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**Predictors of Posttraumatic Stress Disorder in an
Australian Community Sample of Young People**

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Clinical Psychology)

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Abstract

Posttraumatic stress disorder (PTSD) is one of the most common disorders following exposure to trauma. It is a serious, often chronic, and debilitating disorder affecting children, young people, and adults. However, relatively little is known about the prevalence, risk factors, and maintenance of this disorder in Australian young people. This study describes the lifetime prevalence, chronicity, and psychological comorbidity of PTSD in an Australian birth cohort of 706 young people oversampled for maternal history of depression. Risk factors for the development of PTSD in young people were explored. Using a prospective, longitudinal design, data was collected on children from birth to age 20 years. Parent- and/or self-report data were available with diagnostic interviews conducted at 15 and 20 years. Logistic regression analyses were used to examine within-individual and environmental risk factors for PTSD and depression. A total of 6.4% of young people received a diagnosis of clinical PTSD with an additional 4.2% who had subclinical PTSD. PTSD was more common amongst females than males. The mean age of onset was 15 years and 4 months, and the mean duration of symptoms was 1 year and 11 months at the 20-year follow-up. The most common triggering event was a reported sexual assault and the second most reported triggering event was the witnessing of a severe injury or death of another person. PTSD was highly comorbid with lifetime diagnoses of major depression, other anxiety disorders, substance use disorders, behavioural disorders, and suicide attempts. A total of 19% of young people with clinical PTSD reported at least one lifetime suicide attempt. When compared to young people with no PTSD symptoms, female gender,

premorbid anxiety, stressful life events and maternal abuse history significantly predicted PTSD symptoms. When compared to young people with depression, female gender, premorbid depression and substance use, mothers' anxiety, and stressful life events predicted PTSD symptoms in young people. Results help to identify young people most at risk of PTSD and are discussed in relation to prevention and treatment efforts.

Statement of Originality

This work has not previously been submitted for a degree or diploma in any university.

To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself.

Signed: _____

Date: _____

Naomi Michelle Beutel

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First and foremost, I wish to sincerely thank and acknowledge the participants of the Mater University Study of Pregnancy, almost 200 of whom I personally interviewed over hundreds of hours of intensive psychological interviewing across three years. I feel humbled by their openness and their willingness to welcome me, a complete stranger, into their homes and divulge their life stories. For those with posttraumatic stress disorder (PTSD), and other psychological disorders for that matter, this disclosure must not have been easy, though I am sincerely grateful and honoured that they have felt sufficient trust to share openly with me. I believe, as I'm sure many others do, that one of the most awe-inspiring human qualities is the ability to overcome adversity. Time and time again, I felt inspired by their real life stories of surviving tragedy and horror, and was at times shocked by the severe impairment caused by anxiety disorders. Interviewing people in their homes gave me a clearer snapshot of their lives, and I soon recognised the severity and pervasive disability often associated with PTSD. It became easy for me to be guided by my supervisor's interest and specialisation and turn to studying PTSD in this sample. Interest is easy to shift in this amazing and fascinating field that we call psychology. I wish it were nearly as easy to analyse PTSD in a study that was not designed to do so!

Unfortunately, research papers do little to capture the pain, loss, confusion, and gut wrenching tragedy behind PTSD. I urge the reader to please remember that behind every PTSD 'number' in this thesis, there is a real life story of hurt and pain in the life of an Australian young person. Some of these stories will be forever etched into my

memory and although throughout interviewing we, as psychologists, remain professional and composed, even today these stories can still stir some strong emotions for me. For instance, I will never forget hearing the devastating emotional agony experienced by a young person who lost family members in a gruesome and brutal homicide; or the young man who felt that his world was turned upside down by a terrifying home invasion; the young people who have testified against their own parents; the girls who told of their rape experiences; the young person who has never been the same since witnessing and trying to save their partner who was accidentally strangulated in a freak accident; the young man so paralysed by terror that not only did he smoke at least a packet of cigarettes between panic attacks during his interview (spread across two days), but who begs his parents to take him to hospital each night because he is certain that the symptoms he feels are signalling his imminent death; the ones who just happened to be in the wrong place at the wrong time and witnessed a suicide; the girl who lives in a world of fantasy and still talks to her dolls as if they are real people; and, the young person who cannot forgive himself for the death of his friend who was killed in a vehicle accident, the same accident that he survived. These are only some of the many stories of young people affected in a severe way by the trauma they have experienced. Have they not been through enough without suffering for months or even years after the event? We owe it to them to understand their troubles and find a way to prevent the ongoing, often devastating, sequelae of posttraumatic stress disorder.

In addition to the participants, I have two supervisors to thank, and will do so in order of involvement. I will always be grateful to Dr Michael Free who saw

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Last but not least I would like to acknowledge and thank my beautiful family. I love you dearly and am grateful for your support, laughter, and practical help. I have done my absolute best to juggle family, study, work, and recreation and thanks to your resilience I have managed to do this and also have a happy and healthy family. I'm looking forward to having fun by the beach with my growing children, and enjoying stacks of fun surfing and waterskiing. Yippee, finally mummy can drag herself away from this laptop and play!



Wallpaper of photos of MUSP babies on display at the new Museum of Brisbane (MoB) at its Opening Exhibition from October 2003 to April 2004 (Photograph courtesy of MoB).

CHAPTER 1

Literature Review

In Australia, the one year prevalence of mental health problems experienced by children and young people is at an overwhelming 19% (Sawyer, Arney, Baghurst, Clark, Graetz, & Kosky, 2000). According to these figures, approximately one in five young Australians will suffer from a mental health problem. The prevalence of childhood mental health problems is not as well documented as that of adults, however, it appears that the overall incidence of psychosocial disorders in children and young people has increased substantially over more recent years (Rutter & Smith, 1995). Highlighting the importance of early psychological development, epidemiological studies have shown that 75% of people suffering from a psychological disorder in adulthood had an age of onset prior to 24 years old (Kessler, Bergland, Demler, Jin, & Walters, 2005). Adolescence is a critical period for the development of psychological disorders and for young people, mental health disorders decrease ability to participate in normal education and social activities, and are the greatest cause of diminished quality of life and reduced productivity (Public Health Group, 2005). Thus, the issue of the mental health of Australian children and young people is critical and is worthy of more attention than it currently receives.

Recent natural and manmade disasters, together with acts of community violence, have activated growing interest in the impact of traumatic events on children and young people. The majority of children, young people, and adults are resilient to trauma exposure and do not develop long-term emotional disturbance or psychological

disorder. A minority however, do experience problematic symptoms after exposure to trauma. Posttraumatic stress disorder (PTSD) is one of the most common psychological disorders following trauma exposure. However, relatively little is understood about the prevalence, risk factors, maintenance and treatment of this chronic and debilitating disorder in young people. A thorough understanding of PTSD among Australian children and young people is important given that the effect of untreated PTSD on individuals and families can be severe and the influence on society as a whole can be far reaching. Anxiety and depression constitute the two single largest causes of non-fatal disease burden in Australia (Australian Institute of Health and Welfare, 2010) with the potential for severe developmental implications for children and young people if left untreated. An understanding of the risk factors of PTSD in children and young people can help guide important prevention and treatment efforts. This thesis aims to firstly, explore the frequency, duration, precipitating events, and comorbidity of PTSD in a large Australian community sample of children and young people; and secondly, examine risk factors for the development of PTSD symptomatology in young people.

Definition of Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is a serious, often chronic, and debilitating anxiety disorder that may occur following a severe trauma in children, young people or adults. Although extreme symptomatic reactions to trauma have been observed for centuries, the diagnosis of PTSD was only formally recognised in the psychiatric nomenclature when included in the Diagnostic and Statistical Manual of

Mental Disorders – Third Edition (DSM-III) (American Psychiatric Association, 1980). The earlier diagnosis of traumatic neurosis, from the end of the nineteenth century to the 1960's, was not explained as a sole consequence of trauma.

Psychoanalysis of the early twentieth century focused on early developmental experience rather than the impact of adult experience on individual functioning. The personality and individual 'weaknesses' of the victims themselves were questioned (Rechtman, 2004) despite the fact that even Freud himself (Freud, 1922) emphasised that traumatic neurosis was a different phenomenon from other forms of psychopathology. German and Austrian soldiers with traumatic neurosis after World War I were suspected of faking, and were often accused of weakness or a lack of moral fibre (Brunner, 2000). At the time, war was considered to be part of the normal human experience and people were expected to cope with the atrocities of war with no ill effect. As a result, the condition of traumatic neurosis was generally viewed with suspicion. People often received little empathy from physicians, and despite the horrific experiences in combat, psychoanalytic theory focussed on attributing early developmental factors as the primary aetiological factor (McFarlane, 2004).

The definition of PTSD that arose in the 1980's included in the DSM-III, was a turning point for the current conceptualisation of this psychological disorder. The clinical features of PTSD were similar to the previous diagnosis of traumatic neurosis, though the political and sociological meanings of trauma had changed dramatically. The inclusion of PTSD in the psychiatric nomenclature was the result of the evident long-term suffering of a vast number of war veterans and the short-fall of the diagnosis of traumatic neurosis in providing an understanding of their psychological needs.

Despite a long history of reported combat-related psychological disturbance, prior to the inclusion of the PTSD diagnosis, Vietnam veterans were hospitalised in Department of Veterans Affairs psychiatric wards during the 1970's and received diagnoses of schizophrenia and/or other psychotic disorders (Haley, 1974). It was predicted by researchers at the time (Horowitz, 1974) that the U.S. government would need to respond to the widespread psychological problems among the Vietnam veterans, a vast number of whom suffered from a predictable constellation of post-combat symptoms. The requirements of American psychiatry to better understand and manage the psychological needs of these veterans, prompted the inclusion of PTSD in the psychiatric nomenclature (Brett, 1996).

What followed was an uncommon situation in the field of psychology. The political and social context of the 1970's and 1980's in the U.S. paved the way for the acceptance of PTSD as a new category of diagnosis (Rechtman, 2004). Research into the severe psychological sequelae of incarceration in concentration camps during the 1970's (Horowitz, 1974) helped to remove the scepticism surrounding post-trauma responses. During the same period, researchers noted that many rape victims also experienced a range of similar symptoms following assault (Burgess & Holstrom, 1974). The socio-political context of feminism and the battle for women's rights in the 1960's, combined with the return of Vietnam veterans, thrust PTSD to the forefront as a new political condition. The definition of PTSD subsequently provided by the DSM-III attempted to avoid the stigma attached to mental illness, a stigma that still exists amongst many diagnoses today. Based upon the DSM-III definition, for a diagnosis of PTSD to be considered, Criterion A required that an individual be exposed to "a

recognisable stressor that would be expected to evoke significant symptoms of stress in almost all individuals". This criterion implied that PTSD was a normal reaction to abnormal circumstances. Furthermore, the DSM-III indicated that external factors such as trauma magnitude and duration of exposure were the primary determinants of the individual's intensity and scope of PTSD symptoms. Removal of the stressor was no longer considered a guarantee that symptoms would abate and the DSM-III postulated that symptoms might last indefinitely. Thus, the definition of PTSD in the 1980's externalised the cause of traumatic stress reactions away from weaknesses within the individual, to the unusually threatening nature of the trauma.

The definition of the traumatic event as being outside that of normal human experience, arose from a desire to remove the moral suspicion surrounding the theoretical framework of traumatic neurosis (Rechtman, 2004). As the psychopathology was exclusively created by an external event, any blame that could be attributed to the individual was removed. This change in conceptualisation was not based on new empirical findings related to changes in symptomatology or aetiology, but was instead politically and socially motivated. Following the inclusion of PTSD in the DSM-III, studies were conducted on defined populations that had experienced natural and manmade disasters such as the flood of the Buffalo Creek Disaster (Green, Lindy, Grace, Gleser, Leonard, Korol, & Winget, 1990), the Australian Ash Wednesday Bushfires (McFarlane, 1988), and the factory fire in Norway (Weisaeth, 1989). These studies provided evidence about the prevalence and aetiology of PTSD.

Upon the inclusion of PTSD into the DSM-III, little was known about the phenomenon of PTSD in children and young people. The systematic study of

childhood PTSD is relatively recent in comparison to adult PTSD and, with a growing body of research, it is now known that children and young people are indeed susceptible to developing this disorder. Recent research has found that even toddlers may experience symptoms of PTSD following traumatic events (Mongillo, Briggs-Gowan, Ford, & Carter, 2009) including domestic violence (Bogat, De Jonghe, Levendosky, Davidson, & Von Eye, 2006) and severe burns (Stoddard et al., 2006). However, the PTSD diagnostic criteria are designed for adults, not children, and only in 1987, with the publication of the DSM – Third Edition Revised (DSM-III-R) did the DSM even make reference to traumatised children. It was also recognised that children's reactions to trauma may be different to adults. However, despite the frequency of childhood trauma, and the growing body of research, there is currently little theory to understand PTSD in young people.

Diagnosis according to current nosological definition in the fourth edition of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV-TR) expanded further on previous criteria and provided more information about the manifestation of the disorder in children. PTSD is defined by four variables in the DSM-IV-TR (APA, 1994). For reasons that will be discussed in further detail later, Criterion A changed from requiring the trauma to be outside the range of normal human experience, to a stressor definition that recognised that one's personal and subjective reaction to trauma plays a crucial role in the development of PTSD. Firstly, to satisfy diagnostic criteria, one must initially have been exposed to a trauma by personal experience or by witnessing an event or events that constitute a threat to life or physical integrity. The DSM-IV-TR definition requires that the person respond with

intense fear, helplessness, or horror. Given that age may affect the way children respond to trauma and that children may struggle to express subjective symptoms, a qualifier is added to the diagnostic criteria for children: rather than responding with fear or helplessness (a subjective inner experience that children may not be able to verbalise), children may express disorganised or agitated behaviour.

The DSM-IV-TR divides PTSD into three primary symptom clusters. The first symptom cluster includes persistent re-experiencing of the traumatic event. The person must experience intrusive memories, nightmares, flashbacks, or intense distress when events remind them of the original trauma. Children may experience generalised nightmares or frightening dreams with no recognisable content. Furthermore, children may not have a sense that they are reliving the past and therefore, reliving of the trauma may manifest in re-enactment or repetitive play. The second cluster of symptoms involves avoidance behaviours. To meet criteria, the person must experience three symptoms of either: persistent avoidance of thoughts, feelings or conversations associated with the trauma, or other situations that bear a similarity to the trauma; inability to recall an aspect of the trauma; a withdrawal from significant activities and others; a numbing of affect; and a sense of a foreshortened future.

Assessment of avoidance patterns in children can be difficult given children's potential difficulties in describing subjective reactions. Thus, evaluating symptoms in light of collaborating information from parents, teachers and significant others is recommended (APA, 1994). The final cluster of symptoms refers to increased arousal and requires two of the following: sleep difficulties, irritability, concentration difficulties, hypervigilance, or a heightened startle response. Children may also exhibit

physical symptoms such as stomach aches and headaches. For diagnosis, symptoms must persist for more than one month and cause marked distress or impairment in functioning. The symptoms of PTSD usually begin within three months after exposure to the stressor, but may be delayed for months or even years (APA, 1994).

Epidemiology of PTSD in Adults

The prevalence of PTSD has been found to vary, depending on the study. In the early 1980's, the Epidemiologic Catchment Area (ECA) Studies launched by the National Institute of Mental Health (NIMH) in the United States, found in its assessment of 20,000 adults over five epidemiological communities, that the lifetime prevalence of PTSD was approximately 1% in the population as a whole (0.8% among males and of 1.2% among females) (Helzer, Robins, & McEvoy, 1987). Limitations in the methodology of the ECA led to the NIMH funded National Comorbidity Survey (NCS) (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). This more recent epidemiological study based on a representative American sample of over 8,000 persons aged 15-54 years, reported a lifetime prevalence of PTSD at 7.6% (4.8% male and 10.1% female). Clearly, this estimate is significantly greater than the lifetime prevalence of 1% found in the ECA studies and is the estimate used in most current literature. Throughout the current body of PTSD research, the estimated lifetime prevalence of PTSD in the general population ranges from 1% to 14% (APA, 1994; Helzer et al., 1987; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The DSM-IV-TR reports a lifetime prevalence of approximately 8% of the adult population in the United States. Of those people exposed to a significant traumatic event, most

experience some of the symptoms of PTSD in the days and weeks following exposure but recover well from the event. Available data suggests that about 8% of men and 20% of women exposed to trauma subsequently develop PTSD, and approximately one third of these individuals develop a chronic form that persists throughout their lifetime (Kessler et al., 1995).

