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by

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DEDICATION

To all those who were abused as children, especially those with whom I have had the privilege of working: may you find strength even amidst your pain.

"To laugh often and love much; to win the respect of intelligent persons and the affection of children; to earn the approbation of honest citizens and endure the betrayal of false friends; to appreciate beauty; to find the best in others; to give of one's self; to leave the world a bit better, whether by a healthy child, a garden patch or a redeemed social condition; to have played and laughed with enthusiasm and sung with exultation; to know even one life has breathed easier because you have lived—this is to have succeeded."

-Bessie Anderson Stanley

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TABLE OF CONTENTS

List of Tables

List of Figures

Chapter One

Introduction

At both clinical and sub-clinical levels, women are more concerned with weight and thinness than ever before. Ostensibly, it should come as no surprise that by association eating disorders, body dissatisfaction, and pathological attitudes about thinness have become more widespread and have gained increasing attention in recent years. McGrane and Carr (2002) reported that anorexia and bulimia occur in about 1% and 3% of female adolescents, respectively. Considering that of all psychiatric disorders, eating disorders have the highest morbidity rates and are the largest source of premature death (Arcelus, 2007; Berkman, Lohr, & Bulik, 2007; Keel, Dorer, Eddy, Franko, Charatan, & Herzog, 2003; Steinhausen, 2002), it would appear to be imperative that clinicians make active efforts to understand how and why eating disorders develop so that effective treatments can be devised to ameliorate these serious mental health problems. Consistent with this, extensive research has been done to date and there are now a plethora of theories and interventions aimed at addressing the symptoms and their supposed causes.

Despite the burgeoning research and clinical knowledge around the etiology of eating disorders, specifically Anorexia Nervosa, Bulimia Nervosa, and Eating Disorder Not Otherwise Specified (EDNOS), remains murky and the focus of intense debate. For instance, some researchers assert that Anorexia Nervosa and Bulimia Nervosa are fundamentally different disorders whereas other researchers have stated that all eating disorders follow similar etiological paths. Other investigators have asserted that eating

disorders exist on a continuum, with normative eating behavior at one end and the pattern of behaviors, which meets DSM-IV-TR criteria for eating disorders at the other end (Cachelin, Striegel-Moore, & Paget, 1997; Mintz & Betz, 1988; Stice, Killen, Hayward, & Taylor, 1998; Tylka & Subich, 1999). Other studies have supported a discontinuum hypothesis, suggesting that there is a qualitative difference between people with "true" eating disorders and those displaying subclinical disordered eating behaviors (Gleaves, Lowe, Snow, Green, & Murphy-Eberenz, 2000; Ruderman & Besbeas, 1992). And still other researchers who have completed taxometric analyses have suggested that with binge eating behaviors, qualitative differences exist between clinical versus non-clinical individuals, but that the symptoms of anorexia exist more on a continuum (e.g. Williamson et al., 2002).

For the purpose of this dissertation, traits of disordered eating were investigated based on the aforementioned continuum hypothesis. This study investigated what is known as disordered eating, defined as the broad presence of maladaptive thoughts and behaviors related to eating which in and of themselves may not manifest in clinically significant dysfunction but if existing at a high enough frequency and severity combined with other symptoms would be considered symptoms of clinical disorders as per the DSM-IV-TR. Research indicates that eating disorder symptoms appear to be complex and multi-determined (Levitt & Sansone, 2007) and that women who do not meet full diagnostic criteria but instead demonstrate subclinical eating problems nonetheless experience significant distress (Lowe et al., 1996), including low self-esteem (Mintz & Betz, 1988), neuroticism (Tylka & Subich, 1999), anxiety, and depression (Stice et al. 1998). Extending from this, investigations aimed at identifying and preventing disordered eating

may prove invaluable in not only bolstering current understanding of the causes of eating disorders proper but also in helping clinicians to identify and intervene before disordered eating manifests as clinically significant pathology (Mussell, Binford, & Fulkerson, 2000). Therefore, it is important to study eating disturbance in both clinical and nonclinical samples (Hund & Espelege, 2005).

The primary role of early trauma in the development of disordered eating

As noted earlier, extensive research has been conducted to identify causes of eating disorder symptoms. However, while such research has helped to identify candidate factors and agents, results across studies have been inconsistent (Allison, Grilo, Masheb, & Stunkard, 2007). Of the numerous factors studied, the relation of various forms of childhood maltreatment and trauma to different forms of eating disturbance has received a great deal of attention and for good reason-- Trauma, particularly childhood trauma, has been identified as a likely contributing factor in the development of eating disorders. For example, many studies of women with eating disorders have found that between 20 to 69% have histories of traumatic experience (Abrahamson & Lucido, 1991; Bulik, Sullivan, & Rorty, 1989; Curtis, Jason, Olson, & Ferrari, 2005; Folsom et al., 1993; Fonagy et al., 1996; Grave, Rigamonti, Todisco, & Oliosi, 1996; Hall, Tice, Beresford, Wooley, & Hall, 1989; Johnson, Cohen, Kasen, & Brook, 2002; Kaner, Bulik, & Sullivan, 1993; Lacey, 1990; Oppenheimer, Howells, Palmer, & Chaloner, 1985; O'Shaughnessy & Dallos, 2009; Palmer & Oppenheimer, 1992; Ringer & Crittenden, 2007; Root & Fallon, 1988; Sloan & Leichner, 1986; Tagey et al., 2010; Zachrisson & Kulbotten, 2006). Notwithstanding the apparent association of trauma to eating disorder symptomology, some investigators note that not all women with histories of trauma

develop eating disorders and argue that the link of early trauma and the emergence of eating disordered symptomology is likely to be complex (Allison, et al, 2005; Everill & Waller, 1995). This line of argumentation appears to make sense when one considers that other studies have identified factors that appear to be linked to disordered eating which may have moderating and mediating influences on the causal role that trauma plays. Such factors include PTSD, anxiety, depression, dissociation, attachment styles, family environment, body image (Root, 1991), and anxiety (e.g. Brewerton, Brandt, & Gold, 1990; Calam, Waller, Slade, & Newton, 1990; Cooper & Cowen, 2009; Davey & Chapman, 2009; Evans & Wertheim, 2005). Unfortunately, none of the research to date has examined all of these factors in a single study.

In light of this, the purpose of this dissertation was to examine critically the aforementioned factors and how they relate to the emergence of disordered eating symptoms in a non-clinical sample of women. More specifically, this dissertation utilized trauma theory as the basis for conceptualizing the relation of trauma to the development of disordered eating and ultimately diagnosable eating disorders. Trauma theory is based on the idea that repetitive exposure to abusive experiences in childhood is likely to increase the risk for general psychopathology in adulthood (Sansone & Sansone, 2007). Specifically, trauma theory suggests that repetitive exposure to abusive experiences in childhood is likely to increase the risk for eating pathology in adulthood. Several previous studies support this theoretical foundation, demonstrating that a significant proportion of women with eating disorders report a history of childhood abuse (Oppenheimer, Howells, Palmer, & Chaloner, 1985; Hall, Tice, Beresford, & Wooley, 1989; Abramson & Lucido, 1991).

With a basic directional model between trauma and disordered eating as the foundation, this dissertation aimed at testing this association while taking into account a variety of potential moderating and mediating variables that were hypothesized to affect this relationship. In particular, six moderators were considered, namely PTSD symptoms, affective symptoms, attachment styles, agent of abuse (family or nonfamily member), age at onset of trauma (before or after age 14), and chronicity of trauma (one incident v. multiple incidents). At the same time, dissociation, family dysfunction, and body dissatisfaction were studied as three mediating variables. It was the goal of this research project and the hopes of this researcher that this investigation would allow for a more integrated understanding of how and why the myriad of factors mentioned contribute to disordered eating and eating disorders.

Chapter Two

Review of Literature

Traumatic childhood experiences have long been theoretically linked to pathological eating behaviors in adulthood. Research linking the two has yielded mixed results, however, with studies demonstrating varying levels of correlations. Investigators have included a range of variables and attempted to demonstrate ways in which they influence outcomes and lead to various symptoms. Several theoretical models have been proposed to explain the data that have emerged from the vast number of studies conducted in this area. The research to date has focused on isolated variables rather than a comprehensive, and more integrated approach that includes a wide variety of relevant factors. As will be noted throughout this review, the latter is a long overdue approach to studying these phenomena. Throughout the following chapter, an extensive review of the previous literature is presented.

Eating Disorders

An eating disorder involves obsessions about food, weight, and body shape to the point where one's focus on eating or not eating significantly interferes with adaptive functioning. In terms of eating disorders, the Diagnostic and Statistical Manual- Fourth Edition, Text Revision (DSM-IV-TR; APA, 2000) describes Anorexia Nervosa (AN), Bulimia Nervosa (BN), and Eating Disorder Not Otherwise Specified (EDNOS). Anorexia Nervosa is characterized by a persistent fear of fatness, refusal to maintain at least 85% of what would be considered a healthy body weight, loss of menstruation in

females, and a disturbance in one's view of body shape or denial of one's current low body weight. Bulimia nervosa is characterized by repeated episodes of binge eating followed by purging, usually in the form of self-induced vomiting or excessive laxative use to avoid weight gain. EDNOS is a category of eating disorders that contains cases that do not fit the strict symptoms of either anorexia or bulimia as traditionally defined, but have a variety of symptoms related to eating that result in significant functional impairment. EDNOS is actually the most commonly diagnosed type of disordered eating and, as a result, the eating disorder diagnoses underwent a revision with the recent publication of DSM-5 (Machado, 2013). Examples may include significant food obsessions and restrictions without loss of menses or frequent binging without purging (APA, 2000). In addition, many women demonstrate symptoms of eating disorders such as restricting, food obsessions, weight obsessions, binging, or purging that do not cause significant impairments in functioning necessary for a clinical diagnosis. Researchers and clinicians often refer to sub-clinical levels of disordered eating symptomatology as disordered eating (Juarascio, Perone, & Timko, 2011).

Historically, research on eating pathology has focused primarily on adolescent and young adult women, and although this group remains at high risk, recent research has identified a significant group of preadolescent and middle-aged women who struggle with eating pathology (Lewinsohn, Striegel-Moore, & Seeley, 2000; Wilson, Becker, & Heffernan, 2003). Although a growing body of research suggests that men are increasingly struggling with eating dysfunction, preliminary research suggests that the etiology of eating disorders differs depending on gender (Beato-Fernandez & Rodriguez-

Cano, 2005; Orzeck, Rokach, & Chin, 2010; Klump et al., 2012). Thus, this paper focuses exclusively on women.

A variety of theories have been proposed to explain eating disorders, each emphasizing different factors (Rogers & Petrie, 2001). The first theorists were largely psychoanalytic. Freud (1949) asserted that loss of appetite reflected a loss of libido. In contrast, Waller et al. (1940) suggested that anorexia serves as a defense against fantasies of oral impregnation. Others stressed the separation-individuation crisis, suggesting that anorexia and bulimia reflect a fear of maturation and a rejection of adult sexuality (Bruch, 1973; Schmidt, Humfress, & Treasure, 1997). Early researchers believed that by maintaining a childish, body, women can unconsciously avoid both a feminine shape and the associated sexual attention (Bruch, 1973). Scott (1987) provided an extensive literature review of the separation-individuation crisis theories of eating disorders. Later psychoanalytic theorists focused on developmental deficiencies suggesting that eating disorders are a somatic form of neurotic conflicts (Sugarman & Lee, 1990).

Cognitive-behavioral models assert that a person's beliefs about being inadequate are connected to Western culture's values about thinness and reinforcements when weight loss is attempted (Garner & Bemis, 1982; Vitousek & Hollon, 1990). Feminist theories have argued that female roles and personalities promoted by society predispose women to maladaptive eating methods (Orbach, 1986). Family systems theories have pointed to a number of familial characteristics that might be organizing or maintaining factors for eating disorders. Minuchin, Rosman, and Baker (1978), for example, wrote about the families of teenagers with anorexia as enmeshed, rigid, and overprotective. They also

argued that these families tend to have difficulty with conflict resolution and involve child and parent conflict.

Socio-cultural theories underscore the pressure of societal messages promoting the thin ideal, (Silverstein, Peterson, & Perdue, 1986; Wiseman, Gray, Mosimann, & Ahrens, 1992) socialization, and gender role stereotypes (Striegel-Moore, Silberstein, & Rodin, 1986). According to these theorists, culture offers women a substitute, although destructive, system of meaning that may stand-in when an internal system of meaning is missing. Clinicians often note that eating disorder patients exhibit a heightened sensitivity to the needs, moods, and subtle reactions of others compared to patients without eating disorders (Rorty & Yager, 1996a). Not only do these patients appear to be hyper-attuned in regard to interpersonal connections, they also seem to be hyper-aware of cultural expectations. Thus, eating disorder patients may be more vulnerable to overwhelming messages from society that tell women they must be youthful, attractive, and thin in order to have any value as a human being (Rorty & Yager, 1996a). Conversely, as women are being bombarded with these messages encouraging thinness, they are also influenced to eat more due to current social cues (Wansink, 2010). Women who lack an internal sense of purpose tend to be more vulnerable to these messages (Rorty & Yager, 1996a). Excessive dieting and exercise also serves an organizing role for vulnerable women. Women seeking to avoid deeper issues can fill their conscious awareness with thoughts about weight and food issues. Furthermore, rather than being angry with one's self, anger can be directed at society's impossible standards. This seems to be an important defensive mechanism for women with eating disorders, especially those who have a

history of trauma, because it provides a distraction from reminders of trauma (Rorty & Yager, 1996a).

The most recently identified etiological perspective of eating disorders relates to the influence of biological factors (Suisman et al., 2012). Researchers have documented that genetics account for about 55-60% of the variance in eating disordered symptoms (Slane, Burt, & Klump, 2011). Furthermore, neurotransmitters such as GABA and serotonin have been associated with symptom emergence and treatment (Bloss et al., 2011; Pichika et al., 2012). Finally, symptoms have been linked to hormones, such as estrogen and progesterone, and the female menstrual cycle appears to have a distinct role in symptoms such as drive for thinness and body dissatisfaction (Racine, Culbert, Keel Sisk, Burt & Klump, 2012).

Research on the etiology of eating disorders has yielded mixed results even when similar samples and common methodology are used. These mixed results suggest that pathological eating is more complex than original conceptions. Research on eating disorders is complicated partially because of high comorbidity rates with other psychiatric disorders. In fact, eating disorders rarely occur in isolation from other pathology (Fletcher, Kupshik, Uprichard, Shah, & Nash, 2008). Fletcher et al. (2008) suggest that these other symptoms related to comorbid diagnoses, such as depression, anxiety, and interpersonal problems, may be of primary importance to eating disorder patients. Specifically, research has suggested that symptoms of posttraumatic stress disorder may be crucial when it comes to development of eating pathology (Anderson, LaPorte, Brandt, and Crawford, 1997).

Childhood Trauma and Trauma Theory

The DSM-IV-TR defines a traumatic event as "an event where one experiences a serious bodily injury or threat accompanied by intense fear, helplessness, or horror" (APA, 2000, as cited in Seides, 2010, p. 725). People who have experienced childhood trauma often are later diagnosed with one or more of the following: PTSD, depression, anxiety, substance abuse, and eating problems (APA, 2000; Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Jacobi, Hayward, deZwaan, Kraemer, & Agras; 2004; Shipherd, Stafford, & Tanner, 2005). Trauma theory has proposed that the association between child sexual abuse and eating disorders is best understood by a series of complex, intertwined emotional reactions and coping strategies (Schwartz & Gay, 1996).

Everly (1995) asserted that psychological trauma takes place when a psychological crisis exceeds a person's mental capacity to tolerate the stress. Root (1992) argued that the affected person, not the observer, defines trauma. For some, brief separations and parental misattunement or neglect could be experienced as traumatic (Farber, 2008; O'Shaughnessy & Dallos, 2009). It appears that a certain degree of individual variability exists in regard to what is experienced as threatening or to what degree intense fear, helplessness, and/or horror are experienced. Not every individual who experiences what the DSM-IV-TR defines as trauma develops a posttraumatic response that impairs functioning. Therefore, the eventual emergence of pathological symptoms may be influenced by how scary each individual perceived the trauma to be.

In previous studies, childhood trauma has been defined as receiving or witnessing physical, emotional, psychological, and/or sexual abuse and physical or emotional neglect (Sansone & Sansone, 2007). Of these experiences, Sansone and Sansone (2007)

argue that repetitive, purposeful, malignant treatment is associated with the greatest psychological disturbance. Thus, according to these researchers, chronic physical, sexual, and emotional abuse represent the greatest risk for eating disturbances. Others argue however, that the parents' failure to respond to a traumatic environment can also be perceived as trauma by children. That is, the parent's failure to protect, or neglect of the child, may be especially detrimental as an etiological factor. Thus, researchers like Wilbur (1985) and Giovacchini (1986) have argued that chronic failures to respond to distress and chronic disregard for the emotional and physical needs of the child can be in itself traumatic. For these reasons, many researchers have included neglect as a potentially traumatic experience. These theorists have also argued that children who are exposed to neglect tend to detach or dissociate in times of emotional distress. Thus, it is important to keep in mind that those who have experienced chronic neglect or parents who failed to protect them from abusive adults may be prone to engage in eating behaviors that help them to dissociate from their physical environments.

Traumas that are designed by humans and perpetrated by a loved one appear to have the longest lasting psychological effects compared to events such as natural disasters (Allen, 2001; Herman & Harvey, 1997). Furthermore, many studies have confirmed that the cumulative effect of trauma is important (Orzeck, Rokach, & Chin, 2010; Tagay et al., 2010). Child abuse is also unlike many potentially traumatizing experiences that occur at a single time and cause overwhelming feelings such as natural disasters or accidents because child abuse often occurs without a discrete beginning or end (Herman, 1992).

Trauma and Disordered Eating

Several studies have demonstrated that a significant proportion of individuals with eating disorders report a history of childhood abuse. Rodriguez, Perez, and Garcia (2005) also found statistical significance, reporting that 45% of a sample of 270 outpatient women with eating disorders had a history of childhood trauma.

A number of trauma-based theories of eating disorders have been suggested. Many researchers have postulated that for many women, eating disturbance may be part of a post trauma response to victimization (Larkin et al., 1996; Rice, 1996; Root, 1991; Root & Fallon, 1988; Thompson, 1992). Eating problems have been documented as a strategy for coping with various forms of trauma including child sexual and physical abuse, battering, rape, and sexual harassment (Harned, 2000). Researchers have proposed that many maladaptive eating behaviors are used as coping mechanisms for the distress that follows victimization (Larkin et al., 1996; Root, 1991; Schwartz & Cohn, 1996; Thompson, 1992). These behaviors (e.g. binge eating, purging, dieting) may be used to numb depressive feelings, assuage anxiety, produce sleep or dissociate from disturbing memories (Harned, 2000).

Orbach (1986) proposed that women raised in a society that idealizes thinness become obsessed with issues of weight and diet. Because society teaches women to suppress their needs in order to please others, physical urges are ignored in order to meet society's thin ideal. Orbach (1986) noted that these sacrifices are accompanied by a quiet anger that is perceptually impossible for the eating disordered woman to express and thus directed inward. Research has provided some support for these assertions. For example, Williams, Chamove, and Millar (1990) found that patients with eating disorders reported

more self-directed hostility, higher external locus of control, less assertiveness, and less family encouragement than did individuals in dieting and non-dieting control groups. In all of these theories is the idea that certain personality or psychological characteristics exists in women with anorexia and/or bulimia. Theorists ascribing to each perspective have described individuals with eating disorders as feeling out of control and ineffective (Bruch, 1973; Pyle, Neuman, Halvorson, & Mitchell, 1991), obsessional (Crisp & Bhat, 1982), dependent (Bornstein & Greenberg, 1991), and unassertive (Boskind-Lodahl, 1976).

In contrast to studies that did find a link, a fair number of studies have found no link between the severity of eating symptoms and abuse characteristics (Bailey & Gibbons, 1989; Bushnell et al., 1992; Folsom et al., 1993; Fullerton et al., 1995; Pope et al., 1994; Smolak et al., 1990; Vize & Cooper, 1995). Consequently, the extent to which having a history of trauma contributes to the maintenance, severity, and onset of disordered eating habits is not clear (Bailey, 2000). Research has pointed to a number of variables that influence the child's adjustment following abuse (Cohen & Mannarino, 1996; Green, 1993; Kendall-Tackett et al., 1993; Mannarino & Cohen, 1996; Tebbutt et al., 1997). These factors include the child's cognitive construction of the abuse, locus of control, parental reaction, and overall family functioning.

Kong and Bernstein (2009) reported the highest prevalence rate, finding in a sample of 73 outpatient women with eating disorders (39.7% AN, 53.4% BN, 6.8% EDNOS) that 90.4% reported a history of traumatic experiences. Kong and Bernstein's (2009) results have been criticized, however, as an inaccurate picture of the relationship between childhood trauma and eating pathology. Taylor (2009) submitted a formal critique of

these findings, asserting that it is misleading to claim that eating disorders are always caused by trauma and that such a significant proportion of eating disorder patients have a trauma history. Supportive of this line of argumentation, a number of studies have reported that the rates of trauma in women with eating disorders did not differ from other psychiatric disorders (Bryer et al., 1987; Chu & Dill, 1990; Herman, Perry & van der Kolk, 1989; Ogata, et al., 1990). Although correlations between trauma and eating disorders have been high, research to date has not been able to support a causal link. In fact, it has been suggested that high correlations may be due to some other variable or set of variables other than trauma. The mixed findings of studies on reported history of abuse with psychiatric disorders suggests that the link between eating disorders and abuse may not be specific but rather influenced by additional mediating and moderating variables (Connors & Morse, 1993; Pope & Hudson, 1992).

Some research has shown that trauma is more common in the history of patients with bulimic eating disorders than non-bulimic eating disorders (Smolak & Murnen, 2002; Striegel-Moore, Dohm, Pike, Wilfley, & Fairburn, 2002) and has a higher association to psychiatric comorbidity (Sansone & Levitt, 2005). Studies have demonstrated consistently that a high percentage of people with bulimia have a history of trauma (Brewerton, 2007; Thompson & Wonderlich, 2004). The behaviors of binging and purging seem to have specific links to trauma history (Pitts & Waller, 1993; Waller, 1992). Specifically, large-scale studies of nationally representative samples have demonstrated that childhood sexual abuse is a risk factor for bulimia (Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Wonderlich, Wilsnack, Wilsnack, & Harris, 1996).

Many studies have documented trauma history in patients with eating disorders (Dalle Grave, Rigamonti, Todisco, & Oliosi, 1996; Mahon, Bradley, Harvey, Winston, & Palmer, 2001). A number of studies indicate that there are no statistical differences in the prevalence rates of childhood sexual abuse between clinical and nonclinical populations. This observation, however, indicates only that there are no between group differences in the prevalence rates of trauma. It does not eliminate trauma as a causal factor for psychopathology in the clinical subgroup. Rather, this statistical "nonfinding" may directly support the notion that the psychological effects of trauma are highly variable, with some individuals demonstrating unimpaired psychological outcomes in adulthood and others developing various types and degrees of psychopathology (Sansone & Sansone, 2007). The question remains, why do only some women who have experienced childhood sexual abuse develop eating disorders?

Research by Anderson, LaPorte, Brandt, and Crawford (1997) suggested that eating disorders are more severe when a history of child sexual abuse exists. Furthermore, eating disordered patients who had a history of abuse had more comorbidity with depression, borderline personality disorder, and chemical dependency. Anderson, LaPorte, Brandt, and Crawford (1997)'s study also showed that those patients who had a history of abuse were more likely to have subsequent eating disorder-related psychiatric hospitalizations than those who did not have an abuse history. Their study did not demonstrate, however, that the severity of the abuse was correlated with the severity of bulimic symptomology. Also, many studies have found that history of abuse occurs in conjunction with poorer symptom outcomes (Herzog et al., 1991).

Another possibility is that trauma is not related to the etiology of eating disturbance but that it intensifies or complicates eating disorder symptoms (Briere & Scott, 2007). A 2008 investigation by Holzer et al. revealed that abuse victims displayed significantly more eating disorder pathology than comparison subjects. Furthermore, they discovered that when they controlled for the effects of PTSD, the relationship between trauma and eating disorder behaviors were markedly reduced and no longer significant (Holzer et al., 2008). Specifically, high scores on the avoidance component of PTSD were demonstrated to be a nearly perfect mediator of the relationship between trauma and eating disorders.

Rorty, Yager and Rossoto (1993) proposed that all childhood experiences of abuse constitute boundary violations, which lay the foundation for the development of later psychological difficulties. They suggest that all such experiences predispose the individual to psychopathology, including eating disorders, by damaging the self-concept and reducing the ability of the individual to manage strong affect. This vulnerability then interacts with temperament, family dysfunction, and environmental factors, leading in some instances to eating psychopathology.

Sexual Trauma and Disordered Eating

Childhood sexual abuse has been the most well documented trauma in eating disorder patients (Tagay, Schlegl, & Senf, 2010). Sexual abuse has been correlated with both anorexia and bulimia. A great deal of research has been conducted in this area but results of studies in this area have been inconsistent (Connors & Morse, 1993; Deep et al., 1999; deGroot, Kennedy, Rodin, & McVey, 1992; Kinzl et al., 1994; Pope et al., 1994; Waller, 1998; Waller, Halek, & Crisp, 1993). Researchers seem to be divided in their conclusions

(Gleaves, Eberenz, & May, 1998) as some studies have found associations but others have not (Connors & Morse 1993; Pope & Hudson, 1992).

Although definitions of sexual abuse vary greatly, childhood sexual abuse is reported by approximately 20% of women (Gorey & Leslie, 1997; Martin, Anderson, Romans, Mullens, & O'Shea, 1993). In the late 1980s, case reports began to suggest that child sexual abuse was linked with eating disturbances. Modal figures from clinical samples have indicated that about 30% of adults with bulimia have histories of childhood sexual abuse (Steiger & Bruce, 2009; Steiger et al., 2010; Steiger et al., 2011). Carter et al. (2006) reported that 48% of the inpatients in an eating disorder unit affirmed a history of childhood sexual abuse.

Many empirical studies with adults and children have suggested that a history of child sexual abuse is correlated with clinical levels of eating disturbance (Bushnell, Wells, & Oakley-Brown, 1992; Cohen et al., 2010; Johnson, Cohen, Kasen, & Brook, 2002; Orzeck et al., 2010; Pribor & Dinwiddie, 1992; Steiger et al., 2010; Steiger & Zanko, 1990; Tagay et al., 2010; Vize & Cooper, 1995; Welch & Fairburn, 1994; Wonderlich, Wilsnack, Wilsnack, & Harris, 1996; Wonderlich et al., 1996; Wonderlich et al., 2001; Zlotinick et al., 1996). A meta-analysis of 53 studies found a consistent association between childhood sexual abuse and eating disorders. Authors concluded that child sexual abuse is associated with an increased likelihood of eating disorder symptomology (Smolak & Murnen, 2002). Brewerton (2007) also asserted that childhood sexual abuse is a non-specific risk factor for eating disorders. Connors and Morse (1993), Root and Fallon (1988), Rorty, Yager, and Rosotto (1994), and Vanderlinden and Vandereycken (1993) argued child sexual abuse is a risk factor for a variety of psychiatric disturbances,

including eating disorders and suggested that for some people childhood sexual abuse is directly related to eating dysfunction.

Studies of women with eating disorders (Steiger & Zanko, 1990), abuse victims (Pribor & Dinwiddie, 1992; Wonderlich et al., 1996), community-based samples (Bushnell el al., 1992; Garfinkel et al., 1995), and nationally representative samples (Dansky et al., 1997; Wonderlich et al., 1996) have found significant correlations between a history of childhood sexual abuse and disturbances in eating.

Steiger and Zanko (1990) reported a much higher rate of sexual abuse in bulimic anorexics and bulimic patients compared to their normal control group. Two non-clinical studies (Hastings & Kern, 1994; Miller et al., 1993) also demonstrated higher rates for patients with bulimia as compared to controls. Welch and Fairburn (1996) reported that 35% of the cases of bulimia (N=102) in their study had histories of childhood sexual abuse. In a clinical study of bulimic women (N=40), a reported history of sexual violations was associated with more frequent binging (Waller, 1992a). Bulik, Sullivan, and Rorty (1989) investigated the psychiatric and family history of women with bulimia and found that 34.5% had either a history of sexual abuse or reported a sister who had been sexually abused. The women who reported a personal history of sexual abuse displayed higher rates of comorbid depression compared to those with abused family members. Abramson and Lucido's (1991) research reported a significant correlation between bulimia and childhood sexual abuse ($r = .30$, $p < .05$).

Steiger and Zanko (1990) reported that 30% of the women with eating disorders in their study reported histories of childhood sexual trauma. Two studies showed higher rates of eating disorders in abused women compared to non-abused women (Pribor &

Dinwiddie, 1992; Shearer et al., 1990). In two studies of non-clinical samples, Smolak et al. (1990) found that abused students demonstrated significantly higher scores on the Eating Disorder Inventory (EDI) than non-abused students. Folsom et al. (1993) reported that sexually abused eating disorder patients had more psychological disturbance of obsessive and phobic nature than non-abused subjects, suggesting that sexual abuse may lead to increased psychological distress rather than eating symptomology. Vize and Cooper (1995) studied eating disorder patients and non-eating disordered controls and discovered significantly higher rates of childhood sexual abuse among the eating disordered patients. Oppenheimer et al. (1985) found that 64% of eating disorder patients were sexually abused during childhood and/or adolescence. Hall, Tice, and Beresford (1989) reported that 60 out of 158 inpatients with eating disorders had histories of sexual abuse. Research by Bailey (1994) demonstrated that in a sample of 101 outpatients, 50% of the patients who had eating disorders had prior histories of sexual abuse and that 66% of patients with eating disorders had histories of generalized childhood abuse. Bailey (2000) discovered that of 34 females seeking treatment for eating disorders, 74% reported a history of sexual victimization. Oppenheimer et al. (1985) reported that 29.5% of a sample of 78 patients with eating disorders endorsed a history of childhood sexual abuse. Tice et al. (1989) reported that in a sample of 158 inpatients with eating disorders, 60 subjects endorsed a history of sexual abuse. Further, 50% of the subjects had experienced sexual abuse, a clinically significant percentage.

A study by Wonderlich et al. (2000), the first empirical study on this topic to use children as participants, demonstrated that sexually abused children were more likely than controls to be dissatisfied with their weight, restrict food when upset, idealize and

pursue thinness, and purge. Participants were made up of 40 girls, ages 10-15, including a group of 20 sexually abused girls and a control group of 20 non-abused girls. These results were consistent with prior research, which used adults as participants (e.g. Calam, Griffiths, & Slade, 1997; Runtz & Briere, 1986).

Wonderlich et al. (1996) surveyed 1099 women and reported that 34% of women in their study demonstrated binge/purge behaviors that were related to a history of childhood sexual abuse. Garfinkel et al. (1990) reported in a nationwide survey of 8,116 Canadians that 32.7% of the females meeting for criteria for bulimia had a history of sexual abuse versus a comparison rate of 13.9% of females who did not demonstrate bulimic symptoms. In another community study, women with a lifetime history of subclinical disordered eating reported significantly higher rates of intrafamilial sexual abuse than women selected at random (Bushnell et al., 1992). Another community case-control study found that rates of abuse in those with bulimia nervosa were higher than the general population (Welch & Fairburn, 1994).

Harned (2000) reported that 40% of undergraduate women (N=195) experienced at least one incident of sexual abuse or assault in their lifetime. Among the participants in her study, 25% reported that they experienced an attempted rape, 12% reported that they had been raped, and 20% reported that they had been sexually abused. Further, 59% of the sample had experienced physical abuse and 25% had experienced severe physical violence. Sixty-three percent of the sample had experienced lifetime symptoms of eating disorders and 44% were experiencing current eating disorder symptoms. Harned (2000) concluded that abuse is a prevalent experience in women's lives and speculated that the high correlation between abuse and eating disorder symptoms might be due to the

perceived normalcy and pervasiveness of both phenomena. Harned's (2000) work draws attention to the gender-based nature of eating problems and the possible correlations between eating disorders and violence against women.

Additionally, many studies have demonstrated a positive relationship between childhood sexual abuse and subclinical eating dysfunction (Calam & Slade, 1989; Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Garfinkel et al., 1995; Hastings & Kern, 1994; Romans, Gendall, Martin, & Mullen, 2001; Smolak, Levine, & Sullins, 1990; Steiger & Zanko, 1990; Weiner & Thompson, 1997; Zlotnick et al., 1996).

Other investigations, however, have demonstrated no relationship between childhood sexual abuse and subclinical eating disturbances (Casper & Lyubomirsky, 1997; Kent, Waller, & Dagnan, 1999; Kinzl, Traweger, Guenther, & Biebl, 1994) or clinical eating disorders (Pribor & Dinwiddie, 1992; Rorty, Yager, & Rossotto, 1994). In a comprehensive literature review by Wonderlich et al. (1997), the relationship between anorexia nervosa and childhood abuse was deemed unsubstantiated. Four out of 10 studies show no significant differences in abuse history between eating disordered subjects and normal controls (Abramson & Lucido, 1991; Beckman & Burns, 1990; Rorty et al., 1994). Rorty and Yager (1996b) asserted that child sexual abuse is neither necessary nor sufficient for the development of eating disorders. Finn et al. (1986) compared eating disorder rates in abused populations and failed to demonstrate any difference between abused and non-abused women. Kinzl et al. (1994) attempted to replicate finding by Smolak et al. (1990) but were unable to produce significant results in contrast to the original research.

Some researchers and theorists have differentiated between symptoms of bulimia and anorexia in regards to correlations with childhood sexual abuse. A number of researchers have suggested that a history of childhood sexual abuse is strongly correlated with bulimic symptoms specifically, as opposed to anorexia or eating disorders in general (e.g., Connors & Morse, 1993; Palmer et al., 1990; Palmer, Chaloner, & Oppenheimer, 1992; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). The prevalence seems to be greater among women with disorders that have a bulimic component (Waller, 1991; Pribor & Dinwiddie, 1992). Johnson, Cohen, Kasen, and Brooke (2002) and Wonderlich et al. (2007) have suggested that childhood sexual abuse is related to specific features of bulimia, such as binging, purging, and body dissatisfaction, rather than to the disorder as whole.

Considerable debate remains about whether or not a fundamental difference exists between anorexia and bulimia in regards to trauma, and other studies have contradicted these findings (Miller, 1993; Pope & Hudson, 1992; Welch & Fairburn, 1994). For instance, research by Gati, Tenyi, Tury, and Wildmann (2001) suggests that sexual abuse may be a risk factor for the development of anorexia. In 1992, Pope and Hudson concluded that the available data were insufficient to indicate that child sexual abuse was a risk factor in the development of bulimia. They asserted that evidence does not support a linear causal relationship. Everill, Waller, and Macdonald (1995) also demonstrated no significant association between prevalence of reported sexual abuse and diagnostic category. Wonderlich et al. (1997), however, suggested that childhood sexual abuse is a significant risk factor for bulimia nervosa, particularly because there are high degrees of psychiatric comorbidity present. Whether or not childhood sexual abuse is a specific risk

factor for bulimia has been a longstanding question that remains unanswered (Anderson, LaPorte, & Crawford, 2000).

In sum, although some data support a correlation between childhood sexual abuse and eating disorders, a clear relationship has not been well supported by empirical research (Wonderlich et al., 2001). Waller (1992) argued, however, that inconsistent correlations between eating disorders and sexual abuse history do not mean that there is no causal relationship or specific link. Although it would be going far beyond the data to say that sexual abuse causes eating disorders, it appears that the having such a history influences the degree of symptomology, especially in regard to bulimia (Waller, 1992).

Despite contradictions in research findings, psychologists need to be aware that for a great number of patients, sexual abuse histories and eating disorders are related (Calam, Griffiths, & Slade, 1997). Regardless of etiologic significance, clinicians frequently treat patients who present with eating disorders and have histories of sexual abuse (Connors & Morse, 1993). Therefore, the connection between sexual abuse history and eating disorder symptomology needs to be more fully understood. Several theories have been formulated about the possible relationship. One explanation regarding a connection between sexual abuse and eating disorder symptoms is that body-related shame may be an important variable as many children who have experienced sexual abuse have reported increased feelings of shame about their bodies (Andrews, 1995; Kearney-Cooke & Striegel-Moore, 1994). Eating disorder symptoms, including subclinical disordered eating, might develop as an attempt to cope with resulting emotional states and while simultaneously changing some characteristics of the victim's body. Some psychoanalytic theorists have argued that a sexual abuse history explains the often comorbid difficulties

with sexuality often seen in those with eating disorders. Root and Fallon (1988) asserted due to early boundary violations, victims often experience fear, intimidation, and significant physical and/or psychological injury as a result. They termed this effect *traumatic sexualization*. Root and Fallon (1988) asserted that abuse histories increase vulnerability because eating disorder symptoms serve as a way to cope with anger, powerlessness, and depression, which frequently develop after a violation of physical boundaries. Consistent with this hypothesis, Sugarman, Quinian, and Devinis (1982), found that patients with anorexia had difficulty with maintenance of appropriate boundaries. Some other researchers have suggested that what appears to be a correlation between sexual abuse and eating disorders may be coincidental-- a product of the high rates of both occurrences among women (Finn et al., 1986; Pope & Hudson, 1992).

