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INVESTIGATING THE SHARED AND UNIQUE MECHANISMS OF THE  
DEVELOPMENT OF COMORBID EATING DISORDER-ANXIETY SYMPTOMS  
DURING ADOLESCENCE

By

Leigh Cara Brosof  
B.A., Washington University in St. Louis, 2016  
M.S., University of Louisville, 2019

Dissertation  
Submitted to the Faculty of the  
College of Arts and Sciences of the University of Louisville  
in Partial Fulfillment of the Requirements  
for the Degree of

Doctor of Philosophy  
in Clinical Psychology

Department of Psychological and Brain Sciences  
University of Louisville  
Louisville, Kentucky

August 2022

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Dissertation Approved on  
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## ABSTRACT

### INVESTIGATING THE SHARED AND UNIQUE MECHANISMS OF THE DEVELOPMENT OF COMORBID EATING DISORDER-ANXIETY SYMPTOMS DURING ADOLESCENCE

Leigh Cara Brosos

May 4, 2022

**Introduction:** Eating disorders are associated with significant morbidity, psychiatric comorbidity, and impairment. Despite the detrimental outcomes associated with eating disorders, effective treatments for eating disorders are lacking. One factor that has impeded the identification of targets for intervention in eating disorders is the high comorbidity rate with other psychiatric disorders. Comorbidity models can inform treatments by showing which mechanisms are shared in comorbidity and which mechanisms are unique to specific disorders. Anxiety disorders are the most frequently co-occurring disorders with eating disorders, as 85% of individuals with eating disorders have a comorbid anxiety disorder. One mechanistic process in both anxiety disorders and eating disorders is learning. Shared cognitive-behavioral learning mechanisms in both anxiety and eating disorders include repetitive negative thinking and negative affect. Unique cognitive-behavioral learning mechanisms to eating disorders include thinness expectancies and habit formation around eating. In the current study, I investigated the cognitive-behavioral mechanisms related to learning in comorbid eating disorders-anxiety. **Method:** Seventy adolescents (13-15 years old) from the community completed self-report assessments at three time points: at the beginning of school year, the mid-point



of school year (four-month follow-up), and the end of school year (nine-month follow-up). Adolescence may be a critical period for the development of comorbid eating disorder-anxiety symptomatology. First, a cross-sectional path model was constructed to test whether negative affect, repetitive negative thinking, thinness expectancies, and habit formation were related to anxiety symptoms and eating disorder symptoms. Then, a prospective autoregressive model across the three time points (baseline, four-months, nine-months) was constructed to assess a comorbidity model of anxiety and eating disorder symptoms over time, while adjusting for baseline levels of symptoms. Finally, a mediation model from negative affect to eating disorders through thinness expectancies was tested. **Results:** Cross-sectionally, negative affect was a shared cognitive-behavioral mechanism significantly associated with both anxiety and eating disorder symptoms, and thinness expectancies was a unique cognitive-behavioral mechanism associated with eating disorder symptoms. Due to significant attrition, exploratory models including two time points (baseline and four-month follow-up) and excluding habits around eating were conducted. Prospectively, only baseline eating disorder symptoms predicted eating disorder symptoms at four-month follow-up. No mediational effects were found. A post-hoc moderation analysis showed that individuals higher in thinness expectancies were more likely to have higher eating disorder symptoms when also higher in negative affect, compared to lower negative affect. **Conclusions:** This study was a preliminary test of an integrated learning model and hopes to guide future research regarding shared and unique vulnerability factors for eating disorder comorbidity. These findings suggest that the strongest predictor of later eating disorder symptoms in adolescence is earlier eating disorder symptoms. Thus, there is an urgent need to study eating disorder symptoms and comorbidity at an even earlier age, perhaps during preadolescence, before puberty.

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## INTRODUCTION

Eating disorders are serious mental illnesses that affect up to 4.5% of the population across the lifetime (Hudson et al., 2007; Smink, van Hoeken, & Hoek, 2013), though prevalence studies are likely underestimating the true rate of eating disorders because symptoms may be underreported (Udo & Grilo, 2018). In addition, subthreshold symptoms (affecting up to 22% of the population) carry significant impairment and distress (e.g., Jones et al., 2001; Le Grange & Loeb, 2007). Eating disorders are associated with significant morbidity, psychiatric comorbidity, and impairment (Carta et al., 2014; Katzman, 2005; Keel et al., 2003; Rome & Ammerman, 2003) and have the second highest mortality rate of any mental disorder, second only to opioid use disorder (Chesney, Goodwin, & Fazel, 2014). Despite the detrimental outcomes associated with eating disorders, effective treatments for eating disorders are lacking; the gold-standard treatment, Cognitive Behavioral Therapy – Enhanced (CBT-E; Fairburn, 2008), for bulimia nervosa (BN) and binge eating disorder (BED) is only about 50% effective (Linardon et al., 2017), and there are no existing empirically supported treatments for adults with anorexia nervosa (AN; Watson & Bulik, 2013). Relapse occurs in up to 41% of cases, and about 25% of cases of eating disorders result in a severe and persistent course of illness (Berkman, Lohr, & Bulik, 2007; Carter et al., 2004; Halmi, 2013; McFarlane, Olmstead, & Trottier, 2008; Zerwas et al., 2013). Thus, novel treatments or enhancements to existing treatments are sorely needed to relieve the symptoms and impairment associated with eating disorders.

There is also a lack of understanding of the important underlying etiological factors (i.e., *mechanisms*) contributing to the development and maintenance of eating disorders (Culbert et al., 2015; Stice, 2002; Stice, 2016). Explicating these mechanisms is imperative for the future development of treatments because they elucidate specific targets for intervention. For instance, if negative affect is found to be an important etiological factor relating to the development of eating disorders, then treatment would primarily focus on reducing and learning to tolerate negative affect. In theory, if negative affect underlies eating disorders, then reducing negative affect would also reduce eating disorder symptomatology or, ideally, prevent eating disorders from developing in the first place. Thus, understanding underlying mechanisms of eating disorders can help in both prevention and intervention efforts for these disorders.

One factor that has impeded the investigation of mechanisms in eating disorders is the high comorbidity rate with other psychiatric disorders (e.g., Kessler et al., 2005b). Indeed, eating disorders co-occur with other mental disorders 97% of the time (Blinder, Cumella, & Santhara, 2006). This high rate of comorbidity makes disentangling the mechanistic processes of eating disorders versus other mental disorders difficult. One potential solution to this barrier is to create comorbidity models of eating disorders, which may more accurately reflect how eating disorders develop in the first place (Smith & Keel, 2017). Comorbidity models allow for the investigation of transdiagnostic mechanisms that can be targeted in multiple disorders, which may lead to more effective treatment. For instance, obsessive-compulsive disorder (OCD) and eating disorders overlap up to 60% of the time (Godart et al., 2002; Halmi et al., 2005), but it is unknown which disorder should be treated first or whether these disorders should be treated

concurrently (Simpson et al., 2013). Finding a transdiagnostic mechanism core to both disorders, such as perfectionism, can lead to more parsimonious treatment, such that intervening on that target could reduce symptoms of both disorders simultaneously. Thus, etiological models should reflect that eating disorders almost always develop in conjunction with other disorders, rather than in a vacuum (Culbert et al., 2015).

Below, I propose a testable model for investigating core mechanisms underlying eating disorder and anxiety comorbidity at a critical period of development: adolescence. First, I review eating disorders and eating disorder-anxiety comorbidity. Second, I discuss shared processes between eating disorders and anxiety, as well as unique processes related only to eating disorders. Specifically, I introduce learning as a central process underlying eating disorder-anxiety comorbidity, including shared processes of fear and reward learning, and habit learning related to eating as a unique process related to the etiology of eating disorders. I also discuss how these processes may be particularly relevant to the period of development during adolescence. Finally, based on the above literature, I propose an integrated learning model of eating disorder-anxiety comorbidity that includes neurobiological vulnerabilities, shared vulnerabilities, and unique vulnerabilities that will be tested in the proposed study. This study will provide an initial framework for better understanding how eating disorder comorbidity develops, with implications for improving precision interventions to alleviate the suffering of eating disorders.

### **Eating Disorders**

The Diagnostic and Statistical Manual-5 lists three “major” eating disorders – AN, BN, and binge eating disorder (BED; American Psychiatric Association [APA],

2013). AN is characterized by an extreme fear of weight gain and inadequate food intake leading to low body weight (body mass index [BMI] under 18.5). There are two subtypes of AN: restricting subtype and binge/purge subtype. In AN restricting subtype, individuals maintain low weight by dieting, fasting (i.e., skipping two meals or more in a row; Forbush, Wildes, & Hunt 2014), and/or excessive exercise (i.e., exercising for excessively long periods of time, in a driven manner; Cook et al., 2016; APA, 2013). In AN binge/purge subtype, there is also the presence of binge eating and/or purging behaviors. Binge eating is defined as the consumption of an “unusually large” amount of food within a two-hour period accompanied by a feeling of loss of control (APA, 2013). Purging is defined as behaviors that are intended to compensate for caloric intake including self-induced vomiting and laxative or diuretic use (APA, 2013). Compensatory behaviors are behaviors used to negate caloric intake, including purging, as well as excessive exercise. BN is characterized by binge eating *and* compensatory (i.e., purging or excessive exercise) behaviors that *both* occur at least once per week and have been present at least over the last three months (APA, 2013). BED is characterized by binge eating in the absence of compensatory behaviors (APA, 2013).

A fourth diagnosis is other specified feeding or eating disorder (OSFED), which encompasses individuals who do not fit the symptom profile categories of AN, BN, or BED but have a clinically significant eating disorder that causes distress and/or impairment (APA, 2013; Vo et al., 2017). OSFED is the most common eating disorder diagnosis (Dahlgren, Wisting, & Ro, 2017), with up to 39% of individuals with eating disorders diagnosed with OSFED (Vo et al., 2017). Because most individuals with eating disorders present with OSFED, which is an extremely heterogeneous “catch-all”

category, studies including only certain diagnostic groups may be missing important information regarding the heterogeneity of eating disorder symptoms (Levinson, Vanzhula, & Brosof, 2018). Indeed, even within the same diagnosis (e.g., AN), individuals can display a wide array of cognitive symptomatology and behaviors (Levinson, Vanzhula, & Brosof, 2018). As a result, it has been suggested that investigating symptomatology (e.g., restriction, binge eating, purging), rather than full diagnostic disorders, may be more informative for explicating etiology (Glazer et al., 2019; Stice, 2016). In addition, similar psychosocial impairment arises from subclinical eating disorder presentations in comparison to full-threshold presentations (Byrne et al., 2016; Mehak & Racine, 2021; Vannucci et al., 2012). Subclinical eating disorders are defined as distressing and impairing eating disturbances that do not meet full criteria for an eating disorder due to frequency of behaviors (Dahlgren, Wisting, & Ro, 2017; Stice, Marti, & Durant, 2011a). Subclinical presentations can also result in serious medical complications (e.g., electrolyte abnormalities) due to the presence of binge eating, purging, or restriction behaviors (Rome & Ammerman, 2003).

For these reasons, a significant body of literature supports that eating disorders are better represented as dimensional, rather than categorical syndromes (e.g., Forbush et al., 2017). For instance, individuals with eating disorders frequently pass between diagnostic thresholds (Glazer et al., 2019; Stice, Marti, & Durant, 2011a), and it is not uncommon for one individual to be diagnosed with AN, BN, and OSFED in their lifetime. Given the dimensional nature of these disorders and the fact that they share more similarities than differences (Fairburn, 2008), *eating disorders* is used as an umbrella term for all diagnostic presentations in the current proposal. Several seminal



reviews in the eating disorder literature have called for future studies to investigate mechanisms across presentations, rather than within particular eating disorder diagnostic groups (e.g., Culbert et al., 2015; Frank, 2016; Stice, 2016). Studies utilizing a transdiagnostic eating disorder model that investigate mechanisms of symptomatology will elucidate where important differences exist among symptoms and more accurately reflect the current literature on how eating disorders present.

### ***Eating Disorder Comorbidity***

As mentioned above, one area that may present a significant barrier to effective treatment is psychiatric comorbidity associated with eating disorders. Comorbidity is the rule rather than the exception, as eating disorders present with at least one other psychiatric disorder up to 97% of the time (Blinder, Cumella, & Santhara, 2006). Anxiety disorders are the most frequently co-occurring disorders with eating disorders, as 85% of individuals with eating disorders have a comorbid anxiety disorder (Hudson et al., 2007; Kessler et al., 2005; Pallister & Waller, 2008). Comorbid anxiety disorders complicate eating disorder treatment outcomes and make it more likely that eating disorders will persist over time (Peck, Murray, & Kaye, 2018; Zerwas et al., 2013). Further, comorbid eating disorder-anxiety disorders are associated with greater psychosocial impairment, lower quality of life, and medical complications (Button, Chadalavada, & Palmer, 2010; Peck, Murray, & Kaye, 2018). Despite the substantial impairment associated with comorbid eating disorder-anxiety disorders, little is understood about the mechanisms that underlie this comorbidity (Pallister & Waller, 2008; Swinbourne et al., 2012).

### *Adolescence as a Critical Period for Development of Comorbid Symptoms*

Adolescence may be a critical period for the development of comorbid eating disorder-anxiety symptomatology (e.g., Rojo-Moreno et al., 2015). Anxiety symptom onset often precedes the development of eating disorders (Godart et al., 2000; Swinbourne et al., 2012). The median age of onset for anxiety symptoms is 11 years old (Kessler et al., 2005). Eating disorders onset within the ages of 14-19, on average (Herpetz-Dahlmann, 2015; Volpe et al., 2016). In particular, the transition from early-to-mid adolescence may represent a critical transition risk period for the development of eating disorders (Herpetz-Dahlmann, 2015; Volpe et al., 2016). Both biological (i.e., puberty) and psychosocial changes during this time create a particular vulnerability for eating disorder onset (Culbert et al., 2015; Davis & Smith, 2018; Pearson et al., 2012). Puberty has been well-established as a time period for risk for eating disorders due to hormonal changes that may trigger genetic predispositions for eating disorders in concurrence with environmental vulnerabilities (eating disorders are up to 50% heritable; Bulik et al., 2006; Klump, 2013). These environmental vulnerabilities may include psychosocial changes such as greater dieting behaviors and body dissatisfaction (both risk factors for eating disorders; Stice, 2002) during this age range (Stice & Shaw, 2002). Importantly, studying the transition from low-(or no)-symptomatology to subclinical-symptomatology to clinical-symptomatology is imperative for elucidating potentially causal risk factors for disorder onset (Stice, 2002; Stice, 2016). Thus, adolescence represent an ideal period for studying how mechanisms contribute to the development of this comorbidity and will aid in the creation of evidence-based treatments for eating disorders-anxiety disorders.

## **Models of Multifinality and Divergent Trajectories**

Models of divergent trajectories explain how individuals with overlapping risk factors (e.g., negative affect) for multiple disorders (e.g., eating disorders and anxiety disorders) come to develop one disorder but not another (e.g., someone with elevated negative affect develops an anxiety disorder but not an eating disorder) (Nolen-Hoeksema & Watkins, 2011). Divergent trajectories stand in contrast to models of multifinality, which explain how individuals with overlapping risk factors come to develop comorbid disorders (e.g., someone with elevated negative affect develops *both* an anxiety and an eating disorder). Elucidating models of multifinality and divergent trajectories can inform treatments by showing which mechanisms are **shared** in comorbidity and which mechanisms are **unique** to specific disorders. Thus, such models can aid in developing treatments that are both transdiagnostic and disorder-specific in nature.

As mentioned above, anxiety disorders are present among eating disorders 85% of the time (Hudson et al., 2007); however, eating disorders are only present among anxiety disorders 14% of the time (Swinbourne et al., 2012). In other words, out of all individuals with an eating disorder, 85 out of 100 would also have a diagnosable anxiety disorder on average, but out of all individuals with different anxiety disorders (e.g., social anxiety disorder, generalized anxiety disorder, etc.), 14 out of 100 would also have a clinically significant eating disorder on average (Bulik et al., 1997). This pattern of overlap and the precedence of anxiety symptoms to eating disorder symptoms suggests that there are shared vulnerabilities for both anxiety and eating disorders, as well as *additional* unique vulnerabilities that lead to the development of an eating disorder in addition to anxiety.

Delineating mechanisms of comorbid eating disorders-anxiety thus requires: 1) identifying **shared** mechanistic processes between eating disorders and anxiety, and 2) examining what mechanisms add additional, **unique** risk for each disorder separately.

Below, the shared etiological process of learning (specifically fear and reward learning) is discussed. Next, the unique process of habit learning related to eating is reviewed in the context of eating disorder development and maintenance. Finally, a learning model of comorbid eating disorders-anxiety is proposed that integrates these shared and unique mechanisms, as well as their neurobiological underpinnings. Though the neurobiological mechanisms will be discussed as part of the model, they will not be directly tested in this study due to feasibility. Rather, this will be an initial exploration of the cognitive-behavioral shared and unique mechanisms of the model, which may inform future research testing the neurobiological mechanisms in conjunction with these cognitive-behavioral mechanisms.

### ***Shared Neurobiological Mechanistic Process: Reward and Fear Learning***

One central shared mechanistic process in both anxiety disorders and eating disorders is *learning*. Learning is an umbrella term for the neurocognitive processes implicated in the formation, updating, or replacement of associations based on contingencies (i.e., positive or negative feedback or outcomes) in the environment (Craske, Hermans, & Vansteenwegen, 2006). Specifically, two types of learning have been identified in both eating disorders and anxiety disorders: fear learning and reward learning. **Fear learning**, which is also referred to as *aversive associative learning*, is defined as the process through which relationships between stimuli and aversive outcomes are learned and encompasses the learning of fear, anxiety, and avoidance

behaviors (Arnaudova et al., 2017; Pittig et al., 2018). **Reward learning** is defined as the formation of associations in response to reward in the environment (Berner & Marsh, 2014). Both fear and reward learning are implicated in the development and maintenance of anxiety and eating disorders separately. However, little research has investigated fear and reward learning in the context of eating-disorder anxiety *comorbidity*.

### **Fear Learning.**

Four major areas of learning have been delineated in aversive associative (i.e., fear) learning: 1) *fear acquisition*, or how fears develop, 2) *fear generalization*, or how fears spread beyond the feared target or situation, 3) (deficits in) *fear extinction*, or how fears persist, and 4) *resurgence of fear*, or how fears reemerge after initial extinction (Pittig et al., 2018). All four areas of fear learning (acquisition, generalization, extinction, and resurgence) have been implicated in the development and maintenance of anxiety symptoms and eating disorders, particularly AN (Pittig et al., 2018).

### ***Fear Learning in Anxiety.***

Individuals with high symptoms of anxiety more readily acquire fears during experimental acquisition trainings (Lissek et al., 2005; Pittig et al., 2018) and show deficits in fear extinction (Duits et al., 2015; Lissek et al., 2005; Craske et al., 2008; McLaughlin et al., 2015; Milad et al., 2013; Pittig et al., 2018). It is not entirely clear why anxious individuals display overactive fear learning, or a vulnerability to acquire fears more readily than non-anxious individuals. However, individual differences and biological predispositions may play a role in overactive fear learning (Pittig et al., 2018). Fear acquisition is estimated to be 35-45% heritable (Hettema et al., 2003), and genes involved in fear reactivity, such as memory creation, dopamine signaling, and brain

region activation (e.g., amygdala and prefrontal cortex) are connected to fear acquisition and extinction (Abraham et al., 2014; Lonsdorf et al., 2009; Lonsdorf et al., 2015; Pittig et al., 2018; Soliman et al., 2010; Wendt et al., 2015). In addition, individual differences, such as early life adversity and low estrogen levels, may be related to increased fear acquisition and deficits in fear extinction (Glover et al., 2015; Hwang et al., 2015; Pittig et al., 2018; Remmes et al., 2016; Teicher & Sampson, 2016; Wright et al., 2015).

### ***Fear Learning in Eating Disorders.***

In general, fear learning plays a central role in the development and maintenance of eating disorders, particularly in AN (Murray et al., 2018; Strober, 2004). Individuals with AN may have overactive fear learning around food and catastrophic weight gain (i.e., the belief that one will gain a large amount of weight rapidly; Murray et al., 2018); however, these individuals do not necessarily display overactive fear learning to widespread stimuli (Friederich et al., 2013), meaning that fear learning seems to be impaired specifically around food and weight. Biological predispositions in neural, hormonal, and genetic correlates may drive overactive fear learning around food/weight (Strober, 2004; Guarda et al., 2015). Theoretically, these biological predispositions lead individuals to over-attend to food and weight stimuli in the environment, as well negative outcomes related to these stimuli. In turn, over-attenuation to food and negative outcomes leads to fear conditioning around food and weight gain (Murray et al., 2016; Strober, 2004). For example, individuals with eating disorders have deficits in the insula (Kerr et al., 2016; Oberndorfer et al., 2013; Schienle et al., 2009), which is implicated in interoceptive awareness, or the awareness of bodily sensations (Craig, 2009). Such deficits may lead to over-attending to hunger and fullness cues. In individuals with eating

disorders, such attention to these bodily signals can create anxiety around eating certain amounts of or kinds of food that exacerbate fullness because of a fear of gaining weight (i.e., they equate fullness with weight gain) (Friederich et al., 2013). Further, individuals with AN experience restriction as anxiolytic, negatively reinforcing this fear conditioning around fullness and eating (Kaye et al., 2003). Though these models have been applied to AN, all eating disorders share fear of food and weight gain as central features (Levinson et al., 2017), and it is likely that this overactive fear learning would apply across eating disorder presentations. In addition, fears of food and weight gain drive restriction, binge eating, purging, which are central to the maintenance of eating disorders (Murray et al., 2018).

In summary, fear learning is a central mechanistic process in the development of both anxiety disorders and eating disorders. Thus, better understanding fear learning as a shared process in the comorbidity of these disorders is the next step in delineating how fear learning can be targeted in precision interventions for this comorbidity. Below, reward learning is discussed as a similar shared process in both anxiety disorders and eating disorders.

### **Reward Learning.**

#### ***Reward Learning in Anxiety.***

Unlike fear learning, reward learning has received less attention in the anxiety disorders but has been extensively studied in the eating disorders. In the anxiety disorders, a diathesis-stress model has been proposed for altered reward learning in anxious individuals (Dillon et al., 2014). Specifically, deficits in fear and threat processing neurocircuitry may serve as a vulnerability that only result in blunted reward

learning after exposure to a stressful event (Admon et al., 2013; Dillon et al., 2014). This theory may explain how some individuals develop an anxiety disorder alone (i.e., divergent trajectory) or a comorbid disorder in addition to an anxiety disorder (i.e., multifinality). Specifically, individuals with deficits in fear and threat processing may be predisposed to an anxiety disorder; in some individuals, this vulnerability may be paired with a stressful event that then affects reward learning processes. Thus, after reward learning is altered, an individual may develop an additional disorder as well as the preexisting anxiety disorder. Further, some individuals may be biologically predisposed to both altered fear and reward learning that is then activated by a stressful event. For example, this theory has been used to explain how anxiety-depression comorbidity develops (Dillon et al., 2014). Morris and Rottenberg (2015) found that when individuals with GAD completed a computerized signal detection task, those who demonstrated heightened reward learning during a stressor condition were less likely to have comorbid depression symptoms one month later, and individuals with GAD who were never depressed displayed better reward learning than those who had a history of depression. Thus, individual differences in reward learning may influence whether an individual develops a comorbid disorder. However, there is no literature, to my knowledge, on the role of reward learning in comorbid anxiety-eating disorders.

### ***Reward Learning in Eating Disorders.***

In the eating disorders, it has been suggested that reward learning plays a central role in the development and maintenance of AN and BN (Frank, 2016). Overall, individuals with BN exhibit underactive reward learning, whereas individuals with AN exhibit overactive reward learning, compared to healthy controls (Frank et al., 2012;



Grob et al., 2012). Specifically, individuals with BN show reduced activity in reward areas of the brain (e.g., ventral striatum and orbitofrontal cortex) when presented with a reward compared to healthy controls, indicating that more of the reward stimuli (e.g., food) is needed activate these reward areas (Cyr et al., 2016; Frank et al., 2011). Simultaneously, it has been found that individuals with BN do not activate areas of the brain associated with self-regulation (e.g., inferior frontal gyrus) when presented with reward, indicating that an individual may not be able to regulate their response to reward (Berner & Marsh, 2014; Cyr et al., 2016). In contrast, studies on reward learning in AN-restricting subtype samples indicate that these individuals are not as sensitive to reward, suggesting that individuals with AN-restricting subtype may be able to over-control food intake when presented with palatable foods (Frank et al., 2012; Frank et al., 2018; Haynos et al., 2018; Olsavsky et al., 2019). Specifically, individuals with AN-restricting type exhibit increased response in rewards areas (e.g., ventral striatum, insula) of the brain when presented with food rewards, indicating that only small amounts of rewarding stimuli may be needed, allowing individuals to engage in prolonged periods of food restriction (Frank et al., 2016; Frank et al., 2012; Frank et al., 2018). Thus, differential eating disorder symptoms may be exhibited based on predispositions for underactive (restriction) versus overactive (binge eating) reward learning.

Deficits in reward learning and fear learning may then be considered shared vulnerabilities for the development of both anxiety and eating disorder symptoms.

