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Trauma, emotion dysregulation, and posttraumatic symptoms in an inpatient adolescent  
population: Testing multiple mediators

A Thesis presented for the  
Master of Arts Degree  
Department of Psychology  
The University of Mississippi

Elise Marie Elligett

May 2020

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## ABSTRACT

Recent studies suggest that difficulties in emotion regulation (ER) or emotion dysregulation (ED) mediate the association between trauma exposure and posttraumatic stress symptoms (PTSS) in adults and adolescents. However, elucidating how the specific facets of emotion dysregulation may differentially mediate the development of PTSS following trauma in adolescents could benefit the formation of more targeted interventions for PTSS in traumatized youth. The current study examined whether facets of ED mediated the relationship between exposure to traumatic events and PTSS in an ethnically diverse sample of adolescents residing in an inpatient psychiatric facility in Mississippi. Due to prior scholarship finding gender differences in trauma exposure, difficulties in facets of ER, and the development of PTSS, the moderating effect of gender was also explored. Participants included 154 youth (77 females and males) aged 12-17 ( $M = 14.35$ ,  $SD = 1.44$ ), who reported their trauma history, current PTSS, and difficulties in ER. Results suggest that ED as a total construct mediated the relationship between trauma and PTSD-symptoms, however only the facet of difficulties accessing/engaging ER strategies significantly mediated the same relationship separately. Gender differences were absorbed in frequencies of trauma and types of trauma, PTSS, and ED, but gender did not moderate any of the relationships between the variables. These results highlight the potential of examining facets of ED and ED as a total construct in the development of PTSD symptoms in adolescents. However, future research should validate measures of ED in diverse, understudied, and at-risk populations and explore targeted interventions suited to these populations.

## DEDICATION

This thesis is dedicated to those I love and have lost while completing this thesis: my Nana, Annette; my Papa, Trevor; and my dear friend, Dr. Jenn Caldwell.

## ACKNOWLEDGMENTS

I would like to thank my advisor, Dr. John Young, for his mentorship and patience; Dr. Laura Johnson, for her support and encouragement; and Kristen Johnson for her willingness to talk through concepts and ideas, editing, and her friendship.

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## CHAPTER 1

### INTRODUCTION

#### **1.1 Posttraumatic Stress in Youth**

Epidemiological studies have found that more than half of children in the United States have been exposed to several traumatic events by the time they reach the age of 18 (Copeland, Angold, Shanahan, & Costello, 2014; Costello et al., 1996; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Finkelhor, Ormrod, & Turner, 2009; McLaughlin et al., 2013). The detrimental impact of trauma in childhood and adolescence on mental health outcomes throughout the lifespan has been well documented. Exposure to a single trauma puts youth at greater risk of mood, anxiety and substance use disorders, conduct problems, decreased academic performance, poor physical health outcomes, decreased quality of life, and post-traumatic stress disorder (PTSD; Copeland, Shanahan, Costello, & Angold, 2011; Costello, Angold, & Keeler, 1993; Fairbanks & Fairbanks, 2009; Gustafsson et al., 2009; Pynoos et al., 2009; Song, Singer, & Anglin, 1998; Weisberg et al., 2002). Symptoms of PTSD usually begin within 3 months of experiencing a trauma and fall into four symptom clusters: intrusion, avoidance/numbing, negative cognition and mood, and hyperarousal (see Table 1 for *DSM-V* diagnostic criteria and specific symptoms) (Fletcher, 2003). PTSD is also a potentially chronic condition that causes significant distress and can profoundly impact the social and academic functioning of children and adolescents due to the disruption caused by intrusive symptoms, impaired selective attention for non-trauma related stimuli, sleep disturbances, and an increased propensity for peer-to-peer

violence (Pynoos et al., 1987; Osofsky, 1993; Pine & Cohen, 2002; Holt, Finkelhor & Kantor, 2007; Song, Singer & Anglin, 1998; Gustafsson et al., 2009, Meiser-Stedman et al., 2007).

While exposure to traumatic events in childhood is widespread to the point of being normative, the development of PTSD is not. Prior to the 1980s, it was believed that stress reactions in youth were brief and transient (Masten et al., 1990), and the little research conducted on children following a trauma was either anecdotal (e.g. Terr, 1979; Galante & Foa, 1986) or descriptive (e.g. Frederick, 1985). These early studies also had methodological limitations such as measuring anxiety, mood, or conduct-related outcomes (rather than posttraumatic symptomatology) and no control groups. For example, McFarlane (1987) followed a large sample of Australian children longitudinally after devastating bushfires, but the initial outcome measure was only general ‘behavioral problems’ (although the study did include a control group). When specific posttraumatic symptoms were asked about at a later time point, they were restricted to only four symptoms (nightmares; talking excessively about the fire; playing games or creating pictures related to the fire; distress related to fire-related reminders). Additionally, many early studies relied heavily on parental or teacher reports of children’s distress post-trauma, but it has since been well documented that adults significantly under-report children’s posttraumatic symptoms and distress (Handford et al., 1986; Fletcher, 2003; Dalgleish et al., 2005). The dearth of systematic research of trauma-related symptoms in youth prior to the 1990s resulted in contradictory findings and discrepancies in prevalence rates, although agreement was achieved in concluding that, in some cases, children and adolescents *do* experience trauma-related stress reactions that are similar to those of adults diagnosed with PTSD (Fletcher, 1996; Salmon & Bryant, 2002). This has translated to contemporary taxonomy in that the current *DSM-*

✓ criteria for a diagnosis of PTSD are the same for all ages (despite the fact that the behavioral expression of these symptoms may deviate; see Table 1). The main divergence when assessing for PTSD in children and adolescents as opposed to adults is a consideration of language development when the trauma happened and the role of the family in processing and making sense of the memories (Salmon & Bryant, 2002).

The prevalence rates of PTSD in children and adolescents vary widely in the literature, evidenced by one systematic review of post-traumatic reactions in children and adolescents reporting rates ranging from 0-100% (Dalglish et al., 2005). A meta-analysis conducted by Fletcher and colleagues (1996) that included more than 2,500 child and adolescent participants who had been exposed to trauma found that an average of 36% met criteria for a PTSD diagnosis compared to approximately 25% of adults in similar circumstances (Breslau et al., 1991). Further, the rates of PTSD did not significantly differ across developmentally categorized age groups of children/adolescents (39% in 6 year olds and under; 33% in 7-12 year olds; 27% in 13-17 year olds), although the meta-analysis was limited by the paucity of research that included children younger than elementary school age. The methodological and measurement issues and evolving conceptualization of PTSD in children, the type of trauma experienced, the duration of exposure, and the time elapsed since traumatic exposure can impact the reported prevalence rates among different samples (Dalglish et al., 2005). Similarly, the effect of differing measurement tools and reporters (i.e. parent, child or teacher) is observed even when examining only one type of trauma. For example, prevalence rates of PTSD following childhood sexual abuse were reported as 0% by Livingston (1987) after using parental report but reported as 90% by Kiser and colleagues (1988) when using the children's self-report.

Thus, variability of methods and conceptualization have influenced the field's estimation of the prevalence of potentially traumatic experiences and subsequent sequelae (including PTSD). Some clarity for understanding base rates can be derived from epidemiological studies, which have found a similarly variable range of estimated population base rates for youth and adults. The National Comorbidity Study (Kessler et al., 1995), for example, estimated lifetime prevalence of PTSD and measured other variables associated with trauma (e.g., type of trauma and persistence of symptoms) in a large sample that was representative of the US population (n = 5,877). Although the study did not focus on children and adolescents, the participants in the study were aged from 15 years to 54 years old and therefore included some adolescents. A diagnosis of PTSD using the criteria of the then current *DSM-III-R* (Diagnostic and Statistical Manual, 3rd edition-Revised; American Psychiatric Association, 1987) was assigned following a semi-structured interview and self-report measures, and the results suggest an estimated lifetime prevalence rate of 7.8 percent. Information about the type of trauma/s experienced was collected by numbering a list of types of trauma and presenting it to the participant. The interviewer asked respondents if they had experienced the number beside the qualitative description in an attempt to minimize the under-reporting of traumas, particularly types of trauma that potentially carry stigma (e.g. sexual molestation). The NCS reported the most common types of trauma associated with PTSD for men were combat exposure and witnessing the injury or death of another person, whereas rape and sexual molestation were most common for women. Additionally, it was notable that over half of the sample reportedly experienced at least one trauma in their lifetimes (60.7% of men; 51.2% of women), and that more than half of those who experienced one trauma had experienced multiple traumas.

The NCS also collected information relating to the duration of PTSD symptoms and whether or not participants sought treatment for PTSD. Those who reported receiving treatment were found to have a mean of 36 months of symptoms before remission (when applicable), and participants who reported never seeking treatment had a mean of 64 months of symptoms prior to remission. In both the treatment and no-treatment groups, however, over one-third of the participants did not experience remission of symptoms at any point (even many years after experiencing trauma). These findings indicate that PTSD is often chronic regardless of intervention, and that symptoms may persist for many years even after seeking mental health treatment. Furthermore, the NCS indicated that symptoms persisting for 3-6 months is the strongest risk factor for PTSD taking a chronic course, and that after this duration the likelihood of remission reduced drastically. Due to the risk of chronicity, understanding the variables that increase the risk of developing PTSD following a trauma is critical for the early identification of symptoms in youth to minimize long term impairment.

In 2001, the NCS was replicated, and a parallel study of adolescents aged 13 through 17 years was also conducted (National Comorbidity Study Replication Adolescent Supplement; NCS-A). The NSC-A conducted structured diagnostic interviews with a representative sample of 10,123 adolescents in the United States, as well as 6,428 self-administered questionnaires from one parent or guardian of the adolescent sample (Kessler et al., 2009). The Composite International Diagnostic Interview (CIDI; Kessler & Üstun, 2004) assessed lifetime and past-year *DSM-IV* (Diagnostic and Statistical Manual, 4th edition; American Psychiatric Association, 1994) disorders, and explicitly included items asking about 19 different potentially traumatic events (PTEs) that qualify for the *DSM-IV* A1 criterion. When analysing the data of the

adolescent-parent dyads, McLaughlin and colleagues (2013) reported that 61.8% of adolescents had experienced at least one PTE, with 29.1% reporting a single PTE, 14.1% reporting 2 PTEs, and 18.6% reporting 3 or more PTEs. The lifetime prevalence of PTSD among those exposed to a PTE was 7.6%, and the lifetime prevalence of PTSD in the total sample was 4.7%.

Additionally, it was noted that there was a significant difference in lifetime prevalence of PTSD between females and males in the total sample (females = 7.3%; males = 2.2%).

The authors also examined a number of variables relating to PTEs reported by the adolescents in the sample including the most common PTEs, age that each type of PTE occurred, frequency of PTE type being reported as “worst experienced”, and PTEs with the highest probability of subsequent PTSD diagnosis (McLaughlin et al., 2013). The most common PTEs among adolescents sampled were the unexpected death of a loved one, natural disasters, and witnessing the death or serious injury of another person. The median ages of first exposure to the PTEs in the study were reflective of shifting risks associated with various developmental stages in childhood and adolescence. The PTEs with the lowest median age of exposure were kidnapping, physical abuse by a caregiver, and witnessing domestic violence, whereas PTEs with the highest median age at first exposure were stalking, mugging, experiencing an automobile accident, and being physically assaulted by a romantic partner. The PTEs that were most associated with PTSD onset were the same as those that had the greatest likelihood of being reported as the worst PTE experienced (i.e., rape, kidnapping, sexual assault without penetration, physical assault by a romantic partner, and physical abuse by a caregiver). Additionally, a history of multiple PTE exposures was predictive of elevated risk for PTSD onset. These findings corroborate previous literature indicating that interpersonal traumas, especially those with a

perpetrator known to the victim, as well as polytraumatization, are associated with increased probability of PTSD diagnosis.