Sloan and Leichner (1986) theorized that eating disorders serve a defensive function for these individuals in that they could both avoid painful affect and sexuality as an adult. Goldfarb (1987) expanded on this theory by suggesting that in addition to serving as a method for avoiding painful emotions and sexuality, eating disorders also provide a sense of control for people who experienced a sense of powerlessness as a child.

Also, it has been hypothesized that a sexual abuse survivor may shove food into her mouth as others shoved various objects into her body. Then she may vomit the food to rid her body of those things that were inserted by force, since during the abuse she was unable to rid herself of those objects (Farber, 2008).

Mediational studies of the relationship between sexual abuse and eating disorders have generally been based on a theory that impulsive, dysregulated behavioral mechanisms, including dissociation, impulsive self-destructive behavior, and PTSD

(Wonderlich et al., 1996, 2000), are causal factors (Kent, Waller, & Dagnan, 1999). Another explanation which takes mediating variables into account is that early childhood trauma results in multiple forms of psychopathology (Putnam & Trickett, 1997; van der Kolk, 1987), which in turn increases one's vulnerability to eating disturbances (Brewerton, 1995; Wonderlich et al., 1997). Beck and van der Kolk (1987)'s research found that psychiatric patients with sexual abuse histories had more serious psychiatric problems and more severe psychopathology within their diagnostic categories than those patients without a history of sexual abuse. Also, those with sexual abuse histories tend to be more treatment resistant than their non-abused counterparts. Gleaves and Eberenz (1995) found that those who have both eating disorders and histories of sexual abuse have higher risk for treatment failure.

Nonsexual Trauma and Disordered Eating

Some have criticized the fact that researchers have looked extensively at sexual abuse but have failed to look at other forms of abuse. In studies that have failed to find a link between sexual abuse and eating disorders, a link between other forms of childhood abuse, such as physical or emotional abuse, and eating disorders is often supported (Rorty, Yager, & Rossotto, 1994). This finding suggests that including a broader definition of childhood trauma that includes all forms of abuse may be more accurate in looking at precipitating factors among patients with eating disturbances. The existing research on the relationship between other forms of abuse and eating dysfunction will be described below.

The role of childhood physical abuse has attracted substantially less attention in the eating disorders literature. Although childhood physical abuse was one of the first forms

of abuse to gain the attention of researchers, a dearth of empirical investigations remains when considering the relationship between this form of abuse and eating pathology. Preliminary research has suggested that childhood physical abuse might act as a nonspecific risk factor for some eating disorders, particularly bulimia (Folsom et al., 1993; Root & Fallon, 1988). Twenty-three to 40 percent of these women have histories of physical abuse or battering (Kaner, Bulik, & Sullivan, 1993; Root & Fallon, 1988). Many researchers have contended, however, that these numbers are no different than psychiatric controls (Harned, 2000). Recently, trauma researchers have noticed the lack of research looking at the influence of emotional abuse on eating symptomology and more attempts have been made to include childhood emotional abuse in studies (Groleau et al., 2012). Groleau et al. (2012) found that those with bulimic symptoms were more likely to have histories of childhood emotional abuse and also that childhood emotional abuse seems to mediate the effects of maladaptive affect in those with eating disorder symptoms.

Kent, Waller, and Dagan (1999) demonstrated that in addition to sexual abuse being a risk factor, physical abuse is also correlated with eating disorder symptomology. Physical abuse was significantly correlated with overall EDI scores and body dissatisfaction. The Drive for Thinness scale of the EDI was significantly correlated with sexual abuse. Women with eating disorders and histories of other forms of trauma have also reported high rates of physical abuse, psychological abuse, witnessing of intrafamilial violence, parental separation, loss of a family member, and other adverse events which have also been correlated with later symptoms of eating disorders (Dalle Grave et al., 1996; Mazzeo & Espelage, 2002; Mahon et al., 2001; Piran et al., 1988; Rorty, Yager, & Rossoto, 1994; Schmidt, Tiller, & Treasure, 1993).

More recent literature has also suggested that traumatic peer experiences, such as bullying, are also correlated with eating disordered behaviors (Engstrom & Norring, 2002; Kaltiala-Heino, Rissanen, Rimpela, & Rantanen, 2003; Sweetingham & Waller, 2008). Similarly, childhood experiences of being teased about weight and shape have also been associated with eating and related pathology, including body dissatisfaction, low self-esteem, depression, and disordered eating behaviors (Jackson, Grilo & Masheb, 2000; Neumark-Sztainer, Falkner, Story, Perry, Hannan, & Mulert, 2002). Research has suggested that multiple forms of abuse may have additive or interactive effects (Rorty, Yager, & Rossotto, 1994; Schmidt, Tiller, & Treasure, 1993).

Alternatively, some researchers have argued that emotional abuse is the crux of childhood trauma (Hart & Brassard, 1987). Research has suggested that psychological maltreatment may be more prevalent and more destructive than other forms of childhood trauma (Briere & Runtz, 1988, 1990; de Groot and Rodin, 1999; Hart & Brassard, 1987; Moeller et al., 1993). Parental failures in attunement and responsiveness to the child's emotional life can have a profound and long lasting detrimental psychological impact on the child (Briere & Runtz, 1988, 1990; Emde, 1983; Gross & Keller, 1992; Hoglund & Nicholas, 1995). Relatively little research has been conducted, however, on childhood emotional abuse relative to other forms of trauma (e.g. sexual and physical abuse) (O'Hagan, 1993). Early research looking at abuse and eating psychopathology connections only included physical and sexual abuse (Kent & Waller, 2000). The role of emotional abuse has also been considered (Rorty, Yager, & Rossotto, 1994) as research has suggested that emotional abuse significantly predicts eating disturbance independent of sexual abuse (Wonderlich, 2000).

The lack of research on childhood emotional abuse has been partially due to a lack of an adequate definition (Rosenberg, 1987). Childhood emotional abuse is particularly hard to operationally define; thus, many researchers avoid the issues altogether, failing to offer a definition for what it is they attempt to measure. Despite these difficulties, a number of definitions have been proposed (e.g. Hart & Brassard, 1991; Seides, 2010; Yates & Wekerle, 2009). Recent definitions have been based on a continuum with the recurring, unremitting nature of the acts being a crucial defining feature (O'Hagan, 1995). Although childhood sexual abuse could be indicative of a more discrete parental failure, emotional neglect or attunement failures may be as important in the etiology of eating disorders (de Groot & Rodin, 1997).

Kent and Waller (2000) postulated that childhood emotional abuse may play one of two roles within a multi-factorial framework. The first is that childhood emotional abuse is a causal factor, which leads to psychological and physiological consequences and predisposes children toward developing an eating disturbance. The second possibility is that rather than serving as a causal agent, childhood emotional abuse moderates the impact of other causal factors such as dissociation, shame, and self-denigratory beliefs. The latter hypothesis has been relevant, as studies have shown that childhood emotional abuse has seemed to moderate the impact of other forms of trauma. Other forms of abuse have appeared to lead to more pathological consequences when they occur in the context of an emotionally damaging relationship (Kent & Waller, 2000). Much more has been discovered about the immediate consequences of childhood emotional abuse than the long-term consequences and impact (Kent & Waller, 2000).

Briere and Runtz (1990) investigated three major types of childhood abuse: sexual, physical, and emotional/psychological. They also looked at three types of psychological symptoms: low self-esteem, anger/aggression, and sexual difficulties. In their study, they demonstrated a unique relationship between emotional/psychological abuse and low self-esteem, and hypothesized that such negative self-evaluation may be a result of the child internalizing parental criticism and insults over a prolonged period of time. Other researchers have also found a link between childhood emotional abuse and low self-esteem. Gross and Keller (1992) found that childhood emotional abuse was a better predictor of low self-esteem than childhood physical abuse. Hoglund and Nicolas (1995) found that childhood emotional abuse, not physical abuse, was directly related to shame. They hypothesized that the reason for these results was that childhood physical abuse is more easily viewed as punishment for an act, whereas childhood emotional abuse is more readily interpreted as a direct attack upon the person. Self-esteem problems have been important to understand because of their potential link to eating disorders. Restrictive eating behaviors have been suggested to be an outcome of poor self-esteem and a way to feel like one has control over one part of life (Fairburn et al., 1999; Slade, 1982).

A study by Briere and Runtz (1988) demonstrated that physical and psychological abuses generally occur together. Further, a study by Mullen et al. (1996) found considerable overlap in terms of psychopathology between specific types of abuse. They described several important patterns. First, associations between eating disorders and trauma were higher when looking at sexual and emotional abuse rather than physical abuse. Second, of all three types of abuse, those who were emotionally abused demonstrated the lowest self-esteem. Third, the gender of the abuser was particularly

important in emotional abuse. Those who were emotionally abused by a female caregiver had more problems with general pathology whereas those abused by a male were more likely to have sexual difficulties. Fourth, a disrupted home environment was highly correlated with all types of abuse. Therefore, it is important to consider whether these four variables might be important in terms of later development of eating disturbance.

All forms of child abuse have been associated with long-term physical health problems and psychological problems. Moeller et al. (1993)'s research supported the idea that physical and psychological problems increase in proportion to the number of abuse types experienced. Beyond simply the type of abuse, however, it is important to take into consideration the idiosyncratic meaning of the abuse to each child (Kent & Waller, 2000).

The literature has made clear that the relationship between childhood abuse and the development of eating disorders in adolescence is not a simple matter of diagnostic status (Everill, & Waller, 1995). The question then becomes, why do eating disorders develop in some cases versus other symptoms of pathology (Fletcher et al., 2008). The most logical answer to this question seems to be that other variables play important mediating and moderating roles that make the relationship between the two more complex.

Moderating Influences

PTSD

PTSD has been identified as a possible moderator of the relationship between child abuse and eating disorders (Holzer et al., 2008). Specifically, studies by Dansky and colleagues (Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Dansky, Brewerton, &

Kilpatrick, 2000) suggested that PTSD is a powerful moderator in the relationship between a history of sexual assault and bulimia nervosa. Davidson et al. (1991) demonstrated in a community sample that child sexual abuse survivors were at a nine times greater risk for developing PTSD than those who had not been sexually abused. Further, research by Cohen et al. (2010) demonstrated a relationship between disordered eating, depression, and PTSD. PTSD has also been a difficult topic to study in relation to eating disorders since so many of the symptom patterns overlap and thus multicollinearity can create methodological issues.

Lifetime rates of PTSD in the general population have been reported around one percent (Davidson et al., 1991; Helzer et al, 1987). In contrast, rates of PTSD in those exposed to natural disaster have been reported as high as 59 percent (Green et al., 1992). Schetky (1990) estimated that posttraumatic stress symptomology occurs in about half of child and adult abuse survivors. Dansky, Brewerton, Kilpatrick, and O'Neil (1997) conducted a research study with women in residential eating disorder treatment programs and found that 74% of these patients reported experiencing a prior trauma. Of those who reported a trauma experience, 52% met criteria for PTSD. Their research reported that 36.9% of women with bulimia met criteria for PTSD at one point during their lifetimes. Tagay, Schlegal, and Senf (2010) reported that 63.3% of anorexia patients and 57.7% of bulimic patients have experienced at least one lifetime trauma. Of those who reported lifetime traumas, types of trauma were as follows: sexual traumas: 50% anorexia/53.6% bulimia, nonsexual interpersonal traumas: 26.7% anorexia/36.6% bulimia, and other traumas such as natural disasters, fires, and accidents: 53.3% anorexia/47.9% bulimia.

Tagay, Schlegal, and Senf (2010) reported that the current prevalence of PTSD among patients (N=30) with anorexia was 10% and among patients with bulimia it was 14%. These rates were consistent with research by Brewerton (2007). More recently, however, The National Women's Study indicated that rates of PTSD were as follows: 37% for bulimics, 21% for binge eaters, and 11.8% for EDNOS patients (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). In contrast, Turnbull, Troop, and Treasure (1997) found no difference in prevalence rates of PTSD between anorexic and bulimic subgroups. Trumbull et al. (1997) also reported that severity of PTSD symptoms was correlated with childhood sexual abuse, discord in the home, and high levels of parental control. Tagay et al. (2010) also demonstrated that patients with both eating disorders and PTSD diagnoses had more severe symptoms.

Despite the prevalence of PTSD among those with a history of eating disorders, studies have suggested that clinicians are rarely diagnosing patients with the disorder. In fact, several studies support the idea that PTSD is under-diagnosed in patients who meet diagnostic criteria (Mueser et al., 1998; Samson et al., 1999). For example, Zimmerman and Mattia (1999) found that when they screened patients for PTSD, 25.8% met criteria for PTSD but only 7.2% had actually been diagnosed as such.

In addition to finding a connection between eating disorders and PTSD, researchers have also pointed to a relationship between PTSD and other Axis I disorders, particularly depression and anxiety (Davidson et al., 1991; Green et al., 1992; Helzer et al., 1987). McCallum et al. (1992) reported that 74% of the subjects in their study, eating disorder patients, also had a lifetime diagnosis of depression and 32% were dysthymic. These relationships may be an example of a confounding variable, in that many symptoms of

PTSD overlap with symptoms of other Axis I disorder, particularly depression and anxiety.

Research has also shown that people with PTSD are more prone to somatic symptoms (Davidson et al., 1991). Among female child abuse survivors, particularly of sexual abuse, evidence supports higher levels of somatic symptoms (Golding, 1994; Walker et al., 1992; Walling, 1994). Tegay et al. (2010) demonstrated that people with eating disorders and PTSD reported much higher levels of somatoform symptoms than did eating disorder patients without PTSD. Gendall et al. (2005) found that younger ages of onset for self-induced purging was correlated with heightened somatic complaints. They suggested that children who report high levels of stomach pain might be more sensitive to stress. Furthermore, they suggested that the experience of fullness may have been associated with pain during childhood and thus the child learned that being full is bad.

The core psychological problems of those who have survived child sexual abuse are often consistent with a diagnosis of PTSD (Rowan & Foy, 1993). Wilbur (1985) and Kluft (1985) drew attention to the fact that affect regulation tends to be difficult for both those with PTSD and those with eating disorders. Beahrs (1990) suggested that people with PTSD have developed a lower stress tolerance in response to trauma and are more sensitive to triggers that might be linked to other trauma experiences. Thus, those with PTSD might be more likely to use self-destructive behaviors such as eating disorder symptoms to cope with relatively minor stressors. Those who are abused frequently engage in self-denigration and self-blame as a response to the abuse and the secrecy that usually surrounds abuse (Pitts & Waller, 1993). Waller and Everill (1995) reported that higher levels of self-denigration are linked to greater frequency of purging.

Complex PTSD

Research by Herman (1992a; 1992b), van der Kolk (1987; van der Kolk & Fisler, 1994), and colleagues has described a symptom cluster reliably found in victims of these traumas caused by humans, specifically trauma that is ongoing and begins at a young age. These symptoms include: alterations in regulation of affect and impulses (including chronic dysphoria, suicidal preoccupation, and/or explosive or inhibited anger), alterations of attention or consciousness (including amnesia, dissociative experiences, and flashbacks), alterations in perception of self, others, and perpetrators (including a sense of personal shame and damage, inability to trust others, and a sense of a special relationship with the perpetrator), and alterations in systems of meaning. Herman (1992a; 1992b) referred to this symptom cluster as *Complex PTSD.*

Complex PTSD is based on the notion that exposure to continual, recurring, or multiple traumas, especially during childhood, results in a complex symptom presentation that includes not only posttraumatic stress symptoms, but also other symptoms. These symptoms include disturbances in affective and interpersonal self-regulation such as difficulties with dissociative symptoms, aggressive or socially avoidant behaviors, anxious arousal, and anger management (Cloitre et al., 2009). Kessler (2000) indicated that studying this symptom cluster is important because the majority of trauma survivors fall into this category. To date, child abuse has been the main focus of research on Complex PTSD. Therefore, Complex PTSD has been informed by developmental research, which has shown that child abuse and other childhood hardships (i.e. neglect, emotional abuse, absent, or psychiatrically disturbed parents) result in impairments in developmental processes associated with the growth of emotion regulation and related

skills in effective interpersonal behaviors (Shipman, Edwards, Brown, Swisher, & Jennings, 2005; Shipman, Zeman, Penza, & Champion, 2000). Understanding Complex PTSD can be helpful in making sense of the seemingly contradictory symptoms that often present in patients with trauma histories.

Trauma theorists have claimed that these symptoms emerge because child abuse robs the child of the chance to develop a secure attachment with the caregiver (Herman, 1992). Furthermore, in many cases, the very person who is supposed to protect is perpetrating or allowing the abuse to continue. Instead of developing a secure identity, the child develops a trauma-based identity, which is characterized by conflicting desires. The child simultaneously longs for the comfort and care of relationships but also fears the potential for these relationships to end in exploitation, abandonment, and betrayal (Herman, 1992).

Van der Kolk (1987) called Complex PTSD a "disorder of hope." He said that those who are traumatized before an organized sense of self and world can develop are deprived of the chance to ever develop a secure system of meaning and may find themselves preoccupied by an indescribable sense of anguish and deep-seeded feelings of being worthless, alone, and hopeless. Van der Kolk (1987) asserted that in order for a child to develop a belief in a just world, he or she must experience a secure, predictable environment as a child. This environment allows the child to learn that although life includes disappointments and injustices, attachments with important individuals can be continued. When children experience abuse, previously internalized systems of meaning and beliefs can be destroyed (Herman, 1992; van der Kolk & Fisler, 1994).

Trauma researchers have argued that deficits in self-regulation may be the most devastating and long lasting effects of child abuse and neglect (van der Kolk, 1994). A

secure environment allows children to gradually internalize self-regulating functions such as self-soothing and self-modulating and learn increased frustration tolerance. If this primary bond is disrupted, however, such as in the case of early abuse and trauma, this process that is so critical for healthy development is halted both biologically and psychologically. The child instead is likely to rely on basic defensive functions such as dissociation and the use of external mechanisms such as eating, self-harming, or engaging in sexual behaviors to regulate internal affect (van der Kolk, 1994).

According to Wilbur (1985) and Kluft (1985), when a child perceives an event or ongoing series of events as traumatic, that developmental stage of personality and ego development and all stages thereafter are affected. Wilbur asserted that childhood trauma does not necessarily lead to one specific psychiatric disorder but can result in different presentations based on the following four variables: (1) genetic factors and the response of the environment to the incident(s), (2) types and source of the abuse, (3) age of the individual and duration of the abuse, and (4) the child's ability to manage developmental tasks (Wilbur, 1985).

The process by which people learn to make sense of the world and develop trust in their perceptions is severely distorted in people who have experienced childhood trauma. The traumatized individual is taught about reality from the very environment that is allowing the abuse to take place. The child's original cries against maltreatment and the environmental response to any disclosure has been found to be important (Everill & Waller, 1995; Rorty et al., 1993; Vanderlinden & Vandereycken, 1993; van der Kolk, 1987; Waller, Ruddock, & Pitts, 1993). If the abused person perceives an adverse response or a lack of response when they choose to disclose, negative psychological

effects are likely to ensue (Everill & Waller, 1995; Waller & Ruddock, 1993). If a young girl's disclosure is believed and she receives support and protection, her sense of the world and internal reality remains intact. More often, however, what occurs is that the young girl is told that the abuse is her fault, that she acted in such a way as to bring it upon herself, that she is overreacting, or that she has misinterpreted the situation (Rorty & Yager, 1996a). When this is the case, the young girl may convince herself that she imagined the abuse and become too afraid to ever tell her secret. This scenario can lead to distortions in the girl's logic as she tries to make sense of the connection between the external world and her internal perceptions.

Using the notion of complex PTSD can help make sense of the numerous, sometimes paradoxical facets of disturbed eating behaviors in the traumatized patient. It can also assist with understanding symptoms and their intensity, repetitiveness, and tenacity. It can help explain the tendency to dissociate, to engage in self-harm, and to present as comorbid with other disorders. This conceptualization also provides insight into the historical tendency of eating disorders to be resistant to traditional treatments (Rorty & Yager, 1996a).

The occurrence of trauma in childhood, particularly chronic abuse inflicted by another human being beginning at an early age, has a significant effect on developmental processes. Research has repeatedly confirmed that trauma in childhood leads to pervasive psychobiological dysregulation, which increases the risk of a variety of forms of psychopathology, including eating disorders (de Bellis, Lefter, Trickett, & Puttnam, 1994; de Bellis et al., 1994; Field, 1996; Johnson & Connors, 1987; van der Kolk & Fisler, 1994). Current empirically supported trauma theory is based on the idea that

childhood trauma produces marked changes in psychobiological processes (Kraemer, 1992, Putnam & Trickett, 1997; van der Kolk, 1987) and neurotransmitter functioning (Charney et al., 1993; Post et al., 1994; Putnam & Trickett, 1997; van der Kolk, 1987), which are associated with eating behaviors (Brewerton, 1995).

Herman (1992) has hypothesized that the complex PTSD formation helps explain the relationship between childhood trauma and somatoform disorders. Although research on somatic symptoms in eating disordered patients is limited, the research that has been done has shown that eating disordered patients with comorbid PTSD report many more somatic symptoms than those without PTSD (McFarlane, Atchison, Rafalowicz, & Papay, 2004; Tagay et al., 2010). Somatic and dissociative symptoms often occur together. McCallum et al. (1992) reported that fainting, dizziness, weakness, and menstrual abnormalities are common among abused women with eating disorders. In children, complex causal pathways may be precipitated by trauma, which include a variety of psychopathological states including affective and dissociative and serve to moderate the relationship between traumatic experiences and later disturbed eating.

Affective Symptoms

A longitudinal study by Stice, Killen, Hayward, and Taylor (1998) indicated that negative affect predicts onset of binge eating behaviors. Depression (Briere & Conte, 1993; Briere & Runtz, 1988) and anxiety (Briere & Runtz, 1987; Molnar, Buka, & Kessler, 2001) have been significantly correlated with child sexual abuse. Likewise, both depression and anxiety have been found to co-occur with eating disorders (Bailey, 1994; Fairburn & Cooper, 1984; Garfinkel et al., 1995; Herzog, 1982; Katzman & Wolchik, 1984; Mizes, 1988; Rohde, Lewinsohn, & Seeley, 1991; Valdiserri & Kihlstrom, 1995).

Findings regarding emotional abuse and eating disorders are conflicted (Kent et al., 1999; Mazzeo & Espelage, 2002). Kent et al. (1999) did not find depression to be a moderator, but Mazzeo & Espelage (2002) did.

In the past, research has indicated that both depression and anxiety are specific risk factors in the development of eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Mazzeo, Mitchell, & Williams, 2008). It remains unclear, however, how depression relates to eating disorders. Depression may be a cause, effect, or comorbid condition. Research by Measelle et al. (2006) indicated that symptoms of depression and disordered eating covary together over time. Zaider, Johnson, and Cockell (2000) found adolescents with eating disorders have significant levels of dysthymia. Some have suggested that the most prevalent psychiatric problem for female adolescents is depression and nearly 20% experience clinically significant depressive symptoms as teenagers (Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn et al., 2003). A study by Halmi et al. (1991) reported that 68% of patients with anorexia nervosa also had major depressive disorder. Research has suggested that elevated depressive symptoms significantly predict later onset of bulimia over two- and three-year spans (Stice, Burton, & Shaw, 2004; Stice, Presnell, & Spangler, 2002). Research by Juarascio, Perone, and Timko (2011) demonstrated that depression moderated the relationship between disordered eating and body image dissatisfaction. Body image dissatisfaction involves dissatisfaction with weight and shape, fear of gaining weight, and the desire to lose weight and has been identified as a risk factor for eating disorders (Kluck, 2009).

In a study by Kong and Bernstein (2009) depression was reported as a significant mediator between physical neglect and both drive for thinness and body dissatisfaction.

Kaufman et al. (1997) demonstrated that depressed children who were victims of childhood abuse showed a pattern of hypothalamic/pituitary/adrenal axis responding that differed from depressed children who were not abused. Cooper and Cowen (2009)'s research also cited the relationship between eating disorders and major depression. According to this study, self-loathing, hatred, and disgust were found in those with eating disorders at high rates. This research pointed out that those with eating disorder symptoms seem to be "repelled by the self" (Cooper & Cowen, 2009; p. 158).

Women with bulimia nervosa often have reported body dissatisfaction and depression (Cooper & Fairburn, 1993; Cooper & Hunt, 1998). In a study by Anderson, LaPorte, Brandt, and Crawford (1997), it was demonstrated that inpatients with bulimia who had a history of abuse had higher levels of depression, anxiety, and attitudes consistent with eating disorders than those who did not have a history of abuse. General psychopathology, which includes high levels of anxiety and depression, has been implicated as a moderator of the relationship between anxious attachment and bulimic symptoms (Schembri & Evans, 2008). The most parsimonious explanation for the association between body dissatisfaction and depression is that bulimia serves as a moderator (Keel, Mitchell, Davis, & Crow, 2001). Several studies, however, have suggested that a significant association exists between body dissatisfaction and depression in samples of women without bulimia (Allgood-Merten, Lewishon, & Hops, 1990; Joiner, Schmidt, & Singh, 1994; Keel et al., 1997; Leon, Fulkerson, Perry, & Cudek, 1993; McCabe & Marwit, 1993; Rierdan & Koff, 1997; Roth & Armstrong, 1993; Taylor & Cooper, 1986).

These results could be explained by two possibilities (Keel, Mitchell, Davis, & Crow, 2001). First, the relationship between body dissatisfaction and depression could exist independently from bulimic symptoms. Some research has supported this explanation; the literature on depression has reported connections between increased symptoms of depression and decreased ratings of physical attractiveness in non-eating disordered individuals (Noles, Cash, & Winstead, 1985). If this is the case, then depression might contribute to the onset or maintenance of body dissatisfaction, which may in turn contribute to the development or maintenance of bulimia. A second explanation is that body dissatisfaction and depression are not independent of bulimic symptoms. McCabe and Marwit (1993) and Allgood-Merten, Lewinsohn, and Hops (1990) reported significant correlations, indicating that the relationship may be due to undetected bulimia. Keel et al. (2001) reported that the association between depression and body dissatisfaction existed independently of bulimic symptoms. In their study, both symptoms of depression and bulimia significantly predicted coexisting body dissatisfaction. Further, bulimic symptoms accounted for a larger proportion of the variance in body satisfaction than depression. In a follow up analysis by Keel et al., neither bulimic symptoms nor depression displayed greater significance in predicting body dissatisfaction. Keel et al. (2001) concluded that a reciprocal relationship might exist between body dissatisfaction and depression in which both contribute to each other. Based on their results, they concluded that vulnerabilities to bulimia might develop from increased tendencies toward body and/or weight dissatisfaction as a result of negative affect.

Research has suggested that symptoms of anxiety and eating disorders are common among girls with depression (Measelle, Stice, & Hogansen 2006). Kent et al. (1999) indicated that anxiety significantly moderated the relation between child emotional abuse and eating disorders. Anxiety disorders are one of the most frequent comorbidities of eating disorders (Chesler, 1995; Tagay, Schlegl, & Senf, 2010). Bulik (1995) reported that 75% of eating disorder patients have experienced one anxiety disorder in their lifetime. A higher percentage of women with eating disorders compared with non-eating disordered controls have reported lifetime anxiety disorders that predated the onset of eating disorder symptoms (Brewerton et al., 1995; Bulik, Sullivan, Fear, & Joyce, 1997; Schwalberg et al., 1992; Silberg & Bulik, 2005).

Anxiety has been implicated as a central factor in the pathogenesis and maintenance of anorexia and bulimia nervosa (Fitzsimmons & Bardone-Cone, 2011). A study by Jones, Leung, and Harris (2006) demonstrated that three core beliefs were important in predicting eating psychopathology: fears of abandonment, feelings of being fundamentally flawed or inferior, and beliefs of vulnerability to harm. Additionally, anxiety symptoms often remain after the eating disorder has been successfully treated (Bardone-Cone et al., 2010). Symptoms of anxiety often present even if criteria for diagnosis are not met (Ahren-Moonga, Holmgren, Von Knorring, & Klinteberg, 2008; Kaye et al., 2004; Mizes, 1988). Additionally, the presence of anxiety may predict poor treatment outcomes for eating disorder patients (Fichter, Quadflieg, & Hedlund, 2006).

Fears and anxiety about weight gain are central to the experience of anorexia nervosa. Anxiety over the social evaluation that is associated with excessive concerns about shape, eating and weight is often present (Schwalberg et al., 1992). These factors are often

implicated in the etiology of eating disorders (Fitzsimmons & Bardone-Cone, 2011). Anorexia often includes a phobic-like avoidance of high calorie foods and part of the diagnostic criteria is an intense fear of becoming fat (APA, 2000; Crisp, 1967). Intense fears often extend from not only food to also settings that involve food and eating. Davey and Chapman (2009) found in their research that the experience of disgust is common among those with eating dysfunction. They hypothesized that this may be linked to anxiety or anxious sensations. Further, they suggested that disgust might be related to a fear of contamination and the belief that by ingesting food, one will become contaminated by that substance. In bulimia, purging has been described as an anxiety reducing technique. In both anorexia and bulimia, many women are fearful of weight gain, are intensely self-critical, and fear negative evaluation by others (Bulik, 1995).

Anxiety and eating disorders have been investigated from three perspectives. First, parallels exist in symptomology. The binging and purging present in bulimia and the eating related rituals of anorexia have been compared to the compulsive behaviors of obsessive-compulsive disorder. Also, the negative evaluation found in eating disorders has been compared to social phobias. These similarities have suggested an underlying psychopathology that is similar even though symptom expression may be different. Second, patterns of comorbidity and family history tend to be similar in both samples of women with eating disorders and anxiety disorders. Third, the same behavioral and psychopharmacological treatments have been effective applied for both types of disorders (Bulik, 1995).

Based on these studies, it is plausible that depression and anxiety play similar roles in regard to the relationship between child sexual abuse and disordered eating (Hund &

Espelage, 2005). Some have argued that the reason for the contradictions in this area is the fact that anxiety and depression should not be studied as separate constructs, but instead be investigated for the general component of distress that they share (Hund & Espelage, 2006; Watson et al., 2005). In research by Hund and Espelage (2006) and Watson et al. (2005) anxiety and depression were seen to be overlapping constructs. Juarascio, Perone, and Timko (2011) found that both state and trait anxiety were significantly associated with subclinical levels of eating disturbance. They found that more affective symptoms were associated with more severe eating pathology and body image dissatisfaction. Fairburn, Cooper, and Shafran (2003) suggested that anxiety maymight contribute to mood intolerance. Mood intolerance has been described as an inability to cope appropriately with emotional states. Usually, poor coping emerges during negative emotional states such as depression or anxiety, but for some, even positive intense moods can lead to difficulties with coping. Their research demonstrated that the inability to deal with emotions puts people at risk for engaging in dysfunctional behaviors such as eating pathology.

Multiple cross sectional studies have demonstrated a relationship between emotional dysregulation and a wide range of pathology (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Bohnert et al., 2003; Silk et al., 2003; Sim & Zeman, 2006; Zeman et al., 2002). Thompson (1994) defined emotion regulation as: “the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals” (p. 28). Emotional dysregulation, therefore, can be understood as the opposite of this concept. McLaughlin et al. (2011) demonstrated results confirming these findings in a prospective

analysis, indicating that emotional dysregulation increases the risk for pathology. They showed that emotional dysregulation predicted the subsequent development of multiple forms of pathology including anxiety and eating pathology. Depression, however, was not predicted by emotional dysregulation in this study.

Even though research does not always find a correlation between depression and/or anxiety to be significantly correlated with eating disorders, results often show that up to two thirds of patients with eating disorders also have clinical levels of depression and/or anxiety (Bulik et al., 1996). Also, girls with subclinical anorexia nervosa, bulimia nervosa, and binge eating disorder have been shown to have higher prevalence of separation anxiety diagnosis and generalized anxiety symptoms compared with girls reporting no eating disorders (Touchette et al., 2011).

Both depression and anxiety are forms of negative affect that can be potentially overwhelming to survivors of childhood sexual abuse, motivating them to engage in maladaptive survival strategies such as disordered eating (Hund & Espelage, 2005). Disturbed eating behaviors, while highly destructive, are also highly effective at regulating stress and painful affect (Casper, 1983; Johnson & Connors, 1987). Herman (1992b) asserted that abused children usually discover at some point during development that they have the ability to induce major alterations in affect by inducing autonomic crisis or arousal. Before binging many patients report feeling inadequate and out of control. While binging, many patients continue to report high levels of negative affect (e.g. anger, guilt, feelings of inadequacy). While purging, however, patients report marked decreases in anger and negative affect. These reports have led researchers to speculate that women might binge in order to reduce tension and seek affective balance

(Casper, 1983; Johnson & Connors, 1987; Johnson & Larson, 1982). Purging can become the method through which abused children attempt to regulate their emotions when they do not have any other tools to cope with internal distress (Herman, 1992b).

Lazarus and Folkman (1991) asserted that coping is a multi-dimensional construct that involves thoughts and behaviors used by people as attempts to manage, tolerate, or reduce burdens that they feel exceed their resources. Women in general, and especially women with eating disorders have been found to use emotional coping styles (Davies, Bekker, & Roosen, 2011). Reduction of eating disorder symptoms has been correlated with learning to use more active coping styles and being able to seek social support (Davies, Bekker, & Roosen, 2011; Fitzsimmons & Bardone-Cone, 2011).

Trauma theory also suggests that for some women, negative affect contributes to ongoing eating disorder symptoms. One theory, offered by Schwartz and Gay (1996) stated that overwhelming stress triggers a numbing response that is followed by intrusive negative memories and affect. From a stress-vulnerability perspective, the experience of any form of child abuse is postulated to lead to problems with trust and maintaining appropriate boundaries. Chronic abuse then becomes associated with a decrease in a child's self-concept and self-esteem. According to this theory, this diminished sense of self leads to difficulty in managing strong affect, heightening the risk for general distress such as depression and anxiety and maladaptive coping strategies in adulthood like eating disorders (Follette, Ruzek, & Auberg, 1998). Also, self-denigration has been thought to contribute to the depressing thoughts and emotions that bulimic behaviors serve to reduce (Heatherton & Baumeister, 1991).

Attachment Styles

Since the earliest case descriptions of eating disorders, families have been implicated in their pathogenesis and maintenance (Marce, 1860). Emphasis on these factors has waxed and waned over the years, peaking in the 1970s (Minuchin, Rosman, & Baker, 1978; Palazzoli. 1974). During this decade, Hilde Bruch began writing about more specific difficulties in the mother-daughter dyad (Bruch, 1974b). Her description included the description of a mother who projects her own needs onto her infant daughter in such a way that the infant's needs and her own needs are poorly differentiated. This leads the child to difficulties with the development of a separate identity as well as pervasive problems with feeling ineffective (Ward, Ramsay, Turnbull, Benedettini, & Treasure, 2000).

John Bowlby (1969, 1980, 1982) applied ideas from psychoanalysis, ethology, and learning theory to the mother-infant relationship, developing what would become known as attachment theory. He suggested that the mother-infant relationship is influenced by a drive, which is different from and more powerful than hunger. Adult attachment styles have been thought to be the result of internalized working models that develop from infant drives.

Even in nonclinical samples, attachment problems have been linked to disordered eating behaviors (O'Kearney, 1996; Wara, Ramsay, & Treasure, 2000). Research has demonstrated that high rates of attachment anxiety and avoidance are associated with eating disturbances (Troisi, Massaroni, & Cuzzolaro, 2005; Cash, Theriault, Milkewicz, & Annis, 2004; Evans & Wertheim, 2005; Miljkovitch et al., 2005). Additionally, research has shown that women who have eating disorders have significantly higher

levels of attachment insecurity than women without eating disorders (Barone & Guiducci, 2009; Fonagy et al., 1996; Illing et al., 2010; Illing et al., 2011; Troisi et al., 2006; Ward et al., 2001). Illing et al. (2011) demonstrated that women with eating disorders had significantly higher levels of attachment insecurity compared to other psychiatric patients. Some have hypothesized that people with high levels of attachment anxiety seek out close relationships and idealize others while devaluing themselves (Ainsworth, Blehar, Water, & Wall, 1978; Chassler, 1997; Mikulincer, Shaver, & Pereg, 2003). Those with avoidant attachment styles, however, attempt to meet their own attachment needs, avoiding relationships with others. People with avoidant attachment styles, however, strive to activate their attachment needs and avoid relationships with others.

Swanson et al. (2010) demonstrated that maternal bonding serves as a moderator in the relationship between avoidant coping styles and eating pathology. Further, patients with anorexia reported lower levels of parental care than controls. Also, they reported more negative and avoidant coping styles. In this study, disordered eating was significantly correlated with reports of low maternal care and high levels of control (Swanson et al., 2010).

Greenwood and Pietromonaco (2004) demonstrated that women with high levels of attachment anxiety report identifications with and idealizations of people in the media. They offered that attachment needs might influence this involvement with the media, which may in turn exacerbate body concerns. They also suggested that social comparisons moderate the relationship between attachment anxiety and eating disorders.

This research also supported the idea that people who are highly anxious about relational involvement may also be hypervigilant.

