Precipitating and prophylactic factors contributing to posttraumatic stress disorder symptomatology in maltreated children

Lisa Marie Linning

Repository Citation

https://digitalscholarship.unlv.edu/rtds/1329

Follow this and additional works at: https://digitalscholarship.unlv.edu/rtds

This Thesis is brought to you for free and open access by Digital Scholarship@UNLV. It has been accepted for inclusion in UNLV Retrospective Theses & Dissertations by an authorized administrator of Digital Scholarship@UNLV. For more information, please contact digitalscholarship@unlv.edu.
INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

ProQuest Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

UMI®
PRECIPITATING AND PROPHYLACTIC FACTORS CONTRIBUTING TO
POSTTRAUMATIC STRESS DISORDER SYMPTOMATOLOGY IN
MALTREATED CHILDREN

by

Lisa Marie Linning

Bachelor of Science
Idaho State University
1998

A thesis submitted in partial fulfillment
of the requirements for the

Master of Arts Degree
Department of Psychology
College of Liberal Arts

Graduate College
University of Nevada, Las Vegas
December 2001

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
The Thesis prepared by

Lisa Marie Linning

Entitled

Precipitating and Prophylactic Factors Contributing to Posttraumatic Stress Disorder Symptomatology in Maltreating Children

is approved in partial fulfillment of the requirements for the degree of

Master of Arts

Examination Committee Chair

Dean of the Graduate College

Examination Committee Member

Examination Committee Member

Graduate College Faculty Representative
ABSTRACT

Precipitating And Prophylactic Factors Contributing To Posttraumatic Stress Disorder Symptomatology In Maltreated Children

by

Lisa Marie Linning

Dr. Christopher A. Kearney, Examination Committee Chair
Associate Professor of Psychology
University of Nevada, Las Vegas

This study examined mediating factors that lead to, or buffer against, development of posttraumatic stress disorder (PTSD) after child maltreatment. Fifty-five children aged 8-17 years voluntarily completed self-report questionnaires and a structured diagnostic interview. It was hypothesized that 1) chronicity and severity of abuse amplify PTSD risk when a child has limited support networks, ineffective coping, external locus of control, affect dysregulation, and a dysfunctional family; 2) key family and victim characteristics would help to ameliorate effects of traumatic abuse; 3) the combination of precipitating and prophylactic factors would determine the effectiveness of managing traumatic maltreatment. Results suggest that dysthymia, attention deficit hyperactivity disorder and religion are strong predictors of PTSD in maltreated children. Additionally, as duration of abuse increased, comorbidity increased, particularly for depression, generalized anxiety and phobias. Hypotheses were not supported for coping skills, locus of control, resilience/hardiness, affect regulation, or support networks.

iii

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
TABLE OF CONTENTS

ABSTRACT ............................................................................................................................... iii

ACKNOWLEDGMENTS ......................................................................................................... v

CHAPTER 1 INTRODUCTION ............................................................................................ 1
  Child Maltreatment ................................................................................................................. 1
  Posttraumatic Stress Disorder ............................................................................................... 5

CHAPTER 2 REVIEW OF RELATED LITERATURE ......................................................... 14
  Etiological Theories of Posttraumatic Stress Disorder .................................................... 14
  Risk versus Resilience ......................................................................................................... 21
  Risk Factors of Posttraumatic Stress Disorder .................................................................. 22
  Prophylactic Factors of Posttraumatic Stress Disorder ................................................... 36
  Purpose of the Study ............................................................................................................ 41
  Hypotheses ............................................................................................................................ 42

CHAPTER 3 METHODOLOGY .......................................................................................... 46
  Participants ............................................................................................................................ 46
  Child Measures ..................................................................................................................... 47
  Clinician Assessment Measures .......................................................................................... 50
  Procedure .............................................................................................................................. 53

CHAPTER 4 DATA ANALYSIS ....................................................................................... 54
  Demographics ....................................................................................................................... 54
  Factor Analysis and Regression .......................................................................................... 54
  Comparison of PTSD Groups ............................................................................................ 56
  Distribution-Free Tests ........................................................................................................ 57

CHAPTER 5 DISCUSSION ................................................................................................. 58

APPENDIX ............................................................................................................................... 67

REFERENCES ......................................................................................................................... 69

VITA .......................................................................................................................................... 93
ACKNOWLEDGMENTS

I would like to thank Dr. Chris Kearney for his continued support, encouragement, and guidance during this project. He always had the right suggestions to light the darkened pathway while providing me the freedom to make the choices that allowed learning and growth. I would also like to thank my committee members for their encouragement, support, and suggestions for refinement. Additionally, I want to thank my husband Dave, whose contributions are too numerous to mention, and my family for their love and cheerleading. Finally, I extend my appreciation to all the children who shared their painful stories with me. Their histories created these pages.
CHAPTER I

PRECIPITATING AND PROPHYLACTIC FACTORS CONTRIBUTING TO POSTTRAUMATIC STRESS DISORDER SYMPTOMATOLOGY IN MALTREATED CHILDREN

Within the field of clinical child psychology, one of the areas of greatest concern is the safety of children. Safety of children is dependent on parent rearing; parent discipline; a safe environment to live, learn and grow; and sufficient provision of physical and emotional needs. When the safety of a child is violated, he or she is forced to question the meaning of life and cognitive distortions can occur that can interrupt or interfere with development in every domain of the child's functioning. When the safety and general needs of a child are not met, the child is considered maltreated.

The study of child maltreatment (CM) has burgeoned over the past 30 to 40 years, with a more recent focus on the implications of CM. Included in this field of study are the reactions children have to traumatic violations of their safety--reactions that can lead to childhood posttraumatic stress disorder (C-PTSD). This paper will discuss the development of C-PTSD within the context of CM. Particular focus will be given to the factors that place a child at risk for developing C-PTSD, as well as those that buffer or protect the child from developing C-PTSD.
Child Maltreatment

Child maltreatment (CM) is a rampant and ever-increasing problem. Prevalence rates vary greatly, but incidence reports indicate continually rising rates and suggest that over 3 million children experienced child abuse and neglect in 1997. It is further estimated that 47 of every 1,000 children are reported to child protective services (CPS) as victims of child maltreatment (Wang & Daro, 1998). However, these figures likely represent a gross underestimation of actual prevalence as they indicate reported cases only. To understand the scope of this problem, as well as the sweeping and variable implications of child maltreatment, researchers have artificially divided CM into four separate types (see below). It should be noted, however, that any or all of these types of abuse are commonly experienced by any given child who is maltreated.

Definition of Child Maltreatment

Although there are many definitions of maltreatment, the National Incidence Study (NIS-3, Sedlak and Broadhurst, 1996) provides the following definitions for four types of maltreatment. Physical abuse refers to a child under age 18 years who has suffered injury (harm standard) or risk of injury (endangerment standard) by a parent or parent-substitute. This injury is the result of being hit with a hand or other object or having been kicked, thrown, shaken, burned, choked, or stabbed. Sexual abuse refers to sexual exploitation by physical contact (i.e. anal, genital, oral, or breast) between a child and another person. Emotional abuse refers to verbal abuse, threats of maltreatment, and harsh nonphysical punishment such as being restrained. Neglect refers to physical endangerment due to inadequate nutrition, hygiene, clothing and supervision, or
emotional endangerment by failure to provide adequate emotional support or affection or failure to protect a child from being exposed to domestic violence.

Characteristics of Child Maltreatment

CM has been identified as having both direct and indirect implications for a child, even resulting in pervasive developmental deficits. Developmental research indicates that the impact and outcome of CM for any given child evolves out of multiple interactions between the environment, caregiver adequacy, and individual child characteristics—hence it is a heterogeneous rather than a unitary event. In this regard, any or all of a series of variables may cause the derailment or change in trajectory of the child's development. The effects may be temporary, influencing current functioning at that time only, or they may be permanent by interfering with the child's core foundations or developmental course, even leading to personality changes.

Identification of CM may further influence the developmental trajectory of a child through social responses to reports of maltreatment, such as placing a child in foster care or requiring him or her to testify in court. Detrimental effects or breakdown can occur with continuous risk variables such as the re-traumatization of court or being taking from the family of origin. These detrimental effects can lead a child to increasingly organize or manage his or her world in a maladaptive manner (Azar, 1986; Azar & Bober, 1999; Wolfe, 1987).

Most importantly, abuse affects a specific child, who has specific characteristics (e.g., temperament, locus of control, cognitive delays) that guide interpretation of the abuse in a multitude of ways. The meaning of the abuse may even change from one developmental era (e.g., childhood) to another (e.g. adolescence, parenting). In light of
this variability, abuse may affect a child to greater or lesser degrees in the physical, social, or emotional domains as they try to cope throughout life. Additionally, the "scars" that develop may be more or less visible during different developmental periods.

While some distinctive differences in the behavioral and emotional displays of children occur from each type of maltreatment, all types manifest some similarities. All groups exhibit a larger proportion of anxious or insecure attachment, as compared with non-abused controls; children in each group display significantly more anger, frustration, and non-compliance, lowered self-esteem, less enthusiasm and positive affect, and below-average social and academic performance than controls (Belsky, 1980; England, Sroufe, & Erickson, 1983; Wekerle & Wolfe, 1996).

Child maltreatment that begins early and is chronic can disrupt basic cognitive schema development. Schema distortions can develop in the areas of social interaction, impairment in affective and behavioral self-regulation, emotional conceptions of the self, and general cognitive and intellectual abilities (Azar & Bober, 1999). In extreme cases, abused children's schemas include elements of emotional pain, lack of control, threat of physical harm, a sense of helplessness, and fears concerning survival. A combination of these can help produce posttraumatic stress disorder (PTSD). The child who has experienced abuse as traumatizing may be unable to trust others and be unable to respond to positive attention and love. When an abused child feels overwhelmed by the unpredictability and negative environment they live in, their coping abilities can become maladaptive and ineffectual. These maladaptive patterns can continue into adulthood, leaving the individual with insufficient coping and anger management skills that may lead to further abuse of others.
The deleterious effects caused by CM, particularly in children who develop PTSD, can last many years and negatively impact behavioral, social, emotional, and academic domains. Given the breadth and scope of the developmental deficits that can result from CM, particularly when a child develops posttraumatic stress symptoms, it is important for researchers and clinicians to identify the variables that impact this trajectory and develop interventions and preventative measures to address these ruinous consequences. Significant literature exists concerning many of the precipitating factors; however, there is a paucity of literature that examines the prophylactic factors of PTSD development in children who have been maltreated. This paper will provide a brief overview of posttraumatic stress disorder within the framework of child maltreatment, including history, symptoms, and prevalent theories. Factors that have been identified as risk variables will be described, as well as factors that are believed to protect or buffer against the development of childhood posttraumatic stress disorder (C-PTSD).

While emotional abuse and neglect are also important in the consideration of posttraumatic stress disorder symptomatology, references to child abuse and maltreatment throughout this paper will be specifically related to physical and sexual abuse of children, or the witnessing of such.

**Posttraumatic Stress Disorder**

Definitional History of PTSD

Reactions to traumatic stress were initially viewed according to the psychoanalytical explanation presented by Freud (1959), who argued that traumatization occurs when the ego's "stimulus barrier" is overwhelmed by a barrage of unmanageable stimuli from external stressors. He believed the organism's functioning would be
disrupted if this stimulus barrier was broken, but removal of the external stressor was expected to lead to a quick restoration of functioning (Freud 1920/1959). Freud did note, however, that unmanageable stimuli could, at times, become extreme enough to overpower the coping mechanisms of the individual, even leading to "traumatic neuroses." Thus, in traditional psychoanalytic theory, enduring traumatic reactions were ascribed to the victim rather than to the stressor. Later writers even defined the re-experiencing of symptoms (such as traumatic play in children) as "defensive failures" (Terr, 1991), referencing attempts to manage traumatic exposure using defense mechanisms such as denial, dissociation, projection, and identification with the aggressor.

Some of the earliest accounts of children's reactions to stress were reported during World War II by Bradner in 1943 and others, but the reports were quite infrequent, particularly during the next 25 to 30 years (Saigh & Bremner, 1999). Definitions and theories of traumatic stress in adults continued to evolve over many decades, culminating in a groundbreaking diagnostic definition in DSM-III (American Psychiatric Association, 1980), and following heated political debate over posttraumatic responses to the Vietnam experience. Despite this recognition and classification of PTSD in adults, no formal recognition or acknowledgment of children's reactions to traumatic stress was included until the revised third edition in 1987. One of the first researchers to do extensive work in the area of childhood PTSD was Lenore Terr (1979, 1983) who described children's reactions to the Chowchilla school bus kidnapping in 1978. Following Terr's lead, many researchers began to recognize and investigate the specific trauma of children and the differences in their reactions and symptom
trajectories. Research concerning childhood trauma and childhood PTSD has steadily increased over the past two decades, bringing new assessment measures and intervention programs to the field (Cohen, 1998).

DSM Criteria

DSM-III was the first edition to contain a diagnostic definition of PTSD. Symptoms of posttraumatic stress disorder were clustered into four criteria that have remained the foundation of subsequent DSM editions. Criterion A is unique to DSM classification, as it identifies an etiology as one of the diagnostic criteria: an individual must be exposed to "a recognizable stressor that would be expected to evoke significant symptoms of distress in almost all individuals" (American Psychological Association, 1980). This implies that PTSD is considered a normal reaction to an abnormal circumstance. Additionally, the intensity and scope of individual reactions were expected to parallel the intensity and duration of exposure to the stressor. It was further recognized that removal of the stressor was not necessarily sufficient for symptoms to abate. DSM-III recognized that symptoms may last for an indefinite period of time, and that PTSD reactions were caused by an unusually threatening stressor, rather than by the weakened nature of the victim.