The NCS-A (2013) also examined the differential risk of experiencing trauma and consequent PTSD based on sociodemographic factors (i.e., gender, age, race/ethnicity, US nativity, family structure and income, urban/rural location) and previous mental disorders in adolescents. Prior studies focusing on this age group had not used broadly representative samples or had sampled for a specific trauma type, yielding mixed results regarding the variation of PTE exposure and PTSD diagnosis on the basis of sociodemographic factors and psychiatric history (e.g. Storr, Ialongo, Anthony & Breslau, 2007; Breslau, Wilcox, Storr, Lucia & Anthony, 2004; Giaconia, Reinhertz, Silverman, Pakiz & Frost, 1995; Costello, Erkanli, Fairbank & Angold, 2002). Due to the large and representative sample, NCS-A addressed some inconsistencies founded in previous studies. The NCS-A indicated females were significantly more likely to have experienced physical assault from a romantic partner, stalking, rape/sexual assault, the unexpected death of a loved one, and witnessing or having knowledge of a PTE occurring to a loved one. Males had higher odds of experiencing an accident, physical assault, and witnessing death or serious injury. Race was associated with PTE exposure but not a diagnosis of PTSD, with white non-Hispanic adolescents having a significantly higher incidence of witnessing domestic violence than other races, African-American adolescents having a greater likelihood of experiencing the unexpected death of a loved one, and Hispanic adolescents reporting significantly more exposure to physical assault by a romantic partner. Physical abuse by a caregiver and witnessing trauma to a loved one were also found to be more prevalent among adolescents in urban areas, but urban adolescents had lower odds of automobile accidents

compared to those in rural areas (ostensibly due to lower frequency of use). Pre-existing behavioral disorders also conferred a higher probability of experiencing all types of interpersonal PTEs. Those with pre-existing mental disorders categorized as fear or emotional distress (e.g., unipolar depression or anxiety disorders) were more vulnerable to experiencing half of the 19 probed PTEs (especially PTEs involving knowledge of a PTE happening to someone in their network and witnessing PTEs). Alternatively, Bipolar Disorder was only differentially associated with risk of experiencing rape, sexual assault, and kidnapping. Adolescents living with fewer than two biological parents also had a greater likelihood of experiencing all types of interpersonal violence and witnessing events.

Further analyses of sociodemographic risk factors demonstrated some factors initially significantly associated with exposure to PTEs and PTSD diagnosis became non-significant when controlling for other factors (McLaughlin et al., 2013). At first, results indicated the developmental period of early to late adolescence had the largest risk of experiencing a traumatic event, but this association dissipated when controlling for this group's higher rate of prior mental disorders (which, as outlined above, predicted greater risk of traumatic exposure). Additionally, older age was also associated with diagnosis of PTSD, but this relationship was no longer significant when accounting for prior diagnoses of mental disorders and the type of PTE classified as 'worst experienced' by individuals. Specifically, all mental disorder diagnoses were strongly predictive of developing PTSD following a PTE. Also, the PTEs (i.e., all types of sexual assault and physical assault by romantic partner) with the highest probability of preceding posttraumatic symptomatology were more likely to be experienced at an older median age (with the exception of kidnapping and physical abuse by caregiver), which offered some explanation



for the link between older age and a diagnosis of PTSD. Although living with fewer than two biological parents was associated with increased risk of PTEs and PTSD onset, the relationship between this sociodemographic variable and PTSD was discerned to be due to the differential vulnerability of children and adolescents exposed to multiple PTEs, rather than who they lived with. Finally, McLaughlin and colleagues (2013) emphasized other noted risk factors (i.e., type of PTE and number of prior PTEs experienced, etc.) did not account for female gender being predictive of exposure to PTEs and a diagnosis of PTSD, indicating that gender is an independent vulnerability factor. This finding is notable as previous studies that have conflated the higher incidence of interpersonal traumas among females (also found in the NCS-A) with females being more likely to receive a PTSD diagnosis. Thus, the relationship between gender, traumatic experience, and subsequent experience of symptoms is potentially more complicated than the majority of previous literature suggested.

Importantly, the NCS-A confirmed the previous findings regarding the chronic course of adult PTSD in adolescents. Thirty-three percent of adolescents who met criteria for PTSD during their lifetime also met *DSM-IV* criteria for PTSD in the 30 days preceding their interview. Moreover, of the adolescents no longer fulfilling criteria for a diagnosis of PTSD, the mean recovery time was 14.8 months. The study also reported on factors influencing the odds of recovery from PTSD for adolescents. Interestingly, adolescents born outside of the US were 11 times more likely than their native-born counterparts to no longer meet criteria for PTSD, making this variable the only significant resilience factor for adolescents with PTSD. On the other hand, high poverty and pre-existing mental disorders before the reported worst PTE were predictive of lower rates of recovery. Recovery rates were unaffected by multiple PTEs prior to

the trauma that triggered PTSD symptoms. Experiencing trauma after the onset of PTSD, however, did significantly reduce the recovery rate for adolescents with a history of other mental disorders. Although differentially predictive of initial etiology, the type of PTEs experienced were not predictive of recovery time once an adolescent met criteria for PTSD. For instance, although interpersonal traumas such as sexual assault increased the probability of an adolescent developing PTSD, there was no difference in recovery time regardless of whether the instigating trauma was sexual assault or a natural disaster once an adolescent had a PTSD diagnosis. Consequently, the NCS-A corroborated the findings of the NCS regarding the chronicity of PTSD, expanding the conclusions regarding adults with the disorder to youth who meet criteria for diagnosis. This reinforced the importance of recognizing factors that put some children and adolescents at a heightened risk of developing PTSD to facilitate prompt identification and treatment for the condition.

Another seminal study that investigated the rates of PTSD in youth was the Great Smoky Mountains Study (GSMS). The GSMS distinguished itself from other epidemiological studies due to its focus on an understudied, predominantly rural and impoverished population in the Southeastern region of the United States. It was a multistage population-based longitudinal study of youth that used overlapping cohort groups of 9-, 11-, and 13-year-olds from 11 counties in the southern Appalachian region of North Carolina (Costello et al., 1996). The geographic area that the sample of children were drawn from is sparsely populated, with approximately half of the population living in the only town that is classified as urban. Almost all children in the sample attended public schools, and the area was considered representative of the rural Southeastern region of the United States. The study ran from 1992 to 2003 (Copeland, Angold, Shanahan &

Costello, 2014), with an initial focus on the relation between the development of child and adolescent psychopathology and the use of mental health services. Costello and colleagues selected their sample by collecting parent-report psychological screeners pertaining to externalizing symptoms and substance use from 4,500 of the almost 12,000 children in the counties that met their age criteria. All children that fell into the highest quartile of scores on the screener were given a structured diagnostic interview (Child and Adolescent Psychiatric Assessment; Angold et al., 1995), and 10 percent of the remaining children screened were selected at random to do the same.

The GSMS had a final sample size of 1,015 children and adolescents for the first wave of data collection and interview. The sample was predominantly White (90%) and 34.5% were from a household with earnings under the federal poverty line (Costello et al., 1996; Copeland et al., 2014). The GSMS measured 3-month prevalence rates (i.e., symptoms, distress, and impairment reported from the period of the previous three months prior to interview) of disorders using DSM-III-R taxonomy (American Psychiatric Association, 1987). The 3-month prevalence of a DSM-III-R diagnosis of any type was 20.3%; however, there were less than 5 individuals in total given the diagnosis of PTSD at the first wave of interviewing, a reported 0.02% total weighted estimated prevalence (females = 0.05%; males = 0%). The prevalence of PTSD across six annual interviews was similarly low, (i.e., <0.1 percent; Costello et al., 2003). The GSMS found no racial differences in frequency of overall diagnoses when controlling for household income, with the exception of African American children from lower income families being at a higher risk for functional enuresis. Irrespective of ethnicity, however, children from the poorest families had an increased risk of all diagnoses and were also three times more likely to have comorbid disorders.

One possible reason for the low prevalence rates of PTSD found by the GSMS could be that the parent-report screeners completed prior to selection into the study only asked parents about externalizing symptoms and substance use (i.e., observable symptoms). This method could have over-selected for certain disorders (e.g., ADHD, ODD etc.), although the sample prevalence rates for anxiety disorders and mood disorders were similar to other epidemiological studies. Thus, it seems somewhat unlikely that the selection criteria would have only been biased with regard to under-sampling for PTSD. Another factor potentially contributing to the low prevalence rate in the GSMS is the aforementioned scarcity of research relating to PTSD in children when the study began, influencing the conceptualization of the disorder at the time and measurement tools available.

The few epidemiological studies investigating PTSD in children and adolescents elucidate that while exposure to trauma is necessary for the diagnosis of PTSD, it is not sufficient or deterministic of a diagnosis. Additional factors investigated that have been posited to influence the likelihood of PTSD include polytraumatization, poor family functioning, low social support, type of trauma, and gender. Initial research into PTSD in youth populations focused on children who have been through a single traumatic experience but more attention has recently been given to ‘polyvictimization’ or ‘polytraumatization,’ which refers to exposure to two or more traumatic events. Exposure to one trauma is associated with an increased risk of subsequent traumas, and the severity of the resulting psychological outcomes is compounded (Finkelhor, Ormrod & Turner, 2009). Family functioning variables such as parental anxiety sensitivity, maternal avoidance of trauma-reminders, heightened reactivity of a parent to trauma-reminders, family conflict, and maternal overprotection have also been implicated in increased

trauma-related distress and PTSD diagnosis in youth (Pynoos et al., 1999; McFarlane, 1987; Meiser-Stedman et al., 2005). There have been discrepancies as to whether social support factors influence the development of posttraumatic symptoms (Pine & Cohen, 2002), although factors such as peer group inconsistency, delinquency, disruption, and displacement from an individual's peer group or community as a consequence of a traumatic event may also increase the quantity of symptoms experienced, distress, and impairment (Fergusson & Lynskey, 1997; Laor, Wolmer & Cohen, 2001). This appears to impact both acute and long-term mental health outcomes for children and adolescents with PTSD.

In regards to trauma type, interpersonal trauma has been associated with significantly higher rates of PTSS and PTSD diagnosis as opposed to non-interpersonal traumas (Copeland et al., 2007). Studies have also shown that gender is a significant factor with girls being at a higher risk for PTSD than boys; however, whether this difference is due to females being at higher risk of interpersonal trauma (especially sexual assault) than males has received inconsistent support in the literature, as females appear to still be at an increased risk of PTSD when trauma-type is controlled for (Nooner et al., 2012; Tolin & Foa, 2006; McLaughlin et al., 2013). It has also been suggested that the gender difference found in the development of PTSD is associated with females more being more likely to engage in specific maladaptive cognitions and emotion regulation strategies such as self-blame attribution (Trickey et al., 2012) and rumination (Ehlers & Clark, 2000). Potential gender and individual differences in proclivity for certain emotion regulation strategies present an opportunity for further research into the influence of emotion regulation and dysregulation in the etiology and maintenance of PTSD in youth, especially as it

could be a factor more amenable to modification than sociodemographic variables and previous life experiences.

## **1.2 Emotion Regulation**

Emotion regulation (ER) is a broad term that refers to the deliberate and automatic processes that monitor, evaluate, and modify emotional experiences, including what emotional experiences an individual has, when they have them, and how they experience and express them (Gross, 1999; Zeman et al., 2006). Early research into ER equated ‘regulation’ with the ability to ‘control’ emotional experience, especially the expression of negative emotions (e.g., anger, sadness, anxiety, shame), and a reduction in arousal when experiencing negative emotions (Cortez & Bugental, 1994). More recent conceptualizations, however, have shifted to focusing on the functionality of human emotion and an individual’s ability to regulate their cognitions, behaviors, and the duration of negative emotional states in order to reduce distress, impairment, and maladaptive patterns (Thompson, 1994; Gratz & Roemer, 2004). This shift takes into account the growing body of literature demonstrating an association between emotion regulation deficits (e.g., ability to experience a full range of emotional states; inability to respond spontaneously and appropriately) and mood, anxiety, and conduct disorders across the lifespan (Cole et al., 1994; Greenberg & Paivio, 1998; Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema & Schweizer, 2010; Kring & Sloan, 2010; Campbell-Sills, Ellard & Barlow, 2014; Joormann & Siemer, 2014; Kober, 2014). The necessity of an integrated model of ER has become clear as it has become recognized as a transdiagnostic factor associated with a range of psychopathologies (Neacsiu, Bohus & Linehan, 2014).

