unlikely that a common cause such as *treponema pallidum* will be identified for any of the eating disorders in the near future.

**The Network Approach to Psychopathology.** In response to the limitations of the latent trait approach, a model of psychopathology, referred to as the network approach, was developed in which symptoms and their causal relationships constitute the disorders independent of an underlying causal entity (see Figure 1). Broadly speaking, a network is simply a set of objects and the relationships among those objects. The objects in a network, referred to as nodes, can be anything from people, to genes, to, in the context of psychopathology, symptoms. The relationships among the objects, referred to as edges in a network, are the specific measurement used to quantify the relationships among the objects. For example, an edge connecting the nodes fear of weight gain and restriction would be represented by a correlation between these symptoms. Accordingly, symptoms associated with a specific disorder are modeled as components of a causal system, rather than independent indicators of a latent disorder. As such, disorders arise due to causal relations between symptoms (e.g., fear of weight gain -> restriction -> binge eating -> fear of weight gain) and these relationships are hypothesized to maintain the disorder over time.

In the context of eating disorder psychopathology, these symptom associations encompass both the etiology and the maintenance of eating disorder psychopathology (Fairburn et al., 2003; Murphy et al., 2010). This process is best exemplified by the transdiagnostic model of bulimia nervosa. According to this view, overvaluation of weight, shape, and their control are core features of bulimia nervosa. This experience leads to cognitive restraint (e.g., intention to diet) and non-compensatory weight control behaviors. These restraint behaviors are often accompanied by unrealistically strict and rigid beliefs about diet and weight loss which are
inevitably broken, leading to a brief period of relief from the rigid dietary rules. During this time, a large amount of food is consumed and a sense of loss of control over eating is experienced. Following this binge eating behavior, there are often attempts to remediate the episode by engaging in compensatory behaviors and re-adopting the rigid dietary restriction rules and over-evaluation of weight and shape. As a whole, this process can be interpreted as a causal cycle, whereby each symptom is maintained simply by the causal effect of other symptoms in the system.

It is important to point out that, although the cycle of bulimia nervosa is, indeed, pathological, the experiences themselves may be normative. For example, many individuals engage in dieting behaviors, and only a small percentage of these individuals actually meet diagnostic criteria for an eating disorder. Likewise, an overvaluation of weight and shape is common in American culture, especially among adolescent girls. These experiences alone do not necessarily constitute a disorder and should not be viewed as atypical. In contrast, the process that contributes to the maintenance of an eating disorder diagnosis is not necessarily the specific experiences in isolation, but rather the cyclical relationships among the symptoms that increases their severity over time.

Although the framework mentioned above is oversimplified and likely to vary across individuals, the central feature remains the same: eating disorders arise and are maintained by patterns of behavior which attempt to solve the problem of negative feelings about one’s weight, shape, and eating behaviors. Eating disorders can thus be modeled as a network of nodes (the symptoms) and edges (the relationships between symptoms), the process of which is driven by avoidance and/or control behaviors related to over-evaluation of eating, weight and shape. In this way, equally important to the actual symptoms of eating disorders are the functional
relationships among the symptoms that play an integral role in initiating and maintaining eating disorder symptomatology. In fact, this is precisely where AMTs exert their influence. Rather than overtly altering cognitions such as over-evaluation of eating, weight, and shape, AMTs aim to change one’s response to these experiences such that evaluation of weight and shape is experienced but does not lead to restriction and its consequences.

**Symptom Connectivity.** The process, referred to as experiential avoidance, is best captured through the construct of symptom network *connectivity*. Connectivity refers to the global magnitude of associations among all of the symptoms in a network (see Figure 2). As an example of this construct, let us consider the cases of Ashley and Carla (fictional characters), both of whom have recently experienced heightened sociocultural pressures related to shape and weight during their first month of high school. Ashley began to experience a fear of gaining weight and held the belief that gaining weight would have devastating effects on her social status at school. In an attempt to control this fear, she adopted rigid expectations about what and when she could eat, would often skip meals, and was acutely aware of her calorie consumption throughout the day. However, she would occasionally experience a binge eating episode following an inability to adhere to her strict dietary rules, which would further exacerbate her fear of weight gain. Similarly, Carla also began to experience a heightened fear of gaining weight. However, in contrast to Ashley, this experience was perceived simply as it was (i.e., an experience) and did not lead problematic consequences. By doing so, Carla may have experienced this fear and simultaneously acted according to her values and beliefs which did not encompass strict rules about eating and body image. From a network perspective, Ashley’s symptoms are highly connected, whereas Carla’s symptoms are not.
In order to frame symptom connectivity in the context of eating disorder treatment, it is important to first understand the distinction between symptom connectivity and symptom severity. One way of elucidating this difference is to first understand the distinctive roles related to connectivity and severity among single symptoms by themselves. For example, although the experience of negative affect is an important symptom in transdiagnostic models of eating disorders (Fairburn et al., 2003), it is also a normative human experience. The process that defines negative affect as a dimension of psychopathology is not exclusively the magnitude to which it is experienced, but the trajectory that the experience holds over time. For example, an individual may experience negative affect in a psychologically healthy way following a bad day at school, but may also experience difficulty ridding herself of this emotion over time. Thus, the direct experience of the emotion captures the severity of the symptom, and the inflexible trajectory of the emotional experience over time captures the moment to moment connectivity. This process, also referred to as emotional inertia, has been shown to be predictive of a wide range of psychological maladjustment including low self-esteem and high levels of depression (Kuppens, Allen, & Sheeber, 2010), two important constructs in eating disorder psychopathology. Importantly, the magnitude of moment-to-moment emotional connectedness has been identified as an early warning sign for disorder onset in conditions such as major depressive disorder among individual who were currently not experiencing symptoms of depression (van de Leemput et al., 2014; Wichers, Groot, Psychosystems, Group, & others, 2016), and has been found to predict the emergence of clinical depression among adolescents 2.5 years following the assessment period (Kuppens et al., 2012). These findings highlight the fact that moment-to-moment connectedness among emotional experiences such as negative affect is an important aspect in psychopathology above and beyond the severity of the experience, and
can be detected even among individuals who are not currently experiencing clinical levels of the particular emotion.

This process of pathological moment-to-moment connectedness is also seen among cognitive symptoms of psychopathology and is traditionally referred to as rumination. For example, the thought “I am worthless” may have accompanied the negative experience at school referenced above. From an AMT perspective, the presence of this thought is not in itself pathological as it is hypothesized to serve or have served a functional purpose at one time (Hayes et al., 2011). However, this individual may also have a propensity to exclusively focus on this thought such that its presence does not diminish and may even get worse over time. In other words, the thought has a high moment-to-moment connectedness for this individual, categorized by an impairment in cognitive disengagement (Koster, De Lissnyder, Derakshan, & De Raedt, 2011). Cognitive connectedness has been shown to be associated with a plethora of maladaptive consequences including enhancement of negative thought patterns, impairment of cognitive resources involved in problem solving, exacerbation of psychological disorders, and negatively affecting social support (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). This point has particular salience for eating disorders given that cognitive rumination related to disorder-specific cognitions has been hypothesized as an important component of eating disorder psychopathology (Cooper, Wells, & Todd, 2004; Lee & Shafran, 2004; Rawal, Park, & Williams, 2010).

Understanding moment-to-moment connectivity of symptoms in isolation is helpful in conceptualizing the distinction between symptom connectivity and symptom severity. However, from a network perspective, symptoms exist not in isolation, but as a part of a larger network of mutually-interacting components. Accordingly, the distinctiveness of connectivity and severity
exists beyond each symptom in isolation and should be viewed on the level of the whole disorder. In this way, symptom networks with high connectivity are viewed as risky because experiences of one symptom (e.g., dietary restriction) will create a higher likelihood of causing other symptoms to occur (e.g., binge eating), inevitably creating feedback loops that maintain the disorder. This idea has since been empirically validated. For example, one study assessed depressive symptoms among a clinical sample of 515 individuals with major depressive disorder at baseline and 2-year follow-up (van Borkulo et al., 2015). Results indicated that symptom connectivity was associated with disorder prognosis such that highly connected depressive symptoms indicated disorder persistence at the 2-year follow-up when compared to depressive symptoms with low connectivity. This finding remained significant when controlling for baseline symptom severity and general functional impairment, indicating that symptom connectivity was a meaningful construct in psychopathology above and beyond the level of symptom severity. Similarly, a more recent study with a non-clinical sample found that high connectivity among symptoms was associated with a number of constructs related to general psychological impairment, most notably neuroticism (Bringmann et al., 2016).

Given the proposed importance of connectivity in the etiology and maintenance of psychopathology, a critical aspect of treatment is not only to decrease the overt experience of symptoms for a certain individual, but to decrease the response patterns that causally connect the symptoms to each other moment-to-moment or to other symptoms across time. This distinction is crucial because it underlies the core aspirations of AMTs and provides an indicator of therapeutic progress that is compatible with the theoretical aims of AMT-based treatment. Thus, symptom decoupling may constitute the process of first order change that is a direct result of AMT interventions and that, in turn, may result in decreasing the severity of the symptoms themselves.
Furthermore, this process may account for the effectiveness of AMTs in decreasing eating disorder symptoms by allowing symptoms to be experienced in isolation and without causing a cascade effect, activating the eating disorder symptom network as a whole.

Advancing the understanding of symptom connectivity in the context of eating disorders can thus be viewed as a critical next step in identifying meaningful treatment indicators related to AMTs and will aid in progressing the integration of network theory and connectivity with conceptual models of eating disorder psychopathology. Furthermore, future research investigating symptom connectivity and how it relates to eating disorder severity across time may provide a promising new approach to measuring treatment outcome and identifying indicators of therapeutic change above and beyond symptom reductions. More fundamentally, understanding eating disorder symptom connectivity from a network perspective provides a framework from which AMTs are able to exert their influence. This is, indeed, an important step as, to date, AMTs lack integration with contemporary theories of psychopathology. Improving this integration will provide a new language for therapeutic growth that does not rely solely on symptom reductions, a process which is, paradoxically, at the heart of the problem.
CHAPTER 3

METHOD

The first aim of the study was to examine the relationship between symptom network connectivity and global levels of eating disorder symptom severity. It was hypothesized that greater levels of symptom network connectivity would be related to greater levels of eating disorder symptom severity. The second aim of the study was to investigate whether symptom connectivity accounted for the relationship between mindfulness and eating disorder symptom severity. It was hypothesized that symptom network connectivity would mediate the relationship between mindfulness and eating disorder symptom severity. Lastly, the exploratory aim of the study was to investigate the structure of and associative patterns within the eating disorder symptom network. The remainder of this chapter will describe the method that was used to examine these aims and test these hypotheses.