### ***Unique Neurobiological Mechanistic Process in Eating Disorders: Habit Learning***

In addition to these shared learning processes, an additional unique learning process has also been implicated in eating disorders: **habit learning**, specifically habit

learning around eating and food. Habit learning is defined as learned behaviors that over time through repetition become: 1) fixed, 2) automatic, 3) ordered or structured, and 4) routine (Graybiel, 2008; Seger & Spiering, 2011). Using a cognitive neuroscience perspective, Steinglass and Walsh (2006) and Walsh (2013) conceptualize AN as a disorder of maladaptive habitual behaviors that have become engrained and difficult to break. Specifically, Walsh (2013) proposes that in individuals with AN, dieting behavior becomes habitual, in that is fixed, repetitive, automatic, and generalized to various stimuli (Graybiel, 2008). It is theorized that restriction first becomes learned through classical conditioning, where individuals engage in dieting and receive positive feedback when weight is lost due to the thinness ideal (i.e., thin bodies are the ideal bodies) in society. Overtime, restriction is repeated but rewards are likely to become intermittent (e.g., people commenting on weight). Intermittent reinforcement leads to a further strengthening of these associations (Iversen, 1992), thereby strongly pairing restriction with reward. Eventually, these repetitive, rewarding behaviors become engrained as habits. Habit learning theory explains how learning may perpetuate eating disorders. Though the habit learning theory originally was only used to conceptualize AN, it has been applied to other eating disorder behaviors in addition to restriction, such as binge eating (Voon et al., 2015) and purging (Berner & Marsh, 2015) and thus appears to apply to all *symptoms* across the eating disorder spectrum.

Mechanistically, habit learning may interfere with eating disorder recovery by making extinction of learned information more difficult; in other words, once maladaptive patterns of eating are learned in eating disorders, neurobiological dispositions related to habit may make extinguishing these patterns in favor of more

adaptive, healthful patterns exceedingly difficult. *Extinction* of learned information occurs when a new association is learned that competes with the old association (Goodman & Packard, 2018). As behaviors shift from goal-driven to habitual, neural pathways reflect this shift: initially, the behavior is associated with activity in the prefrontal cortex and the dorsomedial striatum, whereas habits are associated with activity in the dorsolateral striatum (Amaya & Smith, 2018). Though other psychiatric disorders are also linked with habits, such as obsessive-compulsive disorder (OCD; Gillan et al. 2011), individuals with eating disorders may have a unique predisposition to develop eating habits that will help achieve goals related to desired weight and shape. Thus, understanding habit learning around eating may help differentiate individuals who develop anxiety and those who go on to develop comorbid anxiety-eating disorders.

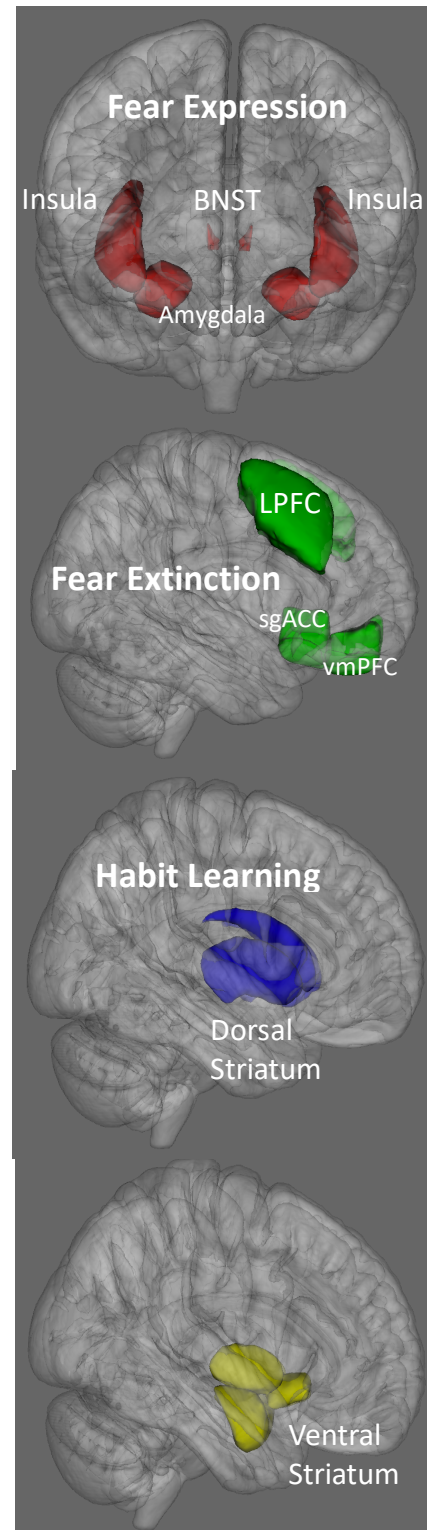
***Summary: Neurobiological Shared and Unique Processes in Comorbid Eating Disorder-Anxiety***

In summary, deficits in reward and fear learning may represent shared processes that create a generalized vulnerability for the development of psychopathology, such as anxiety symptomatology. Then, once the unique process of overactive habit learning around eating is present *in addition to* these deficient reward and fear learning processes, then comorbid eating disorder-anxiety may develop. Thus, though learning deficits have been implicated in the etiology of multiple mental disorders (e.g., anxiety disorders; Pittig et al., 2018), understanding specific learning deficits in the eating disorders can inform why these disorders are centered on overvaluation of shape and weight and develop in addition to anxiety.

Importantly, elucidating learning processes in comorbid eating disorders-anxiety disorders may lead to more effective treatments for these disorders. Indeed, understanding the mechanisms of aversive associative learning has informed the gold-standard treatment for anxiety disorders - exposure therapy (Holmes, Craske, & Graybiel, 2014; Pittig et al., 2018). Exposure therapy is a treatment based on the principles of learning that involves repeatedly exposing an individual to a feared stimulus in order to facilitate approach of the stimulus and overcome maladaptive fear and anxiety (Moscovitch, Antony, & Swinson, 2009). It has been shown that changes in aversive associative learning from exposure therapy correspond to symptom improvement (Helpman et al., 2016; Pittig et al., 2018). Thus, better understanding the shared and unique mechanisms underlying comorbid eating disorder-anxiety can help aid in the development of precision treatments for these disorders.

*Notes:* BNST = bed nucleus of the stria-terminalis; LPFC = lateral prefrontal cortex; sgACC = subgenual anterior cingulate cortex; vmPFC = ventral medial prefrontal cortex

Figure 2. Regions of the brain implicated in anxiety-EDs



## **A Learning Model of Comorbid Eating Disorders-Anxiety**

In order to understand these shared and unique learning processes, it is important to elucidate how they operate mechanistically. In other words, it is crucial to understand how impaired fear, reward, and habit learning come to develop in the first place and then which mechanisms maintain these learning deficits. In this regard, below, I first delineate the neurobiological vulnerabilities that correspond to fear learning, reward learning, and habit learning. Specifically, understanding which regions of the brain may be underactive or overactive in response to environmental stimuli may explain how individuals are conditioned and how this may result in the development of maladaptive learning (e.g., when fear stimuli are over-conditioned). Next, I delineate cognitive-behavioral mechanisms that maintain maladaptive learning processes once they develop by interfering with new learning and extinction of previous learned patterns. I discuss both shared cognitive-behavioral mechanisms, common across both anxiety and eating disorders, as well as cognitive-behavioral mechanisms that are unique and specific to eating disorders. Finally, I propose a learning model of comorbid eating disorders-anxiety integrating these mechanistic processes. The current study will test the cognitive-behavioral mechanisms implicated in the model, with future research needed to test the neurobiological mechanisms.

### ***Neurobiological Regions Implicated in Learning***

As mentioned above, predispositions from overactive or underactive neurocircuitry may create vulnerability for the development of altered fear learning, reward learning, and habit learning (see Figure 2). Specifically, the **amygdala, anterior insula cortex, and the hippocampus** have been implicated in fear acquisition and expression (Fullana

et al., 2016; Greco & Liberzon, 2016; Pittig et al., 2018). These areas display heightened activity in anxiety and eating disorders. For example, individuals with anxiety disorders show higher amygdala activation during fear acquisition compared to healthy controls (Bremner et al., 2005; Milad et al., 2009; Pittig et al., 2018). In addition, the anterior insula shows heightened activity in individuals with anxiety and eating disorders symptoms in relation to aversive stimuli, and it has been theorized that activation in the anterior insula predicts anxiety related to unpleasant cues (Gasquoine, 2014; Kerr et al., 2016; Nunn et al., 2011). The anterior insula is also essential for appetite regulation. Conversely, areas of the brain implicated in fear extinction show blunted responses in individuals with anxiety and eating disorders, meaning that once fear learning takes place, extinguishing the fear association is more difficult. Specifically, the **ventral medial prefrontal cortex [vmPFC], dorsal lateral prefrontal cortex [DLPFC], ventral lateral prefrontal cortex [VLPFC], and subgenual anterior cingulate cortex [sgACC]** are implicated in fear extinction (Greco & Liberzon, 2016; Pittig et al., 2018; Pourtois et al., 2013; Raber et al., 2019). For example, individuals with anxiety have deficits in vmPFC activation during extinction (McLaughlin et al., 2015; Mild et al., 2013; Pittig et al., 2018). There have been no studies, to my knowledge, examining which brain regions are implicated in fear extinction in eating disorders.

In addition to these areas implicated in fear learning, the **ventral striatum and orbitofrontal cortex (OFC)** play a central role in reward learning and are deficient in both individuals with anxiety disorders and eating disorders (Kaye et al., 2013;). For example, deficits in the dopaminergic neural circuitry in the mesolimbic pathway, including the ventral striatum and OFC (Kaye et al., 2013; Wagner et al., 2007), relate to

reward processes in eating disorders and anxiety. In addition, the anterior insula and OFC link to the ventral striatum, which forms reward predictions of food and eating (Kaye et al., 2013). Thus, these brain regions associated with fear and reward learning may represent shared neurobiological vulnerabilities to develop anxiety and eating disorders.

There may also be unique neurobiological vulnerabilities that predispose individuals to eating disorders. Specifically, the **dorsal striatum** is implicated in habit learning and shows heightened activity in individuals with eating disorders (Amaya & Smith, 2018; Goodman & Packard, 2018; Frank et al., 2013; Kerr et al., 2016; Nunn et al., 2011; Wagner et al., 2008). For example, individuals who have recovered from anorexia nervosa show lower activation of the ventral striatum, dorsal striatum, and insula compared to controls when consuming both sucralose and water (Wagner et al., 2008). Specifically, the dorsal striatum regulates automatic, context-driven stimulus-response pairings, meaning that behaviors are more likely to occur independent of higher-level thinking and may take significant cognitive control to interrupt (Goodman & Packard, 2018). Thus, if there is over-activation in the dorsal striatum around food cues, then individuals may be more vulnerable to developing habitual patterns around eating, such as habitual engagement in food restriction or binge eating.

Thus, neurobiological deficits for fear, reward, and habit learning may make individuals more likely to have higher or blunted responses to stimuli, as well as to develop habitual patterns around eating. These responses may then activate the development of cognitive and behavioral mechanisms that perpetuate aberrant learning processes. For instance, an individual who is prone to overactive fear responses may begin to worry about future potential events and also experience greater negative affect

than an individual who is less fearful. In turn, these cognitive and behavioral mechanisms may reinforce previous learning and prevent new learning through avoidance. Below, I first discuss two shared cognitive-behavioral mechanisms that interfere with adaptive learning processes. Then, I review two unique cognitive-behavioral mechanisms implicated in the development and maintenance of eating disorders.

### ***Shared Cognitive-Behavioral Mechanisms Related to Learning Deficits***

Shared cognitive-behavioral mechanisms are cognitive, affective, or behavioral correlates implicated across disorders, such as mood disorders, anxiety disorders, and eating disorders. Two shared mechanisms for eating disorders and anxiety that impact learning processes are: 1) **repetitive negative thinking**, and 2) **negative affect**.

*Repetitive negative thinking* is repeated thoughts about past or future events that focus on negative causes or consequences, including worry and rumination (Kertz, Stevens, & Klein, 2017; Nolen-Hoeksema & Watkins, 2011; Rawal, Park, & Williams, 2010).

*Negative affect* is the experience of negative emotions (Watson, Clark, & Tellegen, 1988; Wilamowska et al., 2010; Wonderlich et al., 2015). Repetitive negative thinking is a transdiagnostic process that operates as a core mechanism in most major mental disorders (e.g., McEvoy et al., 2013; Nolen-Hoeksema & Watkins, 2011; Spinhoven et al., 2015).

Indeed, repetitive negative thinking predicts higher eating disorder symptoms, both momentarily and prospectively (e.g., Rawal, Park, & Williams, 2010; Sala, Brosos, & Levinson, 2019), and is a central symptom in anxiety disorders (e.g., APA, 2013; Borkovec, Alcaine, & Behar, 2004; Mathews, 1990; Olatunji et al., 2010). Similarly, negative affect is a transdiagnostic mechanism across mental disorders (e.g., Brown, Chorpita, & Barlow, 1998; Mikolajewski et al., 2013). Negative affect also predicts



higher eating disorder symptoms momentarily and longitudinally (e.g., Berg et al., 2015; Bodell et al., 2019; Goldschmidt et al., 2014; Lavender et al., 2016; Wegner et al., 2002), as well as higher anxiety symptoms (e.g., Mor et al., 2010; Walz, Nauta, & Rot, 2014).

### **Repetitive Negative Thinking and Learning.**

Both repetitive negative thinking and negative affect may play a role in perpetuating aberrant learning once it has taken place. Repetitive negative thinking may interfere with corrective learning in the anxiety disorders, and it has been proposed that repetitive negative thinking similarly impedes new learning in the eating disorders (Reilly et al., 2018; Paulus & Stein, 2006). Specifically, repetitive negative thinking may increase negative affect and facilitate avoidance of thoughts and behaviors, which may then prevent individuals from learning that the feared stimulus is not as dangerous as previously thought or that the situation (and associated negative affect) can be tolerated (Ehring & Watkins, 2008; Paulus & Stein, 2006; Reilly et al., 2018). It is also possible that repetitive negative thinking uses considerable cognitive (e.g., attentional) resources that interferes with the ability to learn new information (Vanderhasselt, Kuhn, & De Raedt, 2011). Repetitive negative thinking processes are associated with activation in the DLPFC, which plays a role in extinction of learned information (Vanderhasselt et al., 2011; Raber et al., 2019), and the OFC, which processes reward (Buchholz et al., 2016; Kaye et al., 2013; Martin et al., 2009).

### **Negative Affect and Learning.**

Similarly, the experience of negative affect promotes avoidance of behaviors in order to avoid negative emotional states, which then prevents individuals from learning that a given situation is not as bad as previously thought or that the negative emotional state is

tolerable (Paulus & Stein, 2006; Reilly et al., 2018; Selby, Anestis, & Joiner, 2008).

Processing of negative emotionality during cognitive tasks is associated with activity in the OFC, amygdala, and LPFC (Melcher, Born, & Gruber, 2011).

Thus, it may be that these mechanisms (repetitive negative thinking and negative affect) impede extinction of learned information (e.g., aberrant fear and reward conditioning), thus maintaining internalizing symptomatology, such as anxiety.

Additional cognitive-behavioral mechanisms may also play a specific role in developing and maintaining eating disorder symptoms.

### ***Unique Cognitive-Behavioral Mechanisms Related to Learning in Eating Disorders***

Unique cognitive-behavioral mechanisms related to learning that confer specific risk for eating disorders are: 1) **thinness expectancies** (e.g., Annus, Smith, & Masters, 2008; Pearson & Smith, 2015; Smith et al., 2007), and 2) **habit formation** related to eating (Guarda et al., 2015; Steinglass & Walsh, 2006; Walsh, 2013). *Thinness expectancies* are the learned expectation that thinness is necessary for success in life (Pearson & Smith, 2015). *Habits* are behaviors that are learned, linked with specific contexts, automatic, and consistent (Gardner et al., 2012). Previous research shows that thinness expectancies in late pre-teen years and early adolescence predict which individuals will develop later eating disorder symptoms (Pearson & Smith, 2015).

Though there is less research on habit and eating disorders, emerging research suggests that habit formation plays a crucial role in the development and persistence of eating disorder symptoms over time (Davis et al., 2020; Walsh, 2013).

### **Thinness Expectancies and Learning.**

In general, learned expectancies are environmentally-learned associations based on classical and operant conditioning, as well as modeling (Hohlstein et al., 1998). In a society with a particularly narrow definition of beauty, thinness expectancies are often imbued at a young age, teaching children (particularly girls) that thinness is necessary in order to have social status, success, and romantic partners (e.g., Pearson et al., 2012; Stice & Shaw, 2002). Individuals with predispositions to deficits in reward learning may then be particularly vulnerable to rewards in the environment that reinforce thinness expectancies (e.g., receiving compliments based on appearance). These expectancies, in turn, along with reward learning may lead to the overvaluation of weight and shape, as well as engagement in eating disorder behaviors to maintain a thin body. Indeed, numerous studies have shown that thinness expectancies in conjunction with other cognitive-behavioral mechanisms (e.g., negative affect) are predictive of the development of eating disorder symptoms in across adolescence (e.g., Combs, Smith, & Simmons, 2011; Davis et al., 2018; Pearson et al., 2012; Pearson & Smith, 2015; Smith et al., 2007). Thus, thinness expectancies, along with shared vulnerabilities and predispositions for altered reward learning may make an individual prone to the development of an eating disorder in addition to an anxiety disorder.

### **Habit and Learning.**

In addition to thinness expectancies, habit learning around eating may serve as a unique vulnerability for the development of eating disorder symptoms. As mentioned previously, according to the habit theory of eating disorders (Steinglass & Walsh, 2006; Walsh, 2013), eating disorder behaviors, such as binge eating and restriction, initially

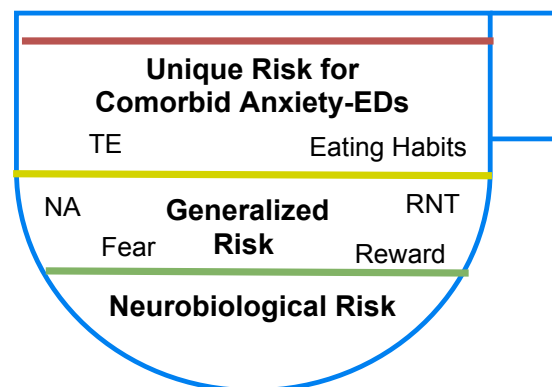
begin as goal-directed behaviors driven by the desire to have a certain weight and shape. Over time, these behaviors shift to become compulsive behaviors that are automatic and driven by environmentally conditioned stimuli (Gasbarri et al., 2014; Guarda et al., 2015). Recent research indicates that habit strength around eating behaviors is greater in individuals with AN versus healthy controls and that habit strength is associated with duration of illness (Davis, 2020). In addition, targeting habit strength in treatment predicts lower eating disorder symptoms after the intervention (Steinglass et al., 2018). Thus, the predisposition for overactive habit learning, particularly around food, along with exposure to an environment with highly prevalent disordered eating behaviors (e.g., dieting, restriction, binge eating) may lead these individuals to develop eating disorder symptoms. Thus, habit learning around eating may work in concert with other altered learning processes (i.e., reward and fear) to uniquely predispose an individual to develop eating disorder symptoms among people also at risk for anxiety.

By testing these neurobiological, shared, and unique mechanisms related to learning, it is possible to understand how mechanisms build upon one another to create additive risk for the development of comorbid eating disorder-anxiety symptoms.

***A Learning Model of Anxiety-Eating Disorder Symptoms***

Fear learning, reward learning, and habit learning can thus be used as a basis for an additive learning model of comorbid eating disorders-anxiety. Specifically, comorbid eating disorder-anxiety

Figure 1. Additive Risk Model for Anxiety- EDs



EDs = eating disorders; TE = thinness expectancies; NA = negative affect; RNT = repetitive negative thinking; Fear = fear learning; reward = reward learning

symptoms can be thought to arise as a result of an additive risk model. This model can be depicted metaphorically as a cup of water, in which each drop of water adds additional risk until a threshold is met for the development of this comorbidity (see Figure 1).

Biology (e.g., neural circuitry) predisposes individuals to be prone to altered fear, reward, and habit learning, as well as to develop cognitive-behavioral mechanisms (e.g., negative affect, repetitive negative thinking, habit formation around eating, and thinness expectancies) that further prompt development of both anxiety and eating disorders. As mentioned above, these mechanisms can be divided into two categories: 1) shared learning mechanisms implicated across a range of psychopathology, including anxiety (i.e., repetitive negative thinking and negative affect), and 2) unique learning mechanisms specific to eating disorders (i.e., thinness expectancies and habit learning around eating). It is not until all three forms of risk are compounded (i.e., neurobiological, shared, and unique) that this comorbidity develops. In this regard, each mechanism is necessary, but not sufficient in the model, meaning that each is needed for the comorbidity to develop but will not lead to comorbidity without the other risk vulnerabilities.

In sum, though the current literature has supported learning processes (i.e., fear learning, reward learning, habit learning) as a key mechanism in the development of eating disorders and eating disorder comorbidity, these learning mechanisms have largely been studied in silos, rather than as integrated, interwoven processes. Further, little work has been done to parse apart which cognitive-behavioral mechanisms may lead to divergent trajectories (i.e., anxiety disorder but not eating disorder symptoms) versus multifinality (i.e., comorbid anxiety and eating disorder symptoms). Thus, this model serves as a guide to begin to answer fundamental questions regarding the development of

eating disorder comorbidity, with the hope of improving precision interventions and prevention efforts.

### **The Current Study**

In the current study, I investigated the cognitive-behavioral mechanisms related to learning in comorbid eating disorders-anxiety. Seventy-three adolescents (13-15 years old) from the community completed self-report assessments at three time points: at the beginning of school year, the mid-point of school year (four-month follow-up), and the end of school year (nine-month follow-up). The transition from early-to-mid adolescence is a critical period for the development of eating disorder symptoms, and peak onset for eating disorders is between 14 and 19 years (e.g., Herpertz-Dahlmann, 2015; Volpe et al., 2016), allowing for investigation of a crucial time period for the development of EDs. Anxiety symptom onset often precedes the development of eating disorders (Godart et al., 2000; Swinbourne et al., 2014). This age is thus an ideal developmental period for examining the increase of eating disorder symptoms in addition to anxiety.

### ***Hypotheses***

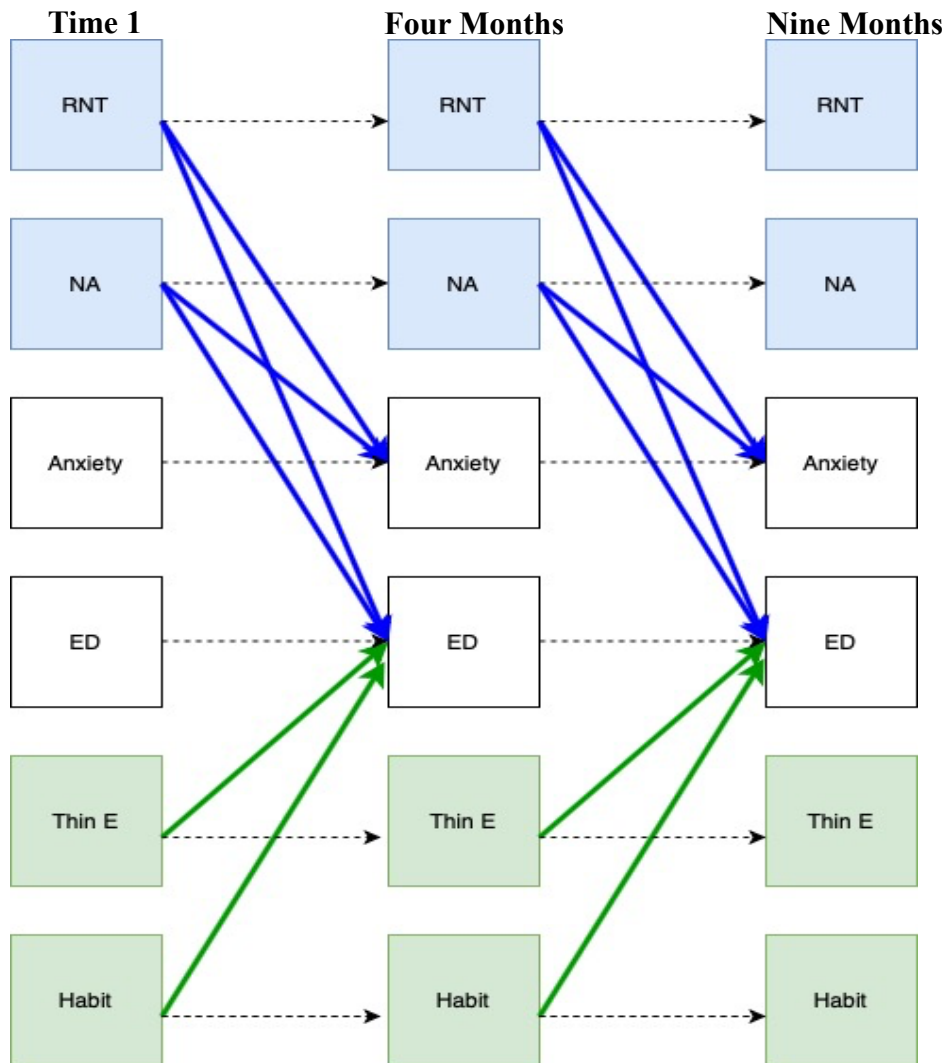
There are two primary research questions and corresponding hypotheses for the current study.

**Primary Research Question and Hypothesis 1.** The primary research question for the current study is: Do shared processes predict both anxiety symptoms and eating disorder symptoms nine-months later? In addition, do unique processes predict eating disorder, but not anxiety, symptoms nine-months later? I hypothesize (See Figure 3) that negative affect and repetitive negative thinking will be transdiagnostic mechanisms that confer risk for the development of both higher anxiety and higher eating disorders

symptoms at nine-month follow-up, while adjusting for baseline eating disorder and anxiety symptoms. Further, thinness expectancies and habit formation related to eating will represent unique mechanisms that predict higher eating disorder symptoms, but not anxiety symptoms, at nine-month follow-up, while adjusting for baseline eating disorder and anxiety symptoms.