The Process Model of Emotion Regulation (Gross, 1998; 2002) attempts to organize the immeasurable number of emotion regulation processes and strategies that can be implemented by individuals across contexts by conceptualizing ER strategies as differing on the basis of *when* they have their core impact on the emotion-generative process. The Process Model is based on the assumption that emotions arise when something important to the individual is at stake. Emotions can be automatic (e.g., recoiling from a spider) or can arise after a deliberate analysis of a situation. In either case, the experience of emotions begins with behavioral, experiential, and physiological reactions that combine to form an individual's overall 'response' to a situation (LeDoux, 1995). Emotions vary on the basis of latency, rise time, magnitude, duration, and offset responses as a function of their behavioral, experiential, and physiological components. While psychological research has historically had a tendency to focus on distress and impairment caused by negative emotional states, both negative and positive emotions are subject to the same regulation processes to some extent (Gross, 2002). For example, a sombre occasion such as a funeral could result in an individual suppressing the expression and experience of happiness after finding out they have been offered a long sought-after job. ER strategies can also be conscious (e.g., leaving the room when a distressing news report begins on the television) or without conscious awareness that a strategy is being used (e.g., ruminating about a problem; Boden & Baumeister, 1997). Finally, the Process Model posits that ER strategies can be 'adaptive' or 'maladaptive', which is heavily dependent on the context in which they are employed, and either antecedent- or response-focused.

In the Process Model of ER, Gross (1998; 2002) describes five stages of emotion generation that can potentially be modified using ER strategies by an individual. The first four

are antecedent-focused, meaning that ER strategies are used before behavioral, experiential, and physiological emotion response tendencies are activated. The first stage is *situation-selection*; i.e., approaching or avoiding people, places, or things to reduce or increase the possibility of a certain emotion-state being activated. Situation-selection can be adaptive (e.g., taking one's dog for a walk after a stressful day to change a negative emotion state to a more positive one; deciding not to answer a phone call from an ex-partner immediately following a break up) or maladaptive (e.g., not accepting an invitation to a party after moving to a new town in order to avoid feeling nervous or potentially embarrassing oneself). The second stage is *situation modification*, which involves the individual altering one's physical environment to change/increase/decrease/maintain an emotion. For example, an adaptive use of situation modification may be to keep distance between oneself and an ex-partner after a recent break up when at a party in order to continue enjoying time with friends and limiting the chance of painful emotions arising. Alternatively, approaching one's ex-partner in this same situation for the purposes of confrontation (and likely concomitant emotional activation) could be viewed as a maladaptive strategy. Thirdly, Gross contends that situations usually have a variety of details that could be differentially salient to an individual in context, *attentional deployment* takes place to facilitate focus on one or multiple details of the situation, which may vary greatly depending upon individual and the specific context. For example, an adaptive use of attentional deployment might be to continue expending effort to concentrate on reading study notes despite a distracting level of ambient noise on a train in order to reduce the possibility of feeling anxious when taking an exam. A maladaptive use of attentional deployment could be distracting oneself with a cell phone while a friend discusses a distressing situation as a way to deliberately reduce the probability of feeling distress. The last antecedent-focused stage of the process model of ER is



*cognitive change*, also known in the literature as cognitive reappraisal. This refers to the meaning that individuals select to apply to a situation. For example, an adaptive use of this could be telling oneself that a friend is probably focused on something else, which explains why he/she did not wave back after a chance encounter. Whereas a maladaptive use of cognitive reappraisal might be to look at the same situation and decide one's friend must be purposely ignoring the interaction, which would increase the likelihood of behavioral, experiential and physiological response tendencies for sadness, confusion, or anger.

The final stage of the process model is response-focused emotion regulation, referred to by Gross (2002) as *response modulation*. Response modulations are the attempts to modify emotion response tendencies once they have been activated. There are many strategies that can be used to try and influence one's emotion-driven responses; however, the most widely studied are 1) the suppression of expressive behavior associated with the experienced emotion and 2) methods of altering the physiological and experiential states associated with an emotion. For example, an individual may suppress the facial expression and behavioral tendencies he/she usually has when angry if that anger has been elicited by their boss in the workplace (likely an adaptive strategy). On the maladaptive side, in order to reduce the unwanted physiological tendencies that come with anxiety at a party when meeting new people, an individual might drink alcohol to reduce his/her heart rate or muscle tension. Given enough alcohol, it is possible that emotions could be transmuted from anxiety to some form of happiness or enjoyment; however, this strategy is physically unhealthy and could result in deferred experience of negative emotions (e.g., embarrassment or shame due to uninhibited behavior, which in turn could increase the probability of anxiety occurring again in similar future situations).

Gross' process model of ER was among the first comprehensive attempts to encapsulate the scope of ER, including many strategies that require complex trade-offs between short-term and long-term consequences associated with regulating emotion states. Additionally, the model incorporates strong attention to context, and thus to contextual conceptualizations of what represents adaptive vs. maladaptive strategies. The model does not, however, explicitly discuss the functional nature of emotion, especially negative or unpleasant affective states. Further, the model does not adequately account for the importance of an individual's awareness and understanding of the emotion being experienced or the functional role of emotion as a necessary foundation to adaptive ER (Thompson & Calkins, 1996; Hayes, Wilson, Follette, & Strosahl, 1996).

The importance of clarity to differentiate between emotions and the awareness of one's own emotional state has been emphasized subsequent to researchers shifting from a perspective of emotional control to one of modulation and functionality (Linehan, 1993; Gross & Munoz, 1995). An accurate assessment of one's own emotional state is fundamental for emotional experiences to be modified in flexible and adaptive ways across contexts (Thompson & Calkins, 1996). For example, the inability to discriminate between anger and other negatively laden emotions such as sadness and embarrassment has been associated with an increased risk of perpetrating violence on intimate partners (Jakupcak, Liser, & Roemer, 2002). Parents/caregivers shape the early development of children's representations of emotion by facilitating an understanding of the causes and consequences of their feelings, the functions of emotion and emotional behavior, and the social expectations around appropriate expression of emotion (Gottman, Katz, & Hooven, 1997; Thompson, 2006). The earliest conversations that caregivers

have with their children about emotion often revolve around labelling the child's emotion for him/her and providing external support for regulation (e.g., a baby or toddler is comforted by the parent with physical contact and the mother applies the label of 'sad' verbally to the child during the interaction). Thus, in some sense, this advances the caregiver's model for emotional regulation and facilitates the development of similar strategies in the child (Thompson, 2014).

Parents who are alert to their child's emotion-driven behaviors and expression can also assist the child in recognizing the physiological correlates of various emotions, the causal events (internal or external) that triggered the emotion, and potential emotion regulation strategies (Eisenberg, Cumberland, & Spinrad, 1998). For instance, a mother of a behaviorally-inhibited/shy 4-year-old child may be attentive to the child standing by silently while watching other children play from a distance. This observation gives the mother the opportunity to talk to the child, label the emotions associated with the child's reticence, and support him/her by suggesting strategies that could help the child move towards his/her goal in the situation. It is imperative in this situation, however, that the parent has awareness and clarity of his/her own emotions and exhibits understanding and acceptance of the child's emotions (Gottman et al., 1997). Gottman and colleagues (1997) differentiate between "emotion coaching" and "emotion dismissing" parenting whereby the coaching parents are attentive to their own emotions and those of their child, and see a children's emotional expressions as a chance to validate their feelings and teach them about emotions and coping. In contrast, dismissing parents have a tendency to be inattentive or suppress their own emotions and belittle emotional expression, and likely see their role as subduing negative outbursts in their children. This style of parenting is associated with children having difficulties discerning between negative emotional states,

thereby impairing the child's ability to learn adaptive ways of regulating various emotional challenges (Hooven, Gottman, & Katz, 1995). Children that receive critical or dismissive responses from parents when facing emotional challenges experience higher rates of distress, including negative emotions such as anger, frustration, and sadness (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Katz, Maliken, & Stettler, 2012; Thompson, 2014). Conversely, parents who affirm that their child's feelings are important, provide support that facilitates coping, and assist the child with managing a situation without taking over may facilitate more adaptive emotion regulation strategies in their children. There is evidence to suggest that these parenting behaviors during upbringing are associated with children who are generally more competent in recognizing and communicating their emotions, show less frustration when completing challenging tasks in laboratories, and are less likely to develop psychopathologies related to emotion dysregulation (Calkins & Johnson, 1998; Caspi et al., 2004).

As children mature and form a more nuanced comprehension of their own emotions and the emotions of others, peer and sibling conversations and evaluations of feelings also impact emotional clarity and awareness. When children reach school age, they tend to talk about their feelings more frequently with friends and siblings than they do with their parents, which may either advance or obstruct the understanding of emotion, depending upon the peer group (Brown, Donelan-McCall, & Dunn, 1996). For example, siblings and peers can contribute positively to the development of awareness and clarity of emotion states by talking through antecedents and consequences together and providing compassion and support. On the other hand, a peer group that is dismissive or punishing of certain emotional experiences or expression (e.g., the

experience of sadness in a male peer group) and that models maladaptive or impulsive regulation strategies (e.g., physical violence in response to embarrassment) can impede the development of emotion regulation.

Acceptance of unpleasant or unwanted cognitions and feelings rather than focusing on changing these experiences has also become a key component of emotion regulation research (Wolgast, Lundh, & Viborg, 2011). Hayes and colleagues (1996; 1999) posited that emotion is an essential part of the human experience, and that acceptance (i.e., fully experiencing the present, including emotions, thoughts, and bodily sensations without trying to change, control or avoid them) and continued engagement in valued behaviors are adaptive strategies to reduce distress. Conversely, excessive focus and efforts to change or avoid unwanted cognitions and feelings potentially exacerbate distress, inhibit psychological flexibility, and become obstacles to living a full and meaningful life (Hayes et al., 1996; 1999). Thus, acceptance is an alternative to engaging in experiential avoidance as a means of reducing contemporaneous distress.

Acceptance influences the unfolding emotional response to an internal or external antecedent by noticing emotional responses and overriding learned automatic responses such as suppression, avoidance, or judgment (Alberets, Schneider, & Martijn, 2012). Lacking acceptance of emotional experiences has been associated with maladaptive secondary emotional states in response to one's own emotions (e.g., experiencing shame in response to feeling anxious), and secondary emotional states are strongly related to difficulties in emotion regulation (Hayes, Strosahl, & Wilson, 1999).

Experiential avoidance, defined as the “unwillingness to remain aware and conscious of a particular private experience” (Dan-Glauser & Gross, 2015, p. 1), is another general emotion

regulation strategy observed across psychopathologies, and it often entails different forms of suppression to avoid the emotion (Hayes, Wilson, Follette, & Strosahl, 1996). Experiential avoidance and suppression have been associated with higher levels of physiological arousal and activation of the sympathetic nervous system (i.e., skin conductance, heart rate, pupil dilation, cortisol levels; Gross & Levenson, 1997; Dan-Glauser & Gross, 2015; Kunzmann, Kupperbusch, & Levenson, 2005), strong and enduring decreases in emotional expressivity (Roberts, Levenson, & Gross, 2008; Gross & Levenson, 1997), and reliably decrease the experience of positive emotions (Strack, Martin, & Stepper, 1988; John & Gross, 2004), yet have no effect on the subjective experience of negative emotion (Roberts et al., 2015). Alternatively, acceptance has been associated with reduced magnitude and duration of negative emotions when watching distressing content (Wolgast et al., 2011), decreased negative expressivity and reported negative mood (Alberts et al., 2012), increased positive emotions (Dan-Glauser & Gross, 2015), and lowering of respiratory rate, increased oxygenation, and significantly smaller changes in heart rate, blood pressure, and pulse amplitude when viewing emotional stimuli compared to emotional suppression (Dan-Glauser & Gross, 2015; Dunn, Billotti, Murphy, & Dalgleish, 2009). Consequently, acceptance and engagement in value-driven and goal-directed behaviors should be considered in a comprehensive conceptualization of emotion regulation to encapsulate not only the subjective experience of emotion, but also the importance of moving toward goals in spite of negative emotional states.