Recruitment and Participants

Three different methods were utilized for participant recruitment. First, flyers were posted around the Northeastern University campus in designated advertisement spaces. The flyers included a brief summary of the study (i.e., “Health and Eating Behaviors”), a statement of reimbursement “up to 45 dollars”, and contact information of the research staff. Second, electronic advertisements were dispersed throughout Northeastern-affiliated social media sites. The content of the electronic advertisements paralleled that of the flyers. Lastly, information regarding the study and contact information for the research staff was uploaded to MyNEU.

Exclusion criteria included age (i.e., younger than 18 years old or older than 40 years old), gender (i.e., not identifying as female), and currently receiving treatment for any mental health disorder. Individuals who did not identify as female were not included in the study due to
the potential differences in gender-specific eating and body image constructs needed to adequately capture eating disorder psychopathology. Furthermore, participants had to own an Apple or Android mobile phone for compatibility with the study’s data collection application. Exclusion criteria were evaluated by an initial e-mail and confirmed during phase 1 of the study. Importantly, potential participants were not screened for their respective level of eating disorder severity. Omitting this step was chosen in light of prior research suggesting that symptom connectivity and symptom network structure may be unbiased to symptom severity as long as the distribution of symptom endorsement is not subject to basal effects skewing the distribution (Fried et al., 2017). However, the items representing eating disorder symptoms in the EMA portion of the study were modified to capture a dimensional nature of disordered eating rather than discrete diagnostic symptoms as specified by the DSM-5 (see below for a detailed description of all items). This was done to account for the uncontrolled nature of eating disorder symptom severity in the sample and to mitigate the possibility basal effects resulting from low symptom endorsement.

The initial sample consisted of 64 participants. Participants who had completed under 30 assessments during phase two of the study were removed (≤ 37.5% response rate; N = 4; see below for a description of the study phases). This response rate was chosen based upon verbal communication with researchers familiar with EMA data collection and mlVAR models as well as with research suggesting a minimum threshold of 30 samples for 10-nodes networks using mlVAR methodology (Epskamp, 2016; Fried, 2016). Furthermore, participants who were affected by a bug in the EMA application midway through the study (time/date of assessment was coded incorrectly due to server malfunction; n = 2) were also excluded. After accounting for these secondary exclusion criteria, six participants were removed from the final dataset and a
total of 58 individuals were included in the subsequent analyses. The mean age of participants in the study was 20.5 (SD = 3.1). The majority of participants identified as Caucasian/white (43.1%) and only 13.7% identified as Hispanic/Latino(a). The majority identified as heterosexual (82.8%) and 91.4% were in their 4th year of college or below. Full sample statistics and demographic information of the final sample can be found in Table 1.

**Procedures**

The participation time for the study was 12 days in total. The study was organized into three sequential phases as follows:

- **Phase 1**: Baseline survey (1 day)
- **Phase 2**: Ecological momentary assessment of eating disorder symptoms (10 days)
- **Phase 3**: Final survey (1 day)

**Phase 1.** Phase 1 of the study consisted of informed consent, baseline data collection, and instructions for downloading and using the mobile phone application utilized in Phase 2 of the study. The online survey software *Qualtrics* was utilized for all steps in phase 1 of the study. Participants were first directed to an electronic informed consent document housed in *Qualtrics*. Upon verification of consent to participant in the current study, participants were electronically enrolled in the study. Demographic data and baseline measures of eating disorder symptom severity and mindfulness constructs were collected. Following completion of the baseline surveys, participants were provided electronically with instructions on how to download and use the mobile phone application (*Instant Survey*) for Phase 2 of the study. The application assigned a unique ID number to each participant, and participants were instructed to input this ID number into *Qualtrics* in order to link their baseline data with the data collected during phase 2 of the study. Phase 1 was marked complete upon entering the unique ID number into *Qualtrics*, and
participants were informed that phase 2 of the study would begin at 9am the following day. A
time-stamped log of participant information and ID numbers were kept in a database on a
password-protected computer for the duration of the study. Phase 1 was completed 100%
electronically.

**Phase 2.** Phase 2 consisted of ecological momentary assessment of eating disorder
symptoms. Data were collected over the course of 10 days. To collect the data, the study used
*Instant Survey*, an ecological momentary assessment data collection application compatible with
both Android and Apple mobile phones. *Instant Survey* sounded an alarm eight times per day on
a fixed basis and ask participants to complete a brief, 1-minute questionnaire that asked
questions about their present-moment emotions, cognitions, and behaviors related to disordered
eating. Thus, there was a total of 80 assessments over the course of 10 days. The application
sampled participants between the hours of 9am and 9pm. Research staff tracked adherence daily
and contacted individuals via email who responded to less than 50% of the prompts by day 2 (N
= 6). Data that were collected using *Instant Survey* were stored in a secure electronic
environment with the first layer of protection being an SSL certificate and the second layer of
protection being MD5 and base64_encode data encryption. Phase 2 of the study was completed
100% electronically.

**Phase 3.** This phase of the study involved participants completing a final questionnaire
measuring the same constructs as phase 1 as well as a participant feedback survey related to the
overall study experience. The purpose of the feedback survey was to screen for participants who
may have encountered technical issues during phase 1 or phase 2 of the study that may have
compromised the quality of their data. Phase 3 took place 100% electronically and was delivered
via Qualtrics. Completion of this final questionnaire marked the end of phase 3 and the end of the study.

Importantly, up to $45 in compensation was provided for participation in the study. Compensation was determined on an individual basis and was based on compliance during phase 2 of the study. Participants who responded to 80% or more of the daily prompts were fully compensated for their participation. Participants who respond to less than 80% of the daily prompts received a compensation amount based on their percentage of responding. Specifically, 10 dollars was subtracted from the full compensation amount for every 10% under the 80% adherence cutoff (e.g., $35 for a 70% or above response rate, $25 for a 60% or above response rate, and so on). Compensation was funded by a Graduate Dissertation Research Grant awarded by the Office of the Provost at Northeastern University.

Measures

Demographic information. Basic demographic information was collected, including data related to participant age, BMI, height and weight, and race/ethnicity (African American/black; Asian; Caucasian/white; Hispanic/Latino(a); Native Hawaiian or other Pacific Islander; American Indian or Alaska Native).

Daily eating disorder symptoms. Thirteen items were used to assess cognitions, emotions, and behaviors related to disordered eating (hereby referred to as eating disorder symptoms) throughout the course of a day. Items that were included in the study were chosen in an attempt to capture the dimensional aspect of disordered eating, rather than simply to identify pathology. For example, an item measuring the construct food intake was included as a normative proxy for binge eating and behavioral restriction in order to capture the dimensional nature of these typically pathological constructs. Items were created specifically for the current
study and were informed by previous eating disorder network studies utilizing non-DSM, dimensional measures of eating disorder symptom constructs (DuBois, Rodgers, Franko, & Robinaugh, 2016). This approach was further justified by prior research indicating that non-DSM symptoms that nonetheless represent noteworthy components of clinical psychopathology models tend to emerge as highly important in the resulting disorder network (Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016).

Ten of these items directly asked the participant to rate how they were feeling at the moment of assessment using a 5-point scale from “0 - Not at all” to “4 - Very much.” Specifically, these items measured the following constructs: perception of control (“I feel out of control”); fear of gaining weight (“I feel afraid of gaining weight”); body dissatisfaction (“I feel dissatisfied with the way my body looks”); preoccupation with thinness (“I am thinking about being thin”); negative thoughts about oneself (“I feel happy with who I am”; reverse coded and labeled in the network as “self-perception”); hunger (“I feel hungry”); negative emotions (“I feel negative emotions”); behavioral control in the context of eating (“I have control over my eating”; labeled in the network as “dietary control”); desire to be thin (“I need to be thin”); and dietary restraint (“I am consciously trying to restrict [cut back] the overall amount that I eat or drink”).

The remaining three items asked the participant to rate their feelings or behaviors since the last assessment using a 5-point scale from 0 to 4 (anchors are item-dependent). Specifically, these items measured the following constructs: weight- or shape-related energy expenditure (“I have engaged in behaviors in order to lose calories or influence my weight or shape [not at all - very much so]”); perception of weight change (“My weight has…[gone down; stayed the same; gone up]”); and food intake (“I have consumed food or drink…[not at all – a large amount]”).
**Daily mindfulness.** State levels of mindfulness were measured throughout the course of each day using three items taken from a previous study on the ecological momentary assessment of mindfulness (Gotink et al., 2016). The items asked the participant to rate how they were feeling at the moment of assessment using a 5-point Likert scale from “1 – not at all” to “5 – very much so.” Items included “I am paying attention to my feelings and sensations in my body” (assessing mindfulness of the present-moment), “I am trying to ignore or avoid my thoughts and feelings” (assessing experiential avoidance), and “I am noticing my thoughts and feelings without having to react to them” (assessing acceptance).

**Eating disorder symptom severity.** Global levels of eating disorder symptom severity were measured during phase 1 and phase 3 of the study using the Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000). The full measure included items assessing DSM-5 eating disorder criteria and used different response formats depending on diagnostic criteria (ranging from binary to Likert-type). Following guidelines from Stice et al., 2000, items were standardized to control for the different response formats and summed to create a composite score of eating disorder symptom severity, with higher scores indicating greater eating disorder symptom severity. Along with its clinical use, the EDDS has been successfully used in non-clinical settings as a tool for screening eating disorder risk in sub-clinical populations (Stice, Fisher, & Martinez, 2004). The EDDS has displayed adequate convergent and discriminant validity (Stice et al., 2004) and the internal consistency of the scale has been shown to be good with Cronbach’s alpha ranging from 0.80 to 0.86 among clinical and sub-clinical samples, respectively (Krabbenborg et al., 2011). In the current study, Cronbach’s alpha was 0.83.