DSM-IV (American Psychiatric Association, 1994) further refined the diagnosis of PTSD with the following criteria: (A) experiencing or witnessing an event with actual or threatened death or serious injury, and a response of intense fear, hopelessness, or horror; (B) re-experiencing of the event through such means as intrusive thoughts, dreams, or play; (C) avoidance or numbing (e.g., pessimism about the future, avoiding reminders); and (D) overarousal such as difficulty sleeping, hypervigilance or irritability.
Associated symptoms of PTSD described by DSM-IV include impaired affect modulation, self-destructive behavior, dissociation, somatic complains, feelings of ineffectiveness and shame, a feeling of being permanently damaged, constant fear of threat, and a loss of previous beliefs. Despite the strides that were made in defining PTSD symptoms in adults, recognition that these symptoms were also manifested in children has had a much shorter history.

Symptoms of PTSD in Children

The changes in definition of the stressor that occurred in DSM-IV Criterion A (American Psychiatric Association, 1994) reflect the controversy that has accompanied this definition, particularly in relation to childhood posttraumatic stress disorder (C-PTSD). Exposure to a life-threatening event (either as victim or witness) is certainly a strong risk factor for a traumatic stress reaction. However, there are differences in the symptom manifestation of children versus adults, which vary with child-specific and stressor-specific variables (Famularo, Kinscherff, & Fenton, 1990). For instance, children have less amnesia for details of a traumatic event than adults. Additionally, chronic physical and sexual abuse in childhood can result in severe psychopathology and interference with development that does not parallel the classic (adult-onset) PTSD symptom trajectory (Amaya-Jackson & March, 1995).

Symptom manifestation of C-PTSD was discussed by Terr (1991), who distinguished acute and chronic traumatic exposure. Type I trauma (acute) is defined as sudden, unpredictable, and single-event, and Type II trauma (chronic) is defined as expected and repeated, such as ongoing childhood physical and/or sexual abuse.

Symptoms of Type I PTSD include nightmares, spontaneous re-experiencing from real
or symbolic exposure, difficulty falling asleep, hypervigilance, exaggerated startle response, omen formation, guilt, and generalized agitation and anxiety. Type II PTSD is manifested by symptoms of detachment or estrangement from others, dissociative episodes, restricted range of affect, sadness or depression, self-blame, and a feeling that life will be hard. Children who have experienced repeated, traumatic abuse may demonstrate behaviors that are contradictory across different contexts, which can be suggestive of multiple personality or other dissociative disorders. Traumatic reenactments may take the form of self-mutilation, sexual or aggressive play, and suicide behaviors. In general, children who endure ongoing abuse exhibit more problems, and more severe disturbance (e.g. psychoses) than children who are exposed to single event abuse.

Additional studies have also found similar differences in symptom presentation of acute versus chronic abuse (Famularo, Fenton, Augustyn, Zuckerman, 1996; Famularo, Kinscherff, & Fenton, 1990; Kiser, Heston, Millsap & Pruitt, 1991). A mixture of acute and chronic symptoms has also been presented by children who have histories of repeated traumatization and re-exposure to reminiscent stimuli over extended periods of time, which is less characteristic of classic PTSD (Famularo et al., 1990; Famularo et al., 1996; Foy, Madvig, Pynoos, Camilleri, 1996; Kiser et al., 1991; Terr, 1991).

Another significant element of C-PTSD symptoms resulting from child maltreatment is the effect of human accountability. When the perpetrator of abuse is a caregiver or an authority figure close to the child, fragile trust is violated. This can result
in long-lasting negative changes in personality, making it difficult for the child to develop meaningful and trusting relationships (Herman, 1992).

The symptomatic and functional impairment of PTSD can manifest as chronic problems with relationships, regulation of self, and consciousness. PTSD symptoms are most often present in children and adults who have experienced extreme traumatization (e.g. child abuse) during critical developmental stages (Ford & Kidd, 1998; Herman, 1992). In reference to the trajectory of symptoms that result from extreme child abuse, a debate is currently taking place. Researchers are questioning whether the complicated symptoms children experience, termed Complex PTSD or Disorder of Extreme Stress Not Otherwise Specified (DESOS; Herman, 1992), represent a distinct entity separate from PTSD or whether the symptoms are a form of C-PTSD that specifically develops from child maltreatment, and interferes with the psychosocial development of young children. Further research is needed in this area, as preliminary field trial subjects with DESOS also met criteria for classic PTSD (Herman, 1992).

**Comorbidity**

It is common for traumatized children to exhibit symptoms that overlap with other disorders (Famularo, Kinscherff, & Fenton, 1992; Kiser et al., 1991; March, 1999; McClosky & Walker, 2000; McLeer, Callaghan, Henry, & Wallen, 1994; Saigh, Yasik, Sack, & Koplewicz, 1999), such as the child who looks depressed or inattentive due to lack of sleep. Comorbid symptoms may be due to PTSD itself or independently due to the stressor, so the assessment of PTSD should include a systematic search for accompanying comorbidities. Disorders that commonly co-occur in children who have been traumatized include internalizing and externalizing behaviors, attention deficit
hyperactivity disorder (ADHD), oppositional defiant disorder, conduct disorder, and enuresis (Famularo, Fenton, Kinscherff, Ayoub, & Barnum, 1994; Fletcher, 1996; McLeer, Dixon, Henry, Ruggerio, Escovitz, Niedda, & Scholle, 1998; McNally, 1996). There are some data that suggest that boys are more likely to exhibit externalizing reactions to trauma, even conduct problems, whereas girls are more likely to exhibit internalizing responses. Results, however, are mixed and more research is needed.

Anxiety symptoms are common among children who have been maltreated, in particular separation anxiety, trait anxiety, phobias, and panic (McLeer, et al., 1998; Wolfe, 1989; Wolfe, Sas, & Wekerle, 1994). Thus, anxiety disorders are commonly comorbid with C-PTSD. This is not surprising, given that PTSD is classified in DSM-IV as an anxiety disorder, with similar hypothesized predispositions for reactions to stress (Jones & Barlow, 1990; Perry, 1995).

Depressive conditions, ranging from demoralization and major depression to obsessive-compulsive disorder (OCD) are also common (Wolfe, Sas, & Wekerle, 1994). Famularo, Fenton, Kinscherff, and Augustyn (1996; Jones & Barlow, 1990) reported that children diagnosed with PTSD frequently demonstrate concurrent anxiety disorders, major depression and dysthymia. McLeer et al. (1998) reported rates of comorbid depression and dysthymia at 14% and 10% respectively, while other studies have reported similar rates for comorbid affective disorders ranging from 12-17 % (Dykman, McPherson, Ackerman, Newton, Mooney, Wherry, & Chaffin, 1997; Famularo et al., 1992; McLeer et al., 1998; Merry & Andrews, 1994; Wolfe & McEachran, 1997).

Famularo et al. (1996) also noted that a significant number of subjects in their study additionally manifested symptoms of brief psychotic disorder or psychotic
disorder NOS. This suggests that reality testing disturbances in C-PTSD may be relatively brief and reminiscent of the trauma, with less symptoms of paranoia, illogicality, flat affect, or bizarre delusions. While psychotic states may be brief, dissociation is commonly comorbid with PTSD, and can become part of an individual's personality and coping style.

Early psychoanalytic theorists such as Janet and Kardiner understood that dissociative processes were fundamental to the trauma experience. Research has shown that dissociation at the time of the trauma is one of the strongest predictors for the development of chronic PTSD. It is a way of organizing information and can occur at the time of the events or after the event as a chronic consequent of traumatic exposure. Dissociation reflects a compartmentalization of information that the individual is unable to integrate into their normal personality state. A study by Saxe, van der Kolk, Hall, Schwartz, Chinman, Hall, Lieberg, and Berkowitz (1993) indicated that 15% of psychiatric inpatients experience a dissociative disorder, invariably connected to childhood histories of sexual abuse.

An increase in suicidal ideation has also been noted (Famularo et al., 1996) among children with C-PTSD whose functioning is complicated by the presence of comorbid psychiatric disorders. Examples include reality distortions associated with Psychotic Disorder NOS or dissociative disorders, impulsivity or low frustration thresholds associated with ADHD, or the sense of hopelessness and despair that accompany affective disorders. When functioning becomes overwhelmingly burdensome or hopeless, suicide may present as a welcome, though maladaptive, relief.
Given the range of comorbid symptoms that are often present in children who have had traumatic exposure, the need for thorough assessment is clear. To more fully understand independent or artifact disorders a child may be experiencing following trauma, typology can be useful.

Type I, Type II Trauma Model

As discussed previously with symptom manifestation, Terr (1991) proposed a model of C-PTSD that suggests a different symptom trajectory, depending on the type of trauma the child has endured. Type I trauma (acute) is characterized as sudden, unpredictable, and single-event in nature. This type of trauma is caused by a nonabusive stressor that happens only once, such as a flood, fire, or transportation accident. Type II trauma is characterized as a chronic or abusive stressor, which includes ongoing or multiple stressors such as chronic illness, repeated surgeries, and war. It also includes incidents of physical and/or sexual abuse, whether the abuse is a single or repeated experience.

Several researchers suggest that the DSM-IV framework is currently less than adequate for evaluating childhood-onset PTSD, as it does not emphasize developmental differences and social contextual factors of the individual (Herman, 1992; March, 1999; Scheeringa, Zeanah, Drell, Larrieu, 1995). To understand the symptom trajectory and debilitating effects of C-PTSD, which can affect so many levels of a child's development, several etiological theories may be considered.
CHAPTER 2

REVIEW OF RELATED LITERATURE

Etiological Theories of Posttraumatic Stress Disorder

Psychodynamic Model

As early as 1922, Freud observed that traumatized persons repeat traumatic events and experience repetition of traumatic memories in recurring nightmares (Freud, 1922/1950). He explained the traumatic neuroses of his patients using an etiological model of posttraumatic symptoms, stating that they were due to "excitations from outside which are powerful enough to break through the protective shield (p.35)" of the internal stimuli-receiving cells in the cortex. Freud believed the breaking of the stimulus barrier disrupts the organism's functioning, but removal of the external stressor was expected to allow quick restoration of functioning. Freud did acknowledge, however, that unmanageable stimuli could overpower the organism's coping mechanisms, leading to an overwhelming sense of helplessness. While feeling helpless, the organism regresses and resorts to primitive defenses and repetitive compensatory behaviors to gain mastery over the stressor-event through dreams, memories, and reenactments. Enduring posttraumatic symptoms were explained by Freud as current stress that has revived infantile conflicts. Thus, enduring traumatic reactions were ascribed to the victim as premorbid characteristics, rather than threatening external stressor characteristics.
Sandor Ferenczi (1929) was a contemporary of Freud who elaborated on the effects of interpersonal violence and victimization on children. Ferenczi posited that a child who is sexually abused feels so helpless, vulnerable, and needing of affection that he or she develops "identification with the aggressor" to defend against the trauma of their victimization. The consideration of a child's response to victimization for the sexual gratification of an adult did not sit well with the psychoanalytic community of the time. This concept was considered too awful and embarrassing to consider in public (van der Kolk, Weisaeth, & van der Hart, 1996), resulting in a total absence of research concerning the traumatic experiences and responses of children until the latter part of the century.

While there was a lack of research concerning PTSD in children, evaluations of stress responses and models of trauma for war veterans and survivors of concentration camps received more attention through the 20th century. Abram Kardiner, who studied under Freud, began his career in 1923 treating traumatized U.S. war veterans. He developed a theory of psychological trauma called the "Traumatic Neuroses of War" (Kardiner, 1941 as cited in van der Kolk et al., 1996), which included symptom descriptions of hysteria, malingering, and "epileptiform" disorders. Kardiner's description of traumatic neuroses detailed the vigilance for and sensitivity to threats in the environment, as well as behavior that manifested as if the original traumatic situation were still in existence. Many of Kardiner's concepts concerning traumatic stress continue to be incorporated into current models of PTSD.

There was a lapse in research and treatment of adult traumatic stress between World War II and the Vietnam conflict. The 1970s brought renewed recognition of the
traumatic stress Vietnam war veterans were experiencing, spurring new vigor for research in this area. It was not until the end of the 1970s that symptoms of traumatized women (e.g. rape trauma syndrome, Burgess & Holstrom, 1974 as cited in van der Kolk et al., 1996) and children (Terr, 1979, 1983) were investigated.

Stress-Response Syndrome

Drawing from the early psychoanalytic theories of traumatic stress, Horowitz (1986) proposed a model of PTSD that continues in the psychodynamic arena, adding cognitive and information processing components. Using the Stress-Response Syndrome, Horowitz described traumatized patients as "striving to metabolize their pathogenic memories through alternating phases of engagement (during which they work at assimilating and accommodating these memories) and withdrawal (an adaptive response to pain generated during the engagement phase)" (Young, 2000 p. 53).

In Horowitz's model of PTSD, the individual is unable to successfully integrate the traumatic event into existing cognitive schemata. Thus, the imposing schema changes that are required cannot be integrated quickly, and reflect normal stress response tendencies gone astray. The individual's coping mechanisms become overwhelmed by active memory representations of the traumatic event, leading to an inhibition of the regulatory system to allow more time for traumatic information assimilation. Intrusive reexperiencing (e.g. flashbacks and nightmares) reflects an inhibitory response that is not strong enough. If the inhibitory responses are overly strong, an avoidance or numbing phase will occur to defend against or ameliorate intrusive thoughts. Thus, the individual is moved alternately through intrusion and avoidance phases as denial reduces anxiety and slows cognitive processes, resulting in intrusion when the denial defenses erode.
Horowitz also recognized the role of neurotransmitter systems (dopamine and norepinephrine), positing that the arousal systems become altered as a response to traumatic events, sending false alarms that signal the intrusion phase to activate.