Given the multifaceted nature of emotion regulation and the transdiagnostic importance of assessing and identifying emotion dysregulation in the treatment of mental disorders, a comprehensive conceptualization and assessment measure was needed. Gratz and Roemer (2004)

developed a multidimensional assessment of emotion regulation and dysregulation by integrating the conceptual and empirical work pertaining to clinically relevant difficulties in ER, resulting in the *Difficulties in Emotion Regulation Scale* (DERS; Gratz & Roemer, 2004). The DERS is a self-report measure that incorporates the evidence-based conceptualizations of ER, highlighting that the distinct approaches taken by various researchers to understand and explain such a complex process are complimentary rather than contradictory. The subscales of the measure relate to the components of regulation that have been identified as the field has progressed from the one-dimensional notion of regulation equating to ‘control’ to a nuanced and more complex, multifaceted construct. Gratz and Roemer (2004) conceptualized difficulties in emotion regulation as deficits in one or more of the following areas: an awareness and understanding of one’s own emotions (corresponding with the Awareness and Clarity subscales of the DERS); modulation of emotional arousal (Strategies subscale); acceptance of emotions rather than avoidance (Nonacceptance subscale); and the ability to act in desired ways regardless of emotional state, as opposed to behaving impulsively or not being able to move past the emotion when necessary (Impulse and Goals subscales). The DERS has seen wide use in emotion regulation research, likely due both to its convenience as a self-report instrument and its ability to measure multiple aspects of emotion regulation/dysregulation.

### **1.3 Emotion Regulation and PTSD**

Although imprecise due to the recency of systematic investigation, the most robust conclusion to be made from the current research is the etiology and maintenance of PTSD in youth is multiply determined and heterogenous, influenced by biological, developmental, psychological, social, and environmental components (Meiser-Stedman, 2002; Pynoos,

Steinberg, & Piacentini, 1999). Traumatic events are complex, idiosyncratic, and the events most associated with negative psychological outcomes and limitations in quality of life (i.e., sexual assault, abuse by caregiver, etc.) are difficult to study prospectively for ethical reasons. As previously discussed, emotion regulation (ER) and dysregulation have been associated with numerous psychopathologies across the lifespan, and the ontogenesis of ER components are important developmental milestones for children and adolescents (Fletcher, 2003; Thompson, 2014). Difficulties in ER have been associated with both the development and maintenance of anxiety and mood-related disorders, occurrence of secondary disorders (i.e., comorbidity; LeBlanc, Essau, & Ollendick, 2017; Riley, Bokszaanin, & Essau, 2017), and engagement in behaviors that can exacerbate poor outcomes (e.g. aggression, self-harm, delinquency, substance use/abuse; Bushman, Baumeister, & Phillips, 2001; Gratz, 2003; Marshall-Berenz, Vujanovic, & MacPherson, 2011). Difficulties in ER have been demonstrated in adults and adolescents with PTSD (Badour & Feldner, 2013; Bardeen, Tull, Stevens, & Gratz, 2015; Tull, Gratz, Salters & Roemer, 2004), although this relationship has almost entirely been studied in adults and predominantly focused on one or two components of ER in comparison studies (e.g., suppression and acceptance; suppression and reappraisal; Wolgast, Lundh, & Viborg, 2011; Xiong et al., 2013). Integrated conceptualizations of ER difficulties, such as the development of the DERS (Gratz & Roemer, 2004), have resulted in clinically relevant components of ER being assessable and quantifiable, which has facilitated new possibilities for developmental psychopathology research (reviewed below).

Adults with emotion regulation difficulties are at greater risk of posttraumatic stress symptoms (PTSS), PTSD diagnosis, and comorbid mood and personality disorders (Badour &



Feldner, 2013; Tull, Barrett, McMillan, & Roemer, 2007). Earlier work implicating emotion regulation difficulties with PTSD symptom severity and functional impairment in adult clinical samples (Cloitre, Miranda, Stoval-McClough, & Han, 2005) was limited by a narrow conceptualization of ER. For instance, Cloitre and colleagues (2005) used the *General Expectancy for Negative Mood Regulation* (Cantazaro & Mearns, 1990) to measure difficulties with emotion regulation; however, this measure only focuses on the down-regulation of negative mood states and an individual's self-efficacy in managing negative emotions. More recent studies have concentrated on multiple components of ER and how participants with a trauma history apply them in experimental procedures. Badour and Feldner (2013), for example, found that female undergraduate students with a history of interpersonal trauma (i.e., sexual assault, intimate partner violence, domestic violence) who had more physiological reactivity to a trauma-related narrative had significantly higher self-reported emotion dysregulation, reported more PTSS, and exhibited greater symptom severity than those who were less reactive. Emotion dysregulation, operationalized as a higher total score on the DERS, mediated the relationship between physiological reactivity and PTS symptoms and severity of symptoms (Badour & Feldner, 2013).

When the six components of the DERS were examined individually in relation to PTSS severity in an undergraduate sample, severity was associated with number of impairments, including: lack of emotional acceptance; difficulties engaging in goal-directed behaviors when experiencing negative emotions; behaving impulsively when upset; limited adaptive emotion regulation strategies; and lack of emotional clarity (Tull, Barrett, McMillan, & Roemer, 2007).

Although measuring adult emotion regulation outcomes, Weiss and colleagues' (2013) examination of childhood trauma, emotion dysregulation, and PTSS in a substance-using sample suggested that difficulties in ER are likely a factor in the maintenance and chronicity of PTSD. They found that adult substance use disorder (SUD) patients who reported childhood sexual, physical, and/or emotional abuse who also exhibited probable PTSD (i.e., meeting criteria using self-report and clinical cut-off criteria rather than diagnostic interview) had significantly higher levels of difficulties engaging in goal directed behavior when upset, controlling impulsive behavior when distressed, engaging in adaptive and effective ER strategies when experiencing negative emotions, and achieving emotional clarity. Furthermore, the relationship between childhood physical and emotional abuse (but not child sexual abuse) and current, probable PTSD diagnosis was mediated by difficulties controlling impulsive behavior. Additionally, higher total difficulties in emotion regulation were significantly associated with more severe childhood abuse experiences. Thus, the trauma of childhood appears to be associated with greater difficulties in emotion regulation as an adult, which in turn was associated with a higher probability of PTSD diagnosis.

The experiences of trauma and posttraumatic symptoms have also been posited to interfere with the normative development of emotion regulation in youth (Shields & Cicchetti, 1998; Maughan & Cicchetti, 2002). Early work examining the emotional responses of children and adolescents who had been maltreated indicated that they had significantly lower levels of emotion expression and higher levels of emotional inhibition or suppression across contexts, both of which have been considered maladaptive and associated with emotional numbing observed in PTSD (Camras et al., 1988; Camras & Rappaport, 1993; Ford, Fraleigh, Albert, &

Connor, 2010; Shipman et al., 2005). Although informative, these studies focused on a limited conceptualization of difficulties with ER, and more recent studies have conducted investigations via a more nuanced model.

Espil and colleagues (2016), for example, investigated the mediating role of emotion regulation in the relation between PTSD and depression in a sample of inpatient adolescents (aged 12-17 years old) in Mississippi. This sample was ethnically diverse (36% African-American; 20% mixed race), approximately gender matched (48% male), low SES, and exhibited severe clinical symptoms (e.g., 40% reported a past suicide attempt; 46% reported a history of self-harm). The authors used only the total score of the DERS rather than the subscales pertaining to individual components of the Gratz and Roemer (2004) conceptualization, and found that PTSD symptom severity was associated with significantly higher levels of emotion dysregulation. Additionally, difficulties in emotion regulation partially mediated the relationship between PTSD and depression, accounting for 37% of the variance in depression symptoms explained by PTSD. The direct relationship between PTSD and depression, however, remained significant after including emotion regulation difficulties in the model. They did not find any significant effects of gender, race, or trauma type but did find a significant relationship between higher age and depression symptoms. This study suggests that adolescents with PTSD are more likely to have difficulties in emotion regulation, and that these difficulties are also associated with more severe symptoms and comorbid depression. Although again informative, this study is one of the very few examining the combination of these constructs in youth.

## **1.4 The Current Study**

On the basis of the literature reviewed and paucity of extant studies in youth, the current study aims to examine whether or not specific aspects of emotion regulation (i.e., Nonacceptance, Goals, Impulsivity, Awareness, Strategies and Clarity, as measured by the DERS) mediate the relationship between experiencing traumatic events and post-traumatic stress symptoms in an inpatient sample of adolescents. It is hypothesized that (1) emotion dysregulation will mediate the relationship between traumatic events and posttraumatic stress symptoms, (2) the six facets of emotion dysregulation will account for differing variances in the relationship between traumatic events and posttraumatic stress symptoms, and (3) gender will moderate the relationship between traumatic events and facets of emotion dysregulation, as well as moderate the relationship between emotion dysregulation and posttraumatic stress symptoms.

## CHAPTER 2

### METHOD

#### 2.1 Participants

Four hundred and fifty-four adolescents, aged 12-17 years old, initially completed self-report measures during their in-patient hospitalization in a psychiatric facility for juveniles in Mississippi. Patients generally arrived in this setting after a long history of behavioral disturbance, which usually entailed aggression toward family members and/or peers. Participants completed self-report measures and demographic information as a standard part of their intake procedures at the facility in the time period spanning May 2012 to June 2015.

**Table 1.** *Demographics of adolescents included in the present study.*

	Female <i>n</i> =77		Male <i>n</i> =77		Total <i>n</i> =154	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Age	14.53	1.33	14.17	1.53	14.35	1.44
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Ethnicity						
White	31	40.26	31	40.26	62	40.26
Black	38	49.35	35	45.45	73	47.40
Mixed	5	6.49	7	9.09	12	7.79
Native	2	2.60	0	0	2	1.30
American						
Other	1	1.30	4	5.19	5	3.25

After participants with missing data were removed ( $n = 248$ ) and participants with problematic data, defined by homogeneity of responses across measures (including reverse

scored items) were removed ( $n = 6$ ), the final sample consisted of 154 adolescents. The sample consisted of 77 females and 77 males, ranging in age from 12-17 years, and a mean age of 14.35 ( $SD = 1.44$ ). Forty-seven percent of the sample identified as Black/African-American, 40% as White, 8% as multiethnic, and 3% as ‘other’, and 1.3% Native American (see Table 1).

**Table 2.** *Internal consistency reliability analyses for continuous variables (N = 154)*

Scale	No. of items	Cronbach's $\alpha$	Range of inter-item correlations	Mean inter-item correlation
DERS	36	.90	-.52-.82	.21
Nonaccept	6	.87	.30-.67	.53
Goals	5	.76	.03-.64	.39
Impulse	6	.85	.09-.82	.47
Aware	6	.74	.23-.47	.32
Strategies	8	.80	-.19-.66	.33
Clarity	5	.63	.11-.45	.26
RPVES	36	.93	-.08-.88	.27
CPSS	24	.89	-.35-.65	.18
Symptom Severity	17	.93	.21-.65	.43
<i>Reexperiencing</i>	5	.81	.35-.65	.47
<i>Avoidance</i>	7	.87	.33-.63	.49
<i>Hyperarousal</i>	5	.78	.21-.56	.41
Impairment Severity	7	.85	.25-.65	.45

## **2.2 Traumatic Events.**

The *Recent and Past Violence Exposure Scale* (RPVES; Singer, Anglin, Song & Lunghofer, 1995; Song, Singer & Anglin, 1998) was used to measure participants' exposure to physical and sexual violence. It contains 24 items assessing recent exposure (defined as 'the past year') and 12 items for past exposure (defined as 'while growing up, not including the past year'). The scale contains six variable clusters that include neighborhood, home, school, and sexual violence. Examples of items include "Saw someone beaten or mugged in your neighborhood" (witnessing neighborhood violence), "Been beaten at home" (witness/victim of home violence), "Saw someone else slapped/hit/punched at school" (witnessing school violence), "Been attacked or stabbed" (witness/victim of a shooting or knife attack), "Been beaten or mugged in your neighborhood" (victim of neighborhood or school violence), and "Been made to do a sexual act against your wishes" (witness/victim of sexual violence).

The 24 items relating to recent exposure are scored on a 7-point Likert-type scale, ranging from 0 (Never) to 6 (Almost Every Day). The 12 items measuring past exposure are scored on a 4-point Likert-type scale, ranging from 0 (Never) to 3 (Very Often). The recent and past item scores are combined to obtain a lifetime exposure to violence, where higher scores indicate greater self-reported exposure to violence. Singer and colleagues (1995) reported Cronbach's alphas for the six variable clusters ranging from 0.66 to 0.87. In the present study, the Cronbach's alpha was .93.