**Trait mindfulness.** The four subscales of the Kentucky Inventory of Mindfulness Skills (KIMS; Baer et al., 2004) were used as multidimensional measures of mindfulness. The KIMS is
a 39-item inventory used for the assessment of mindfulness skills. The four subscales include observing (noticing or attending to various stimuli including internal and external phenomena), describing (labeling or noting of observed phenomena by applying words to them in a non-judgmental way), acting with awareness (being attentive and engaging fully in one’s current activity; hereby referred to solely as “awareness” for simplicity), and acceptance (allowing reality to be what it is without judging, avoiding, changing, or escaping it). Items are rated on a 5-point Likert-type scale ranging from “1 – Rarely” to “4 – Almost always” with higher scores indicating greater magnitude of each facet of mindfulness. Internal consistency of the subscales has been shown to be acceptable to good with a Cronbach’s alpha ranging from 0.65 (observing subscale) to 0.86 (awareness subscale; Baer et al., 2004). In the current sample, Cronbach’s alpha was 0.73 for the observing subscale, 0.73 for the awareness subscale, 0.89 for the accepting subscale, and 0.88 for the describing subscale.

**Body Image Flexibility.** Body image-specific assessments of acceptance and mindfulness may better capture the salutary processes in AMTs for eating disorders when compared to general measures of acceptance and mindfulness. For example, given the importance of overvaluation of weight and shape in eating disorder psychopathology (Fairburn et al., 2003; Murphy et al., 2010), accepting one’s body shape and weight may be particularly difficult among this population when compared to acceptance of internal states more generally. Thus, the Body Image Acceptance and Action Questionnaire (BIAAQ; Sandoz, Wilson, Merwin, & Kellum, 2013) was included as a supplemental measure of the extent to which an individual exhibits a mindful and accepting posture toward negative thoughts and feelings about his or her body shape and/or weight. The measure included 29 items that were rated on a 7-point Likert-type scale ranging from “1 – Rarely” to “4 – Almost always” with higher scores indicating
greater body image flexibility. Internal consistency among a sub-clinical sample has been shown to be good with a Cronbach’s alpha of 0.93 (Sandoz et al., 2013). In the current sample, Cronbach’s alpha was 0.88.

**Usability of Instant Survey.** The System Usability Scale (SUS; Kirakowski & Corbett, 1988) was used to obtain a global view of subjective assessments of usability related to *Instant Survey*. The SUS is a ten-item scale with items rated on a 5-point Likert scale from “1 – Strongly Disagree” to “5 – Strongly Agree.” The scale allows for the calculation of a composite usability score, as well as using each item as a separate aspect of usability. A composite score was not used for the current study because considering individual items rather than a composite score appeared to have higher face validity.

**Data Analytic Plan**

The data analysis was carried out in two stages. The purpose of the first stage was to create the group- and individual-level eating disorder symptom networks. During this stage, the time-series data collected via ecological momentary assessments were analyzed in order to create symptom networks depicting the within-subject and time-variant relationships among eating disorder symptoms. Following that, the second stage of the analysis comprised of using these symptom networks for cross-sectional mediation analysis to test the second research hypothesis.

**Network construction.** At the group level, each node in the network represented a specific eating disorder symptom, and each edge in the network represented the average time-lagged effect of one symptom on another. Given that EMA data violate the assumption of independence of observation, a multilevel approach to the symptom network construction was used. Specifically, a series of multilevel analyses was conducted whereby each symptom $k$ at time $t$ was predicted by all other symptoms $j$ (including itself) at time $t - 1$. This resulted in 13
separate multilevel models, each representing the time-lagged effects of all symptoms on one of the 13 symptoms. Given the exploratory nature of the study, the p value was not adjusted for multiple testing. For example, Equation 1 depicts the multiple regression of an outcome variable k (hunger in this example) for person i at time point t on all other variables j at t – 1 (only four variables depicted in equation for simplicity).

(Equation 1; modified from Bringmann et al., 2015)

\[ \text{Level 1: } \text{Hunger}_{it} = \beta_0 + \beta_{1i}(\text{negative emotions}_{i,t-1}) + \beta_{2i}(\text{self control}_{i,t-1}) + \beta_{3i}(\text{cognitive restraint}_{i,t-1}) + ... + \beta_{13i}(\text{restriction}_{i,t-1}) + \epsilon_{it} \]

Level 2: \[ \beta_{1ij} = \gamma_{ij} + r_{ij}, \]
\[ \beta_{2ij} = \gamma_{ij} + r_{ij}, \]
\[ \beta_{3ij} = \gamma_{ij} + r_{ij}, \ldots \]
\[ \beta_{13ij} = \gamma_{ij} + r_{ij} \]

where \( \epsilon_{it} \sim N(0, \sigma^2) \) and \( r_{ij} \sim N(0, +\sigma_{ij}) \)

A 13 x 13 matrix of the fixed effects was then used as an adjacency matrix to construct an eating disorder symptom network, with each element in the matrix modeled as an edge in the network. The elements \( \beta_{kj} \) represent the extent to which variable j at time t – 1 is related to variable k at time t, while controlling for all other variables. The diagonals \( \beta_{kk} \) of the matrix represent each symptom’s autoregressive effects (i.e., the impact of each symptom on itself over time). Furthermore, the person-specific deviations from the average effects were captured by \( r_{ij} \), and were assumed to come from a multivariate normal distribution. All predictors in the analyses
were person-mean centered, and only edges that were significant at the $p = 0.05$ level were included in the network. The resulting population-level network captured the average influence of the eating disorder symptoms on each other across time. This analysis has since been referred to as a *multilevel vector auto-regressive* (mlVAR) model and has been utilized in a number of psychopathology network studies (Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015; Bringmann et al., 2016; Pe et al., 2015).

Although longitudinal data were utilized for the population-level network construction, it should be noted that the current study’s aims are between-subject in nature and thus inter-individual analyses were not directly used for hypothesis testing. Rather, the population-level network derived from symptom associations across time was utilized as a static model to compute a continuous variable representing the overall connectivity among symptoms for each participant. Thus, each individual in the study had one value representing her overall symptom connectivity as measured across the 10 days of analysis.

In order to calculate the network connectivity for each participant in the study, a separate eating disorder network was created for each individual. Nodes in the network were represented by eating disorder symptoms, and edges were calculated by adding each individual’s random effect value to the fixed effect (i.e., average) of a given edge in the population-level network. For example, the edge from symptom $j$ to symptom $k$ would have the value of $\beta_{jk} + r_{jk}$. Then, the average of the absolute values of the regression weights for each individual was calculated. This average edge value represented the network connectivity for each participant and was utilized as a continuous variable for between-subjects hypothesis testing.

**Preliminary data considerations.** The multilevel model used to analyze the longitudinal data assumed that the error terms were normally distributed and homoscedastic. This assumption
was examined by inspecting the residual versus fitted plots for the error terms of data from phase 2 of the study (Singer & Willett, 2003). It was also assumed that observations were not independent of one another. Given the hypothesized influence of symptoms across time (e.g., restriction at \( t_1 \) causes binge eating at \( t_2 \)), this assumption was likely met. The distributions of the Level 1 variables were assumed to be linear. Considering that the multilevel model was able to handle an unbiased design (i.e., unequal spacing of observations between individuals, as long as the missing-ness is assumed to be random), the study did not impute missing data values for the longitudinal data. Lastly, the network analysis does not allow for trends in the data. A thorough investigation and summary of all data analytic assumptions is presented in chapter 4.

Power analyses for multilevel models are notoriously ambiguous and depend on a number of factors such as the intra-class correlation coefficient and strength of associations among variables (Raudenbush & Bryk, 2001). Assessing adequate observations and sample size for longitudinal network models is no exception. Considering the nested format of the data, power must be determined at two levels: the number of time points per person at Level 1 and the number people at Level 2. Contemporary research from psychological network science has assessed both the number of time points and the number of people required to reach adequate power using a number of different methodologies (Epskamp, 2016; Epskamp & Fried, 2016; Fried, 2016). While a full discussion is beyond the scope of this chapter, research on Level 1 power has indicated that when using roughly 10 nodes, at least 30 observations will result in adequate network parameter specificity and sensitivity and detection of statistical significance. Furthermore, general multilevel modeling guidelines assert that a sample of 30 or more is considered “large” (Snijders & Bosker, 1999). In contrast, the current study utilized up to 80
observations per individual, with a final sample size of 58 participants. Thus, the current study met adequate power requirements to detect statistical significance.

**Hypothesis 1.** In order to examine the association between baseline levels of eating disorder symptom severity and symptom connectivity, composite scores on the EDDI were correlated with individual network connectivity using a Pearson correlation. Percentile bootstrapping (N=1000) was used to determine the 95% confidence interval of the correlation. The analysis controlled for age and BMI using semi-partial correlations, and the distributional qualities of the data were observed to determine the type of correlative analysis that was used. Of note, previous research investigating the association between individual network connectivity and psychopathology found that network connectivity was normally distributed among a college sample (Bringmann et al., 2016).

**Hypothesis 2.** A mediation approach was utilized to examine if symptom connectivity accounted for the relationship between mindfulness and eating disorder symptom severity. Zero-order correlations were calculated among the study variables prior to the mediation analysis in order to explore the presence and magnitude of global associations among the constructs. Significant bivariate correlations among the study variables represented a prerequisite for mediation analyses. This design allowed for up to five separate mediation analyses dependent on the number of significant bivariate relationships that emerge. A different facet of mindfulness (awareness, acceptance, observing, describing, body image flexibility) acted as the independent variable in each of the mediation analyses. All analyses included symptom connectivity as the mediating variable and symptom severity as the dependent variable. This approach to mediation was utilized because the different facets (i.e., subscales) of mindfulness have been shown to have
different associative properties and clinical implications with regard to eating disorders (Baer, 2006; Baer et al., 2005) and psychopathology in general (Kabat-Zinn, 1994).

The percentile bootstrap confidence interval (CI) method was used for testing the mediation models as a whole, as well as the indirect effect of mindfulness on symptom severity through symptom connectivity (Preacher & Hayes, 2004). This method of mediation analysis was chosen primarily due to its reliable performance with small sample sizes (Hayes & Scharkow, 2013). Equation 2 depicts the regression models used for the bootstrapping CI method.

(Equation 2)

Model 1: \( \Delta \text{Severity} = \beta_0 + \beta \Delta \text{Mindfulness} + e \)
Model 2: \( \Delta \text{Severity} = \beta_0 + \beta_1 \Delta \text{Mindfulness} + \beta_2 \Delta \text{Connectivity} + e \)

Using this method, 5,000 bootstrapped samples were utilized to create a sampling distribution of the indirect effects for both models, defined as \( \beta - \beta_1 \). The 2.5 and 97.5 quantiles of these distributions will define the lower and upper bounds of the 95% confidence interval for each indirect effect. Symptom connectivity was identified as mediating the relationship between mindfulness and symptom severity only if the 95% CIs for the indirect effects did span zero. The PROCESS plugin was used for all mediation analyses (Hayes, 2012).
CHAPTER 4

RESULTS

Preliminary Data Analyses

Survey variables. The distribution of EDDS scores was slightly positively skewed (skewness = 0.621), which is to be expected from a sub-clinical college population. Likewise, the mean score for the EDDS was 24.7 (SD = 10.2), indicating sub-threshold eating disorder symptom endorsement. Only one participant in the study reported having received an eating disorder diagnosis in the past. All mindfulness and acceptance-based measures were normally distributed. Table 2 and Figure 4 depict the descriptive statistics and distribution of all survey variables, respectively.