A handful of studies, discussed below, have examined the occurrence of PTSD within specific populations. The National Vietnam Veterans Readjustment Study (NVVRS) investigated the occurrence, risk factors, correlates, and outcome of PTSD among the high risk group of Vietnam veterans (Kulka, Schlenger, Fairbank, Hough, Jordan, & Marmar, 1990). Results showed a lifetime prevalence of 15% among combat troops. The NVVRS highlighted the high frequency of PTSD among Vietnam veterans as well as the harmful and pervasive mental, physical, and social consequences of PTSD, such as marital problems, family problems (including violence) and work difficulties (Zatzick, Weiss, Browner, Metzler, & Golding, 1997). In an American national telephone survey of women only, Resnick and colleagues (1993) found that 12.3% had a lifetime history of PTSD. This equated to 17.9% of women who had experienced a trauma. In another American study, Breslau and associates (1991) examined young urban adults and found that 11.3% of women and 6.0% of men experienced a lifetime history of PTSD. This was equivalent to 30.7% of women, and 14% of men exposed to trauma.

Overall, it is evident that American samples dominate the research literature while the number of Australian studies is sparse in comparison. In 1997, the first Australian National Survey of Mental Health and Wellbeing of Adults (SMHWB)

(McLennan, 1997) examined prevalence estimates for mental disorders and found the 12-month incidence rate of PTSD in a stratified sample of 10,641 Australians was 3.3%. Unlike previous American studies, there was no difference in prevalence between males and females. More recently though, and with differing methodology, the second National SMHWB (ABS, 2007) funded by the Australian Government Department of Health and Ageing (DoHA), surveyed 8,800 Australians aged 16-85 years. This study found that PTSD was the most prevalent anxiety disorder among Australian adults and was even more common than depression. The 12-month incidence rate of PTSD was found to be 6.4%. In contrast with the 1997 SMHWB, the 2007 study found that women experienced higher rates of PTSD than men (8.3% compared with 4.6% respectively). It would seem therefore, that the most recent Australian epidemiology research suggests PTSD prevalence rates that are comparable with our American counterparts.

Epidemiology of PTSD in Children & Young People

Some authors argue that, just as the presentation of PTSD symptoms may differ for children, so too might prevalence rates (Holbrook et al., 2005). The findings discussed above from the ECA and the NVVRS prompted additional study into PTSD in groups of young people. Whilst most studies were conducted with clinical samples, or groups of children and young people who had experienced a shared trauma, three prevalence studies document the frequency of PTSD in non-clinical populations. Firstly, The Giaconia Longitudinal Study of PTSD (Giaconia, Reinherz, & Silverman, 1995) assessed the prevalence of PTSD in a community sample of 384 Massachusetts

adolescents (mean age 17.9 years). This longitudinal study followed the participants from 5 years of age and found lifetime prevalence rates of PTSD to be 6.3%.

Approximately 43% of the adolescents studied had experienced a qualifying trauma and of those, 14.5% developed PTSD.

A second prevalence study, The Cuffe Longitudinal Study of PTSD (Cuffe et al., 1998), examined 490 South Carolina adolescents (aged 16 to 22 years old) enrolled in a longitudinal study of depression and suicidal behaviours. This study reported that the lifetime occurrence of PTSD was 3.5% overall, with approximately 3% of females and 1% of males meeting DSM-IV diagnostic criteria. Of those studied, 16.3% of the South Carolina adolescents had experienced a qualifying trauma, and 21.3% of those reported subsequent PTSD. Therefore in this study, although much fewer had experienced a traumatic event than the Massachusetts sample, more developed PTSD as a result.

A meta-analysis conducted by Fletcher (1996) (which included 34 samples and a total of 2697 children who had experienced a qualifying trauma) found that 36% of children who had experienced a traumatic event met diagnostic criteria for PTSD.

Although there has been some variation in the estimates among the three main studies into child and youth PTSD, it is apparent that the lifetime prevalence of PTSD among children and adolescents significantly exceeds that found by ECA studies of adults in the 1980s (1%). Prevalence of PTSD in Australian children and young people is unclear. Given that the adult lifetime prevalence rate is estimated at 8% (APA, 1994) and studies are already finding between 3.5% and 6.3% for children, either rates of

PTSD are on the rise with each generation studied, or many people first develop PTSD before reaching adulthood.

As mentioned previously, the diagnostic criteria for PTSD was designed for adults and not children, and it is important to highlight research findings that have demonstrated that children who met two of the three diagnostic criteria of re-experiencing (criterion B), avoidance (criterion C), and increased arousal (criterion D) did not differ significantly from children meeting all three criteria with regard to reported distress and impairment (Carrion, Weems, Ray, & Reiss, 2002). This suggests that the DSM-IV-TR diagnostic criteria may not be sufficiently sensitive for children and that the number of children suffering emotional problems after a traumatic event may be much higher than prevalence percentages indicate. Overall, there is limited information on the prevalence of PTSD throughout childhood and youth, and there have been no Australian childhood epidemiological studies conducted to date.

Exposure to traumatic events

PTSD is unlike all other DSM-IV diagnoses in that it is the only diagnosis that requires the presence of an environmental factor, that is, exposure to a traumatic event, as a criterion for the disorder. Estimates of the lifetime prevalence of exposure to a traumatic event are surprisingly high for both adults and young people. Over half of the adults in the National Comorbidity Survey (NCS) (60.7% men and 51.2% women) experienced a qualifying trauma, with the majority of people actually experiencing two or more traumas. Research also indicates that an alarming number of young people are exposed to potentially traumatic events. In a recent US study of 349 adolescents from

nine schools, 76% reported witnessing or experiencing at least one violent event in the 3 months prior (Ozer & Weinstein, 2004). In another study of 2041 urban African adolescents, 80% reported exposure to violence, either as victims or witnesses (Seedat, Nyamai, & Njenga, 2004). Thus, exposure to trauma is high in populations of young people, particularly inner city American young people, and exposure to multiple qualifying events increases risk of PTSD development.

Numerous traumatic events have been shown to precipitate PTSD in children and young people which are discussed and referenced further below, including surviving natural and man-made disasters such as floods; violent crimes, such as kidnapping, rape or murder of a parent, sniper fire, and school shootings; motor vehicle accidents; plane crashes; severe burns; exposure to community violence; war; peer suicide; and sexual and physical abuse. Research has shown that some types of trauma are generally more distressing than others and therefore, carry a greater propensity for the development of PTSD. Earlier studies examining natural disasters typically resulted in lower rates of PTSD than other event types, particularly technological and man-made disasters, with incidence estimates varying between 0% (Earls, Smith, Reich, & Jung, 1988; Handford et al., 1986) and 5% (Lonigan, Shannon, Taylor, Finch, & Sallee, 1994; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993; Shannon, Lonigan, Finch, & Taylor, 1994). However, more recent studies have found higher prevalence rates of 11.3% (McDermott, Cobham, Berry, Stallman, 2010) and approximately 26% (Goenjian, Pynoos, Steinberg, Najarian, Asarnow, & Karayan, 1995) for PTSD in children following a natural disaster. The large degree of variability in prevalence appears to correlate with the proximity and dosage of exposure to the

disaster (Basoglu, Kilic, Salcioglu, Livanou, 2004). Rape, on the other hand, is much more likely to precipitate PTSD (32%) (Resnick et al., 1993). The presence of PTSD following acute physical trauma from motor-vehicle accident occurs in approximately 23% of children exposed (Aaron, Zaglul, & Emery, 1999) and rates of PTSD following violent crimes is generally high and within the range of 27-33% (Schwarz & Kowalski, 1991; Terr, 1983). Rates of childhood PTSD following warfare are also significant, and range between 27% (Saigh, 1991) and 33% (Arroyo & Eth, 1985). A meta-analysis of the impact of childhood sexual abuse suggested an average PTSD rate of 32% in adult survivors (Kendall-Tackett, Williams, & Finkelhor, 1993). One event type with high rates of childhood PTSD following exposure is a sniper attack event (such as school shootings). One year after the incident, more than half of the children exposed (60%) met criteria for PTSD (Pynoos, Frederick, & Nader, 1987). However, by far the highest rates of PTSD have been reported in children exposed to the sexual assault of their mother or the murder of a parent. In two studies examining the psychological impact of witnessing these shocking crimes, all children (100%) exposed were found to have developed PTSD (Malmquest, 1986; Pynoos & Nader, 1989a).

Course and sequelae of PTSD

For children as well as adults, exposure to trauma does not always lead to clinically apparent impairment (Davidson, Inslicht, & Baum, 2000; Sack, Angell, Kinzie, & Rath, 1986; Terr, 1983, 1991) and symptoms often decrease over time (Eksi & Braun, 2009; Green, Grace, Vary, Kramer, Gleser, & Leonard, 1994). Once

established however, PTSD is often chronic and debilitating in childhood, just as it is in adulthood (Green et al., 1991; La Greca, Silverman, Vernberg, & Prinstein, 1996; Pynoos & Nader, 1990). In adulthood, the course of chronic PTSD usually involves periods of symptom increase followed by remission or decrease, although some individuals may experience symptoms that are unremitting and severe. The National Comorbidity Study (Kessler et al., 1995) found that more than one third of adults with PTSD fail to remit after many years of the disorder. The median time to remission was 36 months among adults who sought treatment and 64 months among those who did not. Like the adult form, childhood PTSD is often chronic. A number of studies have suggested that children are not more adaptable or more resilient than adults in the face of adversity and trauma (Morgan & Scourfield, 2003) and that trauma can have long-lasting psychological effects that persist for many years into adulthood (Yule, Bolton, & Nurrish, 2000). Davidson and Smith (1990) reported that PTSD was three times more likely to occur among children when traumatic events were experienced prior to age 11. In van der Kolk's (1985) study into adolescents in combat during the Vietnam War, it was found that a younger age put soldiers at a higher risk of PTSD when compared with adult soldiers. Both authors have concluded that children and adolescents may be at a higher risk of developing PTSD than adults. In respect to the comorbidity of PTSD among children and young people, Yule and colleagues (2000) examined 217 adolescent survivors of a shipping disaster, the sinking of the "Jupiter" in Greek waters. They found that whilst about one third (30%) recovered within one year of PTSD onset, a further one third (34%) were still recovering from PTSD between five and eight years later. Morgan et al (2003) examined the long-term effects

of surviving the 1966 coal slag collapse (land-slide) on primary school students in South Wales, UK. They found that 29% of primary school students continued to suffer from PTSD 33-years after the event. Similarly, Famularo et al (1996) followed a sample of 156 children exposed to severe childhood maltreatment and found that approximately one third (32.7%) of children continued to experience the full criteria of PTSD two years later. Therefore, it would seem that in both children and adults, once PTSD is established, approximately one-third of people experience long-term, chronic and impairing PTSD symptomatology. The long-term effects of traumatic events and the sequelae of PTSD can be severe.

In the short term, PTSD can impair school performance and daytime learning as a result of difficulty in attention and concentration due to intrusive thoughts and sleep disturbance (Pynoos, Steinberg, & Piancentini, 1999). In early childhood, PTSD can interfere with preschool achievement of narrative coherence, normal development of trust, and emergence of autonomy through exploration (Osofsky, 1995). Violent victimisation has been linked to behavioural problems among children aged 11-14 years of age (Bender & Roberts, 2009) also implicating behavioural problems as a possible consequence of PTSD. In adolescence, a negative self-concept may contribute to excessive worries about social evaluation and can lead to social avoidance (Pynoos et al., 1999). Chronic PTSD has significant detrimental effects on the quality of life among adolescents, with additional problems in health, social competence and school performance (Clark & Kirisci, 1996). Problems with mood regulation and anger management can disrupt capacity to restrain aggression and develop assertiveness (Atkins, Stoff, Osborne, & Brown, 1993) potentially leading to conflictual

relationships, behavioural difficulties and potentially, offending behaviour. In the long-term, adults with a history of childhood abuse and PTSD are more likely to experience intimate partner violence (Engstrom, El-Bassel, Go, & Gilbert, 2008), more physical health problems (Baker, Norris, Jones, & Murphy, 2009), and unemployment (Al-Saffar, Borga, & Hallstrom, 2002).

The course and chronicity of PTSD symptoms is also related to trauma type. Generally, researchers have concluded that trauma related symptoms tend to diminish faster in children who have experienced a natural disaster, or a singular unexpected traumatic event, than those who have experienced interpersonal violence (Morgan & Scourfield, 2003). Children with PTSD following chronic trauma of an interpersonal nature, such as repeated childhood maltreatment, often experience a combination of acute and chronic symptoms (Famularo, Fenton, Augustyn, & Zuckerman, 1996; Foy, Madvig, Pynoos, & Camilleri, 1996; Linning & Kearney, 2004). Often symptoms following childhood abuse are more pervasive and complex and include sexual difficulties, low self-esteem and difficulties in relating with others (McDermott, 2004).

Lenore Terr (cited in McDermott, 2004) introduced and conceptualised the terms Type I and Type II trauma to discriminate between the developmental impact of emotional and interpersonal trauma when compared with other forms of trauma. Type I trauma is often sudden, unexpected, a singular traumatic event, that occurs in the course of otherwise normal and healthy development. Type I events would generally include motor vehicle accidents, natural disasters, other accidents and injuries, and singular severe violence in an environment where this would be considered unusual (e.g. school shootings). The content of the fear following Type I events, often clearly

relates to the trauma, or to the nature of the event symbolically. As an example, adolescent survivors of the “Jupiter” shipping disaster became fearful of sea travel and also of any mass transport, and exposure to water (Yule, Udwin, & Murdoch, 1990). Amongst children and young people with PTSD, functional impairment in key child and adolescent developmental areas such as academic performance, and peer and family relationships have been found (McFarlane, 1988; Pynoos et al., 1987). However, it is argued by some researchers that the prognosis for PTSD after a Type I trauma is often better than the prognosis from Type II trauma, given the premorbid context of normal development (McDermott, 2004).

In relation to Type II trauma, the emotional trauma is often unpredictable, repeated, and occurs over a protracted period of time. Repetitive traumatic events prevent the child and young person from returning to their normal developmental trajectory, and therefore impede recovery. Type II trauma’s include childhood abuse and maltreatment, and experiences of children in war zones. It is argued that the sequelae of Type II trauma’s are often more grave with more severe future impairment (McDermott, 2004). One particularly toxic element of child abuse and maltreatment that potentially contributes to poorer outcomes is the violation of basic trust and expectations in relationships (McLeer, Deblinger, Henry, & Orvaschel, 1992).

The impact of trauma on neurobiology (discussed in more detail in latter sections) may also play a significant role in the course and chronicity of PTSD. Researchers have concerns that trauma may be associated with harmful effects on the developing brain that can alter the brain’s structural and functional integrity (Weber & Reynolds, 2004). High levels of glucocorticoids resulting from stress associated with

childhood abuse may be harmful to the hippocampal region of the brain causing hippocampal dysfunction and atrophy (smaller volume) (Bremner et al., 1997; Nutt, 2000), however, there is debate about the direction of this relationship (i.e. hippocampal volume loss may be a consequence of trauma or a pre-existing risk factor). One study (Gurvitis et al., 1996) of Vietnam Veterans found a 26% volume reduction in the left hippocampus and a 22% reduction in the right hippocampus for those with severe PTSD. The limbic system including the amygdala, corpus callosum, prefrontal cortex, as well as the hippocampus are believed to be particularly vulnerable to effects of trauma and stress (Teicher, Glod, Anderson, Dumont, & Ackerman, 1997). The body responds to stress by activating several systems including the immune, neuroendocrine, peripheral autonomic nervous system, and the hypothalamic-pituitary axis which releases cortisol, hormones and other neurochemicals designed to assist with survival (Schwarz & Perry, 1994). Whilst this initial response is aimed at enabling survival, severe, repeated and chronic trauma can lead to a maladaptive response whereby the stress-response system is unable to return to pre-trauma levels, often resulting in chronic abnormal patterns of catecholamine activity (Perry, 2002; Perry, Pollard, Blakley, Baker, & Vigilante, 1995). Sensitisation may occur when the neurochemical changes become irreversible. Ability to regulate mood, impulse control, self-soothing ability, sense of identity, and the ability to form and maintain positive relationships are often impaired by this biological dysregulation (Van der Kolk, 1996). Research has found that very early abuse and neglect is also associated with disorganised infant attachment (Carlson, Cicchetti, Barnett, & Braunwald, 1989),

emerging speech and language deficits (Vondra, Barnett, & Cicchetti, 1990), and suicidal behaviour (Wagner, 1997).

In summary, a child's brain develops new synapses in response to the environmental stimuli and that the majority of the structural organisation of the brain occurs in childhood (Perry et al., 1995). Therefore, it would seem that given the neural plasticity of the child's brain, children are particularly vulnerable to long-term neurological effects of severe stress and trauma. These neurological effects can have far reaching consequences for the course of PTSD and the associated impact on social functioning, educational achievement, speech and language development, emotional regulation and impulse control. Partial PTSD symptomatology is common and may also be significantly impairing even when full criteria are not met (Giaconia et al., 1995; Goenjian et al., 1995). The chronicity and impairment of PTSD is a particular issue for children and young people as the disorder may significantly disrupt important developmental milestones, thus preventing normal development and impeding the ability to live a healthy life for many years to come.

