Understanding correlates and comorbidities in the treatment and recovery of adolescent eating disorders

DISSERTATION

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Abstract

Eating disorders are a serious mental illness characterized primarily by disturbances in eating behaviors and body image. Eating disorders often emerge during adolescence, a period in the life course that is comprised of numerous biological, psychological, and social changes. As a result, adolescent eating disorders can significantly alter the trajectory of one’s life without proper intervention. Although studies examining adolescent eating disorders are abundant, some aspects of the nature of eating disorders in adolescence remain understudied. Despite the complex nature of eating disorders, studies often focus on specific biological, psychological, or social factors associated with eating disorders. The current study, therefore, uses a biopsychosocial approach to investigate correlates and comorbidities associated with eating disorder symptoms and recovery in a clinical sample of adolescents. Secondary administrative data from an urban pediatric hospital’s eating disorder clinic were used for this investigation.

This dissertation is separated into three independent studies. In chapter 2, I examine the prevalence of trauma in a sample of adolescent patient receiving outpatient treatment for an eating disorder. I use bivariate statistics and binomial and multinomial logistic regression models to examine similarities and differences in eating disorder symptoms for those with and without significant trauma exposure. Results show that over
one-third of the sample experienced at least one lifetime traumatic event and traumatic exposure was significantly related to bulimia nervosa and higher weight status (body mass index ‘BMI’ and percent expected body weight). In the following chapter, I examine the prognostic factors associated with early weight restoration in a clinical sample of adolescents receiving outpatient treatment for anorexia nervosa. Findings reveal comorbid anxiety as significant positive predictor of weight restoration at 3 month follow up. Given the importance of comorbid symptoms in the presentation of eating disorders, chapter 4 examines differences in youth and caregiver reports of anxiety and depression symptoms. I find that youth and caregiver reports of comorbid symptoms are significantly discrepant with caregivers reporting higher levels of anxiety and depression than youth. Families with younger adolescents and adolescents with subclinical threshold eating disorder symptoms reported significantly less congruence. I address limitations, future research directions, clinical implications, and implications for overall adolescent development in chapter 5.
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Chapter 1: Introduction

Eating disorders are a great public health concern, holding the highest mortality rate of all mental health disorders for both adolescents and emerging adults, with anorexia nervosa having the most elevated mortality rates (Arcelus, Mitchell, Wales, & Nielson, 2011). Research suggests that the earlier anorexia nervosa is treated the greater likelihood of successful long term outcomes (Lock et al., 2010). However, adolescents often do not want to participate in treatment, not only out of fear associated with weight gain and recovery but also because of the fear of missing out on "normal" adolescent activities (Treasure & Schmidt, 2008). In addition, maladaptive eating disorder behaviors are often perceived by individuals as necessary and purposeful to manage their emotional and physical well-being (Gale, Hol lidsay, Troop, Serpell, & Treasure, 2006). This perception can further fuel their ambivalence to treatment and resistance to change. The goal of this dissertation is to use a biopsychosocial approach (Engel, 1977) to understanding important factors that influence initial presentation and progress over time in a clinical sample of adolescents seeking treatment for an eating disorder, particularly anorexia nervosa, at a Midwestern hospital clinic. This study specifically seeks to examine the relationship between trauma and eating disorders, identify correlates for success and/or failure in treatment over time, and examine the relationship between comorbid anxiety and depression and clinical presentation in adolescents and families seeking treatment.
Literature Review

Definition and Prevalence

Anorexia nervosa (AN) is an eating disorder and serious mental illness characterized by the inability to maintain a healthy body weight due to the restriction of energy intake (American Psychiatric Association, 2013). Diagnostic criteria for AN include the restriction of energy intake, an intense fear of gaining weight, and disturbance in the way one experiences their body weight or shape. Individuals with AN often suffer from body weight that is significantly lower than what is considered normal given their age, sex, and developmental trajectory. Body Mass Index in adults and gender and age specific BMI percentiles in children and adolescents are used to determine severity of the illness. Anorexia can be classified as mild, moderate, severe, or extreme and has two distinctive subtypes, restricting and binge-eating/purging (American Psychiatric Association, 2013). Presentations of restricting subtype are characterized by weight loss due to excessive dieting, fasting, and exercise while binge-eating/purging describes episodes of binge-eating or purging behaviors such as vomiting and the use of laxatives.

Anorexia nervosa is most common among females (10:1 female to male ratio) with onset usually in adolescence or early adulthood. Anorexia nervosa has a prevalence rate of 0.3% to 1.20% in the total population and .3% in adolescents ages 12-22 years-old (American Psychiatric Association, 2013). Approximately 74 adolescent females out of 100,000 will experience AN in their lifetime (Lock et al., 2010). Despite seemingly low prevalence rates, the impact of an eating disorder has significant impairments for an individual’s role functioning (i.e. the ability to participate in one’s expected role within a
system such as a school, family, occupation, or community), somatic symptoms, and additional co-morbid mental and medical illnesses. Common co-occurring mental disorders include anxiety, depression, and substance use (Blinder, Cumella, & Sanathara, 2006; Bulik, 2000; Swinbourne, Hunt, Abbott, Russell, St Clare, & Touyz, 2012). In addition, individuals with anorexia often struggle with low self-esteem and obsessive compulsive patterns around eating and exercise (Lock & Le Grange, 2012).

Despite much speculation over the years about the etiology of AN, it is general consensus that the cause of AN is unknown (Lock & Le Grange, 2012). Although there is no determined cause, research suggests that there are certain risk and prognostic factors. Temperament, environment, and genetic factors appear to play a role in the overall development of AN (American Psychological Association, 2013). Individuals more prone to anxiety disorders are often more likely to develop anorexia. In addition, cultural and societal emphasis on thinness is also attributed to increased risk along with family history of the disorder.

**Eating Disorders and Trauma**

Previous research has reported a significant relationship between traumatic experiences and the development of an eating disorder in both adults and adolescents (Brewerton, 2006; 2007; Johnson, Cohen, Kasen, & Brook, 2002). In addition, patients with an eating disorder who have experienced trauma, also are likely to report other psychiatric comorbidities, especially post-traumatic stress disorder (Brewerton, 2007; Reyes et al., 2011). However, there are significant limitations in the current literature that influence our understanding of the relationship between trauma and eating disorders. First, the majority of studies examining the relationship between trauma and eating
disorders focus primarily on childhood sexual abuse (CSA) (Smolak & Murnen, 2002; Wonderlich, et al., 2001), whereas less is known about the impact of other forms of child abuse (i.e., maltreatment and neglect) on the prevalence and clinical presentation of an eating disorder. Second, past researchers have identified a significant relationship between trauma and bulimia nervosa (BN) (Brewerton, 2007; Wonderlich, Brewerton, Jocic, Dansky, & Abbot, 1997), but not the ways in which trauma is related to AN, specifically AN restricting subtype. The significance of trauma history among those diagnosed with eating disorders warrants our clinical and scholarly attention in an effort to provide the best treatment for what is known to be a pervasive and often difficult to treat mental disorder. This project works to broaden our understanding of the relationship between trauma exposure in childhood and eating disorders in a clinical population of adolescents.

**Predictors of recovery**

Because anorexia is a difficult mental disorder to treat, it is important that clinicians and researchers are educated about the various factors that could potentially impede or increase the likelihood of successful recovery. A large multi-site study of adolescents and adults diagnosed with anorexia found that higher levels of anxiety and the lifetime presence of vomiting were negative prognostic factors linked to lower likelihood of recovery (Zerwas et al., 2013). The same study also found that impulsivity at onset was a positive prognostic factor in predicting recovery (Zerwas et al., 2013). A different study examining five samples of eating disorder patients from randomized clinical trials found that the best predictor of recovery in adolescents for patients with AN was achieving a body weight that is approximately 95% of their expected body weight
This finding is consistent with previous research that suggests attaining greater than 90% of one’s EBW by end of treatment (EOT) is an important indicator for recovery (Courtier & Lock, 2006). Research on other prognostic factors that can predict recovery from AN suggest that very low initial body weight, longer duration of illness, older age at onset of diagnosis, greater intensity and frequency of symptoms, comorbidity with other psychiatric disorders, lower socioeconomic status, and disturbed family relationships may all be significant in predicting sustained recovery (Howard, Evans, Quintero-Howard, Bowers, & Andersen, 1999; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Striegel-Moore & Bulik, 2007). However, research on prognostic factors often focuses on the predictive power of specific factors, focusing on biological, psychological or social factors (Striegel-Moore & Bulik, 2007; le Grange, 2016). An integrative biopsychosocial approach (Le Grange, 2016) has been suggested to help researchers and clinicians better understand the etiology of anorexia and other eating disorders. I further contend that the same integrative biopsychosocial approach can and should be used to better understand the course of anorexia over time and the journey toward recovery. Thus, this project adds to the existing literature by seeking to further our understanding of the biological, psychological, and social prognostic factors that predict recovery and/or decline in adolescent patients seeking treatment for AN. This project further seeks to examine an integrative model of recovery and promote clinical dialogue around ways in which treatment approaches can specifically address multiple factors that affect the likelihood of recovery.
**Anxiety, Depression, and Anorexia Nervosa**

Clinical, epidemiological, and family science research has shown a significant relationship between eating, depression and anxiety disorders (Bulik, 2002). Multiple models exist that attempt to explain the comorbidity between eating, affective, and anxiety disorders. The most compelling model suggests that eating disorders, anxiety, and depression have both shared and specific etiological factors (Bulik, 2002). The clinical manifestation of anxiety in patients with AN is characterized by increased fear and concern over weight, body shape, and appearance. Preoccupation with food is also seen as a manifestation of obsessive compulsive traits of anxiety. Empirical studies have examined the association between malnutrition and anxiety. Some suggest that as nutrition and weight status improve there is a clinically significant decline in anxiety symptoms (Bulik, 2000; Sala, Mirabel-Sarron, Gorwood, Pham-Scottez, Blanchet & Rouillon, 2011). However, other studies provide a lack of evidence for this relationship (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Kezelman, Touyz, Hunt, & Rhodes, 2015). Findings from clinical studies suggest that the onset of anxiety disorders often proceed the development of AN and over half of women diagnosed with AN report a lifetime presence of an anxiety disorder (Bulik, 2000).

The clinical presentation of individuals with AN often reveals depressed affect, low self-esteem, sense of hopelessness and worthlessness, and heightened self-harm and suicidal ideation and behaviors (American Psychiatric Association, 2013; Bulik, 2002). Research suggest that for depression, patterns of onset occur before and after onset of AN. Similar to anxiety and anorexia, some relief from depressed mood may occur as
malnutrition is resolved; however, long term outcome studies suggest that depression may persist even after eating disorder symptoms are resolved (Bulik, 2002; Godart et al., 2007). Results from twin studies have revealed both unique and shared genetic factors that lead to the development of depression and AN (Bulik, 2002; Wade, Bulik, Neale, & Kendler, 2000).

Due to the lack of motivation for change in eating disorder patients, it is often the primary care physician or primary caregiver who initiates treatment for adolescents (Hudson, Hiripi, Pope, & Kessler, 2007). Although low body weight or rapid weight loss may cause some concern, it is often symptoms of anxiety and depression that are noticed by parents and other professionals and propel patients into treatment (Fursland & Watson, 2014). For this reason, the high comorbidity of depression and anxiety in patients with anorexia and other eating disorders is of great concern. Still, little is known about caregiver perceptions of anxiety and depression symptoms in adolescents seeking eating disorder treatment. The onset and/or increased severity of anxiety and depression may serve to increase a family’s urgency to seek treatment sooner than weight loss alone. This project seeks to better understand the influence of depression and anxiety symptoms and co-morbid diagnoses on treatment outcomes over time. It also seeks to address the current gap in the literature around family perceptions of depression and anxiety in adolescents initially seeking eating disorder treatment.

**The Biopsychosocial Model**

The biopsychosocial model (BPS) is a systems-oriented approach to understanding and treating illness and disease (Engel, 1977; 1980). The BPS model reconciles the biological and psychosocial factors relevant in healthcare through the lens
of general systems theory (GST). General systems theory posits that all systems are characterized by the interactions of its components and the nonlinearity of those interactions (von Bertalanffy, 1968). The BPS model was created in response to the linear biomedical model that dominated the medical and mental health fields (Engel, 1977). The BPS model argues that one must understand the biological, psychological and social factors that contribute to an illness and/or help move toward recovery (Engel, 1977; 1980).

Research on the etiology of anorexia suggest that a comprehensive model requires consideration of the influences of genes, environment, and biological/personality factors (Le Grange, 2016). As research indicates, there are a number of risk factors that lead to the development of anorexia. The complex nature of anorexia, and all eating disorders, calls for a holistic and integrative approach to understanding this illness. The biopsychosocial model provides this holistic approach and allows us to examine the multiple systems that interact to influence development, maintenance, treatment, and recovery of AN. Figure 1.1 provides the biopsychosocial model of eating disorder symptomology used in this dissertation. It is important to note that this model assumes the biological, psychological and social risk factors influence and are influenced by one another. The clinical presentation of AN is often accompanied with a number of physiological as well as mental/emotional symptoms and co-morbidities. The following sections outline the biological, psychological, and social factors associated with AN.

**Biological Factors**

Gender, family history of anxiety and eating disorders, and brain chemistry are all significant risk factors for developing anorexia (Lock & Le Grange, 2012). Incidence
rates suggest that females are significantly more likely (10:1) to develop anorexia than their male counterparts (American Psychiatric Association, 2013). Studies focusing on the biological factors that influence the development of anorexia suggest that there are unique genetic factors that contribute to AN. Although no specific gene has been identified, AN has been found to be particularly heritable, with findings that having a first degree relative with an AN diagnosis increases one’s risk of developing the disorder by 54% (Berrettini, 1998). A review of molecular genetic research on anorexia found that genetic effects account for over 50% of the variance in developing AN (Klump & Gobrogge, 2005). Brain imaging studies have attempted to better understand differences in the brains of individuals with and without anorexia. Results from one study found over activity of dopamine receptors in the basal ganglia, an area of the brain that controls reward and reinforcement, of recovered women when compared to women with no history of the disorder (Frank et al., 2005). This finding suggests that dopamine plays a contributing role in the pathophysiology of AN.

**Psychological Factors**

Individuals with AN are often characterized as high achieving and or perfectionistic. Personal values and beliefs may center around unrealistic standards of beauty and thinness or the ability to be in control. Although it is difficult to disentangle certain values from the individual and the influence of the disorder, understanding the cognitions and values of individuals with AN may help to alter and treat cognitive distortions that contribute to the maintenance of disordered eating. The presence of certain psychiatric disorders has also been identified as risk factors for the development of eating disorders. Research has found that individuals who develop an eating disorder
often had a preexisting or primary anxiety disorder (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). In addition, individuals with obsessive compulsive patterns have been found to have a higher risk for developing anorexia (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Cederlöf et al., 2015). Females with eating disorders have significantly higher reports of depression than community controls (Godart et al., 2007). The presence of partial or subthreshold post-traumatic stress disorder (PTSD) has been cited as a potential risk factor for bulimia nervosa and purging symptoms (Brewerton, 2007; Mitchell et al., 2012).

**Social Factors**

Research on sociocultural models of body dissatisfaction and eating disorders have found that exposure to social pressure to adhere to a thin ideal body type can lead to internalization of this unattainable ideal and result in body dissatisfaction and disordered eating behaviors in an effort to solve the discrepancy that exists between self and ideal body type (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; Thompson & Stice, 2001). The Triparte Model suggests that three social influences of, peers, parents, and media, significantly affect body image concerns and eating disturbance by way of internalization of the thin ideal and comparison processes (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999). This model has received empirical support in samples of United States adolescents and young adults (Keery, den Berg, & Thompson, 2004; Thompson & Stice, 2006; Van den Berg, Thompson, Obremski-Brandon, & Coover). Involvement in sports or other activities that emphasize thinness or a specific body type are one example of how societal pressure may be reinforced and result in disordered eating (Lock &
LeGrange, 2012). The presence of stress in the family is also seen as one factor that may often proceed the development of AN as the disorder (Loth, van den Berg, Eisenberg, & Neumark-Sztainer, 2008). The interaction between stress and other predispositions may result in the emergence of the disorder (Berge, Loth, Hanson, Croll-Lampert, & Neumark-Sztainer, 2012). Finally, exposure to trauma has been identified as an important risk factor in the development and maintenance of eating disorders (Brewerton, 2015).

Studies have found a significant relationship between sexual abuse and body dissatisfaction, desire for thinness and dieting and purging behaviors (Preti, Incani, Camboni, Petretto, & Masala, 2006; Wonderlich et al., 2000). Research and clinical data suggest that trauma must be addressed and resolved in order for the full remission of eating disorder symptoms (Brewerton 2004; 2015).

The Current Study

This dissertation examines multiple aspects of adolescent eating disorder treatment and recovery. The project seeks to add to the existing literature on adolescent eating disorder treatment and recovery by achieving the following three objectives. First, this project aims to investigate the significance of trauma exposure in a clinical sample of adolescents and young adults receiving behavioral health services for eating disorders. It seeks to broaden the existing literature on the significance of trauma and eating disorders by examining multiple types of trauma beyond childhood sexual abuse and further exploring the relationship between trauma and AN. Second, this project examines the prognostic factors that predict recovery over time for patients struggling with an eating disorder, with particular emphasis on patients with AN. It broadens the literature by using an integrative approach to simultaneously examine the influence of biological,
psychological, and social risk factors. Third, the project investigates the role of family in the treatment seeking process by examining patient and caregiver’s understandings of comorbid depression and anxiety symptoms in their initial efforts to obtain treatment. This study is the first to provide data on caregivers’ understanding of adolescent comorbidities and congruence between patient and caregiver perceptions of symptoms in a clinical sample of adolescents seeking eating disorder treatment.

Data for this study was taken from a Midwestern urban pediatric hospital’s behavioral health eating disorder clinic. This clinic is composed of providers from multiple health disciplines, including dieticians, physicians, behavioral health therapists, occupational therapist, psychiatrists, and nurses. Prior to January 2014 the clinic offered one level of care, outpatient. The outpatient program provided, medical, dietary, and therapeutic care on a weekly basis or as needed for adolescents and families. In this program, clinicians primarily used cognitive behavioral therapy and dialectical behavioral therapy to work with clients. As of January 1, 2014, the eating disorder clinic expanded to offer three levels of care, outpatient, intensive outpatient, and partial hospitalization. Adolescents seeking treatment at the clinic undergo a diagnostic assessment provided by a therapist, dietician, and physician to determine the appropriate level of care for the patient. Adolescents who are referred to intensive outpatient or partial hospitalization are also referred to the psychiatrist who conducts an additional assessment and provides a final program recommendation.

Across all three levels of care clinicians utilize several evidence based treatments including Maudsley family based therapy (Wallis, Alford, Hanson, Titterton, Madden, & Kohn, 2012), cognitive behavioral therapy (Beck, 2011), and dialectical behavioral
therapy (Linehan & Chen, 2005). Patients receiving outpatient services meet with team members once a week or as needed. Patients participating in the intensive outpatient program (IOP) meet with team members 4 days a week, for an approximate total of 12 hours of nutritional counseling and individual, family, and group therapy. The IOP program last for approximately 6-8 weeks. Patients participating in the partial hospitalization program patients meet six days a week for approximately 50 hours of treatment. Services include group, individual and family therapy sessions, nutritional counseling, occupational therapy and massage therapy. Nursing care, medical stabilization and psychiatric treatment are also provided. This program last for approximately 4-6 weeks.