Daily eating disorder symptoms. A total of 4,056 momentary ratings were completed during Phase 2 of the study. On average, participants completed 71 out of the 80 total EMA prompts (87%), indicating good compliance across the sample (Sonnenschein et al., 2007). Furthermore, 91% of participants rated the EMA application as moderately to extremely simple to use, 90% reported that it was moderately to extremely easy to learn how to use, and 78% reported that they felt moderately to extremely confident using the application.

The EMA application did not allow for missing data within samples, resulting in 0% missing data for surveys that were completed. The assumption of normally distributed residuals (observed value minus predicted value) was checked by creating quantile-quantile plots (i.e., normal probability plots) of the residuals for each separate multilevel model. Results did not raise any significant concerns related to the normality of the weighted residuals. Furthermore, residual plots were used to ensure the homoscedasticity of error terms, and indicated generally consistent variance across the fitted range. EMA items were plotted across time to visually
inspect for trends in the data, and none were identified. Given that the mlVAR model is able to handle unbiased designs (i.e., unequal spacing of observations between individuals, as long as the missing-ness is assumed to be random), no corrections were made for missing prompts.

Figure 2 depicts the time-independent distribution of each EMA item across the sample.

**Hypothesis 1: The Relationship between Symptom Connectivity and Symptom Severity**

It was hypothesized that greater levels of symptom network connectivity would be related to greater levels of eating disorder symptom severity. An individual symptom connectivity score was calculated for each participant in the study using the methods described in chapter III. Figure 8 provides a visual example of these N=1 symptom networks using two randomly selected study participants. The sum of the absolute values of all edges in the network was calculated for each participant network, and the resulting symptom connectivity was normally distributed across the sample with a mean of 5.7 and standard deviation of 1.0. Each participants’ unique symptom connectivity score was subsequently used for the following analyses.

A zero-order correlation between symptom connectivity and symptom severity was conducted as a preliminary step to investigate the relationship between these two constructs. Percentile bootstrapping (N=1000) was used to determine the 95% confidence interval of the correlation. Results from the correlation analysis revealed a significant positive relationship between symptom connectivity and symptom severity (Pearson $r = .48$, $p < 0.01$, 95% CI [0.285 – 0.650]).

Post-hoc hierarchical linear regression was used to investigate the unique variance of symptom severity explained by symptom connectivity above and beyond other clinically-relevant variables. Two separate three-stage analyses were conducted. One analysis controlled for mindfulness as measured by the four subscales of the KIMS, while the other model controlled
for body image flexibility as measured by the BIAAQ. In the first analysis (“regression analysis 1”), participant age and BMI were entered at stage one as control variables, the four subscales of the KIMS were entered at stage two, and symptom connectivity was entered at stage three. In the second analysis (“regression analysis 2”), participant age and BMI were entered at stage one, body image flexibility as measured by the BIAAQ was entered at stage two, and symptom connectivity was entered at stage three. The composite score of the EDDS was used as the dependent variable for both analyses. Figure 9 depicts the bivariate relationships among all study variables included in this analysis.

Results of regression analysis 1 and 2 are depicted in Tables 3a and 3b. In regression analysis 1 (model 1 = age; BMI; model 2 = KIMS subscales; model 3 = connectivity), the final model explained 35% of the variance of symptom severity, $R^2 = 0.35$, $F(1,50) = 3.81$, $p < 0.01$. Only the acceptance subscale was a significant mindfulness-based predictor of symptom severity ($\beta = -0.38$, $p < 0.05$) and symptom connectivity significantly predicted symptom severity above and beyond age, BMI, and acceptance ($\beta = 0.41$, $p < 0.01$). Furthermore, approximately 12% of the variance of symptom severity was uniquely accounted for by symptom connectivity, as evidenced by a significant increase in $R$-squared of 0.13 when connectivity was added to the model, $F(1,50) = 9.92$, $p < 0.01$.

In regression analysis 2 (model 1 = age; BMI; model 2 = BIAAQ; model 3 = connectivity), the final model explained 43% of the variance of symptom severity, $R^2 = 0.43$, $F(1,53) = 9.94$, $p < 0.01$. Body image flexibility was a significant negative predictor of symptom severity, ($\beta = -0.447$, $p < 0.01$) and symptom connectivity significantly predicted symptom severity above and beyond age, BMI, and body image flexibility ($\beta = 0.31$, $p < 0.05$). Furthermore, approximately 8% of the variance of symptom severity in regression analysis 2 was
uniquely accounted for by symptom connectivity, as evidenced by a significant increase in R-squared of 0.08 when connectivity was added to the model, \( F(1,53) = 7.11, p < 0.05 \).

**Hypothesis 2: Mediation Analysis**

The current study also hypothesized that symptom network connectivity would mediate the relationship between mindfulness and eating disorder symptom severity. Two parallel mediation analyses were conducted based on the two different acceptance and mindfulness-based measures included in the study. This was done because the prerequisite of significant bivariate relationships was not met for all subscales of the KIMS. Specifically, the acceptance subscale was the only subscale of the KIMS that was significantly related to either symptom connectivity (\( r = -0.307, p < 0.05 \)) or symptom severity (\( r = -0.353, p < 0.05 \)). Thus, the three remaining subscales of the KIMS were omitted from subsequent analyses.

The first mediation model (“mediation analysis 1”) consisted of acceptance, as measured by the KIMS, as the independent variable, symptom severity as the dependent variable, and symptom connectivity as the mediating variable. The second mediation model (“mediation analysis 2”) consisted of body image flexibility, as measured by the BIAAQ, as the independent variable, symptom severity as the dependent variable, and symptom connectivity as the mediating variable. BMI was included in both models as a covariate, and 5,000 bootstrapped samples were used to calculate the 95% confidence interval of the direct and indirect effects.

Results of mediation analysis 1 (\( X = \text{acceptance}; M = \text{connectivity}, Y = \text{symptom severity} \)) indicated that the relationship between acceptance and symptom severity was partially mediated by symptom connectivity. The unstandardized regression coefficient between acceptance and symptom connectivity was significant (\( B = -0.04, p < 0.01, 95\% \ CI [-0.07, -0.012] \)) as was the unstandardized regression coefficient between symptom connectivity and
symptom severity ($B = 3.39, p < 0.01, 95\% \text{ CI} [0.99, 5.78]$). Approximately 30\% of the variance of symptom severity was accounted for by the predictors, $R$-squared $= 0.305, F (2,55) = 6.48, p < 0.01$. The direct effect of acceptance on symptom severity was -0.323, and the indirect effect was -0.149. A bootstrap estimation approach with 5,000 samples was used to test for statistical significance. Results indicated that this indirect effect was significant ($95\% \text{ CI} [-0.31, -0.02]$), thus supporting the mediation hypothesis for mediation analysis 1.

Results of mediation analysis 2 ($X =$ body image flexibility; $M =$ connectivity, $Y =$ symptom severity) indicated that the relationship between body image flexibility and symptom severity was also partially mediated by symptom connectivity. The unstandardized regression coefficient between body image flexibility and symptom connectivity was significant ($B = -0.04, p < 0.01, 95\% \text{ CI} [-0.07, -0.01])$, as was the unstandardized regression coefficient between symptom connectivity and symptom severity ($B = 2.76, p < 0.05, 95\% \text{ CI} [0.57, 4.96]$). Approximately 42\% of the variance of symptom severity was accounted for by the predictors, $R$-squared $= 0.42, F (2,55) = 6.38, p < 0.01$. The direct effect of acceptance on symptom severity was -0.516, and the indirect effect was -0.114. Again, a bootstrap estimation approach with 5,000 samples was used to test for statistical significance. Results indicated that this indirect effect was significant ($95\% \text{ CI} [-0.29, -0.01]$), supporting the mediation hypothesis for mediation model 2.

**Exploratory Network Analysis:**

**Network structure:** The inferred eating disorder symptom network is depicted in Figure 5. Each symptom is represented as a single node in the network. The relationships among symptoms are depicted by weighted arrows between symptoms (connection strength as indicated by $\beta_{kj}$), and represent the strength of the relationships between a given symptom at time $t - 1$ and all other symptoms, including itself, at time $t$. Red arrows indicate negative relationships,
and green arrows indicate positive relationships. All arrows depicted in Figure 5 were significant at the $p < 0.05$ level.

All symptoms were significantly connected within the network. Self-perception (positive/negative thoughts about oneself) was the only symptom that was connected to the network exclusively through incoming arrows, indicating that this symptom held no significant $t - 1$ relationships with any other symptoms at time $t$. Similarly, dietary control was the only symptom that was connected to the network exclusively through outgoing arrows, indicating that no other symptoms in the network at $t - 1$ held a significant relationship with this symptom at time $t$.

Autoregressive effects (i.e., self-loops) were among the strongest connections in the network, suggesting that the current experience of any given symptom is generally the strongest predictor of the future occurrence of that symptom. This effect was most notable for negative emotions, preoccupation with thinness, body dissatisfaction, and dietary restraint, and was least notable for eating behavior, perception of weight change, and self-perception. Furthermore, bi-symptom (e.g., symptom $k \leftrightarrow$ symptom $j$) and tri-symptom (e.g., symptom $k \rightarrow$ symptom $j \rightarrow$ symptom $i \rightarrow$ symptom $k \rightarrow ...$) feedback loops represent important structural relationships in network psychopathology theory (Borsboom & Cramer, 2013) as well as in cognitive-behavioral theories of eating disorders (Baer et al., 2005; Fairburn, Cooper, & Shafran, 2003; Murphy, Straebler, Cooper, & Fairburn, 2010). In the current network, five bi-symptom feedback cycles and six tri-symptom feedback cycles were identified. Figures 6a and 6b depict the graphical representations of the identified bi-symptom and tri-symptom feedback loops, respectively.