**Figure 3**

*Primary Hypothesis 1*



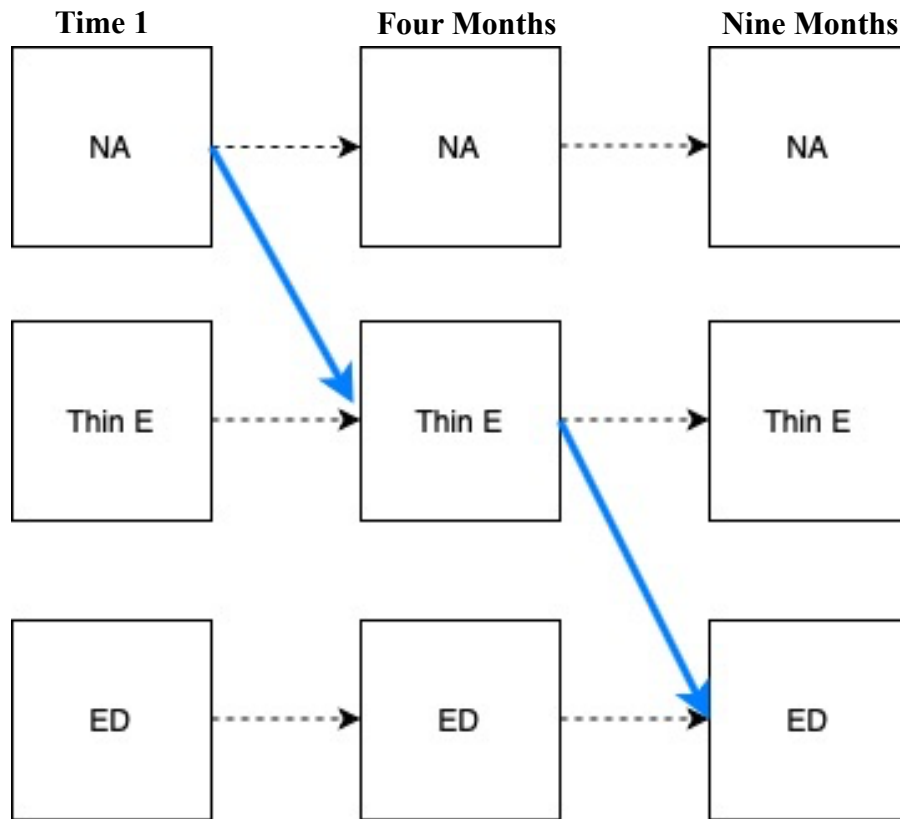
*Note:* RNT = repetitive negative thinking, NA = negative affect; ED = eating disorder symptoms; Thin E = thinness expectancies; habit = habit formation. Dashed lines represent autoregressive pathways. Blue lines represent hypothesized pathways between shared vulnerabilities (in blue squares) and anxiety and EDs; Green lines represent hypothesized pathways between unique vulnerabilities (in green squares) and EDs.

**Primary Research Question and Hypothesis 2.** Can previous models of eating disorder symptoms in adolescents be replicated, showing that unique mechanisms mediate the relationship between a shared mechanism and eating disorder symptoms? To test the hypothesis, I will seek to replicate a previous finding that thinness expectancies (unique) mediate the relationship between negative affect (shared) and eating disorder symptoms (Davis & Smith, 2018). Specifically, in a path model including thinness expectancies, negative affect, and eating disorder symptoms at baseline, four-months, and nine-months, I will test indirect paths from negative affect to eating disorder symptoms through thinness expectancies, while adjusting for autoregressive pathways over time. Further, through mediational testing, I will test thinness expectancies as a mediator between the relation between negative affect and eating disorder symptoms over nine-months. See Figure 4 for a visual representation of this hypothesis.



**Figure 4**

*Primary Hypothesis 2*



*Note.* NA = negative affect; ED = eating disorder symptoms; Thin E = thinness expectancies; Dashed lines represent autoregressive pathways. Blue lines represent hypothesized pathways.

## METHODS

### **Power**

For the total sample, 75 participants at baseline was calculated a priori to provide enough power to detect moderate effects after 10% estimated attrition (estimate effect size = 0.25, power = 80%, alpha = .05, five predictors).

### **Participants**

Participation took place in two separate waves. The first wave of participants completed the questionnaires in the 2019-2020 school year. The first two time points for Wave 1 participants were collected pre-COVID-19 pandemic (August 2019 and January 2020). The third time point was collected in May 2020, after the pandemic began, resulting in significant attrition (see below and **Results** for more information). The second wave of participants completed the questionnaire in the 2020-2021 school year. All participants were high school female freshmen between 13-15 years of age.

### ***Wave 1 Participants***

Forty-two participants participated in the first wave of data collection at Time 1. At Time 2, one participant withdrew from the study due to moving schools, leaving 41 participants. Of the 41 participants, 36 completed their surveys and five did not. At Time 3, of 41 participants, 22 participants completed their surveys. The mean age of participants in Wave 1 was 13.98 ( $SD = 0.35$ ). Participants reported their ethnicities as mostly White ( $n = 36, 85.7\%$ ). Participants self-reported the following diagnoses: Six generalized anxiety disorder (GAD), two social anxiety disorder (SAD), two obsessive-

compulsive disorder (OCD), one panic disorder, one post-traumatic stress disorder (PTSD), one specific phobia, two major depressive disorder, and five attention deficit/hyperactivity disorder (ADHD).

**Wave 2 Participants**

Twenty-eight participants participated in the second wave of data collection at Time 1. All participants completed their Time 2 surveys ( $N = 28$ ). At Time 3, of the 28 participants, eight participants did not complete their surveys. The mean age of participants in Wave 2 was 13.93 ( $SD = 0.38$ ). Participants reported their ethnicities as mostly White ( $n = 23$ , 82.1%). Two participants reported that had been diagnosed with an eating disorder, but did not specify the diagnosis. Participants self-reported the following diagnoses: one GAD, one SAD, one panic disorder, two unspecified anxiety disorders, and three ADHD.

For a direct comparison of Wave 1 and Wave 2 demographic variables please see Table 1:

**Table 1**

*Wave 1 and Wave 2 Demographic Characteristic*

Demographic Characteristic	Wave 1 ( $n = 42$ )		Wave 2 ( $n = 28$ )	
	<i>n</i>	%	<i>n</i>	%
Ethnicity				
African American	1	2.4	1	3.6
Asian American	3	7.1	1	3.6
Multiracial	0	0	2	7.1
Native American	2	4.8	0	0
White	36	85.7	23	82.1
Unreported	0	0	1	3.6
Mean Age (SD)	13.98 (0.35)		13.93 (0.38)	

### ***Total Sample***

The total sample was comprised of 70 high school females. The majority ( $n = 68$ ) of participants reported their gender as cisgender female, one participant reported their gender as transgender female, and one participant reported their gender as nonbinary. The mean age of all participants was 13.96 ( $SD = 0.36$ ). Two participants self-reported a clinical eating disorder diagnosis, and 18 individuals self-reported an anxiety disorder diagnosis. Mean body mass index (BMI) was 22.68 ( $SD = 7.24$ , Range = 14.58-63.09). Fourteen individuals reported BMIs below 18.5, indicating underweight status. Six individuals endorsed comorbid eating disorder-anxiety symptoms at baseline.

### **Measures**

#### ***Demographics***

Participants completed a demographic form with the following information: age, ethnicity, self-reported eating disorder diagnosis (“Have you been diagnosed with an eating disorder?”), self-reported anxiety diagnosis (“Have you been diagnosed with an anxiety disorder?”), whether they had any history of psychological treatment or psychiatric medication, whether they were currently taking birth control, and self-reported height and weight.

#### ***Family Affluence Scale-II***

The Family Affluence Scale-II (FAS-II; Boyce et al., 2006) assesses socioeconomic status (SES) in adolescents through four questions: “Does your family own a car, van or truck?”, “Do you have your own bedroom for yourself”, “During the past 12 months, how many times did you travel away on holiday with your family?”, and “How many computers does your family own?”. The FAS was designed to assess the

relationship between adolescent health outcomes and SES and evidences good internal consistency, test-retest reliability, and criterion validity. The FAS-II was administered at baseline in the current study and was measured to test whether any differences in SES were related to attrition. Internal consistency in this sample was poor ( $\alpha = .20$ ).

Therefore, rather than using the scale dimensionally, it was used ordinally, where scoring between 0-2 indicates low affluence, 3-5 indicates middle affluence, and 6-9 indicates high affluence. This ordinal scale was discussed in the development of the FAS-II as one way of measuring socioeconomic status (Boyce et al., 2006).

### ***Pubertal Development Scale***

The Pubertal Development Scale (PDS; Petersen et al., 1988) measures pubertal onset through five questions for girls (e.g., “Have you notice that your breasts have begun to grow?”). The PDS evidences strong validity and reliability (Coleman & Coleman, 2002). The PDS was used to determine whether girls were pre-pubertal or pubertal (scores above 2.5 indicating onset of puberty; Culbert et al., 2009; Davis & Smith, 2018), which has been shown to impact the development of ED symptoms in adolescent girls (Culbert et al., 2009). However, the PDS evidenced poor internal consistency in the current sample ( $\alpha = .31$ ). Due to poor reliability, puberty will be reported categorically (Yes/No) based on the question: “Have you begun to menstruate (started to have your period)?”

### ***Eating Disorder Examination-Questionnaire***

The Eating Disorder Examination – Questionnaire (EDE-Q; Fairburn & Beglin, 1994) assesses eating attitudes and behaviors over the past 28 days using 28 items. The global score was used as a measure of overall eating disorder symptoms in the current

study. The EDE-Q has been normed in adolescent females from the community, evidences good internal consistency and convergent validity, and can differentiate between individuals with and without a clinical eating disorder (Mond et al., 2011). The EDE-Q evidenced excellent internal consistency at baseline ( $\alpha = .97$ ), four-month follow-up ( $\alpha = .95$ ), and nine-month follow-up ( $\alpha = .96$ ) in the current sample. Fifteen (21.4%) individuals scored above a 2.0 on the EDE-Q, indicating that they endorsed experiencing a subclinical or clinical level of eating disorder symptoms at baseline (Carter, Stewart, & Fairburn, 2001).

### ***Spence Children's Anxiety Scale – Short Version***

The Spence Children's Anxiety Scale – Short Version (SCAS-S; Ahlen, Vigerland, & Ghaderi, 2018) is a 19-item short version of the originally 44-item SCAS (Spence, 1998) designed to measure symptoms of anxiety in children and adolescents. The shortened version of the SCAS was developed to decrease participant burden while retaining the strong psychometric properties of the original measure. The measure consists of five subscales each assessing different facets of anxiety: separation anxiety disorders, social anxiety disorder, panic disorder, specific phobia, and generalized anxiety disorder. The measure also yields an overall global score of anxiety symptoms. The SCAS-S evidences good convergent and divergent validity, similar to the original SCAS. The SCAS-S evidenced good internal consistency at baseline ( $\alpha = .86$ ), four-month follow-up ( $\alpha = .86$ ), and nine-month follow-up ( $\alpha = .85$ ) in the current sample. Twelve (17.1%) individuals scored above a 26.1 on the SCAS–S, indicating that they endorsed experiencing a subclinical or clinical level of anxiety symptoms at baseline.

### ***Repetitive Thinking Questionnaire***

The Repetitive Thinking Questionnaire (RTQ; McEvoy, Mahoney, & Moulds, 2010) is a 31-item questionnaire designed to assess repetitive negative thinking as a transdiagnostic process across psychiatric symptoms. The RTQ was psychometrically tested for use in adolescents (McEvoy et al., 2017). The RTQ evidences excellent internal consistency and convergent validity. The RTQ evidenced good internal consistency at all three time points in the current sample ( $\alpha s \geq .93$ ).

### ***Positive and Negative Affect Schedule***

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) is a 20-item questionnaire that assesses multiple aspects of trait positive and negative affect. The 10-items that comprise the negative affect subscale was used as a measure of trait negative affect. The PANAS has been shown to have good test-retest reliability and internal consistency. The PANAS evidences good psychometric properties in adolescent samples (Huebner & Dew, 1995). The PANAS evidenced adequate-to-good internal consistency at all three time points in the current sample ( $\alpha s \geq .83$ ).

### ***Thinness and Restriction Expectancies Inventory***

The Thinness and Restriction Expectancies Inventory (TREI; Hohlstein, Smith, & Atlas, 1998) is a 44-item questionnaire assessing expectancies that thinness and restriction lead to life success. The adolescent version of the TREI is an eight-item short form. The psychometric properties of the short-form TREI have been tested in adolescent females, evidencing excellent internal consistency and discriminant validity (Davis, Guller, & Smith, 2016; Pearson et al., 2015; Pearson et al., 2012). The TREI evidenced good-to-excellent internal consistency at all three time points in the current sample ( $\alpha s \geq .91$ ).

### ***Self-Report Habit Index***

The Self-Report Habit Index (SRHI; Verplanken & Orbell, 2003) consists of 12-item and assesses the habitual nature of a particular behavior. In the current study, it was used to assess habits around counting calories, a common disordered eating behavior among adolescents (Herpertz-Dahlmann, 2015). The SRHI evidences good internal consistency and has been used in adolescent samples (Kremers, van der Horst, & Brug, 2007). The SRHI evidenced good internal consistency at all three time points in the current sample for calorie counting as a habit ( $\alpha \geq .93$ ).

### ***Social Appearance Anxiety Scale***

The Social Appearance Anxiety Scale (SAAS; Hart et al., 2008) is a 16-item measure of fears of negative evaluation based on one's appearance (i.e., social appearance anxiety). The SAAS was used in the current study as a measure of social appearance anxiety part of the post-hoc analyses. The SAAS evidences good internal consistency and convergent validity (Hart et al., 2008). Internal consistency was excellent in the current study ( $\alpha = .98$ ).

### **Procedure**

Participants were recruited at one all-girls private high school in the southeastern United States. Participants and their parents were sent emails by their school counselor requesting their participation in a study on psychological mechanisms in adolescents. Once participants received parental consent and assented to participate, participants were enrolled in a nine-month longitudinal study, completing self-report questionnaires at baseline, four-month and nine-month follow-up. Self-report questionnaires assessed eating disorder symptoms, anxiety symptoms, repetitive negative thinking, negative



affect, thinness expectancies, and habits related to eating (see **Measures** above). Questionnaires were sent via email and administered through REDCap (Harris et al., 2009), a University-provided, HIPAA compliant questionnaire service. First, participants were sent online baseline self-report questionnaires, which took approximately 30 minutes to complete. Four months after completion of the baseline questionnaires, participants were sent online follow-up questionnaires with the same self-report measures as the baseline questionnaires. Finally, nine months after completion of the baseline questionnaires, participants were sent these same follow-up self-report questionnaires. Participants then received compensation for participation. All procedures were approved by the University of Louisville Institutional Review Board.

## **Data Analytic Plan**

### ***Primary Hypothesis 1***

For any missing data, multiple imputation using Multivariate Imputation by Chained Equations (MICE; van Buuren, 2018) in R was used to estimate missing item-level data (as long as data were missing completely at random [MCAR]). Imputing item-level data is preferable to scale-level data because it retains more participant data (Mazza, Enders, & Euehlman, 2015).

Using MPlus Version 8.0 (Muthen & Muthen, 1998-2014), bootstrapping and path model analyses were conducted using the maximum likelihood estimator with robust standard error (MLR) estimator for standardized path estimates. MLR was chosen because it is robust to non-normality (Muthen & Muthen, 1998). Though MLR also efficiently handles missing data (Muthen & Muthen, 2014), it only handles it at the scale-level (not item-level), which is why MICE was used above for imputation prior to

running further analyses (Mazza, Enders, & Euehlman, 2015). Model fit was evaluated using the: 1) comparative fit index (CFI; Bentler, 1990), 2) Tucker-Lewis incremental fit index (TLI; Tucker & Lewis, 1973), and 3) root mean square error of approximation (RMSEA; Steiger & Lind, 1980). For the CFI and TLI, values of 0.95 and above are considered excellent and values of 0.90 and above are considered adequate. For the RMSEA, values 0.05 and below are considered excellent, and values .08 and below are considered adequate (Hu & Bentler, 1999).

First, a cross-sectional path model was constructed to test whether negative affect, repetitive negative thinking, thinness expectancies, and habit formation were uniquely related (i.e., contributed unique variance, while adjusting for all other independent variables) to anxiety symptoms and eating disorder symptoms. Then, a prospective autoregressive model across the three time points (baseline, four-months, nine-months) was constructed to assess a comorbidity model of anxiety and eating disorder symptoms over time, while adjusting for baseline levels of symptoms.

### ***Primary Hypothesis 2***

Similar to **Primary Hypothesis 1**, MICE was used to impute missing data (as long as data were MCAR). Model fit was assessed using the CFI, TLI, and RMSEA. After establishing adequate model fit for a prospective path model including thinness expectancies, negative affect, and eating disorder symptoms at baseline, four-months, and nine-months, I tested for indirect effects from negative affect to eating disorder symptoms through thinness expectancies using bootstrapping (5000 iterations). Indirect effects are significant when confidence intervals do not include zero.

## RESULTS

### **Handling of Missing Data**

Seventy individuals participated in the current study at baseline; at time 2 (four-month follow-up), 64 individuals completed their surveys; and 42 individuals completed their surveys at time 3 (nine-month follow-up). At Time 1, there were 78 instances of missing data out of a total of 7,560 instances of item-level data, accounting for 1.03% of the data (under 20% has been shown to be ideal for multiple imputation; Dong & Peng, 2013). The data were missing completely at random (MCAR;  $\chi^2[1315] = 883.41, p > .05$ ), and thus, data were imputed using MICE, retaining all 70 individuals at Time 1.

At time 2, there were 1224 instances of missing data out of 7313 instances of item-level data, accounting for 16.7% of the data. The data were MCAR ( $\chi^2[1065] = 617.99, p > .05$ ), and thus were imputed, retaining a total of 70 participants.

At Time 3, due to higher than expected attrition, it was not possible to use MICE to impute item-level data for all cases, as conducted above for Time 1 and Time 2. Though MICE reliably estimates missing data even for large amounts of missingness (Madley-Dowd et al. 2019), it is not advisable to impute large amounts of missing data for cases with no observed data, such as the case with the 28 participants who did not complete the Time 3 assessment (Mazza et al., 2015). In other words, since these individuals did not complete any items at Time 3, using MICE at the item-level could result in greater-than-expected bias in estimates, as well as be extremely computationally demanding.

Thus, there are two different potential strategies for handling missingness in cases of high attrition: 1) not use MI for the 28 cases at Time 3 and run the model with missing data using MLR, which efficiently handles missing data (and which was already selected a priori; Dong & Peng, 2013; Muthen & Muthen, 2014); however, MLR does not impute the data and thus loss of power is still an issue, though bias that would otherwise occur from listwise exclusion is minimized; 2) use MICE to impute the 28 cases at the scale-level, with considerations of observed data from previous time points. Since MICE is able to handle larger amounts of missing data with minimal bias, it is suggested over listwise exclusion (which usually introduces large bias) (Madley-Dowd et al., 2019). For longitudinal data, it is further suggested that MICE is used with multi-level considerations (i.e., that within-person variance over the different time points is taken into account) (Hamidul et al., 2018). For transparency, both strategies were conducted and results are reported for both.

***Strategy 1: MLR with Missingness at Time 3***

Item-level data were not imputed for those who did not complete their surveys at this time point ( $n = 28$ ) because of the large amount of missingness. Rather, item-level data was only considered for imputation for the remaining 42 individuals. In this subsample, there were 68 instances of missing data out of a total of 4326 instances, accounting for 1.57% of the data. These data were also MCAR ( $\chi^2[344] = 39.99, p > .05$ ), and thus were imputed. Analyses then proceeded as outlined in the Data Analytic Plan. Maximum likelihood procedures for imputation have been found to be stable for up to 60% missingness (Dong & Peng, 2013). Thus, models run using this strategy included 70 cases at Time 1 and Time 2 and 42 cases at Time 3.

### ***Strategy 2: MICE at scale-level accounting for multi-level data***

Similar to Strategy 1, item-level data was not imputed for time 3; instead, scale-level imputation was performed, including the complete scale-level data from time points 1 and 2. The literature has supported combining multiple imputation strategies (e.g., item-level and scale-level) in order to avoid listwise deletion (Enders, 2017; Plumpton et al., 2016). There were 277 instance of missing data out of a total of 1680 instances, accounting for 16.49% of the data. These data were also MCAR ( $\chi^2[233] = 233.942, p = .470$ ), and thus were imputed. Analyses then proceeded as outlined in the Data Analytic Plan.

### ***Choosing a Strategy***

Promisingly, both strategies yielded the same results (albeit slightly different parameter estimates). Thus, the results from Strategy 1 (which utilized the originally proposed method of handling missing data) are presented here. The results from Strategy 2 can be found in Appendix B.

### **Differences Between Wave 1 and Wave 2**

Because data were collected in two different waves, differences between Wave 1 and Wave 2 participants in eating disorder symptoms and anxiety symptoms at all three time points were tested. At baseline, independent samples *t*-tests did not reveal significant differences between Wave 1 and Wave 2 participants for either eating disorder ( $t[68] = 0.13, p = .898$ ) or anxiety symptoms ( $t[68] = 0.37, p = .716$ ). Similarly, no differences were found in eating disorder symptoms ( $t[69] = 0.20, p = .841$ ) or anxiety symptoms ( $t[69] = 0.62, p = .537$ ) at Time 2 or ( $t[40] \leq 1.18, ps \geq .245$ ) Time 3.

## **Descriptive Statistics and Zero-Order Correlations**

Please see Table 2 for descriptive statistics and Table 3 for zero-order correlations for all variables at all three time points. Average eating disorder symptom scores were 1.10 ( $SD = 1.36$ ) at Time 1, 0.97 ( $SD = 1.08$ ) Time 2, and 1.20 ( $SD = 1.28$ ) Time 3. These scores obtained on the EDE-Q were similar to community norms of adolescent girls (Carter, Stewart, & Fairburn, 2001). Average anxiety symptom scores were 17.98 ( $SD = 9.02$ ) at Time 1, 16.89 ( $SD = 8.63$ ) Time 2, and 18.05 ( $SD = 8.17$ ) Time 3. These scores obtained on the SCAS-S were similar to community norms of adolescent girls (Ahlen et al., 2018). All variables were significantly correlated with one another ( $ps < .05$ ) except habits around eating. Interestingly, at all time points, habits around eating were not significantly associated with anxiety symptoms ( $rs \leq -.25, ps > .05$ ) and negative affect ( $rs \leq -.23, ps > .05$ ), and were significantly *negatively* correlated with eating disorder symptoms and thinness expectancies. It was determined from the PDS that six girls were pre-pubertal, and 64 (91.43%) participants had undergone puberty. In addition, 13 girls endorsed falling within the middle SES range and 57 (81.43%) participants endorsed falling within the high SES range. No participants indicated falling within the low SES range. All scores were standardized before conducting path analyses, as standardized parameter estimates allow for comparison within and between studies (Lleras, 2005).

### **Primary Hypothesis 1**

#### ***Cross-Sectional Shared and Unique Risk Model***

The cross-sectional model including repetitive negative thinking, negative affect, thinness expectancies, and habits around eating in association with eating disorder and anxiety symptoms was saturated (meaning that there were no degrees of freedom left in

the model to estimate fit; Bentler, 1990). A non-saturated model (i.e., with non-significant paths removed) was conducted to ascertain fit: the model displayed excellent fit: CFI = 1.00, TLI = 1.05, RMSEA = 0.00. Please see Figure 5 for a visual of the model and all paths results. As hypothesized, negative affect was positively associated with both eating disorder ( $b^* = .30, p = .002$ ) and anxiety symptoms ( $b^* = .40, p = .007$ ). However, repetitive negative thinking was only associated with anxiety symptoms ( $b^* = .32, p = .026$ ) cross-sectionally. Further, thinness expectancies ( $b^* = .63, p < .001$ ), but not habits around eating ( $p = .196$ ), were associated with eating disorder symptoms. Neither thinness expectancies nor habits around eating were related to anxiety symptoms ( $ps > .445$ ) cross-sectionally.

#### ***Prospective Shared and Unique Risk Model***

The prospective model including repetitive negative thinking, negative affect, thinness expectancies, and habits around eating, eating disorder and anxiety symptoms at all three time points was not able to be run due to low sample size and non-stable parameter estimates (Muthen & Muthen, 2014). Instead, the prospective model was run using the first two time points with the full sample size: this alternative model still displayed non-stable parameter estimates.

Thus, an exploratory model was run as an alternative that excluded habits around eating due to this variable being uncorrelated with all other variables. This new exploratory model included repetitive negative thinking, negative affect, thinness expectancies, eating disorder, and anxiety symptoms at baseline and at four month follow-up (Time 2); the model displayed perfect fit: CFI = 1.00, TLI = 1.00, RMSEA = 0.00, meaning that it was saturated. As with the cross-sectional model, non-significant

paths were removed to establish fit. Fit was excellent: CFI = 1.00, TLI = 1.05, RMSEA = 0.00. Please see Figure 6 for a visual of the model and Table 4 for parameter estimates.

Contrary to hypotheses, there were no shared predictors of anxiety and eating disorder symptoms. Repetitive negative thinking predicted anxiety symptoms ( $b^* = .36, p = .002$ ) prospectively at four months, while adjusting for baseline levels ( $b^* = .41, p < .001$ ) of anxiety symptoms. Only baseline eating disorder symptoms ( $b^* = .66, p < .001$ ) predicted later eating disorder symptoms at four months. Notably, though non-significant, thinness expectancies did display a moderate effect size ( $b^* = .20, p = .235$ ) predicting later eating disorder symptoms.

## **Primary Hypothesis 2**

### ***Replicating a Shared and Unique Vulnerability Model***

The prospective model including negative affect, thinness expectancies, and eating disorder symptoms at all three time points displayed poor fit, probably attributable to small sample size (Muthen & Muthen, 2014): CFI = .85, TLI = .60, RMSEA = .21. Thus, all results for this model were also considered exploratory. Please see Figure 7 for a visual of the model and Table 5 for all paths results.

Contrary to hypotheses, Time 1 negative affect did not significantly predict Time 2 thinness expectancies ( $b^* = -.20, p = .066$ ), though a moderate effect size was observed. Further, Time 2 thinness expectancies ( $p = .885$ ) did not significantly predict Time 3 eating disorder symptoms. Indirect effects from Time 1 negative affect to Time 3 eating disorder symptoms through Time 2 thinness expectancies were not significant,  $p = .802$ .