**Structure of the Dissertation**

The current dissertation project is comprised of three independent studies focused on eating disorders and disordered eating behaviors among adolescents and young adults. Chapter 2 (study 1) uses retrospective cross-sectional administrative data to examine the prevalence of traumatic experiences in a sample of adolescent patients in outpatient treatment for eating disorders at a Midwestern pediatric hospital clinic. This study explores the relationship between eating disorder symptoms and trauma by examining differences in initial clinical presentation between patients with and without trauma history. Chapter 3 (study 2) is longitudinal study that uses panel administrative data to identify predictors of progress over time in a sample adolescent patients seeking eating disorder treatment. This study also further examines the significance of trauma exposure by identifying differences between patients with and without a history of trauma. Chapter 4 (study 3) examines initial severity of eating disorder and other comorbid symptoms in
patients seeking behavioral health services using cross-sectional data. The study also examines the relationship between patient and caregiver reports of the adolescent’s depression and anxiety symptoms in families initially seeking eating disorder treatment. Finally, chapter 5 concludes by integrating my thoughts and reflections on all three studies and implications for future research and practice working with adolescents and young adults struggling with eating disorders and disordered eating behaviors are discussed.

**Data**

Data from two larger research projects conducted at an urban Midwestern pediatric hospital eating disorder clinic are used in this dissertation. Approval was obtained from institutional review boards at the hospital and The Ohio State University. Data for studies 1 and 2 is from a secondary administrative data project that involves the population of patients seen in outpatient care at the behavioral health eating disorder program from 1/1/12 – 12/31/14. The sampling frame consists of all patients ages 12 -22 diagnosed with an eating disorder using DSM IV criteria over the aforementioned time period. Of note, the DSM V criteria was adopted for use by the hospital in October 2015, thus prior criteria were utilized for consistency in diagnosis over the data collection period. Electronic medical records were reviewed to collect demographic and clinical data regarding baseline psychological and physical health assessment, trauma experiences, and follow-up data related to treatment and prognosis for 9 to 32 months after initiating treatment. Data was collected from multiple time points for each subject when available (i.e. baseline (admission to treatment), 3 month follow-up (f/u), 6 month f/u, 12 month f/u, 18 month, f/u, 2 year f/u, 30 month f/u. Key constructs in studies 1 and
were measured using variables operationalized from pre-existing data available in the medical records. Key constructs in this data set include age, race, ethnicity, sex, weight, BMI, family history of mental illness, trauma exposure, duration of treatment, eating disorder diagnoses, comorbid diagnosis, psychiatric medication, suicidality, self-harm ideation/behavior, past mental health treatment experiences, and responses to intervention.

Data for study 3 is from a cross sectional secondary administrative data project that involves the population of patients and caregivers who completed an initial diagnostic assessment to receive eating disorder treatment at a local behavioral health clinic from 07/1/2014 to 3/1/2016. Electronic medical records were reviewed to collect demographic and clinical data regarding baseline psychological and physical health assessment, and adolescent and caregiver reports of initial symptoms. The sampling frame consists of all patients ages 12-22 who completed an initial diagnostic assessment during the aforementioned time period. Key constructs in study 3 were measured using scores from quantitative assessment measures used during the diagnostic assessment. Key variables include eating disorder symptomology, depression, anxiety, and parental self-efficacy. Additional variables include trauma exposure and family history of physical and mental health concerns.
Figure 1.1 A Biopsychosocial Model of Eating Disorder Symptomology

- **Biological Factors**
  - Sex
  - Family Hx Eating Disorder
  - Family Hx Psychiatric Disorders

- **Psychological Factors**
  - Anxiety
  - Depression
  - Obsessive Compulsive Traits
  - PTSD

- **Social Factors**
  - Trauma Exposure
  - Family Stress

- **Eating Disorder Symptoms**
  - Restriction
  - Compensatory Behaviors
  - Body Dissatisfaction
  - % EBW
Chapter 2. The prevalence of trauma in a clinical sample of adolescent patients with eating disorders

Introduction

Eating disorders have the highest mortality rate of all mental health disorders, with AN having the most elevated rates (Arcelus, Mitchell, Wales, & Nielson, 2011). The prevalence rate of anorexia nervosa is 0.3% to 1.20% in the total population and .3% in adolescents ages 12-22 years-old (American Psychiatric Association, 2013). Approximately 74 adolescent females out of 100,000 will experience AN in their lifetime (Lock et al., 2010). Eating disorders are often characterized by impairments in role functioning (i.e. the ability to participate in one’s expected role within a system such as a school, family, occupation, or community), significant somatic symptoms, and the presence of co-morbid mental and medical illnesses. Previous researchers have reported a significant relationship between traumatic experiences and the development of an eating disorder in both adults and adolescents (Brewerton, 2006; 2007; Johnson, Cohen, Kasen, & Brook, 2002). In addition, patients with an eating disorder who have experienced trauma, also are likely to report other psychiatric comorbidities, especially PTSD (Brewerton, 2007; Reyes et al., 2011). However, there are significant limitations in the current literature that influence our understanding of the relationship between trauma and eating disorders. First, the majority of studies examining the relationship between trauma and eating disorders focus primarily on CSA (Smolak & Murnen, 2002; Wonderlich, et al. 2000), whereas less is known about the impact of other forms of child abuse (i.e., maltreatment and neglect) on the prevalence and clinical presentation of an eating disorder.
disorder. Second, past researchers have identified a significant relationship between trauma and BN (Brewerton, 2007; Wonderlich, Brewerton, Jocic, Dansky, & Abbot, 1997), but not the ways in which trauma is related to AN, specifically AN restricting subtype. Finally, the literature tends to focus on adults, not adolescents. Studies are often retrospective studies with adults reporting on past childhood experiences of trauma and how that may be linked to an eating disorder diagnosis. It is important that clinical and scholarly attention is directed toward the history of trauma among those diagnosed with an eating disorder in order to provide the best treatment options for patients and their families.

**Literature Review**

A significant relationship has been found between trauma exposure and eating disorders among child, adolescent, and adult populations (Brewerton, 2015). This relationship is consistent among male and female populations. Trauma exposure has been identified as a unique and non-specific risk factor for eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Trauma is often described as a deeply distressing or disturbing experience. The greek word for trauma, τραύμα, is translated into wound. Therefore, trauma may simply be defined as a deep wound or affliction. Children and adolescents often experience trauma in two ways, acute traumatic events and chronic traumatic situations (Gerrity & Folcarelli, 2008). Acute traumatic events are defined as experiencing personal injury, witnessing serious injury or harm, facing imminent threat. Acute traumatic events are usually short lived and are accompanied by feelings of fear, terror, and hopelessness (Gerrity & Folcarelli, 2008). Chronic traumatic situations are experiences that occur over a prolonged period of time and include physical abuse, sexual
abuse, domestic violence, and war or community violence. Chronic traumatic situations often produce feelings of fear and loss of trust or sense of personal safety (Gerrity & Folcarelli, 2008). Early research in the field of eating disorders, focused on the influence of childhood sexual abuse. Childhood sexual abuse is the most studied form of trauma in relationship to eating disorders. Empirical studies in clinical populations have found that eating disorder patients with a history of CSA report significantly more severe eating disorder symptomatology, significantly greater psychiatric comorbidity (specifically esteem, and more interpersonal problems (Carter, Bewell, Blackmore, & Woodside, 2006). In addition, research has found that patients who report CSA are more likely to engage in purging behaviors (Carter, Bewell, Blackmore, & Woodside, 2006; Waller, Halek, & Crisp, 1993). This finding is consistent with more recent studies that report trauma exposure is more common in patients with bulimia versus those with other eating disorder diagnoses (Brewerton, 2007; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997).

Despite the initial emphasis on CSA, research on trauma and eating disorders has been extended to include other forms of trauma, including but not limited to sexual assault, experience of significant death or loss, peer victimization and bullying, parental divorce, and physical and emotional abuse (Berge, Loth, Hanson, Croll-Lampert, & Neumark-Sztainer, 2012; Copeland, Bulik, Zucker, Wolke, Lereya, & Costello, 2015; Johnson, Cohen, Kasen, & Brook, 2002). This extension has revealed that potentially traumatic experiences may significantly contribute to the development of an eating disorder (Johnson, Cohen, Kasen, & Brook, 2002; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Therefore, it is important to understand how patients with
more severe trauma histories compared to those without significant trauma exposure. Studies examining the relationship between trauma exposure, PTSD, and eating disorders found that patients with higher PTSD symptoms reported more severe eating disorder psychopathology (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012; Backholm, Isomaa, & Birgegård, 2013; Tagay, Schlottbohm, Reyes-Rodríguez, Repic, & Senf, 2014). Research indicates that eating disorder patients with significant PTSD symptoms are more likely to have poorer therapy outcomes, relapse, or drop out of treatment when compared to their non-traumatized counterparts (Fichter & Quadflieg, 2004; Mahon et al., 2001).

Even with our current knowledge of trauma and eating disorders, the relationship between trauma exposure and AN remains understudied. Studies focusing on differences in trauma exposure and PTSD symptoms across eating disorder subtypes suggest that there are no statistically significant differences in overall trauma exposure or type of trauma experienced across subtypes (Backholm, Isomaa, & Birgegård, 2013; Tagay, Schlottbohm, Reyes-Rodríguez, Repic, & Senf, 2014). Reyes-Rodríguez and colleagues (2011) examined the prevalence of PTSD in a sample of adult women with AN. Their findings reveal no significant differences in the number of traumatic events experienced across AN subtypes. For women who met clinical criteria for PTSD, the majority reported the first traumatic event occurring prior to the onset of anorexia. However, the odds of having PTSD were significantly lower for those with restricting subtype compared to those with purging subtype (Reyes-Rodríguez et al., 2011). Similar findings were reported in a smaller study of adolescent female patients with AN, such that adolescents with AN binge-purge subtype reported a higher frequency of sexual, physical
and emotionally traumatic experiences than those with AN restricting subtype and healthy controls (Jaite, Schneider, Hilbert, Pfeiffer, Lehmkuhl, & Salbach-Andrae, 2011). These findings remain consistent with multiple studies that report a significant relationship between trauma exposure and purging behaviors.

Given the prevalence of trauma among patients with eating disorders, it is important to understand how clinical interventions for patients with eating disorders address the issue of trauma. The significant relationship between trauma, PTSD, and purging behaviors among eating disorder patients may encourage clinicians and researchers to focus on trauma predominantly with patients with BN or AN – binge purge subtype. However, the lack of statistically significant differences in trauma exposure across AN subtypes, and all eating disorder subtypes, suggest a need to assess for trauma exposure and provide appropriate clinical interventions for all eating disorder patients, not only those with purging behaviors.

The purpose of the current study is to describe the prevalence of trauma in a clinical sample of adolescents receiving outpatient treatment for an eating disorder (anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified). This study has two main objectives: 1) Examine the overall prevalence of traumatic experiences in a sample of adolescent patients in outpatient treatment for eating disorders at a Midwestern pediatric hospital clinic. 2) Examine similarities and differences in the initial presentation of eating disorder symptoms in treatment for patients who have experienced trauma and those who have not. Based on these objectives I seek to answer the following research questions, 1) What is the overall prevalence of traumatic experiences in a clinical sample of adolescent patients in treatment for an eating disorder?
2) Is there an influence of various traumatic experiences on initial eating disorder (ED) presentation? There are two main hypotheses for this study:

1. There will be a significant association between trauma and eating disorder presentation
   a. Higher trauma prevalence is predictive of more severe ED symptomatology

2. Trauma history will be significantly positively associated with the presence of other comorbid symptoms/problems.

**Methodology**

**Sample**

Data for this study were obtained from administrative secondary data that involves the population of patients seen in outpatient care at a Midwestern urban pediatric hospital’s behavioral health eating disorder clinic from 1/1/2012 to 12/31/2014. The sampling frame consists of all patients ages 12 -22 (the clinic’s treatment population) who received a primary diagnosis of an eating disorder using DSM IV-TR criteria over this period (N=316). Although more recent diagnostic codes are available (i.e. DSM V published in Mary 2013), DSM IV-TR criteria is used in this study because the hospital’s electronic medical record system was not updated until October 2015. Medical records of patients meeting the criteria were reviewed and demographic and clinical data regarding baseline psychological and physical health assessment and trauma experiences were collected.

The initial sample comprised 316 participants. The analytic sample for this study (n=205) includes only those patients who received a primary diagnoses of an eating
disorder and had full data for variables of interest. For this reason, 111 participants were omitted due to missing data on the following variables: BMI n=55, trauma exposure n=39, PTSD symptom n=10, and race n=6. One additional participant was omitted as an outlier as their %EBW was greater than 200%. Table 2.1 provides a comparison of the full, analytic, and omitted samples. The analytic sample varies from the full sample in that it has a slightly higher number of patients with anorexia (47.8% vs 41.5%). Analyses of group differences between the omitted and analytic samples reveal the analytic sample has a statistically higher percent of patients with anorexia nervosa ($X^2(1) = 9.6936, p <.01$). Results also reveal that the analytic sample had a significantly lower average BMI score than omitted patients ($M = 19.1 \ SD = 3.2$; ($t (259) = 3.46$, p <.01).

Variables

Key variables were operationalized using pre-existing data available in the medical records. Variables include age, race, sex, BMI, trauma exposure, disorder diagnoses, and other comorbid symptoms. Operationalization of these variables are described below. Table 2.2 provides descriptive statistics for key study variables.

Age. Age is measured in years with a range of 12-22 ($M= 14.9 \ SD = 1.7$).

Race. Race is measured based on data reported in the medical records. The following racial categories were recorded: White, Asian, Bi-racial/Multi-racial, Black or African American, Latino/Hispanic/ Black, Latino/Hispanic/Unspecified, Latino/Hispanic/White, and Other. In this study, race is categorized as a dichotomous variable, non-Hispanic White and Other. These two categories were selected as the frequency of all other racial categories were too small to identify significant differences.

Sex. Male or Female as assigned at birth and in the medical records.
**BMI.** BMI is measured as reported in the medical record and was computed as weight in kilograms divided by height in meters squared. BMI score had a range of 12.87-33.67 (M= 19.1, SD=3.2)

**BMI Percentile.** BMI percentile is measured as reported in the medical record at the time of the encounter. BMI percentile is based on the Center for Disease Control’s (CDC) age 2-20 growth charts for boys and females based on height and weight and age. The growth charts were developed with data from five national health examination surveys and supplemental data (Kuczmarski et al., 2002). For more information about the 2000 CDC U.S. Growth Charts see (Kuczmarski et al., 2002).

**Percent Expected Body Weight (%EBW).** Percent expected body weight for age, sex, and height was determined by Centers for Disease Control and Prevention growth charts (http://www.cdc.gov/growthcharts/percentile_data_files.htm). To calculate percent expected bodyweight (%EBW) for a given participant based on his or her height, age, and gender, the 50th percentile BMI for exact age and height at presentation on the CDC BMI-for-age percentiles chart was used (Le Grange, Doyle, Swanson, Ludwig, Glunz, & Kreipe, 2012). A BMI at the 50th percentile would be the expected median in a group of normally developing adolescents, or EBW. The formula for calculating %EBW is listed below:

\[
\text{%EBW} = \frac{\text{BMI}}{50\text{th percentile BMI for age and height}} \times 100
\]

The range for %EBW 65.3 – 165.5 (M= 95.3, SD=15.8).

**Trauma Exposure.** Given the nature of acute vs chronic traumatic exposure it is important to understand how trauma is defined in the present study. Trauma exposure was measured by a yes or no question asked about their past and current experience of the
following: childhood physical abuse, childhood sexual abuse, exposure to domestic violence, parents’ divorce, significant bullying, motor vehicle accident, neighborhood violence, war terrorism, medical procedures, national disaster, and significant death/loss. Although some of these experiences can be considered acute events such as motor vehicle accident, divorce, or medical procedures many of these events can also be defined as chronic traumatic situations (i.e., physical/sexual abuse, domestic violence, etc.). It is important to note that for the current study the focus is not on the typology of traumatic event (i.e., acute or chronic) but the presence of exposure to a traumatic event.

**Lifetime Trauma.** A lifetime trauma exposure variable was created based on trauma exposure information. A dummy variable was coded as 1 if a patient reported at least one current or past experience of any of the above events, and 0 if a patient reported no traumatic experiences.

**Multi trauma.** Multi trauma, or exposure to multiple traumatic events, was measured as a patient’s experience of 2 or more past or current traumatic events in their lifetime. This variable was coded as 1 if a patient reported 2 or more past or current traumatic events, and 0 if a patient reported 1 or 0 traumatic experiences.

Specific trauma dummy variables were also created for four trauma types based on participant reporting past and/or current experience of physical abuse, sexual abuse, bullying, or significant death or loss. These specific trauma types were selected as they were the most prevalent types of trauma reported within the sample. Each dichotomous variable was coded as 1 if a participant reported experiencing this type of trauma, and 0 if they did not.
**Diagnosis.** Eating disorder diagnoses was measured by the listed diagnoses in the chart according to DSM IV-TR criteria. Patients were either diagnosed with anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder otherwise not specified (EDNOS). Table 2.2 provides the diagnostic criteria for all three eating disorders in the study.

**Comorbid Symptoms.** The medical record contains a section in the assessment that asks patients to self-report on the presence of significant mental health symptoms and impairment including depressive symptoms, anxiety symptoms, obsessions and compulsions, and post-traumatic stress responses. These symptoms are collected with yes or no questions. Dummy variables for each of the four comorbid symptoms were created and coded as 0 in the absence of symptoms and 1 if symptoms were reported at the time of encounter. It is important to note that these variables are not indicative of meeting the clinical criteria required for an official diagnosis of major depressive disorder, generalized anxiety disorder, post-traumatic stress disorder or obsessive compulsive disorder. Instead these variables indicate the presence of significant symptoms that impair functioning, whether the patient has another comorbid diagnosis is not indicated by these variables.

**Data Analysis**

All analyses were completed using STATA version 14.1. Independent sample t-tests and chi-square tests of independence were used to check for selection bias as not all participants in the population met criteria for study inclusion. In order to test the
hypotheses univariate and bivariate statistics are used. Chi-square and independent sample t tests were conducted to examine associations between variables of interest. Fisher’s exact test was used to examine associations between specific trauma types (physical and sexual abuse, bullying, and death/loss) and key variables, as the expected counts were less than 5 percent within those contingency tables. Finally, binomial logistic and multinomial logistic regression models were used to examine the ability of trauma to predict percent expected body weight. First, three logistic regression models were used to examine the ability of trauma exposure to predict the likelihood of being under the 85th percentile expected body weight. Model 1 included a single predictor variable, lifetime trauma. Model 2 included lifetime trauma and demographic factors (i.e. age, race, sex). Model 3 included Model lifetime trauma, demographic factors, and the presence of comorbid symptoms (i.e., depression, anxiety, or obsessions and compulsions). In model 3, the presence of comorbid symptoms is measured as a dichotomous variable (0 = no comorbid symptoms, 1 = 1 or more comorbid symptoms). Second, multinomial logistic regression was used to predict the probability of being in a particular weight category (low weight - less than 85 %EBW, mid-range - 85-100% EBW; high weight - greater than 100 %EBW) based on lifetime trauma exposure.

Results

Sample Characteristics

Table 2.1 displays descriptive statistics for the analytic sample. The sample is predominantly white (88%) and female (92%). Of the sample, approximately 48% received a diagnosis of anorexia nervosa, 8% bulimia nervosa, and 44% ED NOS. Over, 75% and 73% of the sample reported depressive and anxiety symptoms, respectively.
Reports of post-traumatic stress symptoms and obsessions/compulsions/hoarding were smaller at 30% and 33%, respectively.

**Prevalence of Trauma**

One third of the sample reported one or more traumatic events during their lifetime (36.1%; n=74). Table 2.3 provides a comparison of key variables based on trauma exposure. Chi-square tests of independence were performed to examine the relation between trauma exposure and categorical key variables. Results reveal a significant relationship between trauma exposure and bulimia nervosa, \( (X^2 (1) = 6.58 \ p < .01) \), where adolescents with bulimia nervosa were more likely to report trauma exposure than those with other diagnoses. A significant relationship between trauma and PTSD symptoms \( (X^2 (1) = 103.5, \ p < .01) \) was also found, such that patients reporting these symptoms were more likely to report trauma exposure. A significant relationship between trauma and depressive \( (X^2 (1) = 4.2, \ p < .05) \) and anxiety symptoms \( (X^2 (1) = 12.7 \ p < .01) \) were also found. Independent sample t-tests revealed a significant relationship between trauma exposure and BMI percentile, BMI and %EBW. Adolescents with trauma exposure had higher BMI \( (M = 20.1 \ SD = 3.8; \ t (203) = -3.41, \ p = .001) \), BMI percentile \( (M=41.8 \ SD =30.8; \ t (203) = -2.3, \ p=.021.) \), and % EBW \( (M =99.6 \ SD= 18.6; \ t(203) = -3.04, \ p = .003) \) than those without any trauma exposure.