**Symptom centrality:** Symptom centrality is depicted in Figure 7. Out-strength centrality refers to the average magnitude of the outward-directed arrows for any given symptom in a
network and conceptually represents the associative influence of each symptom in predicting future network activation. Symptoms with high out-strength centrality are most predictive of future network activation. In the current network, hunger and dietary control displayed the highest out-strength centrality, highlighting their important role as potential time-variant precipitating factors for network activation. Conversely, self-perception had the lowest out-strength centrality, suggesting that this construct is relatively unrelated to the future presence or absence of eating disorder symptoms.

In-strength centrality refers to the average magnitude of the inward-directed arrows for any given symptom in a network and conceptually represents the associative influence of the network as a whole in predicting future activation of a given symptom. Network theory posits that symptoms with high in-strength centrality are strongly influenced by the network as a whole. In the current network, negative emotions and eating behavior (as measured by food intake) displayed the highest in-strength centrality, indicating that global network activation at any given time is highly predictive of later negative emotions and changes to one’s eating patterns. Dietary control had the lowest in-strength centrality, followed closely by perception of weight change.

Lastly, betweenness centrality for a given symptom refers to the number of instances that the symptom lies on the shortest path between two other symptoms in a network and conceptually represents the extent to which each symptom mediates the spread of activation within a network. Often referred to as bridge symptoms, symptoms with high betweenness centrality act as a gateway between different symptom clusters (i.e., subsets) within a network. In the current network, negative emotions and hunger displayed the highest betweenness centrality, suggesting that these constructs mediate the symptom-to-symptom relationships within the
network to the largest degree. Conversely, dietary control, weight change, and self-perception displayed the lowest betweenness centrality.
CHAPTER 5

DISCUSSION

The present study utilized a network approach to elucidate the patterns of relationships among eating disorder symptoms, and to examine how the strength of these relationships related to mindfulness and eating disorder psychopathology. The initial portion of this chapter will synthesize the present results regarding the associative relationship between symptom connectivity, severity, and mindfulness, and the potential role of symptom connectivity as it relates to AMTs. Considering that symptom connectivity is a construct derived from hypothesized relationships among the symptoms at hand, an extended discussion connecting these results to the specific patterns of associations identified within the symptom network will follow.

The Relationship between Symptom Connectivity and Symptom Severity

The present study hypothesized a positive relationship between symptom connectivity and symptom severity. More specifically, it was hypothesized that individuals with higher moment-to-moment connectivity among symptoms would also have elevated eating disorder symptom severity when compared to individuals with lower moment-to-moment symptom connectivity. Preliminary results confirmed this hypothesis by identifying a moderate significant positive correlation ($r = .483$, $p < 0.05$) between symptom connectivity and symptom severity. Furthermore, post-hoc regression analyses revealed that symptom connectivity predicted eating disorder symptom severity above and beyond participants’ age, BMI, and trait levels of acceptance-focused facets of mindfulness.

To our knowledge, this is the first study to date investigating the relationship between these important constructs. Considering the compelling results of recent network studies
identifying symptom connectivity in other mental health conditions as a significant risk factor for poor prognosis (Pe et al., 2015; van Borkulo et al., 2015), a concurrent indicator of disorder severity (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017), and an overall vulnerability factor for psychopathology in general (Borsboom & Cramer, 2013; Fried, van Borkulo, Cramer, Lynn, Schoevers, & Borsboom, 2017), the current results aid in advancing the network approach to eating disorder psychopathology by providing the first evidence for the relationship between individual symptom connectivity and eating disorder symptom severity. The network approach places a strong emphasis on the mutual inter-relationships among symptoms as the primary mechanism by which mental disorders arise and are maintained. As such, the positive relationship found between symptom connectivity and severity acts as a crucial first step in validating the network theory at the individual level. In fact, one of the primary suggestions for future research in network analysis is to use hypothesis-driven research designs to test and confirm overarching theories related to the network approach, rather than the exploratory methods that are commonplace in previous network analysis research (Fried et al., 2017).

More broadly, symptom connectivity and symptom severity may constitute orthogonal dimensions of psychopathology. This is because both constructs are conceptually and statistically distinct from one another, yet together represent the two components that constitute a network: nodes (symptom severity) and edges (symptom connectivity). From a conceptual standpoint, an individual can have low symptom severity and yet still have a highly connected symptom network. According to network theory, this combination would represent elevated risk for the future development of an eating disorder because the experience of any one symptom would have a high likelihood of leading to the experience of many symptoms and activating the
network as a whole. In fact, a number of simulation studies have confirmed this risk profile (Borsboom & Cramer, 2013; Fried et al., 2017).

Furthermore, the statistical methodology for calculating symptom severity parallels this conceptual understanding of connectivity as an orthogonal dimension of psychopathology. This is because the bivariate relationships that are used to calculate symptom connectivity are derived from symptom variance, not symptom means. In this way, an individual could statistically exhibit low symptom severity and elevated symptom connectivity assuming that the distributional properties of the symptoms are generally similar across the severity continuum (i.e., no floor or ceiling effects). This idea is particularly important given that the current study utilized a non-clinical sample. It may be that individuals in the current study who were identified as having high symptom connectivity yet who did not meet a clinical eating disorder cutoff score may nonetheless possess a clinically significant level of symptom connectivity that puts her at a heightened risk for the future development of an eating disorder. However, future research investigating symptom network risk profiles such as this are required to confidently make such claims.

Although subtle, this differentiation has important implications for the study of eating disorder psychopathology because it provides a novel avenue for researching certain features of eating disorder psychopathology at the sub-clinical level. First, it may be that symptom network structure remains stable across clinical and sub-clinical populations due to the omission of symptom means when calculating these networks. If this is the case, researchers may be able to study certain aspects of eating disorder psychopathology, such as symptom centrality, without the requirement of obtaining a clinical sample. Although this is certainly a promising area of study, more research is needed to fully understand the similarities and differences between sub-
clinical and clinical eating disorder networks. Second, symptom connectivity may represent an early warning sign for the development of an eating disorder before the emergence of elevated symptomatology. For example, a phenomenon known as *critical slowing down* has recently been investigated as an early warning sign for the development of psychopathology (Olde Rikker, 2018; Wichers et al., 2016). According to the theory, the transition from a healthy mental state to a mental disorder is marked by a tipping point. This tipping point, known as critical slowing down, is characterized by certain patterns of changes in the connectivity among symptoms and precipitates the full activation of a symptom network. In this way, critical slowing down may be a viable solution for detecting the development of an eating disorder before it happens. This ability would be critically important to the field of eating disorders because improvements in early detection and prevention have been identified as one of the most pressing areas for future research of eating disorders (Stice, Becker, & Yokum, 2013; Stice, South, & Shaw, 2012; Wilksch, 2015). Importantly, the current study sheds preliminary light on this possibility by establishing a method for analyzing the complex relationships among eating disorder-related symptoms in a non-clinical sample. Only when we fully understand these symptom dynamics at a sub-clinical, baseline level can we begin to investigate the structural and functional patterns, such as critical slowing down, that precipitate an eating disorder episode.

**Mindfulness, Symptom Severity, and the Mediating Role of Connectivity**

Preliminary research suggests that AMTs show promise as an effective group of treatments for eating disorders and sub-clinical disordered eating (e.g., Baer, 2003; Baer, Fischer, & Huss, 2005) despite the fact that these protocols advocate against overt attempts to decrease symptoms. Although the process that accounts for the relationship between mindfulness and symptom severity is largely unknown, a recent theory regarding the mechanism of change in
AMTs, referred to as decoupling, has been postulated. However, no prior research has investigated the associative role of symptom connectivity in relation to the relationship between mindfulness and disorder severity. The present study hypothesized symptom connectivity as a mediator of this relationship given that connectivity has been identified as both a treatment target congruent with core AMT principals as well as an indicator of disorder severity and prognosis (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Pe et al., 2015; van Borkulo et al., 2015).

Results from the mediation analyses confirmed this hypothesis by showing that symptom connectivity partially mediated the relationship between symptom severity and mindfulness as measured by acceptance-focused definitions only. Interestingly, acceptance-focused measures of mindfulness (as measured by the BIAAQ and the acceptance subscale of the KIMS) were the only constructs significantly associated with either symptom severity or symptom connectivity, and the describing, awareness, and observing subscales of the KIMS held no significant relationships with severity or connectivity. This finding highlights the subtle yet clinically-relevant distinctions among the different facets of mindfulness.

Particularly in the context of symptom connectivity, this differentiation emphasizes the unique role of acceptance as it relates to the maladaptive cognitive-behavioral-affective cycles that are hypothesized to account for the onset and maintenance of eating disorders. From an AMT perspective, the relationship (or lack thereof) from one given symptom to another is defined in part by the function that is derived from that relationship. For example, experiential avoidance is hypothesized to be a trans-diagnostic risk factor that contributes to eating disorder psychopathology in general, and binge eating disorder in particular (Baer et al., 2005; Kristeller, 1999; Kristeller, Wolever, & Sheets, 2013). In the context of experiential avoidance, overt behaviors such as binge eating arise, in part, as an attempt to avoid or numb emotional pain, but
paradoxically tend to heighten emotional pain over time (Kristeller & Wolever, 2010). Thus, a primary focus of AMTs for binge eating disorder is to combat experiential avoidance by fostering an accepting and open stance toward emotional pain, thereby decoupling this mutually-reinforcing and maladaptive affective-behavioral cycle. In this way, it is reasonable to conclude that describing, observing, and being aware of emotional pain might not be sufficient to stop this cycle given that one could do all those things (i.e., describe, observe, and become aware of the emotional pain) and still want to rid themselves of the experience. However, acceptance implies a unique *willingness* to experience emotional pain without having to react in any particular way, and thus may constitute a particular facet of mindfulness that is particularly relevant to symptom connectivity.

Although the cross-sectional approach to mediation in the current study is not well suited for rigorously investigating the hypothesis of connectivity as a mechanism of change in AMTs, it nonetheless establishes important findings that give promise to such claims. First and foremost, the relationship between mindfulness and eating disorder symptom connectivity has never before been investigated from a network perspective, despite the many clinical theories and treatment protocols that emphasize such a relationship. Our results indicate that individuals with low levels of acceptance tend to have strong moment-to-moment connections among eating disorder symptoms and elevated eating disorder severity. Furthermore, the relationship between acceptance and symptom severity was partially explained by symptom connectivity, suggesting that reducing network connectivity (i.e., decoupling) might constitute the therapeutic process by which acceptance ultimately leads to symptom reduction. Although a pre-post intervention research design is necessary to fully capture the process of decoupling, the preliminary findings are nonetheless critically important for the advancement of AMTs because contemporary
theories regarding mechanisms of change in AMTs have failed to identify a theoretically-sound process that explains the link between acceptance-based practices and reductions in symptom severity. More specifically, these theories have struggled to identify a process of change that can account for how a practice that actively advocates against reducing symptoms can reliably and consistently reduce symptoms. Our findings identifying symptom connectivity as a potential mechanism accounting for this paradoxical relationship are a critically important preliminary step in the development of AMTs. This is because symptom connectivity provides a novel outcome measure for assessing the effectiveness of acceptance- and mindfulness-based intervention that is based in contemporary psychopathology theory, known to be associated with symptom severity, and yet also congruent with the core acceptance-based core beliefs inherent to AMTs.