## **2.3 Posttraumatic Stress Symptoms.**

The Child PTSD Symptom Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001) is a self-report measure that contains 17 items corresponding to the 17 symptoms of Posttraumatic Stress Disorder included in the *DSM-IV TR* (American Psychiatric Association, 2000) and an

additional 7 items assessing the functional impact of symptoms. The measure was designed and validated for use with children and adolescents aged 8 to 18 years old. In Part 1, respondents are directed to indicate how often the symptom described has bothered them in the previous two weeks. Each item in the measure is rated on a 4-point Likert-type scale, ranging from 0 (Not at all) to 3 (5 or more times per week/Almost always). The instrument yields three subscales that correspond with the symptom clusters of PTSD: re-experiencing, avoidance, and arousal. The re-experiencing subscale contains 5 items with a maximum score of 15. The avoidance subscale contains 7 items with a maximum score of 21. The arousal subscale has 5 items with a maximum score of 15. Examples of items include “Having upsetting thoughts or images about the event that came into your head when you didn’t want them to” (re-experiencing symptom), “Trying not to think about, talk about, or have feelings about the event” (avoidance symptom), and “Having trouble concentrating (for example, checking to see who is around you and what is around you)” (arousal symptom). In Part 2, respondents are asked if the symptoms they endorsed in Part 1 have gotten in the way of the seven areas of functioning in their life in the past two weeks. These items are scored dichotomously (1= *Yes* and 0= *No*), yielding a severity of impairment score ranging from 0-7.

The scores obtained can be used to classify respondents as in terms of having a probable diagnosis of PTSD or not. Scoring procedures indicate that responses of 2 or 3 are considered to be clinically significant. In combination with DSM-IV-TR diagnostic criteria that require a minimum endorsement of one re-experiencing symptom, three avoidance symptoms, and two arousal symptoms, the instrument can be used to indicate probably PTSD diagnosis.

Alternatively, Foa and colleagues (2001) found that a clinical cut-off score equal or greater than 11 of the total score in Part 1 yielded a 95 percent sensitivity score and a 96 percent specificity



score for a PTSD diagnosis (i.e., extremely high performance with considerably less scoring complexity).

Additionally, the total score of each subscale in Part 1 (re-experiencing, avoidance, and arousal) and the total score of all 17 items can be used to measure the severity of post-traumatic stress symptoms. In this study, the total symptom severity and impairment score will be used, giving a possible range of 0-58 score will be used, where higher scores indicate more severe post-traumatic stress symptoms and severity and lower scores indicate less symptomology and impairment due to post-traumatic stress. The CPSS Total Severity Score has been shown to have good internal consistency with a Cronbach's alpha of  $\alpha = 0.89$  (Foa et al., 2001). Cronbach's alphas for the subscales are also acceptable, reported as  $\alpha = 0.80$  (avoidance),  $\alpha = 0.73$  (avoidance), and  $\alpha = 0.70$  (arousal). The Total Severity Score has also been shown to have good convergent and discriminant validity and good test-retest validity (Foa et al., 2001). Cronbach's alphas in the present study were congruent with those previously found, ranging from acceptable to excellent with an alpha of .93 for the Total Severity Score (see Table 2 for reliability statistics for CPSS subscales).

#### **2.4 Emotional Regulation.**

The *Difficulties in Emotional Regulation Scale* (DERS; Gratz & Roemer, 2004) is a measure developed to assess clinically relevant emotion regulation (ER) difficulties. The DERS uses an integrative conceptualization of emotion regulation and provides a total score as well as six subscale scores (i.e., Nonacceptance, Goals, Impulse, Awareness, Strategies, and Clarity). There are 36 items that are rated on a five-point Likert-type scale (1 = *almost never* and 5 = *almost always*), where higher scores indicate more difficulties with emotion regulation. Initially developed and validated using an undergraduate population, the internal consistency for the total

score reported in this population was excellent with a Cronbach's  $\alpha = 0.93$  (Gratz & Roemer, 2004). The factor structure, validity, and internal consistency of the six subscales have also been demonstrated for adolescents aged 11-17 years (Neumann, van Lier, Gratz & Koot, 2010). In the present study, the Cronbach's alpha for the total scale was  $\alpha = .90$ , indicating excellent internal consistency.

*Nonacceptance of Emotional Regulation (NONACCEPTANCE)*. The Nonacceptance scale contains six items, yielding a maximum score of 30. Higher scores on this subscale indicate higher levels of distress due to negative self-judgments related to emotional distress. Examples of items include "When I'm upset, I feel guilty for feeling that way" and "When I'm upset, I become angry at myself for feeling that way." Neumann and colleagues (2010) reported the Cronbach's alpha for boys was  $\alpha = 0.73$  and girls was  $\alpha = 0.76$ . The Cronbach's alpha in the present study was  $\alpha = .87$ .

*Difficulties Engaging in Goal-Directed Behavior (GOALS)*. The Goals scale has five items including one reverse-scored item, yielding a maximum score of 25. Higher scores are indicative of greater difficulties engaging in goal-directed behaviors when experiencing distress due to an inability to focus or think about things other than the individual's emotional state. Examples of items include, "When I'm upset, I have difficulty concentrating on other things" and "When I'm upset, I have difficulty thinking about anything else." Neuman and colleagues (2010) reported the Cronbach's alpha for boys as  $\alpha = 0.81$  and for girls as  $\alpha = 0.82$ . In the present study, the Cronbach's alpha was  $\alpha = .76$ .

*Impulse Control Difficulties When Distressed (IMPULSE)*. The Impulse scale contains six items, including one reverse-scored item and has a maximum score of 30, with higher scores indicating more impulse control difficulties when in emotional distress. Examples of items

include, “When I’m upset, I become out of control” and “When I’m upset, I lose control over my behaviors.” Neuman and colleagues (2010) reported the Cronbach’s alpha for boys as  $\alpha = 0.86$  and  $\alpha = 0.83$  for girls. The Cronbach’s alpha in the present study was  $\alpha = .85$ .

*Lack of Emotional Awareness (AWARENESS).* The Awareness scale contains six items giving a maximum score of 30, with all items being reverse-scored. Higher scores on this scale indicate greater difficulties in attending to one’s emotional state and the reason for feeling that way. Examples of items include, “I am attentive to my feelings” and “When I’m upset, I take time to figure out what I’m really feeling.” Neuman and colleagues (2010) reported the Cronbach’s alpha for boys as  $\alpha = 0.73$  and  $\alpha = 0.76$  for girls. In the present study, the Cronbach’s alpha was  $\alpha = .74$ .

*Limited Access to Emotion Regulation Strategies (STRATEGIES).* The Strategies subscale contains eight items, one reverse-scored, giving a maximum score of 40. Higher scores on this subscale indicate deficiencies in knowledge of emotional regulation strategies and/or greater difficulties putting adaptive strategies into use when distressed. Examples of items include, “When I’m upset, I believe that wallowing in it is all I can do” and “When I’m upset, my emotions feel overwhelming.” Neuman and colleagues (2010) reported the Cronbach’s alpha as  $\alpha = 0.80$  for boys and  $\alpha = 0.87$  for girls. The Cronbach’s alpha in the present study was  $\alpha = .80$ .

*Lack of Emotional Clarity (CLARITY).* The Clarity subscale contains five items, with two items reverse-scored, yielding a maximum score of 25. Higher scores on this subscale are indicative of greater difficulties in recognizing the emotional state that one is experiencing. Examples of items include, “I have difficulty making sense out of my feelings” and “I have no idea how I am feeling.” The reported Cronbach’s alphas by Neuman and colleagues (2010) were

$\alpha = 0.74$  for boys and  $\alpha = 0.83$  for girls. In the present study, the internal consistency of this subscale was poor, with a Cronbach's alpha of  $\alpha = .63$

## CHAPTER 3

### RESULTS

#### 3.1 Demographics.

As shown in Table 1, there were no significant difference in ethnicity by gender,  $\chi^2(4) = 4.26, p > .05$ , or age between the genders,  $F(1, 152) = 2.48, p > .05$ . Pearson correlations were computed for age, and all continuous variables to be included in subsequent analyses (see Table 3). Age was positively correlated with trauma exposure but not to the outcome measure of posttraumatic stress disorder or any measures of emotion regulation difficulties,  $r = .220, p < .01$ . As such, it was not included as a covariate in subsequent analyses.

**Table 3.** Means and standard deviations for variables included in moderated-mediation models.

	Female <i>n</i> = 77		Male <i>n</i> = 77		Total <i>n</i> = 154	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
RPVES	31.82	25.50	25.55	23.42	28.68	24.61
CPSS	22.53	11.65	15.87	12.21	19.20	12.35
DERS						
Nonaccept	1.91	1.06	1.79	.95	1.85	.98
Goals	3.04	1.20	2.56	.95	2.80	1.11
Impulse	2.68	1.28	2.64	1.04	2.66	1.16
Aware	3.53	.83	3.09	1.04	3.31	.97
Strategies	2.28	.97	2.08	.82	2.19	.90
Clarity	2.34	.96	2.28	.70	2.31	.84
Total	2.61	.72	2.39	.62	2.50	.68

**Table 4.** Pearson correlation coefficients of Age and all continuous variables included in subsequent analyses.

	Age	RPVES	CPSS	Nonaccept	Goals	Impulse	Aware	Strategies	Clarity	DERS (Total)
Age	1	-	-	-	-	-	-	-	-	-
RPVES	.220**	1	-	-	-	-	-	-	-	-
CPSS	.091	.495**	1	-	-	-	-	-	-	-
Nonaccept	.057	.324**	.536**	1	-	-	-	-	-	-
Goals	.105	.335**	.466**	.529**	1	-	-	-	-	-
Impulse	.095	.374**	.388**	.420**	.689**	1	-	-	-	-
Aware	.029	.183*	.248**	.192*	.346**	.251**	1	-	-	-
Strategies	.111	.411**	.618**	.690**	.755**	.753**	.268**	1	-	-
Clarity	.090	.302**	.450**	.419**	.362**	.304**	.098	.447**	1	-
DERS (Total)	.109	.439**	.613**	.748**	.847**	.806**	.497**	.917**	.531**	1

\*\*  $p < .01$

\*  $p < .05$

**Table 5.** Trauma incidence by type measured by RPVES and gender.

Trauma Type		Female <i>n</i> = 77		Male <i>n</i> = 77		Total <i>n</i> = 154	
		Never	Endorsed	Never	Endorsed	Never	Endorsed
Sexual violence **	%	45	55	88	12	67	33
	<i>n</i>	35	42	68	9	103	51
Victim of violence	%	23	77	29	71	26	74
	<i>n</i>	18	59	22	55	40	114
Witnessed violence *	%	18	82	34	66	26	74
	<i>n</i>	14	63	26	51	40	114
Neighborhood violence	%	29	71	29	71	29	71
	<i>n</i>	22	55	22	55	44	110
School violence	%	22	78	29	71	25	75
	<i>n</i>	17	60	22	55	39	115
Violence with a weapon	%	44	56	55	45	49	51
	<i>n</i>	34	43	42	35	76	78

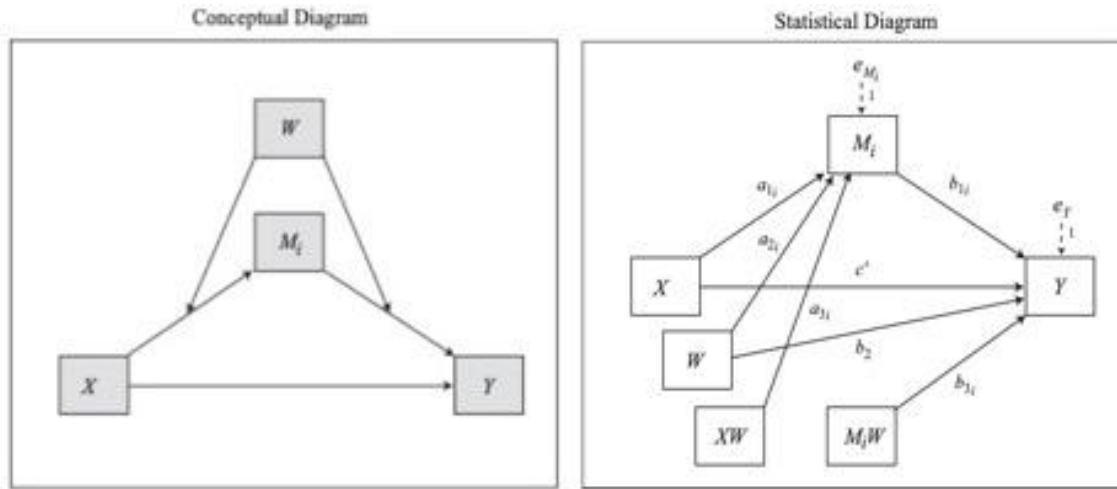
\*\*  $p < .01$

\*  $p < .05$

### 3.2 Analyses.

**Trauma, difficulties in emotion regulation, and posttraumatic stress.** An independent samples t-test indicated that there was not a significant difference in total trauma exposure between males and females,  $t(152) = 1.59, p > .05$ . As shown in Table 5, each type of violent trauma assessed by the RPVES was dichotomized into ‘Never’ and ‘Endorsed’, and female

participants were more likely to have experienced sexual violence,  $\chi^2(1) = 31.93, p < .01$ , and witness violence,  $\chi^2(1) = 31.93, p < .01$ , than male participants.