Comorbidity

PTSD is highly comorbid with other psychiatric disorders and comorbidity is the norm rather than the exception (Kessler et al., 1995). Amongst adult and child populations with PTSD, both clinical and epidemiological data support a high comorbidity with a diverse range of emotional and behavioural problems (Breslau, Davis, Andreski, & Peterson, 1991; Goenjian et al., 1995; Holbrook et al., 2005; Kessler et al., 1995). Adult epidemiologic surveys indicate that the vast majority of

individuals with PTSD meet criteria for at least one other psychiatric disorder and lifetime comorbidity rates range from 62% to 92% (Breslau et al., 1991; Kessler et al., 1995; Yehuda & McFarlane, 1995). In Australia, the National Survey of Mental Health and Wellbeing of adults ($n = 10641$) found that 85.2% of males with PTSD and 79.1% of females with PTSD also experienced another Axis I disorder in the past 12 months (Creamer, Burgess, & McFarlane, 2001). A substantial percentage (nearly 50% of females and 60% of males) have two or more other psychiatric diagnoses (Brady, Killeen, Brewerton, & Lucerini, 2000; Creamer et al., 2001). Australian epidemiological findings are highly consistent with studies from the U.S. which have found that 88% of men and 79% of women met criteria for at least one other diagnosis in addition to PTSD (Kessler et al., 1995).

Diagnoses that most commonly co-occur with PTSD in adults and younger populations are depressive disorders, other anxiety disorders, and substance use disorders. Amongst Australian adults, and consistent with American counterparts, the diagnosis most commonly comorbid with PTSD is major depression (Creamer et al., 2001). Over half (52%) of all Australian men with PTSD, and 65% of Australian women with PTSD were found to have diagnoses of major depression in the past 12 months (Creamer et al., 2001). Generalised anxiety disorder is also commonly comorbid with PTSD (40% of males and 22% of females), followed by alcohol abuse and dependence (38% of males and 12% of females) and other substance abuse and dependence (23% of males and 15% of females) (Creamer et al., 2001).

With respect to children, researchers have repeatedly found that traumatised children and young people also frequently exhibit symptoms that overlap across

multiple psychological diagnoses (Famularo et al., 1996; Perry, 2002; Putman, 2009; Weinstien, Staffebach, & Biaggio, 2000). There is a growing body of evidence to indicate that depression and other anxiety diagnoses are also highly comorbid with PTSD among children and young people, and that substance use disorders commonly co-occur among adolescents and young adults (Breslau et al., 1991; Perkonigg, Kessler, Storz, & Wittchen, 2000). Among children and young people, behavioural diagnoses including ADHD are also highly comorbid with PTSD (Weber & Reynolds, 2004). Perkonigg and colleagues (Perkonigg et al., 2000) examined 3021 young people aged 14-24 years and found high comorbidity rates with 87.5% of all young people with PTSD having at least one additional diagnosis, and 77.5% had two or more additional diagnoses. This study found that major depression was highly comorbid with PTSD in young people (68.5%), as was agoraphobia with or without panic disorder (68.2%) and substance abuse or dependence (70.6%). Similarly, in a smaller study of incarcerated adolescents with PTSD, over half had major depression (58.3%), and alcohol abuse disorders (58.3%) (Ulzen & Hamilton, 2003). In another study of 218 school aged children exposed to an earthquake in Armenia, frequency of comorbid PTSD and depression was associated with proximity to the disaster and ranged from 75% in the heavily impacted areas, to 13% in outer areas (Goenjian et al., 1995).

PTSD has also been shown to be commonly comorbid with bipolar mood disorder amongst 12-17 year old adolescent outpatients (Dislaver, Benazzi, Akiskal, & Akiskal, 2007). In this study of 139 adolescents, PTSD was found to be a comorbid diagnosis with 38.2% of young people with bipolar disorder. Another comorbidity study examining a younger population of 337 children aged 6-12 years old, found that

PTSD highly correlated with symptoms of a number of other psychological disorders including phobias ($r = 0.41$), separation anxiety ($r = 0.37$), and oppositional disorder ($r = 0.36$). Thus, perhaps one of the greatest challenges for clinicians is treating symptoms in children and young people that are orthogonal, that is, having both numbing and hyperarousal symptoms. McCloskey and colleagues (2000) concluded that the commonly referred to distinction in child psychology of internalising and externalising (Achenbach & Edelbrock, 1978) holds little relevance in treating traumatised children, as they are likely to exhibit symptoms from both categories.

Substance use disorder is one such category of externalising disorder that has been found to be commonly comorbid with PTSD. The self-medication hypothesis is generally used to explain the high rates of substance use in individuals with PTSD (Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Chilcoat H.D., 1998; McFarlane, 1998). This hypothesis argues that individuals seek to use specific substances to help manage the distressing symptoms of psychological disorder. Substances are not randomly selected, but rather are chosen based upon their specific psychopharmacological effects on various states of distress (Mueser, Drake, & Wallach, 1998). In examining possible pathways to explain the comorbidity between PTSD and substance use disorders amongst adults, researchers (Jacobsen, Southwick, & Koster, 2001) have found that central nervous system depressants such as alcohol, cannabis, opioids, and benzodiazepines are the substances of choice for people with PTSD and are reported to (temporarily) significantly improve PTSD symptoms.

Experiences of PTSD are also commonly associated with alcohol and other drugs amongst younger populations. Clark and colleagues (1997) found that young

people with substance use disorders reported higher rates of traumatic events such as physical and sexual abuse, violent victimisation, and witnessing of violence when compared with controls, suggesting that the trauma may have precipitated the substance use. In a study of 4,023 young people aged between 12 and 17, those with substance use disorders were significantly more likely to have experienced physical and sexual assault (Kilpatrick, Acierno, Saunders, Resnick, & Best, 2000). Holbrook and colleagues (2005) studied 401 adolescent trauma patients aged 12 to 19 years old. These investigators found PTSD in this age group was associated with drug and alcohol abuse. However, due to the cross-sectional design of these studies, researchers were unable to determine causal pathways. Studies that have led researchers to conclude that the self medication hypothesis is insufficient for explaining the lifetime comorbidity of PTSD and alcohol and other substance use disorders are discussed later.

In childhood, PTSD symptoms may easily mimic many other externalising psychological disorders and, perhaps due to the myth that PTSD is primarily an internalising disorder, children are often misdiagnosed and mistreated (Weber & Reynolds, 2004). Weber and colleagues (2004) argued that the hyperarousal characteristics of PTSD made it easy to misdiagnose PTSD as attention deficit hyperactivity disorder (ADHD) or bipolar mood disorder if comprehensive historical information about trauma exposure was not gathered. Weinstien et al. (2000) have reported that sexually abused children often receive a diagnosis of ADHD rather than PTSD. Amongst sexually abused children diagnosed with PTSD, 54% have been found to also meet criteria for ADHD (McLeer, Callaghan, Henry, & Wallen, 1994).

However, accurate diagnosis and treatment are vital to the recovering of PTSD symptoms. Treatments for many of the externalising symptoms that mimic PTSD in children (such as ADHD and conduct disorder) differ to the treatments for PTSD. For instance, stimulant medication is a common treatment for ADHD, however it is argued that stimulants are harmful to children with PTSD as they may exacerbate their neuropsychological symptoms (Weber & Reynolds, 2004). Therefore, differential diagnosis is crucial, but is difficult given that comorbidity tends to be the rule rather than the exception with children diagnosed with PTSD. Assessing PTSD in children is also more difficult than for adults and ethical problems include mandatory reporting of abuse, confidentiality boundaries, and hesitancy to cause distress to children by probing sensitive areas (Putman, 1996). Paediatric neuropsychologists, Weber and Reynolds (2004) argue for thorough history taking and the utilisation of neuropsychologists when working with children who have been exposed to trauma. They argue that “purely traditional psychological assessments of personality and emotion will be insufficient to elucidate accurate, effective treatments and to avoid actually designing treatment plans that violate the prime directive of health care: first, do no harm” (Weber & Reynolds, 2004, p. 127).

In addition to the aforementioned diagnoses, there is also a growing body of evidence linking PTSD with suicidal ideation and suicide attempts. In fact, PTSD has been found to be a stronger predictor of suicide in adult community samples than major depressive disorder (Davidson, Hughes, Blazer, & George, 1991). The relationship between depression and suicide and suicidal ideation has received a large amount of attention over the years, and is the primary rationale underpinning policies

aimed at decreasing societal costs of depression. Until recently, the role of PTSD in suicidal ideation has been relatively ignored, despite some evidence of significance. An extensive review of the adult PTSD literature, suggests that individuals with PTSD have been found to be at higher risk of suicide (Kotler, Iancu, Efroni, & Amir, 2001). The Carolina subset of the ECA study found that, of individuals with a lifetime history of PTSD, 19.8% had attempted suicide compared to 3.9% with other diagnoses and 0.8% of the general population (Davidson et al., 1991). Adults with PTSD were 14.9 times more likely to have attempted suicide than the non-PTSD participants. After controlling for comorbid depression, those with PTSD were still 8.2 times more likely to attempt suicide. Confirming previous results that PTSD patients show high levels of suicidal ideation and attempts, Tarrier and colleagues (Tarrier & Gregg, 2004) found that over half (56.4%) of a civilian sample with chronic PTSD reported some aspect of suicidality, 38% reported suicidal ideation, and 18% progressed further to making plans or carrying out attempts. In relation to adolescents with PTSD, an increased risk of suicidal ideation and suicide attempts has also been documented. Dilsaver et al (2007) found that PTSD outpatients, aged between 12 and 17 years old, were 4.5 times more likely to have attempted suicide when compared with patients with depression and other diagnoses. Even after controlling for bipolar disorder, PTSD patients were still 4.2 times more likely to have attempted suicide. The authors concluded that PTSD might represent an independent risk factor for suicide. Findings of a more recent study examining 1698 young adults (with a mean age of 21 years old) support the conclusion that PTSD independently increases the risk of suicide attempt (Wilcox, Storr, & Breslau, 2009). These researchers found a robust relationship between PTSD and

suicide attempt, even after adjusting for major depression, alcohol and substance use disorders, and exposure to traumatic events.

In Australia, alarmingly high rates of suicidality have been reported amongst Australian young people and completed suicide remains the second highest cause of death among young Australians (AIHW, 2007). The Child and Adolescent Component of the Australian National Survey of Mental Health and Wellbeing (Sawyer, Arney et al., 2000) found that 12% of Australian young people reported seriously contemplating suicide, 8.9% had made serious suicide plans, and 4.2% had actually attempted suicide. Young people with more emotional and behavioural problems reported substantially more suicidal ideation (42%) with one-quarter (25%) following through with a suicide attempt. The relationship between attempted suicide and PTSD in an Australian adult population was demonstrated by The Australian National Mental Health and Wellbeing Survey (ABS, 2007) which found a stronger relationship between PTSD and suicide attempt than depression and suicide attempt. Current depression diagnoses had an odds ratio of 7.2 for lifetime suicide attempt, versus 14.6 for current PTSD diagnoses. Thus, compared with adults with depression, Australian adults with PTSD were found to be twice as likely to attempt suicide.

To the best of this author's knowledge, there is no published research examining the suicidality among Australian young people with PTSD. The Australian Institute for Suicide Research and Prevention, Griffith University, Qld has recently received an Australian Research Council (ARC) grant to examine indicators for child suicide, but research is yet to begin. Given the high rates of suicidality amongst Australian young people, the fact that suicide attempts are one of the strongest

predictors of completed suicide (Kessler, Borges, & Walters, 1999), and data that suggests that PTSD may be a stronger predictor of attempted suicide than depression, this issue is of high priority and requires further research, but is yet to be seriously embraced in clinical practice.

The neurobiology of PTSD

Research has focussed on clarifying the alterations in neurobiological systems involved in stress regulation amongst people with PTSD. The hypothalamic-pituitary-adrenal (HPA) axis has been implicated, as well as various neurotransmitters and neuropeptides involved in the regulation of the fear and stress response. Efforts have been made to link specific features of PTSD (such as altered mechanisms of learning and extinction, sensitisation to stress, and arousal) with neurobiological changes. Researchers have also attempted to elucidate whether neurobiological changes in PTSD are a consequence of trauma exposure or pre-existing vulnerabilities.

The HPA axis is the primary neuroendocrine stress response system and has been closely examined in people with PTSD. The classic HPA axis response to stress is organised in a series of successive steps consisting of complex interactions among the hypothalamus, pituitary gland and the adrenal glands. Upon exposure to stress, the secretion of hypothalamic corticotrophin-releasing factor (CRF) is increased which then stimulates the production and release from the pituitary of adrenocorticotropin hormone (ACTH). In turn, ACTH stimulates the adrenocortical release of cortisol. Cortisol is a major stress hormone affecting many tissues in the body, including the brain. The primary function of cortisol is to increase blood sugar through

gluconeogenesis, suppress the immune system, and assist in fat, protein, and carbohydrate metabolism i.e. adjusting physiological responses and behaviour to the stressor. Feedback loops are important to the function of the HPA axis. Cortisol produced in the adrenal cortex exerts negative feedback to inhibit both the hypothalamus and the pituitary gland by binding to receptors.

A number of studies, discussed below, have found a dysregulation of the HPA axis amongst people with PTSD. Acute stress would normally activate the HPA axis. However, a number of studies have found paradoxically lower levels of blood and urine cortisol concentration amongst people with PTSD when compared with control groups (Heim & Nemeroff, 2009; Yehuda, 2006). Findings are controversial, differ across studies, and may result from differences in trauma exposure amongst other variables (Meewisse, Reitsma, de Vries, Gersons, & Olf, 2007). Lowered cortisol levels (hypocortisolemia) are believed to be a consequence of increased sensitivity of the HPA axis to negative feedback, a hypothesis supported by low dose dexamethasone suppression tests which have found increased binding of cortisol to receptors (Yehuda, 2006). Given that cortisol is normally important in restoring homeostasis after the stress response, it is thought that trauma survivors with low cortisol experience a longer and more distressing response, rendering them at risk for PTSD (Heim & Nemeroff, 2009). However, whilst cortisol levels may be lowered, increases in CRF have been measured in the spinal fluids of people with PTSD (Baker, West, Nicholson, Ekhtor, Kasckow, et al. 1999; Bremner, Licinio, Darnell, Krystal, Owens et. al, 1997). Increased CRF activity in the central nervous system has been hypothesised to promote some of the key symptom features of PTSD e.g. increased

startle response, conditioned fear response, sensitisation to exposure to stressors, and increased arousal. Thus, PTSD may involve elevated levels of hypothalamic CRF activity and a corresponding down-regulation of CRF receptors (Yehuda, 2006). The discrepant findings may be a reflection of a compensatory adaptation of the HPA axis to persistently elevated levels of central CRF secondary to the traumatic experience.

Changes that are adaptive during periods of acute stress cease to be adaptive beyond the period of danger. The sensitisation of neurobiological systems involved in the stress response, may contribute to heightened sensitivity and vigilance when exposed to future stressors. Prospective studies have demonstrated that low levels of cortisol in the immediate aftermath of a traumatic event may be associated with the development of PTSD (Resnick, Yehuda, Pitman, & Foy, 1995; Yehuda, McFarlane, & Shalev, 1998) suggesting that hypocortisolemia may be a pre-existing risk factor associated with maladaptive responses to stress. Administration of hydrocortisone treatment (aimed at increasing cortisol levels) directly after exposure to trauma has been shown to prevent PTSD (Aerni, Traber, Hock, Roozendaal, Schelling, Papassotiropoulos, Nitsch, Schnyder, & de Quervain, 2004; Shellinga, Kilgera, Roozendaal, de Quervain, Briegela, Dagea, Rothenhauslerb, Krauseneckb, Nollertc, & Kapfhammerb, 2004; de Quervain; 2008), presenting important treatment implications for the prevention of the development of PTSD.