Orzolek-Kronner (2002) suggested that eating disorder symptoms might be an attempt to beckon parental attention, such as what Bowlby described in his concept of proximity seeking. Furthermore, Freud (1949) described the feeding experience as the most primary means of creating physical proximity between mothers and their infants (Freud, 1949). Patients with eating disorders might worry that if their symptoms abate, they will have less contact with their mothers. For example, a girl with bulimia's mother might pay close attention to her, looking for symptoms, which replicates the closeness that existed during early development. Continuing to participate in behaviors such as self-starvation, binging, or purging, might increase the amount of time spent closely with caretakers, both increasing physical and psychological intimacy (Orzolek-Kronner, 2002). A study by Orzolek-Kronner (2002) supported this idea, indicating that adolescents with eating disorders evidenced more proximity seeking behaviors than their nonclinical cohorts.

Interestingly, a hunger for closeness has been reported in families with eating disorders, which often are already too close (Bruch, 1973, 1978; Friedberg & Lyddon, 1996; Minuchin et al., 1978). Theories have been suggested regarding the idea of a mother/daughter enmeshment in anorexic families (Bruch, 1973; Minuchin, Rosman, & Baker, 1978; Zerbe, 1993). Enmeshment is a style of relatedness where family members are highly involved with one another. This style is comprised of excessive togetherness, lack of privacy, intrusiveness, and poorly defined boundaries within the family (Weiss, Katzman & Wolchik, 1985). Children who grow up in these types of families learn that

family loyalty is of utmost importance, which can lead to eventual difficulties with separation and individuation (Blinder, Chaitin & Goldstein, 1988).

Therefore, the separation-individuation predicament might be a precursor to the development of eating disorders, but the actual source might be related to the quality of the original attachment rather than the perceived closeness with the mother (Johnson & Flach, 1985; Kenny & Hart, 1992; Orzolek-Kronner, 2002). Research has supported the idea that in eating disorder cases, Bowlby's secure base might have been compromised, and that symptoms might be an attempt to repair this (Orzolek-Kronner, 2002).

Characteristics of Abuse

Browne and Finkelhor (1986) proposed that three interconnected characteristics influence the outcomes of child sexual abuse: abuse characteristics, the child's personality, and contextual influences. In terms of abuse characteristics, Waller (1992) found that among women with bulimia and history of sexual assault, binging and purging occurred more frequently if the perpetrator was a relative. These symptoms were more marked when the abuse was intrafamilial, involved force, or occurred before the victim was 14 years old. Women who experienced intrafamilial abuse also engaged in more frequent purging (Waller, 1992b). Studies by Calam and Slade (1989) and Abramson and Lucido (1991) also indicated that characteristics of abuse were related to the severity of eating symptomology.

Similarly, Baldo and Baldo (1996) reported that eating pathology occurs at a significantly higher rate among women with histories of intrafamilial sexual assault compared to women with extrafamilial abuse histories. Furthermore, Hastings and Kern (1994) concluded that abuse victims reporting long-term abuse by a parental figure were

more likely to exhibit severe bulimic symptomology. Smolak, Levine, and Sullins (1990), however, found that neither abuse severity (act and frequency) nor familiarity with the perpetrator was related to eating disordered behaviors or attitudes. Finn, Hartmann, Leon, and Lawson (1986) also reported no differences in rates of bulimia when considering relation to the perpetrator.

Some research has also suggested that women with eating disorders report abusive experiences that are different in nature from those reported by women in control groups (Waller, 1992a). The nature and severity of the abuse may also be associated with degree of eating pathology. Waller (1992a; 1992b) also found that if the abuse occurred prior to age 14 or involved physical force, the frequency of binging and purging episodes rose significantly ($p<.001$) and explained 56% and 42% of the variance, respectively. Additionally, Hastings and Kern (1994) demonstrated a relationship between severity of bulimic symptoms and severity of sexual abuse. Anderson, LaPorte, Brandt, and Crawford (1997), however, reported that severity of sexual abuse (specific act and frequency) was not related to severity of eating or overall psychiatric disturbance despite their finding that having a history of abuse was correlated higher levels of eating-disordered attitudes, depression, and anxiety compared to those without a history of abuse.

Much of the research has focused on the direct correlation between child sexual abuse and disordered eating, disregarding the potential relationship of child sexual abuse with other possible risk increasing correlates (Smolak & Murnen, 2002). Since research has underscored the complexity of the relationship between sexual abuse and eating disorders, researchers have tried to sort out the variables that could influence which

women develop disordered symptoms and which do not. Understanding certain characteristics of abuse (e.g., duration of abuse, number of incidents, relationship with perpetrator, age of child, level of aggression) may elucidate some of the remaining mysteries about the developmental etiology of disordered eating symptoms (Lange et al., 1999; Russell, 1984; Sirles & Lofberg 1990). Another difficulty in researching female psychiatric inpatients with regards to childhood sexual abuse and eating disorders is the fact that the work is often complicated by the fact that a majority of them have a history of childhood sexual abuse (Chu & Dill, 1990).

Furthermore, Anderson, LaPorte and Crawford (2000) analyzed several variables that might be relevant (e.g., identity of the perpetrator, number of incidents of abuse, age at time of abuse, use of force, presence of physical abuse, and nature of disclosure) but did not find these factors to be significant predictors of eating pathology differences in a sample of women with clinical levels of bulimia. Research by Waller, Ruddock, and Pitts (1993) demonstrated that another critical factor seems to be the reaction of others to the individual's initial disclosure of abuse. Negative reactions to initial attempts at disclosure were correlated with higher frequencies of binging and purging compared to those who received positive and supportive reactions. Research by Pitts and Waller (1993) supported the idea that self-denigratory beliefs may moderate the relationship between sexual abuse and specific symptoms of bulimic eating disorders. They concluded that vomiting is a response to the self-denigratory cognitions and emotions that follow abuse, rather than to the actual abuse per se.

Although research on this topic has appeared contradictory, mixed findings are consistent with the complexity of sexual abuse and the idiosyncratic nature of each case

(Anderson, LaPorte, & Crawford, 2000). Anderson, LaPorte, and Crawford (2000) suggested that when looking at clinical levels of bulimia, the details of the initial incidents of abuse might be irrelevant. That is, they supposed that when a certain threshold of trauma is reached, the individual details of the incident no longer impact the way eating pathology is expressed. They suggested that further research should include large samples that involve a mixture of clinical and nonclinical participants.

Mediating Influences

Dissociation

A number of studies have concluded that dissociative tendencies are common in women with eating disorders (Abraham & Beumont, 1982; Demitrack, Putnam, Brewerton, Brandt, & Gold, 1990; Vanderlinden, Vandereycken, VanDyck, & Vertommen, 1993). Further, Valdisseri and Kihlstrom (1995) showed that the use of dissociation is high among those with unhealthy eating attitudes in nonclinical populations. As early as 1979, Russell reported that bulimic patients describe episodes of binging as times when they lose awareness. Brown, Russell, Thornton, and Dunn (1999) pointed out that several theorists (Demitrack, Putnam, Brewerton, Brandt, & Gold, 1990; Heatherton & Baumesiter, 1991; McCallum, Lock, Kulla, Rorty, & Wetzel, 1992; Miller, McCluskey-Fawcett, & Irving, 1993; Waller, 1993; Everill & Waller, 1995) have suggested that the binge-purge cycle occurs in a dissociative state or actually serves to create a dissociative state. According to these theorists, intolerable affect is expressed through dissociation and those who have difficulty with affect regulation tend to rely on dissociation in times of distress. Patients who use dissociative coping mechanisms have commonly reported that they attempt to numb feelings.

Specifically, dissociation has been linked to bulimic symptoms (Abraham & Beumont, 1982; Chandarana & Malla, 1989; Everill, Waller, & Macdonald, 1995; Johnson, Lewis, & Hagman, 1984; Torem, 1986). Miller, McCluskey-Fawcett, and Irving (1993) demonstrated that abused women with bulimia dissociated significantly more than nonabused women with bulimia. Patients have often described episodes of binging as being numbing and a way to separate one's self from affective states. Binge-purge cycles seem to provide relief from negative affect (Bailey, 1994).

Early studies have suggested that the relationship between eating disorders and dissociation might be explained by the connection between trauma and dissociation (Binder, 1981, Herzog et al., 1993; McCarthy & Thompson, 2010; Tobin, Molteni, & Elin, 1995; Vanderlinden et al., 1995). Dissociation occurs when children experience a traumatic event that is too overwhelming to incorporate into their psyche and thus repress the event from their consciousness (Bailey, 1994). Research has shown that people who experience dissociation and have a trauma history often have a reported history of sexual abuse (Briere & Runtz, 1988; Sanders & Giolas, 1991; Sanders, McRoberts, & Tollefson, 1989). Dissociation has been shown to reduce negative cognitive states and is often used as a defense mechanism when physical escape is not possible because it helps to reduce awareness of trauma through depersonalization, derealization, amnesia, and absorption. In those who have experienced trauma and present with eating disorder symptoms, dissociation is often used because the past trauma is experienced though current events.

A number of studies have demonstrated that the affective disturbances seen in dissociation are related to eating disorders and an abuse history (Bailey, 1994; Demitrack, Putnam, Brewerton, & Brandt, 1990; Everill, Waller, & Macdonald, 1995; McCallum,

Lock, Kulla, Rorty, & Wetzel, 1992; Miller, McCluskey-Fawcett, & Irving, 1993; Reto, Dalenberg, & Coe, 1993). Some authors have suggested that dissociation might be a mediator in the relationship between early abuse and bulimic symptoms (Everill & Waller, 1995). Binge eating itself has also been suggested to operate as a form of autohypnosis. Concentrating on the task of eating seems to induce numbing, tension reduction, and relief from dysphoric affect. Purging both eliminates the negative consequences of feared weight gain and provides a sense of soothing, cleansing, and relief (Brown, Russell, Thornton, & Dunn, 1999).

A number of authors have contended that there might be a three-way relationship between reported history of sexual abuse, bulimic eating disorder, and dissociation (Miller, McCluskey-Fawcett & Irving, 1993; Vanderlinden et al., 1993). Briere (1992) hypothesized that dissociation might act as a critical mediating factor linking childhood abuse and bulimia. Everill, Waller, and Macdonald (1995) demonstrated that women who reported abuse displayed greater levels of dissociation and that dissociation mediated the relationship between sexual abuse and frequency of binging. They were unable to demonstrate, however, that either dissociation or sexual abuse could account for the severity of purging. This non-finding was consistent with Briere's (1992) model describing eating disorder behaviors as attempts to reduce tension.

Some have argued that dissociation is an underlying factor in the experience of posttraumatic stress disorder (Farber, 2008). PTSD symptoms, especially avoidance symptoms, have been correlated with dissociative symptoms and psychiatric patients with trauma histories have consistently manifested high levels of dissociation (Chu & Dill,

1990; Coons et al., 1989; Nash et al., 1993; Sanders & Giolas, 1991; Saxe et al., 1993; Waldinger et al., 1994).

Lacey et al. (1986) and Root and Fallon (1989) suggested that binging and purging behaviors serve a number of defensive purposes and that people with abuse histories are especially vulnerable to using those defenses. Specifically, some have asserted that a temporary cognitive narrowing is experienced during a binge, which focuses attention on the high priority stimuli. This reduced focus has been proposed to reduce self-awareness (Heatherton & Baumeister, 1991). The tendency to run away from awareness has appeared to utilize cognitive mechanisms that are akin to dissociation. Additionally, Bailey (1994) suggested that a part of the ego takes on the role of the abuser and continues the abuse by self-punishing. This theory is consistent with results from previous studies by researchers such as van der Kolk, Perry, and Herman (1991) who reported strong positive correlations between a childhood trauma history and self-destructive behaviors in adulthood such as cutting, burning, or picking.

Braun (1984) described dissociation as an adaptive defense used by healthy individuals. The potential to rely on dissociation to a pathological degree, however, becomes heightened for those who are raised in chronic, unpredictable, traumatic environments (Braun, 1984). Traumatic environments have been defined in past research as environments that hold any or all of the following: physical abuse, emotional abuse, sexual abuse, domestic violence, family chaos, significant separations from primary caregivers, and neglect (van der Kolk, Perry, & Herman, 1991). Others have described dissociation as an adaptive trauma response that simultaneously allows the psychic escape from inescapable, overwhelming terror and leads to feelings of extreme internal

deadness and disconnection (Rorty & Yager, 1996a). Eating disorder patients have tended to have higher levels of dissociation compared to the general population and those who dissociate tend to have more severe eating disorder symptoms and be more psychiatrically disturbed overall (Demitrack et al., 1990; Everill et al., 1995). Patients with anorexia nervosa have often described starvation as being accompanied by altered perceptions and a sense of distance from reality (Schupak-Neuberg & Nemeroff, 1993).

Many studies have indicated that hormonal changes might trigger symptoms because they serve as a reminder of past trauma (Miller et al., 1993; Putnam & Trickett, 1997; Thomas, 1995; Vanderlinden et al., 1993). Sanders (1986) theorized that dissociative episodes are triggered by the same anxiety that causes binging episodes. He further asserted that dissociation in those who engage in binge eating is more than simply a defense mechanism. He suggested that it might be part of a personality trait, which works to reduce emotions that seem intolerable. Torem (1991) went even further, suggesting that a specific subtype of eating disorders exists, called *dissociative eating disorders*.

Family Dysfunction

The role of the family has long been implicated in bulimic etiology (Connors & Johnson, 1987; Kog & Vandereycken, 1989; Vanderlinden & Vandereycken, 1993; Hastings & Kern, 1994; Waller & Calam, 1994; Everill & Waller, 1995; Rupp & Jurkovic, 1996). Studies have shown that people with eating disorders tend to have more family pathology as compared with controls (Steiger et al., 1991), including more conflict and disorganization (Kog et al., 1985, 1989), lower adaptability and cohesion (Waller et al., 1990), low maternal and paternal care (Palmer et al., 1988), high paternal over-protectiveness (Calam et al., 1990), less cohesion (Crowther, Kichler, Sherwood, &

Kuhnert, 2002), less expressiveness, less orientation towards recreational activities, more conflict, and less emotional support (Grisset & Norvell, 1992; Grotevant & Cooper, 1986; Limbert, 2010; Shisslak et al., 1990).

Although Minuchin's model of the psychosomatic family has been largely discredited (Eisler, Dare, Russell, Szmukler, le Grange, & Dodge, 1997; Fairbairn, Simic, & Eisler, 2011), its influence was once pervasive (Kog & Vandereycken, 1989; Minuchin et al., 1978). Some researchers continue to operate from this model; for example, Kog and Vandereycken (1989) indicated that families of patients with eating disorders had higher levels of conflict avoidance and rigidity. McGrane and Carr (2002) reported that young women at risk for eating disorders endorsed family difficulties in terms of problem solving, roles, affective responsiveness, and general functioning. They also found that these women reported mothers who had significantly more problems with somatization, interpersonal sensitivity, depression, anxiety, anger, hostility, and paranoid ideation (McGrane & Carr, 2002).

Jones et al. (2006) demonstrated that perceived paternal rejection leads to the development of both a fear that significant others will not be able to continue providing emotional support and also underlying feelings of shame and inferiority. In this research, the presence of both of these beliefs significantly predicted eating psychopathology beyond the extent of either belief alone (Jones et al., 2006). Further supporting the important role of families in eating pathology, Munoz, Isreal, and Anderson (2007) demonstrated that activities such as family routines and rituals such as mealtimes serve as protective factors.

Body Dissatisfaction

Studies have demonstrated that body dissatisfaction is the strongest predictor of eating disorders and subclinical eating disturbances (Levine & Smolak, 1996; Stice 2001; Striegel-Moore & Bulik, 2007). Tylka (2004) reported that neuroticism and body surveillance are mediators of eating disorder symptoms and also increase the risk that women with low body satisfaction will demonstrate eating dysfunction. Body surveillance is an indicator of objectification and is made up of routine body monitoring and thinking of one's body in terms of how it looks rather than how one feels (Tylka, 2004). Moreover, body dissatisfaction has been implicated in some research as a mediator in the relationship between unwanted sexual experience and eating disorders (Murray, Macdonald, & Fox, 2008). Research by Calam, Griffiths, and Slade (1997) however, failed to demonstrate that body dissatisfaction was a strong mediator between sexual abuse and eating disorders in a clinical sample of 212 women.

Body dissatisfaction rates have been extremely high among college women. In a study by Klemchuk, Hutchinson, and Frank (1990), over ten percent of women reported pathological weight preoccupation on the drive for thinness scale of the Eating Disorders Inventory (EDI). Pathological weight preoccupation was defined as EDI scores on the drive for thinness scale at or above the mean scaled scores of anorexic patients reported by Garner, Olmstead, Polivy, and Garfinkel (1984). In past research, body dissatisfaction has been associated with depression and poor self esteem (Dykens & Gerrard, 1986; Rosen, Gross, & Vara, 1987). Negative feelings about the body, unrealistic body image, and related cognitive-behavioral patterns have appeared to be important in understanding poor psychological adjustment in women.

Research on connections between child abuse, body image disturbance, and eating pathology has yielded mixed results. Schaaf and McCanne (1994) reported that a history of sexual abuse was not associated with increased body image disturbance. On the other hand, Waller et al. (1993; Waller, 1994a) found that specific aspects of sexual abuse were important. For example, women who reported more recent abuse tended to overestimate their body sizes more than women with earlier abuse or no abuse. No significant associations were found between body image distortion and identity of abuser or use of physical force.

In a study by Dunkley, Masheb, and Grilo (2010), childhood emotional abuse and sexual abuse were significantly linked to body dissatisfaction, whereas childhood physical abuse, physical neglect, and emotional neglect were not significantly associated with body dissatisfaction. Further, according to research by Calam, Griffiths, and Slade (1997), rape is associated with higher levels of body dissatisfaction.

Some researchers have hypothesized that abusive experiences and the later development of eating disorders are connected through characteristics commonly found in survivors of abuse: bodily shame, sense of worthlessness, and self-loathing (Nathanson, 1989). Also, women with a history of childhood abuse often have presented with high levels of self-directed aggression (Carmen, Rieker & Mills, 1984), and it has been hypothesized that vomiting is a form of aggression against the self (Rorty & Yager, 1996). Research has suggested that body-related shame is an important mediator of sexual abuse, physical abuse, and depression. This association has not been accounted for solely by body dissatisfaction or low self-esteem (Andrews, 1995). Pitts and Waller

(1993) demonstrated a correlation between self-denigratory beliefs resulting from sexual abuse and vomiting frequency in bulimic women.

Limitations of Prior Research

Previous literature has had a number of limitations. Taken together, these findings suggest that childhood sexual abuse may indeed be a risk factor for eating disorders, but research in this area is often limited by inadequate research designs and by difficulties in measuring both eating disorders and sexual abuse (Wonderlich et al., 2000). For instance, the methodology in this research has not been consistent and sample sizes have been small (Wonderlich et al., 2001). As well, definitions of abuse vary tremendously, ranging from very broad to very narrow (Rorty & Yager, 1996a). Also, abuse is often treated as a dichotomous variable: either yes it did happen or no it did not happen. Therefore, factors related to the severity often ignored. Also, factors that often influence the experience of the trauma have often been ignored such as the age of onset, the relationship to the perpetrator, the frequency and duration of abuse, the presence or absence of a trusted person in which to confide, etc (Browne & Anderson, 1991; Davidson & Smith, 1990; Roth, Wayland, and Woolsey, 1990; Wyatt & Newcomb, 1990).

Another problem with attempting to measure and research childhood emotional abuse is the probability of underreporting (Claussen & Crittenden, 1991). Methodology is also complicated by repression of abuse experiences or confabulated memories of abuse (Schmidt, Humfress, & Treasure, 1997). Other limitations include different age cutoffs for victims to be considered children, failure to adequately screen comparison groups labeled non-abused, inappropriate comparison groups, and disparate methods of identifying abuse.

Also, like all historical material, adult retrospective reports of abuse in childhood are subject to problems inherent in self-report and recall. Also, most research has been conducted with adults who are asked to recall histories of childhood sexual abuse and eating behavior. Retrospective recall bias has been a weakness of this design, since memories of childhood can be influenced by current circumstances (Wonderlich et al., 2000).

Some people have criticized this research because most of it has been conducted on college age females (Murray, 1999; Shaw & Garfinkel, 1990); however, this population is highly at risk for disordered eating symptoms (Jacobi et al., 2011; Lewisohn et al., 2000), therefore, it seems to be an appropriate population on which to conduct this research.

A study by Wonderlich et al. (2001) addressed many of the aforementioned methodological limitations. These researchers found that childhood sexual abuse increased the risk of eating disorder-related psychopathology. This study did not support the idea that this effect was merely the result of sexual trauma, as rape victims did not show comparable rates of psychopathology. Individuals who experienced both childhood sexual abuse and rape in adulthood demonstrated exceptionally elevated scores on dietary restraint and weight concerns. As a whole, these data suggest that childhood sexual abuse does have a significant effect on eating disorder psychopathology, and furthermore, that early childhood trauma may make women more sensitive to later sexual trauma.

Statement of the Problem

Despite numerous studies being conducted on eating disorders, previous literature has consistently focused on including only one or two variables. The more research is

conducted, the more evidence has suggested that the relationship among proposed etiological factors appear to be extremely complex and interrelated. A further difficulty in the trauma literature is that previous research in childhood abuse has compartmentalized forms of trauma (Rosenbery, 1987), meaning that there is little research that integrates findings across all abuse forms. This approach has meant that specific types of abuse, usually child sexual abuse, have been studied in isolation, with little or no reference to the broader spectrum of abuse experiences (Kent & Waller, 2000). A deficit exists in the literature in terms of looking at how all of the factors mentioned above might interact in order to result in disordered eating behaviors.

In addition, within the existing literature, results have been incredibly inconclusive with regard to a clear explanation for the etiology of disordered eating. Research on the results of childhood trauma has consistently yielded mixed results and supported contradictory findings. Although multiple variables have been implicated as mediators and moderators of eating pathology, a comprehensive model that sufficiently explains etiology has yet to be developed (Tylka & Subich, 2004). Many eating disorder researchers have called for additional research to be conducted, which integrates multiple correlates of eating disorders within models that can predict eating disorder symptomatology. Models are needed to explain how variables combine with one another to predict eating disorder symptoms among women. Further research in this area has been strongly recommended so that clinical prevention and early identification of eating disorders can be accomplished (Hotelling, 2001; Kasubeck-West & Mintz, 2001; Mazzeo & Espelage, 2002; Striegel-Moore & Cachelin, 1999; 2001; Tylka & Subich, 2004).

Purpose and Significance of the Study

The purpose of this study was to address the limitations of current investigations by identifying factors contributing to the development and maintenance of disordered eating behaviors. Due to the pervasive and growing nature of this type of pathology, it is important for clinicians to gain a greater understanding of which variables predict the onset of disordered eating behaviors. By investigating these factors in a nonclinical sample, insight will be gained on these relationships with the goal of eventually providing a basis for further research in a clinical population. Given the importance of early identification in the treatment of disordered eating and its comorbid conditions, practitioners need to have an understanding of what risk factors might lead to the eventual display of clinically significant symptoms. In addition, it is important to understand how multiple variables mediate and moderate the eventual expression of disordered eating. Currently, it remains a mystery as to why some people who experience childhood trauma develop eating pathology as opposed to other forms of pathology or healthy levels of adjustment. This study aimed to elucidate some of the controversies in the research on childhood trauma and the eventual development of disordered eating.

Statement of Hypotheses

Based upon the review of the literature, the following hypotheses were developed:

H1: The presence of at least one type of childhood trauma would be significantly associated with behaviors, cognitions, and affect associated with eating disorders (as measured by the three main scales of the EDI): 1) drive for thinness, 2) bulimia, and 3) body dissatisfaction.

H2: The relationship between childhood trauma and eating disorder symptoms would be moderated by:

a) the presence of PTSD symptomology.
b) the presence of maladaptive affective symptoms, most notably anxiety, depression and low self-esteem
c) the presence of insecure (avoidant or anxious) attachment styles as compared to secure attachment styles.
d) the agent of abuse (i.e. whether the perpetrator was within or outside of the family).
e) the age of the child at the time trauma began, specifically, whether the trauma began before or after the age of 14.
f) the chronicity of the trauma (i.e. whether the trauma was a one-time or ongoing event).

H3: The relationship between childhood trauma and eating disorder symptoms would be mediated by:

a) the presence of dissociation.
b) high levels of family dysfunction
c) high levels of body dissatisfaction.

H4: The relationship between the variables proposed to moderate the relationship between trauma and ED symptoms will themselves be moderated by the mediators specified in hypothesis 3. More specifically:

a) PTSD symptoms will interact with dissociation.
b) PTSD symptoms will interact with family dysfunction.

c) PTSD symptoms will interact with body dissatisfaction.

d) Maladaptive affective symptoms will interact with dissociation.

e) Maladaptive affective symptoms will interact with family dysfunction.

f) Maladaptive affective symptoms will interact with body dissatisfaction.

g) Insecure attachment styles will interact with dissociation.

h) Insecure attachment styles will interact with family dysfunction.

i) Insecure attachment styles will interact with body dissatisfaction.

j) The agent of abuse will interact with dissociation.

k) The agent of abuse will interact with family dysfunction.

l) The agent of abuse will interact with body dissatisfaction.

m) The age at onset of trauma will interact with dissociation.

n) The age at onset of trauma will interact with family dysfunction.

o) The age at onset of trauma will interact with body dissatisfaction.

p) The chronicity of trauma will interact with dissociation.

q) The chronicity of trauma will interact with family dysfunction.

r) The chronicity of trauma will interact with body dissatisfaction.

CHAPTER THREE

METHODS

Participants

Participants were at least 18 years of age in order meet IRB requirements for consent. Based on statistical power analysis software, G*Power 3.1.3 (Faul, Erdfelder, Lang, & Buchner, 2009), a minimum sample size of 189 is required when anticipating a medium effect size with 95% confidence level. In comparison to numbers suggested by power analysis formulas by statisticians such as Tabachnick and Fidell (2007), a sample size of 250 far exceeds minimum requirements. However, due to the many predictors being outlined in this study, a sample size between 250 and 500 was sought based on the number of predictors. Both male and female participants were invited to participate, but only data from female participants were included for the purposes of this study. After eliminating males (n= 101), one case because of age (<18), and one case due to refusal to complete the questionnaires, a total of 334 women remained. For this initial sample, the mean age of the women was 26.65 (range= 18-69).

Measures

Demographics Survey

The survey began with a few demographics questions that inquired about age, sex, ethnicity, and educational level. A copy of these questions is presented in Appendix B.

The *Child Trauma Questionnaire-Third Edition (CTQ)*

The CTQ (Bernstein, Fink, Handelsman, & Foote, 1994) was developed with the intent of providing a brief, standardized, reliable, and valid measure of a broad range of

traumatic experiences occurring in childhood. This measure includes 28 self-report items and inquires about five types of childhood trauma: emotional, physical, and sexual abuse, and emotional and physical neglect. Each type of trauma corresponds to a corresponding scale. The emotional abuse scale inquires about verbal assaults on a child's sense of worth or well-being, or any humiliating, demeaning, or threatening behavior directed toward a child by an older person. The physical abuse scale asks about bodily assaults on a child that result in injury or risk of injury. The sexual abuse scale refers to sexual contact or conduct between the child and an older person including explicit coercion. The emotional neglect scale inquires about the failure of caretakers to provide for the child's basic psychological and emotional needs such as love, encouragement, belonging, and support. The physical neglect scale asks about the failure of caregivers to provide for a child's basic physical needs such as food, shelter, safety, supervision, and health. This measure also includes a 3-item minimization/denial scale for the purpose of detecting false-negative trauma reports. Respondents indicate their exposure to abuse and neglect on a 5-point Likert scale, ranging from 1 ("never true") to 5 ("very often true"). Item scores are summed to yield scale scores that quantify the severity of maltreatment in each area. Thresholds for detecting likely cases of abuse are included.

Excellent psychometric properties have been established and other measures have been compared to the CTQ in terms of these properties (Fink, Bernstein, Handelsman, & Foote, 1995; Bernstein, Ahluvalia, Pogge, & Handelsman, 1997). In terms of reliability, four factors were assessed: 1) physical and emotional abuse, 2) emotional neglect, 3) sexual abuse, 4) physical neglect and internal consistency (Cronbach's alpha) rates were

reported as follows: .94, .91, .92, and .79, respectively. For the total scale, internal consistency was reported at .95. Test-retest reliability was reported between .80 and .83 for the factors, with the total scale test-retest reliability reported at .88 over 2 to 6 month intervals (Bernstein et al., 1994). These results were also replicated in a later study (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997).

In addition to the questions posed by the CTQ, several questions were added in order to determine the source of the abuse, the age at the onset of trauma, and the chronicity of the trauma. These questions appeared after being asked about childhood traumas and were presented only to those who endorsed a history of childhood physical, sexual, emotional abuse and/or neglect. A copy of the CTQ and additional questions can be found in the Appendix C.

The *Trauma Symptom Inventory- Third Edition (TSI)*

The TSI (Briere, Elliot, Harris, & Cottman, 1995) was developed for the purpose of assessing a wide range of psychological sequalae often seen in people with histories of abuse. In addition to screening for the existence of high levels of symptoms associated with acute and chronic posttraumatic symptoms, it also screens for a variety of pathologies commonly seen in conjunction. The TSI is a self-report measure that includes 100 items, which respondents answer on a 4-point rating scale ranging from 0 (“never”) to 3 (“often). The TSI has 10 clinical scales and 3 validity scales. The clinical scales include: 1) Anxious Arousal (AA), 2) Depression (D), 3) Anger/Irritability (AI), 4) Intrusive Experiences (IE), 5) Defensive Avoidance (DA), 6) Dissociation (DIS), 7) Sexual Concerns (SC), 8) Dysfunctional Sexual Behavior (DSB), 9) Impaired Self-

Reference (ISR), and 10) Tension Reduction Behavior (TRB). The validity scales include: 1) Response Level, 2) Atypical Response, and 3) Inconsistent Response.

The TSI has demonstrated favorable psychometric properties in many samples (Briere et al., 1995; Smilijanich & Briere, 1993 as cited in Briere et al., 1995). In a university sample (N=279), internal consistency was found to be at .84. The internal consistency of each scale has been reported as follows: AA= .87, D= .90, AI= .89, DA= .88, DIS= .88, SC= .89, DSB= .89, ISR= .87, and TRB= .74 (Briere et al., 1995). Additionally, the TSI predicted PTSD in 91% of cases based upon Astin, Lawrence, and Foy's (1993) joint scoring of the Impact of Events Scale and Los Angeles Symptom Checklist. See Appendix D for a copy of the TSI.

The *Eating Disorder Inventory- Third Edition*

The EDI, originally developed by Garner, Olmstead, and Polivy (1983), was created to assess symptoms and psychological features of eating disorders. The current revision, the EDI-3 (Garner, 2004) is comprised of 91 self-report items. Respondents answer using a 6-point rating scale, ranging from 1= "never" to 6= "always." The EDI-3 is an expansion and improvement over earlier versions, demonstrating better psychometric properties for non-clinical populations (Garner, 2004; Wildes, Ringham, & Marcus, 2010).

The EDI-3 is organized into 12 primary scales including 3 eating disorder specific scales and 9 general pathology scales. The specific scales are: Drive for Thinness (DT), Bulimia (B), and Body Dissatisfaction (BD). The general, but related, scales are: Low Self-Esteem (LSE), Personal Alienation (PA), Interpersonal Insecurity (II), Interpersonal Alienation (IA), Interoceptive Deficits (ID), Emotional Dysregulation (ED),

Perfectionism (P), Asceticism (AS), and Maturity Fear (MF). Six composites are yielded: Eating Disorder Risk, Ineffectiveness, Interpersonal Problems, Affective Problems, Overcontrol, and General Psychological Maladjustment. Clausen, Rosenvinge, Friborg, and Rokkedal (2011) reported good psychometric properties for these scales. Internal consistency for clinical samples and controls were as follows, respectively: DT= .86/.91, B= .92/.87, BD= .90/.93, LSE= .86/.89, PA= .77/.83, II= .80/.83, IA= .75/ .79, ID .89/.85, ED= .77/.78, P= .76/.80, AS= .77/.59, and MF= .86/.78. A copy of the EDI-3 is available in Appendix E.

The *Experiences in Close Relationships Scale- Revised (ECR-R)*

Brennan, Clark, and Shaver (1998) developed the original 36-item self-report measure of adult attachment. It was created by a factor analysis that considered previous research on attachment measures. The measure was revised in 2000 (Fraley, Waller, & Brennan, 2000). Each question requires respondents to answer according to a seven-point Likert Scale (1= "Strongly Disagree" to 7= "Strongly Agree"). This scale includes two dimensions: Attachment Related Avoidance and Attachment Related Anxiety. The Avoidant Dimension has been demonstrated to have an internal consistency of .92-.94 and the Anxiety Dimension an internal consistency of .89-.91 (Fraley et al., 2000; Sibley, Fischer, & Liu, 2005; Holmberg, Lomore, Takacs, & Price, 2011). Appendix F displays a copy of this measure.

Index of Family Relations (IFR)

Hudson (1991) developed the Index of Family Relations (IFR) to assess the severity of family adjustment and relationship problems. The IFR is a 25-item self-report measure of perceived intra-familial stress. Participants respond according to a seven-item

Likert scale ranging from *none of the time* to *all of the time*. The IFR yields scores ranging from 0 to 100, with lower scores indicating lower family discord and higher scores indicating higher levels of perceived intra-familial stress. This measure was normed on 1743 individuals; scores of 30 or higher is identified as a cut-off point indicating significant family problems (Tutty, Babins-Wagner, & Rothery, 2006). Scores above 70 have been linked to severe stress and/or violence in the home. Psychometric properties of the IFR are high. Internal consistency has been reported at .95 (Weisskirch, 2009) and the standard error of measurement has been reported at 3.65. Validity has been indicated at .60 and greater. See Appendix G for a copy of this measure.

Procedure

Data were collected through an anonymous survey which was available online. Participants were recruited via online message boards and email listservs. Fliers were also posted and announcements made at several universities in an urban, mid-western, metropolitan area. Universities included a mixture of private and public institutions and ranged from large to small in population. The Institutional Review Board (IRB) of the University of Detroit Mercy approved this project prior to initiating data collection. See Appendix H for a copy of the IRB approval forms.

Protection of Human Participants

Since this project involved many questions about traumatic childhood experiences, protection of participants was extremely important. It was possible that in completing the survey, participants might have felt distress in response to the personal nature of the questions and possible negative and/or uncomfortable memories of the past. Specifically, for those who reported a history of trauma in their childhoods, completing

these measures could have lead to dysphoria, fear, or other unpleasant outcomes. For this reason, included in each survey was a consent form, which outlined these risks, and provided contact information for the primary investigator and the IRB chairperson in the event that participants had questions or concerns. This consent form thoroughly advised potential participants that the survey they were about to complete included sensitive questions about childhood experiences including exposure to trauma. These risks were reiterated in the instructions for each measure and participants were reminded that they were under no obligation to continue and could withdraw their participation at any time for any reason. In addition, a list of university clinics providing low-cost or free psychotherapy was also provided for those who might haven been interested in seeking professional help. Participants experiencing distress were directed to use these resources and/or contact crisis hotlines. In addition, participants were advised to contact the principal investigator regarding acute distress during or after participation. Past studies using the measures such as the Childhood Trauma Questionnaire (CTQ), which was used in this study, have not reported that the experience has been psychologically damaging for participants. For example, a study by Walker, Newman, Koss, & Bernstein (1997) using the CTQ demonstrated that in a sample of 327 female subjects, fewer than 3% felt strongly that they would not have agreed to participate had they known the survey's contents beforehand. Although the current study held potential risks for participants, the potential contribution to knowledge could change the way that eating disorder etiology is understood and provide valuable information regarding prevention and treatment of eating disorders, which continue to grow in young people and represent the mental illness with the highest rate of premature death (Arcelus, 2007; Berkman, Lohr, & Bulik, 2007;

Keel, Dorer, Eddy, Franko, Charatan, & Herzog, 2003; Steinhausen, 2002). Thus, the potential risk for participants appeared to be warranted compared to the potential for advancement in mental health care. Furthermore, no participants made efforts to contact the principal investigator during data collection.

Data were stored through the online survey software used by the principal investigator. In order to access the data, a unique username and password, known only to the principal investigator, was required. Once the data were downloaded from the online software, it was stored on the principal investigator's computer, which also required a unique username and password for access.

CHAPTER FOUR

RESULTS

Data Cleaning

Prior to completing data analysis, data were thoroughly examined for problematic cases and/or variables and to identify any univariate and multivariate outliers. In the process of data cleaning, it was discovered that item number 40 on the EDI-3 was omitted from the survey. Since none of the participants were presented with this item, a score was imputed for each participant based on the mean score of their responses to all other items belonging to the corresponding subscale, Interoceptive Deficits. Fortunately, according to the EDI-3 manual, Garner (2004) indicates that any subscale can be considered clinically valid so long as it is not missing more than one item. Thus, the subscale and the overall composite scales should be considered valid according to the established psychometrics of the measure.

As stated previously, the original sample consisted of 334 women. From this initial sample, one case was deleted due to system error as this individual failed to respond to two items on the TSI despite the fact that the software was programmed to require a response to all items before allowing participants to continue to the next section. Also, 11 cases were eliminated due to extraordinarily inconsistent responses. Specifically, 3 cases with TSI Inconsistency Scale T-scores at or above 75 and 8 cases with EDI-3 Inconsistency Scale scores of 20 or above, classified as “very atypical,” were eliminated. Thus, the final sample consisted of 322 women.