**Specific trauma exposure**

Of all the types of trauma reported, the four most prevalent trauma types were selected as key variables of interest. Table 2.4 provides descriptive statistics for these trauma types. Childhood physical abuse, sexual abuse, bullying, and significant death/loss accounted for approximately 84% of the trauma reported in the sample.
Bullying was the most prevalent trauma type with 11% of the sample reporting significant history of bullying. Significant death or loss and sexual abused tied at 9% for the second most reported trauma type. Physical abuse was the smallest reported type of trauma with a prevalence rate of 6%.

Exposure to multiple traumatic events or multi trauma was also observed in the sample. Patients reporting 2 or more exposures to particular forms of trauma (bullying, physical abuse, sexual abuse, and death/loss) were identified as having multi trauma. The prevalence of multi trauma in the sample was approximately 6%. Table 2.5 provides bivariate analyses of multi trauma and the four specific types of trauma with key variables. Results from fisher’s exact tests reveal significant relationships between sexual abuse and bulimia nervosa (p = .007), depressive symptoms (p = .008), and PTSD symptoms (p = .000). Race was significantly associated with physical abuse (p = .027) and multi trauma (p = .027). T test results revealed a significant association between sexual abuse and BMI (M = 20.9 SD = 3.8; t (203) = -2.54, p = .012). Patients who experienced physical abuse reported a significantly higher BMI (M = 20.9 SD = 3.9; t (203) = -2.13 p = .034) and %EBW (M = 104.2 SD = 19.6; t(203) = -2.11, p = .036) than their counterparts. A significant relationship between bullying and BMI and %EBW were found. Adolescents who reported bullying had a higher BMI (M = 21.8 SD = 7.6; t (214) = -3.35, p = .001) and %EBW (M = 109.5 SD = 34.7; t (214) = -3.72, p = .0003 than those without exposure to bullying. T test results reveal a significant relationship between multi trauma and BMI percentile, BMI, and %EBW. Adolescents with multi trauma had a higher BMI percentile (M = 51.6 SD = 32.3; t (203) = -2.072, p = .040), BMI (M = 21.0 SD = 4.3; t (203) = -2.273, p = .024), and %EBW (M = 105.7, SD = 19.9; t (203) = -2.473, p = .014) than those without
multi trauma. A significant relationship was found between all five types of trauma respectively (physical and sexual abuse, bullying, death/loss, and multi trauma) and PTSD symptoms (Fisher’s exact test \( p = .000 \)).

**Predicting \% EBW**

Three logistic regression models were conducted to predict the likelihood of being significantly underweight (\%EBW less than 85\%). Table 2.6 presents the results from the three models. Model 1 included lifetime trauma as the sole predictor of being significantly underweight. A test of the full model against a constant only model was statistically significant (\( \chi^2 = 3.83, \text{df} = 1, p < 0.05 \)). Lifetime trauma was marginally significant in predicting weight status (\( p = .057 \)). When demographic variables were added to the model (Model 2), both lifetime trauma exposure and age were statistically significant predictors of weight status. For those with trauma the odds of being underweight are .44 times smaller than those with no trauma reported (\( B = -.82 (\text{.37}); p=.026 \)). In addition, a one-unit increase in age significantly increased the odds of being underweight by 1.2 times (\( B = .20 (\text{.09}); p=.034 \)). Model 3 added the presence of comorbid symptoms to model 2. Results from Model 3 indicate that lifetime trauma and age continue to remain significant predictors of being underweight. Again, for those with significant trauma history the odds of being underweight were .47 times less likely than their counterparts without trauma history (\( B = -.75 (\text{.38}); p=.047 \)). Age significantly increased the odds of being underweight by 1.2 (\( B = -20 (\text{.09}); p=.035 \)).

Table 2.7 present the results from three multinomial logistic regression models predicting percent expected body weight. Two comparisons are presented in Table 2.7: being low weight compared to mid-range, and being high weight compared to mid-range.
Parameter estimates and relative risk ratios are included in Table 2.7. Model 1 in Table 2.7 includes lifetime trauma as the sole predictor of %EBW category. Results from model 1 reveal that lifetime trauma is not a statistically significant predictor of being either low or high weight compared to being in the mid-range category. Given the small sample size, it is important to note that lifetime trauma reached p=.067 in predicting being in the high weight category when compared to mid-range. This marginal finding is consistent with results from logistic regression model 1 that found trauma’s ability to predict %EBW alone was marginally significant (p=.057). Model 2 adds demographic factors to the multinomial logistic regression estimates of percent expected body weight. Results reveal that lifetime trauma is not a statistically significant predictor of %EBW. However, race was found to be significant. Specifically, for whites compared to non-whites, the relative risk for high weight relative to mid-range weight would be expected to decrease by a factor of .177 given the other variables in the model are held constant ($B = -1.73 (.61) p=.004$). The relationship between race and %EBW remained marginally significant when low weight was compared to mid-range holding all other variables constant ($B = -1.26 (.67) RRR = .284; p=.060$). Finally, model 3 adds the presence of comorbid symptoms to the model 2. The effects of race are similar to model 2, controlling for comorbid symptoms. For whites compared to non-whites, the relative risk for high weight relative to mid-range weight would be expected to decrease by a factor of .174 given the other variables in the model are held constant ($B = -1.75 (.61) p=.004$). Again, this finding was marginally significant low weight was compared to mid-range (p=.058). Age was a marginally significant predictor of being low weight vs mid-range in Model 3. Given a one-unit increase in age, the relative risk of being in low weight vs
mid-range weight increased by a factor of 1.21 (p=.058). Lifetime trauma was not a significant predictor of %EBW in Model 3.

**Discussion**

The aim of this study was to examine the overall prevalence and type of trauma exposure in a clinical sample of adolescent eating disorder patients. Over one-third of the sample reported at least one traumatic event. This finding is comparable, albeit slightly higher, to the prevalence of trauma (29.1%) in a national sample of adolescents from the National Comorbidity Survey Replication Adolescent Supplement (NCS-A), a national survey of adolescents aged 13–17 (McLaughlin et al., 2013). Only a small percent of the sample reported exposure to two or more trauma types. The most common form of trauma was bullying. Experiencing a significant death or loss and sexual abuse were the second most common forms of trauma reported. This finding is consistent with results from the NCS-A study that found unexpected death of a love one as the most prevalent traumatic experience among adolescents (McLauglin et al., 2013). Interpersonal trauma (i.e. physical and sexual abuse, bullying, and death/loss) made up the majority of reported traumatic events in the sample. A significant relationship was found between lifetime trauma exposure and bulimia nervosa. Adolescents with bulimia nervosa were more likely to report trauma exposure than those with anorexia nervosa or eating disorder not otherwise specified. This finding is also consistent with previous research that suggests a link between trauma and bulimia nervosa or other eating disorders subtypes that involve purging behaviors (i.e. AN=binge purge type and binge eating disorder) (Brewerton, 2007). In addition, a significant relationship was found between trauma exposure and BMI, BMI percentile, and %EBW, such that patients who experienced at least one
traumatic event were more likely to have a higher BMI and BMI percentile and be closer to their expected body weight. These findings are not surprising given the fact that patients with bulimia nervosa are often at a higher weight than patients with anorexia nervosa due to diagnostic criteria alone.

When assessing for the impact of specific trauma types, results are consistent with existing research that links childhood sexual abuse and bulimia nervosa (Brewerton, 2007; Johnson, Cohen, Kasen, & Brook, 2002; Wonderlich et al., 2000). Sexual abuse was significantly associated with depressive and PTSD symptoms reinforcing the fact that childhood sexual abuse is a non-specific risk factor for eating disorders and other psychiatric comorbidities (Brewerton, 2007). Significant associations were found between bullying, BMI score, %EBW, and PTSD symptoms. These findings may be partly explained by the fact that children and adolescents who are overweight/obese are more likely to be bullied, regardless of other social or academic factors, than their peers (Lumeng, Forrest, Appugliese, Kaciroti, Corwyn, & Bradley, 2010). Furthermore, current research suggests there may be a significant association between bullying and PTSD (Idsoe, Dyregrov, & Idsoe, 2012; Nielsen, Tangen, Idsoe, Matthiesen, & Magerøy, 2015). However, due to the limitations of cross-sectional data we are unable to ascertain whether bullying victimization occurred before or after increased weight status or engaging in overeating, binging, or purging behaviors.

Patients who experienced multi trauma, reporting two or more traumatic experiences, had higher BMI, BMI percentile, and %EBW. Both physical abuse and multi trauma were significantly associated with race with a higher percentage of non-white patients reporting these experiences when compared to other forms of trauma.
measured in the study. Research indicates that racial and ethnic minorities have high rates of exposure to childhood adversities and this differential exposure may be helpful in explaining racial differences in mental health outcomes (Finkelhor, Ormrod, & Turner, 2007). Reporting any of the five trauma types assessed in this study was significantly associated with PTSD symptoms. This finding confirms the importance of assessing for trauma in patients with eating disorders. Research suggests that trauma history in eating disorder patients is associated with greater psychiatric comorbidity including PTSD (Wonderlich et al., 1997). In addition, the presence of partial or sub clinical threshold PTSD symptoms has also been associated with bulimia nervosa (Brewerton, 2007) and anorexia nervosa (Reyes-Rodríguez et al., 2011).

Results from logistic regression models reveal that lifetime trauma is a significant predictor of being underweight (less than 85% EBW). Controlling for demographic factors and comorbid symptoms, lifetime trauma significantly reduces the odds of being underweight. Patients who reported trauma were almost .5 times less likely to be underweight. This finding is consistent with bivariate results that found a significant relationship between trauma history and higher weight status (BMI, BMI percentile, and %EBW) and bulimia nervosa. Multinomial regression results revealed that lifetime trauma alone was a marginally significant predictor of high weight status compared to mid-range. However, this did not hold when demographic factors and comorbid symptoms were added the model. These findings suggest that although trauma may be important in predicting low vs high weight status, it is limited in its ability to predict more specific weight categories, particularly when additional demographic and clinical factors are taken into account. Overall, results from this study replicate previous research
regarding the relationship between trauma and eating disorder symptoms and the association between trauma and co-morbid mental health conditions. Replication research is an important aspect of the scientific process. However oftentimes this part of the scientific method is lacking (Open Science Collaboration, 2015).

The limitations of the present study warrant consideration. First, due to the use of administrative data the sample was limited only to patients who had full data on variables of interest. This resulted in a loss of subjects and decreased the sample which may have limited the ability to find significant multivariate results as evidenced by the marginally significant findings reported in this study. Second, lack of specificity in diagnoses subtypes and specifiers prevented an examination of the relationship between trauma and particular diagnoses such as anorexia-binge purge type, and atypical anorexia. Although comorbidity was examined in the study, it was limited to examining the relationship between trauma and symptoms only due to the lack of formal diagnoses of depression, anxiety, post-traumatic stress disorder, and obsessive compulsive disorder. Third, because of the nature of these data, the study was limited to one physical measure of eating disorder symptomology, weight status (BMI, BMI percentile, and %EBW). Although weight is an important physiological marker in eating disorder treatment and research it is not the only marker and future studies would benefit from measuring both physical and psychological factors. Fourth, although information on trauma exposure was available in the data set, the use of secondary administrative data does limit the reliability of this information. Because this data was not initially collected for research purposes, issues around selective reporting and failure of clinicians to ask the same questions of each patient should be noted. The data used in this study reported the presence of any
current or past trauma and specific forms of trauma exposure. Inconsistencies across these reports were found when analyzing the data. I did correct these inconsistencies by matching reviewing reports of trauma subtypes and ensuring that these cross-matched with reports of lifetime trauma exposure. However, the presence of these inconsistencies alone suggest that other inconsistencies may be present. Finally, the use of cross-sectional data does not allow for an examination of the impact of timing on the relationship between variables of interest. The ambiguity surrounding timing of a traumatic event or onset of symptoms does not allow for a discussion of causality or modeling of the pathway between associations.

**Conclusions and Clinical Implications**

In summary, the lifetime trauma prevalence in this sample of adolescent eating disorder patients is 36.1%. The prevalence of trauma in this clinical sample of adolescents speaks to the importance of assessment and treatment when working with adolescent eating disorder patients, especially those with comorbid trauma history and PTSD symptoms. Trauma history is significantly linked to PTSD symptoms. Research has consistently shown that trauma history in eating disorder patients is associated with significant psychiatric comorbidity (Brewerton, 2007 Wonderlich et al., 1997). The complex and pervasive nature of eating disorders often results in a long and tedious recovery process for children and adolescents. The presence of psychiatric comorbidities often lengthens this process. As a result, it is important that an initial and thorough trauma assessment is completed as well as a comprehensive assessment for other psychiatric comorbidities. I argue that treatment for eating disorder patients with significant trauma
history and PTSD should look different from treatment of patients without significant comorbid symptoms. Many eating disorder treatment centers have begun to provide subsequent trauma treatment for eating disorder patients when necessary or created separate programs for those patients struggling with full or sub-threshold PTSD. The early detection of trauma exposure, PTSD symptoms, and other psychiatric comorbidities would allow healthcare professionals to tailor a more holistic treatment approach for clients that may be at increased risk for relapse given treatment as usual (Fichter & Quadflieg, 2004; Mahon et al., 2001).
Table 2.1. Comparison of Full Sample vs Analytic Sample on Key Variables

<table>
<thead>
<tr>
<th>Sample</th>
<th>Full (N=316)</th>
<th>Analytic (n=205)</th>
<th>Omitted (n=111)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>15.0 (1.8)</td>
<td>14.9 (1.7)</td>
<td>15.2 (2.0)</td>
<td>.147</td>
<td>t-test</td>
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<tr>
<td>% White</td>
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<td>89.2</td>
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<td>chi square</td>
</tr>
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<td>47.8</td>
<td>29.7</td>
<td>.002**</td>
<td>chi square</td>
</tr>
<tr>
<td>Bulimia Nervosa (%)</td>
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<td>8.3</td>
<td>6.3</td>
<td>.525</td>
<td>chi square</td>
</tr>
<tr>
<td>ED NOS (%)</td>
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<td>43.9</td>
<td>46</td>
<td>.727</td>
<td>chi square</td>
</tr>
<tr>
<td>BMI percentile, mean(SD)</td>
<td>37.3 (29.6)</td>
<td>35.7 (28.9)</td>
<td>41.8 (2.0)</td>
<td>.121</td>
<td>t-test</td>
</tr>
<tr>
<td>BMI, mean(SD)</td>
<td>19.5 (4.0)</td>
<td>19.1 (3.2)</td>
<td>21.1 (5.9)</td>
<td>.001**</td>
<td>t-test</td>
</tr>
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<td>%EBW</td>
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<td>95.6 (16.1)</td>
<td>n/a</td>
<td></td>
<td>t-test</td>
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<td>Depressive symptoms (%)</td>
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<td>75.6</td>
<td>80.3</td>
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<td>Anxiety symptoms (%)</td>
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<td>75.4</td>
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<td>PTSD Symptoms (%)</td>
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<td>30.9</td>
<td>.868</td>
<td>chi square</td>
</tr>
<tr>
<td>Obsessions/Compulsions (%)</td>
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<td>33.2</td>
<td>25.4</td>
<td>.259</td>
<td>chi square</td>
</tr>
<tr>
<td>Trauma Exposure (%)</td>
<td>36.5</td>
<td>36.1</td>
<td>37.7</td>
<td>.819</td>
<td>chi square</td>
</tr>
</tbody>
</table>

*p≤.05 **p≤.01

1 This p value is a result of the bivariate comparison of the analytic sample (n=205) and the omitted subjects (n=111).
Table 2.2 DSM IV TR Diagnostic Criteria for Eating Disorders

<table>
<thead>
<tr>
<th>DSM IV TR Diagnostic Criteria for Eating Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anorexia Nervosa (307.1)</strong></td>
</tr>
<tr>
<td>1. Body weight that is significantly lower than what is considered normal given their age and height</td>
</tr>
<tr>
<td>2. Intense fear of gaining weight</td>
</tr>
<tr>
<td>3. Disturbance in the way one experiences their body weight or shape</td>
</tr>
<tr>
<td>4. Absence of three consecutive menstrual cycles in postmenarcheal females (amenorrhea)</td>
</tr>
</tbody>
</table>

| **Bulimia Nervosa (307.51)**                   |
| 1. Recurrent episodes of binge eating          |
| 2. Recurrent inappropriate compensatory behavior in order to prevent weight gain (i.e. vomiting misuse of laxatives, fasting, or excessive exercise) |
| 3. Bingeing and compensatory behaviors occur at least twice a week for 3 months |
| 4. Self-evaluation is unduly influenced by body shape and weight |
| 5. Disturbance does not occur exclusively during episodes of anorexia nervosa |

<table>
<thead>
<tr>
<th><strong>Eating Disorder Not Otherwise Specified (307.50)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>ED NOS is for disorders of eating that do not meet the criteria for any specific eating disorder. Examples include:</td>
</tr>
<tr>
<td>1. All of criteria for AN met except amenorrhea in females</td>
</tr>
<tr>
<td>2. All of criteria for AN are met, except that despite significant weight loss, individual’s current weight is in the normal range</td>
</tr>
<tr>
<td>3. All of criteria for BN are met except binge eating and compensatory behaviors occur less than twice a week or for a duration of less than 3 months</td>
</tr>
<tr>
<td>4. The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food</td>
</tr>
<tr>
<td>5. Repeatedly chewing and spitting out but not swallowing large amounts of food</td>
</tr>
<tr>
<td>6. Binge eating disorder: recurrent episodes of binge eating without regular use of compensatory behaviors</td>
</tr>
</tbody>
</table>

---

2 American Psychiatric Association (2013)
Table 2.3 Comparison of Characteristics based on Trauma Exposure on Key Variables

<table>
<thead>
<tr>
<th>Sample</th>
<th>None (n=131)</th>
<th>Any Trauma (n=74)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y, mean (SD)</strong></td>
<td>14.8(1.8)</td>
<td>15.1(1.6)</td>
<td>.319</td>
<td>t-test</td>
</tr>
<tr>
<td><strong>% White</strong></td>
<td>65.8</td>
<td>34.2</td>
<td>.131</td>
<td>chi square</td>
</tr>
<tr>
<td><strong>% Female</strong></td>
<td>65.1</td>
<td>34.9</td>
<td>.228</td>
<td>chi square</td>
</tr>
<tr>
<td>Anorexia Nervosa (%)</td>
<td>68.4</td>
<td>31.6</td>
<td>.203</td>
<td>chi square</td>
</tr>
<tr>
<td>Bulimia Nervosa (%)</td>
<td>35.3</td>
<td>64.7</td>
<td>.010**</td>
<td>chi square</td>
</tr>
<tr>
<td>ED NOS (%)</td>
<td>64.4</td>
<td>35.6</td>
<td>.886</td>
<td>chi square</td>
</tr>
<tr>
<td>BMI percentile, mean(SD)</td>
<td>32.2(27.3)</td>
<td>41.8 (30.8)</td>
<td>.021*</td>
<td>t-test</td>
</tr>
<tr>
<td>BMI, mean (SD)</td>
<td>18.5 (2.6)</td>
<td>20.1 (3.8)</td>
<td>.001**</td>
<td>t-test</td>
</tr>
<tr>
<td>%EBW mean (SD)</td>
<td>92.8 (13.4)</td>
<td>99.7 (18.6)</td>
<td>.003**</td>
<td>t-test</td>
</tr>
<tr>
<td>Depressive symptoms (%)</td>
<td>60</td>
<td>40</td>
<td>.041*</td>
<td>chi square</td>
</tr>
<tr>
<td>Anxiety symptoms (%)</td>
<td>56.7</td>
<td>43.3</td>
<td>.000**</td>
<td>chi square</td>
</tr>
<tr>
<td>PTSD Symptoms (%)</td>
<td>11.5</td>
<td>88.5</td>
<td>.000**</td>
<td>chi square</td>
</tr>
<tr>
<td>Obsessions/Compulsions (%)</td>
<td>61.8</td>
<td>38.2</td>
<td>.653</td>
<td>chi square</td>
</tr>
</tbody>
</table>