Some studies have also found changes in brain structure and function amongst people with PTSD. Whilst many parts of the brain are likely to be involved in PTSD, there are three areas of the brain in particular that may be functionally altered: the hippocampus, prefrontal cortex, and the amygdala. The hippocampus is involved in the

control of stress responses, memory and fear conditioning and is responsible for inhibiting the HPA axis. A number of structural imaging studies have shown reduced volume of the hippocampus among people with PTSD (Bremner, Elzinga, Schmahl, & Vermetten, 2007), in Vietnam war veterans (Bremner, Randall, Vermetten, Staib, Bronen, et al., 1995; Gurvitis, Shenton, Hokama, Ohta, Lasko, Gilbertson, Orr, Kikinis, Jolesz, & McCarley, 1996) and women with abuse related PTSD (Stein, Koverola, Hanna, Torchia, & McClarty, 1997), but not children with PTSD (De Bellis, Keshavan, Clark et al., 1999). In these studies small hippocampal volumes were generally associated with the severity of the trauma and the memory impairments of those with PTSD symptoms. Researchers hypothesise that hippocampal volume reduction in PTSD may reflect either toxic effects of chronically elevated levels of glucocorticoid exposure, increased sensitivity to cortisol, or that a small hippocampus may be a pre-existing vulnerability factor to PTSD (Heim & Nemeroff, 2009). Hippocampal deficits may contribute to PTSD symptoms by promoting the activation of, or the failure to shut down the stress response.

The medial prefrontal cortex comprises of the anterior cingulate cortex, subcallosal cortex and the medial frontal gyrus. The medial prefrontal cortex is connected with the amygdala and may play a role in the stress response through inhibitory effects upon the amygdala (Heim & Nemeroff, 2009; Newport & Nemeroff, 2003), mediating the extinction of conditioned fear. People with PTSD have exhibited reduced volume of the prefrontal cortex (Rauch, Sin & Segal, 2003) and the anterior cingulate cortex (Woodward, Kaloupek, Streeter, Martinez, Schaer, & Eliez, 2006; Yamasue, Kasai, & Iwanami, 2003). A recent twin study suggests that the volume loss

in the anterior cingulate cortex is correlated with the development of PTSD, rather than being a pre-existing risk factor (Kasai, Yamasue, Gilbertson, Shenton, Rauch, & Pitman, 2008). In addition, some studies have demonstrated reduced activation of the medial prefrontal cortex in people with PTSD in response to emotional stimuli related and unrelated to the trauma (for a summary see Heim & Nemeroff, 2009). Combined, findings suggest that alterations in prefrontal cortical activity may explain the deficits in explicit memory function and the extinction of fear often seen in PTSD.

The amygdala is an important limbic structure involved in emotional processing, the formation of emotional memories (especially fear-related memories), and the acquisition of the fear response. There is no evidence of structural changes to the amygdala in PTSD but functional neural imaging studies have found hypersensitivity of the amygdala in PTSD during presentation of traumatic scripts, cues and reminders of the trauma (Liberzon, Sripada, 2008; Shin, Rauch, & Pitman, 2006). Increased amygdala responses to emotional stimuli unrelated to the trauma has also been shown in people with PTSD (Shin, Rauch, & Pitman, 2006). The direction of this relationship remains unclear and it is currently unknown whether increased amygdala activity represents a biological risk factor for PTSD or a consequence of trauma. Robust evidence from prospective research is required to determine the direction of this relationship.

Neurotransmitter systems comprising a connected network of brain regions involved in the regulation and integration of the stress and fear responses have also been implicated. The bulk of the research has focussed on Norepinephrine (NE) which is understood to play a central role in regulating arousal and autonomic stress

responses, as well as promoting the encoding of emotional memories. Interestingly, increased urinary concentrations of both norepinephrine and epinephrine (EPI) have been found in people with PTSD (Heim & Nemeroff, 2009; Newport & Nemeroff, 2003) and this finding has been replicated in children experiencing abuse-related PTSD when compared with a control group and children with non-trauma related anxiety (De Bellis, Baum, Birmaher, Keshavan, Eccard, Boring, Jenkins, & Ryan, 1999). Research has also demonstrated that the administration of the 2 receptor antagonist yohimbine, which results in increased NE activity, induces flashbacks and panic attacks in people with PTSD (Southwick, Morgan, Charney, & High, 1999). Therefore, a number of researchers have concluded that increased central nervous system NE activity may contribute to some of the core symptoms of PTSD, such as hyperarousal, increased startle, and encoded fear memories (Heim & Nemeroff, 2009; Strawn & Geraciotti, 2008).

In summary, neurobiological alterations have been observed in PTSD, however, findings provide an incomplete picture of the neurobiology of PTSD and provide a template for further research. It remains unclear whether neurobiological alterations are a direct consequence of PTSD, a consequence of biological adaptations to trauma exposure independent of PTSD, or are pre-existing risk factors for the development of PTSD. As previously stated, robust evidence from prospective research is required to determine the direction of relationships between PTSD and neurobiological alterations. Future neurobiological research is likely to focus on integrating the disparate findings within and among multiple biological systems.

Models of Childhood PTSD

Models of childhood PTSD have been proposed (Fletcher, 1996; Pynoos et al., 1999) or adapted from adult cognitive behavioural models of PTSD (Ehlers & Clark, 2000; Meiser-Stedman, 2002) to help understand PTSD in children and adolescents. Two models, Fletcher (1996) and Pynoos et al. (1999) are most suited to the examination of within individual and environmental risk factors in the development of PTSD and are discussed herein.

Fletcher's working model of the context for the development of childhood PTSD

Fletcher (1996) proposed a multi-factorial model similar to that of Pynoos and colleagues (1999) delineating the process by which multiple factors contribute to the development of childhood PTSD. Fletcher's (1996) emphasises the importance of considering the contribution of multiple factors, in addition to the traumatic event, to achieve a thorough understanding of PTSD in children and adolescents. As illustrated in Figure 1, there is an emphasis on moderating characteristics of both the individual and the broader social environment of the child operating throughout the conceptualisation of childhood PTSD. Individual characteristics such as genetic or biological vulnerabilities, psychological strengths and vulnerabilities, history of psychological problems, history of stressful events, gender, age, ethnicity, cognitive capacity, and coping styles are incorporated as are environmental factors such as social support, parenting style, family composition and stress, family history of psychopathology, socio-economic status, and post-trauma stressors.

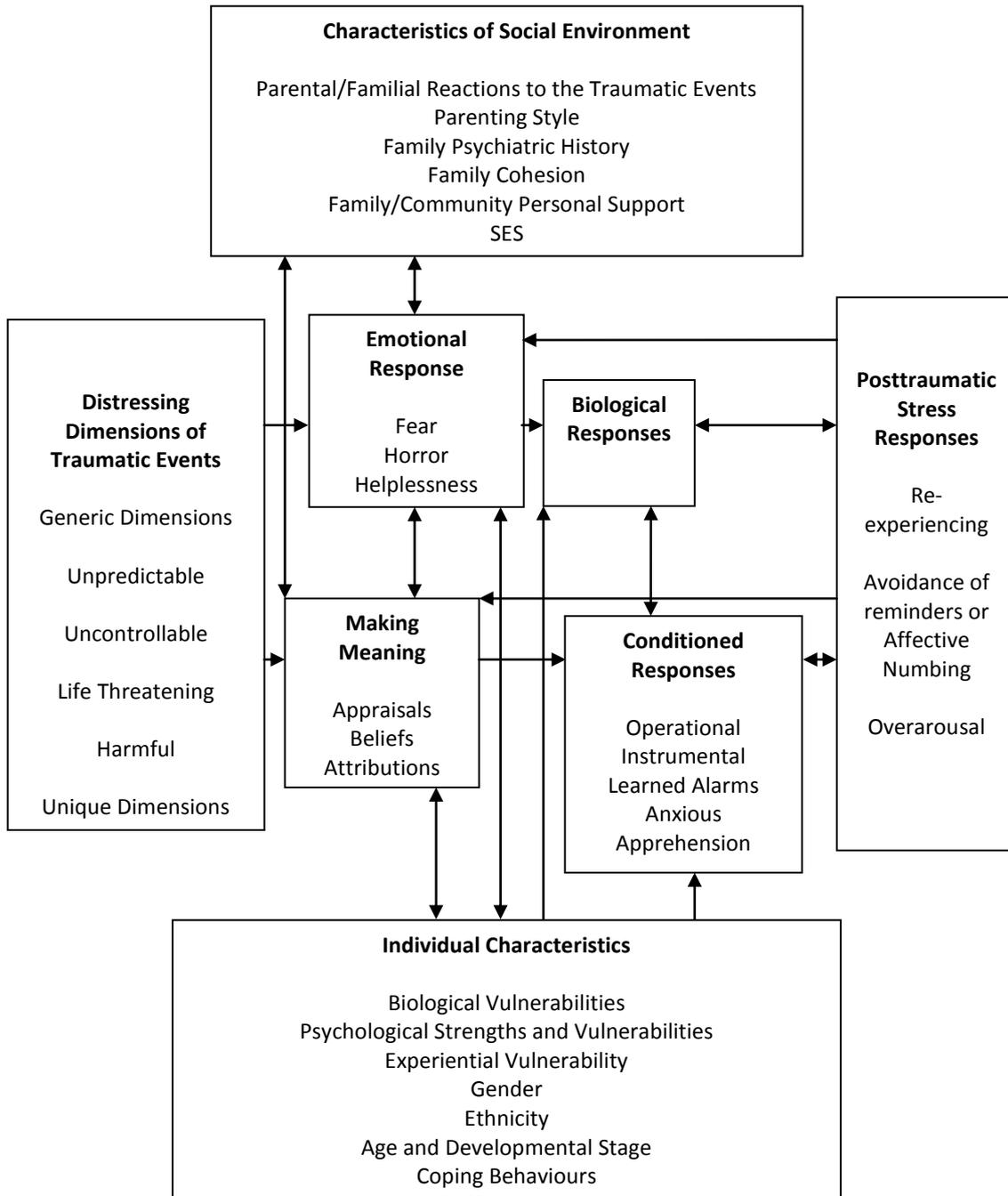


Figure 1. Working model of the context for the development of childhood PTSD (Fletcher, 1996).

Characteristics of the traumatic event (e.g. type, cause, severity, duration) and the aforementioned individual and environmental factors interact with the following four responses to contribute to the development of PTSD in children and young people: (i) cognitive (e.g. cognitive appraisal, beliefs and attributions); (ii) emotional (e.g. the experience of fear, horror and helplessness); (iii) physiological (e.g. biological responses and neurobiological changes), and; (iv) behavioural (e.g. conditioned responses to internal and external cues and anxious apprehension).

Pynoos' developmental psychopathology model of childhood traumatic stress

Pynoos and colleagues (Pynoos, 1994; Pynoos, Steinberg, & Goenjian, 1996; Pynoos, Steinberg, & Piacentini, 1999; Pynoos, Steinberg, & Wraith, 1995) have proposed a developmental life-trajectory model to explain the complexity of child and adolescent reactions to trauma as illustrated in Figure 2. After exposure to a potentially traumatic event, a child's short-term reaction to a trauma is considered to be moderated by four groups of factors: (i) proximal trauma reminders (e.g., external and internal reminders of the trauma, emotional and physiological reactions); (ii) proximal secondary stresses (e.g., changes to family and community circumstances, parental loss, disruption to schooling); (iii) the "ecology" of the child (e.g., parental, family, school, peer, and other social factors); and (iv) factors intrinsic to the child (e.g., genetic predisposition, age, gender, developmental competencies, temperament, pre-existing psychopathology, attachment styles, and previous experiences). Pynoos and colleagues (1999) argued that the etiology of acute posttraumatic distress is related to

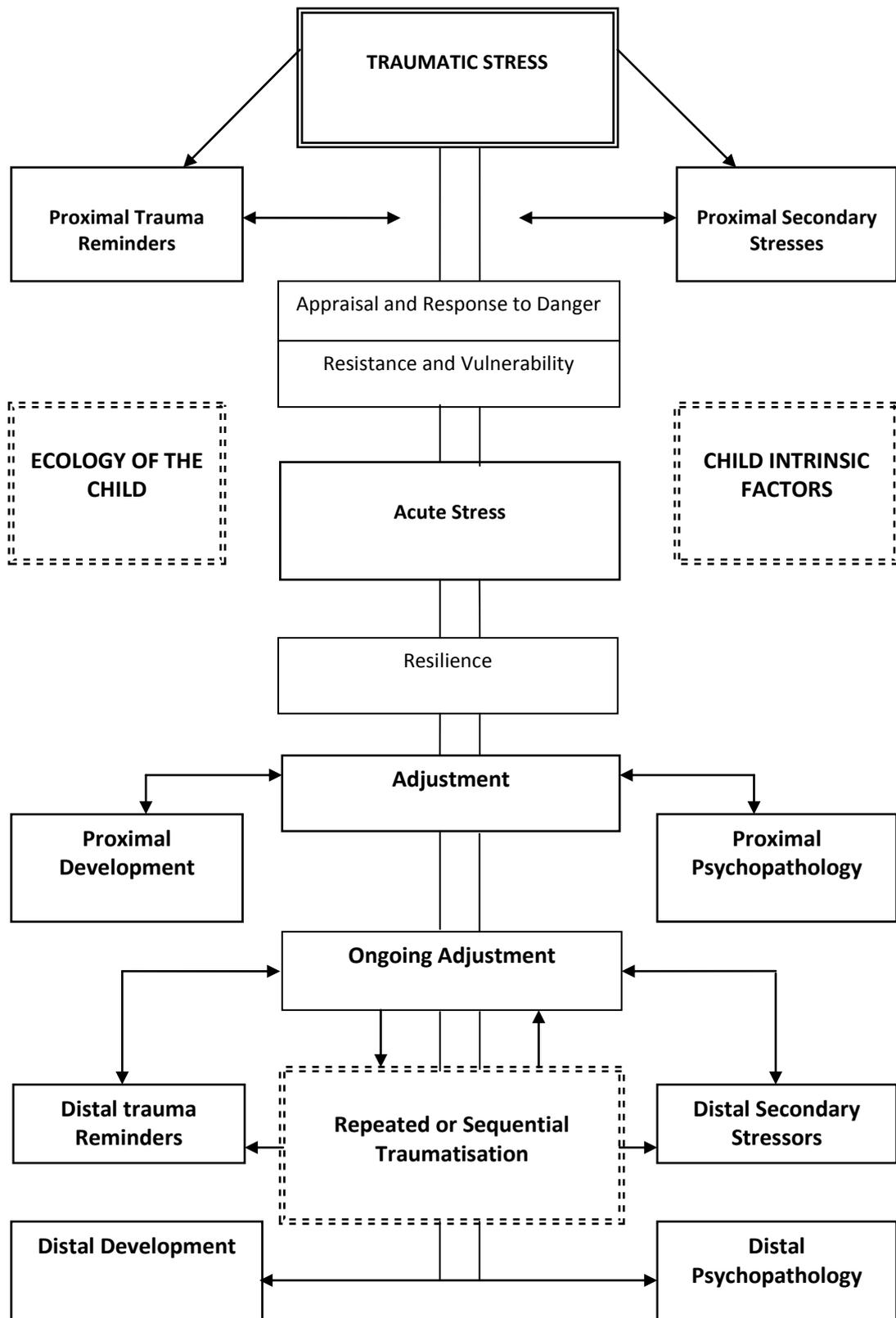


Figure 2. Developmental model of childhood traumatic stress (Pynoos, Steinberg, Piacentini, 1999).

the nature of the traumatic experience(s) and from the subsequent traumatic reminders and secondary stressors. Ongoing reminders of the trauma and persistent secondary stressors (e.g., chronic adversities, physical disability, and judicial proceedings) are likely to be related to the child's longer term (distal) reactions and adjustment and may impede recovery. The holistic conceptualisation of child and adolescent PTSD proposed by Pynoos et al. (1999) recognises and integrates complex acute, short-term, and long-term interactions amongst a multitude of variables that impact on a child's adjustment and developmental outcomes. Particularly relevant for children, the developmental conceptualisation proposed by Pynoos et al. (1999) recognises stage-related vulnerabilities in the aftermath of trauma and emphasises the potentially dramatic impact of the sequelae and legacy of trauma.

Summary of conceptual models of childhood PTSD

A number of conceptual models have been proposed to delineate the complex interaction of multiple factors in the development of post-trauma reactions among children and adolescents. The two models discussed above (Fletcher, 1996; Pynoos et al., 1999) are particularly relevant to the exploration and understanding of within individual and environmental risk factors, and also incorporate the complex interactions between cognitive, emotional, physiological, and behavioural responses in light of the current research enhancing our understanding of PTSD among children and young people. Both models integrate existing psychodynamic, familial, cognitive-behavioural, and psycho-pharmacological approaches to child and adolescent trauma reactions and help to explain the large degree of individual variability following

exposure to potentially traumatic events. Whilst Fletcher's (1996) model is somewhat less complex than the model proposed by Pynoos and colleagues (1999), the latter model places greater emphasis on developmental aspects including the acute, short-term and long-term factors involved in the development of post-trauma psychopathology and the multitude of possible outcomes in addition to PTSD.