## **Post-hoc Analyses**

### ***Post-hoc Power Analysis***

Taking into consideration small sample size, it is unclear whether some of the parameter estimates are non-significant due to lack of power. It should be noted that post-hoc power analyses have been increasingly scrutinized for their utility and validity (e.g., Althouse, 2021; Hoenig & Heisey, 2001; Zhang et al., 2019). Thus, any post-hoc analysis should be interpreted with caution, particularly for more complex statistical analyses, including path analysis and mediation models. For example, Zhang and colleagues (2019) found that post-hoc power analyses do not provide reliable results about the ability to detect statistical significance, even in simplistic models. Therefore, it is sometimes recommended to perform a post-hoc power analysis on a **specific outcome** rather than an entire model. A post-hoc power analysis was conducted on the ability to detect significant predictors of later eating disorder symptoms: ( $N = 70$ , observed  $R^2 = 0.11$ ,  $\alpha = .05$ , five predictors) = 0.56. Taken with caution, the post-hoc power analysis indicates that this study was underpowered to detect effects for factors that predict eating disorder symptoms over four-months.

### ***Post-hoc Variance Analyses***

Another possibility contributing to non-significant findings is whether there was enough variance to be explained by the model. For instance, if Time 1 eating disorder symptoms explains 85% of the variance in Time 2 eating disorder symptoms, then there is only 15% total left to explain. It is then unlikely in this case that any one variable would explain enough variance to be significant in the model. One potential reason that there would be a low amount of variance to explain would be lack of time for change to

occur in symptomatology.

Thus, variance analyses were conducted to see how much variance in Time 2 and Time 3 eating disorder symptoms was explained by Time 1 eating disorder symptoms, as well as anxiety symptoms. Time 1 eating disorder symptoms explained 84.1% of the variance in Time 2 and 66.9% in Time 3 eating disorder symptoms. Time 1 anxiety symptoms explained 67.3% of the variance in Time 2 and 57.2% in Time 3 anxiety symptoms. Thus, eating disorder symptoms more strongly predicted later eating disorder symptoms and left less variance to be explained by other factors than anxiety symptoms.

### ***Differences in Retained versus Unretained Participants***

The higher than expected attrition, particularly in light of the COVID-19 pandemic, raised the question of whether individuals who completed all three time points (retained participants) were different than those who were lost to follow-up or withdrew from the study (unretained participants). Thus, independent *t*-tests were conducted to test whether retained versus unretained participants differed on eating disorder or anxiety symptoms at baseline. It was found that there were no differences between groups for either eating disorder ( $t[68] = 0.70, p = .491$ ) or anxiety ( $t[68] = -0.10, p = .924$ ) symptoms. In addition, because other demographic variables, such as socioeconomic status (SES), have been shown to influence attrition, an independent *t*-test was conducted to test differences in SES between groups; no differences ( $t[68] = -0.20, p = .841$ ) were found between the retained ( $M = 7.07, SD = 1.44$ ) versus unretained ( $M = 7.00, SD = 1.58$ ) groups.

### ***Post-hoc Moderation Analysis***

Because both negative affect and thinness expectancies were significantly

associated with eating disorder symptoms cross-sectionally, but not prospectively, it was thought that these variables perhaps had a moderating effect, rather than a mediating effect, on eating disorder symptoms. Thus, a moderation was conducted to test the interaction of negative affect and thinness expectancies in association with eating disorder symptoms. Baseline negative affect, thinness expectancies, as well as the interaction between negative affect and thinness expectancies on eating disorder symptoms at baseline were included in the model.

In this model, negative affect ( $b^* = .26, p = .001, \text{part } r = .21$ ), thinness expectancies ( $b^* = .54, p < .001, \text{part } r = .44$ ), and the interaction between negative affect and thinness expectancies ( $b^* = .22, p < .001, \text{part } r = .22$ ) were all uniquely related to eating disorder symptoms. Specifically, individuals higher in thinness expectancies were more likely to have higher eating disorder symptoms when also higher in negative affect, compared to lower negative affect. For individuals lower in thinness expectancies, negative affect did not affect the relationship between thinness expectancies and eating disorder symptoms. Please see Figure 8 for the interaction graph.

### ***Post-hoc Alternative Shared Vulnerability Factor***

In the prospective models, though exploratory, it was surprising that no variable emerged as a shared vulnerability factor for eating disorder and anxiety symptoms. It was hypothesized that another untested factor may be more potent in explaining both eating disorder and anxiety symptomatology. One factor that has consistently been found in studying this comorbidity is **social appearance anxiety**, or the fear of negative evaluation based on one's appearance (e.g., Brosos & Levinson, 2017; Levinson & Rodebaugh, 2012; Levinson et al., 2013; Koskina et al., 2011). Thus, a prospective model

was tested with social appearance anxiety, anxiety, and eating disorder symptoms at baseline and four-month follow-up. Fit for this model was saturated; after removing non-significant paths, fit was adequate-to-excellent: CFI = 0.98; TLI = 0.95; RMSEA = 0.09. Interestingly, social appearance anxiety ( $b^* = .26, p = .036$ ) significantly predicted anxiety, but not eating disorder symptoms ( $b^* = .01, p = .956$ ), accounting for baseline symptoms of anxiety ( $p < .001$ ). Once again, only baseline eating disorder symptoms ( $b^* = .71, p < .001$ ) significantly predicted later eating disorder symptoms. Please see Figure 9 for the full model and Table 6 for parameter estimates.

### ***Post-hoc Cross-sectional Model Replication***

Due to sample size limitations, it was not possible to run a stable model with all parameters at all time points. It is well-known that cross-sectional findings do not necessarily generalize to prospective models. However, it is possible to test whether cross-sectional findings replicate between time points, which may give us more confidence in our findings or information that symptoms may be differentially related to one another at different times. Thus, I tested an identical cross-sectional model (presented above) using the data at Time 2, rather than baseline.

The model including repetitive negative thinking, negative affect, thinness expectancies, and eating habits associated with anxiety and eating disorder symptoms was saturated; after removing non-significant paths, model fit was poor-to-good: CFI = .89, TLI = .73, SRMR = .09. Interestingly, when negative affect, though non-significant, was placed back into the model, fit improved to excellent: CFI = .99, TLI = .97, SRMR = .03. In this cross-sectional model at Time 2, only thinness expectancies ( $b^* = .56, p < .001$ ) was associated with eating disorder symptoms. In addition, only repetitive negative

thinking ( $b^* = .36, p = .003$ ) was associated with anxiety symptoms. Interestingly, negative affect (a shared correlate in the original cross-sectional model) was not associated with either eating disorder symptoms ( $b^* = .15, p = .183$ ) or anxiety symptoms ( $b^* = .22, p = .148$ ) at Time 2. Please see Figure 10 for full model results.

### ***Post-hoc Repetitive Negative Thinking Tests***

Interestingly, against hypotheses, repetitive negative thinking was not related to eating disorder symptoms at baseline, nor did it predict later eating disorder symptoms over time. Upon further investigation of the literature, it may be that repetitive negative thinking is differently related to different eating disorder symptoms, particularly in adolescent samples (Smith et al., 2018; Wong, Christian, & Levinson, 2021b). For instance, repetitive negative thinking is associated with binge eating, but not fasting or global eating disorder symptoms. Thus, I tested additional models: whether repetitive negative thinking was associated with 1) binge eating, or with 2) restricting behaviors, both cross-sectionally and over four months. The same cross-sectional and prospective models including repetitive negative thinking, negative affect, thinness expectancies, and anxiety symptoms as above were tested, but binge eating or restriction served as the eating disorder dependent variable rather than global eating disorder symptoms.

First, I tested the cross-sectional and prospective models with binge eating as the dependent variable. The cross-sectional binge eating model was saturated; after removing non-significant paths, fit was good: CFI = 1.00, TLI = 1.03, SRMR = 0.02. Similarly to the original eating disorder symptoms model, thinness expectancies was associated with binge eating ( $b^* = .32, p = .028$ ), and negative affect ( $b^* = .31, p = .053$ ) was marginally significant with a moderate effect size in association with binge eating. In addition,

repetitive negative thinking ( $b^* = .32, p = .026$ ) and negative affect ( $b^* = .40, p = .007$ ) were positively associated with anxiety symptoms cross-sectionally. Fit was also good for the prospective model: CFI = 1.00, TLI = 1.04, SRMR = 0.03. Prospectively, results also mirrored the original eating disorder symptom model, where only baseline binge eating symptoms ( $b^* = .89, p = < .001$ ) predicted later binge eating. Please see Figure 11 and Table 7 for the cross-sectional and Figure 12 and Table 8 for the prospective binge eating models, respectively.

Next, I tested the restriction models. Fit for the cross-sectional restriction model was saturated; after removing non-significant paths, fit was excellent CFI = .99, TLI = .99, SRMR = 0.02. Repetitive negative thinking ( $b^* = .36, p = .025$ ) was positively associated with restriction, while negative affect ( $p = .265$ ) and thinness expectancies ( $p = .999$ ) were not related to restriction cross-sectionally. Please see Figure 13 and Table 9 for the full model. The prospective model was saturated; after removing non-significant paths, fit for the model was adequate: CFI = .94; TLI = .88; RMSEA = .05. Repetitive negative thinking ( $b^* = .32, p = .023$ ), anxiety symptoms ( $b^* = -.36, p = .002$ ), and restriction ( $b^* = .66, p < .001$ ) at baseline predicted later restriction. Negative affect ( $p = .685$ ) and thinness expectancies ( $p = .352$ ) did not predict later restriction. Please see Figure 14 and Table 10 for the full model.

## DISCUSSION

### **Summary of Findings**

The current study was conducted to better understand shared and unique learning mechanisms contributing to the development of comorbid eating disorder-anxiety symptoms over the course of girls' freshmen year of high school, a critical period for the development of eating disorder symptoms (Herpertz-Dahlmann, 2015; Rojo-Moreno et al., 2015). It was hypothesized that shared learning mechanisms (i.e., negative affect and repetitive negative thinking) would relate to both eating disorder and anxiety symptoms cross-sectionally and prospectively. Additionally, it was hypothesized that unique learning mechanisms (i.e., thinness expectancies and habits around eating) would relate to eating disorder symptoms, but not anxiety disorder symptoms, cross-sectionally and over time. Finally, it was hypothesized that I would replicate a previous adolescent risk model, showing that thinness expectancies (unique) mediate negative affect (shared) and eating disorder symptoms, in support of a compounding shared and unique risk model.

Cross-sectionally, negative affect was the only shared factor associated with both eating disorder and anxiety symptoms. In addition, thinness expectancies was a unique factor related to eating disorder symptoms. Prospectively over four months, no shared vulnerability factor was found for comorbid eating disorder-anxiety symptoms. However, all prospective findings ended up being exploratory in nature due to limitations of sample size and higher than expected attrition due to COVID-19. Instead, repetitive negative thinking was the only factor that predicted later anxiety symptoms; the strongest predictor of later eating disorder symptoms in adolescents was earlier eating disorder symptoms.

Interestingly, post-hoc testing revealed that repetitive negative thinking was a shared predictor for both anxiety symptoms and restriction behaviors, but not for binge eating behaviors. Below I discuss these findings in more depth, as well as potential implications and limitations.

## **Unique and Shared Correlates of Eating Disorder-Anxiety Symptoms**

### ***Shared Cross-sectional Correlate***

Only negative affect was found to be a shared correlate for comorbid symptomatology. It is possible that affective factors are more salient in *comorbid* eating disorder-anxiety symptoms in this age group, whereas cognitive factors like repetitive negative thinking may be more specific to each disorder (Mikolajewski et al., 2013; Stice et al., 2021). Negative affect has been established as a transdiagnostic risk factor across different psychopathologies, including anxiety, eating disorder, depressive, and obsessive-compulsive symptoms (e.g., Mikolajewski et al., 2013;). Negative emotions may spur individuals to engage in behaviors to regulate negative affect (Paulus & Stein, 2006; Selby, Anestis, & Joiner, 2008), which depending on the individual, may get expressed in different ways, including avoidance (anxiety) and binge eating, purging, or restriction (eating disorder). Thus, this finding contributes to a robust literature on negative affect transdiagnostically.

In this study, repetitive negative thinking was only related to anxiety and not eating disorder symptoms. Interestingly, there is a dearth of literature on repetitive negative thinking related to eating disorders in adolescent girls (Smith et al., 2018; Wong, Christian, & Levinson, 2021b). The literature that does exist shows mixed results, where repetitive negative thinking may be specifically related to binge eating rather than fasting



or global eating disorder symptoms in younger populations. These findings may help explain why the current study did not find repetitive negative thinking to be a shared correlate.

### ***Unique Cross-sectional Correlates***

As hypothesized, negative affect and repetitive negative thinking were both associated with anxiety symptoms. Worry is one of the main etiological factors of generalized anxiety disorder and is a component of repetitive negative thinking (McEvoy et al., 2013; Olatunji et al., 2010). Worries about the future or ruminating about the past may spur anxiety, which then leads to overpreparation to “succeed” in a given situation, reinforcing continued worry about future situations (McEvoy et al., 2017). In addition, thinness expectancies was uniquely associated with eating disorder symptoms. Thinness expectancies may be associated with eating disorder symptoms because of the belief that thinness will bring happiness and success, thus motivating eating disorder cognitions and behaviors in order to control weight and shape (Combs, Smith, & Simmons, 2011; Davis et al., 2018; Pearson et al., 2012; Pearson & Smith, 2015; Smith et al., 2007). Thus, cross-sectionally, many of the hypothesized paths were supported.

### **Unique and Shared Risk Factors of Eating Disorder-Anxiety Symptoms**

I did not find that these cross-sectional relationships remained significant over time as predicted. These findings reinforce previous research showing that cross-sectional models do not necessarily generalize to prospective models (e.g., Jacobi et al., 2004). It also supports evidence that the strongest predictor of later eating disorder symptoms in adolescents is earlier eating disorder symptoms (e.g., Davis et al., 2016; Herle et al., 2020). Indeed, baseline eating disorder symptoms explained almost 85% of the variance

of later eating disorder symptoms over four months, limiting the variance to explain with additional factors. Eating disorder symptoms may need to be investigated at an even younger age to understand what spurs development, or over a longer course of development (Bufferd et al., 2022). Indeed, onset of eating disorders is occurring earlier (Favaro et al., 2009; Murray et al., 2022), and there is often a spike in eating disorder symptoms around the start of puberty (Klump, 2013), which is also occurring earlier (Eckert-Lind et al., 2020). The median age of puberty onset in girls in the United States is estimated to be between 8.8 and 10.3 years (Eckert-Lind et al., 2020). The current sample was almost entirely post-pubertal, potentially indicating that the early onset of eating disorder symptoms was missed. In addition, we do not necessarily understand when many of these shared and unique factors develop (e.g., Stice, 2016); previous research has examined negative affect and thinness expectancies as important developmental risk factors for psychopathology but less is known about repetitive negative thinking, habits around eating, and social appearance anxiety in adolescent populations (Christian et al., 2019; Smith et al., 2018; Walsh, 2011). However, all prospective findings ended up being exploratory in nature due to limitations of sample size and higher than expected attrition due to COVID-19. Thus, these results are potential indicators for future research rather than confirmatory findings.

### **Replication of Shared and Unique Vulnerability for Eating Disorders**

These mechanisms were also investigated in a mediation model to elucidate whether negative affect leads to thinness expectancies which then leads to eating disorder symptoms, which would replicate findings of a previous model of disordered eating risk in adolescents (Davis & Smith, 2019). Surprisingly, neither thinness expectancies, nor

negative affect predicted later eating disorder symptoms at any time. Rather, eating disorder symptoms were the only predictor over time in the model, predicting negative affect at nine-month follow-up. This persistence of eating disorder symptoms over time indicates that without treatment, these symptoms are only likely to continue, and early treatment is imperative to reduce the impact of eating disorder symptoms in adolescent girls' lives. In addition, negative affect displayed a moderate effect size in association with thinness expectancies. These findings depart from a large body of literature supporting the relationship between thinness expectancies, negative affect, and eating disorder symptoms (e.g., Combs, Smith, & Simmons, 2011; Davis et al., 2018; Pearson et al., 2012; Pearson & Smith, 2015; Smith et al., 2007).). The current tested model may have been affected by lack of power to detect significant effects. This model also needs to be interpreted with caution due to its exploratory nature.

## **Post-hoc Investigation**

### ***Moderating Correlates***

Due to the association of both negative affect and thinness expectancies with eating disorder symptoms, and the surprising lack of relation over time, it was investigated whether thinness expectancies moderated the relationship between negative affect and eating disorder symptoms. This moderation effect was supported, in that individuals who experience higher negative affect are *more likely* to engage in eating disorder behaviors if they *also* are high in the belief that thinness will bring happiness (compared to individuals with lower thinness expectancies). The relationship between negative affect and eating disorder behaviors may be amplified by thinness expectancies because individuals higher in thinness expectancies may specifically try to regulate their

negative emotions by controlling their shape and weight (rather than engaging in a different behavior to regulate negative emotions), making it even more likely that eating disorder behaviors will occur. These conclusions should be investigated in future research, particularly if this finding may replicate longitudinally in a different sample.

### ***Alternative Shared Vulnerability Factor***

Finally, post-hoc testing also failed to elucidate a shared vulnerability factor for eating disorder and anxiety symptoms. Social appearance anxiety has consistently been found as a possible connecting mechanism between eating disorder and anxiety symptoms (e.g., Brosf & Levinson, 2017; Levinson & Rodebaugh, 2012; Levinson et al., 2013; Koskina et al., 2011), and thus an alternative model with social appearance anxiety was tested. Surprisingly, in the model, social appearance anxiety only predicted later anxiety, not eating disorder symptoms. It is possible that the study timeframe (i.e., four months) was not long enough to capture this relationship. Indeed, some previous studies investigating social appearance anxiety have been six-months to a year (Brosf & Levinson, 2017; Levinson & Rodebaugh, 2012; Levinson et al., 2013). Another potential explanation is lack of power to detect significant effects. Future research should continue to investigate other possible shared factors for comorbid eating disorder-anxiety symptoms.

### ***Cross-sectional Model Replication***

Because of limited power to detect effects, an additional cross-sectional model at Time 2 was conducted to investigate whether it replicated the cross-sectional model at baseline. Overall, the cross-sectional model did replicate: repetitive negative thinking was associated with anxiety symptoms, and thinness expectancies was associated with eating

disorder symptoms. Though negative affect was not significantly associated with both outcomes in this replication model, interestingly, model fit was poor when negative affect was removed from the saturated model and became excellent when added back in. Additionally, negative affect had moderate effect sizes in association with anxiety and eating disorder symptoms, suggesting that the model may have been underpowered to detect this association. This replication provides further confidence in our cross-sectional findings and supports further prospective testing of these relationships with larger sample sizes and more power.

### ***Repetitive Negative Thinking Tests***

As mentioned above, though repetitive negative thinking has not been extensively investigated as a predictor of eating disorder symptoms in adolescents (Smith et al., 2018; Wong et al., 2021b), the literature that does exist indicates that repetitive negative thinking may not be associated with global eating disorder symptoms, per se, but rather specific behaviors, such as restriction. It may be that if repetitive negative thinking is associated only with certain behaviors, then its association with overall eating disorder symptoms may be obscured. In order to test this hypothesis, post-hoc tests were conducted testing models with binge eating or restriction as the eating disorder outcome, rather than global eating disorder symptoms. In line with previous literature, repetitive thinking was not associated with binge eating behaviors (and the binge eating model had the same results as the global eating disorders model); however, repetitive negative thinking was associated with restriction cross-sectionally and predicted restriction prospectively, along with anxiety symptoms.

It may be that repetitive negative thoughts regarding shape and weight motivate

an individual to restrict their food intake to prevent their feared outcome. However, restriction may only temporarily relieve these thoughts, which then return and thus maintain restriction over time. In terms of binge eating, it is possible that other factors more robustly predict these behaviors over time. For instance, previous research indicates that impulsivity (i.e., negative urgency, or impulse behavior in reaction to negative emotions) may play more of a role in binge eating than restriction behaviors (e.g., Davis et al., 2018; Pearson & Smith, 2015; Smith et al., 2007). It is also possible that restriction mediates the relationship between repetitive negative thinking and binge eating. In eating disorder models, restriction almost always precedes a binge eating episode (e.g., Fairburn, 2008). In order to test this hypothesis in the future, more time points with a larger sample would be necessary.

Though these findings are exploratory in nature, they suggest that future research may want to study comorbidity models focusing on specific *behaviors*, rather than global symptomatology, or diagnoses. Indeed, there has been a push in the eating disorder field to conceptualize eating disorders as dimensional on binge-eating-type behaviors and restriction behaviors (e.g., Culbert et al., 2015; Forbush et al., 2017). Future prospective models should be tested to see whether the finding of repetitive negative thinking as a shared vulnerability factor for anxiety and restriction symptoms replicates, as well as test other potential shared vulnerability factors predictive of binge eating and anxiety symptoms.

### **A Shared and Unique Risk Model of Comorbid Eating Disorder-Anxiety Symptoms**

Taken together, these findings may inform future tests of a model incorporating shared and unique risk factors for comorbid eating disorder-anxiety symptoms. In terms

of cross-sectional findings, these results were in line with previous studies investigating eating disorders in adolescents. For instance, Pearson and colleagues (2015) found that negative affect and thinness expectancies predict the development of eating disorder symptoms over four years from middle school to early high school. Thinness expectancies may serve as a crucial learning mechanism that interacts with and/or mediates shared factors to be expressed as eating disorder symptoms (e.g., Pearson et al., 2012). One of the main questions that the field continues to investigate is why eating disorders manifest from many other risk factors that generalize across internalizing disorders (e.g., Smith & Keel, 2017). Thinness expectancies, along with other promising factors, like social appearance anxiety, should continue to be investigated as possible mechanistic links from shared factors to comorbid eating disorder symptoms. Negative affect has been extensively studied as a vulnerability factor shared across internalizing disorders (e.g., Brown et al., 1998; Mikolajewski et al., 2013). Negative affect promotes the avoidance of behaviors that lead to negative emotional states, which then prevents individuals from learning that a given situation is not as bad as previously thought or that the negative emotional state is tolerable (Paulus & Stein, 2006; Reilly et al., 2018; Selby, Anestis, & Joiner, 2008). However, research has also begun to question whether a *specific facet* of negative affect may be more related to certain disorders. For instance, shame is a facet of negative affect that contributes to the maintenance of eating disorder symptoms (Wong et al., 2021a). It may be that in this short of a time period, looking at these specific facets may have led to more robust findings. Future research should continue to study momentary and longitudinal effects of negative affect on eating disorder-anxiety comorbidity, including elucidating the type of negative affect (e.g.,

shame) that may be contributing to comorbid symptomatology.

Interestingly, repetitive negative thinking and habits around eating did not relate to global eating disorder symptoms as hypothesized, either cross-sectionally or prospectively. Though repetitive negative thinking has been implicated in eating disorders (e.g., Rawal, Park, & Williams, 2010; Sala, Brosos, & Levinson, 2019), the literature suggests that repetitive negative thinking *around eating, weight, and shape* (e.g., worries around eating too many calories, or rumination about a past meal) may be more important for the maintenance of global eating disorder symptoms than general repetitive negative thinking (e.g., worries about school work or ruminating about a social situation) (Sala et al., 2019; Wong et al., 2021b). Thus, it is possible repetitive negative thinking broadly is related to anxiety symptoms, whereas repetitive negative thinking about eating, weight, and shape may be shown to be as a *unique* maintenance factor for global eating disorder symptoms. Unfortunately, repetitive negative thinking specifically around eating, weight, and shape was not assessed in the current study. It may also be possible that repetitive negative thinking may simply not relate to global eating disorder symptoms due to its association with some eating disorder behaviors (i.e., restriction) and not others (i.e., binge eating), as mentioned above. Future research should seek to measure both aspects of repetitive negative thinking to further parse out how this mechanism contributes to comorbidity, as well as specifically to different eating disorder symptoms.

In addition, habits around eating did not relate to eating disorder symptoms either cross-sectionally or over time. Similar to repetitive negative thinking, habits around eating has largely been studied as a maintenance mechanism of eating disorder



symptoms, rather than as a risk factor. Indeed, Walsh (2013) proposed that habits around eating develop over time as an eating disorder progresses. For instance, habits may develop when behaviors such as restriction are repeated and become automatic and fixed over time. Theoretically, habits around eating was posited as a potential explanation as to why eating disorders are persistent, difficult to treat, and relapse occurs frequently. Because the current study investigated a community sample of adolescents at a critical period for the *development* of eating disorder symptoms, it is more than possible that eating disorder behaviors (i.e., calorie counting, skipping meals) were not present long enough to become engrained as habits and thus were not related to eating disorder symptoms more generally. Indeed, a recent review found that eating disorder symptoms are not related to a predisposition to greater habit development (Schaefer & Steinglass, 2021). In addition, the current study is the first test of the Self-Report Habit Index specifically applied to calorie counting. It is possible that other forms of measurements may better capture habits around eating, especially regarding different forms of restriction (i.e., calorie counting, fasting, skipping meals, etc.) to better assess the heterogeneity of eating disorder symptoms.

In sum, due to the exploratory nature of the current study, no definitive conclusions can be made regarding a shared and unique risk model of eating disorder symptoms. However, the study provides preliminary evidence that negative affect, repetitive negative thinking, and thinness expectancies should continue to be investigated as shared and unique learning correlates of comorbid eating disorder-anxiety symptoms. Other investigations should also inform future model testing, including what other shared and unique factors may play etiological roles in the development of this comorbidity.