The Eating Disorder Symptom Network

Despite the fact that all cognitive-behavioral theories of eating disorders rest on the assumption that time-variant relationships exist between eating- and body image-related cognitive, affective, and behavioral experiences, there is a paucity of research examining these complex relationships among a group of symptoms. The majority of studies utilize cross-sectional data analytic techniques which, by nature of their design, cannot capture the intra-individual processes that define these theories (Fried et al., 2017). In light of this, the current study puts forth an important contribution to the field by examining these complex patterns of relationships within at the individual level using a network approach. To our knowledge, the current study is the first within-subject network analysis of eating disorder symptoms to date.

As Figure 5 clearly depicts, all symptoms included in the study were connected to one another across time, with each symptom displaying its own unique patterns of relationships and
relative importance within the network. For example, some symptoms (e.g., dietary control) were highly predictive of the future state of many other symptoms in the network, whereas other symptoms (e.g., self-perception) appeared to be a result of the previous state of the network with little to no predictive power as an exogenous variable. This is, indeed, an important consideration because the prevailing latent-trait conceptualization of mental disorders in general, and eating disorders in particular, assumes that symptoms are independent indicators of a latent disorder and are not mutually influenced by one another (Borsboom & Cramer, 2013). Our results not only provide evidence that this is not the case, but put forth specific mechanisms by which symptoms can exist independently from a latent trait.

One such mechanisms is a feedback loop, whereby symptoms mutually reinforce one another either bi-directionally (symptom k ↔ symptom j) or in groups (symptom k → symptom j → symptom i → symptom k → …). Often referred to as cycles, feedback loops are important because their existence alone is sufficient to explain the presence of any one of their symptoms without the need for a latent trait. Furthermore, they provide the context for understanding the clinical relevance of symptom connectivity. For example, binge eating and compensatory behavior are hypothesized to exist in a feedback loop whereby binge eating leads to compensatory behavior which, in turn, leads back to binge eating, and so on (Kristeller & Wolever, 2013). In this way, individuals who predictably engage in both compensatory behavior after binge eating and in binge eating after compensatory behavior (i.e., individuals with high connectivity between binge eating and compensatory behaviors) are at higher risk for maintained eating disorder behavior because the experience of any one of these symptoms will naturally reinforce the other. In fact, many CBT (e.g., Murphy et al., 2010) and AMT (e.g., Juarascio et
al., 2013) protocols for eating disorder treatment at least partially account for these maladaptive cycles as important targets for intervention.

In the current study, a number of clinically-relevant feedback loops were identified (Figures 6a and 6b). For example, negative emotions and food intake temporally predicted one another. Specifically, the experience of negative emotions lead to a subsequent increase in food intake which, in turn, further increased negative emotions. From a network perspective, it can reasonably be assumed that feedback cycles such as this represent important behavioral patterns that maintain a disorder over time. In fact, contemporary accounts of binge eating disorder emphasize this particular feedback loop as a primary mechanism by which the disorder is maintained over time (Kristeller & Wolever, 2010; Kristeller, Wolever, & Sheets, 2013).

Similarly, a preoccupation with thinness, compensatory behavior, and hunger were found to predict each other in a temporal fashion (preoccupation → compensation → hunger → preoccupation). Tri-symptom feedback loops such as this represent more complex maladaptive cycles that span the cognitive-behavioral-affective continuum and can likewise be used to better understanding the negative effects of high symptom connectivity on disorder maintenance.

Moreover, these models provide useful information regarding how different intervention strategies might influence different aspects of the disorder at hand. For example, changing one’s appraisal of hunger cues and reducing cognitive fusion are two interventions that have been shown to support eating disorder treatment (Jaurascio et al., 2013; Kristeller, Wolever, & Sheets, 2013). It might be that interventions such as these break up different components of maladaptive cycles such that, for example, changing hunger cue interpretation targets one path of the cycle (hunger cue → preoccupation with thinness) while cognitive defusion exercises target another
(preoccupation with thinness → compensatory behavior). However, more research is certainly needed to confirm whether or not this is the case.

Another important consideration of the network structure in the current study is that of symptom centrality (see chapter IV for a definition of all centrality indices included in the analysis). Hunger and dietary control were among the symptoms with the highest out-degree centrality, meaning that their existence at a given moment in time is highly predictive of a multitude of other eating disorder symptoms being activated in the future, specifically within one and a half hours. Although dietary control is indeed included in many clinical formulations of eating disorders (Fairburn et al., 2003; Murphy et al., 2010), the role of hunger in eating disorder psychopathology is not well understood and often de-emphasized in these formulations. Our results suggest that, from a network perspective, hunger is indeed an important component in eating disorder psychopathology and may play a role in influencing affective (e.g., negative emotions), cognitive (dietary restraint), and behavioral (eating behavior) components of the disorder. Together with the direct relationships among eating disorder symptoms, hunger’s high betweenness centrality also indicates that it may also play an indirect role by mediating the spread of activation across the network as a whole.

Furthermore, cross-sectional network analyses of eating disorder symptoms among clinical samples (DuBois et al., 2017; Forbush et al., 2016) and sub-clinical (DuBois, Rodgers, Franko, & Robinaugh, 2016) tend to identify cognitions and emotions as the most central symptoms in the network, with behaviors holding more peripheral roles. Although the current results do not confirm or disconfirm these findings, they do shed light on important differences when investigating eating disorder networks at the individual level. One, behavioral symptoms in the current network ranged from low to high centrality. For example, dietary control was among
the most central symptoms, deviating from clinical cross-sectional findings that suggest this construct as low in centrality (DuBois et al., 2016). Two, the ability to infer direction of relationship in the current analysis, depicted by arrows in the network, greatly improves the ability to draw meaningful interpretations of centrality indices. For example, strength centrality from cross-sectional network analysis provides information about which symptoms hold the highest degree of association with the network but provide no information about the direction of relationship. It might be that symptoms identified as highly central in cross-sectional network analyses are highly influenced by the symptoms in the network but have no temporal influence on these symptoms. This distinction is critical because nodes identified as highly central in cross-sectional network analyses are often assumed to be influential to the disorder and thus important targets for treatment. Our results suggest that considering the direction of relationship is critically important when interpreting centrality because symptoms identified as central in cross-sectional analyses may simply represent highly predictive outcomes of the network at any given time, with poor clinical utility for intervention. Lastly, our results suggest that symptoms with low centrality may nonetheless play an important role in maintaining certain aspects of the network. For example, preoccupation with thinness was identified as low centrality in the network. However, it nonetheless plays an important role in the network because it is one of two primary symptoms that predict a major diagnostic indicator of eating disorders, that of compensatory behavior. Although some researchers have developed centrality indices to better account for these differences (e.g., expected influence; Robinaugh, Millner, & McNally, 2016), care should be taken not to over-interpret low centrality symptoms as unimportant to the disorder at hand.

Limitations and Future Directions
The current study had a number of noteworthy limitations. One, the sample used in the study was a convenience sample of college students who were not diagnosed with an eating disorder and who scored under a clinical cut-off for eating disorder psychopathology. Despite the fact that current trends in psychopathology and diagnosis emphasize a dimensional approach to mental health classification (Clark, Cuthbert, Lewis-Fernandez, Narrow, & Reed, 2017), it is unknown whether the results of the current study will generalize to a clinically diagnosed eating disorder population. Although there is a lack of research studies comparing the network structure across clinical and non-clinical populations, some researchers theorize that network structure is not highly influenced by disorder severity given that the edges (i.e., correlations) which constitute the networks are derived solely from symptom variability, not symptom means (Fried et al., 2017). In this way, using network analysis to identify similarities and differences across the spectrum of disorder severity may provide the opportunity to identify novel clues related to eating disorder risk and development without the necessary requirement of clinical populations. Notwithstanding this possibility, further research is needed to better understand the stability and generalizability of network analysis across clinical and non-clinical samples.

Two, the current study did not assess for the process of symptom decoupling as it is theorized to unfold across time, nor did it assess whether AMTs have the ability to decouple symptom networks (i.e., reduce symptom connectivity). This is an important limitation to consider given that a significant correlation between symptom connectivity and mindfulness does not infer causation. Although these results suggest that individuals high in specific mindfulness skills tend to have uncoupled symptom networks, we do not yet know whether changes in mindfulness will lead to reductions in symptom connectivity and, in turn, improve outcomes. Leveraging more rigorous research designs, such as longitudinal mediation and pre-
post intervention assessments to determine the presence of decoupling, is warranted in order to better assess the complex relationships among mindfulness, symptom connectivity, and disorder outcomes.

Third, a number of implicit assumptions inherent in the creation of the eating disorder symptom network may have influenced the results in unknown ways. For example, the symptom network captured the relationships among symptoms across the span of roughly 1.5 hours. However, it might be that the associative patterns among the symptoms differ as a function of time, such that different network structures may arise given different intervals of time between assessments. Although the process of symptom identification for the current study was driven, in part, to minimize this concern (e.g., symptoms hypothesized to have lower variability and more stability across time, such as weight and shape overvaluation, were excluded from the analysis), it remains unclear what time interval best captures the true cause and effect relationships among these across time. Relatedly, the process of symptom identification itself has been hypothesized to influence network structure. For example, centrality may be inflated when pairs or groups of symptoms within the network are highly related to one another (conceptually or through measurement invariance). This is because the mutual relationships among these similar constructs will naturally produce an inflated correlation coefficient, which, in turn, will bias the centrality indices of these symptoms relative to the other, more distinct symptoms in the network. Selecting symptoms that are different enough from one another to reduce this effect, yet related enough to one another to maintain a functional role in the network model, is critically important and yet has received little research to date.