*p < .05 **p < .01
Table 2.4. Selected Types of Trauma Exposure

<table>
<thead>
<tr>
<th>Type</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifetime Trauma</td>
<td>74</td>
<td>36.1</td>
</tr>
<tr>
<td>Physical Abuse</td>
<td>13</td>
<td>6.3</td>
</tr>
<tr>
<td>Sexual Abuse</td>
<td>18</td>
<td>8.8</td>
</tr>
<tr>
<td>Bullying</td>
<td>22</td>
<td>10.7</td>
</tr>
<tr>
<td>Death/Loss</td>
<td>18</td>
<td>8.8</td>
</tr>
<tr>
<td>Multi trauma</td>
<td>13</td>
<td>6.3</td>
</tr>
</tbody>
</table>
Table 2.5 Comparison of Characteristics based on Specific Trauma Types on Key Variables

<table>
<thead>
<tr>
<th>Trauma Type</th>
<th>Physical Abuse (n=13)</th>
<th>Sexual Abuse (n=18)</th>
<th>Bullying (n=22)</th>
<th>Death/Loss (n=18)</th>
<th>Multi (n=13)</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>15.1 (1.8)</td>
<td>15.7 (1.91)</td>
<td>14.4 (1.4)</td>
<td>14.7 (1.6)</td>
<td>14.5 (1.4)</td>
<td>t-test</td>
</tr>
<tr>
<td>% White</td>
<td>69.2*</td>
<td>83.3</td>
<td>81.8</td>
<td>88.9</td>
<td>69.2*</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>% Female</td>
<td>92.3</td>
<td>88.9</td>
<td>95.5</td>
<td>88.9</td>
<td>84.6</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>Anorexia Nervosa (%)</td>
<td>53.9</td>
<td>33.3</td>
<td>40.9</td>
<td>33.3</td>
<td>38.5</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>Bulimia Nervosa (%)</td>
<td>15.4</td>
<td>27.8**</td>
<td>18.2</td>
<td>11.1</td>
<td>23.1</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>ED NOS (%)</td>
<td>30.8</td>
<td>38.9</td>
<td>40.9</td>
<td>55.6</td>
<td>38.5</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>BMI percentile, mean (SD)</td>
<td>48.2 (31.7)</td>
<td>45.0 (31.6)</td>
<td>46.3 (29.7)</td>
<td>36.1 (32.4)</td>
<td>51.6 (32.3)*</td>
<td>t-test</td>
</tr>
<tr>
<td>BMI, mean (SD)</td>
<td>20.9 (3.9)*</td>
<td>20.9 (3.8)**</td>
<td>20.5 (4.6)*</td>
<td>19.1 (3.5)</td>
<td>21.0 (4.3)*</td>
<td>t-test</td>
</tr>
<tr>
<td>%EBW mean (SD)</td>
<td>104.1 (19.6)*</td>
<td>101.9 (18.3)</td>
<td>103.8</td>
<td>95.7 (16.17)</td>
<td>105.7 (19.9)**</td>
<td>t-test</td>
</tr>
<tr>
<td>Depressive symptoms (%)</td>
<td>92.3</td>
<td>100.0**</td>
<td>(21.9)**</td>
<td>77.8</td>
<td>92.3</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>Anxiety symptoms (%)</td>
<td>84.6</td>
<td>88.9</td>
<td>90.9</td>
<td>88.9</td>
<td>100</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>PTSD Symptoms (%)</td>
<td>100**</td>
<td>88.9**</td>
<td>81.8</td>
<td>66.7**</td>
<td>84.6**</td>
<td>fisher’s exact</td>
</tr>
<tr>
<td>Obsessions/Compulsions (%)</td>
<td>38.5</td>
<td>16.7</td>
<td>63.6**</td>
<td>22.2</td>
<td>23.1</td>
<td>fisher’s exact</td>
</tr>
</tbody>
</table>

* p ≤.05 **p ≤.01
Table 2.6 Logistic regression analysis for predicting % expected body weight (n=205)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Model 1</th>
<th></th>
<th>Model 2</th>
<th></th>
<th>Model 3</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifetime Trauma</td>
<td>( B \ (SE) )</td>
<td>OR</td>
<td>( B \ (SE) )</td>
<td>OR</td>
<td>( B \ (SE) )</td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>-.67 + (.35)</td>
<td>.51</td>
<td>-.82* (.37)</td>
<td>.44</td>
<td>-.75* (.38)</td>
<td>.47</td>
</tr>
</tbody>
</table>

Model X\(^2\) = 3.83*  
Pseudo R\(^2\) = .0161

Note: The dependent variable in this analysis is %EBW coded so that 0 = greater than or equal to 85% expected body weight and 1 = less than 85% EBW.

Model 1 includes lifetime trauma only; Model 2 = Model + demographic variables (age, sex, race); Model 3 = Model 2 + comorbid symptoms (comorbid symptoms code as 0 = no symptoms 1 = reports present of at least one set of anxiety, depressive or OCD symptoms).

*\( p \leq .05 \)  \quad *\( p \leq .07 \)
### Table 2.7. Multinomial logistic regression for predicting % expected body weight (n=205)

**Comparison category: Mid=range (85-100% EBW)**

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low weight</td>
<td>High weight</td>
<td>Low weight</td>
</tr>
<tr>
<td><strong>B (SE)</strong></td>
<td><strong>RRR</strong></td>
<td><strong>B (SE)</strong></td>
<td><strong>RRR</strong></td>
</tr>
<tr>
<td>Lifetime trauma</td>
<td>-.53 (.36)</td>
<td>.59 (.41)</td>
<td>1.97</td>
</tr>
<tr>
<td>Model X²</td>
<td>6.56</td>
<td>p=.038</td>
<td>16.81</td>
</tr>
<tr>
<td>Pseudo R²</td>
<td>.0169</td>
<td>.0433</td>
<td>.0450</td>
</tr>
</tbody>
</table>

Note: The dependent variable in this analysis is %EBW coded so that 0 = less than 85% EBW, 1 = 85-100% EBW, and 2 = greater than 100% EBW.

Model 1 includes lifetime trauma only; Model 2 = Model + demographic variables (age, sex, race); Model 3 = Model 2 + comorbid symptoms (comorbid symptoms code as 0 = no symptoms 1= reports present of at least one set of anxiety, depressive or OCD symptoms).

+ p ≤ .07
Chapter 3. Identifying predictors of recovery among adolescent patients with anorexia nervosa

Introduction

Anorexia nervosa (AN) often presents during adolescence and can continue into adulthood. It is a serious, and at times, life threatening eating disorder that is characterized by significant weight loss and food restriction (American Psychological Association, 2013). The etiology of AN remains unknown despite a vast amount of clinical, epidemiological, and genetic research on the subject (le Grange, 2016). Over the years, different biological, psychological, and social models have often been cited as providing a better understanding of the risk factors associated with AN. Researchers agree that the cause of anorexia nervosa appears to be multifactorial in nature (Lock & le Grange, 2005). Recent scholars argue that an integrated biopsychosocial approach (Engel, 1980; le Grange, 2016) may help to uncover the etiologic factors that result in the disorder (le Grange, 2016; Rogers, Paxton, & McLean, 2014). An integrated biopsychosocial approach would also add to our understanding of the course and recovery from AN. Multiple biological, psychological, and social risk factors are known to be associated with the development of anorexia (Lucas, 1981; le Grange, 2016;). However, less is known about the way in which these factors are influential throughout the treatment process and into recovery. Identifying factors associated with the course and recovery could have a number of benefits for patients, families, and clinicians.
Patients and families could be provided a more thorough understanding of their illness which may allow the family system to organize in such a way that it enhances their protective factors and mitigates the presence of risk factors. In addition, clinicians would be better able to focus on important factors and individualized care given the clinical context of the patient. This would give patients and families a clearer understanding of what to expect in treatment and a different perspective to help understand successes, setbacks, and effective relapse prevention.

**Literature Review**

Anorexia is often accompanied by high treatment dropout rates in adolescent and adult populations (Fassino, Pierò, Tomba, & Abbate-Daga, 2009; DeJong, Broadbent, & Schmidt, 2012). Patients who remain in treatment are often involved in a lengthy treatment process ranging from 12 months to 3 years or more. For this reason, it is important to understand the factors that promote success or failure in recovery from this illness. Weight restoration is often used as a measure of success or recovery in treatment. Studies have shown that early weight gain is a significant predictor of remission at end of treatment (EOT) (Le Grange, Accurso, Lock, Agras, & Bryson, 2014). In addition, weight gain, specifically early weight gain, was found to be significantly associated with greater improvement in psychological outcomes in adolescent populations (Accurso, Ciao, Fitzsimmons-Craft, Lock, & Le Grange, 2014). A systematic review of dropout from treatment of outpatients with anorexia suggest that illness severity and type of treatment offered may significantly contribute to dropout rates. A negative correlation was found between BMI at 1 year and dropout rates such that those with a lower BMI at 1 year of treatment were more likely to drop out of treatment (DeJong, Broadbent, &
Schmidt, 2012). These findings suggest that early weight gain and restoration is an important factor in recovery from anorexia nervosa and some argue that it is essential in predicting long term outcomes in adolescent patients (Le Grange, Accurso, Lock, Agras, & Bryson, 2014).

Various studies have examined prognostic factors that may predict recovery from anorexia nervosa. One study found that adolescents and adults with higher levels of anxiety and the lifetime presence of vomiting were less likely to recover (Zerwas et al. 2013). Impulsivity at onset was also found to be positively associated with recovery (Zerwas et al. 2013). An examination of multiple randomized clinical trials found that the best predictor of recovery in adolescents with anorexia nervosa was achieving a body weight that is approximately 95% of their expected body weight (EBW) (Lock, Agras, Le Grange, Couterier, Safer, & Bryson, 2013). This finding is consistent with previous research that suggests attaining greater than 90% of one’s EBW by EOT is an important indicator for recovery (Courtier & Lock, 2006). Other factors such as very low initial body weight, longer duration of illness, older age at onset, greater intensity and frequency of symptoms, comorbidity with other psychiatric disorders, lower socioeconomic status, and disturbed family relationships have also been cited as significant predictors of sustained recovery (Howard, Evans, Quintero-Howard, Bowers, & Andersen, 1999; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Striegel-Moore & Bulik, 2007).

Despite much speculation over the years about the etiology of anorexia nervosa, it is general consensus that the cause of anorexia nervosa is unknown (Lock & Le Grange, 2012). Although there is no determined cause, research suggests that temperament, environment, and genetic factors appear to play a role in the overall development of
anorexia nervosa (American Psychological Association, 2013). The biopsychosocial model (Engel, 1977; 1980) provides a way to simultaneously examine multiple prognostic factors and their influence on weight restoration in patients with anorexia nervosa. Figure 3.1 provides the biopsychosocial model of anorexia maintenance and recovery used in this chapter. This model suggests that biological factors (sex, family history), psychological factors (comorbid psychiatric diagnoses), and social factors (trauma exposure) can independently and collectively influence the likelihood of early weight restoration in treatment for anorexia nervosa. The following section outlines the current knowledge about the biopsychosocial factors used in this study.

**Biological Factors**

Gender, family history of anxiety and eating disorders, and brain chemistry are all significant risk factors for developing anorexia (Lock & Le Grange, 2012). Females are ten times more likely to develop anorexia than males (American Psychiatric Association, 2013). In addition, the onset of anorexia often begins during adolescence which is a time of great biological change (i.e. puberty). This rapid change may be one reason for the emergence of eating disorder pathology during adolescence (Kaye, 2008). AN has been found to be particularly heritable, having first degree relative with AN increases one’s risk of developing the disorder by over 50% (Berrettini, 1998). Similarly, anxiety has also been found to be heritable and associated with the development of anorexia nervosa (Strober, Freeman, Lampert, & Diamond, 2007).

**Psychological Factors**

The presence of certain psychiatric disorders has been identified as a risk factor for the development of eating disorders. Research has found that individuals who develop
an eating disorder often had a preexisting or primary anxiety disorder (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Strober, Freeman, Lampert, & Diamond, 2007). In addition, individuals with obsessive compulsive patterns have been found to have a higher risk for developing anorexia (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Cederlöf et al., 2015). Females with eating disorders have significantly higher reports of depression than community controls (Godart et al., 2007). The presence of partial or subthreshold PTSD has been cited as a potential risk factor for bulimia nervosa and purging symptoms (Brewerton, 2007; Mitchell et al., 2012).

**Social Factors**

Exposure to trauma has been identified as an important risk factor in the development and maintenance of eating disorders (Brewerton, 2015). Studies have found a significant relationship between sexual abuse and body dissatisfaction, desire for thinness and dieting and purging behaviors (Preti, Incani, Camboni, Petretto, & Masala, 2006; Wonderlich et al., 2000). Research and clinical data suggest that trauma must be addressed and resolved in order for the full remission of eating disorder symptoms (Brewerton 2004; 2015).

The purpose of the current study is to examine the prognostic factors associated with weight restoration at 3 month follow up in a clinical sample of adolescents who received outpatient treatment for anorexia nervosa. This study seeks to identify biological, psychological, and or social factors that increase or hinder the likelihood of early weight restoration in anorexia nervosa. I seek to answer the following research questions, 1) What percentage of adolescent anorexia nervosa patients obtain weight restoration within the first 3 months of treatment?
2) What prognostic factors increase or decrease the likelihood of obtaining one’s expected body weight at 3 month follow up?

Methodology

Sample

Data for this study were obtained from medical records of patients who have undergone outpatient treatment at a Midwestern urban pediatric hospital behavioral health eating disorder program from 1/1/2012 to 12/31/2014. This program is composed of a multidisciplinary provider team that includes dieticians, physicians, behavioral health therapists, occupational therapist, psychiatrists, and nurses. The primary treatment modalities of the clinic are Maudsley family based therapy (Wallis, Alford, Hanson, Titterton, Madden, & Kohn, 2012), cognitive behavioral therapy (Beck, 2011), and dialectical behavioral therapy (Linehan & Chen, 2005). The sampling frame consists of all patients ages 12 -20 who received a primary diagnosis of an anorexia nervosa using DSM IV-TR criteria over this period (N=98). Although more recent diagnostic codes are available (i.e. DSM V published in May 2013), DSM IV-TR criteria is used in this study because the hospital’s electronic medical record system was not updated until October 2015. Data includes clinical information regarding baseline psychological and physical health and 3-month follow-up data on weight status only. The initial sample included 98 participants. The analytic sample for this study (n=61) includes only those patients who received a primary diagnoses of anorexia nervosa and had full data for variables of interest at baseline and follow up. For this reason, 37 participants were omitted due to missing BMI data at time 2. Table 3.1 contains comparisons of the full, analytic, and omitted samples. Bivariate results reveal that the analytic sample has a higher presence of
comorbid anxiety ($X^2 (1) = 3.8261 \ p \leq .05$) and depression diagnoses than the omitted sample (fisher’s exact = .000).

**Variables**

Key constructs in this study were measured using variables operationalized from pre-existing data available in the medical records. Key constructs in this data set include age, race, sex, weight, percent expected body weight (%EBW), family history of eating disorder, trauma exposure, and comorbid diagnosis. Data were collected at two time points in the study, baseline (Time 1) and 3 month follow up (Time 2). All variables are collected at time 1. Only outcome variables (weight and %EBW percentile) are collected at time 2. Operationalization of these variables are described below. Table 3.2 provides descriptive statistics for key study variables.

**Age.** Age is measured at baseline in years with a range of 12-20 ($M= 15.2 \ SD = 1.73$).

**Race.** Race is measured based on data reported in the medical records. The following racial categories were recorded: White, Asian, Bi-racial/Multi-racial, Black or African American, Latino/Hispanic/ Black, Latino/Hispanic/Unspecified, Latino/Hispanic/White, and Other. In this study, race is categorized as a dichotomous variable, non-Hispanic White and Other. These two categories were selected as the frequency of all other racial categories were too small to identify significant differences.

**Sex.** Male or female as assigned at birth and in the electronic medical records.

**Weight.** Weight is measured as weight at time of encounter and is measured in kilograms. Weight is measured at both time points in the study.
Percent Expected Body Weight (%EBW). Percent expected body weight for age, sex, and height was determined by Centers for Disease Control and Prevention growth charts (http://www.cdc.gov/growthcharts/percentile_data_files.htm). To calculate percent expected bodyweight (%EBW) for a given participant based on his or her height, age, and gender, the 50th percentile BMI for exact age and height at presentation on the CDC BMI-for-age percentiles chart was used (Le Grange, Doyle, Swanson, Ludwig, Glunz, & Kreipe, 2012). A BMI at the 50th percentile would be the expected median in a group of normally developing adolescents, or EBW. The formula %EBW is (%EBW = BMI/50th percentile BMI for age and height x 100). Percent expected body weight EBW was used to create three weight status variables in the study.

Underweight. An underweight variable was created using Time 1 %EBW. A dummy variable was coded as 1 if a patient was less than 85% EBW and 0 if greater than or equal to 85% EBW.

Weight Restoration. A weight restoration variable was created using Time 2 %EBW. A dummy variable was coded as 1 if a patient reached 95% EBW or higher at Time 2 or 0 if a patient was less than 95% EBW.

Weight Change. A continuous weight change variable was created by using the following formula (Follow up weight – Baseline weight = weight change).

Lifetime Trauma. Trauma exposure was measured by a yes or no question asked about their past and current experience of the following: childhood physical abuse, childhood sexual abuse, exposure to domestic violence, parents’ divorce, significant bullying, motor vehicle accident, neighborhood violence, war terrorism, medical procedures, national disaster, and significant death/loss. A lifetime trauma exposure
variable was created based on trauma exposure information. A dummy variable was coded as 1 if a patient reported at least one current or past experience of any of the above events, and 0 if a patient reported no traumatic experiences.

**Comorbid Diagnoses.** Comorbid diagnosis is measured as the presence of an additional Axis 1 DSM IV-TR diagnosis listed in the medical chart. Diagnoses of interest are anxiety and depressive disorders, obsessive compulsive disorder (OCD), and PTSD. Dummy variables for each of the four comorbid diagnoses were created and coded as 0 in the absence of a disorder and 1 if a diagnosis was reported at baseline.

**Family history of eating disorder.** Family history was collected at baseline and includes data about any first degree relative who has a DSM IV-TR mental illness including an eating disorder. Examples of diagnoses include depression, anxiety, eating disorder, schizophrenia, etc.

**Data Analysis**

All analyses were completed using STATA version 14.1. Descriptive statistics are used to present demographic information and study variables. Univariate analyses were done to examine factors that are associated with %EBW. Chi- square values, fisher’s exact test, or independent sample t tests are reported. Both single predictor and multiple logistic regression analyses were conducted to examine the predictive power of biological, psychological, and social factors on weight restoration at Time 2.

**Results**

**Sample characteristics**

Table 3.2 displays descriptive statistics for the analytic sample. The sample is predominantly white (85%) and female (92%). Over a third of the sample reported
comorbid diagnosis of depression or anxiety at baseline. Comorbid diagnoses of OCD and PTSD were significantly smaller at 8% and 5% respectively. Approximately 36% of the sample reported exposure to at least one traumatic event during their lifetime, and 14% of patients reported a first degree relative with an eating disorder. The average weight of the sample at baseline was 46.5 kg (102.5 lbs) SD= 7.2; range 32.1- 68.749.