**Figure 1.** Conceptual and statistical diagram of moderated-mediation model tested (Model 58; Hayes, 2013).

Seven moderated mediation analyses were conducted using Hayes (2013) PROCESS macro for SPSS, with ‘Model 58’ as the specific model tested (see Figure 1 for conceptual and statistical diagram of the model). In all analyses, trauma exposure was included as the predictor variable (X), posttraumatic stress symptoms as the outcome variable (Y), and gender as the hypothesized moderator variable (W). Specifically, gender was hypothesized to moderate the relationship between the predictor variable and hypothesized mediating variable, and the relationship between the mediating variable and the outcome variable. Each analysis only differed by the hypothesized mediator, and included covariates. As shown in Table 3, the six subtests of the DERS (Nonaccept, Goals, Impulse, Aware, Strategies, and Clarity) had significant positive correlations with each other, with an exception being the relationship between Aware and Clarity,  $r = .10, p > .05$ . Subsequently, the remaining 5 subtests were

included as covariates in the models testing Nonaccept, Goals, Impulse, and Strategies as the mediating variable, and 4 subtests were included as covariates in the models testing Aware and Clarity to account for multicollinearity. All variables were mean-centered by the PROCESS macro, and the bootstrap method was set at 5000 iterations. In order to test for unconditional mediation, a simple mediation analysis was ran using ‘Model 4’ from Hayes’ (2013) PROCESS macro as ‘Model 58’ tests only for conditional mediation based on levels the hypothesized moderator (i.e. for gender, male or female), whereas ‘Model 4’ provides unstandardized and standardized coefficients for the indirect mediation pathway. The standardized covariates will be reported for these pathways. All covariates included in the simple mediation models were identical to those included in the moderated-mediated (Model 58) analyses.

**Table 6.** *Mediation pathways of trauma exposure on posttraumatic stress symptoms.*

Mediator	Coefficient ( $\beta$ )	SE	95% Bootstrap CI	
			Lower	Upper
Nonaccept	.005	.010	-.010	.031
Goals	-.005	.009	-.027	.008
Impulse	-.009	.011	-.035	.008
Aware	.019	.014	-.003	.051
Strategies	.180 **	.043	.098	.266
Clarity	.048	.026	-.006	.098
DERS Total	.202 **	.045	.113	.290

\*\*  $p < .01$

*Nonaccept.* It was hypothesized that nonacceptance of emotional distress (Nonaccept) would mediate the relationship between trauma exposure and posttraumatic stress symptoms, and gender would moderate the relationship between trauma exposure and nonacceptance, and the relationship between nonacceptance and posttraumatic stress symptoms. In the first step of the moderated-mediation model testing, Nonaccept was regressed on trauma exposure and gender,



and their interaction term was tested. Trauma was not a significant predictor of nonacceptance of emotional distress,  $t(145) = .791, p > .05$ , nor was gender predictive of nonacceptance of emotional states,  $t(145) = 1.21, p > .05$ , and gender did not moderate the relationship,  $t(145) = .356, p > .05$ . In total, the first regression of the model that predicted nonacceptance of emotional states was significant,  $F(8, 145) = 19.68, p < .0001, R^2 = .52$ , accounting for 52% of the variance in Nonaccept in the model.

**Table 7.** Model 58 testing Nonaccept as mediator.

<b>Trauma Exposure (X) to nonacceptance of emotional distress (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	.002	.003	-.003	.007
Gender (W)	.145	.120	-.092	.382
Trauma exposure (X) * Gender (W)	.002	.005	-.008	.011
<b>Nonacceptance of emotional distress (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Nonacceptance of emotional distress (M)	1.79	1.04	-.266	3.84
Gender (W)	-3.45*	1.50	-6.42	-.475
Nonacceptance of emotional distress (M)* Gender (W)	1.35	1.49	-1.60	4.30
Trauma exposure (X)	.133**	.032	.070	.196

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariates: Nonaccept; Goals; Impulse; Strategies; Clarity. Bootstrap samples for 95%CIs= 5,000.

In the second step of the model, reported PTSD symptoms were regressed on Nonaccept, trauma exposure, and gender. The interaction effect of gender and Nonaccept was also tested to determine moderation. The overall model was significant,  $F(9, 144) = 18.16, p < .0001, R^2 = .53$ , although specifically Nonacceptance of emotional distress did not predict reported PTSD symptoms,  $t(144) = 1.72, p > .05$ . However, gender did predict PTSD symptoms,  $t(144) = -2.29,$

$p = .02$ . Specifically, females reported significantly higher levels of PTSD symptomology than males when controlling for other variables in the model. However, gender did not moderate the relationship between nonacceptance of emotional distress and posttraumatic symptoms as hypothesized,  $t(144) = .905, p > .05$ . Exposure to trauma was a significant predictor of posttraumatic stress symptoms,  $t(144) = 4.19, p < .0001$ , however this relationship was not mediated by nonacceptance of emotional distress and the hypothesized model was not supported,  $\beta = .005, SE = .010, p > .05, 95\% CI [-.010, .031]$ .

*Goals.* It was hypothesized that difficulties engaging in goal directed behavior (Goals) would mediate the relationship between trauma and posttraumatic stress symptoms, and gender would moderate the relationship between trauma and goals and the relationship between goals and posttraumatic stress symptoms. In the first step, Goals was regressed on trauma exposure and gender, and their interaction term was tested. The overall model was significant,  $F(8, 145) = 19.32, p < .0001, R^2 = .52$ , although trauma was not a significant predictor of difficulties engaging in goal directed behavior when experiencing distress,  $t(145) = -.849, p > .05$ . The main effect of gender was significant,  $t(145) = -2.69, p < .01$ , however the interaction term was not significant,  $t(145) = .637, p > .05$ . Although females reported significantly more difficulties engaging in goal directed behavior when distressed, this difference was not related to trauma exposure.

In the second step of the analysis, PTSD symptoms were regressed on Goals and gender, the interaction effect of Goals and gender was tested, and the direct effect of trauma as a predictor of PTSD symptoms was tested when accounting for the other variables in the model. Although the overall regression model was significant,  $F(9, 144) = 18.52, p < .0001, R^2 = .54$ ,

difficulties engaging in goal directed behavior did not predict reported PTSD symptoms,  $t(144) = .922, p > .05$ , but the main effect of gender as a predictor of PTSD symptoms was significant,  $t(144) = -2.20, p < .05$ . The interaction of gender and Goals was not significant,  $t(144) = 1.54, p > .05$ . The main effect of trauma as a predictor of posttraumatic stress symptoms when all variables in the model are accounted for was significant,  $t(144) = 4.01, p = .0001$ , and was not mediated by difficulties engaging in goal directed behavior when experiencing distress,  $\beta = -.005, SE = .009, p > .05, 95\% CI [-.027, .008]$ . Thus, the hypothesized model was not supported, although there were significant differences by gender in self-reported difficulties engaging in goal directed behavior and posttraumatic stress symptoms. Females reported more difficulties engaging in goal directed behavior and more posttraumatic stress symptoms than males but these findings were not related to one another in the model.

**Table 8.** Model 58 testing Goals as mediator.

<b>Trauma Exposure (X) to difficulty engaging in goal directed behavior (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	-.003	.003	-.008	.003
Gender (W)	-.358**	.133	-.621	-.095
Trauma exposure (X) * Gender (W)	.003	.005	-.007	.014
<b>Difficulty engaging in goal directed behavior (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Goal directed behavior (M)	.846	.918	-.968	2.66
Gender (W)	-3.29*	1.50	-6.25	-.336
Goal directed behavior (M) * Gender (W)	2.10	1.37	-.605	4.81
Trauma exposure (X)	.127**	.032	.064	.191

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariates: Nonaccept; Impulse; Aware; Strategies; Clarity. Bootstrap samples for 95%CIs= 5,000.

*Impulse.* It was hypothesized that impulsiveness when distressed (Impulse) would partially mediate the relationship between trauma and posttraumatic stress symptoms, and gender would moderate the relationship between trauma and Impulse and the relationship between Impulse and posttraumatic stress symptoms. In the first step, Impulse was regressed on trauma exposure and gender, and their interaction term was tested. Even though the overall model predicting impulsiveness when distressed was significant,  $F(8, 145) = 23.46, p < .0001, R^2 = .56$ , trauma exposure was not a significant predictor of impulsiveness when distressed,  $t(145) = 1.24, p > .05$ , but the main effect of gender was a significant predictor of impulsiveness when distressed,  $t(145) = 2.00, p < .05$ . The hypothesized moderating effect of gender on the relationship between trauma and Impulse was not supported,  $t(145) = -.225, p > .05$ .

In the second step of the model, reported posttraumatic stress symptoms were regressed on Impulse and gender, the interaction term of Impulse and gender was tested, as well as the direct effect of trauma on posttraumatic stress symptoms. Impulsiveness when distressed was not a predictor of posttraumatic stress symptoms,  $t(144) = -1.17, p > .05$ , gender was a significant predictor of posttraumatic stress symptoms as a main effect,  $t(144) = -2.24, p < .05$ , but gender did not moderate the relationship between Impulse and PTSD symptoms,  $t(145) = -.225, p > .05$ . As in previous models, females reported significantly more posttraumatic symptoms compared to males. The direct effect of trauma as a predictor of posttraumatic stress symptoms was significant,  $t(144) = 4.06, p < .0001$ , as was the overall model,  $F(9, 144) = 18.30, p < .0001, R^2 = .53$ , however impulsiveness when distressed did not mediate the relationship between trauma and posttraumatic stress,  $\beta = -.009, SE = .011, p > .05, 95\% CI [-.035, .008]$ . Therefore, the hypothesized model was not supported.

**Table 9.** Model 58 testing Impulse as mediator.

<b>Trauma Exposure (X) to impulsiveness when distressed (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	.004	.003	-.002	-1.37
Gender (W)	.268*	.134	.004	.533
Trauma exposure (X) * Gender (W)	-.001	.005	-.012	.009
<b>Impulsiveness when distressed (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Impulsiveness when distressed (M)	-1.07	.92	-2.89	.743
Gender (W)	-3.36*	1.50	-6.32	-.40
Impulsiveness when distressed (M) * Gender (W)	1.52	1.28	-1.00	4.05
Trauma exposure (X)	.130**	.032	.067	.193

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariaes: Nonaccept; Goals; Aware; Strategies; Clarity. Bootstrap samples for 95%CIs= 5,000.