Horowitz also implicated positive social support networks as a potential buffer against development of PTSD. If these cohesive support networks are not in place, pathological stress responses can decrease endurance, thus leading to PTSD symptomatology.

Though there are some oversights in this model (e.g., failure to incorporate control and coping perceptions, and the differential development of PTSD symptoms in individuals exposed to the same stressor), Horowitz addresses development, maintenance, and delayed onset of symptoms fairly well. It has been suggested that this model contributed to the development of the original DSM-III diagnostic criteria for PTSD (Young, 2000).

Neurobiological Framework

Based on the animal model of inescapable and/or unavoidable shock, van der Kolk, Boyd, Krystal, and Greenburg (1984) proposed a biological model of the etiology of PTSD. They posited that neurotransmitter activity leads to behavioral changes in three significant ways. First, "exposure to inescapable shock increases norepinephrine turnover, increases plasma catecholamine levels, depletes brain norepinephrine, and increase 3-methoxy-4-hydroxyphenylglycol (MHPG) production...In addition, brain dopamine and serotonin are decreased, and acetylcholine is increased" (p.126). These changes in neurotransmitter function are believed to result in exaggerated and chronic noradrenergic activity, giving rise to increased startle responses and aggressive behavior.

Second, a conditioned reaction of endogenous opioids is said to mediate a stress-induced analgesic response after reexposure to inescapable shock, providing the individual with
an illusion of control. Third, in response to the invoked opioid responses, the victim becomes "addicted" to the trauma, thus voluntarily placing himself or herself in situations reminiscent of the original traumatic event. Subsequent research supports the role of neurotransmitters in development and maintenance of PTSD, but suggests it is not as clear cut as van der Kolk et al. (1984) implied. This model also fails to account for the delayed onset of symptoms that some victims experience.

Another neurobiological model that implicates the role of neurochemical activity following traumatic events was proposed by Kolb (1987). This model suggests that, in addition to changes in neurotransmitter activity, neuronal pathways may change, which has particular implications for children. Kolb posits that because children do not have fully developed neuronal pathways, they are less able to accommodate the neurochemical reactions to traumatization. Consequently, even if symptoms from the original trauma or disorder remit, permanent changes may have occurred that render the individual vulnerable to disordered arousal and affect (McFarlane & Yeduha, 1996).

Another neurobiological framework of PTSD in children was proposed by Perry in 1995 (cited in Amaya-Jackson et al., 1995). Following the work of van der Kolk et al. (1984), Kolb (1987), and others, this model suggested that prolonged "alarm reactions" create abnormal patterns in catecholamine activity, resulting in altered development of the central nervous system. Consequently, dysregulation of the cardiovascular system, behavioral impulsivity, and affective lability can develop as well as sleep abnormalities, anxiety, and increased startle responses. Perry further projected that C-PTSD can be considered a developmental disorder, as it renders the child’s developing brain vulnerable to abnormal neurotransmitter and hormone transmission.
After reviewing the psychobiological literature, Kaplan, Pelcovitz, and Labruna (1999) provided support for Perry's model. They concluded that child maltreatment, particularly when chronic and traumatic, can be implicated in decreased hippocampal size, memory impairment, frontotemporal and anterior brain electrophysiological abnormalities, altered brain development, delayed growth, impaired sleep, and significantly lowered pain threshold levels.

Behavioral Model

Kirkpatrick, Veronen and Best (1985) used an elaboration of Mowrer's Two-Factor Conditioning Model (1939) to posit a dual conditioning learning theory model of PTSD. They defined PTSD as a stimulus-driven anxiety disorder influenced by classical and instrumental conditioning. Stressor events (unconditioned stimulus) are said to elicit extreme fear and the perception of helplessness (unconditioned response) in the child. "Traumatic reminders" of the event (conditioned stimulus) are formed through the cognitive, affective, physiological and environmental cues that are present during the traumatic event. These reminders can then become a conditioned response through stimulus generalization, manifesting in the form of PTSD symptoms. Stimulus generalization is also fostered by the trial and error of the child's attempts to reduce PTSD symptoms by way of cognitive and behavioral avoidance or anxiety-reducing rituals. In a separate but parallel study, Keane, Fairbank, and Caddel (1985) found very similar results.

Cognitive-Behavioral Model

The cognitive-behavioral perspective, as defined by Foa, Steketee, and Rothbaum in 1989 (cited in Amaya-Jackson et al., 1995), recognizes that an individual's
response to trauma is influenced by perceived (subjective) and actual (objective) threat. This model posits that children with PTSD develop "fear structures" that become conditioned through the pairing of the event and the PTSD symptomatology. Internal and external cues that are reminiscent of the initial traumatic event activate the fear structures by way of verbal, somatic and behavioral mediums. The combination of these reminiscent cues can thus determine the child's meaning of whether an event is indeed traumatic. As basic beliefs become questioned after a stressful event (e.g., a rape victim may lose her sense of the world as safe and just), alterations in meaning influence whether or not the event is considered "traumatic." Persistent PTSD is attributed to transformed representations of self and the world, assigning danger as the default interpretation.

Davidson and Foa (1993) revised this theory to include a severity threshold. They posited that certain extreme traumatic events are above the severity threshold, thus inducing PTSD in most individuals who experience them. Lower magnitude stressors (below the severity threshold), considered minimally stressful to most people, may have an additive effect, given a certain combination of trauma severity and predisposition. Development of PTSD is thus mediated by both internal and external variables.

As PTSD is classified as an anxiety disorder, the model of Jones and Barlow (1990) offers a heuristic for PTSD development based on elementary components of anxiety in behavioral, cognitive, and physiological response systems. This model combines data available from diverse research, suggesting that PTSD develops out of complex interactions between psychological and biological predispositions, exposure to a stressful event and accompanying internal alarms, anxiety, and the coping strategies
and social support available to the individual. Jones and Barlow further explained the anxiety connection, illustrating the phenomenological similarities between Panic Disorder (PD) and PTSD such as the presence of anxiety symptoms, discrete fear responses in the form of emotional alarms, and linking of alarms to various internal and external stimuli. Both disorders also have the common feature of avoidance of affect-related stimuli, which elicit alarms such as flashbacks or panic attacks. Thus, the combination of traumatic events and emotional alarms (true or false), coupled with chronic emotional anxiety or distress and accompanying distortions of information processing, present an individual with sufficient symptoms for a disorder. Additionally, the experienced anxiety is focused on anxious apprehension of reexperiencing the intense affect that occurs with alarms; thus, hypervigilence and narrowed attention actually ensure reexperiencing of emotion-laden material. Hence, in the search for a more thorough understanding of the etiology of PTSD, it may be useful to research hypothesized etiological variables of anxiety disorders to discover underlying mechanisms of PTSD.

Risk versus Resilience

The impending question confronting PTSD researchers asks why only some survivors of traumatic events develop PTSD, while others do not, even given similar traumatic events? The factors that contribute to the transition from health to disorder and then recovery are key to understanding the longitudinal course of PTSD. While the interplay of a complex matrix of biological, social, psychological and temperamental factors determines expression of symptoms and adaptive ability, certain factors
contribute more to vulnerability and other factors to resilience. Ultimately, the ability to tolerate suffering may be a relevant determinant of long-term adaptation.

*Risk Factors of Posttraumatic Stress Disorder*

There are challenges associated with the discovery, disclosure, validation, and study of physical and sexual abuse of children, as many of these traumatic events occur in secret. Additionally, it is often difficult for children to verbalize or relate their experiences due to the private and specific fears that may develop concerning aspects of the abuse, as well as language deficits due to a child's young age (Wekerle & Wolfe, 1996). Recent development of diagnostic measures for PTSD—appropriate for use with children and adolescents—will surely help clinicians and researchers further isolate factors that moderate and mediate the impact and reactions children experience as a result of child maltreatment. Despite the challenges involved, there have been some significant discoveries in the identification of precipitating factors of PTSD.

**Demographic Variables**

*Gender, Age, Ethnicity, Intelligence.* Several researchers have found differences in gender among traumatized children. Girls are more likely to experience trauma and are more likely to report their victimization, placing them at higher risk for developing PTSD (Breslau, Davis, Andreski, & Peterson, 1991; Davis & Siegel, 2000; Wolfe & Mosk, 1983; Wolfe, Sas, & Wekerle, 1994). However, other studies have suggested that, while there is a slight increase in reported sexual victimization of girls, physical maltreatment was reported with approximately equal frequency in boys and girls (Fitzpatrick & Boldizar, 1993; Powers, Eckenrode, & Jaklitsch, 1990). Kiser, Ackerman, Brown, Edwards, McColgan, Puch, & Pruitt (1988) described a gender difference in
reactions to stress, as girls were more likely to exhibit internalizing behaviors and boys more likely to exhibit externalizing behaviors following traumatization (Jaffe, Wolfe, Wilson, & Zak, 1986.)

Although symptom manifestation of PTSD in children may differ depending on level of development and age of the child, risk for developing C-PTSD after a traumatic stressor has also yielded mixed results. Studies of refugee children in the United Kingdom exposed to the traumas of war (including physical and sexual maltreatment) indicate that young children may be protected from traumatic events due to cognitive immaturity, particularly if they are shielded by their parents (Hodes, 2000). Wolfe, Sas, and Wekerle (1994) found higher rates of PTSD among sexual abuse victims above the age of 12 years than younger children. In contrast, nonsignificant differences in distribution by age of children with PTSD have been found in numerous studies (Fitzpatrick & Boldizar, 1993; Livingston, Lawson, & Jones, 1993; Nader, Pynoos, Fairbanks, & Frederick, 1990; Pynoos, Frederick, Nader, Arroyo, Steinberg, Eth, Nunez & Fairbanks, 1987).

Studies of ethnicity have also yielded mixed results. LaGreca, Silverman, Vernberg, and Prinstein (1996) reported significantly higher rates of PTSD in Hispanic and black youth, whereas Shannon, Lonigan, Finch, and Taylor (1994) reported nonsignificant differences regarding race. While intelligence has been grossly underreported in the literature, data suggest that higher intelligence may mitigate traumatic stress effects (Fletcher, 1996); however, more research is needed.

Socioeconomic Status. Socioeconomic status (SES) can be considered a risk factor (Foy et al., 1996), as children from low SES families are more likely to experience
interpersonal conflict (stress of their environment) and more likely to witness or become
a victim of community violence. Children who come from an abusive home
environment, and those who are exposed to crime, have increased risk of PTSD
(McClosky & Walker, 2000).

Severity and Duration of Abuse. Exposure severity and longer duration of child
maltreatment are additional etiological variables that consistently show a positive
suggested a positive relationship between severity of the abuse and the number of
perpetrators with PTSD after physical abuse, and duration of abuse with PTSD after
sexual abuse. Length of time post-trauma has shown to have a negative relationship with
PTSD symptoms, indicating that, for many children, symptoms will decrease over time.
However, waning of symptoms can be dependent on the type of abuse (Foy et al., 1996;
Wolfe et al., 1994). Results of a clinical study of women who experienced incest in
childhood indicated that PTSD symptoms had been particularly pervasive and enduring
in most subjects, even as long as 17 years following the trauma (Famularo et al., 1996).

Perpetrator Characteristics. One factor that is consistently supported as a risk
factor for PTSD is violation of trust by a caregiver. PTSD is more common in children
who were maltreated by parents, adult caregivers, or adult strangers as compared to
victimization by another child or a sibling (Browne & Finkelhor, 1986; McLeer,
Deblinger, Atkings, Foa, & Ralphe, 1988). In the case of sexual and/or physical
maltreatment, there is a betrayal of trust and physical violence, often accompanied by
coercion or deceit. The perpetrator's attempts to coerce, threaten, confuse or even
console the child can interfere with the child's efforts to disclose the abuse.
unpredictability of the event, and a feeling that it is beyond the victim's control, elicits the use of coping reactions that may generalize to a global perception of the world as unsafe and unloving, as previously safe or supportive people and places become associated with fear and danger. As the child searches for meaning of this change, he or she may experience self-blame or guilt that they did not exercise enough control to stop the abuse, further hampering recovery from the trauma (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989; Wolfe, Sas, & Wekerle, 1994).

Type I vs. Type II Trauma. As explained earlier, type I trauma is an acute or single event-based trauma (e.g., natural disasters or car accidents), whereas type II trauma is chronic or ongoing (e.g., physical or sexual abuse). Although both types can result in PTSD symptoms, children exposed to type II trauma often experience a longer duration of symptoms and more character changes and severe overall problems (e.g., dissociation, eating disorders, social deficits, substance abuse; Davis & Siegel, 2000; Fletcher, 1996; Green, 1993; McClosky & Walker, 2000; Terr, 1991). Green characterized long-term abuse as "the repeated infliction of sexual and aggressive stimulation superimposed on a chronic background of pathological family interaction, including stigmatization, betrayal, role reversal, sexualization, and a breaching of the child's physical and psychological boundaries (Green, 1993, pp. 897).