Risk Factors for PTSD

Unlike the changes in conceptualisation proposed by the DSM-III that were prompted by the political and social context of the 1970's, the second turning point in the conceptualisation of PTSD was born from empirical data. Large-scale epidemiological surveys during the 1990's failed to support the notion that PTSD was a normal reaction to an abnormal circumstance. The U.S. National Comorbidity Survey (Kessler et al., 1995) indicated that 41.2% of women and 61% of men had been exposed to traumatic events. Similar rates of traumatic exposure were replicated in Australia (Creamer et al., 2001) and over the years, evidence has accumulated to indicate a wide variation in the development of PTSD after exposure to different stressors, supporting the notion that exposure alone does not sufficiently explain the development of PTSD. Furthermore, other diagnoses of major depression, anxiety, and substance use have also been directly linked to trauma (Breslau, Davis, & Shultz, 2003). Thus, there is a growing awareness that the development of PTSD may vary across individuals as a function of both environmental and within-individual risk factors, and current models of PTSD underscore the role of premorbid vulnerability in the aetiology of the disorder.

Although not radically different to the DSM-III-R, the description of PTSD has been refined in the DSM-IV. The current emphasis on premorbid vulnerability is a reversal from the initial conceptualisation in the DSM-III and can be partially credited to the well-known Detroit Area Survey of Trauma directed by Naomi Breslau (Breslau et al., 1991). This cross-sectional study examined a random sample of 1007 young adults aged 21-30 years from a large health maintenance organisation in Detroit, Michigan, U.S. Breslau et al (1991) demonstrated that individual vulnerabilities acted as potential risk factors, contributing to the development of PTSD in young adults. A number of factors were associated with exposure to trauma in this population, such as, low education, male sex, early conduct problems, extraversion and a history of psychological disorder or substance abuse or dependence. Following exposure to trauma, four factors were associated with the development of PTSD: early separation from parents, neuroticism, premorbid anxiety and depression, and a family history of anxiety. Thus, Breslau et al concluded that personal predispositions to the PTSD effects of traumatic events might be responsible for a substantial part of the development of PTSD in this population. One significant limitation was the cross-sectional nature of this study, and Breslau et al (1991) called for prospective longitudinal studies in which risk factors are measured before onset of PTSD.

This new paradigm readdresses the issues of vulnerability and risk factors that the DSM-III definition had withdrawn. However, instead of returning to the previous view of suspicion and blame, the elaborated theory of PTSD has introduced notions of risk factors, vulnerability and resilience (Rechtman, 2004). The new paradigm, heralded as a “rebirth of PTSD” by Rechtman (2004), can easily incorporate issues of

comorbidity, degree of exposure, and protective factors that were unable to fit into a model that viewed PTSD as a normal reaction to an abnormal experience. It would seem that the pendulum has returned to the centre, with many of the conceptual ideas inherent to the diagnosis of traumatic neurosis returning to the theoretical framework, but with a very different moral meaning advancing our understanding and empathy. Just as developmental factors interact to contribute to risk or resilience for most medical and psychological problems (for example, not all cigarette smokers develop cancer, regardless of using the same amount and type of tobacco), similarly, not all individuals exposed to the same type or severity of trauma will develop symptoms of PTSD. Pynoos, Foy and colleagues (Foy et al., 1996; Foy, Osato, Houskamp, & Neumann, 1992; Pynoos et al., 1999) have been instrumental in developing new aetiological models of PTSD incorporating new data and the new paradigm. These authors propose interactional models that include both environmental ('ecology of the child') and within-individual ('child intrinsic') factors in the development and maintenance of PTSD symptoms.

More recently, Koenan and colleagues (Koenen, Moffit, Poulton, Martin, & Caspi, 2007) also concluded that both within-individual childhood characteristics and environmental conditions increased risk of developing PTSD in their longitudinal study design. Koenan and colleagues (Koenen et al., 2007) have been one of the first to examine childhood risk factors for the development of PTSD in adults using a longitudinal design. These researchers examined onset of PTSD both prior to age 26 (part A), and between ages 26 and 32 (part B), and used a New Zealand birth cohort. The first study (part A) was limited in that the interview did not ascertain age of onset

of PTSD. Therefore as childhood factors were measured before age 11, it was inferred that these preceded PTSD onset. Thus, this component of the study must be interpreted with caution as it is not a prospective longitudinal design. The authors did however, in the same paper, follow with an examination of risk factors for PTSD onset after age 26 in a sub-sample of 35 adults who developed PTSD between the ages of 26 and 32 (part B). Therefore the authors were able to document with certainty, using a prospective longitudinal design, the childhood risks that were antecedent to PTSD after age 26 in this adult sample. They found that IQ at age five years old, and antisocial behaviour in childhood were robust predictors of PTSD after age 26 years old. Family environmental factors were less significant predictors for PTSD in adults, except for low socio-economic status, which remained a strong predictor of PTSD.

A range of individual and environmental factors have been implicated in the development of PTSD symptoms after trauma exposure. However, throughout the growing body of PTSD literature, the findings of many previous risk factor studies are limited by a number of methodological problems such as cross-sectional retrospective study designs, use of single informants, small sample sizes, reliance upon questionnaire results rather than diagnostic interviews, and a reliance upon clinical rather than community samples. Although childhood factors have been associated with an increased risk of developing PTSD in adulthood, most studies have assessed only a limited number of childhood factors and, as discussed, have often used retrospective study designs.

The largest sample in which PTSD has been studied is male combat veterans, but it is unknown whether risk factors discovered in combat veteran studies, generalise

to civilian mixed gender, child and youth samples. Furthermore, there is limited research directly focussing on samples of Australian children and young people. Much research has attempted to infer risk factors by comparing participants who had already been exposed to trauma or developed PTSD, with a control group. PTSD diagnostic criteria requires that the cluster of PTSD symptoms continue for at least one month. Therefore, many prospective studies have begun assessment within days of trauma exposure, before a diagnosis of PTSD can be made, but are not truly prospective in that they have not measured factors prior to trauma exposure (Brewin, Andrews, & Valentine, 2000). It can be argued that post-trauma risk factors may differ from pre-trauma risk factors and that exposure to trauma and early PTSD symptoms may bias retrospective reports. There have been very few prospective longitudinal studies of PTSD that have measured risk factors before the exposure to trauma and subsequent development of PTSD.

Whilst recent research has begun to shed more light onto specific vulnerabilities and risk factors, many questions remain unanswered. To the best of this author's knowledge, no published study has yet used a prospective longitudinal design to test possible pre-trauma environmental and within-individual risk factors of PTSD in Australian children or young people. Therefore, prospective longitudinal studies examining risk factors for PTSD, measured pre-trauma, are cutting edge in the world literature on PTSD. Consistent with this assertion, the consensus statement update on PTSD from the International Consensus Group on Depression and Anxiety (Ballenger et al., 2004) has concluded that further longitudinal research on the natural course of PTSD or other psychological disorders in people who have experienced early life

trauma is needed. Whilst these authors acknowledged that there was some data on the influence of childhood adversity on the psychiatric development during adolescence and adulthood, it was identified at this conference and in the subsequent publication, that additional studies were needed. The current study aims to help fill this void in the current PTSD literature. Given that this thesis aims to test a larger number of risk factors for the development of PTSD in young people, the risk factors have been divided into two main clusters as suggested by Pynoos and colleagues (Pynoos et al., 1999). These clusters are 1. Within Individual Factors and, 2. Environmental Factors. This study also aims to explore a range of such risk factors using a prospective longitudinal design. The following section discusses these risk factors in further detail.

Within Individual Risk Factors

Female Gender. One of the most consistent findings in the epidemiology of PTSD is the higher risk of this disorder in women (Breslau, Davis, Andreski et al., 1997; Giaconia et al., 1995; Green et al., 1991; Green, 1991; Holbrook & Hoyt, 2004; Holbrook et al., 2005; Holbrook, Hoyt, Stein, & Sieber, 2001, 2002; Lonigan, Shannon, Finch, Daugherty, & Taylor, 1991; Michaels et al., 1999; Pynoos, Goenjian, & Tashjian, 1993; Shannon et al., 1994; Stein, Walker, Hazen, & Forde, 1997; Storr, Ialongo, Anthony, & Breslau, 2007). In a large meta-analysis of adult risk factors, female gender was found to reliably increase risk for PTSD in civilian samples (Brewin et al., 2000). Current research indicates higher rates of PTSD for women for three reasons. Firstly, women's increased risk for PTSD may be explained, in part, by a greater exposure to traumas with a higher probability of PTSD (such as interpersonal

assaults like sexual abuse and rape), and at a younger age. Thus, women may experience a situational vulnerability related to type of trauma exposure.

A growing number of studies have found that a higher female risk of exposure to potentially more traumatising events cannot sufficiently explain gender differences in PTSD. For example, the National Comorbidity Survey estimated the risk of developing PTSD after trauma exposure at 8.1% for men and 20.4% for women. These findings are consistent with other studies indicating a gender difference in the probability of PTSD after exposure to trauma. One Scandinavian study found that controlling for trauma type did not account for gender differences, but controlling for experienced distress did, suggesting that gender differences in PTSD are partly related to a greater vulnerability to stress in women (Frans, Rimmo, Aberg, & Fredrickson, 2005). Therefore, the second reason behind higher rates of PTSD for women, may be due to an intrinsic vulnerability to the effects of traumatic events. Other studies have found that women have stronger perceptions of threat and loss of control and higher levels of peritraumatic dissociation (Olf, Langeland, Draijer, & Gersons, 2007).

Thirdly, men may be less likely to receive a diagnosis of PTSD due to our current definition of the disorder. The current diagnostic criteria for PTSD is centred around the subjective cognitive and emotional responses to traumatic events and it may be that post-traumatic symptoms manifest differently in men. Men may be less likely to report anxious and depressed symptoms, and more likely to report behaviour and substance use problems, irritability, anger and violence. In summary, there might be a number of reasons why women are more likely than men to develop PTSD once exposed to trauma.

Despite considerable support for gender differences in American samples, Australian studies have found mixed results. Earlier Australian epidemiological studies did not find a gender difference between rates of PTSD for Australian men and women (McLennan, 1997). The more recent 2007 Study of Mental Health and Wellbeing of Adults confirmed that PTSD was more common amongst Australian women (8.3% 12-month incident rate) than Australian men (4.6% 12-month incident rate). In addition, gender differences in PTSD have been found to increase with age, as they do for many internalising symptoms (Vogel & Vernberg, 1993). No epidemiological studies of Australian children and young people have reported results for PTSD, therefore exact prevalence rates and the degree to which there are gender differences in Australia remains unknown at this time (Sawyer et al., 2001). The National Survey of Mental Health and Wellbeing: The Child and Adolescent Component (Sawyer, Kosky et al., 2000) found no difference for anxiety/depression for boys (3.9%, $n = 2082$) and girls (3.2%, $n = 2001$) although rates of PTSD were not reported (Sawyer et al., 2001). A handful of American studies have examined gender differences in children. Cuffe and colleagues (Cuffe et al., 1998) found gender differences in rates of PTSD for adolescents, with approximately 3% of females and 1% of males experiencing clinical PTSD. The National Survey of Adolescents in the U.S. also found a gender difference, with 10% of girls and 6% of boys aged 12-17 years old meeting criteria for PTSD (Acierno, Kilpatrick, Resnick, & Saunders, 2000). Similarly, the data from the youngest cohort of the American National Comorbidity Survey (ages 15-24 years old) found that 10% of females and 5% of males experienced a lifetime diagnosis of PTSD (Kessler et al., 1995). Amongst high risk populations, Green and colleagues (1991)

examined children aged 5 to 15 years that had been exposed to the Buffalo Creek dam collapse and found that 44% of females compared with 30% of males met criteria for PTSD after the disaster. Gwadz and colleagues (2007) found gender differences amongst a sample of 85 homeless youth. Once again, about 10% of girls and 6% of boys met diagnostic criteria for PTSD.

Although females were more likely to meet criteria for PTSD amongst a sample of 5687 American school children exposed to a natural disaster (Shannon et al., 1994), Hurricane Hugo in South Carolina, this finding was not replicated among Australian school students exposed to bushfires. McDermott and colleagues (McDermott & Palmer, 2002) surveyed primary and secondary school students between the ages of 8 and 19 years old following exposure to bushfires in the state of New South Wales, Australia, and in contrast to most other published studies, these researchers did not find gender differences in reported anxiety and depression symptomatology amongst this sample. In summary, there appears to be a clear pattern that females of all ages are usually more likely than males to develop PTSD after trauma. The above competing findings from Australian research raise the question of whether gender differences exist among Australian samples of children and young people.

Internalising Symptoms. Research on the structure and organisation of psychological disorders in children suggests that patterns of psychological problems tend to cluster along the dimensions of internalisation and externalisation. The distinction between internalising and externalising disorders originated from research

in the area of childhood behaviour disorders (Achenbach & Edelbrock, 1978) and has become well known in the field of child psychology. The two constructs have been examined by recent factor-analytic studies of the structure of adult psychological disorders (Kendler, Prescott, Myers, & Neale, 2003). Studies suggest that the primary personality characteristics for the internalising disorders is high negative emotionality, whilst low constraint or impulsivity has been implicated as the primary characteristic for the externalising disorders (Krueger, McGue, & Iacono, 2001). Internalising behaviour problems affect the child's internal psychological environment rather than the external world and include withdrawn, anxious, inhibited, and depressed behaviours (Eisenberg et al., 2001). PTSD, as it is currently defined, primarily loads on the internalising dimension (Cox, Clara, & Enns, 2002) and is highly comorbid with other internalising disorders (Kessler et al., 2005; Yehuda et al., 1995). Given that PTSD is strongly associated with internalising symptoms, and that children with internalising behaviour problems are more likely to grow up to become depressed and anxious (APA, 1994), it is logical to include internalising behaviour as a potential risk factor for PTSD in young people.

Few studies have examined the association between early internalising behaviours and PTSD, and those that have been published have found mixed results. Aaron et al. (1999) examined 40 children between the ages of 8 and 17 after hospitalisation for a severe or life threatening physical injury. This study found that parent reported pre-trauma internalising behaviours were associated with increased symptoms of PTSD following injury. However, Breslau and colleagues (2006) found that high internalising scores measured at age 6, did not increase risk of PTSD in

young people at age 17 on The Teacher Rating Form (Achenbach, 1991c). This finding seems to contradict other findings linking anxiety sensitivity with PTSD and requires further research. Studies examining specific internalising disorders (depression and anxiety) and anxiety sensitivity as risk factors for PTSD are discussed below.

Premorbid depression and anxiety disorders. A large body of research has found a link between premorbid psychological problems and the development of PTSD in adult populations (Ozer, Best, Lipsey, & Weiss, 2003). Pre-existing psychopathology is believed to be a risk factor for PTSD in children, young people and adults (Brewin et al., 2000; Ozer et al., 2003), although research with child and youth samples is limited. As previously discussed clinical and epidemiological data in PTSD populations support a high comorbidity with a broad range of emotional and behavioural problems (Breslau et al., 1991; Goenjian et al., 1995; Holbrook et al., 2005; Jordan et al., 1991; Kessler et al., 1995).

With respect to the association between PTSD and major depression, a number of cross-sectional studies using retrospective self reported lifetime histories, have identified pre-existing major depressive disorder as a risk factor for exposure to trauma (Breslau et al., 1991; Breslau, Davis, Peterson, & Schultz, 1997; Bromet, Sonnega, & Kessler, 1998; Kessler et al., 1995) and for increasing susceptibility to PTSD following exposure to traumatic events (Breslau, Davis, Peterson et al., 1997; Bromet et al., 1998; Fullerton et al., 2000; Kessler et al., 1995). Overall, cross-sectional findings suggest that the association between PTSD and major depression might be explained by a number of causal pathways. Firstly, pre-existing major depression may

increase susceptibility to the PTSD-inducing trauma effects and predict PTSD symptoms after exposure (Breslau, Davis, Peterson et al., 1997). Pre-existing depression may influence the cognitive appraisal of traumatic events contributing to a cognitively-based sensitivity and greater susceptibility to distressing emotional reactions after traumatic exposure. Secondly, major depression may be involved in PTSD sequelae, as an outcome of the PTSD disorder. As previously discussed, PTSD is chronic and impairing and causes significant distress to those with this diagnosis. Given that PTSD has been found to increase the risk of onset of first episode of major depression (Breslau, Davis, Peterson et al., 1997), the relationship between the two diagnoses may be bi-directional. Thirdly, major depression may increase the likelihood of experiencing a PTSD inducing traumatic event (Breslau, Davis, Peterson et al., 1997). Longitudinal studies measuring psychological functioning prior to trauma, and examining the temporal sequencing of PTSD and other highly comorbid disorders is needed to clarify whether these disorders are distinct syndromes and whether some disorders operate as risk factors for the development of others.