Descriptive Statistics

Demographic Variables. Table 4-1 displays the frequencies, percentages, ranges, means, and standard deviations for all cases, as applicable, used in the study on the demographic variables of age, ethnicity, and education. For the final sample of 322 women, the mean age was 26.6 (standard deviation= 10.92; range= 18-69 years). The modal age, however, was 18, with 19.9% of the sample being 18 years old and 13.7% of the sample being 19 years old. In terms of ethnicity, 67.7% of the sample identified as Caucasian, 12.4% identified as African-American, and 8.1% identified as multiethnic. In terms of education levels, the largest group was comprised of individuals who had attended some college, but had not received a degree (56.2%). Following that group were individuals with associates degrees, bachelor's degrees, and high school diplomas/equivalents (11.8%, 10.9%, and 9.6%, respectively).

Abuse Variables. Table 4-2 describes the frequencies and percentages of the abuse related variables. For the final sample of 322 women, 146 (45.3%) indicated that they experienced abuse as a child. Of those 146 women, 132 (90.4% of the abused sample; 41% of the total sample) women indicated that they experienced child abuse before the age of 14. In terms of familial abuse, 111 (76% of the abused sample; 34.5% of the total sample) women reported that a family member abused them during childhood. In terms of chronicity, 37 (25.3% of the abused sample; 11.5% of the total sample) women reported that they were abused on only one occasion, while 109 (74.7 percent of the abused sample; 33.9% of the total sample) women reported that they were abused on repeated occasions. Sixty-four participants (43.8% of the abused sample; 19.9% of the

Table 4-1

Frequencies, Percentages, Ranges, Means, and Standard Deviations of Demographic Variables (N=322)

Variable	n	Percent	Range	M	SD
Age	322		18-69	26.6	10.92
Ethnicity					
Asian	12	3.7			
African American	40	12.4			
Caucasian	218	67.7			
Hispanic	15	4.7			
Indian	1	0.3			
Other	10	3.1			
Multiethnic	26	8.1			
Education					
12^{th} grade or less	11	3.4			
High School	31	9.6			
Some college-no degree	181	56.2			
Associate degree	38	11.8			
Bachelors degree	35	10.9			
Post-graduate degree	26	8.1			

Table 4-2

Frequencies and Percentages of Abuse Related Variables (N=322)

Variable	n	Percent of Total Sample	Percent of Abused Sample
Child Abuse	146	45.3	100
Child Abuse Before Age 14	132	41	90.4
Child Abuse After Age 14	98	30.4	67.1
Child Abuse By Family Member	111	34.5	76.0
Child Abuse By Non-Family Member	69	21.4	62.2
Child Abuse on One Occasion	37	11.5	25.3
Child Abuse on Repeated Occasions	109	33.9	74.7
Childhood Physical Abuse	64	19.9	43.8
Childhood Emotional Abuse	112	34.8	76.7
Childhood Sexual Abuse	77	23.9	52.7
Childhood Neglect	36	11.2	24.7

total sample) endorsed physical abuse, 112 (76.7 of the abused sample; 34.8 of the total sample) women endorsed emotional abuse, 77 (52.7 percent of the abused sample; 23.9 percent of the total sample) of the abused women endorsed sexual abuse, and 36 (24.7% if the abused sample; 11.2% of the total sample) of the abused women endorsed neglect.

Psychometric Measures. The Index of Family Relations (IFR) generates one total score, ranging from 0-100, that indicates the extent, severity, and magnitude of problems between family members. The total score is generated using a five-step process. First, 12 of the 25 questions are reversed scored: 1, 2, 4, 5, 8, 14, 15, 17, 18, 20, 21, and 23. Second, the sum of the items is calculated. Third, the number of completed items is subtracted from the sum. Fourth, multiply this number by 100. Fifth, divide this number by the number of items completed times six. Higher scores predict a greater number of family problems.

Furthermore, Hudson (1992) proposed two cutting scores: 30 and 70. A score below 30 is indicative of the absence of clinically significant problems. Scores between 30 and 70 indicate the presence of potentially clinically significant problems. Scores of 70 or above nearly always indicate severe stress and potential for violence. Regarding the total score, the mean score was 30.15 and the standard deviation was 22.28 (Range= 0-97.33; N=322). Since the first significant cut-off for this measure is 30 or greater, the mean score of 30.15 indicates that the average respondent endorsed family dysfunction indicative of potentially clinically significant problems. However, due to the relatively large standard deviation, a great deal of variation appears to exist among respondents.

Thus, results indicate that the sample is representative of a wide continuum of experiences with regard to level of family dysfunction.

The Experiences in Close Relations (ECR) yields two subscale scales related to the two types of insecure attachment: anxious and avoidant. The first 18 items comprise the attachment-related anxiety scale and items 19-36 relate to the attachment related-avoidance scale. Items 9, 11, 20, 22, 26, 27, 28, 29, 30, 31, 33, 34, 35, and 36 are reverse keyed. Then the scores for each scale are summed and averaged. The mean score for attachment related anxiety was 3.42 (SD=1.53; Range=1.0-7.00; N=322); while the mean score for attachment related avoidance was 3.21 (SD=1.43; Range=1.0-7.00; N=322). Although Fraley (2000) does not suggest cut off scores for categorical assignment, higher scores indicate higher levels of insecure attachments for the respective categories.

Tables 4-3 through 4-5 outline the means and standard deviations for the CTQ, EDI, and TSI, categorized by measure. Raw scores are included due to the fact that T-score norms were developed on clinical populations and the population represented in this sample was nonclinical.

Table 4-3 displays the means, standard deviations, and ranges for the Childhood Trauma Questionnaire (CTQ) scales. The CTQ produces three abuse scales (physical, emotional, and sexual), two neglect scales (physical and emotional), and one validity scale (minimization/denial). With the exception of the validity scale, scale scores use raw scores and range from 5 to 25, with higher scores indicating higher levels of maltreatment. The scores were designed to provide a quantitative index of the categorization and severity of maltreatment. Bernstein and Fink (1998) suggest levels of categorization for each type of abuse based on a nonclinical sample. For physical abuse, a

raw score of at least 8 is required. Scores of 8-9 are considered low to moderate, while scores of 10-12 are considered moderate to severe, and scores over 13 are considered extreme. Classification of emotional abuse requires a raw score of at least 9 and a score of 13 or qualifies as moderate to severe. To be classified as sexually abused, individuals must have a raw score of at least 6. Scores of 6-7 are considered low to moderate, while scores of 8-12 are considered moderate to severe, and scores of 13 or above are considered severe to extreme. A raw score of 8 is required for classification of physical neglect. Scores of 8-9 are considered low to moderate and the moderate to severe cut off begins at 10. For emotional neglect, the raw score cut off is 10 and the moderate to severe classification begins at 15. The Minimization/Denial scale indicates the probability of underreporting or denial of maltreatment. Scores on this scale are calculated from responses to three items (10, 16, and 22). Responses of "5-very often true" count as one point and responses of 1-4 count as zero points. Scores range from 0-3, with higher scores indicating greater probability of underreporting. The mean score for this scale was 0.36, which indicates that the overall level of denial/underreporting was within normative parameters. Regarding the five abuse/neglect scales, the highest means were emotional abuse (mean=10.25) and emotional neglect (mean= 10.18). Thus, both emotional abuse and emotional neglect means were higher than the minimum cut-off scores suggested by Bernstein and Fink (1998).

Table 4-3

Descriptive Statistics for CTQ Scales (N=322)

Scale	M	SD	Range
Minimization/Denial	0.36	0.72	0-3
Emotional Abuse	10.25	5.13	5-25
Physical Abuse	7.85	3.95	5-25
Sexual Abuse	8.13	5.93	5-25
Emotional Neglect	10.18	4.66	5-24
Physical Neglect	7.28	2.95	5-18

Table 4-4 presents the means, standard deviations, and ranges for each scale of the Eating Disorder Inventory (EDI-3) for both raw scores and T-scores. EDI-3 scores were calculated first by summing raw scores for each of the twelve scales. Next, raw scores were converted to T-scores by comparing the raw scores to the U.S. Adult Clinical Sample. Both raw scores and T-scores are presented since the current sample is non-clinical and the EDI-3 does not provide a nonclinical sample for T-score calculation. The three eating disorder specific scales (Drive for Thinness, Bulimia, and Body Dissatisfaction) are based on the T-scores from Eating Disorder Not Otherwise Specified (EDNOS) clinical norms. EDNOS clinical norms were chosen since EDNOS is the most prevalent clinical diagnosis, and also the most likely to be pervasive in nonclinical samples since it does not require strict adherence to the current DSM-IV-TR disorders of Anorexia and Bulimia. Since the Eating Disorders Risk Composite (EDRC) is calculated by summing the T-scores of the three eating disorder specific scales, the composite T-score is also based on EDNOS norms. For the remaining scales and composites, T-scores are based on the general normative clinical sample. Composite scores are generated from

Table 4-4

Descriptive Statistics for EDI-3 Scales- Raw Scores and T-Scores (N=322)

Scale	Raw Scores			T-Scores		
	M	SD	Range	M	SD	Range
Drive for Thinness*	10.73	8.25	0-28	34.84	11.96	19-60
Bulimia*	6.59	7.31	0-32	45.07	9.15	37-77
Body Dissatisfaction*	19.09	11.42	0-40	40.09	11.42	21-61
Low Self Esteem	6.75	6.00	0-24	39.50	10.07	28-68
Personal Alienation	8.18	6.40	0-28	41.33	10.18	28-73
Interpersonal Insecurity	8.76	6.08	0-28	45.41	9.90	31-77
Interpersonal Alienation	8.08	5.75	0-26	45.90	10.62	31-79
Interoceptive Deficits	9.06	8.30	0-33	40.30	10.27	29-70
Emotional Dysregulation	5.71	5.82	0-29	45.69	10.33	36-87
Perfectionism	11.24	5.74	0-24	45.67	9.75	27-67
Asceticism	6.61	5.06	0-27	39.22	8.86	28-75
Maturity Fears	9.57	6.41	0-32	48.16	9.30	34-81
Eating Disorder Risk Composite*	120.01	28.69	77-198	39.29	10.22	26-71
Ineffectiveness Composite	80.82	19.28	56-141	44.53	10.83	28-79
Interpersonal Problems Composite	91.30	19.24	62-152	41.40	10.84	29-79
Affective Problems Composite	85.99	18.96	65-151	40.62	9.26	23-73
Overcontrol Composite	84.89	15.63	55-140	40.08	10.36	24-78
General Psychological Maladjustment Composite	391.17	66.24	287-632	36.34	12.48	18-70
Infrequency Scale	0.48	1.05	0-7			
Negative Impression	9.25	10.96	0-68			
Inconsistency	8.36	4.08	0-19			

*=T-Scores are based on EDNOS norms (all others based on general normative sample T-Scores)

subscales. Specifically, the Ineffectiveness Composite (IC) is comprised of Low Self Esteem and Personal Alienation, the Interpersonal Problems Composite (IPC) is comprised of Interpersonal Insecurity and Interpersonal Alienation, the Affective Problems Composite (APC) is comprised of Interoceptive Deficits and Emotional Dysregulation, the Overcontrol Composite (OC) is comprised of Perfectionism and Asceticism, and the General Psychological Maladjustment Composite (GPMC) is comprised of Low Self Esteem, Personal Alienation, Interpersonal Insecurity, Interpersonal Alienation, Interoceptive Deficits, Emotional Dysregulation, Perfectionism, Asceticism, and Maturity Fears. Proposed cut-offs for T-scores are as follows: Low Clinical= below the 25th percentile, Typical Clinical= 25th percentile to 66th percentile, Elevated Clinical= 67th to 99th percentile. Respective corresponding T-scores for the 25th and 67th percentiles are as follows: EDRC: 45 and 55; IC: 43 and 55; IPC: 42 and 55; APC: 43 and 54; OC: 43 and 55; GPMC: 43 and 55.

Regarding the EDI-3 validity scales, higher scores on the Infrequency Scale are indicative of a tendency to endorse items in a way that differs from how most people would respond. High scores may indicate random responding, attention difficulties, or comprehension problems. For the Infrequency Scale, scores of 0-2 are considered Typical, scores of 3-4 are considered Atypical, and scores of 5-10 are considered Very Atypical. Data indicate that mean score on the Infrequency scale (.48) was in the typical range.

The Negative Impression Scale is calculated by counting the number of items for which a score of 4 was endorsed. It is a measure of respondents' tendencies to endorse items that demonstrate overly negative symptom patterns. Participants with particularly

severe symptoms may show elevations on this scale, but high scores may also indicate a tendency to exaggerate or overemphasize negative symptoms. According to EDI-3 norms, scores of 0-44 = Typical, scores of 45-54 = Atypical, and scores of 55-90 = Very Atypical. Data indicate that the mean score meets criteria for classification as Typical.

The Inconsistency Scale is a measure of the tendency to endorse items in a contradictory manner. Participants who have higher scores on this scale may have difficulty with attention, comprehension, or be randomly endorsing items. To calculate the score for this scale, the following items are compared: 2 and 12, 9 and 55, 10 and 50, 17 and 65, 21 and 26, 30 and 54, 31 and 59, 34 and 57, 37 and 41, and 45 and 62. The lesser number is subtracted from the greater number for each paring and then summing these differences generates the inconsistency score. Classification of scores are as follows: Typical=0-15, Atypical=16-19, and 20-40= Very Atypical. Data indicate that the mean Inconsistency Score meets criteria for classification as Typical.

Table 4-5 presents the means, standard deviations, and ranges for the Trauma Symptom Inventory (TSI) scales. Both raw scores and T-scores are listed for the ten main indices and three validity scales. Raw scores are calculated using simple sums of items and can be converted to T-scores using comparisons to a clinical population based on age and gender norms. The TSI provides separate standards for men and women and also for women aged 18-55 and women aged 55 and over. Higher scores indicate higher levels of difficulty in each category and T-scores of 65 or higher are considered the clinical cut off for scales. In terms of its validity scales, the Atypical Response Scale is a similar measure

Table 4-5

Descriptive Statistics for TSI Raw Scores and T-Scores (N=322)

Scale	Raw Scores			T-Scores		
	M	SD	Range	M	SD	Range
Anxious Arousal	10.23	5.97	0-24	54.94	11.46	35-81
Depression	9.76	6.82	0-24	54.37	11.19	38-78
Anger/Irritability	11.90	7.31	0-27	55.81	11.76	37-80
Intrusive Experiences	8.80	6.90	0-24	55.04	12.41	39-82
Defensive Avoidance	10.54	6.71	0-24	55.57	10.92	38-77
Dissociation	9.18	6.90	0-27	57.37	13.79	39-93
Sexual Concerns	6.47	6.56	0-26	54.48	12.53	42-92
Dysfunctional Sexual Behavior	5.16	6.40	0-27	57.08	15.94	44-100
Impaired Self-Reference	9.97	7.10	0-27	55.87	11.93	39-85
Tension Reducing Behavior	5.31	5.32	0-21	57.88	15.75	42-100
Atypical Response	3.43	4.81	0-26	55.66	14.50	45-100
Response Level	1.11	1.61	0-10	46.39	7.66	41-89
Inconsistent Response	3.83	2.16	0-10	49.96	8.24	35-73

to the Infrequency scale of the EDI-3 as scores on this scale rise as participants endorse items in ways that most people would not. Thus, as participants endorse more severe symptoms or experiences that most people do not have, their scores become elevated. The Response Level Scale serves as a screener for the common tendency to mark zeros indiscriminately on symptom checklists. Thus, it identifies the ten items least likely to be marked zero in a normative sample and demonstrates the extent to which the respondent denies symptoms. The Inconsistent Response scale identifies respondents who respond to the checklist in a way that is contradictory and inconsistent. Random respondents or participants who have reading or comprehension difficulties, poor concentration, or dissociative tendencies are likely to obtain higher scores on this scale.

Inter-correlations Between Scales and Subscales

Next, Pearson product-moment correlations were conducted to examine the relationship between the specific types of traumas to each other. All types of trauma: emotional abuse, physical abuse, sexual abuse, emotional abuse, and physical neglect correlated significantly with each other type of trauma ($p<.001$) and ranged from $r=.35$ between emotional neglect and sexual abuse to $r=.69$ between emotional neglect and emotional abuse. Also, a statistically significant, inverse relationship was noted between each type of trauma and the tendency to minimize and/or deny symptoms. Results are displayed in Table 4-6.

Table 4-6

Pearson Product-Moment Inter-Correlations Between CTQ Scales

	Minimization /Denial	Emotional Abuse	Physical Abuse	Sexual Abuse	Emotional Neglect
Emotional Abuse	-.31***				
Physical Abuse	-.19**	.60***			
Sexual Abuse	-.16*	.48***	.45***		
Emotional Neglect	-.39***	.69***	.47***	.35***	
Physical Neglect	-.27***	.62***	.46***	.34***	.67***

Note: *p<.05, **p<.01, ***p<.001

Pearson product-moment correlations were also used to compare each scale of the EDI-3 to all other scales of the EDI. Statistically significant correlations were discovered between all EDI-3 scales; most were $p<.001$. Perfectionism and Maturity Fears generally yielded correlations of small effect sizes with other EDI-3 scales. The remaining correlations generally yielded medium effect sizes. Results are described in Table 4-7.

Table 4-7

Pearson Product-Moment Inter-Correlations Between EDI-3 Scales

	DT	B	BD	LSE	PA	II	IA	ID	ED	P	A
B	.66***										
BD	.75***	.56***									
LSE	.62***	.65***	.58***								
PA	.55***	.60***	.51***	.82***							
II	.42***	.47***	.33***	.59***	.69***						
IA	.45***	.51***	.39***	.67***	.80***	.76***					
ID	.52***	.60***	.40***	.62***	.71***	.60***	.65***				
ED	.42***	.55***	.32***	.58***	.66***	.47***	.61***	.69***			
P	.29***	.26***	.15**	.21***	.27***	.23***	.29***	.29***	.20***		
A	.59***	.67***	.43***	.56***	.57***	.45***	.56***	.62***	.59***	.41***	
MF	.22***	.25***	.15***	.38***	.39***	.31***	.33***	.33***	.26***	.13*	.27***

Note: *p<.05, **p<.01, ***p<.001; DT= Drive for Thinness, BD= Body Dissatisfaction, LSE= Low Self Esteem, PA= Personal Alienation, II=Interpersonal Insecurity, IA= Interpersonal Alienation, ID= Interoceptive Deficits, ED= Emotional Dysregulation, P= Perfectionism, A= Asceticism, MF= Maturity Fears

Further, Pearson product-moment correlations were used to investigate the relationship between each of the scales on the EDI-3 and each of the EDI-3 composites. As expected, statistically significant correlations were found between each scale and each composite. All of these correlations were significant at the .001 level and most of them could be categorized as having large effect sizes. Details are outlined in Table 4-8.

To study the EDI further, Pearson product-moment correlations were conducted between each EDI-3 composite and all other EDI-3 composites. Statistically significant relationships were established between each of the five composites of the EDI-3. All of these correlations were significant ($p<.001$) and all could be classified as having large effect sizes, with the exception of one correlation (Affective Problems with Overcontrol), although the correlation ($r=.48$) approached a large effect size. Details are reported in Table 4-9.

Table 4-8

Pearson Product-Moment Correlations Between EDI-3 Scales (Raw scores) and EDI-3 Composites (Raw scores)

	EDI Composite				
	EDRC	IC	IPC	APC	OC
Subscale					
DT	.93***	.61***	.46***	.51***	.51***
B	.82***	.65***	.52***	.63***	.54***
BD	.89**	.57***	.39***	.39***	.65***
LSE	.70**	.95***	.67***	.65***	.45***
PA	.62***	.95***	.79***	.74***	.50***
II	.46***	.67***	.93***	.59***	.40***
IA	.50***	.77***	.94***	.68***	.50***
ID	.56***	.70***	.67***	.92***	.53***
ED	.48***	.65***	.58***	.92***	.46***
P	.26***	.25***	.28***	.27***	.86***
A	.63***	.60***	.54***	.65***	.82***
MF	.23***	.40***	.34***	.32***	.24***

Note: *p<.05, **p<.01, ***p<.001; DT= Drive for Thinness, BD= Body Dissatisfaction, LSE= Low Self Esteem, PA= Personal Alienation, II=Interpersonal Insecurity, IA= Interpersonal Alienation, ID= Interoceptive Deficits, ED= Emotional Dysregulation, P= Perfectionism, A= Asceticism, MF= Maturity Fears; EDRC= Eating Disorder Risk Composite, IC= Ineffectiveness Composite, IPC= Interpersonal Problems Composite, APC= Affective Problems, OC= Overcontrol Composite

Table 4-9

Pearson Product-Moment Correlations Between EDI-3 Composites (Raw Scores)

	EDRC	IC	IPC	APC	OC
Ineffectiveness Composite (IC)	.69***				
Interpersonal Problems Composite (IPC)	.51***	.77***			
Affective Problems Composite (APC)	.57***	.73***	.68***		
Overcontrol Composite (OC)	.52***	.50***	.48***	.54***	
General Psychological Maladjustment Composite (GPM)	.67***	.90***	.87***	.87***	.71***

Note: *p<.05, **p<.01, ***p<.001

The next step was to compare the relationship of each TSI scale with each other TSI scale with Pearson product-moment correlations. Again, this analysis resulted in

statistically significant correlations (p<.001) between each scale and all correlations had large effect sizes. All coefficients are shown in Table 4-10.

Table 4-10

Pearson Product-Moment Correlations Between TSI Scales

	AA	Dep	A/I	IE	DA	Dis	SC	DSB	ISR
Dep	.80***								
A/I	.81***	.73***							
IE	.73***	.70***	.68***						
DA	.70***	.70***	.63***	.85***					
Dis	.81***	.78***	.74***	.77***	.73***				
SC	.62***	.64***	.62***	.66***	.69***	.69***			
DSB	.52***	.56***	.55***	.62***	.60***	.60***	.77***		
ISR	.77***	.82***	.76***	.72***	.72***	.83***	.71***	.64***	
TRB	.71***	.72***	.77***	.74***	.68***	.75***	.76***	.85***	.78***

Note: *p<.05, **p<.01, ***p<.001; AA= Anxious Arousal, Dep= Depression, A/I= Anger/Irritability, IE= Intrusive Experiences, DA= Defensive Avoidance, Dis=Dissociation, SC= Sexual Concerns, DSB= Dysfunctional Sexual Behavior, ISR= Impaired Self-Reference, TRB= Tension Reducing Behavior

ECR scales were also analyzed using Pearson product-moment correlations to investigate the correlation between Attachment Related Anxiety and Attachment Related Avoidance. A statistically significant relationship with a medium effect size was found between these two variables as well (r=.54, p<.001).

The CTQ scales were also compared to IFR and ECR scales. Statistically significant relationships were found between each CTQ scale and IFR scores. A statistically significant, inverse, medium effect size relationship was found between the IFR and the tendency to engage in minimization and denial. Correlations yielded medium-large effect sizes. Also, significance was determined between CTQ scales and both the Anxious Attachment (small-medium effect sizes) and Avoidance Attachment (small-medium effect sizes) scales of the ECR. Full details are noted in Table 4-11.

Table 4-11

Pearson Product-Moment Correlations Between CTQ Scales and Scales of the IFR and ECR

	CTQ Scale					
	Minimization /Denial	Emotional Abuse	Physical Abuse	Sexual Abuse	Emotional Neglect	Physical Neglect
IFR- Total Score	-.39***	.69***	.40***	.37***	.66***	.53***
ECR- Anxious Attachment	-.29***	.45***	.24***	.27***	.38***	.28***
ECR- Avoidance Attachment	.26***	.40***	.26***	.28***	.41***	.30***

Note: *p<.05, **p<.01, ***p<.001

CTQ scales were also compared to TSI scales. Statistical significance was established between each scale of the TSI and each scale of the CTQ. Significant, inverse relationships and small effect sizes were noted for all correlations between the CTQ Minimization/Denial Scale and TSI scales. The Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect scales of the CTQ were also significantly related (p<.001) to all TSI scales. Full results are described in Table 4-12.

Table 4-13 outlines the relationships between each TSI scale with IFR Total Score, ECR Attachment Related Anxiety, and ECR Attachment Related Avoidance. As shown below, each TSI scale was significantly correlated (p<.001) with IFR Total Scores, ECR Attachment Related Anxiety and ECR Attachment Related Avoidance. Effect sizes were generally medium-large.

Table 4-12

Pearson Product-Moment Correlations Between CTQ Scales and TSI Scales (Raw Scores)

	CTQ-Minimization/Denial	CTQ-Emotional Abuse	CTQ-Physical Abuse	CTQ-Sexual Abuse	CTQ-Emotional Neglect	CTQ-Physical Neglect
TSI- AA	-.21***	.45***	.27***	.38***	.31***	.31***
TSI- Dep	-.22***	.46***	.31***	.35***	.38***	.33***
TSI- A/I	-.23***	.41***	.25***	.31***	.28***	.30***
TSI- IE	-.15**	.45***	.33***	.41***	.31***	.37***
TSI- DA	-.18**	.47***	.32***	.37***	.34***	.38***
TSI- Dis	-.18**	.49***	.32***	.40***	.40***	.40***
TSI- SC	-.23***	.39***	.31***	.45***	.33***	.32***
TSI- DSB	-.20***	.30***	.29***	.39***	.25***	.29***
TSI- ISR	-.24***	.44***	.27***	.31***	.36***	.32***
TSI- TRB	-.21***	.40***	.32***	.41***	.31***	.33***

Note: *p<.05, **p<.01, ***p<.001; AA= Anxious Arousal, Dep= Depression, A/I= Anger/Irritability, IE= Intrusive Experiences, DA= Defensive Avoidance, Dis=Dissociation, SC= Sexual Concerns, DSB= Dysfunctional Sexual Behavior, ISR= Impaired Self-Reference, TRB= Tension Reducing Behavior

The next table indicates the relationship between the scales and composites of the EDI with the TSI scales. In Table 4-14, results indicate a statistically significant relationship between scores on each EDI scale and composite and each scale of the TSI. Correlations for TSI scales of with EDI scales and composites generally had medium-large effect sizes and were significant at the p<.001 level.

Next, EDI scales and composites were compared with the IFR Total Score, the ECR Attachment Related Anxiety Scale, and the ECR Attachment Related Avoidance Scale. As shown in Table 4-15, all EDI scales and composites were significantly related to IFR total scores and ECR scores and generally yielded medium-large effect sizes. The

Table 4-13

Pearson Product-Moment Correlations Between TSI Scales, IFR Scales, and ECR Scales

	IFR-Total Score	ECR-Attachment Related Anxiety	ECR-Attachment Related Avoidance
TSI- AA	.48***	.57***	.38***
TSI- Dep	.53***	.67***	.43***
TSI- A/I	.45***	.56***	.38***
TSI- IE	.47***	.53***	.38***
TSI- DA	.49***	.56***	.42***
TSI- Dis	.51***	.57***	.41***
TSI- SC	.47***	.54***	.45***
TSI- DSB	.40***	.49***	.33***
TSI- ISR	.50***	.69***	.46***
TSI- TRB	.44***	.59***	.36***

Note: *p<.05, **p<.01, ***p<.001; AA= Anxious Arousal, Dep= Depression, A/I= Anger/Irritability, IE= Intrusive Experiences, DA= Defensive Avoidance, Dis=Dissociation, SC= Sexual Concerns, DSB= Dysfunctional Sexual Behavior, ISR= Impaired Self-Reference, TRB= Tension Reducing Behavior

exception to this was the non-significant relationship between IFR Total Score and the Maturity Fears scale of the EDI (r=.06). A final set of Pearson product-moment correlations was calculated to examine the relationship between Total Score on the IFR and the two scales of the ECR, Attachment Related Anxiety and Attachment Related Avoidance. IFR scores were significantly related to both types of attachment difficulties: Anxiety r=.47 (p<.001) and Avoidance r= .44 (p<.001). Both of these correlations can be classified as having medium-large effect sizes.

Since the correlations between the CTQ scales and the EDI-3 scales were central to Hypothesis 1, they are included in the next section, which discusses the statistical analyses central to that hypothesis.

Table 4-14

Pearson Product-Moment Correlations Between EDI Scales and Composites (Raw Scores) and TSI Scales (Raw Scores)

	TSI- Anxious Arousal	TSI-Depression	TSI-Anger/Irritability	TSI-Intrusive Experiences	TSI- Defensive Avoidance	TSI- Dissociation	TSI- Sexual Concerns	TSI- Dysfunctional Sexual Behaviors	TSI- Impaired Self-Reference	TSI-Tension Reduction Behavior
EDI- DT	.47***	.48***	.41***	.35***	.38***	.44***	.36***	.27***	.47***	.40***
EDI-B	.52***	.54***	.45***	.44***	.41***	.52***	.41***	.31***	.53***	.47***
EDI-BD	.38***	.41***	.35***	.27***	.29***	.37***	.34***	.21***	.39***	.31***
EDI-LSE	.66***	.74***	.58***	.50***	.51***	.63***	.53***	.41***	.69***	.57***
EDI-PA	.69***	.80***	.63***	.57***	.59***	.73***	.60***	.50***	.75***	.62***
EDI- II	.51***	.55***	.46***	.38***	.44***	.52***	.41***	.26***	.57***	.38***
EDI-IA	.62***	.66***	.60***	.56***	.58***	.65***	.64***	.50***	.66***	.56***
EDI- ID	.64***	.67***	.59***	.60***	.62***	.71***	.51***	.44***	.71***	.61***
EDI- ED	.65***	.66***	.70***	.60***	.54***	.64***	.55***	.53***	.62***	.68***
EDI-P	.30***	.22***	.26***	.24***	.26***	.25***	.18**	.13*	.22***	.21***
EDI- A	.53***	.49***	.48***	.46***	.45***	.52***	.47***	.35***	.52***	.49***
EDI- MF	.28***	.33***	.28***	.21***	.25***	.30***	.23***	.19***	.37***	.27***
EDI- EDRC	.51***	.53***	.46***	.39***	.40***	.50***	.42***	.29***	.52***	.44***
EDI-IC	.71***	.81***	.64***	.57***	.58***	.71***	.59***	.48***	.76***	.62***
EDI-IPC	.60***	.65***	.57***	.50***	.54***	.62***	.56***	.41***	.65***	.51***
EDI- APC	.70***	.72***	.70***	.65***	.63***	.73***	.58***	.53***	.72***	.70***
EDI- OC	.49***	.41***	.44***	.41***	.42***	.45***	.38***	.28***	.43***	.41***
EDI- GPM	.74***	.78***	.69***	.62***	.64***	.75***	.62***	.50***	.77***	.66***

Note: *p<.05, **p<.01, ***p<.001; DT= Drive for Thinness, BD= Body Dissatisfaction, LSE= Low Self Esteem, PA= Personal Alienation, II=Interpersonal Insecurity, IA= Interpersonal Alienation, ID= Interoceptive Deficits, ED= Emotional Dysregulation, P= Perfectionism, A= Asceticism, MF= Maturity Fears; EDRC= Eating Disorder Risk Composite, IC= Ineffectiveness Composite, IPC= Interpersonal Problems Composite, APC= Affective Problems, OC= Overcontrol Composite, GPM= General Psychological Maladjustment Composite

Table 4-15

Pearson Product-Moment Correlations Between EDI Scales and Composites (Raw Scores), IFR Scales, and ECR Scales

	IFR-Total Score	ECR-Attachment Related Anxiety	ECR-Attachment Related Avoidance
EDI- Drive for Thinness	.28***	.48***	.31***
EDI-Bulimia	.35***	.50***	.32***
EDI-Body Dissatisfaction	.31***	.43***	.29***
EDI-Low Self Esteem	.47***	.67***	.41***
EDI-Personal Alienation	.56***	.68***	.54***
EDI- Interpersonal Insecurity	.46***	.53***	.61***
EDI-Interpersonal Alienation	.58***	.64***	.71***
EDI- Interoceptive Deficits	.34***	.63***	.49***
EDI- Emotional Dysregulation	.42***	.53***	.35***
EDI-Perfectionism	.24***	.28***	.26***
EDI- Asceticism	.33***	.52***	.36***
EDI- Maturity Fears	.06	.29***	.18**
EDI- Eating Disorder Risk Composite	.36***	.53***	.35***
EDI-Ineffectiveness Composite	.54***	.71***	.50***
EDI-Interpersonal Problems Composite	.55***	.63***	.71***
EDI- Affective Problems Composite	.42***	.63***	.46***
EDI- Overcontrol Composite	.34***	.47***	.37***
EDI- General Psychological Maladjustment	.53***	.72***	.59***

Note: *p<.05, **p<.01, ***p<.001

Analysis of Hypothesis 1

Bivariate Correlations

Pearson product-moment correlations were calculated to investigate the first hypothesis that self-reported childhood trauma would be significantly associated with the three main eating disorder scales of the EDI-3. Endorsement of childhood trauma was found to be correlated significantly with the EDI-3 scales: Drive for Thinness: r=.22 (p<.001); Bulimia: r=.32 (p<.001); and Body Dissatisfaction: r=.28 (p<.001). Based on Cohen's (1988) guidelines for determining effect sizes among correlations in areas without established standards (.10=small, .30=medium, .50=large), the greatest effect size exists for the relationship between bulimia and child abuse (medium), followed by body dissatisfaction and drive for thinness, respectively (small-medium).

CTQ scale scores were then compared to the EDI scale raw scores using Pearson product-moment correlations. Full descriptions of these relationships are outlined in Table 4-16. All EDI scales and composites that demonstrated significant correlations with the Minimization/Denial scale of the CTQ [Drive for Thinness (r=-.14, p<.05, small effect size), Body Dissatisfaction (r=.-15, p<.01, small effect size), Low Self Esteem (r=-.18, p<.01, small effect size), Personal Alienation (r=-.24, p<.001, small effect size), Interpersonal Insecurity (r=-.18, p<.01, small effect size), Interpersonal Alienation (r=.26, p<.001, small-medium effect size), Interoceptive Deficits (r=-.16, p<.01, small effect size), Emotional Dysregulation (r=-.15, p<.01, small effect size), Asceticism (r=-.15, p<.01, small effect size), Eating Disorder Risk Composite (r=-.16, p<.01, small effect size), Ineffectiveness Composite (r=-.22, p<.001, small effect size), Interpersonal

Table 4-16

Pearson Product-Moment Correlations Between CTQ Scales and EDI Scales and Composites (Raw Scores)

	CTQ-Minimization/ Denial	CTQ-Emotional Abuse	CTQ- Physical Abuse	CTQ- Sexual Abuse	CTQ-Emotional Neglect	CTQ-Physical Neglect
DT	-.14*	.33***	.18**	.28***	.25***	.17**
B	.12*	.41***	.24***	.34***	.28***	.29***
BD	-.15**	.34***	.20***	.26***	.28***	.19**
LSE	-.18**	.44***	.22***	.32***	.34***	.29***
PA	-.24***	.50***	.31***	.37***	.42***	.36***
II	-.18**	.40***	.25***	.31***	.40***	.30***
IA	-.26***	.50***	.32***	.39***	.46***	.38***
ID	-.16**	.37***	.26***	.28***	.32***	.32***
ED	-.15**	.39***	.33***	.38***	.32***	.29***
P	-.05	.25***	.16**	.15**	.12*	.12*
A	-.15**	.38***	.26***	.33***	.28***	.29***
MF	.02	.03	-.02	-.02	-.04	-.07
EDRC	-.16**	.40***	.23***	.33***	.31***	.24***
IC	-.22***	.49***	.28***	.36***	.39***	.34***
IPC	-.23***	.48***	.31***	.38***	.46***	.36***
APC	-.17**	.41***	.32***	.36***	.35***	.33***
OC	-.12*	.37***	.25***	.28***	.23***	.24***
GPM	-.20***	.49***	.32***	.38***	.40***	.35***

Note: *p<.05, **p<.01, ***p<.001; DT= Drive for Thinness, B=Bulimia, BD= Body Dissatisfaction, LSE= Low Self Esteem, PA= Personal Alienation, II= Interpersonal Insecurity, IA= Interpersonal Alienation, ID= Interoceptive Deficits, ED= Emotional Dysregulation, P= Perfectionism, A= Asceticism, MF= Maturity Fears, EDRC= Eating Disorder Risk Composite, IC= Ineffectiveness Composite, IPC= Interpersonal Problems Composite, APC= Affective Problems Composite, OC= Overcontrol Composite, GPM= General Psychological Maladjustment Composite

Problems Composite (r=-.23, p<.001, small effect size), Affective Problems Composite (r=-.17, p<.01, small effect size), Overcontrol Composite (r=-.12, p<.05, small effect size), General Psychological Maladjustment Composite (r=-.20, p<.001, small effect size)], demonstrated inverse relationships with the exception of Bulimia (r=.12, p<.05, small effect size). Perfectionism and Maturity Fears were not significantly correlated with the Minimization/Denial scale of the CTQ. Furthermore, the Maturity Fears scale was not significantly correlated with any of the CTQ abuse scales. All other EDI scales were significantly correlated with each CTQ abuse scale.