### *A Note on Effect Sizes*

As discussed above, a post-hoc power analysis indicated that there was not enough power to detect predictors of later eating disorder symptoms. Although this power analysis should be interpreted with caution (Althouse, 2021; Hoenig & Heisey, 2001; Zhang et al., 2019), it is very likely that power was an issue in the current study due to the final sample size and greater than expected attrition. When power is low,  $p$ -values may not be the best indicator of true effects due to Type II error (Sullivan & Feinn, 2012). Rather, effect sizes may be better indicators of whether a particular variable might be a true predictor if power was adequate. Though these effect sizes should be interpreted with extreme caution, due to the ultimately exploratory nature of these models, it is worth exploring which predictors with moderate-to-large effect sizes, in an effort to inform future studies. Specifically, though non-significant, baseline thinness expectancies had a moderate association with later eating disorder symptoms. Thinness expectancies was significantly related to eating disorder symptoms in the cross-sectional model, and there is extensive literature linking earlier thinness expectancies with the development of later eating disorder symptoms (e.g., Combs, Smith, & Simmons, 2011; Davis et al., 2018; Pearson et al., 2012; Pearson & Smith, 2015; Smith et al., 2007). Thus, it may be useful for future studies to continue to investigate the prospective relationship between thinness expectancies, eating disorder symptoms, and other potential shared predictors of both eating disorders and anxiety symptoms.

### **Strengths and Limitations**

The current study should be considered in the context of its limitations. First and

foremost, data from both waves of this study were collected during COVID-19, and thus represent an atypical time period. Students were largely engaging in virtual learning and normative social and daily routines were significantly disrupted. Thus, it is unclear how changes in symptomatology can be generalized to non-pandemic development. In addition, the COVID-19 pandemic led to much higher attrition than expected (closer to 40% rather than the anticipated 10%), mostly due to onset of COVID-19 at the end of Wave 1 data collection (before Time 3). Though no differences were found in individuals who were lost to follow up versus those who completed the study on anxiety and eating disorder symptomatology at baseline, it is possible that there were other important differences that were not assessed to explain attrition. Additionally, even though SES was assessed, internal consistency in the current sample was poor, indicating that the measure used was not reliable in this sample. Thus, it is possible that SES was still a factor in attrition and should be more thoroughly examined in future research. Further, because of higher than expected attrition, power was somewhat limited for the proposed prospective modeling tests, especially for mediation analyses, which yield smaller effect sizes and thus require larger sample sizes (Thoemmes, MacKinnon, & Reiser, 2010). Thus, it is possible that some significant effects were not detected in the current study.

Another notable limitation of the current study is the homogeneity of the sample in terms of race, ethnicity, SES, and gender. The sample consisted of majority white, affluent individuals. Data were collected from an all-girls private high school for feasibility of recruitment but limited the ability to obtain a diverse sample. Thus, the current sample may not be entirely generalizable to all high school freshmen females. In addition, an only-female sample was collected due to higher risk for females developing

eating disorder symptoms and for feasibility (the sample size would have had to be doubled to test gender effects); however, the current study's findings are not generalizable to males, and may be different among non-binary or transgender individuals.

Further, this sample consisted of a community sample, which was ideal for the aims of this study; understanding development of comorbidity requires a heterogeneous sample of both low and high symptomatology to be able to track change over time (Pearson, 1903; Taylor et al., 2006). However, only a small percentage of the sample endorsed clinical symptomatology, which may make detecting significant effects difficult. In addition, the results of this study cannot be generalized to maintenance factors of psychopathology, which may be better captured in a clinical sample.

Another limitation is the length of follow-up used in the study; Nine-month follow-up is a notable length of time, but ideally, we need to understand how these processes are developing over longer periods of time. It would be ideal to capture the shift from disordered eating to clinical eating disorder symptoms over the course of several years (Davis & Smith, 2018; Pearson et al., 2012; Pearson et al., 2015). It may also be useful to utilize more frequent measurement (e.g., ecological momentary assessment) in order to capture dynamics between factors over shorter periods of risk (e.g., Mason et al., 2019; Smyth et al., 2001). In addition, though longitudinal methods can capture temporal precedence, they cannot explain causality – only experimental studies can elucidate causal mechanisms of eating disorder comorbidity (Cole & Maxwell, 2003).

Finally, only self-report measures were used. Unfortunately, COVID-19

precluded the ability to collect behavioral and functional magnetic resonance imaging (fMRI) data. Self-report measures possess inherent limitations, including retrospective and social desirability bias (Trull & Ebner-Priemer, 2009). A more accurate understanding of developmental mechanisms of eating disorder comorbidity require use of multiple levels of analysis (Culbert et al., 2014), including neural, behavioral, and self-report assessments.

Even within the context of these limitations, this study has significant strengths; perhaps most importantly, few studies have tested the development of comorbid symptomatology or comorbidity models (Brososof, Caleb, & Levinson, 2019). As psychopathology is inherently heterogeneous and comorbidity is the rule rather than exception (e.g., Blinder et al., 2006; Levinson et al., 2018), we should utilize methods and analytic techniques that capture real-world conditions of how mental disorders present. Thus, this study is an important preliminary step in creating and rigorously testing models of comorbidity. Second, this study used a longitudinal design, with follow-up at three time points; longitudinal studies are sorely needed in the eating disorders to better understand mechanisms of development and inform precision interventions (e.g., Stice, 2016). The use of three time points is also critical to be able to test mediation pathways (Cole & Maxwell, 2003; Maxwell & Cole, 2007) and track change over time. Finally, this study introduces and tests a novel theoretical model that seeks to integrate two previous separate lines of research in the eating disorders. This theoretical model can be used for future research to elucidate the development and maintenance of eating disorder comorbidity.

### **Future Directions and Implications**

The current study was an initial test of a novel, integrated theoretical learning model of comorbid eating disorder-anxiety symptoms in adolescence. Though the prospective findings were largely exploratory, this study can still be used for the development of future research. Specifically, the findings can inform hypothesis generation for confirmatory studies, as well as provide guidance to improve upon methodological limitations in the current study.

First, it will be essential to test the proposed integrated theoretical learning model with larger sample sizes that are more diverse, and that are designed with a longer follow-up time (i.e., several years). Ideally, future research would incorporate large enough samples to yield a sizeable subsample with subclinical and clinical-level symptomatology to be able to detect small effects, such as in mediation (Thoemmes et al., 2010). In addition, to make the findings more generalizable, it is important to include adolescents that represent different racial, ethnic, and social economic status groups adequately. This consideration of racial diversity is particularly relevant because disordered eating behaviors and cognitions differ across race and ethnicity (Levinson & Brosf, 2015). For example, African American women may endorse lower levels of body dissatisfaction and may engage in more binge eating behavior than White women (Levinson & Brosf, 2016). Further, low SES is associated with higher risk for the development of eating disorder symptoms (Accurso, Buckelew, & Snowden, 2021; Huryk, Drury, & Loeb, 2021), potentially due to food insecurity (Becker et al., 2017; Christensen et al., 2021). It will also be important in future research to investigate potential differences in this integrated learning model across genders, including males and non-binary/transgender folks. Importantly, eating disorder symptoms present differently across different genders

(Gordon, Moore, & Guss, 2021; McClain et al., 2016; Murray, 2017). For instance, males have higher focus on drive for muscularity (Murray, 2017), and transgender/non-binary folks have higher rates of eating disorders than their cisgender counterparts (Gordon et al., 2021). Thus, future samples would need to be large enough to test differences between gender groups.

Larger samples would also enable testing of mediational pathways, which were not able to be tested due to only being able to include two time points (Cole & Maxwell,). In the current study, there was not enough power to test all parameters at all time points, thus creating an unstable model that is not reliable. Testing over three time points (baseline to four months and four months to nine months) would better illustrate how these variables relate over time. Understanding temporal precedence of how mechanisms affect comorbidity is imperative for creating precision intervention by elucidating which mechanism to target. In addition, larger sample sizes would allow for testing of invariance between waves, which was not possible in the current study due to inadequate sample size (< 30) in Wave 2. In order to test invariance, at least 30 participants per group is necessary to have enough power to detect significant effects, though even larger sample sizes might be needed to test models with many parameters. Though I tested whether eating disorder and anxiety symptoms at all time points were different between waves, it would be useful to see whether the entire model is invariant across waves (or other groups, such as different genders or ethnicities). Testing model variance allows for capturing differences in comorbid outcomes with all variables, rather than only testing one variable at a single time point. Thus, these tests may show differences that are not able to be captured by *t*-tests. In the future, if comorbidity models are shown to differ by

gender or ethnicity, then it may indicate different interventions targets for different groups, helping to refine our interventions.

Second, future research should capture earlier versus later transitions to better understand developmental processes of eating disorder comorbidity (e.g., Stice, South, & Shaw, 2012). As mentioned above, age 13-15 represents a critical period for development of eating disorder symptoms; however, puberty is a key part of this critical period (Culbert et al., 2009; Klump, 2013), as hormonal changes undergone during this time can prime risk in combination with environmental factors (e.g., dieting, body dissatisfaction; Stice, 2016), particularly in individuals already genetically predisposed to develop an eating disorder (Culbert et al., 2015). Puberty can onset earlier than 13 (Pearson et al., 2012; Stice et al., 2011a; Stice, 2016), and thus future research may want to begin study at even earlier ages (i.e., 11) through 15 or 16 years of age, in line with need for longer periods of follow-up. Indeed in the current sample, 64 out of 70 individuals had already undergone puberty at baseline. Thus, studying adolescents earlier, even in pre-adolescence, and following them for a longer period of time may yield better information about development of eating disorder comorbidity, particularly with the ability to adjust for or include hormonal indicators in future models. Finally, since many comorbidities develop before eating disorders, including anxiety disorder symptoms (e.g., Swinbourne et al., 2012), studying younger samples with longer follow-up may also help better elucidate shared versus unique risk factors in eating disorder comorbidity.

Third, as mentioned in the Strengths and Limitations section above, including both objective behavioral and self-report assessments is essential to accurately capture developmental processes, particularly around fear and reward learning. For instance, the



FLARE app is a mobile app that was developed to study fear acquisition, generalization, and extinction processes in real-world, rather than laboratory, settings and evidences good psychometric properties similar to comparable lab tasks (Purves et al., 2019). Such behavioral tasks can be administered at each time point to map onto self-report data. By collecting longitudinal objective data, as well as self-report data, we can be more confident in our findings regarding vulnerability mechanisms that need to be targeted in precision interventions of eating disorder comorbidity.

Fourth, future research should also seek to formalize an integrated learning theory using computational modeling (Fried, 2020). Formal models are those represented mathematically and yield predictions about how data would look if those theories are borne out (Haslbeck et al., 2021). Importantly, these predictions often do not map onto what researchers expect to see in the data (Fried, 2020). Thus, instead of mapping the data onto our theories, we can directly compare our predicted model versus observed model, and update our theories accordingly. Learning is studied extensively in other areas of mental health, such schizophrenia, depression, and anxiety, using computational models (e.g., Anticevic, Murray, & Barch, 2015; Bishop & Gagne, 2018). These models are also able to integrate multiple levels of analysis, including predictions from behavioral, neural, and self-report assessments (Anticevic et al., 2015). Despite the promise of these models to move hypothesis and theory testing in our field forward, no formal models of eating disorders exist. Thus, a feasible next step would be applying current anxiety computational models to the study anxiety-eating disorder comorbidity.

Finally, a logical next step for future research would be the collection of fMRI data at baseline to predict later behavioral outcomes and understand neurobiological

vulnerabilities, as was originally proposed in this dissertation; please see below for more information about potential future fMRI studies investigating this integrated learning theory.

### ***A Note About Neurobiological Vulnerabilities***

As noted in the Acknowledgements page at the beginning of the study, the original proposal of this dissertation included a neuroimaging component (see Appendix A) that could not be completed due to the COVID-19 pandemic. However, the current study can be used as preliminary findings to guide further research involving fMRI. First and foremost, neurobiological vulnerabilities including reward and fear learning could not be directly assessed in the current study. Elucidating these vulnerabilities is crucial for understanding how neurobiology may inform the development of cognitive-behavioral learning mechanisms, such as trait negative affect and thinness expectancies. For instance, someone with overactive fear learning (i.e., the ability to acquire fears and activate previous fear learning more readily on average) may over time come to have high trait negative affect due to being in a fearful state more often. It is also possible that someone who has overactive reward learning (i.e., having an accentuated response to rewarding stimuli on average) may experience being thin as extremely rewarding, which then with other factors, can contribute to learned expectancies that thinness will lead to success and other positive outcomes (i.e., thinness expectancies).

The current study can help guide future research by elucidating the cognitive-behavioral mechanisms that may directly relate to neurobiological vulnerabilities, i.e., negative affect and thinness expectancies. Originally, a fear learning task and implicit food association task were proposed for task-based fMRI to assess fear learning, reward

learning, and the tendency to form habits around food. Based on these preliminary findings, particularly that habits around eating may not be as important in the *development* of comorbid eating disorder-anxiety symptoms, a different reward task may be more applicable. For instance, the drifting double bandit task is a probabilistic reward learning task that has been extensively used, including in the eating disorder field (Schaefer & Steinglass, 2021).

It may also be helpful to assess fear and reward learning generally, as well as fear and reward learning specifically around eating, weight, and shape. For fear learning, the proposed acquisition and extinction task was general, but it may be possible to include a counterbalanced block that has food stimuli. It is also possible to conduct reward studies where participants are given a sucrose solution as the rewarding outcome. For instance, in one study, participants who were recovered from AN and matched controls were given tastes of sucrose and water while undergoing fMRI (Wagner et al., 2008). Women recovered from AN showed blunted activation in the ventral striatum, dorsal striatum, and insula compared to healthy controls when presented with sucrose, indicating that sucrose may be less rewarding to those who have had AN. Thus, there are multiple methods that can assess both general and specific fear and reward learning. Future research should seek to incorporate both of these methods and select tasks with specific hypotheses in mind as to how general versus specific learning around eating, shape, and weight may contribute to eating disorder comorbidity.

Finally, it would be useful for future studies to incorporate multiple levels of analysis with neuroimaging beyond self-report, such as behavioral tasks outside of the scanner. For instance, the FLARE app assesses fear learning and can be accessed on a

smart phone (Purves et al., 2019). Pairing task-based fMRI data with other real-time behavioral data over time can provide more information regarding how these mechanisms are working in concert with one another. In this instance, it would be possible to track fear learning at the different time points (not just at baseline when the fMRI component takes place), allowing for change over time, as well as direct comparison with cognitive-behavioral mechanisms. In sum, task-based fMRI examining fear and reward learning is a crucial step in understanding comorbid eating disorder-anxiety symptoms, and the current study provides guidance on task-selection and hypothesis generation for future studies.

### ***Potential Clinical Implications***

If such future research as described above does indeed support an integrated learning model of comorbid eating disorder-anxiety symptoms, then several clinical implications could be made. Importantly, the current study does not yet provide enough evidence to warrant definitive conclusions regarding clinical implications. First, exposure therapy is the gold-standard treatment for anxiety disorders (Holmes et al., 2014; Pittig et al., 2018) and has shown immense promise in application to eating disorders (e.g., Reilly et al., 2017). By better understanding the mechanisms underlying this comorbidity, exposure therapy can be tailored to more precisely target these mechanisms. For instance, if negative affect is supported as the shared mechanism between eating and anxiety disorders, exposures can emphasize tolerating the distress that arises when approaching feared stimuli. Such exposures can integrate reward, as well as fear, learning principles. For example, individuals may select a reward to consistently pair with the exposure, such as engaging in a pleasurable activity or engaging in meaningful interpersonal interactions. Thus, exposures would then be tailored to better target tolerating negative affect while

simultaneously *increasing* positive affect. Future research could also elucidate the specific type of negative affect needed to be targeted, such as shame, which may be more socially-driven than other types of negative affect (Levinson, Byrne, & Rodebaugh, 2016; Wong et al., 2021a), and thus require social exposures. In addition, repetitive negative thinking could be targeted when intervening on restriction behaviors specifically by challenging thoughts, such as in CBT-E (Fairburn, 2008).

Finally, if future research shows that risk factors for this comorbidity can be found in preadolescence, then existing prevention efforts, such as The Body Project, which is currently only validated in high school and college-aged populations (Stice et al., 2009b; 2011b), could be applied earlier in middle school, or late elementary school. Also, if we better understand the shared vulnerabilities between eating and anxiety disorders, we may be better able to predict which individuals are at high risk for the development of these disorders and intervene earlier in this high risk population. Thus, while there are several important clinical implications stemming from this model, significant future research needs to be conducted before implementing any such clinical interventions.

## **Conclusions**

In conclusion, cross-sectionally, negative affect was the shared correlate between eating disorder and anxiety symptoms, and thinness expectancies emerged as the unique correlate related to only eating disorder symptoms. In addition, negative affect and thinness expectancies moderated eating disorder symptoms, showing a compound effect of these factors. Prospectively, no shared vulnerability factors were found, but post-hoc tests indicated that repetitive negative thinking is a shared vulnerability factor for anxiety

and restriction symptoms. Interestingly, there were no factors that predicted global eating disorder symptoms at follow-up, above and beyond baseline eating disorder symptoms.

Taken together, these findings suggest that the strongest predictor of later eating disorder symptoms in adolescence is earlier eating disorder symptoms. In addition, different vulnerability factors may predict different eating disorder behaviors. Thus, there is an urgent need to study eating disorder symptoms and comorbidity at an even earlier age, perhaps during preadolescence, before puberty. This study was a preliminary test of an integrated learning model and hopes to guide future research regarding shared and unique vulnerability factors for eating disorder comorbidity.

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**Table 2***Descriptive Statistics*

Variable	Mean (Standard Deviation)	Range
T1 Eating Disorder Symptoms	1.10 (1.34)	0-5.74
T2 Eating Disorder Symptoms	0.97 (1.08)	0-4.69
T3 Eating Disorder Symptoms	1.21 (1.28)	0-4.91
T1 Anxiety	17.98 (9.02)	2.00 - 45.00
T2 Anxiety	16.89 (8.63)	3.00 - 35.00
T3 Anxiety	18.05 (8.17)	4.00 - 37.00
T1 Repetitive negative thinking	25.93 (12.36)	10.00 - 50.00
T2 Repetitive negative thinking	24.30 (11.69)	10.00 - 49.00
T3 Repetitive negative thinking	26.56 (12.49)	10.00 - 50.00
T1 Negative affect	22.02 (8.35)	10.00 - 48.00
T2 Negative affect	22.70 (7.73)	10.00 - 42.00
T3 Negative affect	23.73 (8.94)	10.00 - 46.00
T1 Thinness expectancies	2.98 (2.02)	1.00 - 7.00

T2 Thinness expectancies	2.79 (1.61)	1.00 – 6.75
T3 Thinness expectancies	3.13 (1.74)	1.00 – 6.63
T1 Habits around eating	53.47 (9.15)	16.00 - 60.00
T2 Habits around eating	53.54 (9.18)	12.00 – 60.00
T3 Habits around eating	55.90 (7.70)	28.00 – 60.00

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*Note.* T1 = Time 1 ( $n = 70$ ); T2 = Time 2 (four-month follow-up;  $n = 70$ ); T3 = Time 3 (nine-month follow-up;  $n = 42$ )

**Table 3a.***Zero-order Correlations*

	T1ED	T2ED	T3ED	T1Anx	T2Anx	T3Anx	T1RNT	T2RNT	T3RNT
T1ED	-								
T2ED	<b>.83**</b>	-							
T3ED	<b>.62**</b>	<b>.67**</b>	-						
T1Anx	<b>.56**</b>	<b>.43**</b>	<b>.33**</b>	-					
T2Anx	<b>.52**</b>	<b>.53**</b>	.30	<b>.67**</b>	-				
T3Anx	.19	.21	<b>.35*</b>	<b>.49**</b>	<b>.49**</b>	-			
T1RNT	<b>.67**</b>	<b>.56**</b>	<b>.47**</b>	<b>.66**</b>	<b>.66**</b>	<b>.38**</b>	-		
T2RNT	<b>.50**</b>	<b>.53**</b>	.23	<b>.32**</b>	<b>.60**</b>	.28	<b>.54**</b>	-	
T3RNT	<b>.41**</b>	<b>.38*</b>	<b>.37**</b>	.29	<b>.42**</b>	<b>.56**</b>	<b>.58**</b>	<b>.58**</b>	-
T1NA	<b>.66**</b>	<b>.55**</b>	<b>.49**</b>	<b>.67**</b>	<b>.55**</b>	<b>.47**</b>	<b>.76**</b>	<b>.44**</b>	<b>.49**</b>
T2NA	<b>.50**</b>	<b>.57**</b>	<b>.52**</b>	<b>.44**</b>	<b>.57**</b>	<b>.56**</b>	<b>.59**</b>	<b>.76**</b>	<b>.65**</b>
T3NA	<b>.56**</b>	<b>.58**</b>	<b>.55**</b>	.30	<b>.48**</b>	<b>.56**</b>	<b>.51**</b>	<b>.42**</b>	<b>.50**</b>
T1TE	<b>.79**</b>	<b>.73**</b>	<b>.52**</b>	<b>.48**</b>	<b>.51**</b>	.14	<b>.67**</b>	<b>.40**</b>	.30
T2TE	<b>.61**</b>	<b>.74**</b>	<b>.51**</b>	<b>.25*</b>	<b>.42**</b>	.14	<b>.41**</b>	<b>.47**</b>	.21
T3TE	<b>.58**</b>	<b>.62**</b>	<b>.76**</b>	<b>.34*</b>	<b>.33*</b>	<b>.31*</b>	<b>.39*</b>	.19	.08
T1Habit	<b>-.25*</b>	<b>-.24*</b>	-.26	-.16	-.09	-.04	<b>-.26*</b>	-.11	-.23
T2Habit	-.21	<b>-.36**</b>	-.06	-.16	-.21	.25	.03	-.23	-.05
T3Habit	-.09	-.06	<b>-.51**</b>	-.18	.02	-.25	-.06	.20	<b>-.55**</b>

*Note.* T1 = Time 1; T2 = Time 2 (four-month follow-up); T3 = Time 3 (nine-month follow-up); ED = eating disorder symptoms; Anx = anxiety disorder symptoms; RNT = repetitive negative affect; NA = negative affect; TE = thinness expectancies; Habit = habits around eating; \* =  $p < .05$ , \*\* =  $p < .01$ . Significant correlations are bolded for clarity.



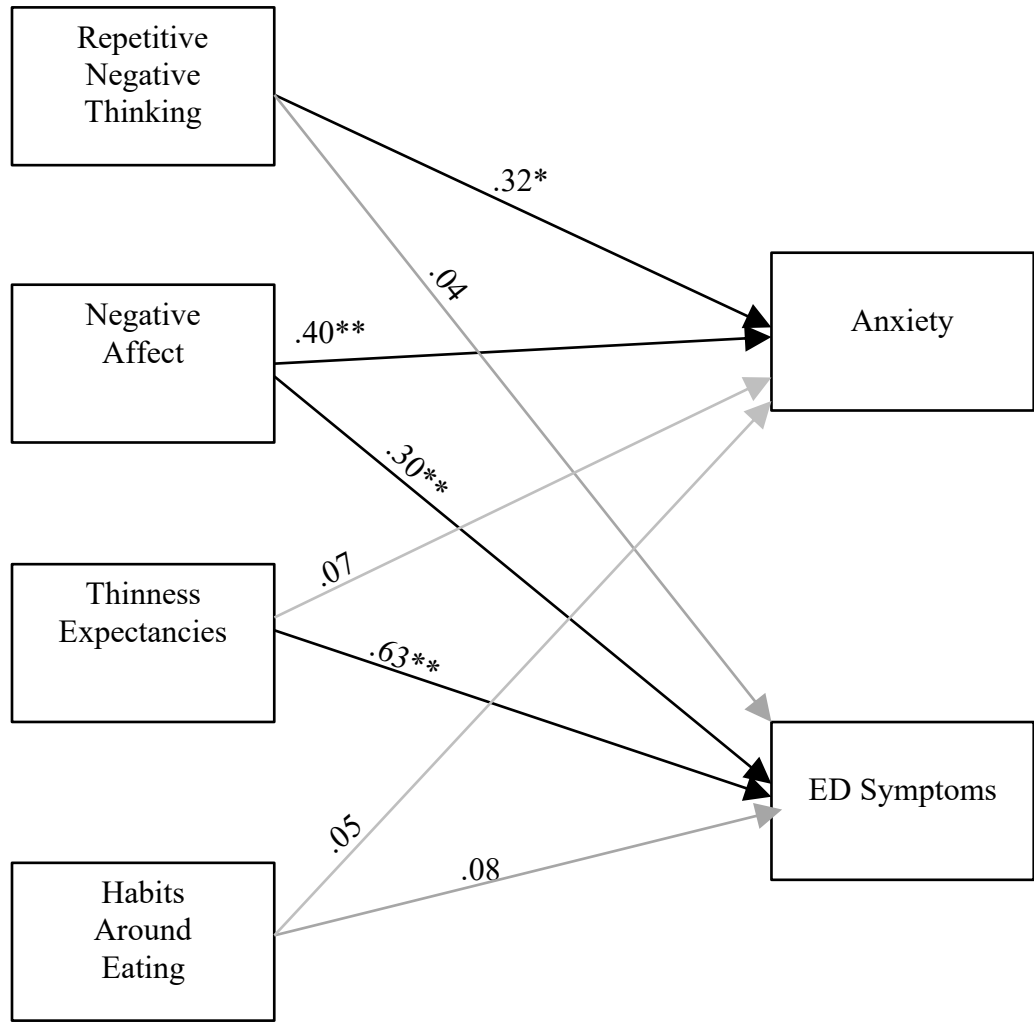
**Table 3b.***Zero-order Correlations*

	T1NA	T2NA	T3NA	T1TE	T2TE	T3TE	T1Hab	T2Hab	T3Hab
T1NA	-								
T2NA	<b>.54**</b>	-							
T3NA	<b>.61**</b>	<b>.71**</b>	-						
T1TE	<b>.55**</b>	<b>.42**</b>	<b>.41**</b>	-					
T2TE	<b>.29*</b>	<b>.53**</b>	<b>.31*</b>	<b>.60**</b>	-				
T3TE	<b>.43**</b>	<b>.49**</b>	<b>.50**</b>	<b>.61**</b>	<b>.68**</b>	-			
T1Hab	-.20	-.20	-.22	<b>.41**</b>	<b>-.25*</b>	<b>.42**</b>	-		
T2Hab	-.01	-.23	-.10	<b>-.29*</b>	<b>-.31*</b>	-.16	.09	-	
T3Hab	-.14	.04	-.10	-.24	-.14	<b>.55**</b>	.09	.02	-

*Note.* T1 = Time 1; T2 = Time 2 (four-month follow-up); T3 = Time 3 (nine-month follow-up); ED = eating disorder symptoms; Anx = anxiety disorder symptoms; RNT = repetitive negative affect; NA = negative affect; TE = thinness expectancies; Hab = habits around eating; \* =  $p < .05$ , \*\* =  $p < .01$ . Significant correlations are bolded for clarity.