In a recent study of 222 adult assault survivors, self-reported premorbid problems with depression and anxiety were found to be among three variables that best predicted PTSD in the six months following the trauma (Kleim, Ehlers, & Glucksman, 2007). This study was similar to other PTSD risk factor studies (Ozer et al., 2003), in that it combined premorbid depression with anxiety as a single predictor which were assessed after exposure to trauma. Therefore, a major limitation in this and other studies include the retrospective nature of psychological histories. Another large study (Zatzick et al., 2007) found that self-reported premorbid depression independently

increased the risk for PTSD up to 12 months post trauma. Once again, whilst longitudinal in design, subjects were recruited post trauma exposure, and self-reported psychological histories were also gathered post-trauma. Studies examining risk factors using retrospective self-reports obtained after trauma exposure have significant limitations given that reports may be biased by current levels of distress and impairment. Moreover, Harvey and Bryant (2000) demonstrated that 75% of individuals with current post-trauma symptoms tend to inaccurately report previous symptoms and concluded that retrospective reports of symptoms should be interpreted with caution.

Ozer et al (2003) conducted a large meta-analytic study of the PTSD risk factor research and reviewed 2647 studies of PTSD. They included 68 studies in a meta-analysis of a number of predictors including prior psychological adjustment. Consistent with the assertions contained in this dissertation, Ozer and colleagues commented that the PTSD literature almost exclusively consisted of retrospective assessment methodology and highlighted that, although it would have been far preferable, the large majority of predictors studied in the meta-analysis were not assessed prior to the development of PTSD symptoms. Overall, 23 studies and a combined total of 6797 participants, contributed to the studies used to assess prior adjustment as a risk factor. It was concluded that individuals who reported problems in psychological adjustment prior to experiencing the target stressor reported higher PTSD symptoms, on average, but the effect was small.

In relation to anxiety as a risk factor for PTSD, although not examining anxiety disorder specifically, three genuinely prospective studies have found that elevated

precombat neuroticism, as measured on the Minnesota Multiphasic Personality Inventory (MMPI), predicted PTSD among war veterans from WWII (Lee, Vaillant, Torrey, & Elder, 1995), the Vietnam War (Shnurr, Friedman, & Rosenberg, 1993), and the U.N. Protection Force mission in the former Yugoslavia (Bramsen, Dirkzwager, & Van-der-Poloeg, 2000). Other studies examining anxiety as a risk factor are cross-sectional in design and have turned more to ‘anxiety sensitivity’ rather than diagnoses, presumably in part due to the methodological difficulties associated with measuring premorbid anxiety diagnoses. Anxiety sensitivity extends beyond anxiety disorders and captures trait like fears of anxiety related sensations. Such studies have used questionnaires to measure anxiety sensitivity soon after trauma, or after the development of PTSD, and have found a strong association between PTSD in adults and anxiety sensitivity amongst female victims of partner violence (Lang, Kennedy, & Stein, 2002) and trauma survivors of car accidents and non sexual assaults (Bryant & Panasetis, 2001). In both studies, subjects with PTSD symptoms were compared with those who had experienced a similar trauma, but had not developed PTSD. Conclusions could not be drawn as to whether anxiety sensitivity was a consequence of trauma and involved in the development or maintenance of PTSD, or a pre-existing vulnerability. Prospective longitudinal study designs would be required to unravel this temporal sequencing.

Studies examining young people have found that, similar to adults, anxiety and depression may act as risk factors for the development of PTSD (Holbrook et al., 2005). The literature is dominated by retrospective study designs. Depression symptoms assessed shortly after violent injury in adolescents aged 12 to 17 years have

been found to be a useful marker for later development of PTSD (Pailler, Kassam-Adams, Datner, & Fein, 2007). However, date of onset for depression was unclear and may or may not have been precipitated by the trauma. Another cross sectional study found trait anxiety to be a strong risk for the development of severe post-traumatic reactions in children (aged 9 to 19 years) exposed to Hurricane Hugo (Lonigan et al., 1994). More than three quarters of the children exhibiting PTSD symptoms scored in the upper quadrant of the distribution of the Revised Children's Manifest Anxiety Scale (RCMAS) scores. Once again, although this is a trait measure of anxiety, conclusions cannot be drawn as to whether results were impacted by exposure to trauma or pre-existing trait anxiety vulnerabilities and prospective longitudinal study designs that measure the factors prior to trauma exposure would be required to determine this with certainty.

There are currently few genuinely prospective longitudinal studies examining depression and anxiety as risk factors for the development of PTSD in children and young people. Storr and colleagues (2007) examined premorbid symptoms as antecedents of PTSD in an American sample of 1698 followed from entry to first grade of a public school system to 21 years. These researchers found that high levels of depressive and anxious problems (as opposed to disorders) at the start of first grade, predicted an increased risk of PTSD following exposure to traumatic events. Not only did depressive and anxious problems at the time of entry into primary school constitute early signs of major depression and anxiety disorders, they also identified children at risk of PTSD in response to traumatic experiences, by age 21 (Storr et al., 2007). Breslau and colleagues (2006) found that the risk for PTSD at age 17 years was

increased for young people with DSM-III-R anxiety disorders at age 6 years. Major depression was not assessed in this study due to a very low prevalence of this disorder amongst 6 year old children. (Breslau, Lucia, & Alvarado, 2006). This study found that the risk for PTSD was increased for American young people with DSM-III-R anxiety disorders. Major depression was not assessed in this study due to a very low prevalence of this disorder amongst six-year-old children (Breslau, Lucia, & Alvarado, 2006).

In conclusion, there is some evidence that premorbid depression and anxiety symptoms may be a risk for PTSD development among young people, but this requires further research and empirical evidence using Australian samples and prospective longitudinal designs.

Externalising and behavioural disorders. In contrast to internalising behaviours, the construct of externalising behaviour problems refers to a cluster of behaviours that are manifested outwardly and reflect negative behaviours in the external environment (Eisenberg et al., 2001). The externalising disorders consist of disruptive, hyperactive, undercontrolled, and aggressive behaviours. Difficult temperament, disruptive behaviours, and hyperactivity are various manifestations of poor self-regulation and difficulty to modulate behaviour according to various situations, and may place a child or young person at increased risk of exposure to trauma. Koenan and colleagues (2003) argued that people with externalising and behavioural problems may also be more likely to develop PTSD due to poor affect tolerance deemed necessary for processing the traumatic event. Instead, they are more

likely to angrily act out and engage in avoidance strategies, such as substance abuse, which are shown to interfere with PTSD recovery in community samples (Koenen, Stellman, Stellman, & Sommer, 2003).

Difficult temperament, antisocial behaviour and being unpopular have been found to significantly increase the odds of developing PTSD as compared to children without such problems (Koenen et al., 2007). Similarly, Breslau et al. (2006) found that young people who had subclinical or clinical externalising problems at age 6 years (measured using the Child Behaviour Checklist - Teacher Rating Form) (Achenbach, 1991c) were at elevated risk for subsequent exposure to any traumatic event at 17 years. Furthermore, they were at increased risk of developing PTSD after trauma exposure.

Conversely, Storr and colleagues (2007) found that children rated as disruptive in Grade 1 at school were at greater risk of exposure to assaultive traumatic events, but not PTSD, at age 21 years. Thus, further research is needed to examine the risk of earlier externalising behaviours, including substance use specifically, for exposure to trauma and PTSD development in an Australian sample.

Premorbid substance use disorders. When considering the dangers of substance use, it is important to reflect on the Australian legal drinking age of 18 years and the high rates of underage drinking, and binge drinking in Australia. Alcohol and the abuse of substances, and the associated reduced inhibitions and impaired judgement may contribute to placing young people in high-risk situations where exposure to traumatic events may be more likely. Substance use disorders may not

only facilitate exposure to trauma, but may also contribute to a diminished ability to cope with trauma afterwards. Biologically, premorbid substance use disorders may also lead to increased levels of arousal, as well as sensitisation of neurobiological stress symptoms. Most research examining the causal pathways for PTSD and substance use problems has used adults or young adults from age 19 years and originates in the US where the legal drinking age is 21 years (Reed, Anthony, & Breslau, 2007). It remains unclear as to whether these results can be generalised to Australia where there exists a younger legal drinking age. Given that published studies tend to focus on adult populations and are cross-sectional in design, further exploration of the temporal sequencing of substance use disorders and PTSD in Australian young people aged 15 to 20 years is needed. The degree to which alcohol and substances play a role in the risk for PTSD may be age specific, with younger alcohol and other substance users at increased risk for subsequent PTSD.

As previously discussed, alcohol and substance use disorders are highly comorbid with PTSD and there is substantial research to indicate that pre-existing PTSD increases the risk of subsequent substance use disorders (Chilcoat & Breslau, 1998; Reed et al., 2007), supporting the self medication hypothesis. Breslau and colleagues (Reed et al., 2007) recently found that prior PTSD was associated with excess risk for drug abuse or dependence, after controlling for trauma exposure and childhood factors (including conduct problems, academic achievement, socio economic status, cognitive problem solving and temperament). In support of the self-medication hypothesis, these authors confirmed that PTSD might be a causal determinant of drug use disorders in young adults (aged 19-24 years old). However,

this study was designed to specifically explore the sequelae of trauma and PTSD and only examined young adults without prior drug use disorders.

In one study of over 300 young people, a lifetime diagnosis of PTSD by age 18 significantly increased the risk of other lifetime diagnoses including alcohol and drug dependence (Giaconia et al., 1995). Giaconia and colleagues (1995) examined the temporal sequencing of these disorders and concluded that PTSD preceded the onset of drug dependence in 67% of young people with PTSD. Similarly, 45.5% had PTSD prior to alcohol dependence. However, despite being widely reported in the youth PTSD literature, these conclusions were drawn from very small sample sizes ($n = 4$ and $n = 5$ respectively). Therefore, one must interpret the results with caution.

Some studies have found that the self-medication hypothesis may not fully explain the comorbidity between PTSD and substance use disorders, and that drug and alcohol use disorders increase the risk of exposure to trauma. In 1991, Breslau and colleagues first found that substance abuse was a risk factor for exposure to trauma amongst young adults (Breslau et al., 1991) but in later examination, Chilcoat and Breslau (1998) did not find evidence to support this view. Examining a slightly younger cohort of 3021 German young people aged 14 to 24 years, other researchers have also found that premorbid substance use disorders increased risk of exposure to traumatic events, but did not predict the onset of PTSD (Perkonigg et al., 2000). In addition, other studies have found that binge drinking is a major risk factor for head trauma from assaults, falls, and biking accidents amongst young people (Savola, Niemela, & Hillbom, 2005), and that alcohol and other substance use (predominantly cannabis) are also linked to motor-vehicle trauma among young Australians (Sugrue et

al., 1995). However, there is little evidence from prospective longitudinal studies to indicate that premorbid alcohol and substance use increases the risk for the development of PTSD. Rather studies have found that substance use disorders are generally secondary to onset of PTSD in young people (Perkonigg et al., 2000; Reed et al., 2007).

In 1992, data from the previous cross-sectional ECA study suggested that, in a significant proportion of adult participants, drug and alcohol abuse was self reported to have preceded onset of PTSD (Cottler, Compton, Mager, Spitznagel, & Janca, 1992). Studies examining the temporal sequencing of PTSD and substance use disorders amongst young people have found mixed results. Another study examining the rates of PTSD amongst young people (aged 15-19 years) with substance use disorders, concluded that onset of substance use disorders and PTSD were intertwined (Deykin & Buka, 1997).

There may be gender differences in the temporal sequencing of PTSD and substance use symptoms. Deykin and associates (1997) found that PTSD tended to precede the onset of chemical dependence among female adolescents aged 15-19 years, supporting the self-medication hypothesis that females use alcohol and other drugs as a way of deadening the psychic discomfort of PTSD. However, the reverse was true for males of the same age. Among males, substance dependence appeared to precede PTSD, suggesting that substance use led to behaviours and interactions that enhanced trauma occurrence and perhaps vulnerability to the effects of trauma. Their results showed that 59% of the females in the study developed PTSD prior to substance use disorder (in support of the self medication hypothesis for females). In

contrast, 72% of the males had substance use disorders prior to PTSD suggesting an alternative pathway for males.

The temporal sequencing of PTSD and substance use may impact on treatment options and their effects. Back and colleagues (2005) explored differences in clinical presentation and the response to cognitive-behavioural substance-use therapy, comparing the order of onset of the two disorders. They examined 94 men and women with comorbid alcohol dependence and PTSD and found differing results amongst men and women. Women with alcohol dependence as the primary diagnosis and men with PTSD as the primary diagnosis presented with higher levels of stress and depression. In general, those with PTSD as the primary diagnosis gained greater benefit from the CBT program than those with alcohol dependence as the primary diagnosis. Women with primary alcohol dependence that was followed by PTSD were particularly vulnerable to ongoing psychiatric distress and depression at the conclusion of treatment (Back, Jackson, Pharm, & Brady, 2005).

From the above discussion it appears that most research has demonstrated that although substance use disorders increase the risk of experiencing trauma, substance use disorders are generally secondary to PTSD onset. For the most part, researchers have concluded that PTSD is the primary disorder that temporally precedes alcohol dependence and use of other substances (Jacobsen et al., 2001). However, other researchers (Deykin & Buka, 1997) have demonstrated that the conclusion that substance use disorders are secondary to PTSD may be too simplistic, and that gender differences may apply. Substance use disorders may increase the risk of PTSD in males and not females. Given the younger legal drinking age in Australia, further

research is needed to clarify the temporal sequencing of substance use disorders and PTSD amongst young Australian's. Whilst most published data supports the self-medication hypothesis, the majority of this data is based upon the adult population and uses American samples, where the legal drinking age is generally older than in Australia (21 years versus 18 years). As Breslau and colleagues note (Reed et al., 2007), whether samples from other places produce similar associations requires further research. Given that the temporal sequencing of PTSD and substance use disorders may also impact on treatment outcomes, with a worse prognosis for those with substance use disorders as the primary diagnosis (Back et al., 2005), the pattern of temporal sequencing among Australian young people requires further investigation.

Intelligence. Although traumatic events may result in cognitive difficulties affecting academic and work performance, low intelligence may constitute a risk factor for PTSD (McNally & Shin, 1995). In a study of Vietnam combat veterans, McNally and Shin (1995) found that IQ predicted PTSD symptoms even after they controlled for effects of combat exposure and years of education. They found that the lower the IQ, the more severe were the PTSD symptoms. This finding was further supported by another study of Vietnam combat veterans (Macklin, Metzger, Litz, McNally, & Lasko, 1998) where average intelligence compared to above average intelligence predicted severity of PTSD symptoms. To add even further support to these results, in their meta-analysis of 77 PTSD studies, intellectual disadvantage was found by Brewin (2000) to be a small but reliable predictor of PTSD in adult populations. A recent prospective longitudinal study extended these results to a New Zealand community

birth cohort and found that low IQ (measured at age 5) predicted PTSD symptoms that occurred in adulthood (Koenen et al., 2007). It would seem that above average cognitive ability may enhance an individual's ability to cope with stressors, thereby protecting against the development of PTSD.

Some studies have examined IQ as a risk factor for PTSD in children and young people. In a pre-school population, children enrolled in a Head Start program because of special educational or emotional need were found to be at higher risk of psychological problems after a severe flood than those who were enrolled because of poverty (Burke, Borus, Burns, Millstin, & Beasley, 1982). A study of inner city trauma-exposed children and adolescents found that higher IQ was the best predictor of resilience against PTSD (Silva et al., 2000). Breslau and colleagues (2006) also found that having an IQ over 115 (measured at age 6 years) decreased the risk of both exposure to trauma and the development of PTSD after a traumatic experience amongst a birth cohort of 713, 17-year-old young people. They concluded that the pervasive real-life benefits of high IQ might include the successful avoidance and prevention of traumatic experiences and their PTSD effects. Therefore, paralleling the findings of studies using adult populations, cognitively disadvantaged children and young people may have more difficulty than others in coping with traumatic events and may also be at increased risk of developing PTSD.

The mechanism by which low IQ predicts PTSD is not well understood. One possible explanation is that people with a higher cognitive capacity are better able to translate the traumatic experience into a narrative and make meaning of the experience (Koenen et al., 2007). Threat appraisal has been shown to be predictive of PTSD, and

it is possible that IQ capacity affects the person's interpretation of the event and the degree of threat posed by the event (Ehlers & Clark, 2000). In addition, some researchers have found that individuals with a greater working memory capacity are better at suppressing unwanted thoughts when instructed to do so (Brewin & Beaton, 2002). This finding suggests that another explanation may be that people with a higher IQ might be able to better manage the involuntary and intrusive memories that are characteristic of the PTSD phenomenon, including flashbacks, nightmares, and intrusive recollections of the traumatic experience. These findings offer some explanation as to why low intelligence, which is strongly related to working memory capacity, is a risk factor for PTSD (Brewin & Holmes, 2003). Prospective studies that measure premorbid IQ and working memory among young Australians are required to test the temporal sequencing in this population.