**Correlates with weight status at baseline**

Independent sample t test results reveal a significant relationship between percent expected body weight and depression diagnosis. Those with a comorbid depression diagnosis had a higher average %EBW (M = 91.9) at baseline than those with no depression diagnosis (M=85.4) t(59) = -2.14; p =.0363. No other significant relationships were found between baseline percent expected body weight and demographic and clinical characteristics. Table 3.3 contains results from bivariate analysis between sample characteristics and being underweight at baseline.

**Predicting weight restoration**

Approximately 51% of the sample reached weight restoration at 95%EBW or higher three months after beginning treatment. The average weight at follow up was 51.4 kg (113.3 lbs) SD= 7.8; range 32.9- 71.99. The average weight increased by 4.9 kg from baseline to follow up. Bivariate analyses of key variables and weight restoration were conducted. Table 3.4 presents these results. A significant relationship between Anxiety diagnosis and weight restoration was found ($\chi^2 = 5.44$, df = 1, $p =.020$). Chi square results suggest a marginally significant relationship, given the small sample size, between comorbid depression diagnosis and weight restoration at 3 month follow up ($\chi^2 = 3.06$, df
In addition, weight restoration was significantly associated with weight change such that patients who reached 95% EBW or higher had a greater weight change (6.5 (5.2)) compared to those who failed to reach weight restoration (3.2 (4.3)) t(59) = -2.7032; p=.009).

Single predictor logistic regression models were conducted to predict weight restoration after 3 months of treatment. Results reveal that the presence of an anxiety disorder is a statistically significant predictor of weight restoration at time 2 (see Table 3.5). A test of the full model against a constant only model was statistically significant ($\chi^2 = 5.58, df = 1, p = .0182$). The odds of reaching an %EBW of 95 or greater were 3.75 times higher than those without comorbid anxiety. Comorbid depression was marginally significant in predicting weight restoration ($\chi^2 = 3.10, df = 1, p = .084$) increasing the odds of being weight restored by 2.6 times.

Three logistic regression models were estimated to predict the likelihood of being weight restored at follow up (%EBW $\geq$ 95%). Table 3.6 presents the results from the three models. Model 1 includes the psychological factors of comorbid depression and anxiety diagnosis. In model 1 comorbid anxiety is a marginally significant predictor of weight restoration ($\chi^2 = 6.31, df = 2, p = .078$). Model 2 includes model 1 and adds the biological factor of sex to the model. This model reveals that comorbid anxiety remains a marginally significant predictor of weight restoration ($\chi^2 = 8.73, df = 3, p = .056$) Model 3 includes Model 2 and adds the social factor of trauma exposure to the model. Again, the presence of comorbid anxiety remains a statistically significant predictor ($\chi^2 = 9.38, df = 4, p = .050$). In all three models, only the presence of a comorbid anxiety diagnosis was significant in predicting the likelihood of being weight restored at three month follow up.
Those who had comorbid anxiety were approximately three times more likely to be weight restored at 3 months than their peers without the comorbid diagnosis across all models.

**Discussion**

I examined biological, psychological, and social predictors of early weight restoration in a clinical sample of 61 adolescent patients with anorexia nervosa. About half of the sample reached weight restoration, greater than or equal to 95% EBW, at 3 month follow up. Among the biopsychosocial factors that were examined in the study, only comorbid anxiety was a significant factor in predicting the likelihood of weight restoration at time 2. Comorbid depression was marginally related to the outcome variable, weight restoration; however, this relationship was not statistically significant and did not remain in the full model which included all biopsychosocial factors. Patients with a comorbid anxiety diagnoses were significantly more likely to reach weight restoration at follow up. This finding is interesting given research that has found that anxiety is a negative prognostic factor for recovery (Yackobovitch-Gavan et al., 2009; Dellava et al., 2010; Zerwas et al., 2013). These studies suggest that patients with anxiety may be predisposed to AN and have a higher risk for illness development and persistence. Clinical features of AN such as food restriction and compulsive exercise often provide short term relief from anxiety symptoms (Kaye, 2008). For this reason, patients may find it extremely difficult to relinquish these behaviors which could result in illness persistence or relapse. However, there have been inconsistencies in the research literature regarding the relationship between anxiety and prognosis in patients with anorexia nervosa (Kezelman, Touyz, Hunt, & Rhodes, 2015), especially for children and
adolescents (Hughes, 2012; Hughes, Goldschmidt, Labuschagne, Loeb, Sawyer, & Grange, 2013).

I present two hypotheses for the positive relationship between comorbid anxiety and weight restoration found in this study. The first hypotheses, an individual level effect, follows along the lines of the fear conditioning model of AN proposed by Strober (2004) that argues that anxiety in patients with AN may lead to fear based phobia of weight gain. However, I argue that patients with comorbid anxiety may apply this fear based learning to recovery in such a way that they develop a fear of continued illness and severe complications (i.e. bradycardia, osteopenia, higher levels of care such as residential treatment, and death) as well as restrictions of their ability to participate in “normal” adolescent activities. The second hypotheses, a system level effect, argues that this sample of patients with comorbid anxiety were more likely to obtain weight restoration because during this time period the eating disorder program was highly focused and specialized in treating a particular clinical presentation of anorexia nervosa, the highly anxious restrictive adolescent. Therefore, patients with comorbid anxiety may have been able to succeed in treatment because it was tailored to their specific needs and focused on their comorbid anxiety as well as their eating disorder symptoms.

**Strengths and Limitations**

Strengths of the present study include a clinical sample of adolescent patients with AN, information regarding clinical comorbid diagnoses versus comorbid symptoms, and a consistent definition of recovery (i.e. weight restoration). Although recovery from anorexia nervosa is not solely a physiological endeavor, weight restoration is an
important predictor of long term success and future research would benefit from using both physiological and psychological indicators of recovery.

The use of retrospective data does have its limitations. First the sample is limited to patients who have complete data on variables of interest and both time points. This reduced our sample size by approximately by one-third and limited the power available in our analyses. This limitation may be reason for the lack of significant biological and social predictors in this study. Second, although early weight restoration is important, maintenance of weight restoration over time is critical in understanding long term recovery. Due to the lack of follow up data after the 3 month time period, it was not possible to assess if patients were able to maintain their EBW and subsequently what factors may predict weight maintenance over time.

These findings confirm previous research that comorbid anxiety is of significant clinical interest for clinicians, patients and families of adolescents with anorexia nervosa. Although results from this study add to mixed findings regarding the nature of the relationship between anxiety and anorexia nervosa, future research should focus on the ability of anxiety and other comorbidities to delineate between patients with various clinical presentations. Developing our understanding of the risk and protective factors of recovery will only help us better serve adolescents and families struggling with this pervasive disorder.
Figure 3.1 A Biopsychosocial Model of Weight Restoration for adolescent patients with anorexia nervosa

Biological Factors
- Sex
- Family Hx Eating Disorder
- Family Hx Psychiatric Disorders

Psychological Factors
- Anxiety
- Depression
- OCD
- PTSD

Social Factors
- Trauma Exposure

Eating Disorder Symptoms
- % EBW
- Weight Restoration
<table>
<thead>
<tr>
<th>Sample</th>
<th>Full (N=98)</th>
<th>Analytic (n=61)</th>
<th>Omitted (n=37)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>15.2 (1.8)</td>
<td>15.2 (1.7)</td>
<td>15.1 (2.0)</td>
<td>.696</td>
<td>t-test</td>
</tr>
<tr>
<td>% White</td>
<td>85.7</td>
<td>85.2</td>
<td>86.5</td>
<td>.865</td>
<td>chi square</td>
</tr>
<tr>
<td>% Female</td>
<td>93.9</td>
<td>91.8</td>
<td>97.3</td>
<td>.404</td>
<td>fisher’s</td>
</tr>
<tr>
<td>Depressive Disorder (%)</td>
<td>25.5</td>
<td>37.7</td>
<td>5.4</td>
<td>.000**</td>
<td>chi-square</td>
</tr>
<tr>
<td>Anxiety Disorder (%)</td>
<td>27.6</td>
<td>34.4</td>
<td>16.2</td>
<td>.050*</td>
<td>chi-square</td>
</tr>
<tr>
<td>OCD (%)</td>
<td>7.1</td>
<td>8.2</td>
<td>5.4</td>
<td>.603</td>
<td>chi-square</td>
</tr>
<tr>
<td>PTSD (%)</td>
<td>3.1</td>
<td>4.9</td>
<td>0</td>
<td>.171</td>
<td>chi-square</td>
</tr>
<tr>
<td>Trauma Exposure (%)</td>
<td>31.6</td>
<td>36.1</td>
<td>24.3</td>
<td>.226</td>
<td>chi-square</td>
</tr>
<tr>
<td>Family Hx ED (%)</td>
<td>12.8</td>
<td>14</td>
<td>10.7</td>
<td>1.00</td>
<td>chi-square</td>
</tr>
<tr>
<td>%EBW, mean (SD)</td>
<td>88.9 (11.4)</td>
<td>87.8 (11.9)</td>
<td>90.6 (10.5)</td>
<td>.248</td>
<td>t-test</td>
</tr>
<tr>
<td>Weight, kg, mean (SD)</td>
<td>47.2 (7.4)</td>
<td>46.5 (7.2)</td>
<td>48.3 (7.6)</td>
<td>.255</td>
<td>t-test</td>
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</table>

*p<.05 **p<.01
<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
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</tr>
<tr>
<td>% White</td>
<td>85.3</td>
</tr>
<tr>
<td>% Female</td>
<td>91.8</td>
</tr>
<tr>
<td>%EBW, mean (SD)</td>
<td>87.8 (11.9)</td>
</tr>
<tr>
<td>Depressive disorder (%)</td>
<td>37.7</td>
</tr>
<tr>
<td>Anxiety disorder (%)</td>
<td>34.4</td>
</tr>
<tr>
<td>PTSD (%)</td>
<td>4.9</td>
</tr>
<tr>
<td>OCD (%)</td>
<td>8.2</td>
</tr>
<tr>
<td>Trauma exposure (%)</td>
<td>36.1</td>
</tr>
<tr>
<td>Family Hx ED (%)</td>
<td>14</td>
</tr>
<tr>
<td>%EBW, mean (SD)</td>
<td>87.8 (11.9)</td>
</tr>
<tr>
<td>Weight, kg, mean (SD)</td>
<td>46.5 (7.2)</td>
</tr>
</tbody>
</table>
### Table 3.3 Comparison of Characteristics based on %EBW underweight category at baseline

<table>
<thead>
<tr>
<th>Sample</th>
<th>&lt; 85% EBW (n=30)</th>
<th>≥ 85% EBW (n=31)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>15.2 (.35)</td>
<td>15.3 (.29)</td>
<td>.897</td>
<td>t-test</td>
</tr>
<tr>
<td>% White</td>
<td>86.7</td>
<td>83.9</td>
<td>1.00</td>
<td>fisher’s</td>
</tr>
<tr>
<td>% Female</td>
<td>90</td>
<td>93.6</td>
<td>.671</td>
<td>fisher’s</td>
</tr>
<tr>
<td>Depressive Disorder</td>
<td>30</td>
<td>45.2</td>
<td>.212</td>
<td>chi square</td>
</tr>
<tr>
<td>Anxiety Disorder</td>
<td>33.3</td>
<td>35.5</td>
<td>.860</td>
<td>chi square</td>
</tr>
<tr>
<td>OCD</td>
<td>6.7</td>
<td>9.7</td>
<td>1.00</td>
<td>fisher’s</td>
</tr>
<tr>
<td>PTSD</td>
<td>0</td>
<td>9.7</td>
<td>.238</td>
<td>fisher’s</td>
</tr>
<tr>
<td>Trauma Exposure</td>
<td>26.7</td>
<td>45.2</td>
<td>.133</td>
<td>chi square</td>
</tr>
<tr>
<td>Family Hx ED</td>
<td>16.7</td>
<td>11.5</td>
<td>.697</td>
<td>fisher’s</td>
</tr>
</tbody>
</table>

*p<.05  **p<.01  *p<.10
Table 3.4. Comparison of characteristics based on weight restoration at 3 month follow up

<table>
<thead>
<tr>
<th>Sample</th>
<th>&lt; 95% EBW (n=30)</th>
<th>≥ 95% EBW (n=31)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>15.3 (.37)</td>
<td>15.1 (.25)</td>
<td>.648</td>
<td>t-test</td>
</tr>
<tr>
<td>% White</td>
<td>83.3</td>
<td>87.1</td>
<td>.731</td>
<td>fisher’s</td>
</tr>
<tr>
<td>% Female</td>
<td>86.7</td>
<td>96.8</td>
<td>.195</td>
<td>fisher’s</td>
</tr>
<tr>
<td>Depressive Disorder (%)</td>
<td>30</td>
<td>45.2</td>
<td>.080*</td>
<td>chi square</td>
</tr>
<tr>
<td>Anxiety Disorder (%)</td>
<td>20</td>
<td>48.4</td>
<td>.020*</td>
<td>chi square</td>
</tr>
<tr>
<td>OCD (%)</td>
<td>10</td>
<td>6.5</td>
<td>.671</td>
<td>fisher’s</td>
</tr>
<tr>
<td>PTSD (%)</td>
<td>3.3</td>
<td>6.5</td>
<td>1.00</td>
<td>fisher’s</td>
</tr>
<tr>
<td>Trauma Exposure (%)</td>
<td>33.3</td>
<td>38.7</td>
<td>.662</td>
<td>chi square</td>
</tr>
<tr>
<td>Family Hx ED (%)</td>
<td>13.0</td>
<td>14.8</td>
<td>1.00</td>
<td>fisher’s</td>
</tr>
</tbody>
</table>

*p ≤ .05  **p ≤ .01  + p ≤ .10
Table 3.5 Single predictor logistic models of weight restoration at 3 month follow up (n=61)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>B(SE)</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression Dx</td>
<td>.95 (.55) *</td>
<td>2.6</td>
</tr>
<tr>
<td>Anxiety Dx</td>
<td>1.3 (.58) *</td>
<td>3.75</td>
</tr>
<tr>
<td>Female</td>
<td>1.5 (1.1)</td>
<td>4.6</td>
</tr>
<tr>
<td>Lifetime Trauma</td>
<td>.23 (.53)</td>
<td>1.3</td>
</tr>
</tbody>
</table>

Note: The dependent variable in this analysis is %EBW coded so that 0 = less than 95% expected body weight and 1 = greater or equal to 95% EBW.

*p ≤ .05  **p ≤ .01  +p ≤ .10
Table 3.6. Logistic regression analysis for predicting weight restoration at 3 month follow up  (n=61)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Model 1</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE)</td>
<td>OR</td>
<td>B (SE)</td>
<td>OR</td>
<td>B (SE)</td>
<td>OR</td>
</tr>
<tr>
<td>Depression Dx</td>
<td>-.52 (.61)</td>
<td>1.7</td>
<td>.41 (.62)</td>
<td>1.5</td>
<td>-.33 (.63)</td>
<td>1.4</td>
</tr>
<tr>
<td>Anxiety Dx</td>
<td>1.1 (1.8)*</td>
<td>3.0</td>
<td>1.3 (.66)*</td>
<td>3.5</td>
<td>1.3 *</td>
<td>3.7</td>
</tr>
</tbody>
</table>

Model $X^2 =$ 6.31*  
Pseudo $R^2 =$ .0747

Model $X^2 =$ 8.73*  
Pseudo $R^2 =$ .1033

Model $X^2 =$ 9.38*  
Pseudo $R^2 =$ .1109

Note: The dependent variable in this analysis is %EBW coded so that 0 = less than 95% expected body weight and 1 = greater or equal to 95% EBW.
Model 1 includes psychological factors - comorbid depression and anxiety diagnosis only; Model 2 = Model 1 + biological factor of sex; Model 3 = Model 2 + the social factor of lifetime trauma

*p ≤ .05  *p ≤ .10
Chapter 4. Examining youth and caregiver reports of depression and anxiety in families seeking eating disorder treatment

Introduction

Research in the areas of mental health, epidemiology, and family science has shown a significant relationship between eating disorders and depression and anxiety disorders (Bulik, 2002). Multiple models attempt to explain the comorbidity between eating, affective, and anxiety disorders. Scholars agree that eating disorders, anxiety, and depression have shared etiological factors (Bulik, 2002). Given the high prevalence of depression and anxiety in the clinical manifestation of eating disorders it is important to better understand initial presentation of these symptoms. Specific to adolescents with eating disorders, it is important to understand the youth and family’s understanding of their depression and anxiety symptoms in their efforts to obtain treatment. Research shows that recovery from an eating disorder is much less likely the longer the duration of the illness (Eisler, Dare, Russell, Szmukler, le Grange, & Dodge, 1997; Von Holle et al., 2008). Early recognition and intervention have been cited as important prognostic factors for long term success and recovery (Russell, Szmukler, Dare, & Eisler, 1987; Treasure & Russell, 2011; Reas, Williamson, Martin, & Zucker, 2000). However, adolescents often do not want to participate in treatment, not only out of fear associated with weight gain and recovery but also because of the fear of missing out on "normal" adolescent activities (Lock & Le Grange, 2012). Family treatment approaches have been presented as an
effective strategy for the successful treatment of adolescent eating disorders (Russell, Szmukler, Dare, & Eisler, 1987; Couturier, Kimber, & Szatmari, 2013). These approaches often utilize the primary caregiver’s desire for recovery as the impetus to treatment instead of focusing on the adolescent’s initial motivation to change. For this reason, it is helpful to understand the youth’s self-reported symptoms as well as the caregiver’s perception of the child’s symptoms. Discrepancies between youth and caregiver reports of youth psychopathology are of great interest as parent reports are often used to help assess and diagnose mental disorders, such as eating disorders, depression, and anxiety in adolescents. However, research has found that parent and adolescent reports of general well-being and mental health are often discrepant (Achenbach, McConaughy, and Howell, 1987; De Los Reyes & Kazdin, 2005). Discrepancies among informants could be problematic because this information is often used by clinicians and researchers to make important decisions. Clinicians use informant reports to generate treatment plans and assess progress over time. Researchers use reports to draw conclusions about empirical work and treatment effectiveness. Discrepancies in reports can cause uncertainties in treatment delivery and the ability to draw solid conclusions from empirical data (De Los Reyes et al., 2015). Little is known about families’ initial understanding of adolescent comorbid symptoms for those struggling with disordered eating. What we do know is that families and loved ones often struggle to understand DE pathology and related anxiety or depressive symptoms (Couturier, Kimber, Jack, Niccols, Van Blyderveen, & McVey, 2013; Eisler, 2005). The current study fills this gap in the literature by examining the congruence among youth and
caregiver reports of the adolescent’s depression and anxiety symptoms in families seeking eating disorder treatment.