**Figure 5**

*Cross-sectional Shared and Unique Risk Model for Comorbid Eating Disorder-Anxiety Symptoms*



*Note.* Significant paths are bolded in black for clarity. Non-significant paths are depicted in gray. \*  $p < .05$ . \*\*  $p < .01$ .

**Table 4**

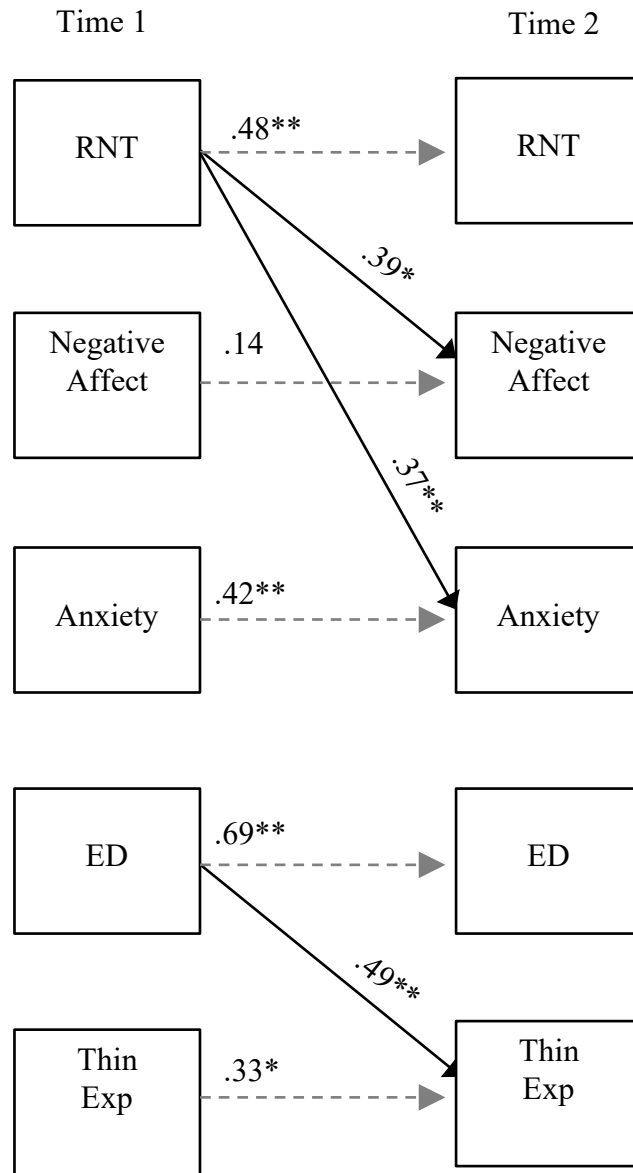
*Prospective Shared and Unique Risk Model Parameter Estimates for Comorbid Eating Disorder-Anxiety Symptoms*

Variable	Parameter Estimate	<i>p</i> -value
T2 ED		
T1 RNT	.01	.967
T1 NA	.06	.503
T1 Anxiety	-.08	.411
T1 ED	.68	< .001
T1 Thin Exp	.21	.243
T2 Anxiety		
T1 RNT	.37	.002
T1 NA	-.07	.629
T1 Anxiety	.42	< .001
T1 ED	.02	.916
T1 Thin Exp	.10	.543
T2 RNT		
T1 RNT	.48	.003
T1 NA	.02	.915
T1 Anxiety	-.14	.387
T1 ED	.37	.072
T1 Thin Exp	-.14	.407
T2 NA		
T1 RNT	.39	.011
T1 NA	.14	.432
T1 Anxiety	.02	.897
T1 ED	.23	.221
T1 Thin Exp	-.08	.657
T2 Thin Exp		
T1 RNT	.10	.502
T1 NA	-.19	.091
T1 Anxiety	-.09	.450
T1 ED	.49	.001
T1 Thin Exp	.33	.026

*Note.* RNT = repetitive negative thinking; NA = negative affect; Thin Exp = thinness expectancies; T1 = Time 1; T2 = Time 2.

**Figure 6**

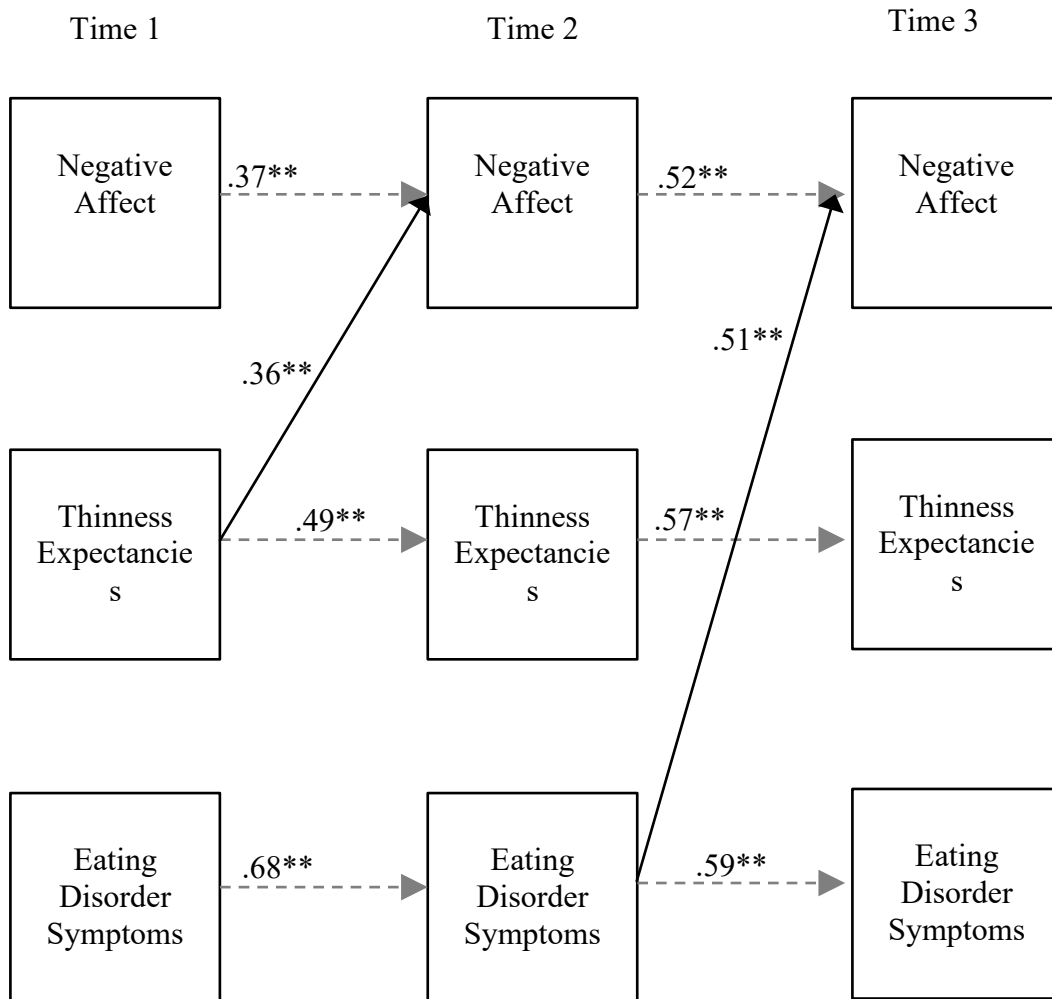
*Prospective Shared and Unique Risk Model for Comorbid Eating Disorder-Anxiety Symptoms*



*Note.* Significant paths are bolded in black. Autogressive paths are dashed and faded in gray. Non-significant paths are not shown for clarity. \* =  $p < .05$ , \*\* =  $p < .01$ . ED = eating disorder symptoms; RNT = repetitive negative thinking; NA = negative affect; Thin Exp = thinness expectancies; T1 = Time 1; T2 = Time 2.

**Figure 7**

*Replicating a Shared and Unique Vulnerability Model for Eating Disorders Symptoms*



*Note.* Significant paths are bolded in black. Autoregressive paths are dashed and faded in gray. Non-significant paths are not shown for clarity; \* =  $p < .05$ , \*\* =  $p < .01$ .

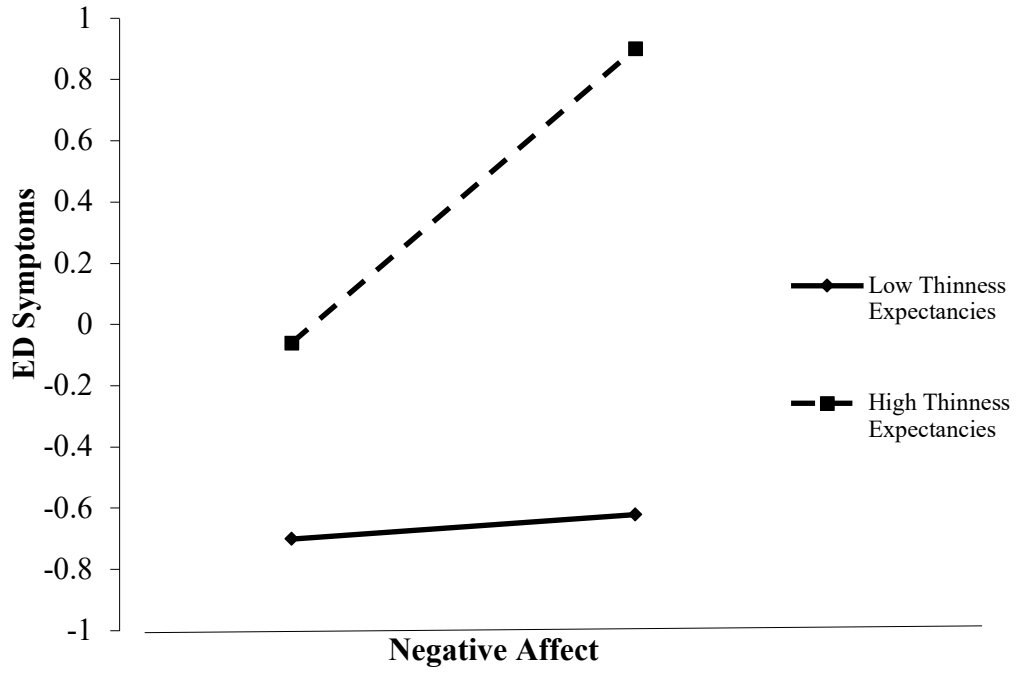
**Table 5.***Parameter Estimates for Shared and Unique Vulnerability Model of Eating Disorders Symptoms*

Variable	Parameter Estimate	<i>p</i> -value
T3 Eating Disorder		
T2 Negative Affect	.18	.117
T2 Eating Disorder	.59	.001
T2 Thinness Expectancies	.03	.886
T3 Negative Affect		
T2 Negative Affect	.52	< .001
T2 Eating Disorder	.51	.004
T2 Thinness Expectancies	-.23	.114
T3 Thinness Expectancies		
T2 Negative Affect	.20	.123
T2 Eating Disorder	.04	.821
T2 Thinness Expectancies	.57	< .001
T2 Eating Disorder		
T1 Negative Affect	.01	.911
T1 Eating Disorder	.68	< .001
T1 Thinness Expectancies	.20	.243
T2 Negative Affect		
T1 Negative Affect	.37	.009
T1 Eating Disorder	.25	.199
T1 Thinness Expectancies	.05	.749
T2 Thinness Expectancies		
T1 Negative Affect	-.20	.064
T1 Eating Disorder	.49	.001
T1 Thinness Expectancies	.36	.012

*Note.* T1 = Time 1; T2 = Time 2.

**Figure 8**

*Interaction Between Negative affect and Thinness Expectancies on Eating Disorder Symptoms*



*Note.* All scores are standardized. ED = eating disorder.

**Table 6**

*Parameter Estimates for a Post-hoc Shared Vulnerability Model with Social Appearance*

*Anxiety*

Variable	Parameter Estimate	<i>p</i> -value
T2 ED		
T1 Anxiety	-.05	.541
T1 ED	.86	< .001
T1 SAA	.01	.956
T2 Anxiety		
T1 Anxiety	.45	< .001
T1 ED	.09	.471
T1 SAA	.26	.036
T2 SAA		
T1 Anxiety	-.15	.151
T1 ED	.20	.136
T1 SAA	.71	< .001

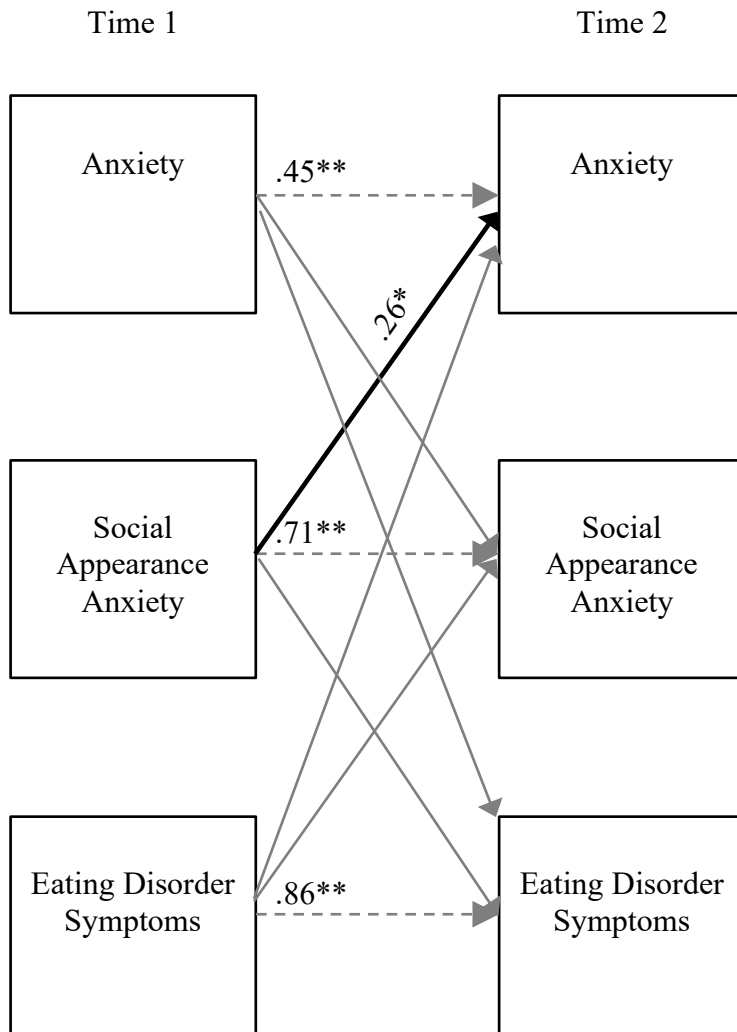
*Note.* ED = eating disorder symptoms; SAA = social appearance anxiety; T1 = Time 1; T2 = Time 2.



**Figure 9**

*Parameter Estimates for a Post-hoc Shared Vulnerability Model with Social Appearance*

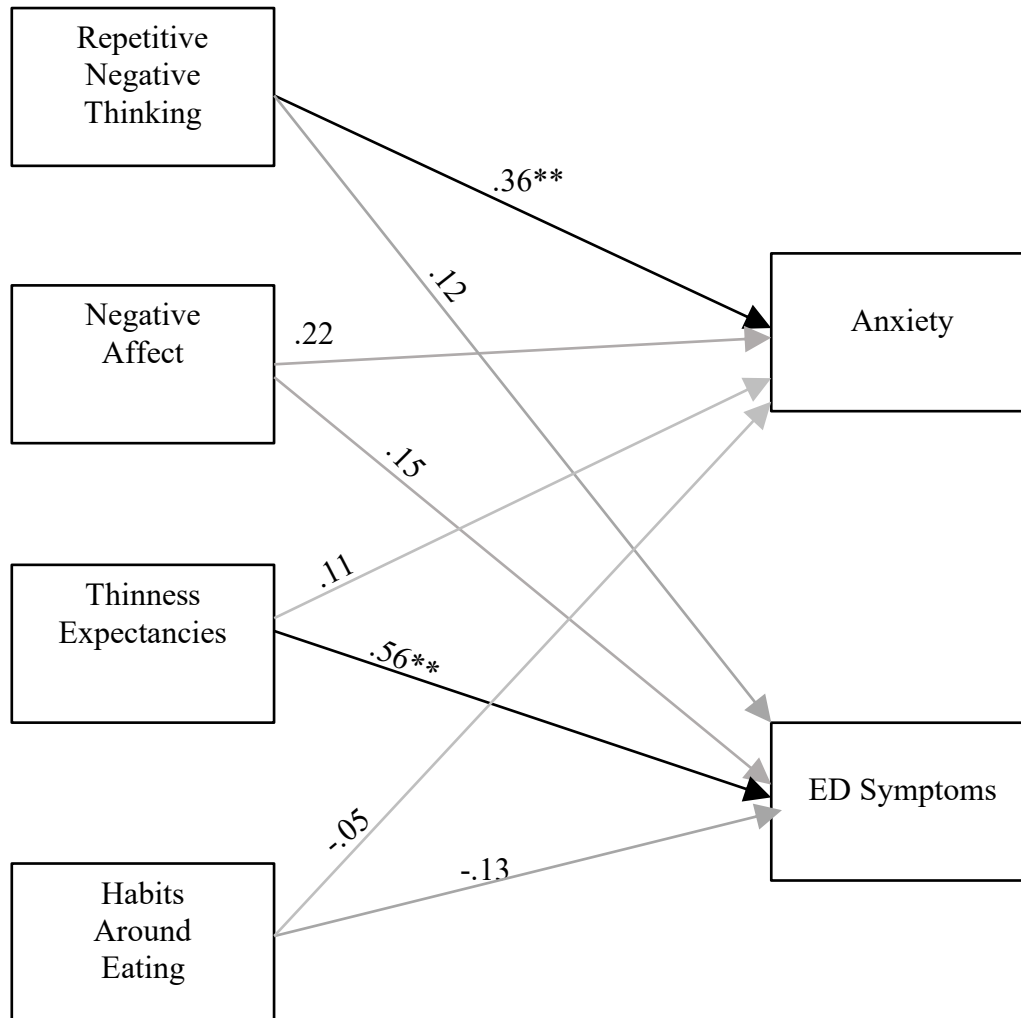
*Anxiety*



*Note.* Significant paths are bolded in black. Autoregressive paths are dashed and faded in gray. Non-significant paths are in gray;  $* = p < .05$ ,  $** = p < .01$ . ED = eating disorder symptoms; SAA = social appearance anxiety; T1 = Time 1; T2 = Time 2.

**Figure 10**

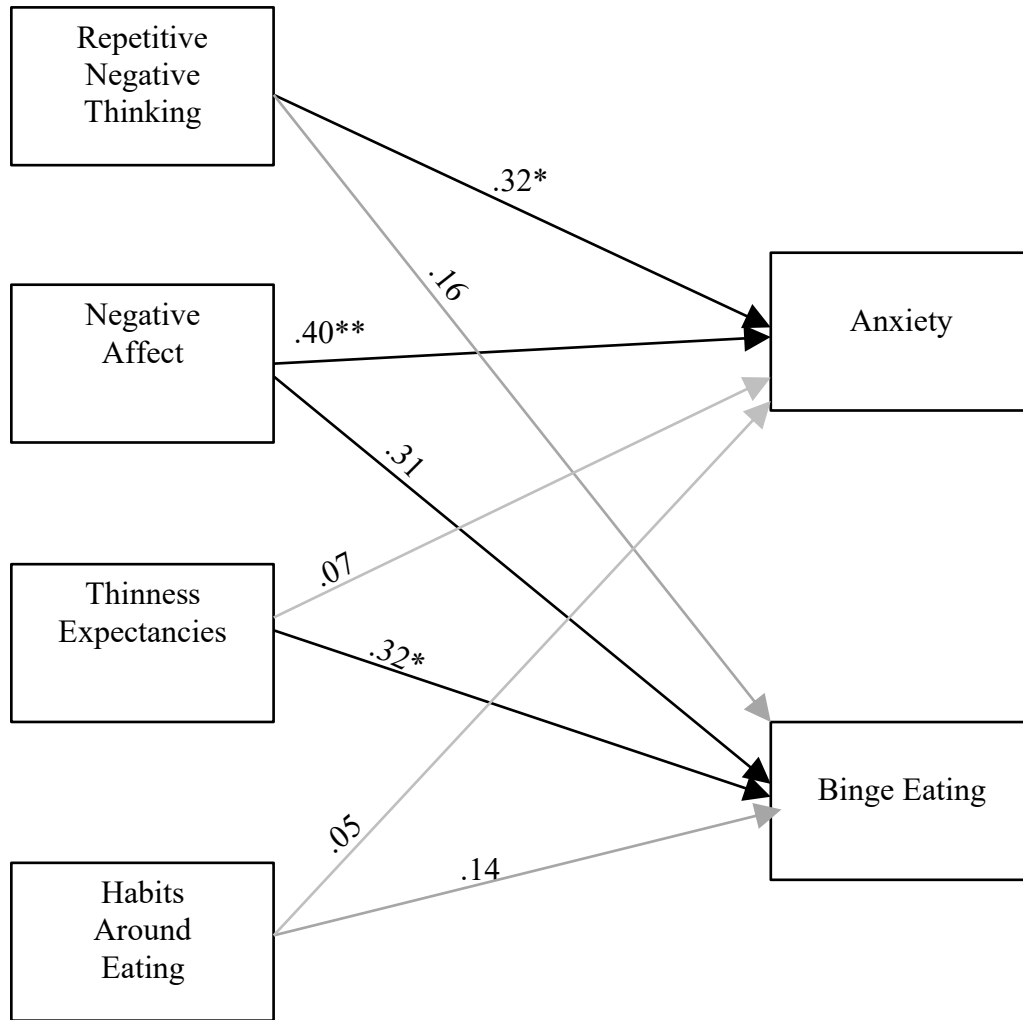
*Post-hoc Replication Cross-sectional Model at Time 2*



*Note.* Significant paths are bolded in black for clarity. Non-significant paths are depicted in gray. \*  $p < .05$ . \*\* $p < .01$ .

**Figure 11**

*Post-hoc Cross-sectional Binge Eating Model*



*Note.* Significant paths are bolded in black for clarity. Non-significant paths are depicted in gray. \*  $p < .05$ . \*\*  $p < .01$ .

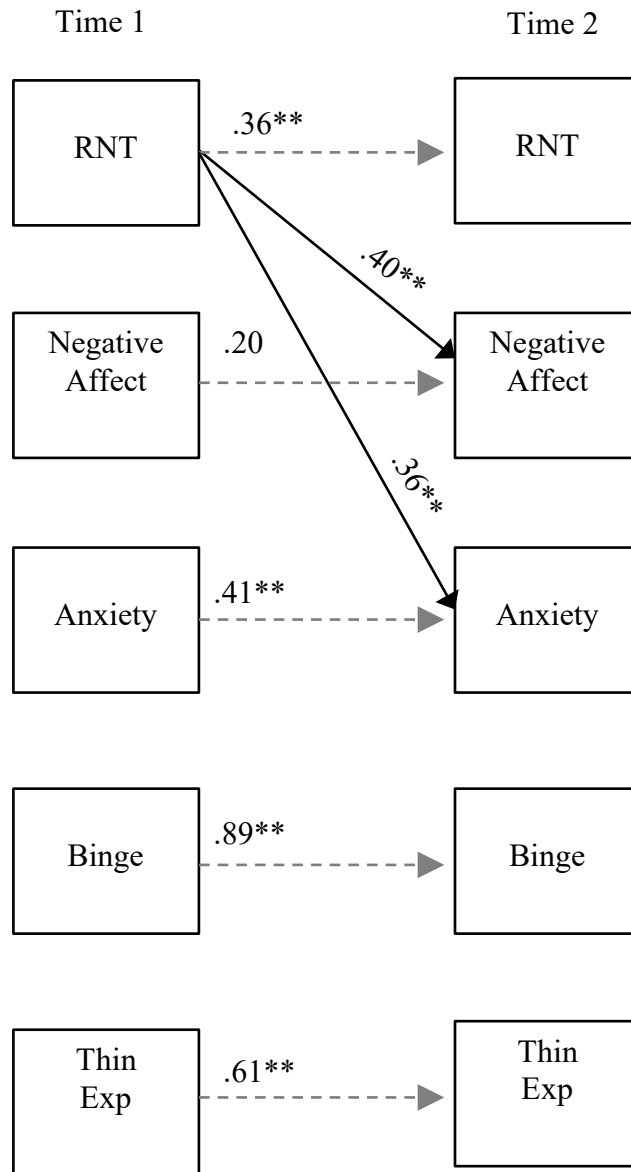
**Table 7***Post-hoc Prospective Binge Eating Model Parameter Estimates*

Variable	Parameter Estimate	<i>p</i> -value
T2 Binge		
T1 RNT	-.14	.240
T1 NA	-.02	.887
T1 Anxiety	-.04	.472
<b>T1 Binge</b>	<b>.89</b>	<b>&lt; .001</b>
T1 Thin Exp	.08	.741
T2 Anxiety		
<b>T1 RNT</b>	<b>.36</b>	<b>.003</b>
T1 NA	-.09	.491
<b>T1 Anxiety</b>	<b>.41</b>	<b>.001</b>
T1 Binge	.09	.399
T1 Thin Exp	.08	.473
T2 RNT		
<b>T1 RNT</b>	<b>.47</b>	<b>.004</b>
T1 NA	.09	.635
T1 Anxiety	-.13	.460
T1 Binge	.11	.382
T1 Thin Exp	.04	.751
T2 NA		
<b>T1 RNT</b>	<b>.40</b>	<b>.009</b>
T1 NA	.20	.303
T1 Anxiety	.04	.831
T1 Binge	-.01	.934
T1 Thin Exp	.04	.777
T2 Thin Exp		
T1 RNT	.10	.523
T1 NA	-.07	.590
T1 Anxiety	-.06	.689
T1 Binge	-.03	.835
<b>T1 Thin Ex</b>	<b>.61</b>	<b>&lt; .001</b>

*Note.* Binge = binge eating symptoms; RNT = repetitive negative thinking; NA = negative affect; Thin Exp = thinness expectancies; T1 = Time 1; T2 = Time 2.