Type of Exposure (experienced, witnessed, indirect exposure). While it is clear that C-PTSD develops from experienced maltreatment and witnessed interpersonal violence, posttraumatic stress symptoms may also develop through indirect exposure from a near-miss experience. A child who left the Chowchilla bus before it was kidnapped also developed PTSD symptoms (Terr, 1990). Contagion effects in the
community, detailed media coverage, or retelling of the death or injury of a loved one by family members can also precipitate or exacerbate PTSD symptoms in children (Amaya-Jackson et al., 1995).

Multiple Forms of Abuse. The children who were both a witness to violence and also a target of the same abusive event seem to carry the highest PTSD risk (Livingston et al., 1993; Pynoos & Nader, 1989). Witnessing abuse of their mothers was significantly distressing and traumatic for very young children who were confused about the event and unable to process it (McNally, 1996; Pynoos & Nader 1989). Particularly distressing was threatened loss of a loved one, likely due to the high dependency needs of childhood (McClosky & Walker, 2000).

McClosky and Walker (2000) also reported that children who were both a target and witness met PTSD criteria 100% of the time. Children who have witnessed spouse abuse, experienced child abuse, or both, have been shown to be at increased risk for socioemotional, behavioral, and academic difficulties, including PTSD symptomatology (Trickett, 1998). Shipmann, Rossman and West (1999) supported previous findings that children who have been exposed to multiple forms of maltreatment (including witnessing parental violence) are at increased risk for emotional dysregulation, PTSD development, and a decreased capacity to calm themselves when distressed. Clearly there is increased trauma when a child’s family is threatened or is threatening.

Victim Characteristics

Affect Regulation. Affect regulation has been defined as "the intra- and extraorganismic factors by which emotional arousal is redirected, controlled, modulated, and modified so that an individual can function adaptively in emotionally challenging
situations" (Cicchetti & Toth, 1997, p. 325). It is during early parent-child interactions that children learn emotional competency. However, maltreated children demonstrate numerous deficits in their emotional-regulation abilities. Additionally, maltreated children evidence distortions in their initial patterns of affect differentiation, either displaying excessive negative affect or blunted patterns of affect (Cicchetti, 1991; Cicchetti & Toth, 1997). These aberrant patterns of affect differentiation reveal problems in the processing and modulating of physiological arousal, hindering an ability to make rational assessments of stressful or ambiguous situations.

Temperament. Dykman et al., (1997) reported that a child's premorbid temperament can predict risk. Children with "sensitive" nervous systems (e.g., increased sensitivity to loud noise, increased startle response) were more vulnerable to PTSD following abuse. Jones and Barlow (1990) further postulated that there may be a genetic component that predisposes an individual to diffuse stress responsivity, which manifests as chronic autonomic overarousal and noradrenergic lability. It has also been suggested that children who are less consolable, have more difficult temperaments, or were more difficult infants may have an increased risk for maladaptive coping and more vulnerability to developing PTSD after traumatic stress (Wertlieb, Weigel, Spinger, & Feldshein, 1987; Wyman, Cowen, Work, & Parker, 1991).

Locus of Control. The way a child comes to view her or his victimization, as well as their own personal characteristics and locus of control, can greatly influence their risk of PTSD. Lack of personal efficacy has been associated with chronic PTSD symptoms, suggesting that a child's locus of control is instrumental in the induction and/or maintenance of PTSD (Amaya-Jackson & March, 1995; March, 1999). A child with an
external locus of control feels that they have no control over their environment, which can contribute to feelings of guilt concerning the abuse (Jones & Barlow, 1990).

Wolfe et al. (1994) studied child victims of sexual abuse, reporting that children with PTSD self-reported more abuse-related fears, anxiety, depression and feelings of guilt than children without PTSD. The no-PTSD children were more likely to report self-blame and exhibited more externalizing behaviors. Perceived threat and self-blame influence a child’s coping responses, and their development of a success-based orientation (e.g. degree of control). The success-based orientation can thus be thwarted by maltreatment, particularly when the abuse takes place in a usually safe place, the perpetrator is an adult they know or trust, or when they are violated within their usual (safe) environment and routines. Children whose feeling of safety has been violated are particularly vulnerable to attribution formation of self-blame and guilt (Kiser et al., 1991; Wolfe et al., 1994). The child’s locus of control can be seen in the initial response to trauma, which contributes to the meaning they ascribe to the event and to themselves following the event.

*Initial Trauma Reaction.* It has been hypothesized that the immediate physiological reaction a child has to crisis is a crucial link to acute distress, which is shaped by concurrent mediating variables (precipitating or protective) from psychological, biological and social domains (Foy, Osato, Houskamp, & Neumann, 1992; Foy, et al., 1996). Jones and Barlow (1990) suggested that some victims of PTSD may have a genetic predisposition to hyperarousal and a higher resting heart rate, as observed in subjects with panic disorder. Increased hyperarousal may exacerbate the..
child’s reactions to stress, increasing fear and helplessness, particularly when previously
safe and supportive persons or places become associated with danger and fear.

Perceived control appears to be another important aspect of the initial response,
as children who experience the use of threat or force are significantly more likely to
meet PTSD criteria (Wolfe et al., 1994). Investigations of psychophysiological
vulnerability suggests that lack of experience with control can contribute to feeling
helpless and even change levels of various neurochemicals that have been correlated
with PTSD symptoms, such as hyperarousal. Jones and Barlow (1990) suggested that a
person’s coping strategy reflects their perceived control. They reported that non-avoidant
or problem-focused coping strategies are used by people who feel they have some
control in life, and are more adaptive in the long run than emotion-focused and
avoidance strategies. Individuals who use emotion-focused and avoidant strategies may
view their traumatic events as uncontrollable, which render them less amenable to
problem-focused strategies. These studies also suggest that children with prior histories
of emotional problems may be more vulnerable to long-term adjustment problems than
children exposed to trauma that do not have such histories (Jones & Barlow, 1990; van
der Kolk, 1989).

Several researchers have reported disturbances in memory and processing during
the traumatic event, and indicate that there is a linear relationship between trauma
exposure and risk for C-PTSD (Foy et al., 1996; March, 1999; Pynoos & Nader, 1989;
Saigh et al., 1999). Deficits in short-term memory and disturbances in dissociative
memory can occur, particularly if physical coercion is used. Children may also omit
moments of extreme life threat or duration from memory, or may distort proximity to
minimize their life threat (March, 1999). These findings, in part, explain why some children develop PTSD following lower levels of traumatic exposure and others do not develop PTSD following severe trauma.

Foy et al. (1992, 1996), and Shipman, Rossman, and West (1999) have also suggested that "independent causes" of distress can act in an additive fashion to increase PTSD risk; however, additional research in this area has yielded mixed results. Hyperactivity and concerns over school achievement have also been reported to be significantly discriminative of PTSD in a sample of abused children (Kiser et al., 1991).

Coping Skills and Experience with Success. Though the initial response to a traumatic event can frame how a child ascribes meaning to the event, coping behaviors can also affect the meaning and the trajectory of symptoms the child experiences after maltreatment. Wertlieb et al. (1987) distinguished between children's coping behaviors that 1) focus on self, environment, or others, 2) serve to solve problems or manage emotions, and 3) engage one of the following: support seeking, information seeking, direct action, inhibition of action, or intrapsychic coping. Coping strategies/styles are thought to change depending on the developmental stage of the child, such as decreased use of self-calming behaviors at younger ages and decreased distraction and avoidance (e.g., read a book) at older ages. There is also a decrease in anger use among girls with age, but boys increase anger use from ages 10 to 12 years (Rossman, 1992).

More important to the moderation of the effects of trauma than the type of coping is successful coping. Coping that is effective relieves distress, allows the maintenance of self-worth feelings, sustains the ability to form reinforcing social contacts, and provides the capacity to meet task demands. However, success is dependent on coping efforts that
match the circumstances of the traumatic event and the resources available to the child (Shalev, 1996). For example, passive surrender, acceptance, and cognitive reframing may be most appropriate when the situation is uncontrollable or inescapable, whereas seeking help or avoiding threatening situations may be most adaptive in other circumstances. Achieving individual coping goals results in decreased distress and an increased sense of control and mastery.

Conversely, when coping behavior is not available or is not effective, changes may be set in motion that induce vulnerability to disorders such as PTSD. Studies on the behavioral, biochemical, and pathological sequelae of inescapable stress in animals and humans have suggested that uncontrollable stress exposure can have debilitating effects. These include interference with subsequent learning (Maier & Seligman, 1976), immunosuppression (Laudenslager, Ryan, Drugan, Hyson, & Maier, 1983), opiate stress-induced analgesia (Drugan, Ader, & Maier, 1985), and gastric ulceration (Weiss, 1971). It has also been reported that many of the pathological effects of stress can be ameliorated when the subject is allowed to alter the pattern, onset, duration, or intensity of stress by active escape behavior (coping; Maier & Seligman, 1976; Weiss, 1971), which fosters a feeling of experience with control and success.

Previous Negative Life Events. Studies with adults have reported a correlation between negative life changes and increased traumatization (Ruch, Chandler, & Harter, 1980). Similarly, researchers and policy advocates interested in understanding the effects of trauma and negative life events on children have examined areas such as domestic and child maltreatment, community violence, war, parental divorce, parental psychopathology, and substance abuse. In light of the breadth of areas involved, an
ecological/transactional framework has proven useful for assessing how the confluence of these experiences can exert a negative impact on a child's development. Studies by Cicchetti, Toth, & Hennessy (1993) and Cicchetti and Toth (1997) have shown that culture, community, and family, combined with premorbid adaptation attained by a child, conjoins to influence child developmental outcomes. Over the course of development, vulnerability stressors such as divorce, chronic poverty, community violence, and unsupportive social networks can compromise competent and successful adaptation to stress and promote a pathological organization of the developmental systems (biological, emotional, cognitive, linguistic, interpersonal, and representational). These studies also suggested that the immediate family context is vital in predicting how a child will cope with adversity. If a child's home environment has not sufficiently and positively contributed to effective coping and adaptation prior to a trauma, the child's ability to function adaptively will be compromised when faced with a traumatic event.

Family Characteristics.

Shipman et al. (1999) reported that the family factors that best predict PTSD development include lower SES, high numbers of family stressors, poor maternal mental health, and neighborhood violence.

Family Functioning. Maltreating families can have disruptions in many, if not all, aspects of family relationships. Maltreating parents are less interactive and nurturing with their children, and display more negative affect toward their children than nonmaltreating parents. Additionally, husbands and wives in maltreating families are less warm and supportive, less satisfied with their marriages, and more aggressive toward their spouses than parents in nonabusive families. Overall, anger and conflict are
pervasive in maltreating families; thus, profound family dysfunction can exacerbate the experience of child maltreatment, rendering the child less effective in coping with their traumatic experiences (Cicchetti & Toth, 1997; Trickett & Susman, 1988).

Shipman et al. (1999) also indicated that children exposed to more than one type of family violence (e.g., child abuse, spouse abuse) exhibited more behavioral and socioemotional problems than their nonexposed peers. Children from violent families had significantly greater difficulty regulating their emotional experiences. These children also had poorer academic performance, suggesting that child maltreatment can interfere with cognitive development as well as socioemotional and behavioral regulation. Children from violent homes were reported at greater risk for other adaptational failures such as child psychopathology and poor peer relations (Famularo et al., 1990; Kiser et al., 1991; Shipman et al., 1999). Another surprising factor of PTSD risk identified in the Shipman et al. (1999) study was greater frequency of physical punishment experienced by the father during childhood. The full ramifications of this factor are not clear, except that fathers who experienced more physical punishment were also more likely to use greater physical punishment with their own children. It is likely that modeling parents is a key element of this factor. Parent psychopathology and a family history of psychiatric illness has also been reported as a significant risk factor for C-PTSD (Breslau & Davis, 1992; McFarlane, 1992).

**Maternal Adjustment.** Research has identified the effects family functioning can have on a child’s risk for PTSD development. However, the mental health of mothers appears to be particularly predictive of the child’s adjustment following child maltreatment (Foy et al., 1996). Famularo et al. (1996) reported that 36% of children
whose mothers had PTSD also met criteria for PTSD. Sexually abused children who were able to report and receive emotional support from their mother were less symptomatic than children who did not receive support. Conversely, children whose mothers reacted negatively to their report of abuse experienced more severe problems (Deblinger, Steer, & Lippmann, 1999).

Additional findings suggest that the mother’s level of depression is positively related to the child’s total PTSD symptoms, as well as mother-reported increase in the behavior problems of their children. Depressed mothers are less engaged and involved with their children, and use more forceful control strategies or avoid conflict with children by accommodating child demands (Cicchetti & Toth, 1997). It is not completely clear whether depressed mothers are less available emotionally to their children, thus leading to increased child symptomatology, or if depressed parents have less tolerance and energy, leading them to the perception that their child is more symptomatic. Spouse abuse has also been indicated in a decrease of maternal availability and comfort (Shipman et al., 1999), and a parent’s response to a shared trauma can be a strong mediating factor (positively or negatively) of the child’s PTSD symptom development (Foy et al., 1996).

**Parenting Style and Discipline.** Parenting style is another family factor that has been shown to be a contributor to children’s PTSD symptomatology and levels of child-reported depression. Children who experience their mother’s parenting style as hostile and rejecting were more likely to have a negative self-view and were consequently more predisposed to depression (Deblinger et al., 1999). Alternately, it is also possible that children with elevated levels of depression experience parental responses as more
negative than non-depressed children. Deblinger and colleagues (1999) reported that mothers' parenting methods that were more intrusive and controlling, rather than encouraging of autonomy, contributed unique variance to the child's PTSD symptoms. Maternal use of guilt and anxiety-provoking parenting strategies increased children's feelings of more responsibility and self-blame for the abuse which, in turn, increased symptomatology.