**Literature Review**

**Disordered Eating and Eating Disorders in Adolescence**

Disordered eating behaviors (DE) and eating disorders (ED) warrant considerable attention given their ability to impair the physical and psychosocial functioning of adolescents and adults (Golden et al., 2003). Epidemiological research reports that over the last two decades the incidence rate of anorexia has increased for females ages 15-19 and there has been an elevated mortality rate across all eating disorder types (Smink, Van Hoeken, & Hoek, 2012). Disordered eating behaviors often begin during adolescence and can persist over time, developing into full blown eating disorders that are often complex and difficult to treat. Disordered eating is defined as a range of abnormal eating behaviors that can negatively impact a person’s biopsychosocial well-being (American Psychiatric Association, 2000). Examples of DE include restriction of food intake, self-induced vomiting, binge-eating, and overuse of laxatives/diuretics. All eating disorders include DE but are distinguished by the severity and frequency of engagement in DE behaviors. One study estimated that approximately 55% of 9th and 12th grade females and 30% of 9th and 12th grade males reported DE (Croll, Neumark-Sztainer, Story, & Ireland, 2002). A more recent Norwegian study of high school youth found a 64% prevalence rate of DE among females and 45% rate among males (Torstveit, Aagedal-Mortensen, & Stea, 2015). Studies have shown that consistent participation in DE in adolescence significantly increases the likelihood of continued used of DE and/or development of an ED into young adulthood (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011).
Comorbidity Depression and Anxiety in ED

Individuals struggling with DE or a clinical ED often struggle with other comorbid physical and psychological problems as well. A review found that adolescents with DE behaviors were more likely than their peers without DE to report mood disorders such as anxiety or depression and struggle with substance use and abuse (Chamay-Weber, Narring, & Michaud, 2005). This relationship between psychological comorbidities, such as depression and anxiety, and DE has also been verified in adolescents with clinical EDs (Hughes, 2012; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). Similar rates of a lifetime depressive disorder in patients with anorexia and bulimia nervosa are reported, ranging from 16 - 50% (Herpertz-Dahlmann, 2009). Despite inconsistent prevalence rates, it is clear individuals with eating disorders are more likely to experience a lifetime depression or anxiety disorder than those in the general population (Hughes, 2012; Swinbourne & Touyz, 2007; Swinbourne, Hunt, Abbott, Russell, St Clare, & Touyz, 2012). The relationship between comorbid depression and anxiety and initial presentation of eating disorder symptoms has begun to be studied in more recent years. A cross-sectional study examining 371 children and adolescents’ intake assessments from a specialty ED clinic found that while 58% of the sample had no comorbid diagnosis, 23% had a comorbid mood disorder, 9% had a comorbid anxiety disorder, and 10% had both comorbid anxiety and mood disorder (Hughes, Goldschmidt, Labuschagne, Loeb, Sawyer, & Grange, 2013). Results from this study suggest that comorbid depression and anxiety may be differentially related to clinical characteristics and severity of ED symptoms in youth. Children and adolescents with comorbid depression presented with more severe and complex presentations of ED.
than those with no comorbid diagnosis or comorbid anxiety alone (Hughes, Goldschmidt, Labuschagne, Loeb, Sawyer, & Grange, 2013).

Caregivers understanding of comorbid depression and anxiety

The prevalence of comorbid depression and anxiety in youth with DE and ED warrants clinical and scholarly attention. Despite their complex nature, eating disorders are treatable. However, the presence of additional psychiatric comorbidities, such as anxiety and depression, may increase the needs of patients and families seeking treatment and could provide additional obstacles for recovery. Getting individuals, particularly adolescents to seek treatment can be an uphill battle. Adolescents with DE and/or ED often minimize the frequency, severity, and impact of their DE behaviors (American Psychiatric Association, 2013). Due to the secretive nature of eating disorders, caregivers of adolescents may not be aware of the severity of eating pathology until well into the course of the illness. However, caregivers may be more aware of changes in the adolescent’s mood or daily functioning that may change as a result of depressed mood or chronic anxiety. For this reason, psychiatric comorbidities may be more of an impetus to initial assessment and treatment seeking than ED symptoms alone (Fursland & Watson, 2014). However, little is known about caregiver’s understanding of adolescent’s ED symptoms and even less is known about their perceptions of comorbid symptoms of anxiety or depression in adolescents with DE or an ED. Understanding caregiver initial perceptions of comorbid symptoms and discrepancies between youth and caregivers’ perceptions of comorbid depression and anxiety is important in conducting effective family based interventions to promote a full biopsychosocial recovery. In addition, understanding caregiver perceptions of psychiatric comorbidities may indicate the
likelihood of family involvement in treatment and willingness to adopt treatment recommendations.

Discrepancies between youth and caregiver reports of mental health

Previous research has revealed that reports of social, emotional, and behavior problems among youth and parents are discrepant (Achenbach, McConaughy, & Howell, 1987). Achenbach and colleagues’ (1987) seminal meta-analysis of 119 studies provided robust evidence for the lack of agreement (r ≤ .20) between multiple informants (youth, parent, teacher) of youth behavior problems (internalizing and externalizing). This study found that youth and parents had the lowest level agreement between any dyad. Since its publication, several studies have confirmed that youth and caregivers’ reports remain discrepant (Seiffge-Krenke, & Kollmar, 1998; Jensen et al., 1999; Vierhaus, Rueth, & Lohaus, 2016; Waters, Stewart-Brown, S. & Fitzpatrick, 2003; Weissman et al., 1987). When focusing specifically on mental health concerns, studies have found that youth in community samples report significantly higher mean scores for internalizing and externalizing symptoms than parents (Duhig, Renk, Epstein, & Phares, 2000; van der Ende, Verhulst, & Tiemeier, 2012; Waters, Stewart-Brown, S. & Fitzpatrick, 2003). However, this relationship has been found to be inverted among clinical samples (Kristensen, 2001).

Although research consistently shows discord between youth and caregiver reports of youth psychiatric problems, very few studies have examined the relationship between youth and caregiver reports of psychological problems in adolescents with ED. To my knowledge, only one study has examined correspondence between youth and caregiver reports of psychiatric problems in adolescents with EDs (Salbach-Andrae et al.,
Salbach-Andrae and colleagues (2008) examined parent and youth self-report agreement on emotional and behavioral symptoms in adolescents age 11-18 with ED. Results revealed that agreement between parents and youth were low across all symptoms. In addition, adolescents reported significantly lower levels of internalizing symptoms than parents (Salbach-Andrae et al., 2008). Discrepancies among youth and caregivers are important among this clinical population because parents are often the initiators of treatment and may dictate treatment success or failure particularly in family based treatment approaches. Furthermore, lack of parental understanding of illness severity and psychiatric and physical comorbidities associated with ED has been cited as a significant barrier to successful treatment uptake by providers (Couturier, Kimber, Jack, Niccols, Van Blyderveen, & McVey, 2013). Given the scarcity of research in this area specific to adolescent ED patients, it is important to examine whether or not discrepancies between youth and caregiver reports of additional mental health concerns are consistently discordant and if they can be explained by other contextual factors such as ED severity. The purpose of this study was to examine the difference between youth and caregiver self-reports of youth anxiety and depression symptoms in a clinical sample of families seeking ED treatment. I evaluate whether demographic factors (race, and age) and ED symptom severity were associated with youth and caregiver reports of depression and anxiety symptoms. There are three main hypotheses for this study:

1. Youth with higher ED symptoms will report higher anxiety and depression scores.
2. Caregivers will report higher depression and anxiety symptoms than youth.
3. Caregivers of youth with more severe ED symptoms will exhibit more congruence in reports of depression and anxiety.
Methodology

Sample

Data for this study was obtained from administrative secondary data that involves the population of families seeking treatment at an urban Midwestern pediatric hospital eating disorder clinic from July 1, 2015 to March 1, 2016. Data from the initial diagnostic assessment was reviewed including demographic and clinical information regarding baseline psychological and physical health.

The sampling frame consists of all youth ages 12 -18 and their primary caregivers who completed a diagnostic assessment at the clinic (N=99). Some of the sample was omitted due to missing data. Forty subjects were omitted due to lack of primary caregiver’s report of depression/anxiety symptoms, 8 were omitted due to lack of youth’s self-reported depression symptoms, and 2 were omitted for lack of youth self-reported anxiety symptoms. The analytic sample for this study is 49 adolescent and caregiver pairs who completed all self-report measures included in the study (Age: M=15 years, SD=1.6; Race: White 88.9% Other 11.1%). Table 4.1 contains comparisons of the full, analytic, and omitted samples. T-test results reveal that the analytic sample has significantly higher average anxiety (M = 55.7 SD =16.3; (t (61) = -2.28, p=.026) than the omitted sample.

Variables

Age. Age is measured in years at the time of assessment with a range of 12-18 (M= 15 SD = 1.6)
Race. Race is measured based on data reported in the medical records. Due to the limited variability across racial groups, race is measured as White and Other (including Asian, Black, and African). No ethnicity information was available for this sample.

Sex. Sex is measured as male or female as assigned at birth and in the electronic medical records.

Eating Disorder Symptomatology. Eating disorder symptoms were measured using the global score from the sixth edition of the Eating Disorders Examination Questionnaire (EDE-Q) (Fairburn & Beglin, 2008). This sample had a mean global score of 2.2 (1.8) with a range of 0 to 5.65. The EDE-Q is 41-item self-report test which measures behaviors in four subscales (restraint, eating concern, weight concern, and shape concern) and provides a global score for each subject. The EDE-Q focuses on the past 28 days and is scored using a 7-point rating format. The EDE-Q has been found to have good concurrent and acceptable criterion validity in a community sample (Mond, Hay, Rodgers, Owen, & Beumont, 2004). Normative EDE-Q data is available for a number of subgroups including adolescents and young women (Carter, Stewart, & Fairburn, 2001; Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis, 2014; Mond, Hay, Rodgers, & Owen, 2006). EDE-Q norms are essential for the interpretation of tests scores and comparisons across samples. Based on previous normative data, EDE-Q scores $\geq 4$ are indicative of clinically significant disordered eating pathology (Carter, Stewart, & Fairburn, 2001; Mond, Hay, Rogers, & Owen, 2006).

Two separate measures were used to assess for depressive symptoms. The first measure, the Quick Inventory of Depressive Symptomatology - Self Report (QIDS), was used to obtain a short term measure of depressive symptoms in the week prior to
assessment. The second measure, the Revised Child Anxiety and Depression Scale (RCADS-C) was used to obtain a more global understanding of the youth’s depressive symptoms and distinguish between those above and below the clinical threshold for major depression. The RCADS-C was also utilized in order to compare youth and caregiver reports of adolescent depressive symptoms as the RCADS has a parental counterpart utilizing the same scales. Descriptions of these measures are presented below.

**Depression Severity.** The Quick Inventory of Depressive Symptomatology - Self Report 16 (QIDS) was used to assess for severity of depressive symptoms over the last seven days prior to assessment. The QIDS is a 16 item measure that evaluates the overall severity of each of the nine criterion symptom domains used to diagnose a major depressive episode as defined by the DSM-IV (i.e. sleep, appetite/weight, concentration, energy, sad mood, suicidal ideation, self-concept/guilt, psychomotor activity, and interest) (Rush et al., 2000, 2003; Trivedi et al., 2004). For three domains (sleep, psychomotor, appetite/weight disturbances), multiple questions are used. For the sleep domain, the highest score on any of the four sleep items; for the appetite/weight domain, the highest score on any one of the four items, and for psychomotor changes, the highest score on either one of the two items were chosen to represent the domain. Each symptom item is scored on a 0–3 scale. The QIDS total score is computed by adding scores for the nine criterion domains. Total score range from 0 to 27 and severity is measured by the total score using the following categories, none (1-5), mild (6-10), moderate (11-15), severe (16-20), and very severe (21-27). The QIDS has been found to have good internal consistency (Cronbach’s $\alpha = .86$) (Rush et al., 2003) and found to be reliable ($\alpha \geq 0.8$) and
suitable for use with adolescents (Bernstein et al., 2010). The average QIDS score in this sample fell into the mild to moderate category at 10.4 (SD = 5.7) with a range of 0 to 22.

**Major Depression.** Youth self-reported depressive symptoms were measured using the t score results of the major depression subscale of the Revised Child Anxiety and Depression Scale (RCADS-C). The RCADS-C is a 47-item, youth self-report questionnaire that assesses symptoms of depression and anxiety based on DSM IV criteria (Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000;). The RCACDS-C is comprised of 6 subscales including: separation anxiety disorder, social phobia, generalized anxiety disorder, panic disorder, obsessive compulsive disorder, and major depressive disorder. For all RCADS subscales, scores between 65-69 are considered borderline and scores of 70 or higher are above the clinical threshold. The average depression score was 63.0 (SD=18.2) with a range of 32 to 105.

**Anxiety.** Youth’s self-reported anxiety symptoms were measured using the t score results of the total anxiety scale of The Revised Child Anxiety and Depression Scale (RCADS-C). The average anxiety score was a 55.7(SD=16.3) with a range of 30 to 94. The RCADS yields a total anxiety Scale (sum of the 5 anxiety subscales). Items are rated on a 4-point Likert-scale from 0 (“never”) to 3 (“always”).

**Caregiver perception of youth anxiety.** Caregiver perception of youth anxiety symptoms was measured using the t score results for the total anxiety scale using the Revised Child Anxiety and Depression Scale – Parent Version (RCADS-P). The average caregiver perceived anxiety score was a 60.0(SD=15.8) with a range of 37 to 90. The RCADS-P similarly assesses parent report of youth’s symptoms of anxiety and depression across the same subscales as the RCADS-C. The RCADS-P has been found
to have high internal consistency and convergent validity, and has been shown to accurately assess anxiety and depression symptoms in youth (Ebesutani et al. 2010).

Evidence for the clinical and research utility of RCADS has also been demonstrated in clinical and international samples (Chorpita, Moffitt, and Gray, 2005; de Ross, Gullone, and Chorpita, 2002).

**Caregiver perception of youth depression.** Caregiver perception of youth depressive symptoms was measured using the t score results for the major depression subscale using the Revised Child Anxiety and Depression Scale – Parent Version (RCADS-P). The average caregiver perceived depression score was 71.9 (SD=15.8) with a range from 40 to 105.

**Data Analysis**

All analyses were completed using STATA version 14.1. Independent sample t-tests, chi-square tests of independence, and fisher’s exact test were used to check for selection bias as not all participants in the population met criteria for study inclusion. Independent samples t-tests were used to compare EDE-Q Global and subscale scores to previously published norms (Carter, Stewart, & Fairburn, 2001; Mond, Hay, Rodgers, & Owen, 2006; Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis, 2014). In order to test the hypotheses correlations and paired sample t tests were conducted to examine associations between variables of interest.

**Results**

**Descriptive data and clinical cutoffs**

Demographic information and means and standard deviations for the EDE-Q global and subscales for the analytic sample are presented in Table 4.1. The sample is
predominantly white (87%) and female (94%). The average age of the adolescent was 15 and the majority of primary caregivers in the sample were the biological mother (87%). Based on previous normative data, EDE-Q scores ≥ 4 are indicative of clinically significant disordered eating pathology (Carter, Stewart, & Fairburn, 2001; Mond, Hay, Rogers, & Owen, 2006). Based on these cutoffs, approximately 20% (n=10) of the sample scored in the clinically significant range on the Global scale. In addition, 10 (18%), 7 (14%), 17(35%), and 13 (27%) participants scored in the clinically significant range on the restraint (R), eating concern (EC), shape concern (EC), and weight concern (WC) subscales respectively. Comparisons of EDE-Q mean and standard deviations for normative samples of adolescents compared to the current sample are provided in Table 4.2. Overall, the current sample had a higher average score on all 5 subscales and the global subscale compared to normative samples of adolescent females (Carter, Stewart, & Fairburn, 2001; Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis, 2014) and adolescent males (Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis, 2014). A larger percentage of adolescents in this sample endorsed clinically significant weight (35% vs 13%) and shape concern (27% vs 20%) than the normative samples of young adolescent females (Carter, Stewart, & Fairburn, 2001).

Table 4.3 presents mean differences between the current sample and normative adolescent data for the EDE-Q subscales. The current sample reported significantly higher levels of ED symptomology (Global, t (855) =2.69, p=.007; R, t (855) = 2.67, p=.007; EC, t (855) = 4.56, p=.000; SC, t (855) = t=2.36, p=.018; WC, t (855) = 2.37, p=.018) than the young adolescent normative sample (Carter, Stewart, & Fairburn, 2001). Compared to adolescent females ages 12 – 18 (Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis, 2014).
Sarll, K., & Lewis, 2014), the current sample endorsed significantly higher, Global pathology ($t$ (1181) =1.81, $p$=.070), R ($t$ (1181) = 2.12, $p$=.034), EC ($t$ (1181) =2.43, $p$=.015), WC ($t$ (1181) =4.55, $p$=.000). Groups did not differ on SC, $t$ (1181) = 1.08, $p$ = .282.

**Prevalence of clinically significant depression and anxiety**

Youth completed two self-report measures to assess for the presence (RCADS-C) and severity (QIDS) of depressive symptoms. The distribution of depressive severity is presented in Table 4.4. Based on results from the QIDS, 24% of youth did not endorse clinically significant depressive symptoms while the remainder of the sample reported experiencing mild (31%) to very severe (4%) depression.

Table 4.5 displays the percentage of youth and caregivers that reported depressive and anxiety symptoms in the clinically significant range. Approximately, 33% (16) of youth reported depression symptoms above the clinical threshold, t-score $\geq$70, as measured by the RCADS-C. In regards to overall anxiety, approximately 20% (10) of youth reported anxiety symptoms above the clinical threshold for the total anxiety scale of the RCADS-C. In addition, 25% (12), 22% (11), 16%, 10%, and 10% of youth reported symptoms above the clinical threshold for panic disorder, separation anxiety, social phobia, generalized anxiety, and obsessive compulsive disorder respectively (see Table 4.5). Results from the child and parent versions of the RCADS reveal that caregivers reported consistently higher levels of clinically significant symptoms than youth for major depression, panic disorder, generalized anxiety, social phobia, and the total anxiety scale (see Table 4.5). Very minimal differences were reported between
caregiver and youth reports of clinical significant symptoms of separation anxiety and obsessive compulsive disorder.

**Association between ED symptoms and reports of internalizing symptoms**

Table 4.6 presents correlations between ED, anxiety, and depression symptoms. Correlation results reveal a strong positive correlation between youth reports of anxiety and depression using the RCADS-C (.75). Caregiver perceptions of adolescent depression and anxiety were also strongly correlated (.74). However, correlations between adolescent reported depression and caregiver perceived depression were moderate (.57). A similar correlation was found for adolescent anxiety and caregiver perceived anxiety (.54). An examination of the relationship between eating disorder symptoms and internalizing symptoms found that depression severity and Global EDE-Q score were strongly positively correlated, $r (49) = .70$, $p=.000$. Depression Severity was also strongly positively correlated with eating concern, $r (49) = .73$ $p =.000$, and moderately positively correlated with restraint (.58), shape concern (.65) and weight concern (.63) ($p=.000$). The relationship between clinically significant scores on the Global EDE-Q and depression severity was also examined. There was a moderately positive correlation between clinically significant ED symptoms and depression severity, $r (49) = .62$, $p=.000$. No significant relationship was found between EDE-Q score and total anxiety or specific anxiety disorders. There are also weak positive correlations between clinically significant eating disorder symptoms and major depression, $r (49) =.29$, $p=.041$ and total anxiety, $r (49) =.27$, $p=.064$ (see Table 4.6).
**Clinically significant ED symptoms and internalizing symptoms**

Independent t tests were conducted to examine the relationship between self-reported clinically significant ED symptoms and self-reported depression and anxiety. Results, presented in Table 4.7, reveal a significant mean difference in reports of depression severity between clients with ED symptoms above the clinical threshold and those below the threshold. Youth above the clinical threshold reported more severe depression (M = 17.3 SD = 3.6; t (47) = -5.46, p = .000) with a mean difference of 8.7. In addition, youth with clinically significant ED symptoms reported significantly higher scores on the major depression subscale of the RCADS-C than those below the clinical threshold (mean difference: 13.1), M = 73.4 SD = 22; t (47) = -2.1, p = .041). A marginally significant difference was found between youth with clinically significant ED symptoms and overall anxiety score such that youth with clinically significant ED symptoms endorsed higher anxiety symptoms overall than those with lower ED symptoms (M = 64.2 SD = 19; t (47) = -1.89, p = .064). Altogether these results provide positive support for hypothesis 1 (i.e. youth with higher ED symptoms will report higher anxiety and depression scores). In particular, the positive relationship between ED symptoms and depression symptoms was strong while support for the relationship between anxiety and ED symptoms was weak with marginally significant associations.