Lastly, the process of selecting the daily eating disorder symptoms, and corresponding measurement items during phase 2 of the study, was largely hypothesis-driven rather than
reflecting formal diagnostic symptoms based on DSM-5 criteria for eating disorders. This was done in order to minimize the potential effects mentioned above, and was informed by previous network models of eating disorder symptoms among a college population (DuBois et al., 2016). Furthermore, the symptoms included in the network analysis aimed to capture a dimensional approach to eating disorders whereby low levels of symptomatology may be found in the general population. In fact, recent studies using network analysis for depression have indicated that a number of non-DSM symptoms are highly central in the depression network, suggesting that it may be beneficial to include normative experiences that span a larger range of psychopathology than traditional symptoms as represented in the DSM-5 (Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016). However, care should be taken when diverging from formal symptom classification because results may not be generalizable to other research studies utilizing different symptomatology. In fact, creating standardized guidelines for the use of eating disorder symptoms in network models represents a critical development in the field of eating disorders and network analysis. Despite this need, the current study puts forth a preliminary network model that can be tested and refined both within and across different samples.
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Figure 1. A graphical representation of the latent trait approach (left) and network approach (right) for eating disorder psychopathology. Shaded circles represent symptoms of an eating disorder and lines represent causal relationships.
Figure 2. Depiction of Connectivity

Figure 2. A graphical representation of a hypothetical low connectivity (left) and high connectivity (right) eating disorder cycle. Arrow size and color intensity represents the magnitude of influence across time.
### Table 1. Demographic information

<table>
<thead>
<tr>
<th>Sample size (N)</th>
<th>58</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age (SD)</td>
<td>20.5 (3.1)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sex</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Male, n (%)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>58 (100)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnicity, n (%)</th>
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</thead>
<tbody>
<tr>
<td>Hispanic/Latino</td>
<td>8 (13.7)</td>
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<tr>
<td>Not Hispanic/Latino</td>
<td>50 (86.3)</td>
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</table>

<table>
<thead>
<tr>
<th>Race*, n (%)</th>
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</thead>
<tbody>
<tr>
<td>American Indian/Alaska Native</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Black/African American</td>
<td>3 (5.2)</td>
</tr>
<tr>
<td>Asian</td>
<td>16 (27.6)</td>
</tr>
<tr>
<td>Native Hawaiian/Other Pacific Islander</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>White</td>
<td>25 (43.1)</td>
</tr>
<tr>
<td>Other</td>
<td>6 (10.3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sexual Orientation*, n (%)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Heterosexual</td>
<td>48 (82.8)</td>
</tr>
<tr>
<td>Homosexual</td>
<td>4 (6.9)</td>
</tr>
<tr>
<td>Bisexual</td>
<td>6 (10.3)</td>
</tr>
<tr>
<td>Other</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year in College, n (%)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>13 (22.4)</td>
</tr>
<tr>
<td>2nd</td>
<td>12 (20.7)</td>
</tr>
<tr>
<td>3rd</td>
<td>16 (27.6)</td>
</tr>
<tr>
<td>4th</td>
<td>12 (20.7)</td>
</tr>
<tr>
<td>5th or higher</td>
<td>5 (8.6)</td>
</tr>
</tbody>
</table>

*Note: Individuals were given the option to select more than one category for Race and Sexual Orientation*
Table 2. Survey Variable Descriptive Statistics

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
<th>Cronbach’s alpha</th>
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</thead>
<tbody>
<tr>
<td>EDDS</td>
<td>24.8</td>
<td>10.3</td>
<td>0.62</td>
<td>0.69</td>
<td>0.83</td>
</tr>
<tr>
<td>BIAAQ</td>
<td>33.0</td>
<td>8.9</td>
<td>-0.07</td>
<td>-0.43</td>
<td>0.88</td>
</tr>
<tr>
<td>KIMS_Observing</td>
<td>37.4</td>
<td>6.7</td>
<td>0.52</td>
<td>-0.02</td>
<td>0.73</td>
</tr>
<tr>
<td>KIMS_Awareness</td>
<td>28.9</td>
<td>5.8</td>
<td>-0.15</td>
<td>-0.04</td>
<td>0.73</td>
</tr>
<tr>
<td>KIMS_Acceptance</td>
<td>28.9</td>
<td>8.3</td>
<td>-0.541</td>
<td>-0.165</td>
<td>0.89</td>
</tr>
<tr>
<td>KIMS_Describing</td>
<td>26.7</td>
<td>6.2</td>
<td>0.045</td>
<td>0.254</td>
<td>0.88</td>
</tr>
</tbody>
</table>
Figure 3: Distribution of Survey Variables
Figure 4: Distribution of EMA Items
Figure 5: Group-Level Symptom Network
Figure 6a: Bi-Symptom Feedback Loops
Figure 6b: Tri-Symptom Feedback Loops
Figure 7: Symptom Centrality

![Symptom Centrality Chart]

- Perceived Weight Change
- Self Perception
- Preoccupation with Thinness
- Perception of Control
- Negative Emotions
- Hunger
- Fear of Gaining Weight
- Compensatory Behavior
- Food Intake
- Drive for Thinness
- Dietary Restraint
- Dietary Control
- Body Dissatisfaction

Standardized Centrality
Figure 8: Individual Symptom Networks from Two Randomly Selected Participants
Table 3a:

*Summary of Hierarchical Regression Analysis for KIMS and Connectivity Predicting Eating Disorder Symptom Severity (N = 58)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Stage 1</th>
<th></th>
<th></th>
<th>Stage 2</th>
<th></th>
<th></th>
<th>Stage 3</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
<td>β</td>
<td>B</td>
<td>SE B</td>
<td>β</td>
<td>B</td>
<td>SE B</td>
<td>β</td>
</tr>
<tr>
<td>Age</td>
<td>0.07</td>
<td>0.43</td>
<td>0.02</td>
<td>0.23</td>
<td>0.45</td>
<td>0.07</td>
<td>0.40</td>
<td>0.41</td>
<td>0.21</td>
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<tr>
<td>BMI</td>
<td>0.64</td>
<td>0.35</td>
<td>0.24</td>
<td>0.76</td>
<td>0.33</td>
<td>0.28*</td>
<td>0.40</td>
<td>0.33</td>
<td>0.15</td>
</tr>
<tr>
<td>Acceptance (KIMS)</td>
<td>-0.47</td>
<td>0.18</td>
<td>-0.38*</td>
<td>-0.27</td>
<td>0.18</td>
<td>-0.22*</td>
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</tr>
<tr>
<td>Observing (KIMS)</td>
<td>0.08</td>
<td>0.21</td>
<td>0.05</td>
<td>0.10</td>
<td>0.09</td>
<td>0.06</td>
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<tr>
<td>Describing (KIMS)</td>
<td>-0.20</td>
<td>0.22</td>
<td>-0.12</td>
<td>-0.31</td>
<td>0.21</td>
<td>-0.19</td>
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<tr>
<td>Awareness (KIMS)</td>
<td>0.11</td>
<td>0.27</td>
<td>0.06</td>
<td>0.08</td>
<td>0.25</td>
<td>0.05</td>
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</tr>
<tr>
<td>Connectivity</td>
<td></td>
<td></td>
<td></td>
<td>3.93</td>
<td>1.25</td>
<td>0.42**</td>
<td></td>
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<tr>
<td>$R^2$</td>
<td>0.06</td>
<td></td>
<td></td>
<td>0.22</td>
<td></td>
<td></td>
<td>0.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2$ change</td>
<td></td>
<td></td>
<td></td>
<td>0.16</td>
<td></td>
<td></td>
<td>0.13</td>
<td></td>
<td></td>
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<tr>
<td>$F$ for change in $R^2$</td>
<td>1.68</td>
<td></td>
<td></td>
<td>2.62*</td>
<td></td>
<td></td>
<td>9.92**</td>
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</table>

*p < .05.  **p < .01.
Table 3b:

*Summary of Hierarchical Regression Analysis for BIAAQ and Connectivity Predicting Eating Disorder Symptom Severity (N = 58)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Stage 1</th>
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<tr>
<td></td>
<td>B</td>
<td>SE B</td>
<td>β</td>
<td>B</td>
<td>SE B</td>
<td>β</td>
<td>B</td>
<td>SE B</td>
<td>β</td>
</tr>
<tr>
<td>Age</td>
<td>0.07</td>
<td>0.43</td>
<td>0.02</td>
<td>0.18</td>
<td>0.36</td>
<td>0.05</td>
<td>0.35</td>
<td>0.35</td>
<td>0.11</td>
</tr>
<tr>
<td>BMI</td>
<td>0.64</td>
<td>0.35</td>
<td>0.24</td>
<td>0.42</td>
<td>0.30</td>
<td>0.16</td>
<td>0.22</td>
<td>0.29</td>
<td>0.08</td>
</tr>
<tr>
<td>BIAAQ</td>
<td>-0.63</td>
<td>0.13</td>
<td>-0.55**</td>
<td>-0.52</td>
<td>0.13</td>
<td>-0.45*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Connectivity</td>
<td>2.97</td>
<td>1.11</td>
<td>0.31**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.06</td>
<td></td>
<td></td>
<td>0.35</td>
<td></td>
<td></td>
<td>0.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2$ Change</td>
<td>0.29</td>
<td></td>
<td></td>
<td>0.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$F$ for change in $R^2$</td>
<td>1.68</td>
<td></td>
<td></td>
<td>24.54**</td>
<td></td>
<td></td>
<td>7.11**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .05. **p < .01.
Figure 9: Bivariate Relationships among Study Variables

Note: Colored cells indicate significant relationships at the $p < 0.05$ level.
Appendix A: Demographic Questionnaire

What is your age? __________

What is your year in school (e.g., 1st, 2nd, 3rd, ...)? __________

Do you consider yourself to be:

☐ Heterosexual or straight?
☐ Gay or lesbian?
☐ Bisexual?
☐ Other?

Have you ever been diagnosed with an eating disorder?

☐ Yes
   a) What year ______
☐ No

Are you currently receiving psychological treatment for an eating disorder?

☐ Yes
☐ No

What is your height?

_____ Feet
_____ Inches

What is your weight?

_____ Weight in Pounds

What is your identified race/ethnicity?