For the Emotional Abuse scale, correlations were as follows with EDI scales and composites: Drive for Thinness (r=.33, p<.001, medium effect size), Bulimia (r=.41, p<.001, small effect size), Body Dissatisfaction (r=.34, p<.001, medium effect size), Low Self Esteem (r=.44, p<.001, medium effect size), Personal Alienation (r=.50, p<.001, large effect size), Interpersonal Insecurity (r=.40, p<.001, medium effect size), Interpersonal Alienation (r=.50, p<.001, large effect size), Interoceptive Deficits (r=.37, p<.001, medium effect size), Emotional Dysregulation (r=.39, p<.001, medium effect size), Perfectionism (r=.25, p<.001, small-medium effect size), Asceticism (r=.38, p<.001, medium effect size), Eating Disorder Risk Composite (r=.40, p<.001, medium effect size), Ineffectiveness Composite (r=.49, p<.001, medium-large effect size), Interpersonal Problems Composite (r=.48, p<.001, medium-large effect size), Affective Problems Composite (r=.41, p<.001, medium effect size), Overcontrol Composite (r=.37, p<.001, medium effect size), General Psychological Maladjustment Composite (r=.49, p<.001, medium-large effect size).

For the Physical Abuse scale, correlations were as follows with EDI scales and composites: Drive for Thinness (r=.18, p<.01, small effect size), Bulimia (r=.24, p<.001, small effect size), Body Dissatisfaction (r=.20, p<.001, small effect size), Low Self Esteem (r=.22, p<.001, small effect size), Personal Alienation (r=.31, p<.001, medium effect size), Interpersonal Insecurity (r=.25, p<.001, small-medium effect size), Interpersonal Alienation (r=.32, p<.001, medium effect size), Interoceptive Deficits (r=.26, p<.001, small-medium effect size), Emotional Dysregulation (r=.33, p<.001, medium effect size), Perfectionism (r=.16, p<.01, small effect size), Asceticism (r=.26, p<.001, small-medium effect size), Eating Disorder Risk Composite (r=.23, p<.001, small effect size), Ineffectiveness Composite (r=.28, p<.001, small-medium effect size), Interpersonal Problems Composite (r=.31, p<.001, medium effect size), Affective Problems Composite (r=.32, p<.001, medium effect size), Overcontrol Composite (r=.25, p<.001, small-medium effect size), General Psychological Maladjustment Composite (r=.32, p<.001, medium effect size).

For the Sexual Abuse scale, correlations were as follows with EDI scales and composites: Drive for Thinness (r=.28, p<.01, small-medium effect size), Bulimia (r=.34, p<.001, medium effect size), Body Dissatisfaction (r=.26, p<.001, small-medium effect size), Low Self Esteem (r=.32, p<.001, medium effect size), Personal Alienation (r=.37, p<.001, medium effect size), Interpersonal Insecurity (r=.31, p<.001, medium effect size), Interpersonal Alienation (r=.39, p<.001, medium effect size), Interoceptive Deficits (r=.28, p<.001, small-medium effect size), Emotional Dysregulation (r=.38, p<.001, medium effect size), Perfectionism (r=.15, p<.01, small effect size), Asceticism (r=.33, p<.001, medium effect size), Eating Disorder Risk Composite (r=.33, p<.001, medium

effect size), Ineffectiveness Composite (r=.36, p<.001, medium effect size), Interpersonal Problems Composite (r=.36, p<.001, medium effect size), Affective Problems Composite (r=.38, p<.001, medium effect size), Overcontrol Composite (r=.28, p<.001, small-medium effect size), General Psychological Maladjustment Composite (r=.38, p<.001, medium effect size).

For the Emotional Neglect scale, correlations were as follows with EDI scales and composites: Drive for Thinness (r=.25, p<.001, small-medium effect size), Bulimia (r=.28, p<.001, small-medium effect size), Body Dissatisfaction (r=.28, p<.001, small-medium effect size), Low Self Esteem (r=.34, p<.001, medium effect size), Personal Alienation (r=.42, p<.001, medium effect size), Interpersonal Insecurity (r=.40, p<.001, medium effect size), Interpersonal Alienation (r=.46, p<.001, medium-large effect size), Interoceptive Deficits (r=.32, p<.001, medium effect size), Emotional Dysregulation (r=.32, p<.001, medium effect size), Perfectionism (r=.12, p<.05, small effect size), Asceticism (r=.28, p<.001, small-medium effect size), Eating Disorder Risk Composite (r=.31, p<.001, medium effect size), Ineffectiveness Composite (r=.39, p<.001, medium effect size), Interpersonal Problems Composite (r=.46, p<.001, medium-large effect size), Affective Problems Composite (r=.35, p<.001, medium effect size), Overcontrol Composite (r=.23, p<.001, small effect size), General Psychological Maladjustment Composite (r=.40, p<.001, medium effect size).

For the Physical Neglect scale, correlations were as follows with EDI scales and composites: Drive for Thinness (r=.17, p<.01, small effect size), Bulimia (r=.29, p<.001, small-medium effect size), Body Dissatisfaction (r=.19, p<.01, small effect size), Low Self Esteem (r=.29, p<.001, small-medium effect size), Personal Alienation (r=.36,

p<.001, medium effect size), Interpersonal Insecurity (r=.30, p<.001, medium effect size), Interpersonal Alienation (r=.38, p<.001, medium effect size), Interoceptive Deficits (r=.32, p<.001, medium effect size), Emotional Dysregulation (r=.29, p<.001, medium effect size), Perfectionism (r=.12, p<.05, small effect size), Asceticism (r=.29, p<.001, small-medium effect size), Eating Disorder Risk Composite (r=.24, p<.001, small effect size), Ineffectiveness Composite (r=.34, p<.001, medium effect size), Interpersonal Problems Composite (r=.36, p<.001, medium effect size), Affective Problems Composite (r=.33, p<.001, medium effect size), Overcontrol Composite (r=.24, p<.001, small effect size), General Psychological Maladjustment Composite (r=.35, p<.001, medium effect size).

Multiple Regressions

Standard multiple regressions were performed using the EDI scales as criterion variables and the five scales of the CTQ as predictors. All analyses were done using SPSS standard forward regression. For the EDI scales, raw scores were used.

In the first analysis, Drive for Thinness scale was the dependent variable Table 4-17 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the five CTQ scales collectively predict Drive for Thinness (Multiple R=.37, R^2=.14, R^2_{adj} =.12, F(5, 316)=9.95, p<.001). According to Cohen's (1988) guidelines for categorizing effect sizes for standardized regression weights in areas of psychological research without established baselines, values of .14 should be considered small, values of about .39 should be considered medium, and values of .59 or greater should be considered large.

Based on these recommendations, this effect size is categorized as small. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, also emerged as significant (see Table 4-17). Upon examination of squared semi-partial correlations, 28.6% of the variance within R^2 is uniquely attributable to emotional abuse and 14.3% of the variance is uniquely attributable to sexual abuse.

Table 4-17

Standard Multiple Regression between the EDI Drive for Thinness Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.47	.14	.29	3.50**	.04
Physical Abuse	-.14	.14	-.07	-.96	
Sexual Abuse	.23	.09	.17	2.73**	.02
Emotional Neglect	.14	.14	.08	1.02	
Physical Neglect	-.26	.21	-.09	-1.27	

Note: Df=(5,316); F=9.95; R=.37; R^2=.14; R^2_{adj}=.12; *p<.05, **p<.01, ***p<.001

For the second regression, EDI Bulimia was the dependent variable. Table 4-18 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Bulimia (Multiple R=.45, R^2=.20, R^2_{adj} =.19, F(5, 316)=5.72, p<.001). This R^2 value can be categorized as having a small effect size. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, also emerged as significant (see Table 4-18). Squared semi-partial correlations indicated that 25% of R^2 is uniquely predicted by emotional abuse after controlling for all other CTQ scales; 20% of R^2 uniquely predicted sexual abuse.

Table 4-18

Standard Multiple Regression between the EDI Bulimia Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.48	.12	.34	4.15***	.05
Physical Abuse	-.12	.12	-.07	-.99	
Sexual Abuse	.24	.07	.20	3.36**	.04
Emotional Neglect	-.04	.12	-.03	-.37	
Physical Neglect	.15	.18	.06	.86	

Note: Df=(5,316); F=15.72; R=.45; R^2=.20; R^2_{adj}=.19; *p<.05, **p<.01, ***p<.001

In the third regression, EDI Body Dissatisfaction scale was the dependent variable. Table 4-19 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Body Dissatisfaction (Multiple R= .34, R^2=.14, R^2_{adj} =.12, F(5, 316)=9.99, p<.001). The effect size for this regression can be categorized as below the recommended baseline for small. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, were also found to be significant (see Table 4-19). Squared semi-partial correlations indicated that emotional abuse uniquely predicted 8.8% of the variance after controlling for all other CTQ scales; sexual abuse uniquely predicted 2.9% of the variance.

Table 4-19

Standard Multiple Regression between the EDI Body Dissatisfaction Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.59	.19	.27	3.17**	.03
Physical Abuse	-.13	.20	-.05	-.67	
Sexual Abuse	.25	.12	.13	2.15*	.01
Emotional Neglect	.34	.20	.14	1.74	
Physical Neglect	-.37	.29	-.10	-1.28	

Note: Df=(5,316); F=9.99; R=.34; R^2=.14; R^2_{adj}=.12; *p<.05, **p<.01, ***p<.001

For the fourth regression, EDI Low Self Esteem Scale was the dependent variable. Table 4-20 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Low Self Esteem (Multiple R=.47, R^2=.22, R^2_{adj} =.21, F(5, 316)=18.10, p<.001). According to aforementioned guidelines, this regression can be categorized as having a small effect size. In addition to the overall model being significant, three specific scales, emotional abuse, physical abuse, and sexual abuse, were also found to be significant (see Table 4-20). Squared semi-partial correlations indicated that emotional abuse uniquely predicted 14.9% of the variance after controlling for all other CTQ scales, physical abuse predicted 2.1% of the variance, and sexual abuse uniquely predicted 6.4% of the variance.

Table 4-20

Standard Multiple Regression between the EDI Low Self Esteem Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.46	.09	.40	4.97***	.07
Physical Abuse	-.20	.10	-.13	-1.99*	.01
Sexual Abuse	.16	.06	.16	2.76**	.02
Emotional Neglect	.08	.10	.06	.80	
Physical Neglect	.02	.14	.01	.16	

Note: Df=(5,316); F=18.09; R=.47; R^2=.22; R^2_{adj}=.21; *p<.05, **p<.01, ***p<.001

In the fifth regression, EDI Personal Alienation Scale served as the dependent variable. Table 4-21 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Body Dissatisfaction (Multiple R=.53, R^2=.279, R^2_{adj} =.268, F(5, 316)=24.473, p<.001). The effect size for this regression can be classified as small. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, were also found to be significant (see Table 4-21). Squared Semi-partial correlations indicated that emotional abuse uniquely predicted 11.3% of the variance after controlling for all other CTQ scales; sexual abuse uniquely predicted 5.7% of the variance.

Table 4-21

Standard Multiple Regression between the EDI Personal Alienation Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.41	.10	.33	4.30***	.06
Physical Abuse	-.06	.10	-.04	-.61	
Sexual Abuse	.19	.06	.17	3.07**	.03
Emotional Neglect	.18	.10	.13	1.78	
Physical Neglect	.06	.15	.03	.43	

Note: Df=(5,316); F=24.473; R=.53; R^2=.28; R^2_{adj}=.27; *p<.05, **p<.01, ***p<.001

For the sixth regression, EDI Interpersonal Insecurity scale was the dependent variable. Table 4-22 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Interpersonal Insecurity (Multiple R=.45, R^2=.21, R^2_{adj} =.19, F(5, 316)=16.25, p<.001). This regression indicates a small effect size. In addition to the overall model being significant, three specific scales, emotional abuse, sexual abuse, and emotional neglect, were also found to be significant. Squared semi-partial correlations indicated that emotional abuse uniquely predicted 4.4% of the variance after controlling for all other CTQ scales, sexual abuse uniquely predicted 4.4% of the variance, and emotional neglect 6.7% predicted of the variance.

The seventh regression used the EDI Interpersonal Alienation scale as the dependent variable. Table 4-23 displays the unstandardized regression coefficients (B),

Table 4-22

Standard Multiple Regression between the EDI Interpersonal Insecurity and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.24	.10	.20	2.47*	.02
Physical Abuse	-.06	.10	-.04	-.61	
Sexual Abuse	.15	.06	.15	2.53*	.02
Emotional Neglect	.30	.10	.23	3.03**	.03
Physical Neglect	-.02	.15	-.01	-.102	

Note: Df=(5,316); F=16.248; R=.45; R^2=.21; R^2_{adj}=.19; *p<.05, **p<.01, ***p<.001

the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Interpersonal Alienation (Multiple R=.55, R^2=.30, R^2_{adj} =.29, F(5, 316)=27.40, p<.001. This regression can be categorized as having a small effect size. In addition to the overall model being significant, three specific scales, emotional abuse, sexual abuse, and emotional neglect were also found to be significant (see Table 4-23). Squared semi-partial correlations indicated that emotional abuse uniquely predicted 7.3% of the variance after controlling for all other CTQ scales, sexual abuse uniquely predicted 7.3% percent of the variance, and emotional neglect uniquely predicted 5.5% of the variance.

For the eighth regression, EDI Interoceptive Deficits served as the dependent variable. Table 4-24 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model

Table 4-23

Standard Multiple Regression between the EDI Interpersonal Alienation Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.30	.08	.27	3.52***	.04
Physical Abuse	-.06	.09	-.04	-.65	
Sexual Abuse	.20	.05	.20	3.67***	.04
Emotional Neglect	.26	.09	.21	2.91**	.03
Physical Neglect	.05	.13	.02	.35	

Note: Df=(5,316); F=27.402; R=.55; R^2=.30; R^2_{adj}=.29; *p<.05, **p<.01, ***p<.001

significantly predicts Interoceptive Deficits (Multiple R=.40, R^2=.16, R^2_{adj} =.15, F(5, 316)=12.24, p<.001). This R^2 value falls below the standard small effect size categorization. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, were also found to be significant (see Table 4-24). Squared semi-partial correlations indicated that emotional abuse uniquely predicted 5% of the variance after controlling for all other CTQ scales; sexual abuse uniquely predicted 2.5% of the variance.

In a ninth regression, EDI Emotional Dysregulation was the dependent variable. Table 4-25 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Emotional Dysregulation (Multiple R=.46, R^2=.21, R^2_{adj} =.20, F(5, 316)=16.70, p<.001). This regression has a small effect size. In addition to the overall model being

significant, two specific scales, emotional abuse and sexual abuse, were also found to be significant (see Table 4-25).Squared semi-partial correlations indicated that emotional abuse uniquely predicted 8.7% of the variance after controlling for all other CTQ scales; sexual abuse uniquely predicted 10.5% of the variance.

Table 4-24

Standard Multiple Regression between the EDI Interoceptive Deficits and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.31	.13	.19	2.33*	.02
Physical Abuse	.00	.14	.00	.03	
Sexual Abuse	.18	.08	.13	2.11*	.01
Emotional Neglect	.11	.14	.06	.79	
Physical Neglect	.32	.21	.11	1.54	

Note: Df=(5,316); F=12.24; R=.40; R^2=.16; R^2_{adj}=.15; *p<.05, **p<.01, ***p<.001

Table 4-25

Standard Multiple Regression between the EDI Emotional Dysregulation Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.19	.09	.17	2.13*	.04
Physical Abuse	.11	.10	.07	1.12	
Sexual Abuse	.23	.06	.23	3.97***	.05
Emotional Neglect	.08	.10	.07	.85	
Physical Neglect	.07	.14	.03	.46	

Note: Df=(5,316); F=16.70; R=.46; R^2=.21; R^2_{adj}=.20; *p<.05, **p<.01, ***p<.001

The tenth regression used EDI Perfectionism scale as the dependent variable. Table 4-26 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Perfectionism (Multiple R=.26, R^2=.07, R^2_{adj} =.05, F(5, 316)=4.50, p<.01). Effect size is below the baseline for categorization as small for this regression. In addition to the overall model being significant, one specific scale, emotional abuse was also found to be significant (see Table 4-26). Squared semi-partial correlations indicated that emotional abuse uniquely predicted of the 11.5% variance after controlling for all other CTQ scales.

Table 4-26

Standard Multiple Regression between the EDI Perfectionism Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.32	.10	.28	3.23**	.03
Physical Abuse	.03	.10	.02	.32	
Sexual Abuse	.04	.06	.04	.63	
Emotional Neglect	-.11	.10	-.09	-1.10	
Physical Neglect	-.03	.15	-.01	-.17	

Note: Df=(5,316); F=4.499; R=.26; R^2=.07; R^2_{adj}=.05; *p<.05, **p<.01, ***p<.001

For the eleventh regression, EDI Asceticism scale was the dependent variable. Table 4-27 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model significantly predicts Asceticism (Multiple R=.42, R^2=.18, R^2_{adj} =.16, F(5, 316)=13.40, p<.001). This

regression is approaching categorization as a small effect size. In addition to the overall model being significant, two specific scales, emotional abuse and sexual abuse, were also found to be significant (see Table 4-27). Squared semi-partial correlations indicated that emotional abuse uniquely predicted 7.1% of the variance after controlling for all other CTQ scales; sexual abuse uniquely predicted 7.1% of the variance.

Table 4-27

Standard Multiple Regression between the EDI Asceticism Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.25	.08	.25	3.08**	.03
Physical Abuse	-.02	.09	-.01	-.218	
Sexual Abuse	.16	.05	.19	3.14**	.03
Emotional Neglect	-.02	.08	-.02	-.202	
Physical Neglect	.15	.12	.09	1.24	

Note: Df=(5,316); F=13.40; R=.42; R^2=.18; R^2_{adj}=.16; *p<.05, **p<.01, ***p<.001

In the last regression, EDI Maturity Fears scale was the dependent variable. Table 4-28 displays the unstandardized regression coefficients (B), the standard error (SE), the standardized regressions (β), t values, the squared semi-partial correlations (sr^2), R, R^2, and adjusted R^2. Regression results indicate that the overall model does not significantly predict Maturity Fears. Furthermore, no specific scales within this model appeared to be statistically significant.

Table 4-28

Standard Multiple Regression between the EDI Maturity Fears Scale and CTQ Scales

	B	SE	β	t	sr^2
Emotional Abuse	.22	.11	.17	1.93	
Physical Abuse	-.06	.12	-.05	-.59	
Sexual Abuse	-.03	.07	-.03	-.42	
Emotional Neglect	-.07	.12	-.05	-.59	
Physical Neglect	-.26	.17	-.12	-1.52	

Note: Df=(5,316); F=1.11; R=.13; R^2=.02; R^2_{adj}=.00; *p<.05, **p<.01, ***p<.001

Analysis of Hypothesis 2: Moderation Analysis

Moderators Relating to Entire Sample (H2: A-C)

Since half of the moderators only applied to those who experienced abuse as children, the sample was divided into two groups for moderation analysis. Hypotheses 2: A-C related to PTSD symptomology, maladaptive affect, and attachment style, and thus could be tested for both abused and non-abused members of the sample. Hypotheses 2: D-F related to the agent of abuse, the age of onset of the abuse, and the chronicity of the trauma, and thus, these sub-hypotheses were only applicable to the portion of the sample that reported childhood abuse. In order to adequately test H2, two different statistical approaches were used involving the use of correlations and tests of group differences (i.e., ANOVAs), respectively.

For the correlational approach, product-moment correlations were first calculated between all variables of interest including interaction/product variables and then compared using Steiger's Z. The interaction variables were calculated by multiplying the

hypothesized moderating variables with each of the expected moderator variables (e.g., PTSD was multiplied with trauma to produce an interaction variable which was used in analyses). In order to make the testing of the hypothesis more straightforward and to reduce the need to run extensive numbers of statistical tests, it was decided that childhood abuse would be represented as total composite score for the CTQ by summing the five CTQ subscales. Since there was no scale included in this study that directly measures PTSD, raw scores of the following three TSI scales were summed to produce a PTSD composite: Anxious Arousal (AA), Intrusive Experiences (IE), and Defensive Avoidance (DA). In a similar vein, a maladaptive affect composite score was created by summing the scores of the EDI's Low Self Esteem scale, the EDI's Emotional Dysregulation, and the Depression scale of the TSI created the maladaptive affect composite.

Steiger's Z is a test that compares bivariate correlations within a single sample and tests to determine if the correlations are statistically different in magnitude. Table 4-29 reports each of the Steiger's Z values and significances. When the Steiger's Z is significant, a statistically significant difference between correlations is supported. As stated previously, the relationship between the main effect of the CTQ composite with each of the main EDI scales were as follows: Drive for Thinness= .32 ($p<.001$), Bulimia=.41 ($p<.001$), and Body Dissatisfaction=.33 ($p<.001$). Thus, the main effects were significant for each main EDI scale. This hypothesis, however proposes that these main effects are not straightforward, but actually a result of moderated relationships.

After completing Steiger's Z tests, a series of univariate analyses of variance (ANOVA)'s were conducted in order to examine the moderators more closely. The

second statistical approach to testing Hypothesis 2, a series of univariate Analyses of Variance (ANOVAs), were completed where the EDI scales served as dependent variables and the CTQ composite, along with the hypothesized moderator variable (i.e., PTSD composite, Maladaptive Affect composite, and attachment variables) were used as independent variables. In order to facilitate these analyses, high versus low groups were generated on the CTQ composite, PTSD composite, maladaptive affect composite, and attachment variables using a mean split of the sample. Those falling above the mean were categorized as "1=high" and those scoring below the mean were categorized as "0=low."

A: PTSD

Steiger's Z testing indicated that for PTSD, the interaction effects were indeed significantly higher for the EDI scales of Drive for Thinness and Bulimia, but not for Body Dissatisfaction. Drive for Thinness yielded a statistically significant interaction effect of .43, and when tested using Steiger's Z, this correlation was significantly higher ($Z=-3.67$; $p<.001$). Bulimia yielded a statistically significant interaction effect of .53, and when tested using Steiger's Z, this correlation was significantly higher ($Z=-4.23$, $p<.001$). Body Dissatisfaction yielded a statistically significant interaction effect of .37, but when tested using Steiger's Z, this correlation was not significantly higher ($Z=-1.32$, $p=.188$). ANOVA results for the three EDI scales using PTSD and CTQ composites as independent variables are presented in Table 4-30. As can be seen, the CTQ composite produced a significant main effect for the CTQ composite for all three EDI scales (Drive for Thinness: $F(1,318)=14.74$, $p<.001$, partial $\eta^2=.04$; Bulimia: $F(1,318)=30.75$, $p<.001$, partial $\eta^2=.09$; Body Dissatisfaction: $F(1,318)=22.98$, $p<.001$, partial $\eta^2=.07$) as did the

Table 4-29

Bivariate Correlations between Drive for Thinness, Bulimia, and Body Dissatisfaction with CTQ Scores and the Interaction of CTQ Scores with Proposed Moderators A-C, including Pairwise Comparison of the Correlations (Steiger's Z Values) and Significance Levels (N=322)

	DT	B	BD
CTQ Comp	.32***	.41***	.33***
CTQ x PTSD	.43***	.53***	.37***
Steiger's Z	-3.67***	-4.23***	-1.32
CTQ Comp	.32***	.41***	.33***
CTQ x Affect	.46***	.57***	.42***
Steiger's Z	-7.31***	-8.87***	-4.65***
CTQ Comp	.32***	.41***	.33***
CTQ x Anxious Attachment	.47***	.54***	.45***
Steiger's Z	-5.06***	-4.60***	-4.03***
CTQ Comp	.32***	.41***	.33***
CTQ x Avoidant Attachment	.36***	.42***	.34***
Steiger's Z	-1.35	-0.35	-0.34

Note: *p<.05, **p<.01, ***p<.001

Table 4-30

ANOVA Summary Table: Interaction effects of PTSD Composite and CTQ Composite with Main EDI Scales (N=322)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	828.43	14.72***	.04
	PTSD	1582.72	28.16***	.08
	CTQ x PTSD	406.98	7.24**	.02
Bulimia				
	CTQ	1235.95	30.75***	.09
	PTSD	1456.61	36.24***	.10
	CTQ x PTSD	194.23	4.83*	.02
Body Dissatisfaction	CTQ	2591.62	22.98***	.07
	PTSD	1159.58	10.28**	.03
	CTQ x PTSD	250.65	2.22	

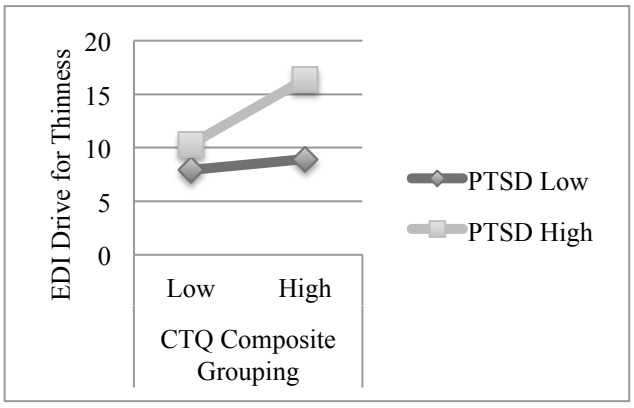
Note: *p<.05, **p<.01, ***p<.001

PTSD Composite (Drive for Thinness: $F(1,318)=28.16$, $p<.001$, partial $\eta^2= .08$; Bulimia: $F(1,318)=36.24$, $p<.001$, partial $\eta^2=.10$; Body Dissatisfaction: $F(1,318)=10.28$, $p<.01$, partial $\eta^2= .03$). For Drive for Thinness ($F(1,318)=7.24$, $p<.01$, partial $\eta^2= .02$) and Bulimia ($F(1,318)=4.83$, $p<.05$, partial $\eta^2= .02$), the interaction effects between the CTQ Composite and the PTSD Composite were significantly significant, but the interaction effects for Body Dissatisfaction were not statistically significant ($F(1,318)=2.22$, $p=.14$).

Figure 4-1 provides a graphical representation of the interaction between the CTQ groupings and the PTSD groupings on the EDI scale of Drive for Thinness. For the low CTQ-low PTSD group, the mean was 7.91 (SD=6.93, N=139). For the low CTQ-high PTSD group, the mean was 10.34 (SD=7.98, N=59). For the high CTQ-low PTSD group, the mean was 8.97 (SD=7.54, N=39). For the high CTQ-high PTSD group, the mean was 16.41 (SD=8.01, N=85).

Figure 4-1

Graphical Representation of Interaction effects of PTSD Composite Grouping and CTQ Composite Grouping with EDI Drive for Thinness Scale (N=322)

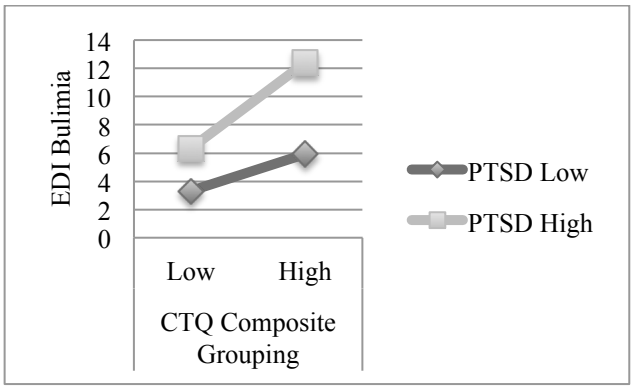


Note: $F(1,318)=7.24$, $p<.01$

Figure 4-2 provides a graphical representation of the interaction between the CTQ groupings and PTSD groupings on the EDI scale of Bulimia. For the low CTQ-low PTSD group, the mean was 3.32 (SD=4.32, N=139). For the low CTQ-high PTSD group, the mean was 6.32 (SD=6.07, N=59). For the high CTQ-low PTSD group, the mean was 5.95 (SD=5.66, N=39). For the high CTQ-high PTSD group, the mean was 12.41 (SD=9.03, N=85).

Figure 4-2

Graphical Representation of Interaction effects of PTSD Composite Grouping and CTQ Composite Grouping with EDI Bulimia Scale (N=322)

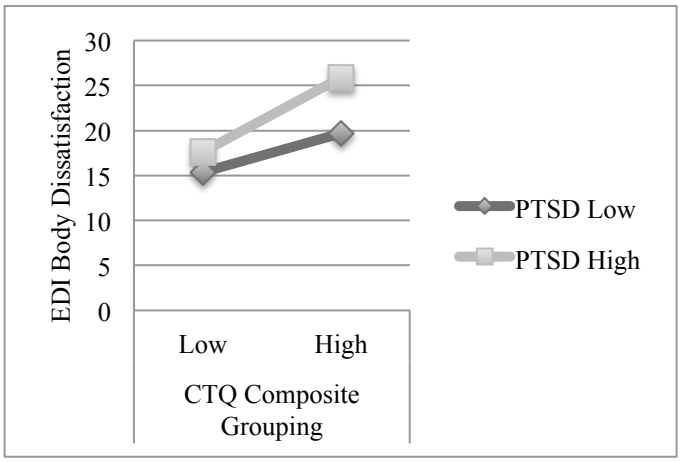


Note: F(1,318)=4.83, p<.05

Figure 4-3 provides a graphical representation of the interaction between the CTQ groupings and PTSD groupings on the EDI scale of Body Dissatisfaction. For the low CTQ-low PTSD group, the mean was 15.37 (SD=10.09, N=139). For the low CTQ-high PTSD group, the mean was 17.63 (SD=10.34, N=59). For the high CTQ-low PTSD group, the mean was 19.72 (SD=12.65, N=39). For the high CTQ-high PTSD group, the mean was 25.91 (SD=10.66, N=85).

Figure 4-3

Graphical Representation of Interaction effects of PTSD Composite Grouping and CTQ Composite Grouping with EDI Body Dissatisfaction Scale (N=322)



Note: F(1,318)=2.22, p=.137

B: Maladaptive Affect

Steiger's Z testing indicated that for Maladaptive Affect, the interaction effects were significantly higher for the all of the EDI scales. Drive for Thinness yielded a statistically significant interaction effect of .46, and when tested using Steiger's Z, this correlation was significantly higher (Z=-7.31; p<.001). Bulimia yielded a statistically significant interaction effect of .57, and when tested using Steiger's Z, this correlation was significantly higher (Z=-8.87, p<.001). Body Dissatisfaction yielded a statistically significant interaction effect of .42, and when tested using Steiger's Z, this correlation not significantly higher (Z=-4.65, p<.001).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of the Maladaptive Affect Composite and CTQ Composite with the three Main EDI scales. ANOVA results, presented in Table 4-31, showed a significant

main effect for the CTQ composite for all three EDI scales (Drive for Thinness: $F(1,318)=5.20$, $p<.05$, partial $\eta^2=.02$; Bulimia: $F(1,318)=16.69$, $p<.001$, partial $\eta^2=.05$; Body Dissatisfaction: $F(1,318)=11.58$, $p<.001$, partial $\eta^2=.04$) and the Maladaptive Affect Composite for all three EDI scales (Drive for Thinness: $F(1,318)=97.31$, $p<.001$, partial $\eta^2= .23$; Bulimia: $F(1,318)=123.65$, $p<.001$, partial $\eta^2=.28$; Body Dissatisfaction: $F(1,318)=49.20$, $p<.001$, partial $\eta^2= .13$) were statistically significant. For Drive for Thinness ($F(1,318)=6.40$, $p<.05$, partial $\eta^2= .02$) and Bulimia ($F(1,318)=6.28$, $p<.05$,

Table 4-31

ANOVA Summary Table: Interaction effects of Maladaptive Affect Composite and CTQ Composite with Main EDI Scales (N=322)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	245.02	5.20*	.02
	MalAff	4587.75	97.31***	.23
	CTQ x MalAff	301.87	6.40*	.02
Bulimia	CTQ	539.23	16.69***	.05
	MalAff	3995.22	123.65***	.28
	CTQ x MalAff	202.81	6.28*	.02
Body Dissatisfaction	CTQ	1170.20	11.58**	.04
	MalAff	4970.23	49.20***	.13
	CTQ x MalAff	276.70	2.74	

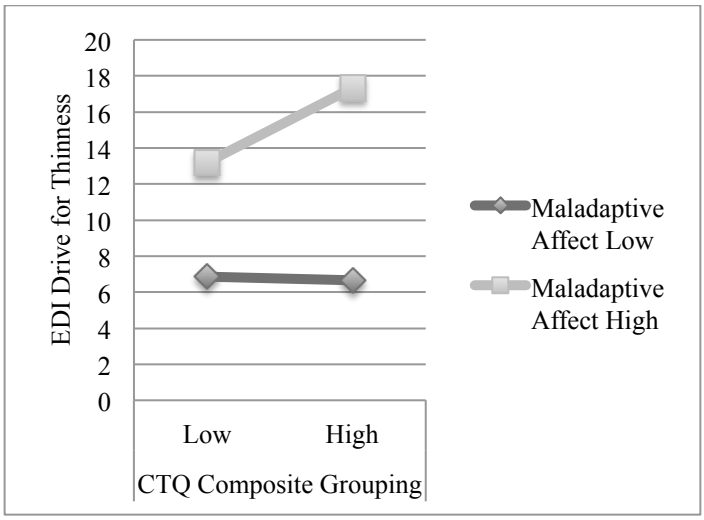
Note: *p<.05, **p<.01, ***p<.001

partial η^2= .02), the interaction effects between the CTQ Composite and the PTSD Composite were significantly significant, but the interaction effects for Body Dissatisfaction were not statistically significant.

Figure 4-4 provides a graphical representation of the interaction between the CTQ groupings and the maladaptive affect groupings on the EDI scale of Drive for Thinness. For the low CTQ-low maladaptive affect group, the mean was 6.87 (SD=6.10, N=143). For the low CTQ-high maladaptive affect group, the mean was 13.20 (SD=8.27, N=55). For the high CTQ-low maladaptive affect group, the mean was 6.65 (SD=6.19, N=38). For the high CTQ-high maladaptive affect group the mean was 14.07 (SD=8.57, N=86).

Figure 4-4

Graphical Representation of Interaction Effects of Maladaptive Affect Composite Grouping and CTQ Composite Grouping with EDI Drive for Thinness Scale (N=322)



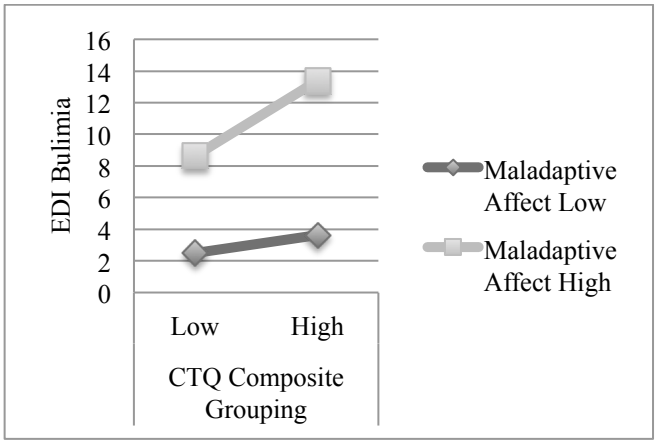
Note: F(1,318)=6.40, p<.05

Figure 4-5 provides a graphical representation of the interaction between the CTQ groupings and the maladaptive affect groupings on the EDI scale of Bulimia. For the low CTQ-low maladaptive affect group, the mean was 2.50 (SD=2.97, N=143). For the low

CTQ-high maladaptive affect group, the mean was 8.65 (SD=6.57, N=55). For the high CTQ-low maladaptive affect group, the mean was 3.63 (SD=4.13, N=38). For the high CTQ-high maladaptive affect group the mean was 13.36 (SD=8.45, N=86).

Figure 4-5

Graphical Representation of Interaction Effects of Maladaptive Affect Composite Grouping and CTQ Composite Grouping with EDI Bulimia Scale (N=322)

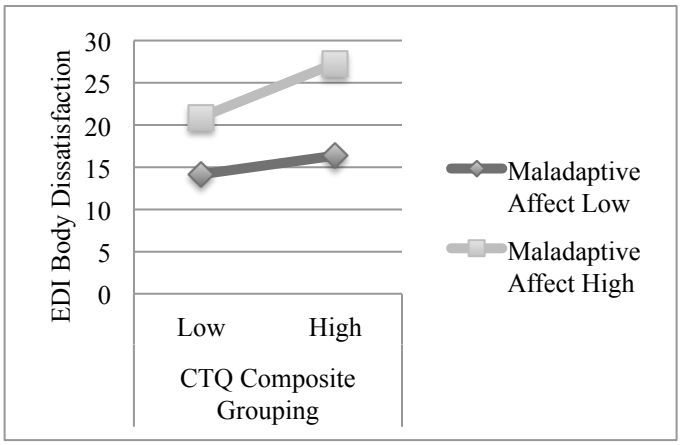


Note: F(1,318)=6.28, p<.05

Figure 4-6 provides a graphical representation of the interaction between the CTQ groupings and the maladaptive affect groupings on the EDI scale of Body Dissatisfaction. For the low CTQ-low maladaptive affect group, the mean was 14.16 (SD=9.88, N=143). For the low CTQ-high maladaptive affect group, the mean was 20.93 (SD=9.41, N=55). For the high CTQ-low maladaptive affect group, the mean was 16.37 (SD=12.35, N=38). For the high CTQ-high maladaptive affect group the mean was 27.31 (SD=9.61, N=86).