**Figure 12**

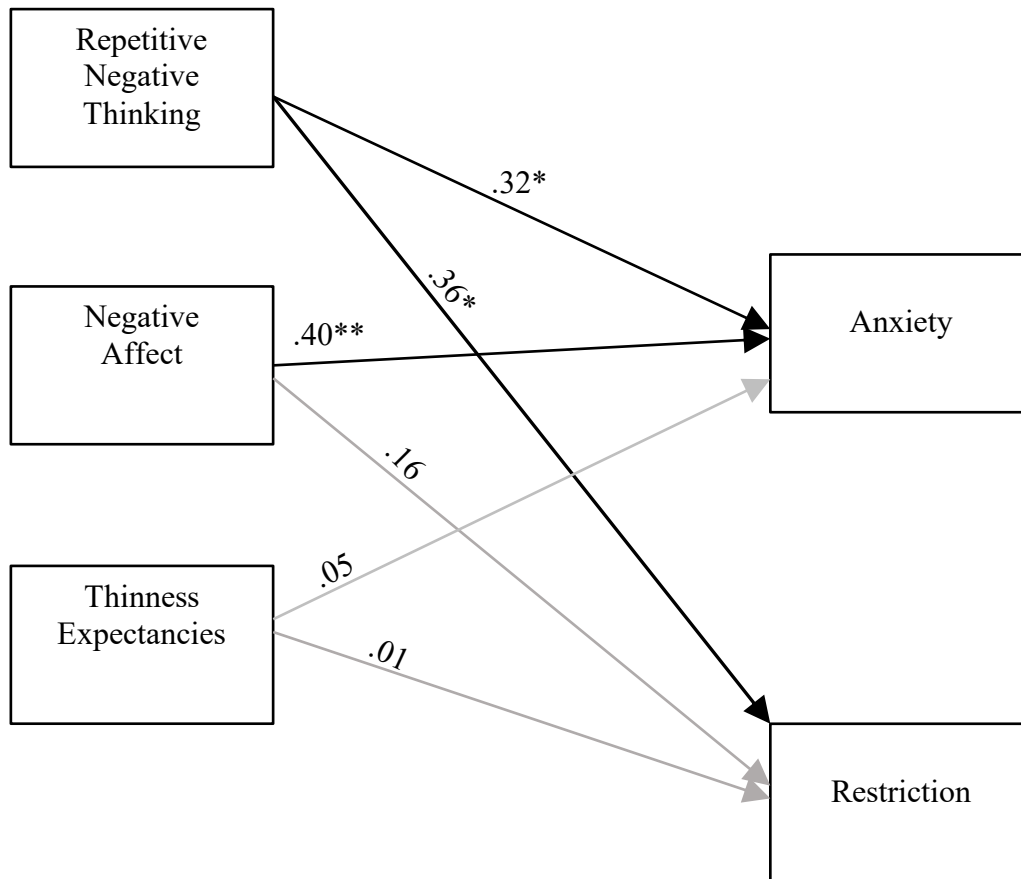
*Post-hoc Prospective Binge Eating Model*



*Note.* Significant paths are bolded in black. Autogressive paths are dashed and faded in gray. Non-significant paths are not shown for clarity. \* =  $p < .05$ , \*\* =  $p < .01$ . Binge = binge eating symptoms; RNT = repetitive negative thinking; NA = negative affect; Thin Exp = thinness expectancies; T1 = Time 1; T2 = Time 2.

**Figure 13**

*Post-hoc Cross-sectional Restriction Model*



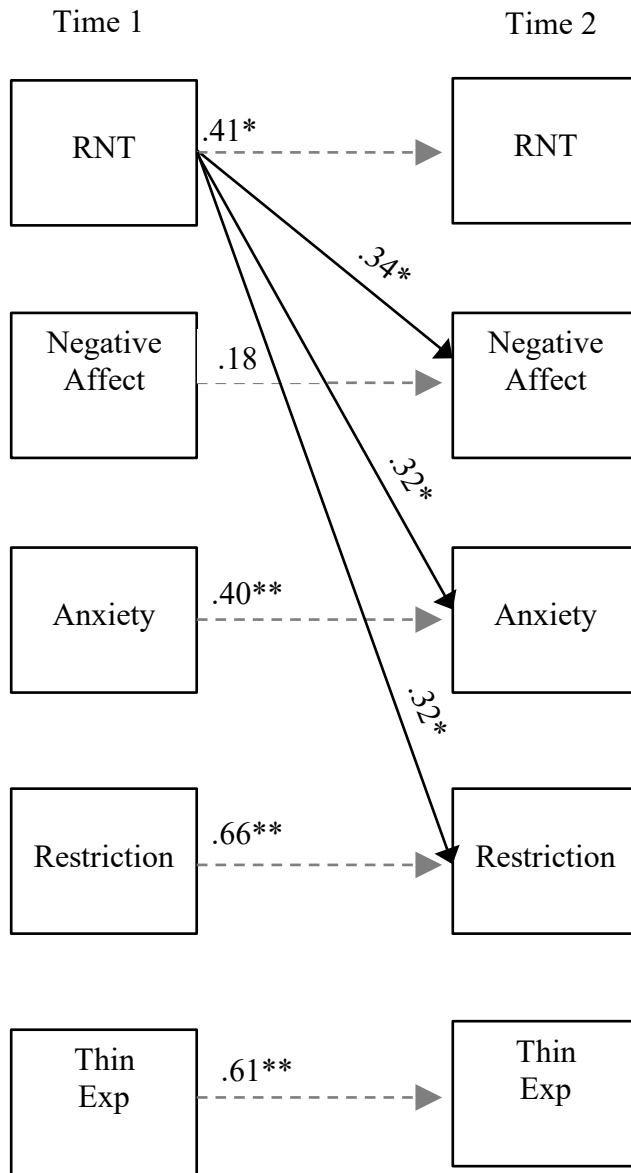
*Note.* Significant paths are bolded in black for clarity. Non-significant paths are depicted in gray. \*  $p < .05$ . \*\*  $p < .01$ .

**Table 8***Post-hoc Prospective Restriction Model Parameter Estimates*

Variable	Parameter Estimate	<i>p</i> -value
T2 Restrict		
<b>T1 RNT</b>	<b>.32</b>	<b>.023</b>
T1 NA	.05	.685
T1 Anxiety	-.36	.002
<b>T1 Restrict</b>	<b>.66</b>	<b>&lt; .001</b>
T1 Thin Exp	.10	.352
T2 Anxiety		
<b>T1 RNT</b>	<b>.32</b>	<b>.012</b>
T1 NA	-.08	.528
<b>T1 Anxiety</b>	<b>.40</b>	<b>.001</b>
T1 Restrict	.14	.117
T1 Thin Exp	.10	.345
T2 RNT		
<b>T1 RNT</b>	<b>.41</b>	<b>.015</b>
T1 NA	.09	.630
T1 Anxiety	-.15	.381
<b>T1 Restrict</b>	<b>.26</b>	<b>.040</b>
T1 Thin Exp	.07	.566
T2 NA		
<b>T1 RNT</b>	<b>.34</b>	<b>.046</b>
T1 NA	.18	.313
T1 Anxiety	.01	.964
T1 Restrict	.17	.229
T1 Thin Exp	.03	.770
T2 Thin Exp		
T1 RNT	.08	.624
T1 NA	-.08	.500
T1 Anxiety	-.07	.622
T1 Restrict	.06	.571
<b>T1 Thin Ex</b>	<b>.61</b>	<b>&lt;.001</b>

**Figure 14**

*Post-hoc Prospective Restriction Model*



*Note.* Significant paths are bolded in black. Autogressive paths are dashed and faded in gray. Non-significant paths are not shown for clarity.  $*$  =  $p < .05$ ,  $**$  =  $p < .01$ . Restrict = restriction symptoms; RNT = repetitive negative thinking; NA = negative affect; Thin Exp = thinness expectancies; T1 = Time 1; T2 = Time 2.



## **Appendix A. Neurobiological Vulnerability Exploration Research Questions, Hypotheses, Tasks, and Data Analytic Plan**

**Exploratory research question and hypothesis 1:** The first exploratory research question is as follows: Does fear acquisition occur more readily and fear extinction occur more slowly in comorbid ED-anxiety, compared to controls? Is there less activation and functional connectivity in brain regions associated with fear extinction in comorbid ED-anxiety compared to controls?

In addition, do individuals with comorbid ED-anxiety attend to highly palatable foods more quickly than controls, potentially indicating that these individuals are more likely to form habits around eating? Is there greater activation and functional connectivity in brain regions associated with fear expression, habit learning and reward in comorbid ED-anxiety compared to controls?

I hypothesize that the high eating disorder-high anxiety group will behaviorally rate greater fear acquisition and less fear extinction than the low eating disorder-low anxiety group. In addition, I hypothesize that there will be less activation and functional connectivity in fear extinction brain regions (i.e., dorsal lateral prefrontal cortex [DLPFC], ventral lateral prefrontal cortex [VLPFC], subgenual anterior cingulate cortex [sgACC], ventral medial prefrontal cortex [vmPFC]), and increased activation and functional connectivity in fear acquisition/expression brain regions (i.e., amygdala, bed nucleus of the stria-terminalis [BNST], and insula) observed in the high eating disorder-high anxiety group compared to the low eating disorder-low anxiety group.

Further, I hypothesize that individuals with behaviorally react more quickly when attending to highly palatable foods in the high eating disorder-high anxiety group, in line with the ability to be primed to form habits around eating more easily (and finding these foods more rewarding) than those in the low eating disorder- low anxiety group. I also hypothesize increased activation and functional connectivity in regions implicated in reward and implicit/habit learning processes (i.e., orbitofrontal cortex, dorsal and ventral striatum, and insula) will be observed in the high eating disorder-anxiety low group compared to the low eating disorder-low anxiety group.

**1.1:** Functional activation during the Fear Acquisition and Extinction Task (FAET) will be decreased in brain regions associated with fear extinction (i.e., DLPFC, VLPFC, sgACC) and increased in brain regions associated with fear acquisition/expression (i.e., amygdala, BNST, insula) in the high eating disorder-high anxiety group compared to the low eating-low anxiety group.

**1.2:** During completion of the FAET task, individuals in the high eating disorder-high anxiety group will exhibit reductions of functional connectivity in the expression of fear extinction neural networks (i.e., DLPFC, VLPFC, sgACC), as well as reductions in connectivity between those fear extinction networks and fear expression networks, as compared to low eating disorder-low anxiety individuals.

**1.3:** During completion of the FAET task, individuals in the high eating disorder-high anxiety group will behaviorally rate acquisition higher (indicating greater fear acquisition), as well as rate extinction lower (indicating lesser fear extinction), compared to the low eating disorder-low anxiety group.

**1.4:** Functional activation during the Food Attention Visual Probe Task will be increased in implicit/habit learning and reward regions (i.e., orbitofrontal cortex [OFC], insula, dorsal striatum and ventral striatum) in the high eating disorder-high anxiety group compared to the low eating disorder-low anxiety group in relation to food stimuli.

**1.5:** Functional connectivity during the Food Attention Visual Probe Task will be increased in habit learning and reward regions (i.e., OFC, insula, dorsal striatum, and ventral striatum) in the high eating disorder-high anxiety group compared to the low eating disorder-low anxiety group in relation to food stimuli.

**1.6:** During completion of the Food Attention Visual Probe Task, reaction time will be faster in the high palatable trials compared to the lower palatable trials (i.e., more quickly attend to highly palatable foods) in the high eating disorder-high anxiety group compared to the low eating disorder-low anxiety group.

**Exploratory Research Question and Hypothesis 2:** The second exploratory research question is: Do deficits in fear expression and extinction areas of the brain correspond with higher comorbid symptomatology at baseline, as well as greater development of anxiety and eating disorder symptoms over nine months? In addition, do deficits in reward and habit learning areas of the brain correspond with greater development of eating disorder, but not anxiety, symptoms both at baseline and over nine months?

I hypothesize that increased activation in the fear expression, habit learning, and reward brain regions (i.e., amygdala, BNST, insula, OFC, dorsal striatum, and ventral striatum), as well as decreased activation of fear extinction regions (i.e., DLPFC, VLPFC, sgACC, vmPFC), and decreased functional connectivity between these networks during

baseline will correspond to higher eating disorder-anxiety symptom development both cross-sectionally, as well as predict higher comorbid symptoms at nine-month follow-up, while adjusting for baseline levels of eating disorder and anxiety symptoms. More specific hypotheses for each task are as follows:

**2.1:** Increased functional activation of fear expression regions (i.e., amygdala, BNST, and insula) and decreased activation of fear extinction regions (i.e., DLPFC, VLPFC, sgACC, vmPFC) during the FAET will be associated with higher comorbid eating disorder-anxiety symptoms cross-sectionally, as well as predict higher comorbid symptoms at nine-month follow-up, adjusting for baseline levels of eating disorder-anxiety symptoms.

**2.2:** Decreased functional connectivity between the networks associated with fear expression and fear extinction regions, during the FAET, will be associated with higher comorbid eating disorder-anxiety symptoms cross-sectionally, as well as predict higher comorbid symptoms at nine-month follow-up, adjusting for baseline levels of eating disorder-anxiety symptoms.

**2.3:** Increased functional activation of implicit/habit learning and reward regions (i.e., OFC, insula, dorsal striatum, and ventral striatum) during the Food Attention Visual Probe Task will be associated with higher comorbid eating disorder-anxiety symptoms cross-sectionally, as well as predict higher comorbid symptoms at nine-month follow-up, adjusting for baseline levels of eating disorder-anxiety symptoms

**2.4:** Decreased functional connectivity between the networks associated with habit learning and reward, during the Food Attention Visual Probe Task, will be associated with higher comorbid eating disorder-anxiety symptoms cross-sectionally, as well as

predict higher comorbid symptoms at nine-month follow-up, adjusting for baseline levels of eating disorder-anxiety symptoms.

### **Neuroimaging Tasks**

*Data Acquisition. Structural* –A Siemens 3-Tesla SKYRA MR scanner will be used. T1-weighted Magnetization-Prepared Rapid Gradient-Echo sequence will be used [echo time (TE) = 2.26ms, repetition time (TR) = 1700ms, flip angle = 9.0°, field of view (FoV) = 204mm, 256 x 256 matrix, 208 sagittal slices, voxel size = 0.8mm isotropic].

*Functional* – Images will be collected using gradient echo T2\*-weighted echoplanar imaging [(EPI); TE = 30ms, TR = 2000 ms; flip angle = 72°, multi-band accelerated factor 3; FoV = 192 mm; 128 x 128 matrix, 72 transverse slices (oriented obliquely along the AC-PC line), voxel size = 1.5mm isotropic].

*Fear Acquisition and Extinction Task (FAET; Naaz, Knight, & Depue, 2019).* The FAET consists of two phases: 1) an acquisition phase, and 2) an extinction phase. First before either phase, participants rate cue faces using seven-point Likert scale to assess valence (1 = Extremely pleasant to 7 = Extremely unpleasant, with 4 = Neutral) to provide a behavioral measure of baseline. During the acquisition phase, a neutral face is paired with two conditioned stimuli (CS): a red border (CS+) on 50% of trials and a blue border (CS-) on 50% of trials (see Figure 15). Each trial contains a cue presented for 1000 milliseconds (ms) immediately followed by the presentation of either a fearful face with a human scream (unconditioned stimulus [US]) for CS+ trials (red border), or a neutral face with conversational chatter for CS- trials (blue border). Stimuli (face and

audio) are presented for 2000 ms, followed by an inter-trial interval (ITI) of 1000 ms (total trial duration is four seconds). All trials also include an additional pseudo-random variable ITI (jitter) (4000-14000 ms). Jitter allows for the separation of neural activity in response to stimuli versus feedback. During acquisition, four different stimuli for each

Figure 15. Fear Acquisition and Extinction



condition (CS+/CS-) are repeated six times (24 trials per condition total). Blocks will be broken into a mini-block design that lasted 16 seconds (four trials of either CS+ or CS-) in pseudo-

random order. After acquisition, participants again rate all cue faces with the seven-point Likert scale to provide a behavioral measure of acquisition. During the extinction phase, cues of previous neutral faces and borders are presented without fear expressions and human screams, and instead, all trails are paired with conversational chatter. All eight stimuli from the acquisition phase are repeated six times with identical duration as the acquisition phase. Finally, participants rate all cue faces using the same seven-point Likert scale to provide a behavioral measure of extinction.

***Food Attention Visual Probe Task (Shank et al., 2015).*** Please see Figure 16 for a picture of what participants see in the scanner. This visual probe task is used to measure impairment in implicit learning related to food. The task consists of 180 trials in which pairs of color photographs are presented. The task uses 90 photos from one of three categories: 30 high palatable foods (HP; e.g., pizza, donuts), 30 low palatable foods (LP;

e.g., pineapple, mushrooms), and 30 neutral images of household items (NF; e.g., paper shredder, paintbrush). Across the task, pictures are paired into groups of HP-LP; HP-NF; and LP-NF. For each trial, stimuli are presented side-by-side (e.g., HP-LP) and then a

Figure 16. Food Attention Visual Probe Task



Following each picture pair, you will see an arrow, either pointing to the left or to the right. If you see a right arrow, please press your right index finger (number 2). If you see a left arrow, please press your left index finger (number 7). This will repeat many times. At the end of the experiment, you will be asked to rate your anxiety from 1-10. This task will take around 15 minutes.



fixation cross appears for 2000ms. A left or

right arrow then appears. Participants are instructed to press their right index finger for right arrow stimuli and left index finger for left arrow stimuli.

Participants are asked to respond as quickly as possible and reaction time is assessed. This task has evidenced

good psychometric properties in eating disorder samples and is shown to relate to eating disorder outcomes, such as body mass index (Shank et al., 2015). The task will be used as a measure of *implicit/habit learning* specifically related to food and weight cues.

**Functional Activation Analyses for the FAET.** Standard group level contrasts for the two groups will be included with the regressors for FAET (CS+>CS- during both acquisition and extinction phases). We will conduct a whole brain and region of interest (ROI) analysis (ROIs – DLPFC, VLPFC, sgACC, amygdala, BNST and insula).

Comparisons of neuroimaging data between the groups will confirm that the two groups differ, and the changes observed in the a priori ROIs will indicate decreases of activation in frontal control regions (i.e., DLPFC, VLPFC, sgACC) and increases of activation in fear expression regions (amygdala, BNST and insula) in individuals with high eating disorder-high anxiety symptoms, as compared to individuals with low eating disorder-

low anxiety symptoms.

***Functional Connectivity Analyses for the FAET.*** Independent Components Analysis (ICA) decomposition of neural networks underlying the FAET will be compared and contrasted across the two groups (high eating disorder-high anxiety symptoms versus low eating disorder-low anxiety symptoms). Specifically, we will test whether high eating disorder-high anxiety individuals exhibit reductions of functional connectivity in the expression of fear extinction neural networks (i.e., DLPFC, VLPFC, and sgACC), as well as reductions in connectivity between those fear extinction networks and fear expression networks (i.e., amygdala, BNST, insula), as compared to low eating disorder-low anxiety individuals. We will also examine seed-based connectivity between all combinations of a priori ROIs, to establish differences between the two groups.

***Regressor Analyses for FAET.*** The functional activation of the a priori ROIs and connectivity of the networks in the FAET using individual differences in symptomatology as regressors (as outlined above in **Exploratory Hypothesis 1**) will be examined. In addition, within group contrasts, as well as between group contrasts, controlling for baseline levels of eating disorder-anxiety symptoms, will be conducted.

***Functional Activation Analyses for the FVP Task.*** The between group analysis of high eating disorder-high anxiety versus low eating disorder-low anxiety differences will be conducted using a whole brain and ROI approach (ROIs – OFC, insula, dorsal striatum, and ventral striatum) on the functional activation exhibited by both groups. Comparisons of neuroimaging data between the groups will confirm that the two groups differ, and the changes observed in the predicted ROIs will indicate decreases in OFC and increases in insula, dorsal striatum, and ventral striatum activation in high eating



disorder-high anxiety individuals, as compared to low eating disorder-low anxiety individuals.

***Functional Connectivity Analyses for the FVP Task.*** ICA decomposition of neural networks underlying implicit learning during the task will be compared and contrasted across the two groups (high eating disorder-high anxiety symptoms versus low eating disorder-low anxiety symptoms). Specifically, we will test whether high eating disorder-high anxiety individuals exhibit reductions of functional connectivity between higher-order reward and learning networks (i.e., OFC) and saliency/striatal networks, as compared to low eating disorder-low anxiety individuals. We will also examine seed-based connectivity between all combinations of ROIs, to establish differences between the two groups.

***Regressor Analyses for the FVP Task.*** The functional activation of the a priori ROIs and connectivity of the networks in the Food Attention Visual Probe Task using individual differences in symptomatology as regressors (as outlined above in **Exploratory Hypothesis 2**) will be examined. In addition, within group contrasts, as well as between group contrasts, controlling for baseline levels of eating disorder-anxiety symptoms, will be conducted.

## **Appendix B. Results from Alternative Imputation Strategy**

### **Primary Hypothesis 1: Cross-Sectional Shared and Unique Risk Model**

The cross-sectional model including repetitive negative thinking, negative affect, thinness expectancies, and habits around eating in association with eating disorder and anxiety symptoms was saturated (meaning that there were no degrees of freedom left in the model to estimate fit; Bentler, 1990). A non-saturated model (i.e., with non-significant paths removed) was conducted to ascertain fit: the model displayed excellent fit: CFI = 1.00, TLI = 1.05, RMSEA = 0.00. Similarly to Imputation Strategy 1, negative affect was positively associated with both eating disorder ( $b^* = .30, p = .002$ ) and anxiety symptoms ( $b^* = .40, p = .007$ ). However, repetitive negative thinking was only associated with anxiety symptoms ( $b^* = .32, p = .026$ ) cross-sectionally. Further, thinness expectancies ( $b^* = .63, p < .001$ ), but not habits around eating ( $p = .196$ ), were associated with eating disorder symptoms. Neither thinness expectancies nor habits around eating were related to anxiety symptoms ( $ps > .445$ ) cross-sectionally.

### **Primary Hypothesis 1: Prospective Shared and Unique Risk Model**

Similarly to Imputation Strategy 1, the prospective model including repetitive negative thinking, negative affect, thinness expectancies, and habits around eating, eating disorder and anxiety symptoms at all three time points was not able to be run due to low sample size and non-stable parameter estimates (Muthen & Muthen, 2014). Instead, the prospective model was run using the first two time points with the full sample size: this alternative model still displayed non-stable parameter estimates, which was also true of

### Imputation Strategy 1.

Thus, an exploratory model was run as an alternative that excluded habits around eating due to this variable being uncorrelated with all other variables. This new exploratory model included repetitive negative thinking, negative affect, thinness expectancies, eating disorder, and anxiety symptoms at baseline and at four month follow-up (Time 2); the model displayed perfect fit: CFI = 1.00, TLI = 1.00, RMSEA = 0.00, meaning that it was saturated. As with the cross-sectional model, non-significant paths were removed to establish fit. Fit was excellent: CFI = 1.00, TLI = 1.05, RMSEA = 0.00.

Once again, results were identical to Imputation Strategy 1: Repetitive negative thinking predicted anxiety symptoms ( $b^* = .37, p = .002$ ) prospectively at four months, while adjusting for baseline levels ( $b^* = .42, p = <.001$ ) of anxiety symptoms. Only baseline eating disorder symptoms ( $b^* = .68, p = <.001$ ) predicted later eating disorder symptoms at four months. As can be compared with the original results section, even the effect sizes are nearly identical, indicating that this strategy and the first yielded the same results.

### **Appendix C. A NOTE ABOUT COVID-19**

This dissertation was proposed in May 2020, at the beginning of the COVID-19 pandemic. At the time, it was impossible to have predicted that the pandemic would last almost two years and that the world would continue to be significantly impacted for a long length of time. As a result, a revised proposal was approved in November 2020, omitting a neuroimaging component from the dissertation, as in-person scanning was simply infeasible. Though I am thoroughly disappointed that my original proposal could not be completed, I am incredibly proud of the work that was able to be completed despite the pandemic.

This dissertation, from data collection through analysis and to write-up, was completed almost 100% during COVID-19. It is a testament to perseverance, embracing uncertainty, and flexibility. Though I think that the work in this dissertation stands without a ‘COVID clause,’ I also hope that readers will appreciate the work in the context in which it was conducted.

## CURRICULUM VITAE

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### EDUCATION

**University of Louisville**, Louisville, Kentucky Started: July 2016  
Clinical Psychology Doctoral Student  
Clinical Psychology, Masters **Received:** May 2019

**Washington University in St. Louis**, St. Louis, MO **Received:** May 2016  
Psychology, Bachelor of Arts  
Minor: Anthropology

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### GRANTS, AWARDS, AND HONORS

#### **National Institute of Health F31 Ruth L. Kirschstein Predoctoral Individual National Research Service Award (Scored, Not Funded)**

- Shared and Unique Neurobiological and Psychological Mechanisms of Comorbid Eating Disorder-Anxiety Symptoms During Adolescence
- Submitted 2018; Impact Score: 44; 40<sup>th</sup> percentile

#### **National Science Foundation Graduate Research Fellowship Program (GRFP) 2017 Honorable Mention**

- Does concern over mistakes impair cognitive control leading to food intake: an experimental study?