**Youth and caregiver congruence in reports of depression and anxiety**

Paired t-tests were used to compare youth and caregiver reports of youth anxiety and depression. Table 4.8 shows mean differences between youth and caregiver perception of anxiety and depression using results from the child and parent version of the RCADS. First, a significant mean difference exists between youth and caregiver
reports of major depression with caregivers reporting higher depression scores (M = 71.9 SD =15.8; (t (48) = -3.93, p=.000). Second, a significant mean difference exists between youth and caregiver reports of generalized anxiety disorder with caregivers reporting higher scores (M=58 SD= 14.6; (t (48) = -4.6, p=.000). Third, a significant mean difference was also found between youth and caregiver reports of social phobia with caregivers reporting higher scores (M=57.9 SD =2.0; t (48) = -2.08, p=.043). Finally, a marginally significant mean difference was found between youth and caregiver reports of overall anxiety symptoms (M=60.0 SD = 15.8; (t (48) = -1.93, p = .060) with caregivers again reporting higher levels of symptomatology. Altogether these findings provide evidence to support hypothesis 2 (i.e. caregivers will report higher depression and anxiety symptoms than youth).

Effect of age, race, and eating disorder symptoms on congruence

Table 4.9 presents the paired comparisons of mean differences between major depression and total anxiety scores for youth and caregivers stratified by age, race, and ED symptomatology. Overall, no significant differences existed between youth and caregivers when reporting obsessive compulsive disorder (OCD) or panic disorder. When stratified by age, results suggest that caregivers of younger adolescents (ages 13-15) are less congruent, reporting significantly higher major depression scores (MDD column in Table 4.9) than caregivers of older adolescents (mean difference -12.8, p=.000). Similarly, caregivers of younger adolescents also reported significantly higher social phobia (SP) scores (mean difference -7.0, p=.033) and total anxiety scores (mean difference -6.4, p=.060). Results from the generalized anxiety scale (GAD), suggest that there is a significant lack of congruence between youth and caregiver reports regardless
of age. When stratified by race, a significant difference was found for major depression (mean difference -9.5, p=.000) and social phobia (mean difference -4.3, p=.041) with White caregivers reporting higher levels of disagreement than non-White caregivers. It is important to note that the lack of racial variability in the study sample may have influenced these findings. Finally, when stratified by ED symptoms, results suggest that there are no significant differences between youth and caregiver reports of major depression and total anxiety in youth with more severe eating disorder symptoms. A significant difference between youth and caregiver reports was found for both the major depression scale (mean difference -10.1, p=.000) and the total anxiety scale (mean difference -5.3, p=.049) for patients below the clinical threshold on the Global EDE-Q scale, while those with above clinical threshold symptoms were statistically congruent. In addition, caregivers of patients with below threshold ED symptoms also reported significant disagreement on the social phobia scale (mean difference -4.8, p=.047). Again, results suggest a significant lack of congruence between youth and caregiver reports regardless of eating disorder symptoms. These findings provide support for hypothesis 3. Caregivers of youth with more severe ED symptoms did exhibit more congruence in reports of depression and anxiety than those caregivers of youth with less severe ED symptomology.

Discussion

Using a small clinical sample of families seeking eating disorder treatment, this study examines discrepancies between youth and caregiver reports of anxiety and depression. Previous research has examined the association between disordered eating, eating disorders, and comorbid anxiety and depression, still little is known about
caregivers’ understanding of depression and anxiety in youth seeking treatment for eating pathology. I compare youth and caregiver reports of adolescent anxiety and depression symptoms in relation to youth self-reported eating disorder symptoms, depression severity, and demographic factors (i.e. age and race). Findings from this study provided support for all three hypotheses. Approximately one-third and one-fifth of youth in the study reported comorbid major depression and anxiety respectively. These numbers are similar to those reported in previous clinical and epidemiological research (Hughes, Goldschmidt, Labuschagne, Loeb, Sawyer, & Grange, 2013; Hughes, 2012). In addition, youth with clinical ED symptoms reported more severe depression and higher scores on the major depression and total anxiety scales of the RCADS-C than their peers with subclinical ED symptoms.

Regarding agreement between youth and caregiver reports, findings replicate previous research regarding lack of concordance between youth and caregiver reports of child and adolescent mental health concerns (Achenbach, McConaughy, & Howell, 1987; De Los Reyes & Kazdin, 2005). In the present sample, correlations between youth and caregiver reports of depression (.57) and anxiety (.54) were moderate, positive, and significantly correlated. Caregivers reported significantly higher scores of depression and anxiety than adolescents. These findings also replicate previous research that has shown parents report higher levels of psychopathology than children and adolescents in clinical samples (Kristensen, 2001).

In addition to replicating previous findings, this study also adds new information. This study is unique in that it examines congruence between youth and caregiver reports of comorbid internalizing problems in families whose primary concern is obtaining
treatment for eating pathology. When severity of ED symptoms was taken into account, significant differences were found for youth with clinical ED symptoms and those with subclinical ED symptoms. There were no significant mean differences between youth and caregiver reports of major depression, panic disorder, separation anxiety, social phobia, obsessive compulsive disorder, or total anxiety (RCADS) for youth with clinically significant ED symptoms. Caregivers of youth with more severe ED symptoms exhibited more congruence with youth reports of depression and anxiety. On the other hand, there was a significant mean difference between youth and caregivers with scores below the clinical threshold on the EDE_Q for major depression, generalized anxiety, social phobia, and total anxiety. Caregivers of youth with subclinical ED symptoms reported significantly higher levels of depression and anxiety than youth. Interpretation of this discrepancy between youth and caregivers is not obvious. One explanation may simply be an underestimation of depression and anxiety by youth. Another reason may be caregiver’s willingness to more easily attribute pathological behavior to depression and anxiety instead of complications of DE or ED. Berg-Nielsen, Vika, & Dahl (2003) argue that subjective psychological variables such as caregiver’s mental health or adolescent’s self-esteem may be useful in interpreting informant discrepancies. Although, examining these subjective variables was not possible in the present study, examining caregiver mental health, particularly their own depression and anxiety, as well as adolescent self-esteem may provide more insight into understanding the cause of these discrepancies.

The present study did examine the influence of objective variables, age and race, on congruence between reports and found higher levels of agreement between caregivers
and older adolescents (ages 16-18) than younger adolescents (ages 12-15). Significant mean differences exist between younger adolescents and caregiver reports of major depression, social phobia, generalized anxiety, and total anxiety, with caregivers reporting higher scores than youth. These findings add to the inconsistencies present in previous research regarding the influence of age on informant discrepancies.

Achenbach’s seminal study found that agreement between informants was higher for younger children compared to older children (Achenbach, McConaughy, & Howell, 1987). However, several studies have failed to replicate this finding or found the reverse (Grills & Ollendick, 2003). De Los Reyes & Kazdin (2005) argue that these inconsistent findings regarding age and discrepancies may be due in part to inconsistencies in the methods used to examine age across studies. Many studies that failed to find an age effect have often used smaller samples which significantly limits the statistical power available to detect age effects. Some studies also limit their sample to youth age 16 and below and as a result create older and younger age groupings with limited variability (De Los Reyes & Kazdin, 2005). The current study did utilize a smaller sample and categorized age into two groupings, older (16-18) and younger (12-15). However, the current study did find an age effect with older youth having higher levels of agreement than younger children. One reason for this may be the ability of older adolescents to effectively express their internalizing symptoms to their caregivers in a way that is easily understood. Younger adolescents may not be able to articulate low mood or increased anxiety. As a result, caregivers may interpret their behaviors as boredom, fatigue, anger, or adolescent moodiness rather than signs of depression or anxiety.
Few studies have examined the relationship between informant discrepancies and racial identity and those that have focus mostly on White and African-American families. Although some have identified lower agreement among African-American families (Roberts, Alegria, Roberts, & Chen, 2005; Walton, Johnson, & Algina, 1999), most have found no difference in agreement based on race (Duhig, Renk, Epstein, & Phares, 2000). A study examining caregiver and youth reports of youth’s quality of life found a higher level of agreement between Black adolescents and their care providers than white adolescents (Pratt, Lamson, Lazorick, Swanson, Cravens, & Collier, 2011). When race was considered in the current study, a significant mean difference was found between white caregivers and youth when reporting major depression and social phobia only. This difference was not found for non-White caregivers. Sample characteristics suggest that this may be a sample specific finding and may not be generalizable to other adolescent populations. Racial information for this sample was particularly homogenous. The majority of the sample was White and no ethnicity information was made available. The number of non-White families in this sample was small therefore limiting the statistical power present to detect any race effects. Given the lack of significant racial differences in previous research, replication of these findings is necessary to ensure confidence in this particular clinical population. Because the current sample was primarily white it may be more interesting to examine differences in agreement based upon socioeconomic status (SES) instead of race. The population that obtains treatment from the clinic, and ED treatment in general, is primarily White and middle to upper middle class. Future research may benefit by examining differences in youth and caregiver agreement in relation to SES.
Limitations

One important limitation of this study was the small sample size (n=49 caregiver/youth dyads). Although more than 49 families were assessed during the time period, missing or incomplete data resulted in losing half of the potential families. Although small, this sample size did allow for the examination of agreement or discrepancies among youth and caregiver reports of internalizing symptoms and is similar to samples used in previous research studies. However, the sample size did not limit our ability to examine differences based on important contextual factors such as age or race due to lack of variability. In addition, due to restrictions in accessing administrative data, we were unable to obtain information regarding patient’s initial diagnosis, level of care recommendation, or clinician assessment. This additional information would have been useful in providing a more holistic picture of psychopathology and may have accounted for potential underreporting of symptoms by youth and/or caregivers.

Clinical Implications

The urban pediatric hospital’s eating disorder clinic provides a family based treatment approach to eating disorders and uses an interdisciplinary multiaxial assessment process. Based on my results, I believe several key findings are important for clinicians to consider during the assessment and subsequent treatment process. First, caregivers of youth seeking ED treatment often report higher levels of depression and anxiety symptoms than youth themselves, especially when the patient is a younger adolescent. It is important that providers do not dismiss youth reports but obtain information from both youth and caregivers, as well as complete their own clinical assessment of comorbid depression anxiety in adolescents seeking treatment in order to obtain a more holistic
understanding of the client’s internalizing problems. Second, note that youth, and caregivers of youth, with more severe ED symptoms are more likely to agree on the presence of significant anxiety and depressive disorders. This agreement between family members may provide providers with a source of leverage to encourage treatment uptake for youth and caregivers who desire a reduction in depression and anxiety symptoms even if they are unsure of motivation to change eating behaviors. Third, comorbid depression and anxiety is common among adolescents seeking ED treatment. It is important to consider the ways in which providers can incorporate interventions that address these comorbid symptoms that may or may not be exacerbated by ED behaviors. It is also important to educate families around issues of internalizing problems and acknowledge the fact that these comorbid symptoms may persist even after eating concerns are resolved.

Research Recommendations

Family based treatment for eating disorders has been demonstrated to be an effective approach to promoting recovery from DE and ED for adolescents (Wallis et al., 2012; Lock & Le Grange, 2010). It is important that researchers use appropriate measures (such as the EDE-Q and RCADS) to examine ED symptoms and other psychological comorbidities at initial assessment, over the course of treatment, and longitudinally after end of treatment. It also important that researchers examine the role of the family in their understanding of the adolescent’s ED and comorbid symptoms and the way in which family member’s perceptions may hinder or promote recovery. I argue that tracking caregiver-youth discrepancies longitudinally may provide clinicians and researchers insight into the process of relapse and recovery that is common among ED patients.
Future research could examine whether families with higher levels of agreement over time demonstrate a higher likelihood of sustained recovery than those families with lower levels of agreement. Future research could also examine the influence of subjective variables such as caregiver mental health and family functioning on informant discrepancies and treatment outcomes among families involved in ED treatment.
### Table 4.1 Comparison of Full Sample vs. Analytic Sample on Key Variables

<table>
<thead>
<tr>
<th>Sample</th>
<th>Full (N=99)</th>
<th>Analytic (n=49)</th>
<th>Omitted (n=50)</th>
<th>p value</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>14.9 (1.6)</td>
<td>15.1 (1.6)</td>
<td>14.8 (1.6)</td>
<td>.805</td>
<td>t-test</td>
</tr>
<tr>
<td>% White</td>
<td>89.9</td>
<td>86.8</td>
<td>93.8</td>
<td>.242</td>
<td>chi</td>
</tr>
<tr>
<td>% Female</td>
<td>91.9</td>
<td>93.9</td>
<td>96.1</td>
<td>.715</td>
<td>square</td>
</tr>
<tr>
<td>EDE-Q global score, mean (SD)</td>
<td>2.4 (1.8)</td>
<td>2.2 (1.8)</td>
<td>2.6 (1.9)</td>
<td>.355</td>
<td>fisher</td>
</tr>
<tr>
<td>Restraint, mean (SD)</td>
<td>2.0 (1.9)</td>
<td>2.0 (1.9)</td>
<td>2.0 (2.1)</td>
<td>.921</td>
<td>t-test</td>
</tr>
<tr>
<td>Eating concern, mean (SD)</td>
<td>1.8 (1.6)</td>
<td>1.7 (1.6)</td>
<td>2.0 (1.7)</td>
<td>.374</td>
<td>t-test</td>
</tr>
<tr>
<td>Shape concern, mean (SD)</td>
<td>3.0 (2.2)</td>
<td>2.8 (2.1)</td>
<td>3.2 (2.3)</td>
<td>.321</td>
<td>t-test</td>
</tr>
<tr>
<td>Weight concern, mean (SD)</td>
<td>2.7 (2.0)</td>
<td>2.4 (2.0)</td>
<td>2.9 (2.1)</td>
<td>.215</td>
<td>t-test</td>
</tr>
<tr>
<td>Depression Severity, mean (SD)</td>
<td>12.0 (6.4)</td>
<td>10.4 (5.7)</td>
<td>12.6 (6.0)</td>
<td>.195</td>
<td>t-test</td>
</tr>
<tr>
<td>Patient Anxiety, mean (SD)</td>
<td>53.3 (16.4)</td>
<td>55.7 (16.3)</td>
<td>44.6 (14.4)</td>
<td>.024</td>
<td>*t-test</td>
</tr>
<tr>
<td>Patient Major Depression, mean (SD)</td>
<td>60.9 (17.9)</td>
<td>63.0 (18.2)</td>
<td>53.5 (15.2)</td>
<td>.082</td>
<td>t-test</td>
</tr>
<tr>
<td>Caregiver Report Anxiety, mean (SD)</td>
<td>60.4 (15.7)</td>
<td>60 (15.8)</td>
<td>62.3 (16.1)</td>
<td>.646</td>
<td>t-test</td>
</tr>
<tr>
<td>Caregiver Major Depression, mean (SD)</td>
<td>72.6 (16.4)</td>
<td>71.9 (15.8)</td>
<td>74.9 (18.5)</td>
<td>.531</td>
<td>t-test</td>
</tr>
</tbody>
</table>

*p ≤ .05 **p ≤ .01
Table 4.2 EDE-Q mean score compared to normative data for adolescents and young adults

<table>
<thead>
<tr>
<th>Mean (SD)</th>
<th>Global Score</th>
<th>Restraint</th>
<th>Eating Concern</th>
<th>Shape Concern</th>
<th>Weight Concern</th>
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</thead>
<tbody>
<tr>
<td>Current Study</td>
<td>2.2(1.8)</td>
<td>2.0(1.9)</td>
<td>1.7(1.6)</td>
<td>2.8(2.1)</td>
<td>2.4(2.0)</td>
</tr>
<tr>
<td>Young adolescent females (12-14)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Carter, Stewart, &amp; Fairburn (2001) (N=808)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.6 (1.4)</td>
<td>1.4(1.5)</td>
<td>1.0(1.0)</td>
<td>2.2(1.7)</td>
<td>1.8 (1.7)</td>
<td></td>
</tr>
<tr>
<td>Adolescent females (12-18)</td>
<td>1.8 (1.5)</td>
<td>1.5 (1.6)</td>
<td>1.2 (1.4)</td>
<td>2.5 (1.9)</td>
<td>1.2 (1.8)</td>
</tr>
<tr>
<td>Adolescent males (12-18) c</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>.61 (.86)</td>
<td>.54 (.97)</td>
<td>.4 (.72)</td>
<td>.8 (1.2)</td>
<td>.67 (1.05)</td>
<td></td>
</tr>
</tbody>
</table>

a Carter, Stewart, & Fairburn (2001) (N=808)

b Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis (2014) (n= 1134)

c Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis (2014) (n= 530)
<table>
<thead>
<tr>
<th>Normative Data</th>
<th>EDE-Q Subscale</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Global Mean Difference</td>
<td>Restraint Mean Difference</td>
<td>Eating Concern Mean Difference</td>
<td>Shape Concern Mean Difference</td>
<td>Weight Concern Mean Difference</td>
</tr>
<tr>
<td>Young adolescent females (12-14) a</td>
<td>.60**</td>
<td>.60**</td>
<td>.70**</td>
<td>.60*</td>
<td>.60*</td>
</tr>
<tr>
<td>Adolescent females (12-18) b</td>
<td>.40**</td>
<td>.50*</td>
<td>.50*</td>
<td>.30</td>
<td>1.2**</td>
</tr>
<tr>
<td>Adolescent males (12-18) c</td>
<td>1.59**</td>
<td>1.5**</td>
<td>1.3**</td>
<td>2.0**</td>
<td>1.73**</td>
</tr>
</tbody>
</table>

* a Carter, Stewart, & Fairburn (2001) (N=808)
  b Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis (2014) (n= 1134)
  c Mond, Hall, Bentley, Harrison, Gratwick-Sarll, K., & Lewis (2014) (n= 530)
  *p<.05  **p<.01  ***p< .07  *p< .10
<table>
<thead>
<tr>
<th>Depression Severity</th>
<th>Percent (Frequency)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>24% (12)</td>
</tr>
<tr>
<td>Mild</td>
<td>31% (15)</td>
</tr>
<tr>
<td>Moderate</td>
<td>29% (14)</td>
</tr>
<tr>
<td>Severe</td>
<td>12% (6)</td>
</tr>
<tr>
<td>Very Severe</td>
<td>4% (2)</td>
</tr>
</tbody>
</table>

a. Depression severity was measured using the QIDS_SR 16 and severity scores were based on the following categorization: None (0-5), Mild (6-10), Moderate (11-15), Severe (16-20), and Very Severe (21-27).
Table 4.5 Comparison of youth and caregiver reports of clinically significant depression and anxiety

<table>
<thead>
<tr>
<th></th>
<th>Youth</th>
<th>Caregiver</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Depression</td>
<td>33% (16)</td>
<td>45% (22)</td>
</tr>
<tr>
<td>Total Anxiety Scale</td>
<td>20% (10)</td>
<td>33% (16)</td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>25% (12)</td>
<td>33% (16)</td>
</tr>
<tr>
<td>Separation Anxiety</td>
<td>22% (11)</td>
<td>20% (10)</td>
</tr>
<tr>
<td>Generalized Anxiety</td>
<td>10% (5)</td>
<td>25% (12)</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>16% (8)</td>
<td>27% (13)</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>10% (5)</td>
<td>8% (4)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>1. EDE-Q, Global</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Restraint</td>
<td></td>
<td>.87**</td>
</tr>
<tr>
<td>3. Eating concern</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Shape concern</td>
<td></td>
<td>.96**</td>
</tr>
<tr>
<td>5. Weight concern</td>
<td></td>
<td>.95**</td>
</tr>
<tr>
<td>6. Depression Severity (QIDS)</td>
<td></td>
<td>.70**</td>
</tr>
<tr>
<td>7. Youth Total Anxiety</td>
<td>.19</td>
<td>.22</td>
</tr>
<tr>
<td>8. Youth MDD</td>
<td>.18</td>
<td>.18</td>
</tr>
<tr>
<td>9. Caregiver Report Anxiety</td>
<td>.16</td>
<td>.14</td>
</tr>
</tbody>
</table>