Figure 4-6

Graphical Representation of Interaction Effects of Maladaptive Affect Composite Grouping and CTQ Composite Grouping with EDI Body Dissatisfaction Scale (N=322)



Note: F(1,318)=2.74, p=.10

C1: Anxious Attachment

Steiger's Z testing indicated that for Anxious Avoidance, the interaction effects were also significantly higher for the all of the EDI scales. Drive for Thinness yielded a statistically significant interaction effect of .47, and when tested using Steiger's Z, this correlation was significantly higher (Z=-5.06; p<.001). Bulimia yielded a statistically significant interaction effect of .54, and when tested using Steiger's Z, this correlation was significantly higher (Z=-4.60, p<.001). Body Dissatisfaction yielded a statistically significant interaction effect of .45, and when tested using Steiger's Z, this correlation not significantly higher (Z=-4.03, p<.001).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of Anxious Attachment and the CTQ Composite with the three main EDI scales. ANOVA results, presented in Table 4-32, showed a significant main effect

Table 4-32

ANOVA Summary Table: Interaction Effects of Anxious Attachment and CTQ Composite with Main EDI Scales (N=322)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	596.93	11.08**	.03
	EcrAnx	2462.75	45.72***	.13
	CTQ x EcrAnx	173.43	3.22	
Bulimia	CTQ	1124.15	28.34***	.08
	EcrAnx	1659.20	41.83***	.12
	CTQ x EcrAnx	155.27	3.92*	.01
Body Dissatisfaction	CTQ	2078.19	18.99***	.06
	EcrAnx	2254.19	20.60***	.06
	CTQ x EcrAnx	52.63	0.48	

Note: *p<.05, **p<.01, ***p<.001

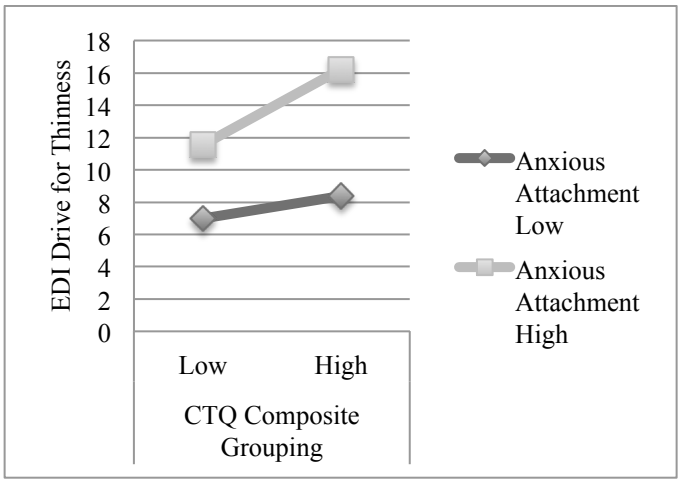
for the CTQ composite for all three EDI scales (Drive for Thinness: $F(1,318)=11.08$, $p<.01$, partial $\eta^2=.03$; Bulimia: $F(1,318)=28.34$, $p<.001$, partial $\eta^2=.08$; Body Dissatisfaction: $F(1.318)=18.99$, $p<.001$, partial $\eta^2=.06$) and the Anxious Attachment score for all three EDI scales (Drive for Thinness: $F(1,318)=45.72$, $p<.001$, partial $\eta^2=.13$; Bulimia: $F(1,318)=41.83$, $p<.001$, partial $\eta^2=.12$; Body Dissatisfaction: $F(1,318)=20.60$, $p<.001$, partial $\eta^2=.06$). For Anxious Attachment, however, the only significant interaction effect was found with Bulimia ($F(1,318)=3.92$ $p<.05$, partial $\eta^2=.01$); both Drive For Thinness and Body Dissatisfaction yielded statistically insignificant interaction effects.

Figure 4-7 provides a graphical representation of the interaction between the CTQ groupings and insecure anxious attachment on the EDI scale of Drive for Thinness. For the low CTQ-low anxious attachment group, the mean was 6.98 (SD=6.40, N=126). For the low CTQ-high anxious attachment group, the mean was 11.53 (SD=7.97, N=72). For the high CTQ-low anxious attachment group, the mean was 8.38 (SD=6.67, N=34). For the high CTQ-high anxious attachment group the mean was 16.22 (SD=8.24, N=90).

Figure 4-8 provides a graphical representation of the interaction between the CTQ groupings and insecure anxious attachment on the EDI scale of Bulimia. For the low CTQ-low anxious attachment group, the mean was 2.93 (SD=3.80, N=126). For the low CTQ-high anxious attachment group, the mean was 6.46 (SD=6.18, N=72). For the high CTQ-low anxious attachment group, the mean was 5.56 (SD=6.57, N=34). For the high CTQ-high anxious attachment group the mean was 12.20 (SD=8.66, N=90).

Figure 4-7

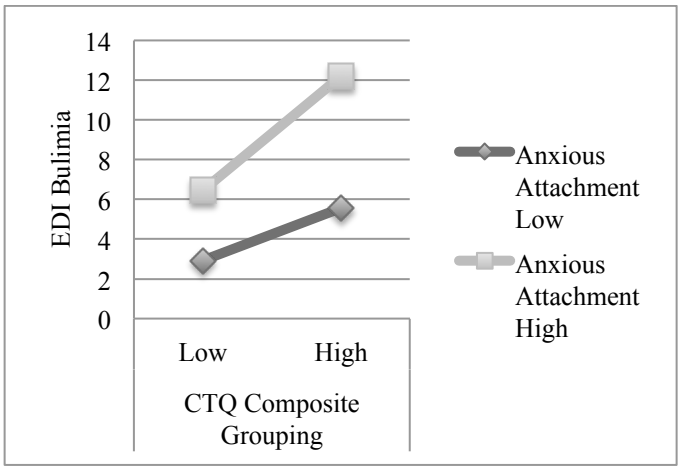
Graphical Representation of Interaction Effects of Anxious Attachment and CTQ Composite with EDI Drive for Thinness Scale (N=322)



Note: F(1,318)=3.22, p=.07

Figure 4-8

Graphical Representation of Interaction Effects of Anxious Attachment and CTQ Composite with EDI Bulimia Scale (N=322)

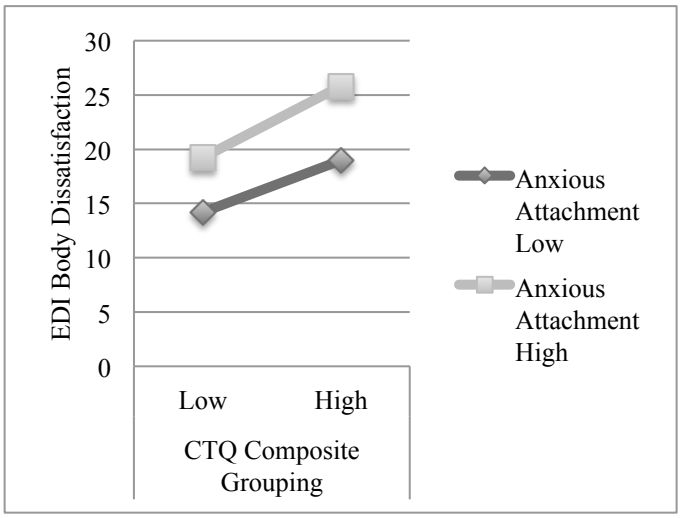


Note: F(1,318)=3.92, p<.05

Figure 4-9 provides a graphical representation of the interaction between the CTQ groupings and insecure anxious attachment on the EDI scale of Body Dissatisfaction. For the low CTQ-low anxious attachment group, the mean was 14.21 (SD=10.21, N=126). For the low CTQ-high anxious attachment group, the mean was 19.24 (SD=9.39, N=72). For the high CTQ-low anxious attachment group, the mean was 19.0 (SD=12.59, N=34). For the high CTQ-high anxious attachment group the mean was 25.83 (SD=10.74, N=90).

Figure 4-9

Graphical Representation of Interaction Effects of Anxious Attachment and CTQ Composite with EDI Body Dissatisfaction Scale (N=322)



Note: F(1,318)=.48, p=.49

C2: Avoidant Attachment

Steiger's Z testing indicated that for Avoidant Attachment, the interaction effects were not significant higher for any of the three main EDI scales, despite the respective interaction effects being significant on their own (Drive for Thinness: Z=-1.35, p=.177; Bulimia: Z=-.35, p=.726; Body Dissatisfaction: Z=-.34, p=.736).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of Avoidant Attachment and the CTQ Composite with the three main EDI scales. ANOVA results, presented in Table 4-33, showed a significant main effect for the CTQ composite for all three EDI scales (Drive for Thinness: $F(1,318)=21.60$, $p<.001$, partial $\eta^2=.06$; Bulimia: $F(1,318)=41.16$, $p<.001$, partial $\eta^2=.12$; Body Dissatisfaction: $F(1.318)=26.86$, $p<.001$, partial $\eta^2=.08$) and the Avoidant Attachment score for all three EDI scales (Drive for Thinness: $F(1,318)=9.61$, $p<.01$, partial $\eta^2=.03$; Bulimia: $F(1,318)=10.27$, $p<.01$, partial $\eta^2=..03$; Body Dissatisfaction: $F(1,318)=6.63$, $p<.01$, partial $\eta^2=.02$). For Avoidant Attachment, none of the interaction effects yielded significant results.

Figure 4-10 provides a graphical representation of the interaction between the CTQ groupings and insecure avoidant attachment on the EDI scale of Drive for Thinness. For the low CTQ-low avoidant attachment group, the mean was 7.43 (SD=6.54, N=127). For the low CTQ-high avoidant attachment group, the mean was 10.77 (SD=8.17, N=71). For the high CTQ-low avoidant attachment group, the mean was 12.25 (SD=8.88, N=36). For the high CTQ-high avoidant attachment group the mean was 14.82 (SD=8.37, N=88).

Figure 4-11 provides a graphical representation of the interaction between the CTQ groupings and insecure avoidant attachment on the EDI scale of Bulimia. For the low CTQ-low avoidant attachment group, the mean was 3.40 (SD=4.34, N=127). For the low CTQ-high avoidant attachment group, the mean was 5.66 (SD=5.95, N=71). For the

Table 4-33

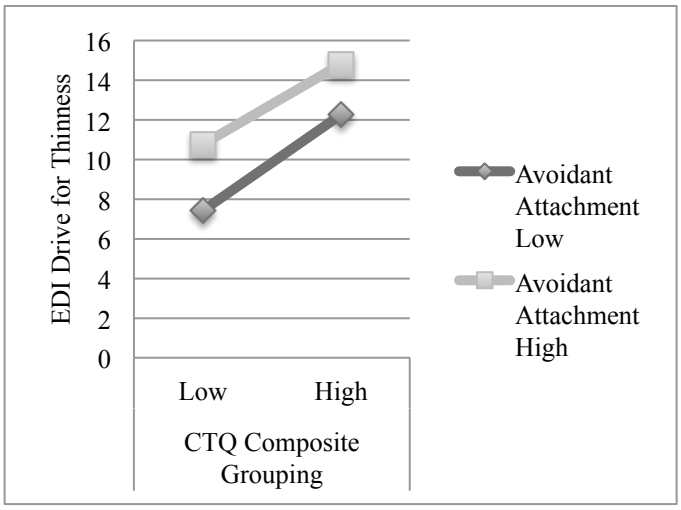
ANOVA Summary Table: Interaction Effects of Avoidant Attachment and CTQ Composite with Main EDI Scales (N=322)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	1284.91	21.60***	.06
	EcrAvoid	571.61	9.61**	.03
	CTQ x EcrAvoid	9.79	0.17	
Bulimia	CTQ	1788.26	41.16***	.12
	EcrAvoid	446.18	10.27**	.03
	CTQ x EcrAvoid	8.03	0.19	
Body Dissatisfaction	CTQ	3041.41	26.86***	.08
	EcrAvoid	750.40	6.63**	.02
	CTQ x EcrAvoid	102.38	0.90	

Note: *p<.05, **p<.01, ***p<.001

Figure 4-10

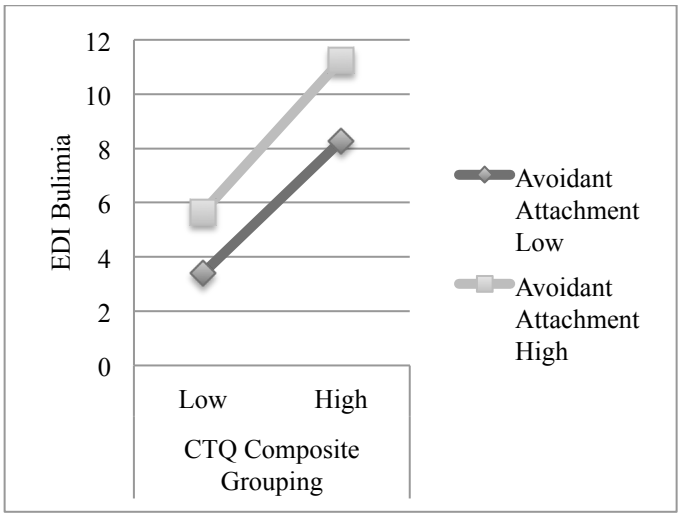
Graphical Representation of Interaction Effects of Avoidant Attachment and CTQ Composite with EDI Drive for Thinness Scale (N=322)



Note: F(1,318)=0.17, p=.69

Figure 4-11

Graphical Representation of Interaction Effects of Avoidant Attachment and CTQ Composite with EDI Bulimia Scale (N=322)



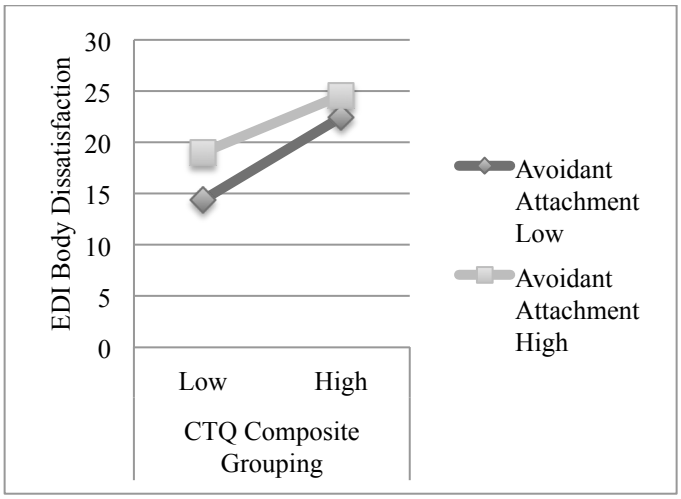
Note: F(1,318)=.0.19, p=.67

high CTQ-low avoidant attachment group, the mean was 8.28 (SD=8.33, N=36). For the high CTQ-high avoidant attachment group the mean was 11.24 (SD=8.67, N=88).

Figure 4-12 provides a graphical representation of the interaction between the CTQ groupings and insecure avoidant attachment on the EDI scale of Body Dissatisfaction. For the low CTQ-low avoidant attachment group, the mean was 14.38 (SD=9.92 N=127). For the low CTQ-high avoidant attachment group, the mean was 19.01 (SD=10.05, N=71). For the high CTQ-low avoidant attachment group, the mean was 22.44 (SD=12.90, N=36). For the high CTQ-high avoidant attachment group the mean was 24.58 (SD=11.09, N=88).

Figure 4-12

Graphical Representation of Interaction Effects of Avoidant Attachment and CTQ Composite with EDI Body Dissatisfaction Scale (N=322)



Note: F(1,318)=.90, p=.34

Moderators Relating Only To Abused Sample (H2: D-F)

As stated previously, the last three moderators (D-F) were tested only on the subset of the sample that endorsed experiencing childhood abuse since they were not applicable to the rest of the sample. Steiger's Z tests were also performed to analyze the remaining moderators for the presences of significant interaction effects. These results are displayed in Table 4-34. As a reminder, the main effects for the main eating disorder scales with the CTQ comp were as follows: Drive for Thinness: .32 ($p<.001$), Bulimia: .41 ($p<.001$), and Body Dissatisfaction: .33 ($p<.001$).

D: Agent of Abuse

Interestingly, Steiger's Z testing indicated that for Agent of Abuse, the interaction effects were significantly lower for the all of the EDI scales. Drive for Thinness yielded an interaction effect of .12, which alone did not emerge as significant, but when tested using Steiger's Z, this correlation was significantly lower than the main effect ($Z=2.99$; $p<.001$). Bulimia yielded an interaction effect of .15, which also did not emerge as significant, but when tested using Steiger's Z, it was significantly lower than the main effect ($Z=3.99$, $p<.001$). Body Dissatisfaction yielded an interaction effect of .16, which again was significant, but when tested using Steiger's Z, emerged as significantly lower ($Z=2.57$, $p<.01$).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of the agent of abuse and the CTQ composite with the three main EDI scales. ANOVA results, presented in Table 4-35, showed a significant main effect of the CTQ with all three EDI scales (Drive for Thinness: $F(1,142)=19.81$, $p<.001$, partial

Table 4-34

Bivariate Correlations between Drive for Thinness, Bulimia, and Body Dissatisfaction with CTQ Scores and the Interaction of CTQ Scores with Proposed Moderators D-F, including Pairwise Comparison of the Correlations (Steiger's Z Values) and Significance Levels (N=146)

	DT	B	BD
CTQ Comp	.32***	.41***	.33***
CTQ x Agent	.12	.15	.16
Steiger's Z	2.99**	3.99***	2.57**
CTQ Comp	.32***	.41***	.33***
CTQ x Age	.34***	.32***	.24**
Steiger's Z	-.44	2.00*	1.94
CTQ Comp	.32***	.41***	.33***
CTQ x Chronicity	.26**	.27**	.28**
Steiger's Z	1.54	3.66***	1.29

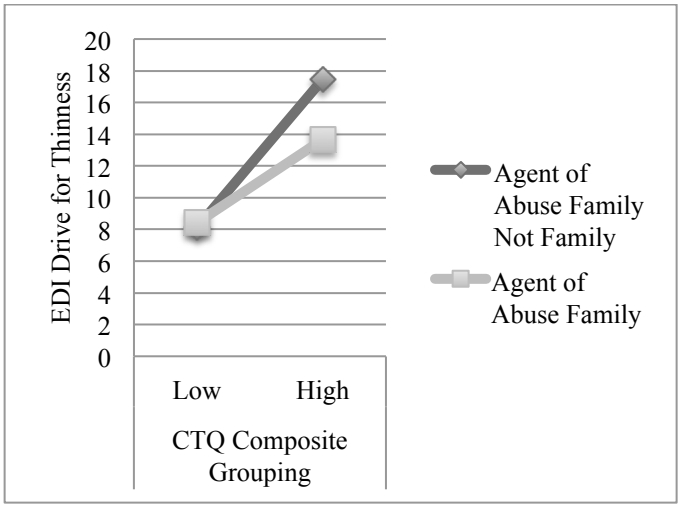
Note: *p<.05, **p<.01, ***p<.001

η^2=.12; Bulimia: F(1.142)=17.35, p<.001, partial η^2=.11; Body Dissatisfaction: F(1,142)=13/07, p<.001, partial η^2=.08). The Agent of Abuse (family or non family member) was not significant as a main effect for any of the EDI scales nor was the interaction effect significant for any of the EDI scales.

Figure 4-13 provides a graphical representation of the interaction between the CTQ groupings and the agent of abuse on the EDI scale of Drive for Thinness. Agent of abuse was categorized as "family member" or "non-family member." For the low CTQ-non-family member group, the mean was 8.13 (SD=7.93, N=16). For the low CTQ-family member, the mean was 8.43 (SD=5.78, N=23). For the high CTQ-non-family member, the mean was 17.47 (SD=8.64, N=19). For the high CTQ-family member group the mean was 13.65 (SD=8.25, N=88).

Figure 4-13

Graphical Representation of Interaction Effects of Agent of Abuse and CTQ Composite with EDI Drive for Thinness Scale (N=146)



Note: F(1,142)=1.60, p=.21

Table 4-35

ANOVA Summary Table: Interaction Effects of Agent of Abuse and CTQ Composite (N=146)

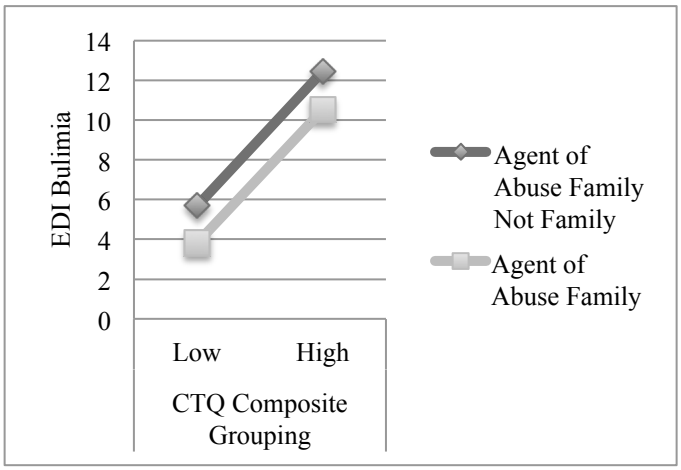
	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	1247.50	19.81***	.12
	Agent	72.74	1.16	
	CTQ x Agent	100.63	1.60	
Bulimia	CTQ	1067.70	17.35***	.11
	Agent	86.02	1.40	
	CTQ x Agent	0.06	.00	
Body Dissatisfaction	CTQ	1661.22	13.07***	.08
	Agent	6.61	0.05	
	CTQ x Agent	145.48	1.15	

Note: *p<.05, **p<.01, ***p<.001

Figure 4-14 provides a graphical representation of the interaction between the CTQ groupings and the agent of abuse on the EDI scale of Bulimia. For the low CTQ-non-family member group, the mean was 5.69 (SD=6.73, N=16). For the low CTQ-family member, the mean was 3.83 (SD=3.69, N=23). For the high CTQ-non-family member, the mean was 12.47 (SD=9.05, N=19). For the high CTQ-family member group the mean was 10.51 (SD=8.50, N=88).

Figure 4-14

Graphical Representation of Interaction Effects of Agent of Abuse and CTQ Composite with EDI Bulimia Scale (N=146)



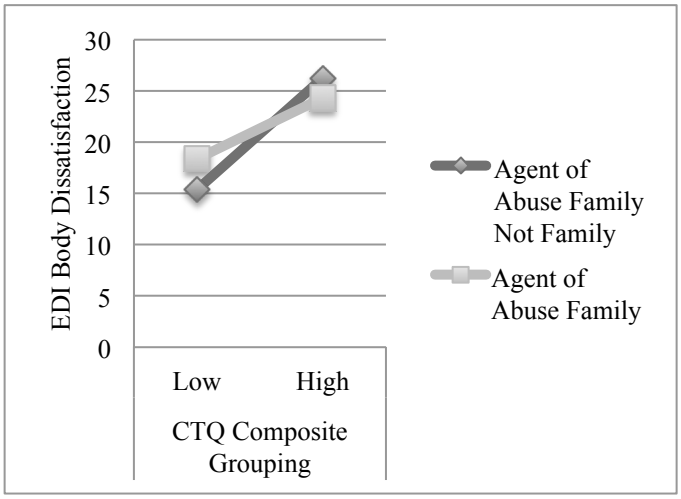
Note: F(1,142)=.00, p=.98

Figure 4-15 provides a graphical representation of the interaction between the CTQ groupings and the agent of abuse on the EDI scale of Body Dissatisfaction. For the low CTQ-non-family member group, the mean was 15.38 (SD=10.41, N=16). For the low CTQ-family member, the mean was 18.39 (SD=10.73, N=23). For the high CTQ-non-

family member, the mean was 26.26 (SD=10.78, N=19). For the high CTQ-family member group the mean was 24.31 (SD=11.65, N=88).

Figure 4-15

Graphical Representation of Interaction Effects of Agent of Abuse and CTQ Composite with EDI Body Dissatisfaction Scale (N=146)



Note: F(1,142)=1.15, p=.29

E: Age at Onset of Trauma

Steiger's Z testing indicated that for Age of Abuse Onset, the interaction effect was significant only for Bulimia, and like Agent of Abuse, was lower than the main effect. Bulimia yielded a statistically significant interaction effect of .32, and when tested using Steiger's Z, this correlation was significantly lower (Z=2.00, p<.05). Although both Drive for Thinness or Body Dissatisfaction yielded statistically significant interaction effects of .34 and .24, respectively, these correlations were not significantly different from the main effect correlations when tested using Steiger's Z (Drive for Thinness: Z=-.44, p=.662; Body Dissatisfaction: Z=1.94, p=.053).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of the age of abuse onset and the CTQ composite with the three main EDI scales. ANOVA results, presented in Table 4-36, showed a significant main effect of the CTQ with Drive for Thinness ($F(1,142)=5.03$, $p<.05$, partial $\eta^2=.03$) and Bulimia ($F(1,142)=7.51$, $p<.01$, partial $\eta^2=.05$), but not with Body Dissatisfaction. Age of onset was not found to have a main effect and none of the interaction effects showed statistical significance.

Figure 4-16 provides a graphical representation of the interaction between the CTQ groupings and the age of abuse on the EDI scale of Drive for Thinness. For the low CTQ-after 14 group, the mean was 5.38 (SD=5.60, N=8). For the low CTQ-before age14 group, the mean was 9.06 (SD=6.77, N=31). For the high CTQ-after age14 group, the mean was 10.17 (SD=6.91, N=6). For the high CTQ-before age 14 group the mean was 14.57 (SD=8.45, N=101).

Figure 4-17 provides a graphical representation of the interaction between the CTQ groupings and the age of abuse on the EDI scale of Bulimia. For the low CTQ-after 14 group, the mean was 2.38 (SD=2.56, N=8). For the low CTQ-before age14 group, the mean was 5.16 (SD=5.53, N=31). For the high CTQ-after age14 group, the mean was 9.00 (SD=8.85, N=6). For the high CTQ-before age 14 group the mean was 10.97 (SD=8.61, N=101).

Table 4-36

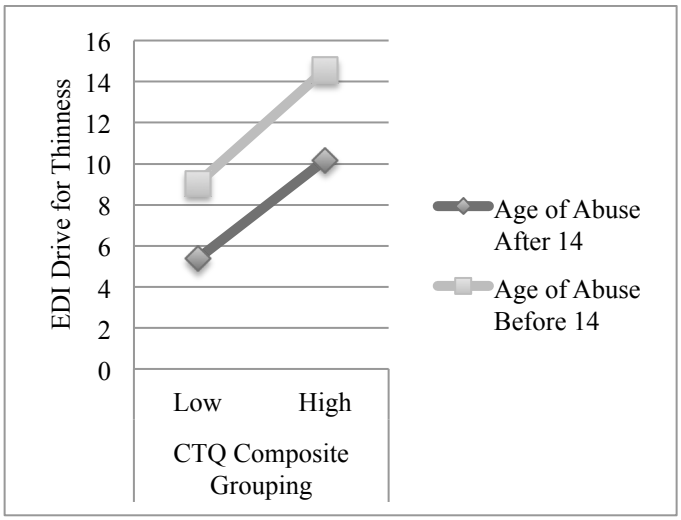
ANOVA Summary Table: Interaction Effects of Age of Abuse Onset and CTQ Composite (N=146)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	317.89	5.03*	.03
	Age	196.40	3.11	
	CTQ x Age	1.54	.02	
Bulimia	CTQ	463.13	7.51**	.05
	Age	67.78	1.10	
	CTQ x Age	2.00	.03	
Body Dissatisfaction	CTQ	311.29	2.45	
	Age	7.18	.06	
	CTQ x Age	114.92	.90	

Note: *p<.05, **p<.01, ***p<.001

Figure 4-16

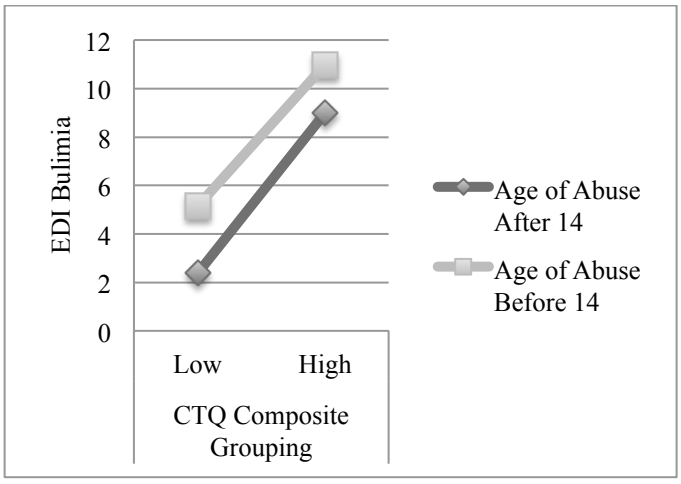
Graphical Representation of Interaction Effects of Age at Onset of Abuse and CTQ Composite with EDI Drive for Thinness Scale (N=146)



Note: F(1,142)=0.02, p=.88

Figure 4-17

Graphical Representation of Interaction Effects of Age at Onset of Abuse and CTQ Composite with EDI Bulimia Scale (N=146)



Note: F(1,146)=.03, p=.86

Figure 4-18 provides a graphical representation of the interaction between the CTQ groupings and the age of abuse on the EDI scale of Body Dissatisfaction. For the low CTQ-after 14 group, the mean was 19.00 (SD=8.07, N=8). For the low CTQ-before age14 group, the mean was 16.68 (SD=11.18, N=31). For the high CTQ-after age14 group, the mean was 21.00 (SD=9.61, N=6). For the high CTQ-before age 14 group the mean was 24.87 (SD=11.58, N=101).

F: Chronicity of Trauma

Steiger's Z testing indicated that for Chronicity, the interaction effect was again significant only for Bulimia, and was lower than the main effect. Bulimia yielded a statistically significant interaction effect of .27, and when tested using Steiger's Z, this correlation was significantly lower ($Z=3.66$, $p<.001$). Although both Drive for Thinness or Body Dissatisfaction yielded statistically significant interaction effects of .26 and .28, respectively, these correlations were not significantly different from the main effect correlations when tested using Steiger's Z (Drive for Thinness: $Z=1.54$, $p=.128$; Body Dissatisfaction: $Z=1.29$, $p=.198$).

A univariate analysis of variance (ANOVA) was conducted to examine the interaction effects of the chronicity of abuse and the CTQ composite with the three main EDI scales. ANOVA results, presented in Table 4-37, showed a significant main effect of the CTQ with Drive for Thinness: ($F(1,142)=15.77$, $p<.001$, partial $\eta^2=.06$), Bulimia: ($F(1,142)=15.40$, $p<.001$, partial $\eta^2=.10$), and Body Dissatisfaction: ($F(1,142)=8.82$, $p<.01$, partial $\eta^2=.06$). Chronicity was not found to have a main effect for any of the three EDI scales and no statistically significant interaction effects were found.

Table 4-37

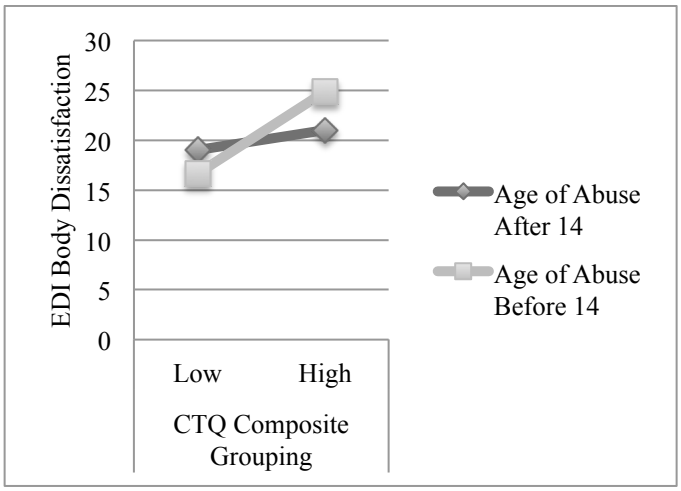
ANOVA Summary Table: Interaction Effects of Chronicity of Abuse and CTQ Composite (N=146)

	Source	MS	F	Partial η^2
Drive for Thinness	CTQ	1014.54	15.77***	.06
	Chronicity	4.70	.07	
	CTQ x Chronicity	24.64	.38	
Bulimia	CTQ	953.91	15.40***	.10
	Chronicity	30.53	.49	
	CTQ x Chronicity	14.08	.23	
Body Dissatisfaction	CTQ	1120.45	8.82**	.06
	Chronicity	136.90	1.08	
	CTQ x Chronicity	0.09	.00	

Note: *p<.05, **p<.01, ***p<.001

Figure 4-18

Graphical Representation of Interaction Effects of Age at Onset of Abuse and CTQ Composite with EDI Body Dissatisfaction Scale (N=146)



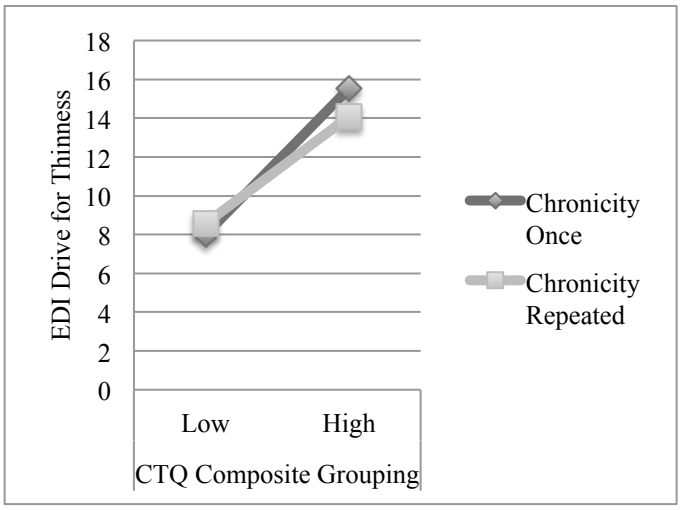
Note: F(1,142)=.90, p=.34

Figure 4-19 provides a graphical representation of the interaction between the CTQ groupings and the chronicity of abuse on the EDI scale of Drive for Thinness. For the low CTQ-once incident group, the mean was 8.00 (SD=7.51, N=18). For the low CTQ-repeated incidents group, the mean was 8.47 (SD=6.00, N=21). For the high CTQ-once incident group, the mean was 15.53 (SD=9.62, N=19). For the high CTQ-repeated incidents group the mean was 14.07 (SD=8.16, N=88).

Figure 4-20 provides a graphical representation of the interaction between the CTQ groupings and the chronicity of abuse on the EDI scale of Bulimia. For the low CTQ-once incident group, the mean was 5.61 (SD=6.34, N=18). For the low CTQ-repeated incidents group, the mean was 3.71 (SD=3.84, N=21). For the high CTQ-once incident group, the mean was 11.16 (SD=9.47, N=19). For the high CTQ-repeated incidents group the mean was 10.80 (SD=8.44, N=88).

Figure 4-19

Graphical Representation of Interaction Effects of Chronicity of Abuse and CTQ Composite with EDI Drive for Thinness Scale (N=146)

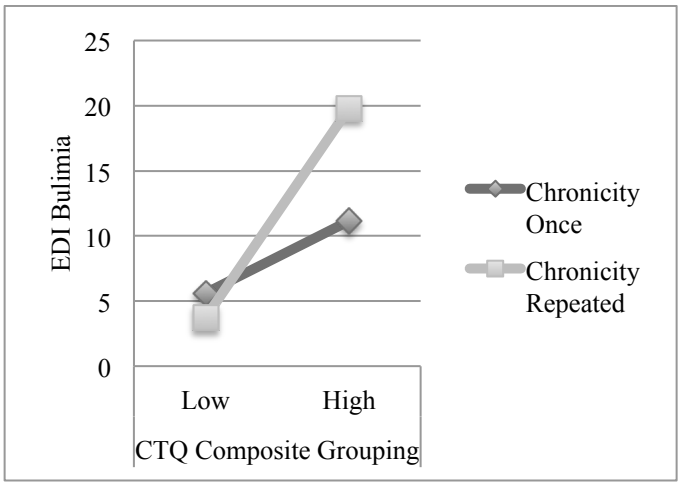


Note: F(1,142)=.38, p=.54

Figure 4-21 provides a graphical representation of the interaction between the CTQ groupings and the chronicity of abuse on the EDI scale of Body Dissatisfaction. For the low CTQ-once incident group, the mean was 15.83 (SD=9.42, N=18). For the low CTQ-repeated incidents group, the mean was 18.29 (SD=11.57, N=21). For the high CTQ-once incident group, the mean was 22.74 (SD=13.12, N=19). For the high CTQ-repeated incidents group the mean was 25.07 (SD=11.12, N=88).

Figure 4-20

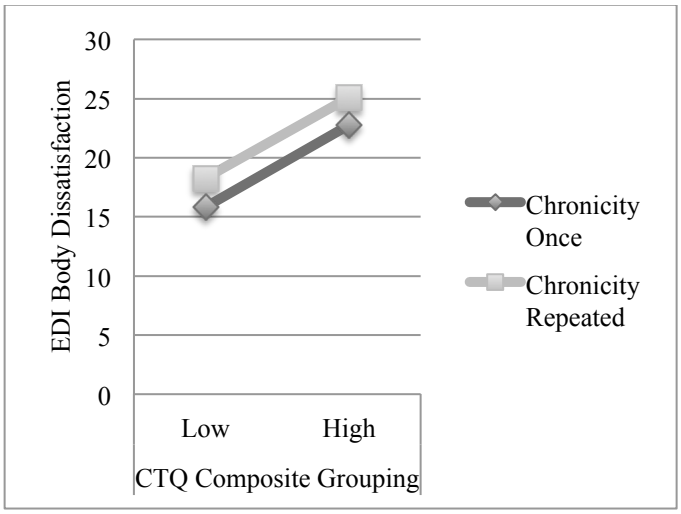
Graphical Representation of Interaction Effects of Chronicity of Abuse and CTQ Composite with EDI Bulimia Scale (N=146)



Note: F(1,142)=.23, p=.63

Figure 4-21

Graphical Representation of Interaction Effects of Chronicity of Abuse and CTQ Composite with EDI Body Dissatisfaction Scale (N=146)



Note: F(1,142)=.09, p=.98

Analysis of Hypothesis 3: Meditational Analysis

Structural equation modeling (SEM) was used to investigate the meditational relationships proposed in Hypothesis 3. SEM is a more powerful approach to multiple regression, since it takes into account many latent independent variable, the indicators that they are measured by, multiple latent dependent variables, their indicators, moderators, mediators, measurement error, and correlated error terms. SEM has multiple advantages compared to multiple regressions. For example, SEM allows interpretation of results even when multicollinearity occurs. Also, SEM is not as susceptible to interpretation error as multiple regression and is therefore a more robust statistical analysis. Furthermore, it allows the researcher to model mediating variables instead of looking at each coefficient individually. For the aforementioned reasons, SEM was chosen as the preferred method for analyzing mediation effects for hypothesis three.