Environmental Risk Factors

Stressful life events. A number of studies have demonstrated that exposure to multiple traumatic events is associated with increased likelihood and severity of PTSD in adults (Green et al., 2000), young people (Suliman et al., 2009), and in children (Copeland, Keeler, Angold, & Costello, 2007; Finkelhor, Ormrod, & Turner, 2007). Supporting a "dose of exposure" theory, an accumulation of adverse life events and exposure to violence has been found to dramatically increase the risk of psychological disorder (Tiet et al., 1998) and may hinder efforts at adjustment. With regard to stressful life events that are not life threatening and do not necessarily meet criteria for a PTSD inducing traumatic event, there is growing body of literature beginning to

demonstrate that the accumulation of multiple stressors combine to lead to more severe psychological outcomes for children and young people. Stressful life events have been linked to behavioural problems (Maclean, Perrin, Gortmaker, & Pierre, 1992) and poor physical health (Heisel, Ream, Raitz, Rappaport, & Coddington, 1973). Recently, Currier and colleagues (2009) assessed the relative contribution of stressful life events to cancer related posttraumatic stress symptoms among 121 children diagnosed with malignant cancer. These researchers found that the lifetime prevalence of stressful life events, both related and unrelated to cancer treatment, was a salient predictor of adjustment to cancer and contributed to the intensity of trauma responses (Currier, Jobe-Shields, & Phipps, 2009). Unfortunately, this study did not distinguish between stressful life events before and after diagnosis and was unable to determine the extent to which premorbid stressful life events contributed to posttraumatic stress symptoms.

Two studies have shown that the sum of stressful life events in the year preceding trauma predicted PTSD. The first (Glynn et al., 2007), examined predictors of chronic PTSD after facial injury in 336 adults and found that prior trauma exposure and the accumulation of stressful life events one-year prior, predicted chronic PTSD symptoms after injury. The second (Lawyer et al., 2006), examined predictors of PTSD within a sample of 2001 New York City residents following the September 11th terrorist attacks and primarily focussed on acute reactions post trauma. After controlling for demographic, historical and exposure related variables, the accumulation of life stressors 12 months prior to the event remained a significant predictor of PTSD. Based upon these findings, it would appear that the accumulation

of stressful life events prior to exposure to trauma, may contribute to the development of PTSD.

Abuse history. Prior exposure to trauma may increase the vulnerability of children, young people and adults at the time of subsequent stress. A number of studies in adult trauma have found that prior exposure to childhood abuse and neglect increases a person's risk for subsequent PTSD following subsequent trauma's (Astin, Ogland-Hand, Coleman, & Foy, 1996; King, King, Foy, & Gudanowski, 1996; Rowan, Foy, Rodriguez, & Ryan, 1994; Widom, 1999; Zaidi & Foy, 1994). In victims of violent crime, the experience of shame has been found to link childhood abuse with a failure to recover from adult traumas (Andrews, Brewin, Rose, & Kirk, 2000). Furthermore, a history of childhood abuse is associated with a prolonged episode of PTSD (PTSD chronicity) (Zlotnick et al., 1999). Finally, in their meta-analysis of risk factors, Brewin and colleagues (2000) found that a reported history of childhood abuse uniformly predicted PTSD across studies with adults. Once again, this is a difficult risk factor to examine in populations of children and young people and research is lacking.

Social integration and social support. The term 'social support' has been widely used to refer to the provision of perceived or received interpersonal help and is therefore hypothesised to buffer people against a stressful environment. Social integration and social support are two related theoretical constructs that refer to the degree to which people are socially connected and have a sense of belonging (Schwarzer & Knoll, 2007). Social integration generally refers to the structure and

quantity of social relationships, such as the size of the overall available social networks and the quantity and frequency of social interaction within those networks. Current social support researchers distinguish social integration from social support, which is defined as the function and quality of social relationships and includes the perceived availability of help, and the support actually received (Schwarzer & Knoll, 2007). Social support can be viewed as the physical and emotional comfort provided by friends and family. It also includes belonging to a community and feeling valued and cared for by that community. Given the large conceptual overlap of social integration and social support, the constructs are generally combined throughout this thesis.

The 1980's saw a surge in research on the role of social support in the prevention of psychological and somatic disorders in the face of stress, leading to 'the buffering hypothesis'. "The hypothesis states that the psychosocial stress will have deleterious effects on the health and wellbeing of those with little or no social support, while these effects will be lessened or eliminated for those with stronger support systems" (Cohen & McKay, 1984, pg 253). Social support (emotional help, practical help, and affirmations) and negative social interactions (highly demanding and critical) have obvious relevance for coping with experiences of trauma. Access to empathy, compassion, practical help, and the opportunity to acknowledge the trauma are clearly aspects of social support that can be expected to assist with coping and recovery. In examining the 'buffering effect' of social support on PTSD, a number of researchers have generally found support for the hypothesis. One Australian prospective study of posttraumatic stress examined the impact of the Newcastle Earthquake on a community sample (Carr et al., 1997). These authors found that a lack of social

support was one of three predictors (including initial exposure to threat and coping style) for long-term distress. Whilst results vary depending on the methodology and measures used, a meta-analysis study of 14 separate risk factors for PTSD found social support to have the strongest effect size (Brewin et al., 2000).

It has been found that a negative social environment is an even stronger predictor of PTSD than a lack of social support (Ullman & Filipas, 2001; Zoellner, Foa, & Bartholomew, 1999). In additional studies, negative appraisal of others' attempts to support the traumatised person, and negative responses rather than positive responses, were significantly associated with PTSD symptoms (Andrews, Brewin, & Rose, 2003; Dunmore, Clark, & Ehlers, 2001). Negative social responses by partners has also been found to predict poorer response to PTSD treatment (Tarrier, Sommerfield, & Pilgrim, 1999). Among inner city young people, traumatic violence, coupled with family dysfunction (presumably including a lack of support), was found to be related to PTSD symptoms (Burton, Foy, Chenga, Johnson, & Moore, 1994).

Children and young people rely heavily on the support provided by parents in particular, and children and their parents tend to respond to each other's stress. Generally, good family relationships tend to be protective for children in traumatic situations and parents can serve as role models for coping. Alternatively, parental distress can hinder recovery (Pynoos & Nader, 1989b). For more independent young people, whilst family relationships continue to play a significant role, the broader social milieu holds additional value and opportunities for support or criticism. Rosenthal and colleagues (2008) investigated the mechanisms of protection for emotional social support in the relationship between exposure to community violence

and psychological distress among 947 older adolescents aged 16 to 19 years old and residing in New York City. These researchers found that emotional support performed a preventative function in that it was negatively associated with exposure to community violence. In addition, whilst social support did not buffer the immediate emotional impact of trauma due to community violence, it did provide compensatory protection after exposure to trauma by helping to decrease emotional distress (Rosenthal & Wilson, 2008). In refugee children and adolescents, reaction to stress has also been found to be mediated by social support (Lustig, Kia-Keating, & Knight, 2004).

In contrast, however, other researchers have found differing results for the buffering effect of social support on community violence trauma's among African-American young people. In a sample of 77 low-income African-American male adolescents recruited from an inner city high school, social support from peers, family members and other adults did not buffer the negative psychological sequelae of community violence (Paxton, La Vome Robinson, & Shah, 2004).

In summary, poor social support factors after exposure to trauma have been implicated as risk factors for the development of PTSD symptoms, suggesting a stronger impact of negative over positive support in the immediate sequelae of trauma exposure in adults. There are a handful of studies to indicate that a similar situation might occur for children and young people. There is however, much more to learn about the role of social support and the development of PTSD. In particular, studies that examine the quality of social support factors prior to traumatic exposure are needed, as are studies that focus on young people.

Maternal psychopathology (anxiety, depression and comorbid anxiety and depression). A number of researchers have found a high familiarity of anxiety and depressive disorders (Livingston, Nugent, Rader, & Smith, 1985; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). A substantial body of literature has examined the impact of maternal psychological functioning on offspring functioning and diagnostic outcome, and documents the broad range of adverse outcomes, evidenced as early as infancy and continuing into adulthood (Beardslee, Versage, & Gladstone, 1998; Cummings & Davies, 1994; Hammen, 1991; Hammen & Brennan, 2001; Hammen, Burge, & Stansbury, 1990; Weissman et al., 1997). Using the same Australian sample examined in this study, principal investigators, Brennan, Hammen and associates examined the risk of depression in offspring of depressed parents and found that the presence of depression in the mother or the father increased the risk of depression in youth aged 15-years old to the same extent as dual presence of depression in both parents (Brennan, Hammen, Katz, & Le Brocque, 2002). These authors found a relationship between parental depression, family stress and functioning, and youth depression and concluded that parental psychopathology disrupts family functioning and in turn leads to deleterious outcomes for offspring. The principal investigators further examined the severity, chronicity, and timing of maternal depression on the risk for adolescent depression and anxiety in this sample (Hammen & Brennan, 2003). They found that 15-year old offspring of depressed mothers were twice as likely to also have depression, or anxiety compared with those who had mothers who had never been depressed. Approximately 11% of children of

depressed mothers had an anxiety disorder by age 15 years compared with about 5% of children of non-depressed mothers. The severity of maternal depression was a better predictor of psychopathology in the children in this sample than the chronicity of depressive symptoms.

Lieb and associates (2002) examined parental major depression among a large sample of 2427 adolescents and young adults in a 4-year longitudinal study in Germany (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002). They found that major depression in parents increased the overall risk in offspring for depression, anxiety and other psychological disorders. Offspring of depressed parents had an increased risk of anxiety disorders (OR, 1.6; 95% CI, 1.3-1.9) and the risk was higher if both parents were affected (OR, 2.1; 95% CI, 1.6-2.8). In a 20-year longitudinal study of 151 children, offspring of depressed parents were found to be at high risks for psychological and medical problems beginning in early childhood and continuing through adulthood (Weissman, Wickramaratne et al., 2006). These researchers found a three-fold increase in the risk of anxiety disorders, major depression and substance use disorders among offspring of depressed parents. In addition, offspring of depressed parents were found to have more social impairment, higher rates of medical problems and higher mortality rates compared to offspring of non-depressed parents. A number of other researchers have examined changes in psychological symptoms and general functioning in children of depressed mothers and found that remission of maternal depression has a positive effect on children's psychological symptoms (Gunlicks & Weissman, 2008; Pilowsky, Wickramaratne, Talati, & Tang, 2008; Weissman, Pilowsky et al., 2006). These researchers have found that remission of maternal

depression was associated with reductions in child diagnoses and symptoms, whereas ongoing maternal depression was associated with increased rates of children's diagnoses.

The impact of maternal depression on children also seems to be heightened when mothers have a comorbid diagnosis of depression and anxiety. Biederman and colleagues (1991) studied children and adolescents (aged 4 – 20 years) of parents with comorbid anxiety and depression. Using questionnaire measures, the prevalence of anxiety disorder was four times higher in the children of parents with depression and anxiety than in those that had neither (Biederman, Rosenbaum, Bolduc, Faraone, & Hirshfeld, 1991). More recently, Biel and colleagues (2008) examined the relationship between major depression and anxiety in parents, and anxiety in 318 children aged six to 17 years. They found that rates of childhood fears were only elevated amongst children of parents with comorbid anxiety and depression, but not with either diagnosis alone. They concluded that comorbid parental depression and anxiety may incur a cumulative risk for anxiety in children. Thus, results have consistently demonstrated that children are adversely affected by maternal depression, and that this impact may be worsened by comorbid depression and anxiety.

In the current PTSD literature, parental psychopathology is recognised as one potentially powerful factor influencing vulnerability to PTSD. Adult studies have consistently reported that pre-existing familial psychopathology increases both the risk of exposure to traumatic events and the risk of developing PTSD following exposure (for a review, see Brewin et al., 2000). More specifically, a history of depression in first-degree relatives has been identified as predictive of PTSD in adulthood (Breslau

et al., 1991; Brewin et al., 2000). A handful of studies have examined maternal depression and anxiety as a risk factor for PTSD in children and young people after exposure to trauma. McFarlane and colleagues examined PTSD symptoms among Australian children exposed to bushfires and showed that PTSD symptoms in children were more closely related to the mother's anxiety levels than the level of exposure to trauma (McFarlane, Policansky, & Irwin, 1987). Parental psychopathology and an irritable or depressed family atmosphere was also found to contribute to the prediction of PTSD in 179 children aged two to 15 years old following the Buffalo Creek dam collapse in 1972 (Green et al., 1991) and to children's psychological symptoms six months after a severe earthquake in Turkey (Kilic, Ozguven, & Sayil, 2004). Kilic and colleagues found that in their sample of 49 children aged seven to 14, the severity of PTSD in children was mainly affected by symptoms of depression and anxiety in parents (Kilic et al., 2004). General parental distress after the hospitalisation for accidental injuries of 48 children aged between seven and 17 years old was found to predict PTSD symptoms one month after trauma (Daviss et al., 2000)

Sack et al., (1995) examined the relationship of war generated PTSD and depression across two generations (parent and adolescent) of Cambodian refugees and found that parental psychopathology often predated adolescent PTSD with 62.5% of parents reporting onset of symptoms prior to the offspring's symptoms (Sack, Clarke, & Seeley, 1995). As discussed, recent research has focussed on the impact of parental distress and psychopathology after trauma exposure as a predictor of PTSD in the child. The question of whether preexisting maternal depression and anxiety predicts PTSD in young people has not been clearly answered, but rather implied.

The mechanism by which family psychiatric history increases the risk of PTSD is not yet clear. Goodman and Gotlib (1999) propose four mechanisms of transmission of risk, the first being that having a depressed mother confers on the child a genetic predisposition to a diverse range of psychopathology. Twin and adoption studies show that genetic factors partially account for psychological problems of adult offspring with depressed parents. The incidence of anxiety and depressive disorders in monozygotic and dizygotic twins revealed that approximately 30%–40% of the variance in occurrence between individuals can be attributed to genetic variation (Hettema, Neale, & Kendler, 2001; Sullivan, Neale, & Kendler, 2000). Other twin studies have found that the genetic predisposition for PTSD among adults confers a risk that accounts for 30% to 35% of the liability to develop PTSD following exposure to traumatic events (True et al., 1993) and also a genetic predisposition for exposure to violent crimes such as assault (20%) and combat-related trauma (35%) (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Exposure to other classes of trauma (vehicle accidents and natural disasters) was influenced by environment alone (Stein et al., 2002). These researchers have examined the genetics of anxiety and depression and have concluded that behaviours related to anxiety and mood disorders are probably related to complex and subtle alterations to multiple genes that each contribute partially to the expression of behaviour. Genetics alone does not sufficiently explain psychological and behavioural outcomes in offspring (Leonardo & Hen, 2006). Gene researchers acknowledge that the environment makes a significant contribution to the development of psychological disorder. The empirical support for heritability of

psychological disorder is less clear for child or adolescent onset than it is for adult onset.

The second mechanism of transmission proposed by Goodman and Gotlib (1999) is that offspring of depressed mothers are born with dysfunctional neuroregulatory mechanisms that interfere with emotional regulation processes and consequently increase vulnerability to psychological disorder. Alder and colleagues (2007) carried out a search on twenty years of research linking prenatal maternal psychological problems and associations with adverse obstetric, foetal and neonatal outcome. Across a total of 35 studies, maternal elevated levels of depression and anxiety were found to be associated with both obstetric outcomes (complications, pregnancy symptoms, pre-term labour and pain relief required during labour) and foetal and neonatal wellbeing and behaviour independently of other medical risk factors. Studies examining this mechanism have generally focussed on both the foetal environment (e.g. neuro-endocrine abnormalities, reduced blood flow to the foetus, poor health behaviours, and the use of antidepressant medications) and how the foetal environment can lead to abnormal development, and also on the behavioural evidence of dysfunctional neuro-regulatory systems among babies and toddlers (Goodman & Gotlib, 1999). Handley and colleagues (1990) found higher levels of cortisol, beta-endorphin and corticotrophin releasing hormones among pregnant depressed women compared with non-depressed pregnant women (Handley, Dunn, Waldron, & Baker, 1980) and cortisol hormones are believed to cross the placenta accounting for 50% of the variance in the foetus' levels of cortisol (Glover, 1997). These higher than normal levels of stress hormones in infants would be expected to be associated with

heightened stress reactivity and abnormal behavioural and affective functioning (Goodman & Gotlib, 1999).