☐ African American/Black
☐ Asian
☐ Caucasian/White
☐ Hispanic/Latino
☐ Native Hawaiian and Other Pacific Islander
☐ American Indian or Alaska Native
☐ Other ____________________
Appendix B: Momentary Assessments

Please rate how you feel in this moment:

I feel out of control
I feel afraid of gaining weight
I feel dissatisfied with the way my body looks
I am thinking about being thin
I feel happy with who I am
I feel hungry
I feel negative emotions
I have control over my eating
I am consciously trying to restrict [cut back] the overall amount that I eat or drink
I am paying attention to my feelings and sensations in my body
I am trying to ignore or avoid my thoughts and feelings
I am perceiving my thoughts and feelings without having to react to them

Please rate the following questions based on your experiences since the last assessment:

I have engaged in behaviors in order to lose calories or influence my weight or shape

[not at all – very much so]

My weight has…[gone down; stayed the same; gone up]

I have consumed food or drink…[not at all – a large amount]
Appendix C: Eating Disorder Diagnostic Scale

### Eating Disorder Diagnostic Scale (EDDS) – DSM-5 Version

Please carefully complete all questions, choosing NO or 0 for questions that do not apply.

**Over the past 3 months...**

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Have you felt fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Have you had a definite fear that you might gain weight or become fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Has your weight or shape influenced how you judge yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. During the past 3 months have there been times when you have eaten what other people would regard as an unusually large amount of food (e.g., a pint of ice cream) given the circumstances?</td>
<td>[ ] YES</td>
<td>[ ] NO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. During the times when you ate an unusually large amount of food, did you experience a loss of control (e.g., felt you couldn’t stop eating or control what or how much you were eating)?</td>
<td>[ ] YES</td>
<td>[ ] NO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. How many times per month on average over the past 3 months have you eaten an unusually large amount of food and experienced a loss of control?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**During episodes of overeating with a loss of control, did you...**

| 7. Eat much more rapidly than normal? |            |           |           |           |           |           |           |           |           |           |           |           |           |           |           |           |           |           |           |           |
8. Eat until you felt uncomfortably full?
[ ] YES
[ ] NO

9. Eat large amounts of food when you didn’t feel physically hungry?
[ ] YES
[ ] NO

10. Eat alone because you were embarrassed by how much you were eating?
[ ] YES
[ ] NO

11. Feel disgusted with yourself, depressed, or very guilty after overeating?
[ ] YES
[ ] NO

12. If you have episodes of uncontrollable overeating, does it make you very upset?
[ ] YES
[ ] NO

In order to prevent weight gain or counteract the effects of eating, how many times per month on average over the past 3 months have you:

13. Made yourself vomit? 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16+
14. Used laxatives or diuretics? 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16+
15. Fasted (skipped at least 2 meals in a row)? 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16+
16. Engaged in more intense exercise specifically to counteract the effects of overeating.

17. How many times per month on average over the past 3 months have you eaten after awakening from sleep or eaten an unusually large amount of food after your evening meal and felt distressed by the night eating?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16+

18. How much do eating or body image problems impact your relationships with friends and family, work performance, and school performance?
   Not at all  Slightly  Moderately  Extremely
   1 2 3 4 5 6

   ______lbs. -or- _____kg.

20. How tall are you? _____ft. _____in. -or- ______cm.

21. What is your highest weight at your current height? _________lbs. -or- _____kg

22. What is your sex?
   [ ] MALE
   [ ] FEMALE

What is your age? ________
Appendix D: Kentucky Inventory of Mindfulness Skills

<table>
<thead>
<tr>
<th>Never or very rarely true</th>
<th>Rarely true</th>
<th>Sometimes true</th>
<th>Often true</th>
<th>Very often or always true</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I notice changes in my body, such as whether my breathing slows down or speeds up.</td>
<td></td>
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<tr>
<td>2. I’m good at finding the words to describe my feelings.</td>
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<tr>
<td>3. When I do things, my mind wanders off and I’m easily distracted.</td>
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<tr>
<td>4. I criticize myself for having irrational or inappropriate emotions.</td>
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<tr>
<td>5. I pay attention to whether my muscles are tense or relaxed.</td>
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<tr>
<td>6. I can easily put my beliefs, opinions, and expectations into words.</td>
<td></td>
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<tr>
<td>7. When I’m doing something, I’m only focused on what I’m doing, nothing else.</td>
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<tr>
<td>8. I tend to evaluate whether my perceptions are right or wrong.</td>
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<tr>
<td>9. When I’m walking, I deliberately notice the sensations of my body moving.</td>
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<tr>
<td>10. I’m good at thinking of words to express my perceptions, such as how things taste, smell, or sound.</td>
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<tr>
<td>11. I drive on “automatic pilot” without paying attention to what I’m doing.</td>
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<tr>
<td>12. I tell myself that I shouldn’t be feeling the way I’m feeling.</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>13. When I take a shower or bath, I stay alert to the sensations of water on my body.</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. It’s hard for me to find the words to describe what I’m thinking.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. When I’m reading, I focus all my attention on what I’m reading.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. I believe some of my thoughts are abnormal or bad and I shouldn’t think that way.</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>17. I notice how foods and drinks affect my thoughts, bodily sensations, and emotions.</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. I have trouble thinking of the right words to express how I feel about things.</td>
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<tr>
<td>19. When I do things, I get totally wrapped up in them and don’t think about anything else.</td>
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<td></td>
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</tr>
<tr>
<td>20. I make judgments about whether my thoughts are good or bad.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>21. I pay attention to sensations, such as the wind in my hair or sun on my face.</td>
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<td></td>
</tr>
</tbody>
</table>
### Symptom Decoupling in Eating Disorder Treatment

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never or very rarely true</td>
<td>Rarely true</td>
<td>Sometimes true</td>
<td>Often true</td>
<td>Very often or always true</td>
<td></td>
</tr>
</tbody>
</table>

___22. When I have a sensation in my body, it’s difficult for me to describe it because I can’t find the right words.
___23. I don’t pay attention to what I’m doing because I’m daydreaming, worrying, or otherwise distracted.
___24. I tend to make judgments about how worthwhile or worthless my experiences are.
___25. I pay attention to sounds, such as clocks ticking, birds chirping, or cars passing.
___26. Even when I’m feeling terribly upset, I can find a way to put it into words.
___27. When I’m doing chores, such as cleaning or laundry, I tend to daydream or think of other things.
___28. I tell myself that I shouldn’t be thinking the way I’m thinking.
___29. I notice the smells and aromas of things.
___30. I intentionally stay aware of my feelings.
___31. I tend to do several things at once rather than focusing on one thing at a time.
___32. I think some of my emotions are bad or inappropriate and I shouldn’t feel them.
___33. I notice visual elements in art or nature, such as colors, shapes, textures, or patterns of light and shadow.
___34. My natural tendency is to put my experiences into words.
___35. When I’m working on something, part of my mind is occupied with other topics, such as what I’ll be doing later, or things I’d rather be doing.
___36. I disapprove of myself when I have irrational ideas.
___37. I pay attention to how my emotions affect my thoughts and behavior.
___38. I get completely absorbed in what I’m doing, so that all my attention is focused on it.
___39. I notice when my moods begin to change.
Appendix E: Body Image Acceptance and Action Questionnaire

Directions: Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following rating scale to make your choices. For instance, if you believe a statement is ‘Always True,’ you would write a 7 next to that statement.

<table>
<thead>
<tr>
<th>Never True</th>
<th>Very Seldom True</th>
<th>Seldom True</th>
<th>Sometimes True</th>
<th>Frequently True</th>
<th>Almost Always True</th>
<th>Always True</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
</tbody>
</table>

1. I get on with my life even when I feel bad about my body.
2. Worrying about my weight makes it difficult for me to live a life that I value.
3. I would gladly sacrifice important things in my life to be able to stop worrying about my weight.
4. I care too much about my weight and body shape.
5. How I feel about my body has very little to do with the daily choices I make.
6. Many things are more important to me than feeling better about my weight.
7. There are many things I do to try and stop feeling bad about my body weight and shape.
8. I worry about not being able to control bad feelings about my body.
9. I do not need to feel better about my body before doing things that are important to me.
10. I don’t do things that might make me feel fat.
11. I shut down when I feel bad about my body shape or weight.
12. My worries about my weight do not get in the way of my success.
13. I can move toward important goals, even when feeling bad about my body.
14. There are things I do to distract myself from thinking about my body shape or size.
15. My thoughts and feelings about my body weight and shape must change before I can take important steps in my life.

16. My thoughts about my body shape and weight do not interfere with the way I want to live.

17. I cannot stand feeling fat.

18. Worrying about my body takes up too much of my time.

19. If I start to feel fat, I try to think about something else.

20. Worrying about my weight does not get in my way.

21. Before I can make any serious plans, I have to feel better about my body.

22. I will have better control over my life if I can control my negative thoughts about my body.

23. I avoid putting myself in situations where I might feel bad about my body.

24. To control my life, I need to control my weight.

25. My worries and fears about my weight are true.


27. I do things to control my weight so I can stop worrying about the way my body looks.

28. When I start thinking about the size and shape of my body, it’s hard to do anything else.

29. My relationships would be better if my body weight and/or shape did not bother me.
Appendix F: System Usability Scale

1. I think that I would like to use this system frequently
   | Strongly disagree | Strongly agree |
   | 1 | 2 | 3 | 4 | 5 |

2. I found the system unnecessarily complex
   | 1 | 2 | 3 | 4 | 5 |

3. I thought the system was easy to use
   | 1 | 2 | 3 | 4 | 5 |

4. I think that I would need the support of a technical person to be able to use this system
   | 1 | 2 | 3 | 4 | 5 |

5. I found the various functions in this system were well integrated
   | 1 | 2 | 3 | 4 | 5 |

6. I thought there was too much inconsistency in this system
   | 1 | 2 | 3 | 4 | 5 |

7. I would imagine that most people would learn to use this system very quickly
   | 1 | 2 | 3 | 4 | 5 |

8. I found the system very cumbersome to use
   | 1 | 2 | 3 | 4 | 5 |

9. I felt very confident using the system
   | 1 | 2 | 3 | 4 | 5 |

10. I needed to learn a lot of things before I could get going with this system
    | 1 | 2 | 3 | 4 | 5 |
Research participants wanted!!!

Participate in an online research study on body image and dieting

Phase 1: Brief online survey
Phase 2: Mobile phone prompts over 10 days
Phase 3: Brief online survey

Receive up to $45 for your participation!!!

Must be an NU student, Age 18-40, not currently receiving psychotherapy for an eating disorder, and own an Android or iPhone mobile phone. Female participants must not be pregnant or lactating.

INTERESTED IN PARTICIPATING?
contact Russell DuBois at dubois.r@husky.neu.edu
With subject line “App study 1”

Research conducted by Dr. Rachel Rodgers: Department of Applied Psychology