The first model (shown in Figure 4-22) depicts the relationship of trauma to eating disorders without the presence of the hypothesized mediators (i.e., dissociation, family dysfunction, and body dissatisfaction). More specifically, this model depicts a directional hybrid structural equation model where the latent construct of trauma, operationally defined as a measurement model comprised of the five abuse scales of the CTQ, is used to predict the latent construct of eating disorders, operationally defined as a measurement model comprised of two EDI scales- Drive for Thinness and Bulimia. This model was tested as a preliminary step to evaluate the potency of trauma in predicting eating disorder symptomology.

According to this model, all paths are statistically significant ($p<.001$). Standardized regression weights indicate that relationships between variables are as

follows: eating disorders with trauma: β =.47 (p<.001; effect size= medium-large), emotional abuse with trauma: β = .88 (p<.001; large effect size), physical abuse with trauma: β = .66 (p<.001; large effect size), sexual abuse with trauma: β =.53 (p<.001; medium-large effect size), emotional neglect with trauma: β =.78 (p<.001; large effect size), physical neglect with trauma: β =.73 (p<.001; large effect size), drive for thinness with eating disorders: β =.72 (p<.001; large effect size), bulimia with eating disorders: β =.92 (p<.001; large effect size). Overall model fit appears to be adequate especially considering the lack of models proposed in the research to date (e.g. Chi-Square=58.51, df=13, p<.001, Chi-square/df=4.50, GFI=.95, AGFI=.89, NFI=.94, RFI=.90, IFI=.95, TLI=.92, CFI=.95, RMSEA=.10, SRMR=.05).

Since the third hypothesis suggests, however, that the relationship is not in fact direct, a second model was tested to the mediational effects of dissociation, family dysfunction, and body dissatisfaction on the trauma-eating disorder association. Dissociation was operationally defined as the score on the Dissociation scale of the TSI, Family Dysfunction was operationally defined as the total score on the IFR, and Body Dissatisfaction was operationally defined as the score on the Body Dissatisfaction scale of the EDI.

In this second model, Dissociation and Body Dissatisfaction emerged as significant mediators in the relationship between trauma and eating disorders. Standardized regression weights were as follows: dissociation with trauma: β =.58 (p<.001; medium-large effect size), Dissociation with Eating Disorders: β=.27 (p< .001; small-medium effect size), body dissatisfaction with trauma: β =.38 (p<.001; small-medium effect size).

Family Dysfunction, however, did not significantly mediate the relationship between trauma and eating disorders. Family dysfunction was significantly related to trauma (β =.78; p<.001) but the regression weight was not significant with regards to eating disorders (β =-.06; p=.385). Additionally, with the addition of the mediators to the model, the direct path between trauma and eating disorders was no longer significant (β =.08; p=.359).

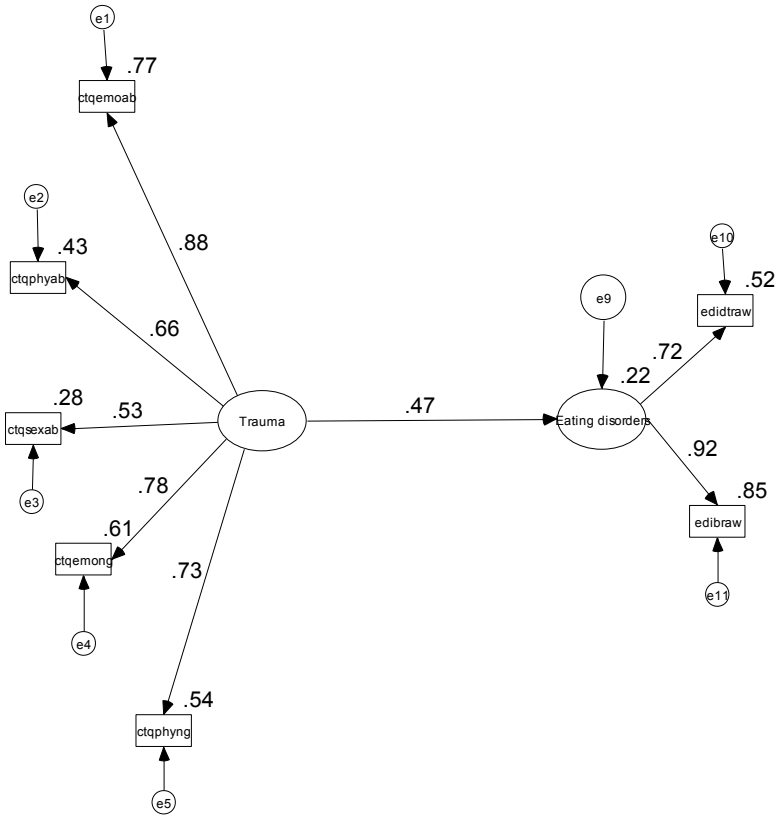
Thus, this second model indicates that Dissociation and Body Dissatisfaction did not partially mediate the relationship between trauma and eating disorders, but in fact, completely mediated the relationship. Overall model fit remains generally adequate (e.g. Chi-Square=139.38, df=31, p<.001, Chi-square/df=4.50, GFI=.92, AGFI=.86, NFI=.92, RFI=.88, IFI=.93, TLI=.90, CFI=.93, RMSEA=.10, SRMR=.06). Figure 4-23 presents the complete model with standardized regression weights included.

The final SEM model, depicted in Figure 4-24, has been adjusted to support the data provided by the second model. Based on those data, the direct path between trauma and eating disorders was removed, as was family dysfunction since it was a non-significant mediator. After making these changes to the model, all regression weights emerged again as being significant. Standardized regression weights for this model were as follows: Dissociation with Trauma: β =.56 (p<.001, medium-large effect size), body dissatisfaction with trauma: β =.37 (p<.001, small-medium effect size), Eating Disorders with Dissociation: β =.28 (p<.001, small-medium effect size), eating Disorders with body dissatisfaction: β=.74 (p<.001, large effect size), emotional abuse with trauma: β =.88 (p<.001, large effect size), physical abuse with trauma: β = .65 (p<.001, large effect size), sexual abuse with trauma: β = .54 (p<.001, medium-large effect size), emotional neglect

with trauma: β = .78 (p<.001, large effect size), physical neglect with trauma: β = .73 (p<.001, large effect size), drive for thinness with eating disorders: β =.89 (p<.001, large effect size), bulimia with eating disorders: β = .73 (p<.001, large effect size). In terms of overall model fit, it remained adequate, despite all paths being significant and most having medium-large effect sizes (e.g. Chi-Square=110.01, df=25, p<.001, Chi-square/df=4.4, GFI=.93, AGFI=.87, NFI=.92, RFI=.89, IFI=.94, CFI=.94, RMSEA=.10, SRMR=.07).

Figure 4-22

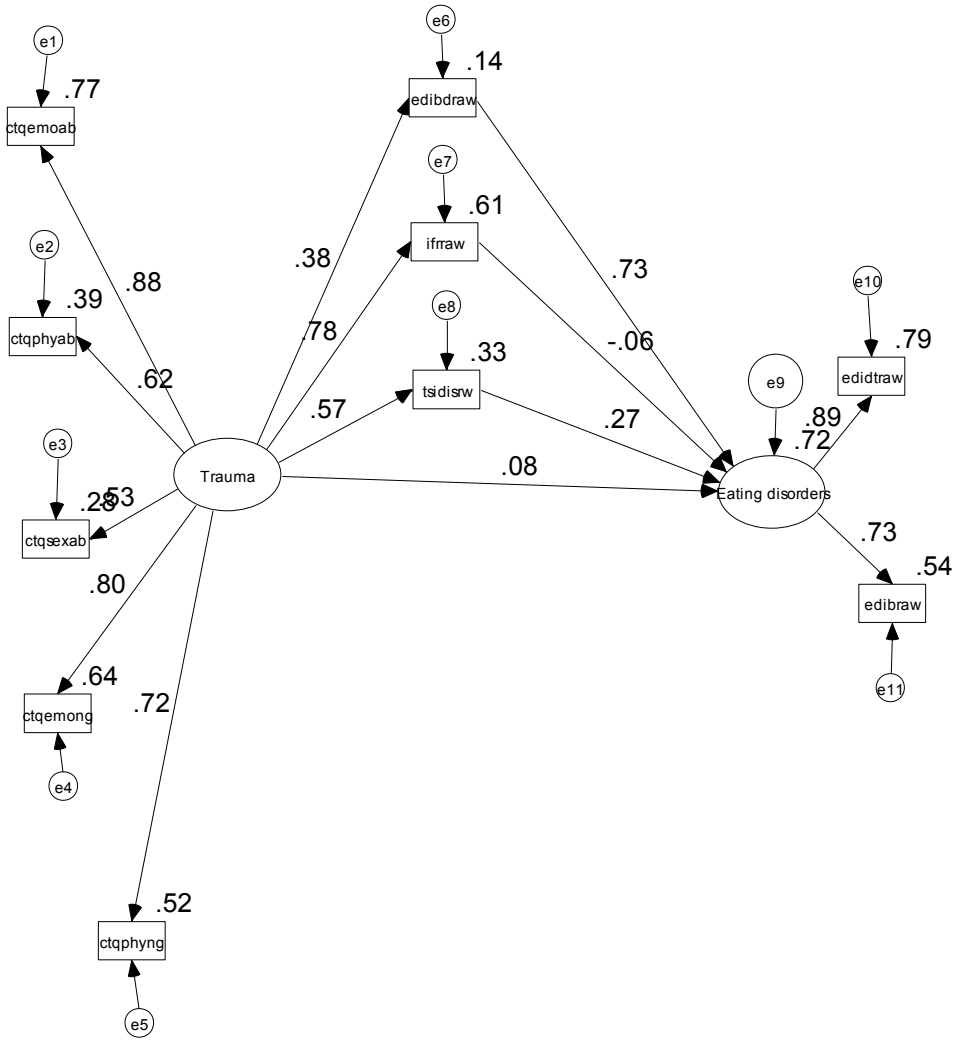
Model Depicting the Relationship between Trauma and Eating Disorders without Hypothesized Mediators with Standardized Regression Weights



Note: ctqemoab= Emotional Abuse; ctqphyab= Physical Abuse; ctqsexab= Sexual Abuse; ctqemong= Emotional Neglect; ctqphyng= Physical Neglect, edibdraw= EDI Body Dissatisfaction Raw Score; ifrraw= IFR Total Raw Score; tsidisrw= TSI Dissociation Raw Score; edidtraw= EDI Drive for Thinness Raw Score, edibraw= EDI Bulimia Raw Score

Figure 4-23

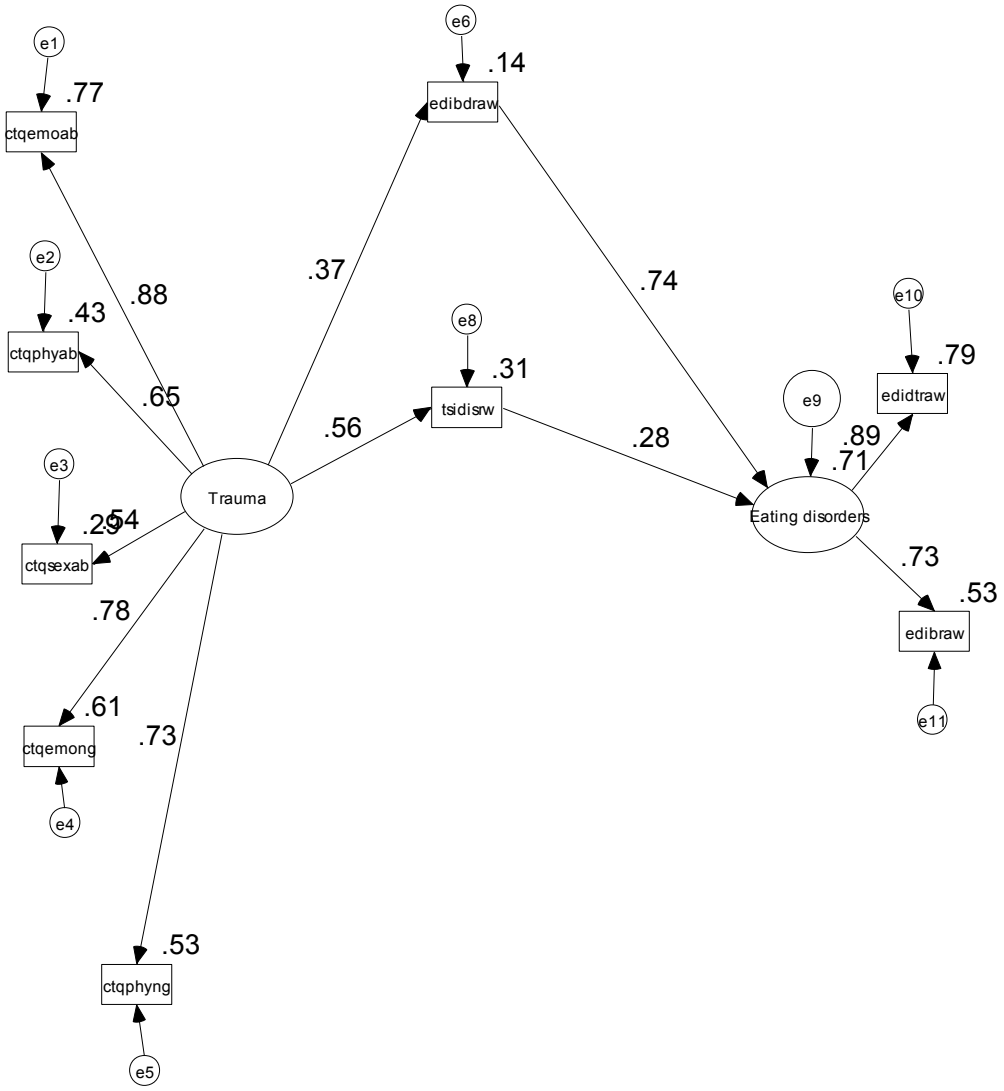
Hypothesized SEM Model With Regression Weights



Note: ctqemoab= Emotional Abuse; ctqphyab= Physical Abuse; ctqsexab= Sexual Abuse; ctqemong= Emotional Neglect; ctqphyng= Physical Neglect, edibdraw= EDI Body Dissatisfaction Raw Score; ifrraw= IFR Total Raw Score; tsidisrw= TSI Dissociation Raw Score; edidtraw= EDI Drive for Thinness Raw Score, edibraw= EDI Bulimia Raw Score

Figure 4-24

Adjusted SEM Model With Regression Weights



Note: ctqemoab= Emotional Abuse; ctqphyab= Physical Abuse; ctqsexab= Sexual Abuse; ctqemong= Emotional Neglect; ctqphyng= Physical Neglect, edibdraw= EDI Body Dissatisfaction Raw Score; tsidisrw= TSI Dissociation Raw Score; edidtraw= EDI Drive for Thinness Raw Score, edibraw= EDI Bulimia Raw Score

Analysis of Hypothesis 4

Based on the statistical analysis of hypotheses one through three, it became evident that the conceptual model driving hypothesis four needed modification. Variables from hypothesis two and three that did not emerge as significant were removed from the conceptual model. Furthermore, where interaction effects emerged as significant, main effects were removed from the model. Because of these findings from previous hypotheses, it became evident that the final nine parts of H4 were not testable. The three sub-hypotheses relating to agent of abuse, age at onset of abuse, and chronicity of trauma were unable to be tested in the model due to the lack of power and the unbalanced distribution of subjects between groups.

Structural equation modeling was then used to test the interaction effects of the remaining variables. Due to the complexity of the model, this approach was more parsimonious than conducting a sequence of multiple regressions and furthermore reduced the probability of error. Due to the high degree of correlation between the latent variables, a mean-centering approach was used to reduce the effects of multicollinearity. Centering is a statistical technique that allows for a clearer analysis of main effects and interaction effects without changing the computational precision of the parameters (Echambadi & Hess, 2004).

The SEM model depicted in Figure 4-25 demonstrates the relationships of the variables that emerged as significant. When the model was originally tested, the error terms were highly correlated between the two forms of neglect (physical and emotional). A measurement error term refers to the measurement error factor associated with a given indicator. In a regression model, the error term is assumed to be zero, but with SEM, the

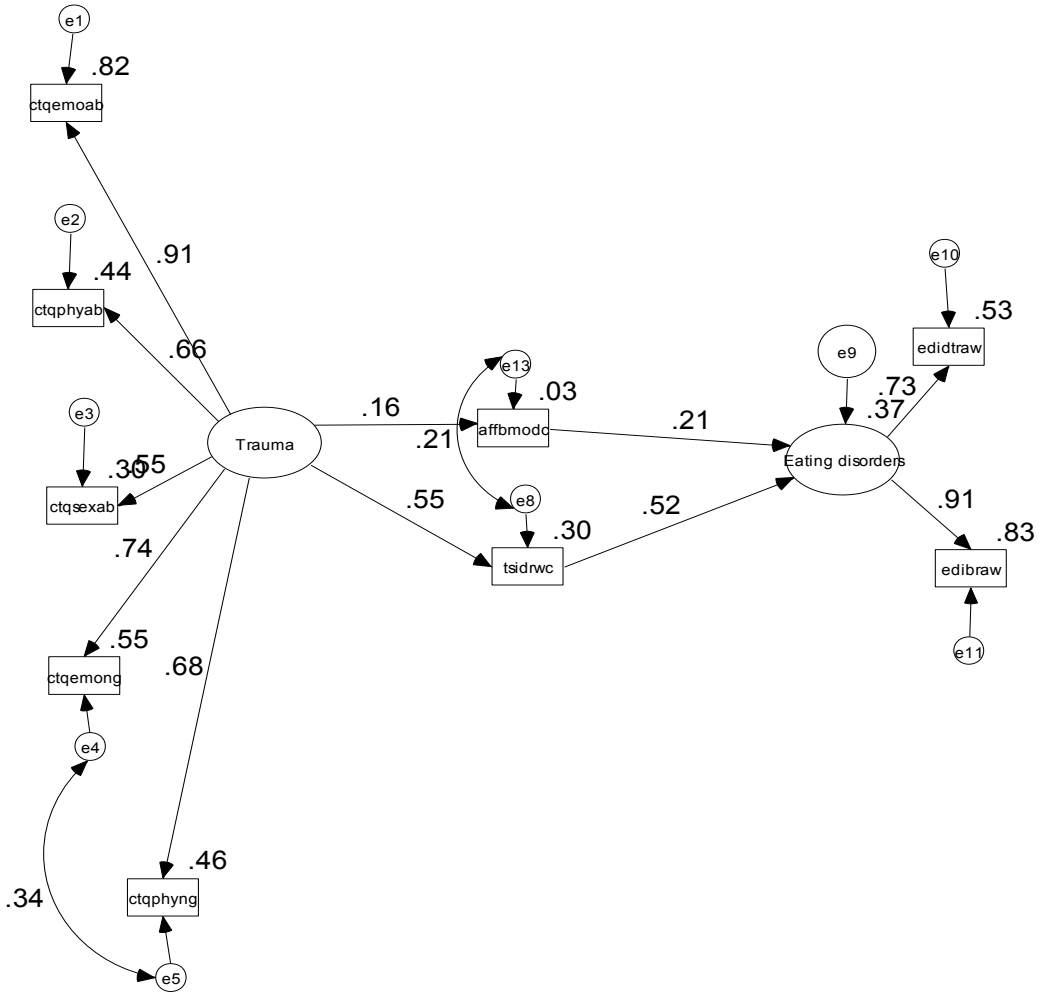
error terms are explicitly modeled. If measurement error is high, SEM path coefficients will be less reliable. For correlated error, knowing the residual of one indicator helps in knowing the residual associated with another indicator. Thus, to improve the model fit and reliability, the error terms between the two forms of neglect were allowed to correlate with each other. Likewise, a high degree of correlated error was found noted the error terms of the moderator, dissociation, and the error of the mediated moderator, maladaptive affect with the error of body dissatisfaction. To improve model fit, based on substantial overlap among these error terms, they were also allowed to correlate with each other.

Standardized regression weights for this model were as follows: dissociation with trauma: $\beta = .55$ ($p < .001$, medium-large effect size), maladaptive affect with body dissatisfaction with trauma: $\beta = .16$ ($p < .01$, small effect size), eating disorders with dissociation: $\beta = .52$ ($p < .001$, medium effect size), eating disorders with maladaptive affect with body dissatisfaction: $\beta = .21$ ($p < .001$, small-medium effect size), emotional abuse with trauma: $\beta = .91$ ($p < .001$, large effect size), physical abuse with trauma: $\beta = .66$ ($p < .001$, large effect size), sexual abuse with trauma: $\beta = .55$ ($p < .001$, medium-large effect size), emotional neglect with trauma: $\beta = .74$ ($p < .001$, large effect size), physical neglect with trauma: $\beta = .68$ ($p < .001$, large effect size), drive for thinness with eating disorders: $\beta = .89$ ($p < .001$, large effect size), bulimia with eating disorders: $\beta = .91$ ($p < .001$, large effect size). For this final model, overall model fit was excellent (e.g. Chi-Square=56.67, df=23, $p < .001$, Chi-square/df=2.46, GFI=.97, AGFI=.93, NFI=.95, RFI=.92, IFI=.97, CFI=.97, RMSEA=.07, SRMR=.06). Figure 4-25 depicts the final model where the relationship between childhood trauma and eating disorders is

moderated by dissociation and by the mediated moderator of maladaptive affect interacting with body dissatisfaction.

Figure 4-25

Final SEM Model with Regression Weights



Note: ctqemoab= Emotional Abuse; ctqphyab= Physical Abuse; ctqsexab= Sexual Abuse; ctqemong= Emotional Neglect; ctqphyng= Physical Neglect, edibdraw= EDI Body Dissatisfaction Raw Score; tsidrwc= TSI Dissociation Raw Score Centered; affbmodc=Maladaptive Affect x Body Dissatisfaction Moderator Centered; ediditraw= EDI Drive for Thinness Raw Score, edibraw= EDI Bulimia Raw Score

CHAPTER FIVE

DISCUSSION

The goal of this dissertation was to examine the relationship between childhood trauma and disordered eating. A comprehensive literature review revealed multiple factors, which appeared to influence how and why these two variables are connected, but empirical evidence was overwhelmingly contradictory and inconclusive. This led the researcher to conclude that the relationship between childhood trauma and disordered eating were not straightforward, but in fact, impacted by many moderating and mediating factors. Furthermore, despite the burgeoning research on the connection between childhood trauma and disordered eating, no prior studies have examined all of the identified variables in one comprehensive study. Given the extensive clinical implications of predicting risk factors and identifying disordered eating early, it is important to have an integrated understanding of these complex factors. The beginning of the discussion will explain the meaning of the results as they relate to each hypothesis. Next, limitations of the study and suggestions for future research will be discussed.

The final sample of 322 was comprised of women, the majority of whom were in their late teens through early twenties (mean=26.6, mode=18), Caucasian (68%), and had attended some college but had not earned a degree (56%). Of those 322 women, 146 (45%) self-reported that they experienced child abuse (before age 18). Seventy-six percent of those abused indicated that a family member had abused them before age 18. Ninety percent of the abused women reported experiencing some abuse before age 14. Seventy-five percent of the abused women reported that they experienced chronic child

abuse (i.e., they were abused on more than one occasion). In terms of types of abuse, the most prevalent type of abuse reported was emotional abuse (77% of the abused women). The second most prevalent type of abuse reported was sexual abuse (53% of the abused women). Sexual abuse was reported by 23.9% of the total sample, which is consistent with prior research reporting a rate of approximately 20% (Gorey & Leslie, 1997; Martin, et al., 1993) but is lower than Harned's (2000) report that 40% of undergraduate women were sexually assaulted in their lifetime. Arguably, however, Harned did not limit the reported sexual abuse to childhood, which could account for the difference. The third most prevalent form of abuse was physical abuse (45% of abused women). This means that about 20% of the total sample reported childhood physical abuse, which is much lower than Harned's sample, since she reported that 59% experienced physical abuse. Neglect was the least commonly reported form of abuse (25% of abused women).

These descriptive statistics revealed several interesting facts. First, it should be noted that the majority of the women who experienced abuse reported chronic, interfamilial abuse, which began before age 14. That is, for those who did experience abuse, a family member, someone who the youngster was supposed to rely on and trust, most often perpetrated it. Second, for the majority of abused women, the idea of one discrete traumatic incident is not applicable. This makes the concept of chronic trauma, and as a logical extension, Complex PTSD (Herman, 1992a; 1992b; van der Kolk & Fisler, 1994), even more relevant to current psychological research and clinical practice. These findings are consistent with the findings of the National Comorbidity Study (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), which also indicated that the

majority of those who experience trauma have experienced complex trauma. The term Complex PTSD was originally coined to capture the unique presenting problems (pervasive difficulties in: affective regulation, relational capacities, attention and consciousness, altered belief systems, and somatic distress) of those who experienced prolonged traumas with early onset (Resick et al., 2012) and the findings of this study support the inclusion of such a diagnosis in upcoming versions of DSM. The diagnosis of Complex Post Traumatic Stress Disorder (CPTSD) was considered in field trials for DSM-5, but did not end up being added due to the current lack of research supporting the diagnosis. Clinicians and researchers should keep in mind that when seeing an individual with a trauma history, the likelihood of Complex trauma and subsequently, Complex PTSD, is high. Furthermore, this finding highlights the importance of educating clinicians on psychological profiles and empirically supported treatments for those who have experienced multiple traumas.

Third, although no specific hypothesis related to the prevalence of abuse, it is important to note that emotional abuse was reported in such a high majority of the abused women. This is especially notable considering the lack of research on emotional abuse, especially compared to the quantity of research that has been conducted looking at sexual abuse (Tagay et al., 2010). Also, it is important to keep in mind that these rates of childhood abuse were self-reported, and thus were based on the respondents' own perception of whether or not she was abused. This appears especially relevant for neglect, which was the least commonly reported form of abuse. Since neglect tends to be more ambiguous and less discrete than other forms of abuse, participants may have underreported in this area due to confusion about what constitutes neglect.

Hypothesis 1

Hypothesis 1 stated that those who experienced at least one childhood trauma would experience significantly higher levels of behaviors, cognitions, and affect associated with eating disorders (as defined by the three main scales of the EDI-3): 1) Drive for Thinness, 2) Bulimia, and 3) Body Dissatisfaction. This hypothesis was first tested using Pearson product-moment correlations. As predicted, the women in this study who experienced childhood trauma also reported significantly higher symptoms when looking at the Drive for Thinness, Bulimia, and Body Dissatisfaction scales of the EDI-3. Effect sizes were largest for the relationship between bulimia and child abuse, but overall were small to medium. Furthermore, when looking at the specific scales of the EDI-3, higher scores on each of the five abuse scales were significantly correlated with all of the EDI-3 scales except for Maturity Fears.

Next, this hypothesis was investigated using multiple regressions employing the five different forms of abuse as predictors and the EDI-3 Eating Disorder scales as criterion variables. Multiple regressions were consistent with bivariate correlations. For all EDI-3 scales, with the exception of Maturity Fears, the overall model was significantly able to predict higher levels of difficulties. For the Drive for Thinness scale, both emotional abuse and sexual abuse emerged as significant predictors of symptoms. Specifically, 28.6% of the variance was uniquely attributable to emotional abuse and 14.3% of the variance was uniquely attributable to sexual abuse. For the Bulimia scale, both emotional abuse and sexual abuse again emerged as significant predictors. In terms of the Bulimia scale, emotional abuse uniquely accounted 25% of the shared variance,

while sexual abuse uniquely accounted for 20% of the variance. For the Body Dissatisfaction scale, like the other two main EDI-3 scales, emotional abuse and sexual abuse were the two types of trauma that emerged as significant. For Body Dissatisfaction, however, abuse seemed to account for a smaller percentage of the variance, with emotional abuse accounting for 8.8% and sexual abuse accounting for 2.9%, respectively.

Each subscale of the EDI-3 was also examined using multiple regressions. Emotional abuse, physical abuse, and sexual abuse all emerged as significant predictors of low self esteem, predicting 14.9%, 2.1%, and 6.4% of the variance, respectively. Emotional abuse and sexual abuse emerged as significant predictors of personal alienation, accounting for 11.3% and 5.7% of the variance, respectively. Emotional abuse, sexual abuse, and emotional neglect emerged as significant predictors of interpersonal insecurity, accounting for 4.4%, 4.4% and 6.7% of the variance, respectively. Emotional abuse, sexual abuse, and emotional neglect also emerged as significant predictors of interpersonal alienation, accounting for 7.3%, 7.3%, and 5.5% of the variance, respectively. Emotional abuse and sexual abuse emerged as significant predictors of interoceptive deficits, accounting for 5% and 2.5% of the variance, respectively. Emotional abuse and sexual abuse also emerged as significant predictors of emotional dysregulation, accounting for 8.7% and 10.5% of the variance, respectively. Emotional abuse emerged as a significant predictor of perfectionism, accounting for 11.5% percent of the variance. Emotional abuse and sexual abuse emerged as significant predictors of asceticism, each accounting for 7.1% of the variance. Consistent with the aforementioned analysis using bivariate correlations, maturity fears did not emerge as significant, and none of the specific forms of abuse emerged as predictors.

Although previous research linking childhood trauma and disordered eating has been contradictory and inconclusive (Allison et al., 2007), the results of this dissertation are consistent with many previous studies which have also provided support for a statistically significant relationship between the two variables (Abrahamson & Lucido, 1991; Bulik et al., 1989; Curtis et al., 2005; Folsom et al., 1993; Fonagy et al., 1996; Grave et al., 1996; Hall, et al., 1989; Johnson et al., 2002; Kaner et al., 1993; Lacey, 1990; Oppenheimer et al., 1985; O'Shaughnessy & Dallos, 2009; Palmer & Oppenheimer, 1992; Ringer & Crittenden, 2007; Root & Fallon, 1988; Sloan & Leichner, 1986; Tagey et al., 2010; Zachrisson & Kulbotten, 2006).

Overwhelmingly, emotional abuse and sexual abuse emerged as having the strongest connections to disordered eating symptoms. These two forms of abuse were consistently found to be predictors of maladaptive symptoms on the relevant EDI-3 scales. Again, considering the lack of research on emotional abuse compared to other forms of abuse, this finding has considerable implications for further research, which will be discussed later in this chapter. The connections between emotional abuse and sexual abuse also emphasize the importance of early identification by clinicians. Clinicians need to be aware of the risk factors that child abuse, especially in these forms, has for the later onset of disordered eating symptoms.

Another important outcome of these analyses was the consistent nonsignficance of the findings for the subscale of Maturity Fears. Both bivariate correlations and multiple regressions showed no significant relationship between childhood trauma and higher scores on this scale. One explanation for this finding could be that the abused women were eager to escape their childhoods, rather than fearing adulthood. People who

experienced abusive childhoods may in fact be less fearful of growing up than their non-abused counterparts, since they would have motivation to be independent from their perpetrators. Another possibility could be that the inclusion of the subscale could be theoretically linked to early theorists (e.g., Bruch, 1974; Minuchin et al., 1978; Orbach, 1986) whose ideas about eating disorders being tied to the rejection of a mature body and reproductive capacities and enmeshed family relationships have been criticized by the current community of eating disorder researchers (Eisler, Dare, Russell, Szmukler, le Grange, & Dodge, 1997; Fairbairn, Simic, & Eisler, 2011).

Hypothesis 2

Hypothesis 2 stated that the relationship between childhood trauma and eating disorders would be moderated by: A) the presence of PTSD symptoms, B) the presence of maladaptive affective symptoms, C) the presence of insecure attachment styles, D) the agent of abuse, E) the age of trauma onset, and F) the chronicity of the trauma. These moderators were classified into two groups based on their applicability to participants. Hypothesized moderators A-C were applicable to the full sample of 322 women, but hypothesized moderators D-F were only applicable to the 146 women who endorsed a history of childhood abuse. Analysis of H2: A-C will be discussed first. These proposed moderators were tested using two statistical methods. First, Pearson product-moment correlations were calculated using the bivariate correlations of a total trauma composite score and moderation variables reflecting the multiplicative product of total trauma and the hypothesized moderators and compared using Steiger's Z. Second, ANOVAs were used to evaluate group differences on the EDI-3 scales and to identify the presence of interaction effects. Each of the proposed moderators was tested separately with regard to

each of the three main EDI-3 scales (Drive for Thinness, Bulimia, and Body Dissatisfaction). As a reminder, this hypothesis was tested using a composite of CTQ scores, which was a sum of all forms of abuse. The correlation between the CTQ composite and all three of the EDI-3 scales were statistically significant ($p<.001$). The relationship of childhood trauma with Bulimia yielded the highest effect size (.41), followed by Body Dissatisfaction (.33), and Drive for Thinness (.32). Although all of these main effects were indeed significant, which is consistent with what was demonstrated in hypothesis 1 for each of the abuse subtypes individually, the findings of this study as described below indicate that the relationship is moderated by several variables.

PTSD. According to Steiger's Z tests, PTSD appears to moderate the child trauma-disordered eating relationship for Drive for Thinness and Bulimia, but not for Body Dissatisfaction. The relationship between child abuse and Drive for Thinness and Bulimia were both significantly moderated by PTSD symptoms ($p<.001$). Using an ANOVA to analyze the relationship, the same pattern emerged. Significant interaction effects emerged when looking at both Drive for Thinness and Bulimia, but PTSD did not act as a significant moderator for Body Dissatisfaction. Both of these statistical analyses provide clear support for PTSD as a significant moderator between childhood trauma and disordered eating when looking at symptoms directly related to the core features of anorexia and bulimia. In terms of body dissatisfaction, however, PTSD does not emerge as a significantly moderator.

Maladaptive Affect. Steiger's Z testing demonstrated that maladaptive affect significantly moderated the relationship between child abuse and disordered eating for all

three of the main EDI-3 scales ($p<.001$). When looking at an ANOVA, however, the same pattern emerged as did for PTSD. Only for Drive for Thinness and Bulimia did the interaction effects emerge as significant; Body Dissatisfaction was not associated with a significant interaction effect. Considering both analyses, the data again indicate that maladaptive affect serves as a strong moderator between child abuse and symptoms of anorexia and bulimia, but that body dissatisfaction seems to follow a different pattern.

Anxious Attachment. Steiger's Z testing demonstrated that anxious attachment significantly moderated the relationship between child abuse and disordered eating for all three of the main EDI-3 scales ($p<.001$). When using an ANOVA to look at anxious attachment, however, a different pattern emerged than did for the other proposed moderators. Only bulimic symptoms seemed to be significantly moderated by anxious attachment, but the other two main EDI-3 scales did not show moderation effects.

Avoidant Attachment. Steiger's Z testing demonstrated that avoidant attachment did not significantly moderate the relationship between child abuse and disordered eating for any three of the main EDI-3 scales. When using an ANOVA, the same results emerged. None of the relationships between the main EDI-3 scales and childhood trauma were significantly moderated by avoidant attachment. A recently published article by Tasca et al. (2013) suggests that insecure attachment styles (avoidant and anxious, equally) actually mediate, rather than moderate, the relationship between childhood trauma and disordered eating. Thus, the lack of support for both types of insecure attachment as moderators could be due to the fact that they are actually mediating the relationship.

To summarize the findings of H2: A-C, PTSD, maladaptive affect, and anxious attachment appear to serve as significant moderators between child abuse and disordered eating, when looking specifically symptoms associated with restriction, desire for thinness, binging, and purging. Considering these results highlights an important finding: body dissatisfaction does not appear to have the same relationship with related variables that symptoms of bulimia and drive for thinness have. Thus, although the authors of the EDI-3 include it as a main scale in terms of predicting disordered eating, it seems to have a different type of relationship with the variables.

Agent of Abuse. The second half of H2 (D-F) looks explicitly at characteristics of abuse and thus only relate to the portion of the sample that self-reported abuse. Interestingly, for the moderators that emerged as significant from D-F, the moderators emerged as having negative relationships. Steiger's Z tests indicated that the interaction of the agent of abuse with childhood trauma was significantly lower than the main effects for Drive for Thinness, Bulimia, and Body Dissatisfaction. When using an ANOVA to examine this relationship further, none of the moderators emerged as significant with any of the main EDI-3 scales.