*Aware.* It was hypothesized that difficulties in emotional awareness (Aware) would mediate the relationship between trauma and posttraumatic stress symptoms, and gender would moderate the relationship between trauma and Aware and the relationship between Aware and posttraumatic stress symptoms. In the first step, Aware was regressed on trauma exposure and gender, and their interaction term was tested. The overall model predicting difficulties in emotional awareness was significant,  $F(7, 146) = 2.50, p < .05, R^2 = .11$ , but trauma exposure was not a significant predictor of difficulties in emotional awareness,  $t(146) = 1.46, p > .05$ , although gender was a significant predictor of difficulties in emotional awareness,  $t(146) = -2.44, p < .05$ . Females experienced more difficulties being aware of their emotions when distressed than males in the sample. However, the interaction term of Aware and gender was not significant,  $t(146) = -.06, p > .05$ , therefore there was no moderation by gender in the hypothesized relationship between trauma and difficulties emotional awareness.

In the second step of the model, posttraumatic stress symptoms were regressed on difficulties in emotional awareness and gender, and the main effect of trauma on posttraumatic stress symptoms was calculated when all variables in the model were taken into account. Even though the overall model predicting posttraumatic stress symptoms in this regression was significant,  $F(8, 145) = 18.42, p < .0001, R^2 = .50$ , difficulties in emotional awareness was not a significant predictor of PTSD symptoms,  $t(145) = 1.72, p > .05$ . Gender was a significant predictor of posttraumatic stress symptoms,  $t(145) = -2.36, p < .05$ , with females reporting greater difficulties in emotional awareness than males, but gender did not modify the relationship between Aware and PTSD symptoms,  $t(145) = -1.10, p > .05$ . The direct effect of trauma

**Table 10.** Model 58 testing Aware as mediator.

<b>Trauma Exposure (X) to difficulties in emotional awareness (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	.005	.003	-.002	-1.37
Gender (W)	.268*	.134	.004	.533
Trauma exposure (X) * Gender (W)	-.001	.005	-.012	.009
<b>Difficulties in emotional awareness (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Difficulties in emotional awareness (M)	-1.07	.92	-2.89	.743
Gender (W)	-3.36*	1.50	-6.32	-.40
Difficulties in emotional awareness (M) * Gender (W)	1.52	1.28	-1.00	4.05
Trauma exposure (X)	.130**	.032	.067	.193

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariates: Nonaccept; Goals; Impulse; Strategies. Bootstrap samples for 95%CIs= 5,000.

exposure as a predictor of posttraumatic stress symptoms was significant,  $t(144) = 4.07, p = .0001$ , but not mediated by difficulties in emotional awareness,  $\beta = .019, SE = .014, p > .05, 95\% CI [-.003, .051]$ .

*Strategies*. It was hypothesized that limited access to emotion regulation (ER) strategies (Strategies) would mediate the relationship between trauma and posttraumatic stress symptoms, and gender would moderate the relationship between trauma and Strategies and the relationship between Strategies and posttraumatic stress symptoms. In the first step, Strategies was regressed

**Table 11.** Model 58 testing Strategies as mediator.

<b>Trauma Exposure (X) to limited access to ER strategies (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	.004*	.002	.001	.007
Gender (W)	-.050	.084	-.217	.117
Trauma exposure (X) * Gender (W)	.002	.003	-.005	.008
<b>Limited access to ER strategies (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Limited access to ER strategies (M)	4.25**	1.48	1.33	7.17
Gender (W)	-3.40*	1.50	-6.37	-.433
Limited access to ER strategies (M) * Gender (W)	1.81	1.65	-1.45	5.08
Trauma exposure (X)	.130**	.032	.067	.193

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariates: Nonaccept; Goals; Aware; Impulse; Clarity. Bootstrap samples for 95%CIs= 5,000.

on trauma exposure and gender, and their interaction term was tested. Trauma exposure was a significant predictor of limited access to emotion regulation strategies,  $t(145) = 1.98, p < .05$ , but gender was not a significant predictor limited access to ER strategies,  $t(145) = -.594, p > .05$ . The interaction term of Strategies and gender was not significant,  $t(146) = -.873, p > .05$ , therefore there was no moderation by gender in the hypothesized relationship between trauma and Strategies as predicted. Taken together, the first regression of the moderated-mediation in prediction of limited access to ER strategies was significant,  $F(8, 145) = 46.74, p < .0001, R^2 = .72$ , accounting for a total of 72% of the variance in limited access to ER strategies.

In the second step of the model, posttraumatic stress symptoms were regressed on limited access to ER strategies and gender, the interaction of Strategies and gender was tested, and the direct effect of trauma exposure on posttraumatic stress was calculated. Higher levels of difficulty accessing ER strategies was a significant predictor of higher incidence of posttraumatic stress symptoms,  $t(144) = 2.88, p < .01$ . Gender also predicted incidence of PTSD symptoms, with females reporting significantly higher rates of symptoms,  $t(144) = -2.27, p = .03$ . However, there was no modifying effects found of gender on the relationship between limited access to ER strategies and posttraumatic stress symptoms,  $t(144) = 1.10, p > .05$ . The direct effect of trauma exposure on posttraumatic stress symptoms was significant,  $t(144) = 4.07, p = .0001$ , however this pathway was also mediated by limited access to ER strategies,  $\beta = .180, SE = .043, p < .05$ , 95% CI [.098, .266]. In all, the regression on posttraumatic stress symptoms was significant,  $F(9, 144) = 18.25, p < .0001, R^2 = .53$ , accounting for 53% of the variance in posttraumatic stress symptoms. The hypothesized model was partially supported, trauma was a significant predictor of limited access to ER strategies, and limited access to strategies was a significant predictor of posttraumatic stress symptoms, and Strategies mediated the relationship between trauma exposure and posttraumatic stress symptoms.

*Clarity.* It was hypothesized that the relationship between trauma exposure ( $X$ ) and posttraumatic stress symptoms ( $Y$ ) would be mediated by lack of emotional clarity (Clarity;  $M$ ). Additionally, it was hypothesized that the relationship between trauma and Clarity, and between Clarity and posttraumatic stress symptoms would be moderated by gender ( $W$ ). In the first step, lack of emotional clarity was regressed on trauma exposure and gender, and their interaction term was tested (i.e.  $X*M$ ). Trauma was not a significant predictor of lack of emotional clarity,



$t(146) = -.444, p > .05$ , nor was gender a significant predictor of Clarity,  $t(146) = -.121, p > .05$ .

There was also no interaction between trauma exposure and gender,  $t(146) = -.873, p > .05$ , indicating gender did not moderate this relationship. Taken together, the first regression of the moderated-mediation in prediction of lack of emotional clarity was significant,  $F(7, 146) = 5.93, p < .0001, R^2 = .22$ , accounting for 22% of the variance of Clarity.

**Table 12.** Model 58 testing Clarity as mediator.

<b>Trauma Exposure (X) to lack of emotional clarity (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	-.001	.003	-.007	.004
Gender (W)	.015	.128	-.237	.268
Trauma exposure (X) * Gender (W)	-.004	.005	-.014	.006
<b>Lack of emotional clarity (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Lack of emotional clarity (M)	2.10*	1.01	.080	.208
Gender (W)	-4.17**	1.50	-7.14	-1.21
Lack of emotional clarity (M) * Gender (W)	-.027	1.83	-3.65	3.59
Trauma exposure (X)	.144**	.032	.080	.208

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Covariates: Nonaccept; Goals; Impulse; Strategies. Bootstrap samples for 95%CIs= 5,000.

In the second step of the model, posttraumatic stress symptoms were regressed on lack of emotional clarity and gender, the interaction of Clarity and gender was tested, and the direct effect of trauma exposure on posttraumatic stress was calculated. Lack of emotional clarity was a significant predictor of higher reported posttraumatic stress symptoms,  $t(145) = 2.09, p < .05$ , and gender was also a significant predictor of posttraumatic stress symptoms,  $t(145) = -2.28, p < .01$ , with females experiencing significantly higher rates of PTSD symptoms than males. Despite these main effects, contrary to the hypothesis, the interaction between Clarity and gender in

predicting PTSD symptoms was not significant,  $t(145) = -.015, p > .05$ . The direct effect of trauma exposure on posttraumatic stress symptoms was significant,  $t(145) = 4.48, p < .0001$ . Overall, the regression model on PTSD symptoms was significant,  $F(8, 145) = 18.72, p < .0001, R^2 = .51$ , with 51% of the variance in posttraumatic symptoms being accounted for by the variables in the model. Despite this, there was no mediation of the trauma and posttraumatic symptoms by lack of emotional clarity as hypothesized,  $\beta = .048, SE = .045, p > .05, 95\% CI [- .006, .098]$ , and gender did not moderate any relationships in the model.

*DERS-Total.* It was hypothesized that the total score on the Difficulties in Emotion Regulation Scale (DERS-Total) would mediate the relationship between trauma exposure and posttraumatic stress symptoms. Further, it was hypothesized that gender would moderate the relationship between trauma and DERS-Total and DERS-Total and PTSD Symptoms. In the first step of the analysis, difficulties in emotion regulation were regressed on trauma exposure and gender. Greater levels of reported trauma exposure was a significant predictor of more difficulties in emotion regulation,  $t(150) = 5.21, p < .0001$ . However, there was no main effect of gender as a predictor of DERS-Total,  $t(150) = -1.53, p > .05$ , nor was there a significant interaction of trauma and gender when predicting difficulties in emotion regulation,  $t(150) = .967, p > .05$ . The overall model at this level of analysis was significant,  $F(3, 150) = 10.76, p < .00001, R^2 = .18$ , with the model accounting for 18% of the variance in difficulties in emotion regulation. Despite the overall significance of the model, the specific hypotheses for this level of analysis were not supported.

**Table 13.** Model 58 testing DERS-Total as mediator.

<b>Trauma Exposure (X) to difficulties in emotion regulation (M)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Trauma exposure (X)	.011**	.002	.007	.015
Gender (W)	-.154	.101	-.353	.045
Trauma exposure (X) * Gender (W)	.004	.004	-.004	.012

<b>Difficulties in emotion regulation (M) to posttraumatic stress symptoms (Y)</b>				
<i>Predictor</i>	<i>b</i>	<i>SE</i>	<i>95%CI<sub>lower</sub></i>	<i>95%CI<sub>upper</sub></i>
Difficulties in emotion regulation (M)	9.11**	1.21	6.73	11.49
Gender (W)	-3.81*	1.48	-6.74	-.892
Difficulties in emotion regulation (M) * Gender (W)	2.55	2.23	-1.85	6.95
Trauma exposure (X)	.133**	.033	.068	.197

\*  $p < .05$ ; \*\*  $p < .01$

$n = 154$ ; X= Independent variable; Y= Dependent variable; M= Mediating variable; W= Moderating variable. Bootstrap samples for 95%CIs= 5,000.

In the second step of the analysis, posttraumatic stress symptoms were regressed on difficulties in emotion regulation and gender, the interaction of DERS-Total and gender was tested, and the direct effect of trauma exposure predicting posttraumatic stress symptoms was tested. Difficulties in emotion regulation predicted higher reported posttraumatic stress symptoms,  $t(149) = 7.55, p < .0001$ . Additionally, gender was also a significant predictor of PTSD symptoms,  $t(149) = -2.58, p < .05$ , with females experiencing more posttraumatic stress symptoms than males. The direct effect of trauma exposure as a predictor of PTSD symptoms was significant,  $t(149) = 4.07, p = .0001$ , and the overall model as a predictor of posttraumatic symptomology was also significant,  $F(4, 149) = 34.37, p < .00001, R^2 = .48$ , accounting for 48% of the variance in PTSD symptoms reported. Contrary to the hypothesis, gender did not moderate the relationship between difficulties in emotion regulation and posttraumatic stress symptoms,  $t(149) = 1.15, p > .05$ . The hypothesized indirect pathway of trauma exposure on posttraumatic

stress symptoms mediated by difficulties in emotion regulation was significant,  $\beta = .202$ ,  $SE = .045$ ,  $p < .05$ , 95% CI [.113, .290].

## CHAPTER 4

### DISCUSSION

The present study examined the role of emotion dysregulation (ED) and specific facets of emotion regulation (ER) in posttraumatic stress symptoms (PTSS) in a sample of adolescent patients receiving inpatient services in a psychiatric facility. Gender differences in how facets of ER mediate the relationship between exposure to trauma and posttraumatic stress symptoms were also investigated. By exploring the associations among these variables, the results contributed to our understanding of the development and maintenance of PTSS, and how traumatic events may manifest into PTSD symptoms in adolescents. Overall, the results supported emotion dysregulation mediating the relationship between trauma and PTSS, however the only specific facet mediating the relationship was access to ER strategies. Gender did not modify any models tested.