Support Networks. One of the factors that has been identified as having both negative and positive relationships with C-PTSD development is family functioning and support systems. In general, a more cohesive, supportive, and functional family is related to fewer symptoms in traumatized children. On the other hand, a more negative and dysfunctional family, along with poor social support, is related to a greater likelihood of a child developing PTSD following abuse.

Summary of Risk Factors of PTSD

Overall, results are mixed with respect to age, gender, ethnicity, and intelligence, but most studies have not found these variables to be significant markers of PTSD (Famularao et al., 1990; Fletcher, 1996; Foy et al., 1996; Wolfe et al., 1994). Socioeconomic status (SES), severity, and duration of abuse have interactive effects with perpetrator characteristics, suggesting that greater duration and severity of abuse is seen in lower SES environments and that violation of trust by a previously trusted perpetrator increases risk for C-PTSD. There is also an additive effect manifested by Type II (chronic) abuse as well as by multiple types of abuse. Individual characteristics of the child such as temperament, locus of control, affect regulation, initial trauma response, and experience with effective coping also interact to define the meaning and assimilation
of the traumatic event, which can be negatively colored by previous negative life events. Ultimately, family dysfunction, poor parenting practices and limited support networks can have direct influences on all of the above, placing the child at increased risk of maladaptive coping with traumatic abusive experiences.

In essence, risk and vulnerability mechanisms do not cause ineffective adaptation, but are indicators of a complex template of mechanisms and processes that can affect or change the developmental course. Thus, maladaptation and/or disorder are manifested in children that have developed a pathological organization of coping capacities and protective resources over the course of their development. In light of severe trauma such as child maltreatment, this maladaptation can result in many debilitating symptoms, including posttraumatic stress disorder.

**Prophylactic Factors of Posttraumatic Stress Disorder**

As indicated above, many variables have a moderating or mediating effect on the development of PTSD development, such as demographics (e.g., SES), characteristics of the individual child (e.g., locus of control), biology (e.g., temperament, arousal), coping strategies (e.g., emotion-focused), and family functioning (e.g., dysfunction, maternal psychopathology). However, many of these variables can also serve an enduring protective function when they are active in the opposite direction. Of particular protective importance is cohesive family functioning, secure attachment, nurturing parental reactions to reported abuse, positive parenting, and a supportive social network. In addition, family factors such as marital status and stability, psychological health of the parents, the family’s social support network, and the child’s own social support can all
serve a protective function for a child who has been victimized by maltreatment (Amaya-Jackson & March 1995; Jones & Barlow, 1990; Kiser et al., 1991).

**Victim Characteristics**

*Affect Regulation.* Physically abused children have been reported to manifest affect regulatory problems coping with interadult anger. Studies have shown that physically abused boys report more fear in response to angry adult behavior, which leads to greater (not less) emotional reactivity (Rieder & Cicchetti, 1989). Hypervigilance and ready assimilation of angry or aggressive stimuli may originate as an adaptive protective function, serving to alert the child to impending danger. However, hypervigilance and aggressive assimilation becomes less adaptive when used with nonthreatening situations (Cicchetti & Toth, 1997).

*Secure Attachment.* Early infant affect regulation experiences and interactions with the primary caregiver are critical in the development of attachment (Sroufe, 1996). To develop stable, secure attachments with the parent (or caregiver), parent-child dyadic interactions must employ relatedness and synchrony, as well as appropriate affective interchange in a goal-corrected partnership (Bowlby, 1969/1982). By developing representational models of attachment figures, of themselves, and of themselves in relation to others, the affect, cognitions, and expectations of children about future interactions are organized and applied to subsequent relationships (Bowlby, 1969/1982; Sroufe, 1996).

Studies with maltreated children indicate that more than 2/3 have insecure attachments (type A-avoidant, or type C-anxious resistant), while the other 1/3 have secure attachments (type B). These figures are directly opposite those of nonmaltreated
children (Ainsworth, Blehar, Waters, & Wall, 1978). Similar studies show that approximately 80% of maltreated children show disorganized attachments (atypical type D pattern), with the other 20% starting as securely attached but deteriorating into insecure attachments over time as maltreatment continues (Carlson, Cicchetti, Barnett, & Braunwald, 1989). Of particular concern with children who develop disorganized attachment is the devastating long-term impairment they can suffer. This devastating psychological insult is believed to result in long-term psychobiological disorders such as PTSD, dissociative disorder, and multiple personality disorder (Cicchetti, 1991; van der Kolk, 1989).

Resilience and Hardiness. Resilience and hardiness have recently received increased attention in the personality literature, particularly in reference to how personality can influence coping and integration of meaning for a traumatic experience. Flach (1990, as cited in Williams, 1999) defines psychobiological resilience as "the efficient blending of psychological, biological and environmental elements that permits human beings...to transit episodes of chaos necessarily associated with significant periods of stress and change successfully." Thus, resilience is not considered an exclusively internal characteristic, but rather an interactive process. Similarly, hardiness is defined by Lyons (1991, as cited in Williams, p. 105) as a personality characterized by commitment, control, and challenge. "Commitment captures the authentic positive state of caring...Control and challenge assess another major element of the authentic being: courage...recognizing hard facts, the personal belief that one is able to exert control over external and internal events, and attributing to stress the meaning of challenge" (p. 106). In essence, individuals with low hardiness or low resilience are more likely to manifest
higher symptoms of maladaptation, while hardy individuals are those who gain
successful experience in coping with stressors. Such positive coping experiences thus
lead to the acquisition of complex schematic networks that are able to metabolize
trauma, and more complex neuronal structures developed from rich histories of learning,
which allow better stimulus discrimination and restricted generalization of anxiety
(Williams, 1999).

Temperament. Literature on resiliency also suggests a relationship between
temperament and risk for PTSD. Several studies (Werner & Smith. 1982; Wertlieb,
Weigel, Springer, and Feldstein, 1987; Wyman, Cowen, Work, & Parker, 1991) indicate
that resilient children are more likely to be characterized as easygoing, positive in mood,
outgoing, adaptable as infants, and overall better able to adapt to stress.

Family Characteristics

Throughout history, people have organized in groups and communities to help
themselves meet the challenges of the outside world. Developing close emotional
relationships helps people anticipate and negotiate challenges. The emotional
attachments they build provide protection against feelings of meaningless and
helplessness. For children, the primary emotional attachment and protection comes from
their family, which is usually an effective source of nurturance and protection from
traumatic experiences. When the family is not able to provide this protection, the child is
left vulnerable, relying solely on whatever coping strategies and information processing
they have been able to develop. In part, this explains the differences in traumatic stress
reactions among children experiencing similar events.
Parental Reaction to Report. Deblinger and colleagues (1999) reported that emotional support from a non-offending adult, particularly if that adult is the mother, significantly reduced symptoms in sexually abused children. A mother's willingness to believe her child's report of abuse and provide comfort can also reduce symptom development. The mother's willingness to believe the child can, however, be predicated by her relationship and dependence on the perpetrator, particularly if the perpetrator resides in the same home as the mother (Amaya-Jackson & March, 1995; Elliott & Briere, 1994).

Family Support. From an examination of the effects of community violence, Richters and Martinez (1993b) reported that community violence was not specifically predictive of adaptational success or failure. Adaptation was most related to the child's home environment, and rose sharply for children who resided in safe and stable homes. Clearly, caregiver functioning can have a moderating effect on negative life events, and is important for assimilation of adaptation and coping skills.

Social Support. In addition to the protective function stable families provide, a child's social network and school environment can provide protection from maladaptive reactions to trauma. Cicchetti, Toth, and Hennessy (1993) reported that maltreated children who were able to attain support at school had lower adverse sequelae than children who did not attain social support from school.

Summary of Prophylactic Factors of PTSD

Clearly, the longitudinal trajectory of PTSD for a given child who has been maltreated can manifest with great variability of symptoms. Basic foundations from infancy such as secure attachment, affect regulation, and temperament begin a child's
experience with assimilation of new information and use of coping strategies in the face of challenge. Whether these early experiences are successful or not largely depends on the support and care of the child's family, and are likewise influenced by social networks. The core foundations the child develops thus influence how a child is able to ascribe meaning to and assimilate an abusive, traumatic event. The core foundations are particularly important following trauma, as trauma can cause the child to question all previous schemas, coping strategies, and definitions of safety. Clearly there is a multitude of risk variables that can precipitate or augment a maladaptive reaction to trauma. A child who is hardy and resilient, with effective coping strategies and a nurturing, supportive environment is best equipped to metabolize and assimilate an abusive event, thus protecting himself or herself from the debilitating outcomes of maladaptive trauma reactions such as PTSD.

Purpose of the Study

The purpose of this study was to explore victim characteristics, demographic factors, and family characteristics that may interact to interfere with, or protect against, a breakdown in the coping abilities of children who had been physically or sexually abused. The primary goal was to identify stressors, mediators, and buffering agents that predict the development of posttraumatic stress disorder in children who had experienced child maltreatment. One advantage of the proposed study is that a well-standardized diagnostic measure was used to provide sound diagnoses of PTSD and comorbidity.

Previous literature has documented that many children develop posttraumatic symptoms following trauma such as physical or sexual abuse, or witnessing
interpersonal violence. It has also been documented that some children do not develop PTSD following trauma; however, there is a paucity of literature concerning these buffering variables. Therefore, particular emphasis was given in this study to the prophylactic factors that serve a protective or ameliorating function for children that would otherwise be at risk to develop posttraumatic stress symptoms.

Research has also shown that many biological and psychological factors contribute to PTSD in children. Several variables were identified to have either a precipitating/high-risk function or a prophylactic/buffering function leading to PTSD in children who have been maltreated. While many of these variables have been studied in isolation, or as small groups (e.g., age, ethnicity, intelligence and SES), no studies were identified that evaluated both precipitating and prophylactic variables collectively. In addition, there is a paucity of literature concerning the buffering variables which, again, have mostly been studied in isolation (e.g., coping skills or resiliency). The structure of this study was developed to incorporate available literature and to examine both groups of variables simultaneously, anticipating further enlightenment concerning the specific variables that serve a risk or resiliency function for children dealing with the trauma of child maltreatment.

This study adds to the current knowledge base of posttraumatic stress disorder in children who have been maltreated by further illuminating precipitating and prophylactic mechanisms as well as contributing comorbidity.

**Hypotheses**

It was originally hypothesized that the following precipitating factors, including SES, severe and chronic duration of abuse, initial trauma response, violation of trust,
multiple types of abuse, ineffective coping, temperament, locus of control, negative life events, poor parenting, dysfunctional families, and non-supportive networks would lead to increased likelihood of PTSD. It was also hypothesized that prophylactic factors, including affect regulation, secure attachment, resilience and hardiness, effective coping, supportive parental reaction to report, and support networks would serve to ameliorate development of PTSD. It was further hypothesized that the combination of both precipitating and prophylactic factors is what determines a child's ability to effectively metabolize a traumatic event such as child maltreatment.

Due to bureaucratic restraints, it was not possible to collect parent data as originally planned. Data were collected only from children and the study was restructured. The revised hypotheses include the following: 1) precipitating factors, including severe and chronic duration of abuse, affect dysregulation, ineffective coping, external locus of control, non-supportive networks, and dysfunctional families will lead to an increased likelihood of C-PTSD; 2) prophylactic factors, including affect regulation, internal locus of control, resilience and hardiness, effective coping, and supportive networks (including religious participation) will serve to ameliorate development of PTSD; 3) additionally, the combination of precipitating and prophylactic factors will determine how effectively they will be able to accommodate and assimilate a traumatic event such as child maltreatment. If the trauma is too overwhelming for the child, PTSD is likely to emerge as a defense against an experience that is just too horrible to effectively process and accommodate.