Age of Abuse Onset. As reflected in the correlational findings, agent of abuse emerged as significant moderator in the relationship between child abuse and Bulimia, but not for Bulimia or Drive for Thinness. Also, just like with the agent of abuse, the correlation with the interaction variable was significantly lower than those with child abuse alone. Again, when using an ANOVA to analyze the relationship, age of abuse did not produce any significant main effects nor did its interaction with child abuse.

Chronicity of Abuse. Again, in the correlational analyses, chronicity of abuse served as a significant moderator in the relationship between child abuse and Bulimia, but not for the other two main EDI-3 scales. But, once again, correlation with the interaction variable was lower than that obtained with child abuse alone. As with agent of abuse and age of abuse onset, the interaction effect did not emerge as significant when tested using an ANOVA.

The difference in results between the Steiger's Z analyses of correlations and the ANOVA results can be best explained by two factors. First, due to the reduction in sample size for this part of the statistical analysis, power was significantly reduced. It is may be that a larger sample of abuse-reporting participants would yield significant results using ANOVAs. This likelihood is greatly supported by investigating the graphical representation of the ANOVAs. Even though the interactions do not emerge as significant, the graphs indicate that the low and high groups intersect, reflective of different slopes, which is indicative of an interaction. Thus, although with this small group of individuals, significance was not achieved, the data indicate that a larger sample could yield significant results. The second factor, which makes the use of ANOVAs difficult to apply to these data, is the skewed distribution of the participants into the low and high groups and also between the binary coded variables listed in D-F. Thus, in a larger sample with more evenly distribute groups, it is likely that the ANOVA results would suggest similar evidence to what Steiger's Z suggests: that significant interaction effects are present.

Hypothesis 3

Hypothesis 3 stated that the relationship between childhood trauma and eating disorder symptoms would be mediated by: A) the presence of dissociation, B) high levels of family dysfunction, and C) high levels of body dissatisfaction. This hypothesis was tested using SEM and the data indicated that the relationship between childhood trauma and eating disorder symptoms is in fact completely mediated by dissociation and body dissatisfaction. Family dysfunction, however, did not play a significant meditational role in the relationship. Thus, two of the three parts of this hypothesis were supported. The model including these two mediators demonstrated adequate fit.

The results of this hypothesis are crucial because the data demonstrate that what appears to be a significant relationship between childhood trauma and disordered eating can be completely explained by the two mediators of dissociation and body dissatisfaction. When controlling for those two variables, the relationship between childhood trauma and disordered eating is no longer significant. This finding is unlike any other published research to date, because no prior research has investigated the relationship in this manner.

Hypothesis 4

Hypothesis 4 originally stated that the relationship between the variables proposed to moderate the relationship between trauma and eating disorder symptoms (PTSD, maladaptive affect, insecure attachment styles, agent of abuse, age of abuse onset, and chronicity of trauma) will themselves be moderated by the mediators specified in hypothesis 3 (dissociation, family dysfunction, and body dissatisfaction). The results from hypotheses 2 and 3, however, suggested that several adjustments to the original data

analytic plan were necessary. First, as shown in hypothesis 2, insecure avoidant attachment did not emerge as significant. Therefore, only insecure anxious attachment was used in the revised model. Second, as demonstrated in hypothesis 3, family dysfunction did not emerge as a significant mediator in this relationship, but body dissatisfaction did. Therefore, to improve model fit, family dysfunction was removed. Third, after seeing how the ANOVAs were impacted by reduced sample size and the extremely uneven distribution of participants between groups in moderators D-F (agent, age, and chronicity of abuse), these factors were not able to be included in the final model.

After making these adjustments, the resulting directional model revealed an excellent fit with all regressions having medium to large effect sizes. The final model depicts the relationship between childhood trauma and disordered eating which is moderated by dissociation and also by the mediated moderator of maladaptive affect with body dissatisfaction. This final model suggests that the interaction of maladaptive affect with body dissatisfaction is significant in predicting disordered eating behaviors. That is, individuals who experienced abuse as children and display high levels of dissociation, and also have high levels of depression, anxiety, low self esteem, and body dissatisfaction are at highest risk of developing symptoms of eating disorders.

Significance of the Study

This study has extensive implications and produces groundbreaking data for contemplation. First and foremost, this appears to be the first study of its kind as it both attempts to simultaneously analyze a complex interaction of variables and is also the first attempt at using structural modeling to explain the relationship between childhood trauma

and disordered eating. Thus, although there is not much prior research to compare these results to, this dissertation serves as a starting point for future researchers. Although this study is by no means able to explain all of the complex interactions between variables, it is able to provide a foundation upon which other researchers and this researcher can continue to build.

In addition to the research significance of this study, it also has great implications for clinical practice. Clinicians overwhelmingly agree that the prognosis of eating disorders is best when identified early and even before the onset of symptoms meeting clinical criteria. The data gained from this study can help clinicians to have a better understanding of risk factors and thus identify women at a young age as being in need of extra support.

This study provides strong support for the correlation between childhood trauma and disordered eating. Consistent with previous research, trauma exposure emerged as a nonspecific risk factor for eating disorders (Mitchell et al., 2012). Thus, clinicians who are aware of a history of child abuse, particularly emotional abuse and/or sexual abuse, should be aware of the increased risk for disordered eating symptoms. Clinicians should be especially vigilant in cases where dissociative symptoms and evidence of body dissatisfaction are present. It is the hope of this researcher that these results will be able to be used in the treatment of young people who have been traumatized. When a clinician encounters a child or adolescent who presents with a trauma history, dissociative symptoms, affective distress, low self-esteem, and body dissatisfaction, the clinician should be able to identify immediately that youngster as being at risk for the later development of disordered eating.

Furthermore, this researcher hopes to develop a psychometric measure based on the outcome of this research to help other clinicians in identifying those who might be at the greatest risk of disordered eating symptomology. If these results can indeed be replicated in a clinical population, the implications for clinical practice are vast. Developing a psychometric assessment measure based on these risk factors would allow clinicians to identify which patients are most at risk for the onset of disordered eating symptoms and target these individuals with preventative psychoeducation prior to the onset of clinical levels of symptomology.

In addition to the practical significance of these findings, important implications for theoretical understanding also emerged. These results contribute to a greater understanding of complex trauma theory and can inform current theories regarding the etiology of disordered eating. In terms of biological theories of disordered eating, this study did not directly address any genetic links, hormonal differences, or neurotransmitter disturbances and thus does not provide insight either way into the validity of these theories (e.g. Racine et al., 2012; Bloss et al., 2011; Brewerton, 1995). Family influences, however, have long been identified as a contributing factor to the development and maintenance of pathological eating behaviors and many clinicians use a family treatment approach to treatment (e.g. Cachelin et al., 1997; Crittenden et al., 1991; Fairbairn, 2011). The findings of this study, however, did not show family dissatisfaction as a significant mediator in the relationship between trauma and disordered. One reason for this could be that family dissatisfaction is highly prevalent in those who have experienced childhood trauma, but that the family dissatisfaction does not explain the

relationship between trauma and disordered eating or increase the likelihood of symptoms.

Negative body image and negative emotions have also been implicated as risk factors and theories often include these variables as causal factors in the development of disordered eating symptoms (e.g. Stice et al., 1998; Briere & Conte, 1993; Briere & Runtz, 1988; Briere & Runtz, 1987). This research supports those theories. In terms of dissociation being theoretically linked to disordered eating (e.g. Brown et al., 1999; Farmer, 2008), this idea was also strongly supported by this project. Body dissatisfaction has also been long identified in theories regarding the etiology of eating disorders (Keel et al., 2001; Levine & Smolak, 1996; Stice, 2001; Striegel-Moore & Bulik, 2007). Overall, theories suggesting a relationship between childhood trauma and disordered eating were substantiated by this research, particularly with regards to emotional and sexual abuse. Furthermore, this research added to those theories by showing that this relationship was completely mediated by body dissatisfaction and dissociation.

Limitations of the Research

Despite the best intentions of the researcher, this study, as with all studies, included several limitations. First and foremost, as stated earlier, this study would have benefitted from a larger sample of abused women. In hindsight, it became clear that many of the analyses, which the investigator had hoped to run, did not have adequate power after the non-abused participants were removed. A related but distinct limitation was the fact that the groups of participants needed to test Hypothesis 2: D-F were enormously lopsided. This study would have benefitted from equal groups of participants endorsing each side of the respective dichotomies.

Another related limitation arises from the sampling method in that the participants were self-selected. Inherent to the self-selected sample are concerns about the representativeness of the sample given the likelihood of those with histories of abuse and/or disordered eating symptoms to be more motivated to participate than those without. The survey was advertised as increasing knowledge about mental health (see Appendix I) but upon clicking the link, it was immediately disclosed in the informed consent (see Appendix A) that the purpose of the research was to learn more about these issues. Furthermore, the survey was advertised heavily in collegiate settings but also on eating disorder related forums and websites. Thus, these specific characteristics of the sample should be kept in mind when interpreting the results.

Additionally, keeping in mind the generalizability of the results, since this study involved a nonclinical sample, the findings should not be too broadly interpreted to clinical samples of eating disordered persons. Furthermore, this study is relevant only to women since the participants were all women and the etiological similarities between men and women in this area have yet to be determined.

A final area of limitation in this project relates to the difficulty in defining some of the latent constructs named as variables. As mentioned in the literature review, one of the reasons such a small amount of research has been conducted on emotional abuse relative to other forms of abuse has to do with the difficulty in operationally defining emotional abuse. For the purposes of this study, emotional abuse was examined with two methods. First, individuals who endorsed a history of abuse were asked simply to report from their subjective view if they experienced emotional abuse as a child. This type of questioning in consistent with theories such as de Groot and Rodin (1997) who argued

that a discrete failure is not necessary so long as the individual perceives attunement failures. The second method of measuring emotional abuse was to use the emotional abuse scale of the CTQ. This scale attempts to provide an objective measurement of emotional abuse based on empirically supported factors. Interestingly, however, both the more subjective reports and the objective reports yielded the same correlations with the variables of disordered eating, suggesting that the experience of trauma is indeed in the eye of the beholder (Root, 1992).

Dissociation and body dissatisfaction are also variables that can be defined using different nuances and the results of this study are limited to the operational definitions used by the TSI and the EDI, respectively. The TSI defines dissociation as "dissociative symptomatology, such as depersonalization, out-of-body experiences, and psychic numbing" (Briere, 1995). Those using a different definition of dissociation should keep that in mind when viewing these results. The EDI's construct of body dissatisfaction looks at the general idea of being dissatisfied with one's body. It is certainly possible that this research is limited in that it did not address the specific types of body dissatisfaction as these might be more predictive of disordered eating symptomology.

Considerations for Future Research

The limitations of this study can be used to fuel further research in this area. First and foremost, expanding on this study with a large-scale study that includes a minimum of 500 abused participants would greatly improve the power necessary to analyze all of the abuse related moderators. Further, researchers might benefit from using stratified sampling techniques to ensure a normal distribution between groups on those variables. In addition, replicating this study with a clinical sample is essential in terms of

confirming the generalizability of the model and expanding current understanding of etiology, so that treatment can also be improved.

Another key finding, which calls attention to the need for further research, is the incredibly high prevalence of self-reported emotional abuse. Given that emotional abuse was the most commonly reported form of childhood trauma in this study, and was continually seen to be a significant predictor of disordered eating symptoms and account for a unique portion of the variance, the current dearth of research in this area is unacceptable. Considerably more research needs to be conducted looking at the relationship between childhood emotional abuse and disordered eating symptomology. Although recent researchers such as Groleau et al. (2011) have begun to focus more attention, this area remains to be under researched despite having such a high degree of clinical relevance.

The findings of this study also have important implications for further editions of the EDI. Consistent with research by Clausen et al. (2011), the model fit of the EDI-3 should be considered minimally acceptable. Several of the items have poor psychometric properties, high correlations among error variances, and lopsided factor loadings. Thus, it appears that many of the EDI-3 items are too ambiguous and do not represent specific indicators of eating pathology. Further revisions of the EDI should take into account the findings of this study, combined with the findings of Clausen et al. (2011) in terms of improving the its validity.

Conclusions

As a whole, this study exhibited several radical discoveries. One of the greatest contributions of this research is the identification of a working model to explain the

complex relationship between childhood abuse and disordered eating. As shown in the literature review, previous research in this area has been chaotic at best, and has left clinicians with more questions than answers about the etiology of eating disturbance. This study aimed to take a first step at answering some of the questions elucidated by the confusing and contradictory research conducted to date. The final SEM model helps explain why some studies have found relationships between childhood abuse and disordered eating and some have not. The contradictions among the previous research findings are likely explained by the significant impact of mediators and moderators. This study shows that what appears to be a correlation between childhood abuse and disordered eating is actually an illusory correlation that is masked by the subtle mediators and moderators. Instead of studying this relationship as straightforward, it needs to be investigated as an incredibly complex, interconnected, multi-determined set of interwoven threads, that when associated in a precise way, create the perfect storm for disordered eating symptoms to occur.

The findings of this study are also so important because by identifying at-risk women, clinicians can attempt to contribute to lower rates of premature death. In addition to reducing unnecessary mortality, clinicians can also help young women to identify risk factors and to develop prevention techniques that will ultimately increase the quality of life for those who have been abused. Abused children are at significantly higher risk of developing eating pathology, but by learning more about this relationship, clinicians can help ensure that not all who are at-risk go on to develop pathology.

Appendix A

INFORMED CONSENT FORM RESEARCH PROJECT DESCRIPTION
Childhood Trauma and the Emergence of Disordered Eating Symptoms

Dear Prospective Participant,

Thank you for taking the time to consider participating in my research project. My name is Änna Pettway. I am a student in the Department of Psychology at the University of Detroit Mercy. I have asked you to agree to be a volunteer in some research I plan to conduct. Before I can accept your consent, I want to make known to you the following information pertaining to the project.

1. Explanation of the Purpose. I am conducting this research as part of my doctoral studies. I am gathering information in order to learn more about childhood trauma and how it relates to eating behaviors. I am seeking 250-500 adults (18 or older) to participate in this study. By agreeing to participate, you are not only helping me finish my doctorate, but also contributing to science in an important way. The goal of this study is to ultimately have a better understanding of how to detect and treat difficulties with eating.

2. Explanation of the Procedures. As a participant, you will be asked to answer questions pertaining to your own childhood, family or origin, eating habits, and body image. Some of these questions may be extremely sensitive in nature and may lead to recollection of upsetting memories and/or traumatic experiences. You will be asked to read the questions and select the answer that best applies to you.

3. Expected Risks. While completing these questions, you may experience discomfort and/or distress due to the sensitive nature of the questions. Most people will not experience this, but in previous studies, about 3% of participants indicated that they would not have chosen to participate due to their discomfort with responding to the questions (Walker, Newman, Koss, & Bernstein, 1997). If you feel uncomfortable at any time and do not feel as if you can continue, please discontinue the survey. You will not be penalized in any way. In addition, if you feel at any time during or after your participation that you would benefit from access to resources to help tolerate feelings that may arise from your involvement, please print out or copy down the resources offered below. If these resources are not adequate in meeting your need, please contact me at annapettway@gmail.com to discuss further access to mental health services.

4. Expected Benefits. There are no particular personal benefits other than participating in an important research project. However, as stated previously, your participation will benefit both the researcher in her attempt to graduate and the field of psychology as a whole in terms of better understanding the relationship between trauma and eating behaviors.

5. Confidentiality. At no time during this process will you be asked for your name or any other potentially identifying information. The researcher has no way of identifying individual participants. All research will be presented in terms of group results rather than

individual experiences. The confidentiality of the records will be maintained unless the law requires disclosure. None of the questions will ask about topics that require legal mandates to disclose. Confidentiality of records will be maintained by Änna C. Pettway, M.A., T.L.L.P. NOTE: In certain cases the FDA may inspect the records and/or the IRB may inspect the records.

6. Offer To Answer Questions. I hereby offer to answer any questions you might wish to ask concerning the procedures used in this research at this time. Furthermore, I may be reached at 248-735-5928 or by e-mail at annapettway@udmercy.edu. You may also contact the chairman of my dissertation, Dr. Douglas A. MacDonald, at by phone at (313) 578-0388 or by email at macdonda@udmercy.edu. If you have questions concerning your rights as a volunteer, you may contact Dr. Elizabeth M. Hill, Chair, UDM Institutional Review Board, 313.578.0405 or hillelm@udmercy.edu. The University of Detroit Mercy is located at 4001 W. McNichols Road, Detroit, MI, 48221.

7. Freedom To Withdraw Consent. If you consent to be a volunteer in this research project, you are nonetheless free to withdraw your consent and discontinue participation at any time without prejudice to you. This will include students participating in research projects within a course and no penalty to a course grade or class standing will precipitate from withdrawal as a subject. You should also understand that the investigator has the right to withdraw you from the research project at any time. For example, volunteers who fail to follow instructions may be removed from the data sample.

8. Compensation. No compensation will be offered for participation in this research project.

9. Availability of Compensation and Medical Treatment for Injury. You, the volunteer, understand that if you experience trauma, no form of compensation is available. However, a list of resources will be provided that provide reduced-cost mental health treatment. Treatment may also be provided at your own expense or at the expense of your health care insurer (i.e. Medicare, Medicaid, BC/B.S., etc.), which may or may not provide coverage. If you have questions, you should contact your insurer.

10. Significant New Findings. If any significant new findings are developed during the course of this research that might relate to your willingness to continue participation, such new findings will be provided you.

By clicking the "agree" button below, I understand that I am giving my consent to participate in this research project. In doing so, I am confirming that I am at least 18 years of age. I understand that I am free to discontinue my participation at any time without penalty.

1. I have read all of the statements above pertaining to the research project entitled and I understand them.

2. I have been given the opportunity to ask any questions I wish concerning this research project and any questions I have asked have been answered to my satisfaction.

3. I understand that I may print a copy of this document or request one via email.

4. I hereby consent to be a volunteer in this research project.

( ) AGREE

( ) DISAGREE

Future Data Use. Occasionally, the same or another researcher will request the permission to review or use previously gathered data from a completed research project for a different project. If confidentiality of the data is protected and if the UDM Institutional Review Board has approved the study, would you be willing to give your permission to the release of your data collected from your participation in the current study without prior notification?

( ) Yes, I give my permission for the future use of data obtained in this study contingent on the preceding conditions.

( ) No, I do not give my permission for the future use of data from this study.

Appendix B

Demographics Questionairre

1) **What is your current age?_____**

2) What is your sex?

( ) Male

( ) Female

3) What is your race? (Check all that apply).

[ ] Asian/Pacific Islander
[ ] Black/African-American
[ ] Caucasian
[ ] Hispanic
[ ] Native American/Alaska Native
[ ] Asian Indian
[ ] Other
[ ] Multiracial/Biracial

4) What is your highest level of education?

( ) 12th grade or less
( ) Graduated high school or equivalent
( ) Some college, no degree
( ) Associate degree
( ) Bachelor's degree
( ) Post-graduate degree

Appendix C

Childhood Trauma Questionnaire

Instructions. These questions ask about some of your experiences growing up as a child and a teenager. Although these questions are of a personal nature, please try to answer as honestly as you can. For each question, mark the response that best describes how you feel.

When I was growing up…	Never True	Rarely True	Sometimes True	Often True	Very Often True
1. I didn't have enough to eat.	.	.	.	.	.
2. I know that there was someone to take care of me and protect me.	.	.	.	.	.
3. People in my family called me things like "stupid," "lazy," and "ugly."	.	.	.	.	.
4. My parents were too drunk or high to take care of the family.	.	.	.	.	.
5. There was someone in my family who helped me feel that I was important or special.	.	.	.	.	.
6. I had to wear dirty clothes.	.	.	.	.	.
7. I felt loved.	.	.	.	.	.
8. I thought that my parents wished I had never been born.	.	.	.	.	.
9. I got hit so hard by someone in my family that I had to see a doctor or go to the hospital.	.	.	.	.	.
10. There was nothing I wanted to change about my family.	.	.	.	.	.
11. People in my family hit me so hard that it left me with bruises or marks.	.	.	.	.	.
12. I was punished with a belt, a board, a cord, or some other hard object.	.	.	.	.	.
13. People in my family looked out for each other.	.	.	.	.	.
14. People in my family said hurtful or insulting things to me.	.	.	.	.	.
15. I believe that I was physically abused.	.	.	.	.	.
16. I had the perfect childhood.	.	.	.	.	.
17. I got hit or beaten so badly that it was noticed by someone like a teacher, neighbor, or doctor.	.	.	.	.	.

When I was growing up…	Never True	Rarely True	Sometimes True	Often True	Very Often True
18. I felt that someone in my family hated me.					
19. People in my family felt close to each other.	.	.	.	.	.
20. Someone tried to touch me in a sexual way, or tried to make me touch them.	.	.	.	.	.
21. Someone threatened to hurt me or tell lies about me unless I did something sexual with them.	.	.	.	.	.
22. I had the best family in the world.	.	.	.	.	.
23. Someone tried to make me do sexual things or watch sexual things.	.	.	.	.	.
24. Someone molested me.	.	.	.	.	.
25. I believe that I was emotionally abused.	.	.	.	.	.
26. There was someone to take me to the doctor if I needed it.	.	.	.	.	.
27. I believe that I was sexually abused.	.	.	.	.	.
28. My family was a source of strength and support.	.	.	.	.	.

Additional Questions Regarding Abuse

1) I experienced some type of abuse or violation (including physical, sexual, or emotional abuse or neglect) when I was a child and/or adolescent.*

( ) TRUE

( ) FALSE

If TRUE:

2) I experienced abuse or violations (including physical, sexual, or emotional abuse or neglect) BEFORE the age of 14:

( ) TRUE

( ) FALSE

3) I experienced abuse or violations (including physical, sexual, or emotional abuse or neglect) AFTER the age of 14:

( ) TRUE

( ) FALSE

4) A person who abused me (including physical, sexual, or emotional abuse or neglect) when I was a child and/or teenager was a member of my family:

( ) TRUE

( ) FALSE

5) A person who abused me (including physical, sexual, or emotional abuse or neglect) when I was a child and/or teenager was not a member of my family:

( ) TRUE

( ) FALSE

6) The abuse I experienced…

( ) occurred only on one occasion.

( ) occurred repeatedly.

7) As a child (prior to age 18), I experienced…

[ ] physical abuse

[ ] sexual abuse

[ ] emotional abuse

[ ] neglect

Appendix D

Trauma Symptom Inventory

In the past 6 months, how often have you experienced?
(Rate from 0 to 3, with 0=Never to 3=Often)

1) Nightmares or bad dreams
2) Trying to forget about a bad time in your life
3) Irritability
4) Stopping yourself from thinking about the past
5) Getting angry about something that wasn't very important
6) Feeling empty inside
7) Sadness
8) Flashbacks (sudden memories or images of upsetting things)
9) Not being satisfied with your sex life
10) Feeling like you were outside of your body
11) Lower back pain
12) Sudden disturbing memories when you were not expecting them
13) Wanting to cry
14) Not feeling happy
15) Becoming angry for little or no reason
16) Feeling like you don't know who you really are
17) Feeling depressed
18) Having sex with someone you hardly knew
19) Thoughts or fantasies about hurting someone
20) Your mind going blank
21) Fainting
22) Periods of trembling or shaking
23) Pushing painful memories out of your mind
24) Not understanding why you did something
25) Threatening or attempting suicide
26) Feeling like you were watching yourself from far away
27) Feeling tense or “on edge”
28) Getting into trouble because of sex
29) Not feeling like your real self
30) Wishing you were dead
31) Worrying about things
32) Not being sure of what you want in life
33) Bad thoughts or feelings during sex
34) Being easily annoyed by other people
35) Starting arguments or picking fights to get your anger out
36) Having sex or being sexual to keep from feeling lonely or sad
37) Getting angry when you didn't want to
38) Not being able to feel your emotions
39) Confusion about your sexual feelings
40) Using drugs other than marijuana

41) Feeling jumpy
42) Absent-mindedness
43) Feeling paralyzed for minutes at a time
44) Needing other people to tell you what to do
45) Yelling or telling people off when you felt you shouldn't have
46) Flirting or "coming on" to someone to get attention
47) Sexual thoughts or feelings when you thought you shouldn't have them
48) Intentionally hurting yourself (for example, by scratching, cutting, or burning) even though you weren't trying to commit suicide
49) Aches and pains
50) Sexual fantasies about being dominated or overpowered
51) High anxiety
52) Problems in your sexual relations with another person
53) Wishing you had more money
54) Nervousness
55) Getting confused about what you thought or believed
56) Feeling tired
57) Feeling mad or angry inside
58) Getting into trouble because of your drinking
59) Staying away from certain people or places because they reminded you of something
60) One side of your body going numb
61) Wishing you could stop thinking about sex
62) Suddenly remembering something upsetting from your past
63) Wanting to hit someone or something
64) Feeling hopeless
65) Hearing someone talk to you who wasn't really there
66) Suddenly being reminded of something bad
67) Trying to block out certain memories
68) Sexual problems
69) Using sex to feel powerful or important
70) Violent dreams
71) Acting "sexy" even though you didn't really want sex
72) Just for a moment, seeing or hearing something upsetting that happened earlier in your life
73) Using sex to get love or attention
74) Frightening or upsetting thoughts popping into your mind
75) Getting your own feelings mixed up with someone else's
76) Wanting to have sex with someone you knew was bad for you
77) Feeling ashamed about your sexual feelings or behavior
78) Trying to keep from being alone
79) Losing your sense of taste
80) Your feelings or thoughts changing when you were with other people
81) Having sex that had to be kept a secret from other people
82) Worrying that someone is trying to steal your ideas

83) Not letting yourself feel bad about the past
84) Feeling like things weren't real
85) Feeling like you were in a dream
86) Not eating or sleeping for 2 or more days
87) Trying not to have any feelings about something that once hurt you
88) Daydreaming
89) Trying not to think or talk about things in your life that were painful
90) Feeling like life wasn't worth living
91) Being startled or frightened by sudden noises
92) Seeing people from the spirit world
93) Trouble controlling your temper
94) Being easily influenced by others
95) Wishing you didn't have any sexual feelings
96) Wanting to set fire to a public building
97) Feeling afraid you might die or be injured
98) Feeling so depressed that you avoided people
99) Thinking that someone was reading your mind
100) Feeling worthless

Appendix E

Eating Disorder Inventory- 3 (EDI-3)

The items below ask about your attitudes, feelings, and behaviors. Some of the items relate to food or eating; other items ask about your feelings about yourself. For each item, decide if the item is true about you ALWAYS (A), USUALLY (U), OFTEN (O), SOMETIMES (S), RARELY (R), or NEVER (N). Respond to all of the items, making sure that you mark the letter for the rating that is true about you.

1. I eat sweets and carbohydrates without feeling nervous.
2. I think that my stomach is too big.
3. I wish that I could return to the security of childhood.
4. I eat when I am upset.
5. I stuff myself with food.
6. I wish that I could be younger.
7. I think about dieting.
8. I get frightened when my feelings are too strong.
9. I think that my thighs are too large.
10. I feel ineffective as a person.
11. I feel extremely guilty after overeating.
12. I think that my stomach is just the right size.
13. Only outstanding performance is good enough in my family.
14. The happiest time in life is when you are a child.
15. I am open about my feelings.
16. I am terrified of gaining weight.
17. I trust others.
18. I feel alone in the world.
19. I feel satisfied with the shape of my body.
20. I feel generally in control of things in my life.
21. I get confused about what emotion I am feeling.
22. I would rather be an adult than a child.
23. I can communicate with others easily.
24. I wish I were someone else.
25. I exaggerate or magnify the importance of weight.
26. I can clearly identify what emotion I am feeling.
27. I feel inadequate.
28. I have gone on eating binges where I felt that I could not stop.
29. As a child, I tried very hard to avoid disappointing my parents and teachers.
30. I have close relationships.
31. I like the shape of my buttocks.
32. I am preoccupied with the desire to be thinner.
33. I don’t know what’s going on inside of me.
34. I have trouble expressing my emotions to others.
35. The demands of adulthood are too great.
36. I hate being less than best at things.
37. I feel secure about myself.

38. I think about bingeing (overeating).
39. I feel happy that I am not a child anymore.
40. I feel confused as to whether or not I am hungry.
41. I have a low opinion of myself.
42. I feel that I can achieve my standards.
43. My parents have expected excellence of me.
44. I worry that my feeling will get out of control.
45. I think my hips are too big.
46. I eat moderately in front of others and stuff myself when they're gone.
47. I feel bloated after eating a normal meal.
48. I feel that people are happiest when they are children.
49. If I gain a pound, I worry that I will keep gaining.
50. I feel that I am a worthwhile person.
51. When I am upset, I don't know if I am sad, frightened, or angry.
52. I feel that I must do things perfectly or not do them at all.
53. I have the thought of trying to vomit in order to lose weight.
54. I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close).
55. I think that my thighs are just the right size.
56. I feel empty inside (emotionally).
57. I can talk about personal thoughts or feelings.
58. The best years of your life are when you become an adult.
59. I think my buttocks are too large.
60. I have feelings I can't quite identify.
61. I eat or drink in secrecy.
62. I think that my hips are just the right size.
63. I have extremely high goals.
64. When I am upset, I worry that I will start eating.
65. People I really like end up disappointing me.
66. I am ashamed of my human weaknesses.
67. Other people would say that I am emotionally unstable.
68. I would like to be in total control of my bodily urges.
69. I feel relaxed in most group situations.
70. I say things impulsively that I regret having said.
71. I go out of my way to experience pleasure.
72. I have to careful of my tendency to abuse drugs.
73. I am outgoing with most people.
74. I feel trapped in relationships.
75. Self-denial makes me feel stronger spiritually.
76. People understand my real problems.
77. I can't get strange thoughts out of my head.
78. Eating for pleasure is a sign of moral weakness.
79. I am prone to outbursts of anger or rage.
80. I feel that people give me the credit I deserve.
81. I have to be careful of my tendency to abuse alcohol.

82. I believe that relaxing is simply a waste of time.
83. Others would say that I get irritated easily.
84. I feel like I am losing out everywhere.
85. I experience marked mood shifts.
86. I am embarrassed by my bodily urges.
87. I would rather spend time by myself that with others.
88. Suffering makes you a better person.
89. I know that people love me.
90. I feel like I must hurt myself or others.
91. I feel that I really know who I am.

Appendix F

Experiences in Close Relationships- Revised (ECR-R)

The statements below concern how you feel in emotionally intimate relationships. We are interested in how you *generally* experience relationships, not just in what is happening in a current relationship. Respond to each statement by clicking the circle that indicates how much you agree or disagree with the statement. Each item is rated on a 7-point scale where 1 = strongly disagree and 7 = strongly agree.

1. I'm afraid that I will lose my partner's love.
2. I often worry that my partner will not want to stay with me.
3. I often worry that my partner doesn't really love me.
4. I worry that romantic partners won't care about me as much as I care about them.
5. I often wish that my partner's feelings for me were as strong as my feelings for him or her.
6. I worry a lot about my relationships.
7. When my partner is out of sight, I worry that he or she might become interested in someone else.
8. When I show my feelings for romantic partners, I'm afraid they will not feel the same about me.
9. I rarely worry about my partner leaving me.
10. My romantic partner makes me doubt myself.
11. I do not often worry about being abandoned.
12. I find that my partner(s) don't want to get as close as I would like.
13. Sometimes romantic partners change their feelings about me for no apparent reason.
14. My desire to be very close sometimes scares people away.
15. I'm afraid that once a romantic partner gets to know me, he or she won't like who I really am.
16. It makes me mad that I don't get the affection and support I need from my partner.
17. I worry that I won't measure up to other people.
18. My partner only seems to notice me when I'm angry.
19. I prefer not to show a partner how I feel deep down.
20. I feel comfortable sharing my private thoughts and feelings with my partner.
21. I find it difficult to allow myself to depend on romantic partners.
22. I am very comfortable being close to romantic partners.
23. I don't feel comfortable opening up to romantic partners.
24. I prefer not to be too close to romantic partners.
25. I get uncomfortable when a romantic partner wants to be very close.
26. I find it relatively easy to get close to my partner.
27. It's not difficult for me to get close to my partner.
28. I usually discuss my problems and concerns with my partner.
29. It helps to turn to my romantic partner in times of need.
30. I tell my partner just about everything.
31. I talk things over with my partner.
32. I am nervous when partners get too close to me.
33. I feel comfortable depending on romantic partners.

34. I find it easy to depend on romantic partners.
35. It's easy for me to be affectionate with my partner.
36. My partner really understands me and my needs.

Appendix G

Index of Family Relations

This questionnaire is designed to measure the way you feel about your family as a whole. It is not a test, so there are no right or wrong answers. Answer each item as carefully and as accurately as you can by placing a number beside each one as follows.

1 = None of the time 2 = Very rarely 3 = A little of the time 4 = Some of the time 5 = A good part of the time 6 = Most of the time 7 = All of the time

1. _____ The members of my family really care about each other.
2. _____ I think my family is terrific.
3. _____ My family gets on my nerves
4. _____ I really enjoy my family.
5. _____ I can really depend on my family.
6. _____ I really do not care to be around my family.
7. _____ I wish I was not part of this family.
8. _____ I get along well with my family.
9. _____ Members of my family argue too much.
10. _____ There is no sense of closeness in my family.
11. _____ I feel like a stranger in my family.
12. _____ My family does not understand me.
13. _____ There is too much hatred in my family.
14. _____ Members of my family are really good to one another.
15. _____ My family is well respected by those who know us.
16. _____ There seems to be a lot of friction in my family.
17. _____ There is a lot of love in my family.
18. _____ Members of my family get along well together.
19. _____ Life in my family is generally unpleasant.
20. _____ My family is a great joy to me.
21. _____ I feel proud of my family.
22. _____ Other families seem to get along better than ours.
23. _____ My family is a real source of comfort to me.
24. _____ I feel left out of my family.
25. _____ My family is an unhappy one.

Appendix H

APPENDIX I

Participants Needed!

It's easy to waste 25 minutes online…why not take that same amount of time and contribute to research on mental illness?

Find out more at:

http://edu.surveygizmo.com/s3/1097601/CHILDHOOD-TRAUMA-AND-THE-EMERGENCE-OF-DISORDERED-EATING-SYMPTOMS

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ABSTRACT

CHILDHOOD TRAUMA AND THE EMERGENCE OF DISORDERED EATING SYMPTOMS: AN INVESTIGATION OF CONTRIBUTING VARIABLES

This dissertation aimed to elucidate the complex relationship between childhood trauma and disordered eating. An extensive literature review demonstrated that the previous research in this area was contradictory and chaotic and that no one study had looked at all of the proposed mediators and moderators together. Thus, in this dissertation, the moderators of: PTSD, maladaptive affect, insecure attachment, agent of abuse, age of trauma onset, and chronicity of abuse are investigated along with the mediators of: dissociation, family dysfunction, and body dissatisfaction. This study was limited to adult women and was conducted on a nonclinical population. The final sample consisted of 322 adult women; of those women, 146 endorsed a history of childhood trauma. Emotional abuse and sexual abuse were the most commonly reported forms of trauma. Participants completed an online survey that was comprised of the Eating Disorder Inventory (EDI-3), the Trauma Symptom Inventory (TSI-3), the Experiences in Close Relationships (ECR-R), the Inventory of Family Relations (IFR), and the Childhood Trauma Questionnaire (CTQ), along with several demographics questions. Participants endorsing abuse also answered questions about their abuse history in terms of chronicity (one incident v. repeated), age of abuse onset (before or after age 14), and agent of abuse (family or non-family member). Hypotheses were analyzed using bivariate correlations, multiple regressions, ANOVAs, and structural equation modeling. Childhood trauma did indeed emerge as a non-specific risk factor for disordered eating symptoms, but this relationship was completely mediated by body dissatisfaction and

dissociative symptoms. Thus, results indicated that disordered eating symptoms can be predicted by childhood trauma, and the most important factors in this relationship are dissociative symptoms, maladaptive affect, low self-esteem, and body dissatisfaction. These findings have substantial implications for clinicians in terms of identifying those at risk for onset of disordered eating symptoms and also in terms of understanding complex trauma.

By

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May 2013

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Major: Psychology (Clinical)

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AUTOBIOGRAPHICAL STATEMENT

Änna Charis (Cotton) Pettway became interested in psychology while still in high school. She graduated in 2004 from Forest Hills Northern High School (Grand Rapids, MI) where she was recognized for excellence in psychology. She began her undergraduate education at Geneva College in Beaver Falls, Pennsylvania and went on to graduate from Spring Arbor University (Spring Arbor, MI) in 2007 with a Bachelor of Arts degree, majoring in psychology. She then went on to work at Hope Network (Grand Rapids, MI) where she gained experience working with traumatic brain injuries in a residential neurobehavioral unit. In 2008, she began graduate studies at the University of Detroit Mercy (Detroit, MI). She received her Masters of Arts in Clinical Psychology in 2011 and subsequently received her educational limited license in the state of Michigan. During the course of her graduate training, she completed a one-year practicum at Oakland County Court Psychological Clinic (Pontiac, MI) where she conducted forensic evaluations for adjudicated youth. She then went on to complete two years of practicum training at Hawthorn Center (Northville, MI) where she conducted therapy and assessments in an inpatient state hospital for children and adolescents. In September 2013, she will begin her pre-doctoral internship at Hawthorn Center, where she will continue to work with children and families. Her clinical and research interests involve serious mental illness in children and adolescents, specifically concerning complex trauma and the long-term interactions with maladaptive symptomology. She plans to continue working as a clinical child psychologist.