The first aim of the study was to establish ER difficulties as a mediator of the relationship between trauma exposure and PTSD symptoms in the current sample. Previous studies of adults (e.g., Tull, Barrett, McMillan, & Roemer, 2007; Badour & Feldner, 2013) and adolescents (e.g., Espil et al., 2016) have consistently found that difficulties in ER, measured by the total score on the ‘Difficulties in Emotion Regulation Scale’ (DERS; Gratz & Roemer, 2004), was a significant mediator of this association. It was hypothesized that this would be found in the present study, and this was confirmed. The second aim of the study, examining each facet of the DERS (i.e. Nonaccept, Goals, Impulse, Aware, Strategies, and Clarity), found that only ‘Strategies’ was a

significant mediator.

The subscale of ‘Strategies’ is operationalized in the DERS as difficulties accessing and/or engaging ER strategies when an individual is experiencing distress. Strategies has been identified as a significant mediator of the relationship between trauma and PTSD symptoms in adults with PTSD diagnoses or probable diagnoses (Tull et al., 2007; McDermott et al., 2009), as well as PTSD with comorbid Substance Use Disorders (SUDs) (Tull et al., 2007; Weiss et al., 2013). In the aforementioned adult findings, however, other facets were also significant. Specifically, Nonaccept, Goals, Impulse, and Clarity were also significant predictors of PTSD symptoms in adults who reported a history of trauma. This contrasts with the current study which found difficulties accessing ER strategies to be the only significant mediator in this study of adolescents. There are a number of possible explanations for this difference, not least of which is the younger age of the participants and the noted developmental changes from adolescence to adulthood in ER and trauma interfering with the normative development of ER in youth samples (Shields & Cicchetti, 1998; Maughan & Cicchetti, 2002).

The Strategies subscale was distinct from the other facets of emotion dysregulation in the current study as far as its correlations with other subscales and the total score on the DERS, which may account for it being the only significant mediator. It accounted for a significantly larger proportion of the variance of the total DERS scores, and had strong to extremely strong positive relationships with three of the other subscales (Nonaccept, Impulse, and Goals). These relationships are stronger than the low-medium positive relationships found by Neuman and colleagues (2010) in their validation study of DERS in adolescents, as well as those found in the original validation study by Gratz and Roemer (2004). The sole validation and factor analysis done in adolescents at the time of writing (Neuman et al., 2010) was conducted on a sample of

urban Dutch school students. This sample is a stark contrast to participants in this study (rural, Southern United States, inpatient, predominantly black), thus it is possible that the factor structure of the DERS in the current sample is not valid. Support for this idea requires further investigation, however the sample utilized in this study also presents a number of other considerations to be discussed further.

The demographics of the participants in the present study are dissimilar from other samples in the current body of literature on trauma, PTSD, and ER difficulties, representing a largely understudied population in the field of psychology. More than half of participants identified their ethnicity as non-white (African-American/Black = 47%; Mixed Race = 8%; Native American = 1%; and Other = 3%), and the sample was collected from an inpatient psychiatric facility that largely provides care to youth from impoverished family backgrounds in Mississippi. Mississippi is a mostly rural state, under-resourced in mental and physical healthcare, and reports the lowest median income of any state in the USA. Although the Great Smoky Mountains Study (GSMS; Costello et al., 1996) aimed to be representative of the rural Southeastern US, the sample was 90 percent white, representative of the small and understudied region it was conducted in but not representative of African-American youth in the South or of Mississippi in particular. Given the dearth of inquiry into such populations, the results of this study provide a basis for further investigation into the significant models found and future research should examine the factor structure of the DERS in similar populations of adolescents, as well as further specify the influence of demographic factors such as race, education, family structure and SES. Additionally, future research should investigate how the complex needs of inpatient samples may influence the impact of ER difficulties and individual facets of ED on the relationship between trauma and PTSD-symptoms.

Although it was hypothesized that gender would moderate the relationship between trauma and ER difficulties, as well as the relationship between ER difficulties and PTSD-symptoms, this hypothesis was not supported. Male and female adolescents in the sample had significant mean differences on a number of variables (e.g. females were more likely to experience sexual violence and witness violence than males, reported more PTSS, and experienced more difficulties in emotion regulation), but there were no mean differences found among the individual facets of the DERS between genders, as well as no moderating effects found. This contrasts with previous studies conducted relating to ER difficulties in adolescents, in which males were more likely to experience difficulties being aware of their emotional state when distressed (Aware), and females experienced higher levels of the five other facets than males (Nonaccept, Goals, Impulse, Strategies, and Clarity) (Badour & Feldner, 2013; Trickey et al., 2012). The lack of previous empirical studies on the population represented in this study may account for no significant differences between genders being found. Additionally, the complex mental health needs and significant trauma history found in inpatient samples could reduce the variability between genders previously found in community samples of adolescents.

Though the diverse sample in this study is a strength, the data set also came with limitations due to the large amount of incomplete or missing data and participants being recruited from an inpatient setting. Although the decision was made to exclude incomplete data, the final sample size was not significantly different from the excluded data demographically and the sample was still large with adequate power to complete the analyses. Specifically, the nature of data collection for this study relied on staff employed at the facility presenting the measures to the adolescents rather than an on-site researcher supervising the completion of questionnaires. Subsequently, 61% of the data collected was incomplete, missing entire measures, or was



problematic (i.e., homogenous responses regardless of item content throughout).

The results of this study have potential clinical implications for the understudied adolescent population represented, pending future research. ‘Strategies’ was the only facet of emotion dysregulation that mediated the relationship between trauma and PTSD symptoms, and investigation is needed into whether this facet may be more amenable to brief interventions targeted at youth. Certainly, the effectiveness of Dialectical Behavioral Therapy (DBT; Miller, Rathus, & Linehan, 2006) indicates that emotion regulation strategies can be taught and may be more concrete and amenable to brief intervention than other difficulties in emotion regulation measured by the DERS. Furthermore, the divergence of the findings from past research in that only one facet mediated the model rather than multiple facets suggests that there may be demographic differences (specifically race, age, and location) in how emotion dysregulation affects the relationship between trauma and PTSD in some populations.

Given the chronicity and adverse outcomes across the lifespan of trauma and posttraumatic stress symptoms, and the compounded effect of trauma experienced during childhood on this course, a greater understanding is needed to provide effective, timely, and accessible intervention for traumatized adolescents. Emotion dysregulation, taken as a total construct, mediated the relationship between trauma exposure and PTSD symptoms in an ethnically diverse group of inpatient adolescents. Moreover, the findings of this study are in contrast to studies done on mostly adult samples that found that multiple facets of difficulties in emotion regulation differentially accounted for PTSD symptoms in people who have experienced trauma. Difficulties accessing and engaging in emotion regulation strategies was the only facet that mediated this relationship independently. Also, gender did not moderate the relationships between trauma exposure, emotion dysregulation or any singular facet of ER difficulties, and

posttraumatic stress symptoms as opposed to past studies. Further inquiry is needed to validate the DERS and its factor structure in an ethnically diverse adolescent population and to elucidate whether brief behavior interventions focusing on emotion regulation strategies would be effective in ameliorating some of the detrimental outcomes of experiencing trauma during childhood and adolescence.

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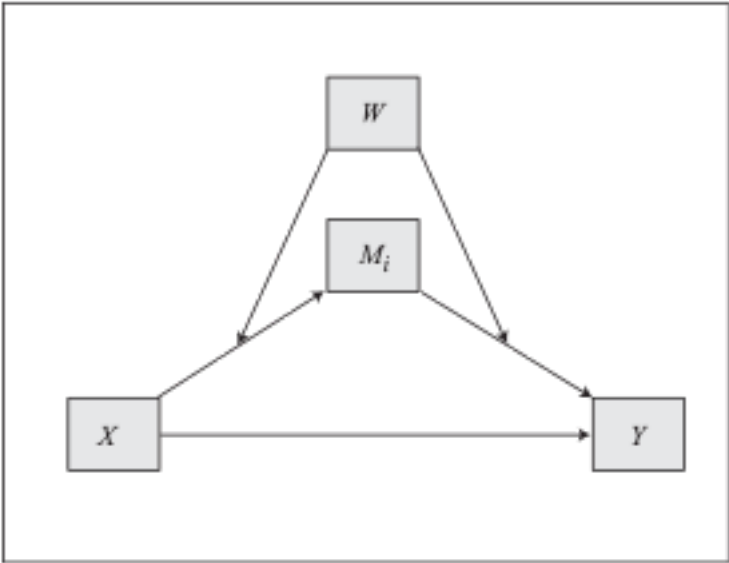
## List of Appendices

Appendix A.

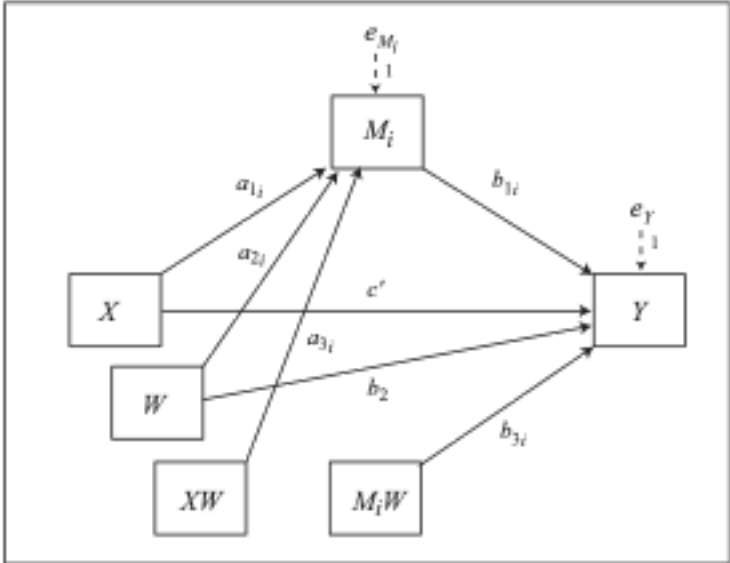
Criterion	A. Exposure to trauma	B. Trauma is persistently re-experienced	C. Avoidance of trauma-related stimuli	D. Negative thoughts or feelings that began or worsened post-trauma	E. Trauma-related arousal and reactivity that began or worsened after the trauma	F. Symptoms last for more than one month	G. Symptoms create distress or functional impairment (e.g., social, occupational)	H. Symptoms are not due to medication, substance use, or other illnesses
	<p><i>One required</i> Direct exposure</p> <p>Witnessing trauma</p>	<p><i>One required</i> Unwanted upsetting memories Nightmares</p>	<p><i>One required</i> Trauma related thoughts or feelings Trauma related reminders</p>	<p><i>Two required</i> Inability to recall key features of trauma Overly negative thoughts and assumptions about oneself or the world</p>	<p><i>Two required</i> Irritability or aggression Risky or destructive behavior</p>	<p><i>Required</i></p>	<p><i>Required</i></p>	<p><i>Required</i></p>
	<p>Learning that a relative/close friend was exposed to trauma</p> <p>Indirect exposure to aversive details of the trauma, usually in the course of professional duties</p>	<p>Flashbacks</p> <p>Emotional distress after exposure to traumatic reminders</p> <p>Physical reactivity after exposure to traumatic reminders</p>		<p>Exaggerated blame of self or others for causing the trauma</p> <p>Negative affect</p> <p>Decreased interest in activities</p> <p>Feeling isolated Difficulty experiencing positive affect</p>	<p>Hypervigilance</p> <p>Heightened startle reaction</p> <p>Difficulty concentrating</p> <p>Difficulty sleeping</p>			

Appendix B.

Conceptual Diagram



Statistical Diagram



## VITA

Elise M. Elligett earned her Bachelor of Psychology (Honors) degree from Macquarie University in Sydney, Australia in 2013. Prior to commencing her Master of Arts in Clinical Psychology, under the supervision of Dr. John Young, she was employed as a clinician in Australia working with children diagnosed with autism, adults with trauma histories and Substance Use Disorder, and families affected by substance use.