Following study revisions, The Anxiety Disorder Interview Schedule for Children-Child Version (ADIS-C), The Children's PTSD Inventory (CPTSDI), When
Bad Things Happen (WBTH), Self-Report Coping Scale (SRCS), Family Environment Scale (FES)—shortened child version, and demographic sheet were retained. Measures removed from the study include ADIS-Parent Version, Alabama Parenting Questionnaire (APQ), Child Behavior Checklist (CBCL), The Child's Upsetting Times Checklist (CUTC), Dimensions of Stressful Events Rating Scale (DOSE), and FES—full version from parent.
Table 1  
Revised Measures Used for Each Variable: Precipitating and Prophylactic

<table>
<thead>
<tr>
<th>PROPHYLACTIC SYMPTOM</th>
<th>MEASURE</th>
<th></th>
<th>PROPHYLACTIC SYMPTOM</th>
<th>MEASURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affect Regulation</td>
<td>WBTH</td>
<td>↑</td>
<td>Resilience</td>
<td>WBTH</td>
</tr>
<tr>
<td></td>
<td>SRCS</td>
<td>↑</td>
<td>Hardiness</td>
<td>FES (IND)</td>
</tr>
<tr>
<td>Locus of Control—internal</td>
<td>WBTH</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>SRCS</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coping Skills—approach</td>
<td>SRCS-</td>
<td>↑</td>
<td>Support Networks</td>
<td>FES—Conflict, Cohesion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PRECIPITATING SYMPTOM</th>
<th>MEASURE</th>
<th></th>
<th>PRECIPITATING SYMPTOM</th>
<th>MEASURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abuse Severity and Duration</td>
<td>CPTSDI</td>
<td>↑</td>
<td>Family Dysfunction</td>
<td>Demo</td>
</tr>
<tr>
<td>Type I, Type II Trauma</td>
<td>ADIS-C</td>
<td>↑</td>
<td></td>
<td>FES</td>
</tr>
<tr>
<td></td>
<td>WBTH</td>
<td>↑</td>
<td></td>
<td>Conflict</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cohesion</td>
</tr>
<tr>
<td>Gender, Age, Ethnicity</td>
<td>Demo</td>
<td>*</td>
<td>Multiple Forms of Abuse</td>
<td>ADIS</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>ADIS-C</td>
<td>↑</td>
<td>Affect Dysregulation</td>
<td>WBTH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SRCS</td>
</tr>
<tr>
<td>Coping Skills—avoidant</td>
<td>SRCS</td>
<td>↑</td>
<td>Locus of Control—external</td>
<td>WBTH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SRCS</td>
</tr>
<tr>
<td>DSM Criteria—Symptoms</td>
<td>ADIS-C</td>
<td>↑</td>
<td>Unstable Support Networks</td>
<td>FES</td>
</tr>
<tr>
<td></td>
<td>WBTH</td>
<td>↑</td>
<td></td>
<td>Conflict</td>
</tr>
<tr>
<td></td>
<td>CPTSDI</td>
<td>↑</td>
<td></td>
<td>Cohesion</td>
</tr>
</tbody>
</table>

* No score obtained; ↑ = elevated scores; ↓ = lowered scores
CHAPTER 3

METHODOLOGY

It was hypothesized in this study that the precipitating factors of severe and chronic duration of abuse, affect dysregulation, ineffective coping, external locus of control, dysfunctional families, and non-supportive networks would lead to increased likelihood of PTSD. Specifically, a high score on each variable would be predictive of a high number of PTSD symptoms. It was also hypothesized that prophylactic factors, including affect regulation, internal locus of control, resilience and hardiness, effective coping, and supportive family and social networks would serve to ameliorate development of PTSD. A low score on these variables is predictive of a low number of PTSD symptoms. It was further hypothesized that the combination of both precipitating and prophylactic factors would predict a child’s ability to effectively metabolize a traumatic event such as child maltreatment.

Participants

The participants for this study were recruited from state and private facilities that provide respite care (e.g., Child Haven in Las Vegas, NV) and/or services to children and families (e.g. Family Service Alliance Center, and Family and Children’s Services in Idaho) who have experienced maltreatment within the past 2 years. Fifty-five children aged 8 to 17 years voluntarily participated in a structured diagnostic interview and were asked to complete several self-report questionnaires. The sample was approximately
33% Caucasian, 33% African-American, 15% Hispanic, and 19% self-reported mixed race.

Demographic analysis indicated that the mean age was 12.65 years. 58% of the sample was female, 60% came from single-parent homes, 78% had a family member who had served time in prison, and 63% reported regular drug/alcohol use in their home. Demographic data also indicated that 31% did not have any religious practice in their home, 27% reported only themselves as religious, and 34% indicated both they and their family were religious. Evaluation of the span of months in which abuse occurred indicated a mean of 48 months for the PTSD group and a mean of 12 months for the NO PTSD group. Children in the PTSD group also had increased incidence of comorbid diagnoses (mean = 3.5) compared to the NO PTSD group (mean = 1.8).

**Child Measures**

*When Bad Things Happen Scale* (WBTH; Fletcher, 1991) is a 90-item self-report measure that assesses DSM-IV PTSD and additional trauma-related behaviors in children. This instrument assesses a wide range of symptoms associated with trauma such as anxiety, depression, omens, survivor guilt, self-blame, fantasy denial, self-destructive thoughts and behaviors, dissociation, antisocial behavior, risk-taking behaviors, and changes in eating habits.

The instrument has been used to measure PTSD in children who have experienced a variety of traumatic exposures, including children exposed to the Gulf War Crisis (Moller-Thau & Fletcher, 1996). Internal consistency was reported with a Cronbach's alpha of .92 for the total score, and alpha values ranging from .70 to .89 for DSM-IV Criteria A-D. Convergent validity with other measures includes correlations.
ranging from .54 and .87 between WBTH and CPTSDI-C, the CPTSDI-P, and the PR-CRS (three other PTSD parent and child measures by Fletcher, 1991). A correlation of .54 was obtained for the PTSD subscale of the Child Behavior Checklist (CBCL; Achenbach, 1991). Additionally, scores on the WBTH were higher for the clinic sample of children than for the community sample children.

The Family Environment Scale (FES; Moos & Moos, 1981) is composed of 10 rationally derived subscales assessing interpersonal relationships among family members, personal growth characteristics emphasized by the family, and the system of organizational features of the family. The FES consists of 90 true-false statements. It is a widely used measure in clinical and research settings. The FES has been shown to have significant correspondence across ratings made by different family members, which is important, as it demonstrates the ability of the FES to generalize scores from one family member to other family members. Moos and Moos (1986) reported internal consistency across the 10 subscales as .75, with a .80 12-month test-retest reliability. Furthermore, the FES was normed on 1,125 families meeting “distressed” or “non-distressed” criteria. Many studies have supported the sound psychometric properties of the FES (Karoly & Rosenthal, 1977; Moos & Moos, 1981, 1986; Scoresby & Christensen, 1976). A shortened version of the FES will be administered to the children, using original questions from the Cohesion (C), Conflict (CON), Independence (I), and Moral-Religious Emphasis (MRE) subscales.

The Self-Report Coping Scale (SRCS; Causey & Dubow, 1992) is a 70-item self-report measure that assesses children's coping strategies, based on 5 coping subdomains. The Approach Scale includes Problem-Solving (PS) and Social Support Seeking (SSS);
the Avoidance Scale consists of Distancing (D) and Emotional Reaction (including Internalizing (I) and Externalizing (E)). After summing subscale items, higher scores suggest a greater endorsement of that specific coping strategy (Approach or Avoidance).

There are two lead questions (i.e., "When I get a bad grade in school, one worse than I normally get, I usually..." "When I have an argument or a fight with a friend, I usually..."), each with 34 response items (e.g., "Get help from a friend."). Each item is linked to a 5-point Likert scale ranging from never (1) to always (5). The children are also asked to rate the degree of control they feel they have when these stressors occur, using the same Likert scale.

Reliability and validity was originally investigated with 4th through 6th graders (Causey & Dubow, 1992) and subsequently with older students (Causey & Dubow, 1993). Principal factor analysis showed that 38% of the variance of each stressor (PS, SSS, D, I, E) was accounted for by the hypothesized 5-factor solution. Internal consistencies ranged from .68 to .84 and two week test-retest consistency ranged from .58 to .78. Cronbach's alphas for Approach Coping were alpha = .89 at Time 1 and alpha = .88 at Time 2; Avoidance Coping was alpha = .82 at Time 1 and alpha = .77 at Time 2. Validity was reported as significantly positively correlated with a companion peer-rating coping measure, yielding $r = .53$ for Coping with a Poor Grade and $r = .47$ for Coping with a Peer Argument. Correlation with Behavioral Self-Esteem and Global Self-Worth (Harter, 1985) showed a modestly positive relation on Problem-Solving with $r = .35$ for the academic stressor and $r = .30$ for the peer stressor. Distancing and Externalizing avoidance showed negative relations of $r = -.29$ for the academic stressor and $r = -.24$ for the peer stressor.
Clinician Assessment Measures

The Children's PTSD Inventory (Saigh, Yasik, Oberfield, Inamdar, Rubenstein, & Nester, unpublished manuscript) is a semi-structured interview with five subscales developed to reflect the DSM-IV criteria for PTSD. The criteria measured include exposure (assesses traumatization through experimental, vicarious, or verbal mediation), re-experiencing (unwanted anxiety-evoking recollections of the trauma), avoidance (divergent symptoms which were not apparent before the trauma), general affect and hyperarousal, and degree of distress. Scores reflect one of four diagnoses: no PTSD, acute PTSD, chronic PTSD, or delayed-onset PTSD. A forced dichotomy was used in this study yielding negative or chronic diagnoses.

Following field testing in 1988 and 1989, preliminary assessment of interrater reliability yielded 100% interrater agreement (kappa = 1.0) by Saigh et al. (unpublished manuscript, reported in Saigh & Bremner, 1999), using an outpatient sample of 25 consecutively referred children with confirmed exposure to physical or sexual abuse. Preliminary data also yielded a kappa coefficient of .92 between clinician derived diagnoses and diagnoses using the Children's PTSD Inventory; sensitivity in this population of 100%; specificity of 92%; and diagnostic efficiency of 96%. They also noted a positive predictive power of .93 and a negative predictive power of 1.0.

Another study by Yasik, Saigh, Oberfield, Green, Halamandaris, & McHugh (1998) was conducted with 82 traumatized and 22 non-traumatized youths (N = 104) at an inpatient psychiatric hospital. At the subtest level, moderate Cronbach alphas (.53 - .89) were achieved, with an alpha of .95 at the diagnostic level. Interrater reliability yielded a kappa of .96 at the diagnostic level, and good to excellent kappas (.66 - 1.00).
for the subtest level. Test-retest reliability was indicated via 97.6% agreement at the
diagnostic level (Saigh, Yasik, Oberfield, Green, Halamandaris, & McHugh, in press).

Criterion and construct validity of the Children’s PTSD Inventory was examined
by Yasik and colleagues (1998) with 73 traumatized and 22 non-traumatized youth at an
inpatient psychiatric hospital. Responses obtained using clinical interviews, Diagnostic
Interview for Childhood Adjustment-Revised (DICA-R; Herjanic & Reich, 1982) and
Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First,
1990) PTSD module administrations were compared to responses on the Children’s
PTSD Inventory. The results were reported as moderate to high levels of sensitivity (.91
- 1.00), specificity (.90 - .97), positive (.68 - .94) and negative (.95 - 1.00) predictive
power, and diagnostic efficiency (.92 - .95). Correlations with the Revised Children’s
Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1985) (r = .74), Children’s
Depression Inventory (CDI; Kovacs, 1992) (r = .65), and Junior Eysenck Personality
Inventory (JEPI; Eysenck, 1963) Neuroticism scale (r = .59) represent significant
evidence for the construct validity of this measure. Convergent and discriminant validity
were assessed through a significant correlation with the CBCL Internalizing scale (r =
.52), with non-significance compared with the CBCL Externalizing scale (r = .19, and
the JEPI Extroversion scale (r = -.08). Overall, the reliability and validity data
concerning the Children’s PTSD Inventory provides support that it demonstrates sound
psychometric properties.

The Anxiety Disorders Interview Schedule for Children (ADIS-C; Silverman &
Nelles, 1988) is a semi-structured interview with a yes/no question format and a visual
rating scale to help young people report the frequency and duration of their worry, fear,
somatic complaints and to assess the interference of these symptoms in their daily life functioning. The questions address symptomatology, symptom history, severity, and precipitating events. Subsections include all anxiety disorders as well as PTSD, dysthymia, major depression, and suicidal ideation. There is also a parallel parent version (ADIS-P), which was not used in this study. The ADIS-C provided descriptive comorbidity data.

The ADIS has demonstrated sound psychometric properties. In a study with 51 subjects, Silverman and Nelles (1988) found interrater reliability to have a kappa coefficients for the ADIS-C (.84), ADIS-P (.83), and a diagnosis composite of the child and parent interviews of .78. With regard to agreement on ratings of symptom severity, they found Pearson product-moment correlations of .71 with the ADIS-C, .76 for the ADIS-P, and .74 with the composite data. Silverman and Eisen (1991) evaluated the test-retest reliability of the ADIS with 50 subjects using a test-retest interval of 10 to 14 days. They reported overall Kappa coefficients of .76 for the ADIS-C, .67 for the ADIS-P, and composite diagnosis of .75. Pearson product moment correlations of severity ratings were .89, .87, and .88 for the child, parent, and composite diagnoses, respectively. Rapee, Barrett, Dadds, and Evans (1994) reported similar interrater reliability (k = .59 to .82) and found parent-child agreement to be poor (k = .11 to .44). They concluded that despite low parent-child agreement, there was little criterion variance for DSM-III-R anxiety disorders (and likely DSM-IV), suggesting that the diagnostic criteria are clear and usable.

Only one study reported specific information concerning the PTSD subscale on the ADIS. For the PTSD subscale, Silverman and Eisen (1991) reported overall test-retest
reliabilities of symptom scores as .43 (p < .002) on the ADIS-C and .25 (p < .05) for the ADIS-P. This finding attests to the utility of the ADIS PTSD subscale as an assessment measure of PTSD, particularly when using the child version.

Procedure

Permission to interview and informed consent was obtained from the Juvenile District Attorney and the administration of the state facility (Child Haven) in Nevada and from parents in Idaho (recruited from the Family Service Alliance Center and Family and Children's Services). Assent from each child was also obtained prior to data collection. The children were interviewed and completed questionnaires at the shelter (NV) or in their homes (ID) during one or two sessions (children who could not read well needed a second session to have the questionnaires read to them). A structured diagnostic interview was conducted with each child individually by the primary investigator, and was approximately one hour in length. An additional graduate student was in attendance for approximately 20% of the interviews for reliability purposes, yielding 100% agreement on clinical diagnoses. The children completed three randomly ordered self-report questionnaires, which took approximately 40 minutes. Two-week test-retest reliability was computed for SRCS and FES-short version for 20% of subjects. Coefficients were calculated for the SRCS approach (r = .81), SRCS avoidance (r = .55), and FES-short version conflict (r = .84), moral/religious (r = .95), independence (r = .66), and cohesion scales (r = .21).