*p<.05  **p<.01
Table 4.7. Mean Differences in self-reported anxiety and depression based on ED severity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Global EDE – Q &lt; 4</th>
<th>Global EDE-Q ≥ 4 a</th>
<th>Mean Difference</th>
<th>T(48)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression Severity (QIDS)</td>
<td>8.6</td>
<td>17.3</td>
<td>-8.7</td>
<td>-5.46</td>
<td>.000**</td>
</tr>
<tr>
<td>Youth Total Anxiety</td>
<td>53.6</td>
<td>64.2</td>
<td>-10.6</td>
<td>-1.89</td>
<td>.064*</td>
</tr>
<tr>
<td>Youth MDD</td>
<td>60.3</td>
<td>73.4</td>
<td>-13.1</td>
<td>-2.1</td>
<td>.041*</td>
</tr>
<tr>
<td>Caregiver Report Anxiety</td>
<td>58.8</td>
<td>64.4</td>
<td>-5.6</td>
<td>-0.99</td>
<td>.325</td>
</tr>
<tr>
<td>Caregiver Report MDD</td>
<td>70.3</td>
<td>77.9</td>
<td>-7.6</td>
<td>-1.36</td>
<td>.179</td>
</tr>
</tbody>
</table>

*a Global EDE-Q ≥ 4 is above the clinical threshold for ED symptoms

Note: Scores on the RCADS anxiety and MDD scales (both parent and child) 70 or higher are considered above the clinical threshold

*p < .05  **p < .01  + p < .07
Table 4.8. Mean Differences in youth and caregiver perceptions of anxiety and depression

<table>
<thead>
<tr>
<th>Scale</th>
<th>Youth Mean</th>
<th>Caregiver Mean</th>
<th>Mean Difference</th>
<th>T(48)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Depression</td>
<td>63.0</td>
<td>71.9</td>
<td>8.9</td>
<td>-3.93</td>
<td>.000**</td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>60.9</td>
<td>64.7</td>
<td>3.8</td>
<td>-1.32</td>
<td>.192</td>
</tr>
<tr>
<td>Separation Anxiety</td>
<td>57.1</td>
<td>56.4</td>
<td>.69</td>
<td>.433</td>
<td>.667</td>
</tr>
<tr>
<td>Generalized Anxiety</td>
<td>48.6</td>
<td>58</td>
<td>9.4</td>
<td>-4.59</td>
<td>.000**</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>53.7</td>
<td>57.9</td>
<td>4.2</td>
<td>-2.08</td>
<td>.043*</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>50.5</td>
<td>52.4</td>
<td>1.9</td>
<td>-1.00</td>
<td>.322</td>
</tr>
<tr>
<td>Total Anxiety</td>
<td>55.7</td>
<td>60.0</td>
<td>4.2</td>
<td>-1.93</td>
<td>.060*</td>
</tr>
</tbody>
</table>

*p<.05 **p<.01 + p< .07
Table 4.9 Comparison of depression and anxiety scores between youth and caregiver by age, race, and eating disorder symptoms

<table>
<thead>
<tr>
<th>Variable</th>
<th>RCADS Subscale</th>
<th>MDD Mean Difference (SD)</th>
<th>Panic Mean Difference (SD)</th>
<th>Separation Mean Difference (SD)</th>
<th>GAD Mean Difference (SD)</th>
<th>SP Mean Difference (SD)</th>
<th>OCD Mean Difference (SD)</th>
<th>Total Anxiety Mean Difference (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;16</td>
<td></td>
<td><strong>-12.8 (15.8)</strong> **</td>
<td>-3.9 (21.8)</td>
<td>-2.2 (10.6)</td>
<td>**-9.7 (13.1) **</td>
<td>**-7.0 (16.0) **</td>
<td>-3.6 (13.3)</td>
<td>-6.4 (17.0) **</td>
</tr>
<tr>
<td>≥16</td>
<td></td>
<td>-4.2 (16.7)</td>
<td>-3.7 (18.8)</td>
<td>4.2 (11.2) **</td>
<td>**-9.0 (16.1) **</td>
<td>-.73 (10.5)</td>
<td>.05 (14.0)</td>
<td>-1.5 (13.1)</td>
</tr>
<tr>
<td><strong>ED symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global EDE-Q &lt; 4</td>
<td></td>
<td>**-10.1 (16.4) ** **</td>
<td>-4.6 (21.1)</td>
<td>.13 (11.2)</td>
<td>**-10.4 (15.6) **</td>
<td>**-4.8 (14.6) **</td>
<td>-2.2 (13.0)</td>
<td>-5.3 (16.2) **</td>
</tr>
<tr>
<td>Global EDE-Q ≥4</td>
<td></td>
<td>-4.5 (13.5)</td>
<td>-.8 (17.6)</td>
<td>2.9 (1.7)</td>
<td>**-5.5 (7.4) **</td>
<td>-1.7 (11.9)</td>
<td>-.9 (16.3)</td>
<td>-.2 (11.6)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td></td>
<td>**-9.5 (15.4) ** **</td>
<td>-3.8 (21.4)</td>
<td>.74 (11.6)</td>
<td>**-8.8 (14.0) **</td>
<td>**-4.3 (13.3) **</td>
<td>-1.0 (13.5)</td>
<td>-3.8 (15.4)</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>-5.4 (19.6)</td>
<td>-4.1 (12.3)</td>
<td>.43 (9.3)</td>
<td>**-13.3 (17.2) **</td>
<td>-3.3 (19.4)</td>
<td>-7.3 (13.0)</td>
<td>-6.7 (16.4)</td>
</tr>
</tbody>
</table>

*p < .05 **p < .01 †p < .07 ‡p < .10
Chapter 5. Conclusions

Eating disorders (ED) are a complex and pervasive mental health condition. Adolescents struggling with an ED often contend with a number of difficulties besides eating. Given the high dropout and relapse rates seen in adolescent eating disorder treatment (Fassino, Pierò, Tomba, & Abbate-Daga, 2009), it is important to understand the various factors that influence symptom presentation and prognosis. Research has identified various biological, psychological, and social factors that are associated with EDs (le Grange, 2016). This dissertation sought to further examine the biopsychosocial factors that influence ED symptoms and treatment outcomes among adolescent patients and provide new insight into the ways in which the factors work separately and collectively to influence overall well-being.

Findings

In chapter 2, I identify the prevalence of trauma in a clinical sample of adolescents receiving outpatient treatment for an eating disorder. Over one-third of the sample reported exposure to one or more traumatic events during their lifetime. Thirty percent reported symptoms of PTSD and over 70% reported symptoms of depression and anxiety. Lifetime trauma was a significant predictor of weight status across all three logistic regression models presented. Specifically, the presence of trauma significantly reduced the odds of being underweight. This finding provided support for hypothesis 1, there was a significant association between trauma and ED presentation. A significant
relationship was also found between lifetime trauma exposure and the presence of psychiatric comorbidities (PTSD, anxiety, and depression) supporting hypothesis 2. My results replicated existing research identifying a significant relationship between trauma exposure, particularly sexual abuse, and bulimia nervosa (Brewerton, 2007; Wonderlich et al., 2000; Johnson, Cohen, Kasen, & Brook, 2002). After controlling for demographic and comorbid symptoms, the presence of lifetime trauma reduced the likelihood of being underweight by approximately half. Results from multinomial regression models suggest that while trauma may be an important predictor in determine low vs high weight, it is unable to determine more specific weight categories. These specific categories are better explained by additional demographic and psychological variables. From this chapter it is apparent that lifetime trauma exposure is common among adolescents seeking ED treatment. Trauma exposure is related to initial weight status, bulimic symptoms, and psychiatric comorbidities. The clinical implications of these findings are that it is imperative that providers conduct a comprehensive trauma assessment when working with clients with EDs and tailor interventions to address comorbid PTSD, anxiety, and depression when present. Although chapter 2 provides useful information on the prevalence and influence of trauma on the initial presentation of ED symptoms it does not provide information on the predictive ability of other biopsychosocial factors on ED symptom presentation. These were further explored in chapter 3.

Chapter 3 examined biological (sex, family history of ED), psychological (depression, anxiety, OCD, and PTSD), and social (trauma exposure) factors associated with early weight restoration in a clinical sample of adolescents who received outpatient treatment for anorexia nervosa (AN). This chapter had two main research questions, 1)
What percentage of patients obtain early weight restoration, and 2) What factors increase or decrease the likelihood of obtaining one’s %EBW at 3 month follow up?

Approximately half of patients (51%) reached weight restoration after 3 months of beginning treatment. Alone, comorbid depression was found to be a marginally significant predictor of weight restoration but this effect diminished when additional factors were added to the model. Only comorbid anxiety was found to be a significant predictor of weight restoration at time 2 across all three models. Adolescents with comorbid anxiety were three times more likely to be weight restored than their peers without the comorbid diagnosis. This finding is in contrast with previous research that suggest that anxiety is a negative prognostic factor for recovery (Zerwas et al., 2013; Yackobovitch -Gavan et al., 2009; Dellava et al., 2010). As a result, I present two hypotheses, one individual and one systemic, about why this positive relationship exists. Although we can only speculate on the nature of the relationship between anxiety and weight restoration in this study, findings do confirm the fact that comorbid anxiety is of significant clinical interest for patients with AN. Despite the small sample size and limited findings in the current study, I argue that we should continue to examine the ability of anxiety and other psychiatric comorbidities, such as depression, to delineate between ED patients with various clinical presentations. For this reason, chapter 4, examines the presence and perception of comorbid anxiety and depression in a sample of adolescents and their families seeking initial assessment for ED treatment.

Chapter 4 investigates the differences between youth and caregiver reports of comorbid anxiety and depression in a sample of families seeking ED treatment.

Approximately one third of the sample reported comorbid major depression and one-fifth
reported a comorbid anxiety disorder. More severe depression and higher total anxiety scores were associated with having ED symptoms above the clinical threshold (support for hypothesis 1). Youth and caregiver reports of depression and anxiety were significantly discrepant. Caregiver and youth reports of depression and anxiety were moderately positively correlated. Overall findings supported hypothesis 2 with caregivers reporting higher levels of anxiety and depression symptoms than youth. These findings replicate previous research regarding discrepancies in reports of internalizing symptoms in clinical and ED samples (Kristensen, 2001; Salbach-Andrae et al., 2008). Families of youth with clinical ED symptoms endorsed higher levels of agreement when reporting ED symptoms. In contrast, families of youth with subclinical ED symptoms endorsed significantly higher levels of disagreement. These result confirmed hypothesis 3 – caregivers of youth with more severe ED symptom will exhibit more congruence. Younger age and race were also found to be significantly positively related to disagreement between youth and caregiver reports of depression and anxiety. In light of these findings, it is important that service providers are aware that youth and caregivers’ reports are often discrepant and utilize both reports in addition to their own clinical assessment to make recommendations for treatment and develop necessary interventions. It is also important to note that families of youth with more severe ED symptoms appear to be in agreement regarding comorbid psychological concerns which may be beneficial in promoting treatment uptake, fostering motivation to change, and handling resistance throughout the process of treatment.
Limitations

Findings in this dissertation highlight the usefulness of a biopsychosocial (BPS) approach to understanding factors associated with initial presentation and prognosis in adolescents seeking ED treatment. It is important to note that although the BPS model is called a model it is more of a framework for making sense of illness and recovery not predicting causality. My ability to examine a multitude of biopsychosocial factors was limited given the data available for this study. However, I was able to examine sex and family history of ED (biological), anxiety, depression, OCD, and PTSD symptoms (psychological), and trauma exposure (social). Results from all three studies revealed significant psychological (depression and anxiety) and social factors (trauma exposure) associated with ED. No biological factors (sex, family history of ED) were found to be significant predictors of symptoms or treatment outcomes. Nevertheless, the BPS model provided a comprehensive, systemic framework that allowed for the creation and testing of hypothesis and research questions that acknowledged the multiple factors that influence ED symptoms, severity, and outcomes. Due to limitations, I was not able to complete an extensive BPS examination of the reciprocal influence of multiple factors on ED symptoms and outcomes over time. A discussion of major limitations is presented below.

Sample Size.

The most prominent limitation across all three studies in this dissertation is the small sample sizes. Although chapter 2 had a final analytic sample of 205, chapters 3 and 4 had 61 and 49 participants, respectively. Due to the nature of clinical research and the population of interest, smaller sample sizes were expected. However, across all three
studies, one third to one-half of the sample was omitted due to missing data. This reduction in sample size significantly limited the statistical power available to detect significant effects and limited the variables available to explore the BPS model. For example, in chapter 3, family history of psychiatric disorders was presented as an important biological factor that could potentially predict ED symptoms and treatment outcomes. However, this variable was subsequently dropped due to missing data.

**Lack of diversity.**

The lack of diversity across samples is also noted as an important limitation. Data from all three research chapters was pulled from one Midwestern ED clinic. This clinic’s treatment population is predominantly white, female, and middle to upper middle class. As a result, the samples in this dissertation were predominantly white and female. No ethnicity or SES information was available for these subjects. As a result, this study is unable to contribute to the limited research base focused on non-White and/or male adolescents with eating disorders. Research on eating disorders among ethnic minority populations suggest that acculturative stress, the stress of moving from one culture to another, may be an important factor in moderating and or predicting ED symptoms and DE behaviors (Van Diest, Tartakovsky, Stachon, Pettit, & Perez, 2014). Van Diest and colleagues (2014) found that the acculturative stress significantly moderated the relationship between body dissatisfaction and ED symptoms among young undergraduate African-American women. In addition, acculturative stress significantly predicting bulimic symptoms among African American, Asian, and Latina women, even after controlling for general life stress (Van Diest, Tartakovsky, Stachon, Pettit, & Perez, 2014). During adolescence, ethnic minority youth may feel increasing pressure to
acculturate to the dominant society in order to develop a socially acceptable identity. In turn, this pressure may increase their risk for engaging in ED behaviors. Future research on ED should focus on the ways in factors such as race and ethnicity, acculturation, and racism may impact the presentation of ED symptoms among adolescents.

**Lack of family and environmental context.**

Adolescents are influenced significantly by their family and their environment. Unfortunately, this dissertation was unable to fully examine the influence of the family and larger social/environment factors on ED symptoms and treatment outcomes. First, youth-caregiver relationship quality is an important factor that would have been useful when examining ED symptoms and differential reports of adolescent mental health concerns. Previous research suggests that adolescents who report more positive relationships with their parents had lower odds of engaging in disordered eating behavior (Haines et al., 2016). Preliminary evidence has also been presented for the longitudinal association between attachment to parental figures and eating pathology (Goossens, Braet, Van Durme, Decaluwé, & Bosmans, 2012). Insecure attachment toward mothers was associated with increased dietary restraint, weight, and shape concern, while insecure attachment toward fathers was predictive of persistence in binge episodes (Goossens, Braet, Van Durme, Decaluwé, & Bosmans, 2012). Second, general family functioning might also be a significant mediator in understanding the influence of certain BPS factors on prognosis over time, especially when utilizing a family based treatment approach. A systematic review found that families with a loved one who has an eating disorder report worse family functioning than control families (Holtom-Viesel & Allan, 2014). In addition, patients who had more positive perceptions of their family functioning
had better treatment outcomes than those with more negative perceptions (Holtom-Viesel & Allan, 2014). A protective relationship has been found between family functioning and DE, such that higher family functioning significantly reduces the likelihood of engaging in DE behaviors in adolescents (Bege, Wall, Larson, Eisenberg, Loth & Neumark-Sztainer, 2014). Third, social/environmental factors such as individual and family stress and SES are also important to consider when examining treatment outcomes or level of agreement between youth and caregivers.

**Research Recommendations and Clinical Implications**

Existing research has demonstrated that eating pathology is common among adolescents (Croll, Neumark-Stainer, Story, Ireland, 2002; Torstveit, Aagedal-Mortensen, & Stea, 2015). Given the complex nature of eating disorders and the increased risk for the development of mental health concerns in adolescence, I argue that comorbid problems should be an area of great consideration within this population. Research focusing on comorbid depression and anxiety among children and adolescents suggests that depression and anxiety may be able to differentiate between ED symptom presentation and treatment outcomes (Hughes, Goldschmidt, Labuschagne, Loeb Sawyer, & Grange, 2013). Findings from chapter 4 suggest that clients with more severe ED symptoms also present with more severe depression and anxiety. Directing our clinical and scholarly attention toward the influence of comorbid symptoms may provide us with quality information about the nature of the presenting problem and the process of recovery than focusing solely on eating pathology.

Trauma was found to be an important social/environmental factor that was associated with bulimia nervosa (BN). Based on previous research (Carter, Bewell,
Blackmore, & Woodside, 2006; Brewerton, Dansky, O’Neil, & Kilpatrick, 2015), I posit that trauma exposure is associated with a range of behaviors characteristic of BN, such as purging and binge eating behaviors. In addition, findings from this study provide further confirmatory evidence that trauma exposure is associated with comorbid psychological problems in adolescents with ED. I believe it is important for service providers to complete a comprehensive assessment, including trauma history, in patients who present with these behaviors.

The question that follows from these findings is, how do we tailor treatment of adolescent eating disorders to address additional mental health concerns? Cognitive behavior therapy (CBT) and family based treatment (FBT) have been cited as producing the most effective outcomes for adolescents struggling with an ED (Fairburn, 2008; Dalle Grave, Calugi, Sartirana, & Fairburn, 2015; Wallis et al., 2012; Lock & Le Grange, 2010). However, research suggests that adolescents with particular comorbidities such as chronic anxiety, depression, or PTSD, may not respond in the same way as their peers without comorbid diagnoses (Wild et al., 2016; Sticker, 2013). Clinicians may desire to create customized treatment plans within more standardized forms of treatment (i.e. trauma focused treatment, treatment focusing on chronic depression or suicidality, etc.). In fact, some service providers are creating treatment modalities that focus on particular comorbid issues such as trauma or depression. However, the research base on the effectiveness of these focused programs is lacking and needs to be bolstered. Findings from chapter 3 suggest that comorbid anxiety is a positive prognostic factor for early weight restoration in patients with AN. Based on the systemic hypothesis presented chapter 3, I argue that the treatment program was set up in such a way to help those
patients succeed, focusing on the restrictive over anxious adolescent. If this is true, this may provide some evidence for the benefit of tailoring interventions toward particular comorbid conditions.

To date, this dissertation includes the second study to examine the level of agreement between youth and caregivers regarding other mental health problems in adolescents seeking ED treatment. This study confirms the findings provided by Salbach-Andrae and colleagues (2008) and provides additional information regarding the influence of eating disorder severity on levels of concordance. Little research is available examining caregivers’ perceptions of ED symptoms. What is available suggests that caregivers and youth often report different sets of ED symptoms (Swanson et al., 2014). No data exists on how discrepancies in reports of ED symptoms and/or comorbid diagnoses in patients with ED may influence treatment outcomes over time. If families of youth with more severe ED symptoms are not discrepant (see chapter 4) perhaps this suggests everyone is on the same page regarding the impact of the disorder. Discrepancies may not speak to whose right or wrong, but may provide valuable information regarding the impact of the illness on family functioning and willingness to participate in treatment.

**Implications for Adolescent Development**

Adolescence is a developmental period that is marked by numerous biological, cognitive, social, and emotional changes. It is also a period in which many mental health disorders emerge and influence the ways in which the adolescent interacts with their environment (Merikangas, & He, 2014). Adolescents and families who struggle with an ED diagnosis face unique challenges as a result of this illness; however, these families
also continue to face the challenges that are characteristic of this period in the life span such as handling biological changes, educational transitions, identity development, role expectations, and developing sexuality (Bandura, 2006). It is clear that experiencing a mental health disorder, such as an ED along with other psychiatric comorbidities such as depression, anxiety, or PTSD, during adolescence increases the risk of lower quality of life and general well-being during adolescence and young adulthood (Sawyer, Whaites, Rey, Hazell, Graetz, & Baghurst, 2002; McCloughen, Foster, Huws-Thomas, & Delgado, 2012). However, research suggests that early resolution and intervention during adolescence can prevent morbidity during adulthood (Patton et al., 2014). Despite the complex nature of ED, intervention and resolution of ED and other comorbidities may provide adolescents and young adults with a greater sense of personal self-efficacy and identity development, such that they are more confident in their ability to achieve their future goals despite challenges.
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