#### **American Psychological Association Dissertation Research Award**

2021

- Dissertation entitled Shared and Unique Mechanisms of Comorbid Eating Disorder-Anxiety Symptoms During Adolescence recognized for excellence in scientific psychology
- Awarded \$1000 to assist with research costs related to dissertation research

#### **Eating Disorders Research Society Best Student Abstract Award**

September 2019

- Prediction of binge eating, restricting, and purging symptoms using machine learning

#### **University of Louisville College of Arts and Sciences Undergraduate Mentored Research Award**

Awarded

Fall 2019 & 2020

- \$1500 internal grant to mentor an undergraduate in research activities
- Funds to be used to teach undergraduate longitudinal data collection and analytic methods

**University of Louisville College of Arts and Sciences Graduate Student Grant for Research and Creative Activities**

Awarded Spring 2019

- \$1000 internal grant for research activities in collaboration with Lindsay Knight, M.S.
- Funds to be used for neuro-imaging for in vivo imaginal exposure study

**University of North Carolina-Chapel Hill Center of Excellence for Eating Disorders (CEED) Summer Fellowship**

Summer 2017

- Summer fellowship was a full-time, 8-week research experience at the UNC-Chapel Hill CEED. Fellows are selected based on research experience and interests in eating disorders

**University of Louisville Clinical Psychology Excellence in Teaching Award**

2019-2020

- Awarded to the graduate student in the program that best exemplifies excellence in teaching. Feedback gathered from student and professor evaluations
- Awarded for Psych 610 & 611 (Graduate Psychology Statistics I and II)

**University of Louisville Graduate Fellowship**

2016-2018

- Fellowship awarded based on undergraduate GPA that provides protected research time during first two years of graduate education

**University of Louisville Clinical Psychology Junior Excellence**

2016-2017

**in Research Award**

- Awarded the clinical psychology program's junior excellence in research award, given to the student who has not yet completed their preliminary exam and has demonstrated productivity and excellence in research for the 2016-2017 school year

**Phi Beta Kappa**

Inducted:

May 2016

**Undergraduate Research Conference Travel Award**

October 2015

- Awarded \$500 to present a poster on research from the Personality and Behaviors Over Time Study at the 2015 annual conference of the Association for Behavioral and Cognitive Therapies in Chicago, IL

**John A. Stern Undergraduate Research Award**

September 2014

- Awarded \$480 for the Personality and Health Behaviors Over Time Study
- Funds used for participant reimbursement in order to meet study recruitment goals and increase retention of participants over the course of the study

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**MANUSCRIPT PUBLICATIONS**

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2. **Brosof, L. C.**, Munn-Chernoff, M. A., Bulik, C., & Baker, J. H. (2019). Associations between eating expectancies and eating disorder symptoms in men and women. *Appetite*, 141, 104309. <https://doi.org/10.1016/j.appet.2019.06.001>
3. **Brosof, L. C.**, Calebs, B., & Levinson, C. A. (2019). Testing for ethnic invariance between Asian and European Americans in a six-month prospective model of eating disorder and social anxiety comorbidity. *Eating Disorders: The Journal of Treatment and Prevention*, 27, 110-122. <https://doi.org/10.1080/10640266.2019.1585678>
4. **Brosof, L. C.**, & Levinson, C. A. (2017). Social appearance anxiety and dietary restraint as mediators between perfectionism and binge eating: A six month three wave longitudinal study. *Appetite*, 108, 335-342. <https://doi.org/10.1016/j.appet.2016.10.015>
5. **Brosof, L. C.**, Williams, B. M., & Levinson, C. A., (2019). Exploring the contribution of exercise dependence to eating disorder symptoms. *International Journal of Eating Disorders*. <https://doi.org/10.1002/eat.23156>
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7. Christian, C., **Brosof, L. C.**, Vanzhula, I., Williams, B., Shankar Ram, S., & Levinson, C. A. (2019). Implementation of a dissonance-based, eating disorder prevention program and investigation of eating disorder comorbidities and risk factors in southern, all-female high schools. *Body Image*, 30, 26-34. <https://doi.org/10.1016/j.bodyim.2019.05.003>
8. Christian, C. Ngo, B., **Brosof, L. C.**, & Levinson, C. A. (2020). Social appearance anxiety moderates the relationship between thin ideal internalization and eating disorder symptoms. *Eating and Weight Disorders: Studies on Anorexia, Bulimia, and Obesity*. doi: 10.1007/s40519-020-01050-y.
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10. Gorrell, S., Reilly, E. E., **Brosof, L. C.**, & Le Grange, D. (2022). Use of telehealth in the management of adolescent eating disorders: Patient perspectives and future directions suggested from the COVID-19 pandemic. *Adolescent Health, Medicine and Therapeutics*, 13, 45-53. <https://doi.org/10.2147/AHMT.S334977>
11. Levinson, C. A., **Brosof, L. C.**, Shankar Ram, S., Pruitt, A., Russell, S., & Lenze, E. J. (2019). Obsessions are strongly related to eating disorder symptoms in anorexia nervosa and atypical anorexia nervosa. *Eating Behaviors*, 34, 101298. <https://doi.org/10.1016/j.eatbeh.2019.05.001>
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- Discharged from Intensive Treatment. *Eating Behaviors*, 67, 45-51.  
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  15. Levinson, C. A. & **Brosof, L. C.** (2016). Cultural and ethnic differences in eating disorders and disordered eating behaviors. *Current Psychiatry Reviews*, 12, 163-174.
  16. Levinson, C. A., Hunt, R. A., Keshishian, A. C., Brown, M. L., Vanzhula, I., Christian, C., **Brosof, L. C.**, & Williams, B. M. (2021). Using individual networks to identify treatment targets for eating disorder treatment: A proof-of-concept study and initial data. *Journal of Eating Disorders*, 9, 147. DOI: 10.1186/s40337-021-00504-7
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26. Sala, M., **Brosof, L. C.**, Rosenfield, D., Fernandez, K., & Levinson, C. A. (2017). Stress is associated differently among individuals with higher and lower levels of eating disorder symptoms: An ecological momentary assessment study. *International Journal of Eating Disorders*, 50, 1413-1420. <https://doi.org/10.1002/eat.22799>
27. Schaumberg, K., **Brosof, L. C.**, Micali, N., & Zerwas, S. (2019). Childhood neuropsychological profiles demonstrate association with adolescent eating disorder symptoms. *European Eating Disorders Review*. <https://doi.org/10.1002/erv.2721>
28. Yilmaz, Z., Schaumberg, K., Halvorsen, M., Goodman, E., **Brosof, L. C.**, Crowley, J. J., Mathews, C., Mattheisen, M., Breen, G., Bulik, C. M., Micali, N., & Zerwas, S. C. (2022). Predicting eating disorder and anxiety symptoms using anorexia nervosa and obsessive-compulsive disorder polygenic risk scores. *Psychological Medicine*, 1-15. DOI: [10.1017/S0033291721005079](https://doi.org/10.1017/S0033291721005079)

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## SYMPOSIA PRESENTATIONS

- Brosof, L. C.**, Trombley, C., & Levinson, C.A. (September 2019). *Prediction of binge eating and purging symptoms using machine learning*. Paper presented at the Eating Disorders Research Society, Chicago, Illinois. Selected for Best Student Abstract Award.
- Brosof, L. C.**, Knight, L., Hunt, K. J., Levinson\*, C. A., & Depue\*, B. (2019, March). *Imaginal exposure eating disorder fear scripts are associated with increased activation related to threat and internally processed thought compared to neutral scripts: A proof-of-concept fMRI study*. Paper presented at the International Conference of Eating Disorders, New York, NY. \*joint senior authorship
- O'Connor, S.S., **Brosof, L.C.**, Vanzhula, I., & Levinson, C.A (2019, April). *Eating disorder fears are uniquely associated with suicidality*. Paper presented at the American Association of Suicidology Conference, Denver, CO.
- Sala, M., **Brosof, L. C.**, & Levinson, C.A. (March, 2019). *Rumination predicts eating disorder behaviors: An ecological momentary assessment study in a clinical eating disorder sample*. Paper presented at the Academy of Eating Disorders, International Conference on Eating Disorders, New York, NY.
- Brosof, L. C.**, Vanzhula, I.A., Shankar Ram, S. Christian, C., Michelson, L., & Levinson, C.A. (November, 2018). *Interacting risk factors: Higher emotional impulsivity and lower maladaptive perfectionism is associated with higher bulimic symptoms*. In **L.C. Brosopf**: Transdiagnostic processes in eating disorders: How shared mechanisms can inform treatment. Paper presented at the Association for Behavioral and Cognitive Therapies, Washington D.C.
- Brosof, L. C.**, Vanzhula, I. A., & Levinson, C. A. (November, 2018). *A combined latent variable and network model of eating disorder symptoms in two eating disorder measures*. In S. Wang: Novel insights on eating disorder treatment and etiology from network and complexity science. Paper presented at the Association for Behavioral and Cognitive Therapies, Washington D.C.
- Levinson, C.A., Fewell, L., **Brosof, L. C.**, Fournier, L.C., Lenze, E.J. (2018, April). *Concern*

- Over Mistakes Prospectively Predicts Obsessive Compulsive Disorder and Anorexia Nervosa Symptoms in Eating Disorder Patients After Discharge from Intensive Treatment.* Paper presented at the International Conference on Eating Disorders, Chicago IL.
- Brosof, L. C.,** Sala, M., Fernandez, K. C., & Levinson, C. A. (2017, November). *Body dissatisfaction moderates the relationship between social anxiety and exercise frequency: An ecological momentary assessment study.* In **L.C. Brosof & M. Sala** (chairs), Exercise and eating pathology. Paper presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Levinson, C. A., Fewell, L. **Brosof, L. C.,** Fournier, L., & Lenze, E. J. (2017, June). *Mealtime eating disorder cognitions predict eating disorder behaviors: A mobile technology based ecological momentary assessment study.* Paper presented at the annual conference of the Academy for Eating Disorders, Prague, Czech Republic.
- Levinson, C. A., Vanzhula, I., **Brosof, L. C.,** Calebs, B., Michelson, L., & Fewell, L. (2017, November). *A trial of online imaginal exposure therapy for eating disorder related fears in individuals with anorexia nervosa.* In N. Farrell (chair). A New Way Forward? Novel Applications of Exposure-Based Therapy in the Context of Eating Disorders. Paper presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Sala, M., **Brosof, L. C.,** Rosenfield, D., Fernandez, K. C., & Levinson, C. A. (2017, November). *Exercise regulates anxiety and stress differently among individuals with higher and lower levels of disordered eating: An ecological momentary assessment study.* In **L.C. Brosof & M. Sala** (chairs), Exercise and eating pathology. Paper presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Smith, T., Levinson, C. A., **Brosof, L. C.,** Bulik, C., & Zerwas, S. (2017, November). Using network analysis to examine how core bulimia nervosa symptoms and symptom networks change with treatment. Paper presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Levinson, C. A., Fewell, L., & **Brosof, L. C.** (2016, October). *Using network analysis to explain eating disorder and obsessive compulsive disorder symptom overlap.* In H.B. Murray & B. Evans (chairs), Network analysis as an innovative approach to understanding eating behavior: Identifying key treatment targets in eating and weight disorders. Paper presented at the Association for Behavioral and Cognitive Therapies, NY, NY.

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## POSTER PRESENTATIONS

- Brosof, L.C.,** & Levinson, C.A. (2020, October). Perfectionism predicts cognitive-behavioral outcomes and food intake: An experimental manipulation of concern over mistakes. Poster presented at the Eating Disorders Research Society, Virtual.
- Brosof, L. C.,** Knight, L., Hunt, K. J., Depue, B., & Levinson, C. A. (2019, March). *Imaginal exposure eating disorder fear scripts are associated with increased activation related to threat and internally processed thought compared to neutral scripts: A proof-of-concept fMRI study.* Poster presented at the International Conference of Eating Disorders, New York, NY.
- Brosof, L. C.,** & Levinson, C. A. (2019, March). *Manipulating concern over mistakes increases restrictive eating: An experimental test of the impact of perfectionism on eating behaviors.* Poster presented at the International Conference of Eating Disorders, New York, NY.

- Schaumberg, K., **Brosof, L. C.**, Lloyd, C., Micali, N., & Zerwas, S. (2019, March). *Childhood neuropsychological predictors of disordered eating behaviors in adolescence*. Poster presented at the International Conference of Eating Disorders, New York, NY.
- Brosof, L. C.**, & Levinson, C. A. (2018, April). *Guilt, Shame, and Fear of Weight Gain are Bridge Symptoms Between Negative Affect and Eating Disorder Symptoms*. Poster presented at the International Conference of Eating Disorders, Chicago, IL.
- Brosof, L. C.**, Levinson, C. A., Goodman, E., Schaumberg, K., Bulik, C., Micali, N., & Zerwas, S. (2018, April). *Purging is a bridge symptom in a network model of eating disorder and obsessive-compulsive symptoms*. Poster presented at the International Conference of Eating Disorders, Chicago, IL.
- Brosof, L. C.**, Munn-Chernoff, M. A., Bulik, C., & Baker, J. H. (2018, April). *Associations between Eating Expectancies and Eating Disorder Symptoms in Men and Women*. Poster presented at the International Conference of Eating Disorders, Chicago, IL.
- Brosof, L. C.**, Ngo, B., & Levinson, C. A. (November, 2018). *Social Appearance Anxiety Moderates the Relationship Between Thin Ideal Internalization and Eating Disorder Symptoms*. Poster presented at the Association for Behavioral and Cognitive Therapies, Washington D.C.
- Brosof, L. C.**, Vanzhula, I., Christian, C., & Levinson, C. A. (2018, April). *Concern over mistakes connects social anxiety and eating disorder symptoms: A network analysis*. Poster presented at the Anxiety and Depression Association of America, Washington D. C.
- Brosof, L. C.**, Adams, W. B., & Levinson, C. A. (November, 2018). *Maladaptive perfectionism uniquely contributes to eating disorder impairment*. Poster presented at the Association for Behavioral and Cognitive Therapies, Washington D.C.
- Christian C., Vanzhula I.A., Calebs, B., **Brosof, L. C.**, Michelson, L. P., Stratigis, K.Y., Hicks- Chambers, A., Toon, A.E., Fewell, L. K., Levinson, C.A. (2018, February). *Effectiveness of Online Imaginal Exposure Therapy for the Treatment of Eating Disorders*. Poster presented at Posters at the Capitol, Frankfort, KY.
- Levinson, C.A., **Brosof, L.C.**, Vanzhula, I.A., Bumberry, L., Zerwas, S., Bulik, C.M. (2018, April). *Perfectionism Group Treatment for Eating Disorders in an Inpatient, Partial Hospitalization, and Outpatient Setting: Preliminary Support for Reducing High Standards but not Concern Over Mistakes*. Poster presented at the International Conference on Eating Disorders, Chicago IL.
- Brosof, L. C.**, Levinson, C. A., & Fewell, L. (2017, June). *Exercise Addiction: Exploring the contribution of exercise dependence to eating disorder symptoms*. Poster presented at the annual conference of the Academy for Eating Disorders, Prague, Czech Republic.
- Brosof, L. C.**, Lee, A. H., & Levinson, C. A. (2017, November). *Higher personal standards but not evaluative concerns perfectionism moderates the relationship between intolerance of uncertainty and eating disorder symptoms cross-sectionally and prospectively over two weeks*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Brosof, L. C.**, & Levinson, C. A. (2017, November). *Gender matters: The roles of perfectionism, shame, and pride are different for eating disorder symptoms in males versus females*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.
- Lee, A. H., **Brosof, L. C.**, & Woodruff-Borden, J. (2017, November). *Examining the roles of intolerance of uncertainty and perfectionism in attentional control*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.

Melton, S. M.\*, **Brosof, L. C.**, & Levinson, C. A. (2017, November). *The roles of mindfulness and impulsivity in eating disorder symptoms*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.

\*denotes student mentee author.

Michelson, L.\*, **Brosof, L. C.**, & Levinson, C. A. (2017, November). *Age moderates the relationship between anxiety about eating and eating disorder symptoms*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, San Diego, CA.

\*denotes student mentee author.

**Brosof, L. C.**, & Levinson, C. A. (2016, October). *Replicating and extending a maintenance model of binge eating in a longitudinal, four wave study across four weeks*. Poster presented at the Association for Behavioral and Cognitive Therapies, New York, NY.

**Brosof, L. C.**, & Levinson, C. A. (2016, October). *Testing for ethnic invariance between Asian and European Americans in a six month prospective model of eating disorder and social anxiety comorbidity*. Poster presented at the Association for Behavioral and Cognitive Therapies, New York, NY.

**Brosof, L. C.**, & Levinson, C. A. (2015, November). *Social appearance anxiety is the domain of social anxiety that predicts binge eating over time*. Poster presented at the annual conference of the Association for Behavioral and Cognitive Therapies, Chicago, IL.

**Brosof, L. C.**, & Levinson, C. A. (2015, May). *Social appearance anxiety mediates the relationship between concern over mistakes and binge eating: A six-month three wave longitudinal study*. Poster presented at the annual conference of the Association for Psychological Science, New York, NY.

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## RESEARCH EXPERIENCE

### **Eating Anxiety Treatment (EAT) Lab**

August 2016 –

present

University of Louisville, Louisville, KY

Faculty Advisor: Cheri A. Levinson, Ph.D.

Role: Graduate Student

**Dissertation: Investigating the shared and unique mechanisms of comorbid eating disorder-anxiety symptoms during adolescence**

### **Anxiety and Psychotherapy Lab**

August 2013 –

May 2016

Washington University in St. Louis, St. Louis, MO

Faculty Advisor: Thomas L. Rodebaugh, Ph.D.

Role: Undergraduate Research Assistant

### **Healthy Mind Lab**

December 2015 –

May 2016

Washington University in St. Louis, St. Louis, MO

Faculty Advisor: Eric Lenze, Ph. D.

Research Supervisor: Cheri A. Levinson, Ph. D.

Role: Undergraduate Research Assistant

**Weight Management and Eating Disorders Research Lab**  
December 2015

June 2014 –

Washington University in St. Louis, St. Louis, MO

Faculty Advisor: Denise E. Wilfley, Ph. D.

Supervisor: Ellen Fitzsimmons-Craft, Ph. D.

Role: Undergraduate Research Assistant

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## **CLINICAL EXPERIENCE**

**Noble H. Kelley Psychological Services Center at the University of Louisville**

Role: Therapist

### **Eating Disorder Specialty Rotation (June 2017 – July 2020)**

Supervisor: Cheri Levinson, Ph.D.

Hours: 4-6 hours per week

- Provided individual psychotherapy to individuals with AN, BN, BED, and OSFED
- Collaborated with multidisciplinary team of dietitians, psychiatrists, and physicians
- Conducted intake assessments and made recommendations for level of care
- Attended weekly didactic training and received weekly group and individual supervision led by a licensed psychologist
- Implemented CBT-E, FBT, exposure therapy, DBT, and ACT
- Facilitated a weekly eating disorder recovery support group

### **Peer Clinical Supervisor (August 2018 – July 2020)**

Supervisor: Cheri Levinson, Ph.D.

Hours: 1-2 hours per week

- Peer-supervised clinical psychology doctoral students weekly
- Reviewed videotaped sessions and provided feedback
- Facilitated case conceptualization and treatment planning
- Trained in clinical supervision from licensed clinical psychologist

### **Assessment Rotation (June 2017 – August 2018)**

Supervisors: David Winch, Ph.D. and Bernadette Walter, Ph.D.

Hours: 3 hours per week

- Administered test batteries to assess cognitive functioning, learning disabilities, and psychiatric diagnoses in adults and children
- Assessments included: intelligence (WAIS-IV and WISC-V), achievement (Woodcock-Johnson III), personality (MCMI-III), and psychopathology (SCID, SCID-P, MINI, CPT-III)
- Completed 3 integrative reports

**Integrated Psychotherapy Rotation**\_(August 2016 – June 2017)

Supervisor: Rich Lewine, Ph.D.

Hours: 4-6 hours per week

- Provided individual psychotherapy to patients with depression, anxiety, and stress
- Conducted structured intake interviews
- Implemented integrated interventions utilizing a range of evidence-based therapies, including mindfulness-based treatments and CBT
- Developed individualized case conceptualizations and treatment plans integrating various theoretical orientations
- Attended weekly didactic training, as well as group and individual supervision led by a licensed psychologist

**Louisville Center for Eating Disorders and Louisville OCD Clinic** August 2019 – present

Role: Therapist

Supervisors: Mark Schirmer, Psy.D., Licensed Psychologist

Street Russell, Psy.D., Licensed Psychologist and Associate Director

- Serve as outpatient and intensive outpatient therapist for clients with eating disorders
- Serve as outpatient therapist for clients with OCD and other anxiety disorders
- Lead meal therapy groups for intensive outpatient program
- Lead weekly exposure group therapy session for intensive outpatient program
- Co-lead group therapy sessions, such as Body Image group and Eating Disorder Thoughts group, for intensive outpatient program

**University of Louisville Outpatient Psychiatry Clinic and Depression Center**

Role: Therapist and DBT Skills Group Co-leader

August 2018-

May 2019

Supervisor: Stephen O'Connor, Ph.D., Assistant Professor, Department of Psychiatry and Behavioral Sciences, Associate Director, University of Louisville Depression Center

- Serve as outpatient therapist for clients of psychiatry clinic and depression center using cognitive behavioral techniques
- Co-lead weekly Dialectical Behavior Therapy (DBT) Skills group
- Serve as therapist for pilot study using Collaborative Assessment and Management of Suicidality (CAMS) approach for individuals following hospitalization for suicide attempt

**Online and In Vivo Imaginal Exposure Studies**

Diagnostic Assessor and Therapist (January 2017-present)

Supervisor: Cheri A. Levinson, Ph.D.

- Conduct structured clinical interviews to determine participant inclusion
- Administer imaginal exposure therapy for clients with eating disorders

**Anorexia Nervosa and Associated Disorders (ANAD) Support Group**

Support Group Leader: April 2017- present

- Serve as a support group leader for individuals with eating disorders

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**COMMUNITY OUTREACH/SERVICE EXPERIENCE**

**The Body Project Eating Disorder Prevention Program**

February 2017-

ongoing

Trainer and Facilitator

- Trained as a high school facilitator in January 2017 and trainer in January 2018
- Trained as a college facilitator and trainer in January 2018
- Served as a facilitator for an empirically supported body acceptance and dissonance-based eating disorder prevention program in local high school and colleges

**Louisville National Eating Disorder Association Walk**

September

2016-2019

- Served as team captain to encourage participation and helped fundraise for the 2016 walk
- Serving on the committee to help plan, recruit, and fundraise for the 2017 and 2018 walks

**Eating Disorder Clinical Training**

August 2019

- Provided two-hour clinical training to Psy.D. interns on eating disorder etiology, assessment, and treatment

**Prospective Graduate Student Transportation Organizer**

January 2017

and 2018

- Aided in organizing transportation for prospective student interviews

**National Depression Screening Day Screener**

October 2017

and 2019

- Screened community members for symptoms of depression and anxiety and provided appropriate resources and referrals on National Depression at the Noble H. Kelley Psychological Services Center

**Kentucky Air National Guard – Louisville Base Talk**

February 2017

- Gave a talk on healthy eating to members of the Kentucky Air National Guard as part of a series trying to increase health behaviors and healthy and mindful eating

**Louisville National Alliance on Mental Illness Walk**

August 2016

- Participated in and fundraised for the 2016 walk

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## TEACHING/MENTORSHIP EXPERIENCE

### Graduate Teaching Assistant

July 2018 –

present

University of Louisville, Louisville, KY

Courses: Advanced Statistics I (Psyc 610) and II (Psyc 611)

- Serve as teaching assistant for advanced statistical courses in psychology for graduate students covering topics from probability to ANOVA statistical techniques
- Teach labs on how to conduct statistical tests in SPSS Statistics
- Guest lecture on relevant topics, such as logistic and multiple regression

### Mentorship of Research Assistants

August 2016 –

present

University of Louisville EAT Lab, Louisville, KY

- I directly supervise research assistants, which includes responsibilities of teaching research skills such as analyzing data SPSS, writing IRB protocols, conducting literature searches, and running participants

### Mentor for High School Science Fair Project

August 2016 –

May 2019

University of Louisville EAT Lab, Louisville, KY

- I helped a student from DuPont Manual High School, located in Louisville, with her 2017 and 2018 science fair projects on body image in adolescents. I taught her independent research skills such as creating an IRB proposal for the study, study creation and design, recruitment, data analysis, and presentation preparation.
  - She received first place in the science fair for her project in 2017 and 2018
- I am continuing to serve as her mentor for her 2019 science fair project

### Guest Lecturer

November 2017-

present

University of Louisville, Louisville, KY

- Served as a guest lecturer on eating disorders for an undergraduate Abnormal Psychology Honors course
- Served as guest lecturer on eating disorders for graduate Psychopathology course

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## STATISTICAL WORKSHOPS

### Curran and Bauer Network Analysis Workshop

June 2017

Chapel Hill, North Carolina

- Attended three-day workshop on network analysis



**ICPSR Machine Learning: Uncovering Hidden Structure in Data**  
2, 2019  
Berkeley, California

July 29-August

- Attended five-day workshop on unsupervised and supervised machine learning methods

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## **PROFESSIONAL AFFILIATIONS**

Kentucky Psychological Association, Student Affiliate present	2016 –
American Psychological Association, Student Member present	2016 –
Academy for Eating Disorders, Student Member present	2016 –
Association for Behavioral and Cognitive Therapies, Student Affiliate present	2014 –
Psi Chi, Student Affiliate present	2014 –