All consent forms and questionnaires were coded by number to ensure subject anonymity and will be kept confidential in a locked file cabinet for a period of 3 years, after which time all data will be destroyed.
CHAPTER 4

DATA ANALYSIS

Demographics

To identify possible differences among participants, comparisons were made across age, gender, race, parental status (e.g., dual-parent home, single-parent home, grandparent/other home), family member who had served jail time, regular drug/alcohol use in the home, and religious participation. Results indicated a significant difference between the PTSD/NO PTSD groups with respect to gender and exposure to drugs/alcohol. Specifically, an approximately equal number of boys with or without PTSD was seen, but 81% of girls met PTSD criteria. Similarly, an approximately equal number of children without PTSD reported use or no use of drugs/alcohol in the home, but 77% of children with PTSD reported such use. No other significant differences were found with respect to demographics.

Factor Analysis and Regression

Factor analysis with varimax rotation was conducted to eliminate redundant or theoretically similar variables. Thirty-one variables were found to load across eleven factors. Results are in Table 1.

Insert Table 2 about here

54
Because the sample included 55 participants, six of the eleven variables were chosen for regression analysis. Choices of variables to retain were guided by hypothesized action (precipitating or prophylactic) and strength of factor loadings. Several comorbid diagnoses loaded on Factor 1, thus the highest loading, dysthymia (.81), was retained. FES Conflict (-.84) had the highest loading of the four FES subscales loaded on Factor 2, so it was retained. The two locus of control (LOC) variables and approach coping loaded on Factor 4, resulting in the retention of the highest LOC variable (LOCGRADE, .82). Avoidance coping (.86) loaded independently on Factor 7, suggesting a distinctively different factor from LOC and approach coping; thus it was retained. ADHDHY loaded separately from other comorbid diagnoses on Factor 10 (-.84), so it was retained. YRSPAN (duration of abuse, .43) was also retained from Factor 10, as it appeared conceptually distinct from the other variables. In summary, the variables retained for regression analyses included dysthymia (.81), avoidant coping (.86), FES conflict (-.84), religion (.85), ADHDHY (-.84) and YRSPAN (.43).

Regression analyses were conducted to identify variables most predictive of, or associated with, high PTSD symptom severity (i.e., total symptoms, reexperiencing, arousal, avoidance, and distress). Results indicated that dysthymia was most predictive of total symptoms, \( F(1, 53) = 19.12, p < .001 \), accounting for 26.5% of the variance. Religion was also significantly predictive, adding another 5.9% variance for a combined total of 32.6% of the variance of total symptoms. Further regression analyses were used to examine elements of total symptoms. Using reexperiencing as the dependent variable, dysthymia was most predictive, \( F(1.53) = 11.57, p < .001 \), accounting for 17.9% of the
variance. Using *avoidance* as the dependent variable, ADHDHY was most predictive, $F(1, 53) = 5.83, p < .05$, accounting for 9.9% of the variance, and dysthymia added another 7.1% of variance, for a total of 17.0% variance ($F(2, 52) = 5.32, p < .01$). Using *arousal* as the dependent variable, dysthymia was most predictive, accounting for 29.5% of the variance, with religion contributing another 7.6% variance, for a combined total of 37.1% of the variance ($F(2, 52) = 15.34, p < .001$). Using *significant distress* as the dependent variable, dysthymia was again most predictive, $F(1, 53) = 21.27, p < .001$, accounting for 28.6% of the variance. Regression analysis of the PTSD group data, using *duration of abuse* as the dependent variable, also indicated that dysthymia was the most predictive comorbid diagnosis ($F(1, 35) = 6.67, p < .05$), accounting for 16% of the variance. Table 2 summarizes regression analysis results.

---

Insert Table 3 about here

---

*Comparison of PTSD Groups*

To examine differences between the PTSD/NO PTSD groups, multiple t-tests were used. Correction for inflated Type 1 error was calculated using a Bonferroni correction for multiple t-tests (.05/$\alpha$; .05/12 = $p = .004$). Comparisons were made for total symptoms, duration of abuse (YRSPAN, calculated by months), FES Conflict, FES Cohesion, FES Independence, FES Moral/Religious Emphasis, reexperiencing, avoidance, hyperarousal, overall distress, comorbidity, and coping style. Results indicated significant differences between the groups with respect to total symptoms ($p < .0001$), comorbidity ($p < .001$), re-experiencing ($p < .0001$), avoidance ($p < .0001$),
arousal ($p < .0001$), and significant distress ($p < .0001$). An independent t-test comparison of duration of abuse (YRSPAN, low = 1 - 24 months, high = 36 months and higher) and number of comorbid diagnoses also indicated a significant difference between high duration and low duration groups (high $M = 3.56$ comorbid diagnoses, low $M = 2.43$ comorbid diagnoses, $p < .05$). Table 3 summarizes the results.

Insert Table 4 about here

Distribution-Free Tests

Because children in the PTSD group had approximately 1.5 more comorbid diagnoses than children in the NO PTSD group (Table 3), comparisons were made with respect to which diagnoses most accounted for this difference. The Mann-Whitney U was used to make qualitative frequency comparisons because comorbid data were qualitatively scored ($0 =$ not present; $1 =$ present). This test assumes the null hypothesis that the two samples were drawn randomly from identical populations and is particularly sensitive to population differences in central tendency. The PTSD group had significantly higher mean rank scores compared to the NO PTSD group for the following comorbid diagnoses: dysthymia ($z = -3.28$, $p = .001$), major depressive disorder ($z = -2.85$, $p < .01$), and attention deficit disorder-hyperactive type ($z = -2.05$, $p < .05$). Results also suggested a trend in greater frequency of diagnoses of specific phobia ($z = -1.73$, $p = .08$) and generalized anxiety disorder ($z = -1.86$, $p = .06$) in the PTSD group.
CHAPTER 5

DISCUSSION

This study is the first to examine maltreated children with and without posttraumatic stress disorder (PTSD) using a well-standardized structured diagnostic interview as well as standardized measures of PTSD symptoms. This study is also the first to evaluate both precipitating and prophylactic factors within the same sample. Overall, childhood posttraumatic stress disorder (C-PTSD) was shown to have a high co-occurrence with dysthymia, attention deficit hyperactivity disorder-hyperactive type (ADHDHY), and moral/religious emphasis. Factors that most distinguished the PTSD and NO PTSD groups were number of total PTSD symptoms and number of comorbid diagnoses; both factors were significantly higher in the PTSD group. In addition, as duration of abuse increased, the number of comorbid diagnoses also increased. Comorbid diagnoses in the PTSD group were significantly more frequent with respect to dysthymia, major depressive disorder, and attention deficit disorder-hyperactive type. A trend was found for generalized anxiety disorder and specific phobias.

Dysthymia was identified as having a high co-occurrence with total PTSD symptoms, reexperiencing symptoms, arousal symptoms, and distress symptoms, and contributed to prediction of (or association with) avoidance symptoms. In addition, it was among the most frequently occurring comorbid diagnosis (including major depression, generalized anxiety, and phobias) as duration of abuse increased. It is
interesting to note that dysthymia, not major depressive disorder, was the type of depression most predictive of PTSD symptoms. This finding suggests that low-grade, long-term, personality-based depression may have a priming effect toward the subsequent development of PTSD following trauma. Children with PTSD often report feelings that include a sense of foreshortened future, that life will just be too hard, or they will never be whole again following a violation such as traumatic child maltreatment. These feelings are consistent with children diagnosed with dysthymia and represent a more negative overall perception of the world, rather than a more optimistic perception. Conversely, however, traumatic events may serve as a catalyst for dysthymic symptoms, and produce a negative feedback loop (i.e., a child with a negative attitude may contribute to less positive interaction from their parent). Thus, more research is needed in this area to separate precursor and reactionary effects.

Another variable that may suggest a negative feedback loop between child and parent is attention deficit disorder (ADHD). ADHDHY (hyperactive type) was another comorbid diagnosis identified as showing a strong co-occurrence with C-PTSD. This is not too surprising given the range of extreme, often irritating behaviors these children can manifest. It may be that as children with ADHD grow up, they are more difficult to parent, thus taxing available family resources. As the child's symptoms stress the family system, parents may increasingly attempt to overcorrect the inappropriate behavior, rendering the child at greater risk to become abused. It is also possible that symptoms of preexisting ADHD (e.g., hyperactivity, distractability, and impulsiveness) can render these children somehow more vulnerable to develop PTSD following traumatic maltreatment. Symptoms such as impulsivity, high arousal, and diminished control of
aggression and anger are common to both C-PTSD and ADHD, and may suggest
distinctions within the nervous system that predispose an individual to maladaptive
reactions rather than cognitive processing to appropriately manage traumatic or
challenging events.

An area that has not received much attention in the C-PTSD literature is the
contribution of religious and moral emphasis in a child's ability to effectively process
and metabolize traumatic events. In general, religion has been shown to have a net
buffering effect for people dealing with stressful events (Koenig & Larson, 1998).
However, results of this study identified religion as another significant predictor of C-
PTSD, which seems to suggest that children with more religious influence in their lives
had a harder time coping with traumatic maltreatment. A note of caution should be used
in relation to these findings as they stem from only one demographic question about
religious participation. If this result is not an artifact, one possible implication is that as
more emphasis is given to moral ("right" or "wrong") behavior, the child's schematic
interpretation of moral behavior may become disrupted and discordant when physical or
sexual violation occurs, particularly if the perpetrator is someone previously trusted.
Children from a strong religious and moral background may also be trying to resolve the
question of why God would allow this terrible thing to happen to them, whereas a child
without views of a supreme protector may be more likely to interpret negative life events
as "just part of life." Additionally, it is possible that families who emphasize religious
teaching may be overly puritanical. These families may be more restrictive and more
likely to use harsher forms of punishment, which can become extreme and abusive.
While these hypotheses seem plausible, again, results from this study concerning
religion stem from one demographic question about religious participation and could be an artifact. Additionally, the FES moral/religious subscale showed a fairly weak relationship with total symptoms and with arousal. More research is thus necessary in this area to further illuminate the role religion may play in development of C-PTSD.

As hypothesized, duration of abuse was strongly related to the development of PTSD. However, hypotheses were not supported with respect to resilience/hardiness, coping strategies, locus of control, affect regulation, or support networks. No significant differences were detected between the PTSD/NO PTSD groups for these variables. Because each of these variables has been shown to contribute to the overall individual adjustment, nonsignificant results were surprising. Inability to collect parent data significantly hampered a thorough investigation of many variables, including these. In the case of resilience, for example, parental information about the child's behavior and mental state before, during, and immediately following trauma would have provided key information.

The variables of coping style (e.g., approach and avoidant) and locus of control (e.g., external or internal) have received little attention in the C-PTSD literature, though much has been written with respect to their significant overall contributions to everyday problem-solving in adults and children (Causey & Dubow, 1992; Folkman & Lazarus, 1984; Shalev, 1996; Wertlieb et al., 1987). Because the results of this study are inconsistent with previous coping literature, many questions arise. First, sampling a clinical population may do more to explain similarities than differences in coping strategies among the PTSD and NO PTSD groups, since all of the children in this study had been victims of child maltreatment and been removed from their homes to shelter-
care. Second, coping behavior can have much to do with how a child manages the
distress of their symptoms as well as their stressful life events. Thus, it is possible that
the situations presented on the SRCS (e.g., getting a bad grade; having an argument or
fight with a friend) were not salient concerns for this maltreated population in the
broader context of their everyday survival needs. Finally, the relatively small sample
size may not have yielded enough power on this measure. More research is needed in
this area.

The clinical implications of this research highlight the need to use sound
diagnostic measures, as children can emit a vast array of responses to traumatic events
that may or may not be directly related to C-PTSD. Using a comprehensive assessment
approach to identify a child’s reactions to traumatic maltreatment, their current
developmental level, comorbidity, as well as individual strengths and weaknesses can be
used in tandem to rationally guide the most effective treatment modalities and treatment
goals. Specifically, broad spectrum diagnostic measures (e.g., the ADIS), full evaluation
of PTSD across different areas of functioning (e.g., PTSD-I), as well as supplemental
measures of behavior (e.g., Child Behavior Checklist) will help clinicians form a clearer
picture of the developmental and environmental elements that should be targeted in
treatment planning.

Currently, there is a paucity of information concerning empirically validated
treatments for child trauma victims. As managed health care exerts pressure for time-
limited treatment, the need is acute for efficient brief therapies that are derived from
sound empirical foundations. Preliminary research suggests some efficacy treating C-
PTSD with Anxiety Management Training (AMT; Foa & Meadows, 1998), Stress
Inoculation Training (SIT; Michenbaum, 1974), cognitive-behavioral therapy (CBT; Debblinger & Heflin, 1996), and various exposure therapies (see Keane, 1998 for review). As many of these treatments are adaptations of treatments designed for adults, additional research may allow for more tailored treatment for child trauma victims.

Because it is evident that maltreated children who develop PTSD manifest a variety of symptoms, tailored treatment, designed with specific therapeutic modules, will address the strengths and weaknesses of an individual child. For example, overall PTSD symptoms could be treated with anxiety management training (e.g., relaxation training, cognitive restructuring, Stress Inoculation Training, and social skills training), and exposure-based techniques (e.g., systematic desensitization, and graduated exposure). However, a child with dysthymia and PTSD could also benefit from cognitive behavioral therapy (CBT) that confronts, challenges, and modifies maladaptive cognitions such as misattributions of abusive events, as well as encouraging the child to increase pleasurable experiences.

A treatment module for a child with PTSD and a religious conflict might include clergy to discuss religious issues or beliefs causing cognitive dissonance for the child. Parent involvement (with the child or parallel to child therapy) may also be beneficial to increase PTSD treatment success, address parent stress, and improve parenting skills. Inclusion of pharmacotherapy may also be indicated for debilitating symptoms of anxiety, depression, or ADHD. Children who manifest the common overlap of PTSD and ADHD symptoms may need a behaviorally oriented treatment plan to address externalizing behaviors (Finkelhor & Berliner, 1995), rather than a more cognitively or exposure-based treatment. Thus, to identify the specific modules needed for a tailored
treatment plan, multi-source, comprehensive assessment is key to identification of precursor, buffering, and maintenance effects.

Future directions of this research should include replication and further evaluation of both precipitating and prophylactic variables with the inclusion of parent data. These results and subsequent findings can be instrumental for continued research and treatment development to thwart the negative developmental trajectory that C-PTSD can cause. Further research on coping skills and resilience factors could contribute to school prevention programs to strengthen children in their ability to confront challenges more effectively. Self-protective education could also be incorporated in school curricula to render children less vulnerable to experiencing and/or reacting negatively to trauma such as child maltreatment. Additionally, parenting classes could be developed to help parents improve their parenting skills and decrease their stress levels, thus limiting the likelihood of resultant child maltreatment. Individual treatment of comorbidity such as depression, ADHD, and generalized anxiety, as well as promotion of social support for isolated children, could also serve to ameliorate the negative symptoms associated with traumatic child maltreatment. Group interactions could also help children feel validated, and come to understand and process their traumatic events more effectively.

Undoubtedly, prevention and treatment would be particularly effective if both children and their parents are involved and taught to function optimally.

While this study has illuminated the area of C-PTSD, there were limitations that subsequent replication could address. A larger sample size may have allowed for a better distinction of predictors, buffering agents, and differences between groups. As with most research of this nature, it can be problematic and unethical to establish a control group,
so clinical samples lend themselves as convenient and available groups of study. It would be useful to include children from the general population to minimize biases and confounds inherent within clinical samples.

A major limitation of this study was the inability to study all desired variables given the barriers to obtaining parent data. This precluded measurement of the child's initial reaction to trauma, parent's reaction to reported maltreatment, survey of the child's negative life events, parent variables such as parenting techniques, parent pathology, maternal adjustment, parental support, attachment and temperament information, and types of exposure/experience with abuse. It also precluded the collection of more complete demographic information such as socioeconomic status, family members in jail (including type of crime committed), and previous experience with therapy (parent and child). Despite the limitations of the study, there were also several strengths. Though subjects were from a clinical sample, the demographic features were diverse and heterogeneous. In addition, the use of a structured diagnostic interview provided objective measurement of comorbidity and a broad analysis of PTSD symptoms in both groups of children.

While there have been advances over the last decade in assessment tools for traumatized children and as research continues to contribute to our knowledge base, clearly we are far from a complete understanding of C-PTSD. Identifying the most salient precipitating and prophylactic variables (e.g., dysthymia, ADHD, religion, duration of abuse) that contribute to C-PTSD is essential for helping researchers and clinicians develop the most efficacious treatments and prevention programs for maltreated children and their families. Using broad spectrum measures in tandem with
thorough PTSD and comorbidity assessment (from multiple sources) will serve clinicians well in providing individualized treatment for a child's symptoms. Because the symptoms of C-PTSD can become severe, long-lasting, and quite debilitating, it is essential that the negative trajectory of C-PTSD be curtailed as early as possible.

Individuals, as well as our society as a whole, pay a great toll for the negative effects of traumatic child maltreatment. This underscores the need to increase available resources both to victims and to scientific endeavors in this area, thus guiding development of knowledgeable child mental health professionals who can provide intervention, prevention, and further research.
APPENDIX A

IRB APPROVAL
DATE: June 7, 2000

TO: Lisa Linning
Psychology
M/S 5030

FROM: Dr. Fred Preston
Chair, Social/Behavioral Committee
of the Institutional Review Board

RE: Status of Human Subject Protocol Entitled:
"Precipitating and Prophylactic Factors Contributing to Posttraumatic Stress Disorder Symptomology in Maltreated Children"

OSP # 113s0500-028

This memorandum is official notification that the Social/Behavioral Committee of the Institutional Review Board has approved the protocol for the project listed above. This approval is for a period of one year from the date of this notification, and work on the project may proceed.

Should the use of human subjects described in this protocol continue beyond a year from the date of this notification, it will be necessary to request an extension.

If you have any questions or require any assistance, please contact the Office of Sponsored Programs at 895-1357.

cc: OSP file
REFERENCES


Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.


<table>
<thead>
<tr>
<th>Variable</th>
<th>Factor Loadings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>YRSPAN</td>
<td>.360</td>
</tr>
<tr>
<td>RELIGION</td>
<td>.118</td>
</tr>
<tr>
<td>DRUGALCO</td>
<td>.158</td>
</tr>
<tr>
<td>AGE</td>
<td>.476</td>
</tr>
<tr>
<td>GENDER</td>
<td>.431</td>
</tr>
<tr>
<td>RACE</td>
<td>.044</td>
</tr>
<tr>
<td>PARENT</td>
<td>-.020</td>
</tr>
<tr>
<td>FAMJAIL</td>
<td>-.499</td>
</tr>
<tr>
<td>APPROCCOP</td>
<td>.056</td>
</tr>
<tr>
<td>AVOIDCOP</td>
<td>.100</td>
</tr>
<tr>
<td></td>
<td>FESCOHES</td>
</tr>
<tr>
<td>--------</td>
<td>----------</td>
</tr>
<tr>
<td>FESCONF</td>
<td>.067</td>
</tr>
<tr>
<td>FESIND</td>
<td>-.145</td>
</tr>
<tr>
<td>FESMRE</td>
<td>.224</td>
</tr>
<tr>
<td>SEPANX</td>
<td>.009</td>
</tr>
<tr>
<td>SOCOPHOB</td>
<td>.082</td>
</tr>
<tr>
<td>SPECPHOB</td>
<td>.338</td>
</tr>
<tr>
<td>PANIC</td>
<td>.199</td>
</tr>
<tr>
<td>PANICAG</td>
<td>.194</td>
</tr>
<tr>
<td>GAD</td>
<td>.722</td>
</tr>
<tr>
<td>OCD</td>
<td>.632</td>
</tr>
<tr>
<td>DYS</td>
<td>.809</td>
</tr>
<tr>
<td>MDD</td>
<td>.795</td>
</tr>
<tr>
<td>ADHDS</td>
<td>-.240</td>
</tr>
<tr>
<td>ADHDDY</td>
<td>-.059</td>
</tr>
</tbody>
</table>
Table 3

Six Regression Analyses

<table>
<thead>
<tr>
<th>DV</th>
<th>PREDICTOR</th>
<th>B</th>
<th>SEB</th>
<th>β</th>
<th>t</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Symptoms</td>
<td>dysthymia</td>
<td>5.58</td>
<td>1.41</td>
<td>.46</td>
<td>3.96***</td>
<td>.326</td>
</tr>
<tr>
<td></td>
<td>religion</td>
<td>1.12</td>
<td>.52</td>
<td>.25</td>
<td>2.16*</td>
<td></td>
</tr>
<tr>
<td>Reexperiencing</td>
<td>dysthymia</td>
<td>1.46</td>
<td>.43</td>
<td>.42</td>
<td>3.40***</td>
<td>.179</td>
</tr>
<tr>
<td>Avoidance</td>
<td>ADHDHY</td>
<td>-3.01</td>
<td>1.36</td>
<td>-.28</td>
<td>-2.21*</td>
<td>.170</td>
</tr>
<tr>
<td></td>
<td>dysthymia</td>
<td>1.18</td>
<td>.56</td>
<td>.27</td>
<td>2.11*</td>
<td></td>
</tr>
<tr>
<td>Arousal</td>
<td>dysthymia</td>
<td>1.53</td>
<td>.36</td>
<td>.48</td>
<td>4.30***</td>
<td>.371</td>
</tr>
<tr>
<td></td>
<td>religion</td>
<td>.33</td>
<td>.13</td>
<td>.28</td>
<td>2.50*</td>
<td></td>
</tr>
<tr>
<td>Significant Distress</td>
<td>dysthymia</td>
<td>1.71</td>
<td>.37</td>
<td>.54</td>
<td>4.61***</td>
<td>.286</td>
</tr>
</tbody>
</table>

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
<table>
<thead>
<tr>
<th>Abuse Duration</th>
<th>dysthymia</th>
<th>34.41</th>
<th>13.34</th>
<th>.40</th>
<th>2.58*</th>
<th>.161</th>
</tr>
</thead>
</table>

(PTSD group)

*p < .05; **p < .01; ***p < .001
Table 4

Comparison of PTSD Groups

<table>
<thead>
<tr>
<th>PTSD GROUP</th>
<th>N</th>
<th>M</th>
<th>SD</th>
<th>SEM</th>
<th>SIG. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comorbid 1</td>
<td>18</td>
<td>1.83</td>
<td>1.20</td>
<td>0.28</td>
<td>0.003</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>3.54</td>
<td>2.17</td>
<td>0.36</td>
<td>0.000</td>
</tr>
<tr>
<td>Approcop 1</td>
<td>18</td>
<td>97.94</td>
<td>20.86</td>
<td>4.92</td>
<td>0.358</td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>91.82</td>
<td>23.35</td>
<td>4.07</td>
<td>0.343</td>
</tr>
<tr>
<td>Avoidcop 1</td>
<td>18</td>
<td>79.22</td>
<td>20.45</td>
<td>4.82</td>
<td>0.349</td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>84.79</td>
<td>19.87</td>
<td>3.46</td>
<td>0.355</td>
</tr>
<tr>
<td>Yrspan    1</td>
<td>18</td>
<td>27.28</td>
<td>30.09</td>
<td>7.09</td>
<td>0.025</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>53.38</td>
<td>43.25</td>
<td>7.11</td>
<td>0.013</td>
</tr>
<tr>
<td>FES Cohes 1</td>
<td>18</td>
<td>42.33</td>
<td>18.14</td>
<td>4.28</td>
<td>0.885</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>41.52</td>
<td>19.64</td>
<td>3.42</td>
<td>0.882</td>
</tr>
<tr>
<td>FES Conflict 1</td>
<td>18</td>
<td>57.44</td>
<td>13.12</td>
<td>3.09</td>
<td>0.318</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>61.97</td>
<td>16.36</td>
<td>2.85</td>
<td>0.288</td>
</tr>
<tr>
<td>FES Indepen 1</td>
<td>18</td>
<td>41.56</td>
<td>15.77</td>
<td>3.72</td>
<td>0.632</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>39.36</td>
<td>15.35</td>
<td>2.67</td>
<td>0.635</td>
</tr>
<tr>
<td>FES Moral/Rel 1</td>
<td>18</td>
<td>51.28</td>
<td>10.39</td>
<td>2.45</td>
<td>0.942</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>51.06</td>
<td>10.02</td>
<td>1.74</td>
<td>0.943</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>--------</td>
<td>-----</td>
<td>------</td>
<td>---</td>
<td>------</td>
</tr>
<tr>
<td>Reexper</td>
<td>1</td>
<td>18</td>
<td>1.59</td>
<td>1.46</td>
<td>.35</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>37</td>
<td>4.05</td>
<td>.91</td>
<td>.15</td>
</tr>
<tr>
<td>Avoidance</td>
<td>1</td>
<td>18</td>
<td>2.35</td>
<td>1.84</td>
<td>.45</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>37</td>
<td>5.11</td>
<td>1.47</td>
<td>.24</td>
</tr>
<tr>
<td>Arousal</td>
<td>1</td>
<td>18</td>
<td>1.41</td>
<td>1.28</td>
<td>.31</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>37</td>
<td>3.32</td>
<td>1.11</td>
<td>.18</td>
</tr>
<tr>
<td>Significant</td>
<td>1</td>
<td>18</td>
<td>.29</td>
<td>.47</td>
<td>.11</td>
</tr>
<tr>
<td>Distress</td>
<td>3</td>
<td>37</td>
<td>2.84</td>
<td>1.01</td>
<td>.17</td>
</tr>
<tr>
<td>Total Symp</td>
<td>1</td>
<td>18</td>
<td>5.65</td>
<td>3.53</td>
<td>.86</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>37</td>
<td>15.32</td>
<td>3.18</td>
<td>.52</td>
</tr>
</tbody>
</table>
VITA

Graduate College
University of Nevada, Las Vegas

Lisa Marie Linning

Home Address:
2906 Apogee Lane
Henderson, Nevada 89014

Degrees:
Bachelor of Science, Psychology, 1998
Idaho State University

Special Honors and Awards:
Psychology Graduate Student Faculty Representative, UNLV, 2001-2002
Graduate Student Research Award, UNLV Psychology Department, 2001
Graduate Student Association Summer Assistantship, UNLV, 2001
Graduate Student Association Grant, UNLV, 2001
Graduate Student Representative, UNLV, 2000-2001

Publications and Posters:


Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
Thesis Title: Precipitating and Prophylactic Factors Contributing to Posttraumatic Stress Disorder Symptomatology in Maltreated Children

Thesis Examination Committee:
   Chairperson, Dr. Christopher A. Kearney, Ph.D.
   Committee Member, Dr. Mark Floyd, Ph.D.
   Committee Member, Dr. Murray Millar, Ph.D.
   Graduate Faculty Representative, Dr. Colleen Peterson, Ph.D.