Thirdly, depressed mothers expose their children to negative or maladaptive cognitions, behaviours, and affect, which place their children at elevated risk for developing psychological disorder (Goodman & Gotlib, 1999). Depressed mothers, in their interactions with their child, have been shown to model poor emotional strategies and have been found to display more sad and irritable affect (Cohn, Campbell, Matias, & Hopkins, 1990) and to be more punitive, negative, angry and hostile (Goodman, Adamson, Riniti, & Cole, 1994; Hammen, 1991). It is believed that through social learning, or modelling, this style of parenting would negatively affect the child's development of social and cognitive skills and place them at greater risk for anxiety and depression. Children of depressed mothers have been found to have deficits in affective, interpersonal, cognitive and behavioural skills. For example, children of depressed mothers have been found to exhibit high levels of anxiety during a mildly stressful situation (Radke-Yarrow & Sherman, 1985). Furthermore, through modelling of poor coping and social learning theory, overt maternal trauma-related fears, avoidance and anxious responses may increase the post-trauma distress of offspring because children acquire the cognitions, behaviours, and affect that resembles that displayed by their parents (Pynoos et al., 1999). Finally, the context of the lives of children in families with depressed mothers, particularly the stressors, contributes significantly to the development of psychopathology in the children. Hammen (1991) demonstrated that negative life events experienced by depressed adults, may be a consequence of their depression, rather than a cause. Furthermore, research has shown

that children of depressed parents experienced more negative life events than children of well parents (Adrian & Hammen, 1993; Hirsch, Moos, & Reischl, 1985). Therefore, offspring of depressed mothers are exposed to a variety of stressors that are associated with maternal depression.

Parental trauma. There is a growing body of evidence that a history of prior trauma and adversity increases the risk of PTSD following later trauma (Brewin et al., 2000) and that the risk is higher after earlier experiences of non-combat interpersonal violence (Ozer et al., 2003). However, it is unclear whether this relationship is also intergenerational. Does early parental exposure to trauma increase the risk of later exposure to trauma and development of PTSD among offspring? There is some evidence to suggest that the risk of exposure to trauma and the subsequent development of PTSD may indeed be intergenerational. Initially, research examining transgenerational trauma involved identifying past traumatic events that continued to impact on following generations (Frazier, West-Olatunji, Juste, & Goodman, 2009) and has been used to explain the immense emotional sequelae of the widespread and collective traumatisation experienced by cultural groups such as Native Americans and African Americans (Dass-Brailsford, 2007), and Australian Indigenous populations (Atkinson, 2002; Ober, Peeters, Archer, & Kelly, 2000). Dass-Brailsford (2007) defines transgenerational trauma as a form of trauma that has been passed down, either directly or indirectly, from one generation to another (Dass-Brailsford, 2007). Some PTSD researchers argue that this type of trauma can occur in the absence of direct exposure to a traumatic event and can be transmitted from a parent who has

experienced a traumatic event (Goodman & West-Olatunji, 2008) and that trauma creates patterns that are repeated from one generation to the next (Frazier et al., 2009).

Due to the convenience of sampling war veterans this group appears to be the most frequently examined across the PTSD literature and as a result the most compelling evidence for a genetic component to PTSD vulnerability is the Vietnam Era Twin Registry study (Goldberg, True, Eisen, & Henderson, 1990; True et al., 1993). This study examined 4,042 male only veteran twin pairs (2,224 monozygotic and 1,818 dizygotic twins) and found genetic influences on extent of trauma exposure (Lyons et al., 1993). These studies highlighted that genetic factors contribute to risk for trauma exposure, perhaps related to similarities in pre-trauma personality characteristics, however, the sample was solely siblings and men exposed to combat. The National Vietnam Veterans Study has since examined the impact of paternal PTSD on offspring of war, and interesting research now shows clear and consistent evidence for an intergenerational transmission of trauma in the children of war veterans (Rosenheck & Fontana, 1998). This study examined 257 fathers who participated in abusive violence in Vietnam and the subsequent behavioural problems in their children aged 6 to 16 years old, some 15 to 20 years after the father's participation in combat. This study found a significant and direct relationship between the father's participation in violent abuse during combat and behavioural problems in children, even after controlling for PTSD, psychological disorders and other veteran problems. This study clearly demonstrated that a paternal history of trauma exposure in the form of combat abusive violence affected parent-child relationships in a way that was deleterious for the offspring, independent of the role of parental PTSD and

other psychological disorder (Rosenheck & Fontana, 1998). In addition the potential for negative intergeneration effects of trauma exposure were also evident, but the mechanism for this intergenerational impact is not clear. One possibility posed by the authors, is that veterans exposed to abusive violence had difficulty in forming an emotional connection with their children or in assertive discipline and boundary setting. As a result, they experienced difficulty in being empathic, firm and consistent (Rosenheck & Fontana, 1998). Although this study did not examine PTSD in offspring, the authors concluded their article with a statement highlighting the importance of exploring parental trauma history when treating child and adult patients.

In an Australian sample of Vietnam Veterans, offspring aged 15 to 30 years rated their families as clinically dysfunctional especially in the areas of responding with appropriate affect (emotional responsiveness) and problem solving when compared with civilians (Davidson & Mellor, 2003). Whilst, offspring of veterans with PTSD were not more likely to themselves develop PTSD, this Australian finding was consistent with other research (Westerink & Giarratano, 1999) demonstrating that disrupted family functioning is a consistent outcome for trauma survivors, especially those with PTSD.

As previously discussed, civilian studies have demonstrated a genetic predisposition for exposure to violent traumas (Stein et al., 2002). In the first study to examine the heritability of trauma exposure and PTSD symptoms outside of the context of military trauma, and the first study to include women, researchers have examined the heritability of trauma exposure in 222 monozygotic and 184 dizygotic civilian male and female twin pairs. Replicating the findings of the Vietnam Era Twin Registry

study, this study found that genetic factors and environmental (shared and non-shared) factors influenced the risk of exposure to interpersonal traumas with assault as the common element. Whilst this was the first study to focus on the genetic vulnerability to trauma exposure among civilians, it is already well established that genetic factors and shared environment are both associated with particular kinds of environmental exposures, including stressful life events and trauma (Foley, Neale, & Kendler, 1996; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Middeldorp, Cath, Vink, & Boomsma, 2005). Middeldorp and colleagues (2005) examined stressful life events including divorce, illness, and death of a significant other, as well as potentially traumatic events including sexual assault, and violent assault among over 3000 monozygotic and dizygotic twins and their siblings. These researchers found strong evidence for a familial aggregation of traumatic events. Both genetic and common environmental effects contributed to exposure to events (Middeldorp et al., 2005).

It is currently unclear whether these findings extend to children of mothers who have experienced trauma, although a number of transgenerational theorists are beginning to argue that mother's history of sexual harm significantly impacts on trauma symptoms among offspring and that a child's sexual assault symptoms often coexist with behaviours associated with a caregiver's own untreated or unresolved child sexual assault symptoms (Frazier et al., 2009). In relation to the intergenerational effects of maternal trauma, other studies supporting the possible transmission of risk from mothers include those that have found an increased risk of sexual abuse between mothers and their daughters. One factor consistently found to be associated with the aetiology of trauma in the form of child abuse, is the mothers' own experiences of

childhood abuse which is estimated to account for one third of the variance in predicting child maltreatment (Haapasalo & Aaltonen, 1999). McCloskey and Bailey (2000) examined 176 girls within families over-sampled for violence, and found that girls whose mothers were sexually abused, were 3.6 times more likely to be sexually victimized themselves (McCloskey & Bailey, 2000). Not only is maternal abuse history a marker for exposure to trauma among offspring, but it also impacts on parenting behaviours (Bert, Guner, & Lanzi, 2009), which may in turn contribute to the intergenerational transmission of abuse either directly through poor maternal parenting, or indirectly by contributing to emotional vulnerabilities in offspring. Recent research by Bert and colleagues (2009) found that maternal exposure to childhood physical and emotional abuse was associated with poorer maternal responsiveness and an increased propensity for abusive behaviour among 681 mothers between the ages of 14 and 36 years.

In addition, children of Holocaust survivors have also showed significantly higher levels of self-reported childhood trauma, particularly emotional abuse and neglect (Yehuda, Halligan, & Grossman, 2001) and the difference was largely attributable to parental symptoms related to their own history of trauma. Lichtman (1984) examined outcomes for survivors of the Holocaust and specifically researched parental communication of their experiences of trauma and the psychological wellbeing of their adult children. Among other psychological factors, the degree of anxiety among the second generation was assessed. This study found that maternal, and not paternal, willingness and frequency of communication about her experiences in the Holocaust was significantly associated with the experience of anxiety in

offspring. Conversely, fathers' willingness and frequency of communication about the Holocaust was associated with positive outcomes in offspring. A significant difference was noted in the communication style of the fathers, and it was suggested that mothers transmit to the family a sense of victimisation in their communication, whereas fathers communicated the identity of being a 'fighter'. It was hypothesised that from the mother's communication style, children perceived their mothers, and also themselves as victims, whereas when the father presented them as 'fighters', they were less likely to feel depressed and anxious. Parental communication that was guilt inducing, and indirect was also associated with poorer outcomes for children. In addition, a significant gender difference was found among the second generation. Females were more likely than males to internalise their feelings about their parent's experiences and experienced more symptoms of fear, withdrawal, somatic complaints and lowered self-esteem. Davidson et al. (1980) has also found that excessive talking about Holocaust experiences, or the opposite, a lack of communication characterised by avoidance and denial (taboo) has been associated with severe psychological effects and hospitalisation of children of Holocaust survivors (Davidson 1980). Whilst these studies do not examine PTSD specifically, they do suggest that parental communication style, and particularly maternal communication about previous trauma experiences may negatively impact on offspring and contribute to anxiety and psychological disorder.

Therefore, offspring of traumatised adults may themselves be traumatised by stories passed on from one generation to the next, and may be vulnerable to the deleterious effects of direct exposure to trauma by the lessons learned from the

experiences of previous generations. For instance, a sense of hyper-arousal and a lack of safety may originate from parental communication and modelling of the view of the world as a dangerous place. Given that parental psychopathology and, more specifically, parental depression and anxiety have been identified as possible risk factors for the development of PTSD in children, it is also reasonable to consider the role of maternal abuse histories in this relationship. Based upon the above findings, it is reasonable to contend that a maternal history of trauma may increase the risk of PTSD in offspring as a result of an individual's genetically determined personality characteristics or behaviour influencing their environmental choices, familial aggregation of events due to common environmental effects, or the influence of parental factors associated with modelling and conditioning leading to emotional vulnerabilities in offspring. Both parental trauma and parental psychological disorder have been found to increase the risk for the development of PTSD in offspring. However, exposure to trauma, especially interpersonal abuse, and depression are also known to be highly associated. To date, studies that have found a relationship between PTSD and maternal trauma history, have generally not taken into account maternal depression. Similarly, studies that have found that familial depression is a risk for PTSD have not taken into account parental abuse histories. Little is written about the intergenerational influences of community based trauma exposure on offspring, particularly in relation to the development of PTSD among mother's exposed to trauma. It is therefore essential to examine the PTSD, parental trauma relationship taking into account depression and anxiety in the mother, which may account for the association.

Summary

In summary, although it would appear that psychological disorders are affecting one in five Australian children before adulthood (Sawyer, Arney et al., 2000) and that the overall incidence of psychosocial problems are increasing in modern times (Rutter & Smith, 1995), the actual prevalence, chronicity and psychological comorbidity of PTSD amongst Australian children and young people is currently unknown. This is particularly surprising in light of recent epidemiological studies that have found that PTSD was more common than depression amongst Australian adults (ABS, 2007). In addition, we know that adolescence is a critical period for the development of psychological disorders (Public Health Group, 2005), and onset of most psychological disorders occurs before age 24 years (Kessler et al., 2005). Perhaps partially accounting for the deficit in research on PTSD in children is the fact that although traumatic symptoms have long been recognised, the inclusion of PTSD as a psychological diagnosis is a relatively new phenomena in the field of psychology. As is often the case, research into PTSD in children and young people has lagged behind the adult research. Whilst there has been a recent surge in research, many of the studies to date are limited by a number of significant methodological problems that are understandable to some extent given the difficulty in predicting exposure to trauma. Therefore, there are very few studies documenting the prevalence, course and comorbidity of PTSD in young Australian samples, and even fewer genuine prospective studies examining risk factors for PTSD prior to onset of disorder. Most PTSD research examines adult populations using American samples.

Whilst the estimated prevalence of exposure to traumatic events is high (Kessler et al., 1995; Ozer & Weinstein, 2004), most children, young people and adults are resilient to trauma exposure and do not develop clinically apparent symptoms (Le Brocque, Hendrikz, & Kenardy, 2009). Even so, it is wrong to presume that children are more adaptable to trauma than adults (Morgan & Scourfield, 2003) and the oversight of PTSD research and treatment amongst our valuable young Australians is unacceptable, and must be remedied. For those who do develop PTSD, it would seem that at least one-third experience long-term, chronic and impairing PTSD symptomatology (Kessler et al., 1995; Le Brocque, Hendrikz, & Kenardy, 2009; Yule et al., 2000). The severe and pervasive developmental consequences for younger populations are a major concern, but this finding is yet to be replicated using young Australian samples. The impact upon learning, school performance, social development, relationships, personality formation, identity, health, emotional regulation, impulse control, and overall quality of life can have far-reaching and at times lifelong consequences for young people (Pynoos et al., 1999). Therefore, studies that help plot the development and course of PTSD, and determine risk factors for PTSD onset, play an important role in determining those most at risk for this debilitating disorder and can help guide important preventative and early treatment interventions. Whilst a number of premorbid factors are known to increase the risk of PTSD amongst adults; such as female gender, premorbid psychological disorder, lower intelligence, early adverse life events, a negative social environment, familial psychopathology, and a family history of trauma; it is unclear whether these results can be applied to a young Australian population.

CHAPTER 2

Study One: Epidemiology Study

The recent National Survey of Mental Health and Wellbeing (NSMHW2) (ABS, 2007) examined 12-month prevalence rates of anxiety, affective and substance use disorders experienced by Australians aged 16 to 85 years, and found that Post Traumatic Stress Disorder (PTSD) was the most common anxiety disorder across this entire (primarily adult) sample. Surprisingly, PTSD was more common than depression (6.4% compared with 4.1% prevalence). Whilst being well overdue, the epidemiological data from the NSMHW2 focussed largely on adults ($n = 8841$), and whilst there were 995 young people between the ages of sixteen and twenty-one years included in the sample, the study did not report the lifetime prevalence of PTSD amongst children and young people specifically. As discussed in detail in chapter one, the prevalence, course, chronicity and psychological comorbidity of PTSD amongst non-clinical populations of younger Australian children and young people currently remains unclear. The prevalence of PTSD amongst American children and young people is estimated at 3.5% (Cuffe et al., 1998) to 6.3% (Giaconia et al., 1995), but it is unknown whether these rates can be generalised to an Australian population. Most published research has examined either American or clinical samples, or has examined groups of children exposed to a shared trauma such as bush fires (McFarlane et al., 1987), motor vehicle accidents (Aaron et al., 1999), accidental injury (Le Brocque, Hendrikz, & Kenardy, 2009), warfare (Saigh, 1991), sexual abuse (Kendall-Tackett et al., 1993), child maltreatment (Famularo et al., 1996), and sniper attacks (Pynoos et al.,

1987). Whilst it is promising that Australian epidemiological studies such as the NSMHW2 have begun to include PTSD diagnoses in their studies, there remains a considerable gap in the literature examining the topography of PTSD in young Australians.

In relation to comorbidity, American studies once again dominate the research and have shown high comorbidity with both internalising disorders such as mood disorders and anxiety disorders, and externalising disorders such as behavioural disorders, and alcohol and substance use disorders amongst child and adolescent populations. However, the extent to which childhood features of PTSD are similar, or different, across American and Australian cultures is unclear. Further to this, there is a growing body of evidence demonstrating a high suicide risk associated with PTSD amongst adults (Davidson et al., 1991; Kotler et al., 2001; Tarrier & Gregg, 2004) with about 18-20% of adults with PTSD attempting suicide. Whilst in Australia there are alarmingly high rates of suicidality amongst young people (Sawyer, Arney et al., 2000), to the best of this author's knowledge, there is no Australian research examining suicidality and PTSD amongst young Australian samples.

The frequency, chronicity and impairment of PTSD is of particular importance for children and young people as the disorder may significantly disrupt important developmental milestones and contribute to the loss of many years of healthy life and normal development, and can prevent a young person from reaching his or her full potential throughout childhood and into adulthood. An understanding of PTSD in childhood and adolescence is important for prevention, detection, and treatment to not only relieve the experience of distress, but to improve functioning in later adulthood

and prevent the intergenerational deleterious effects of psychological dysfunction. Research examining the frequency, course, chronicity, and psychological comorbidity of PTSD in Australian children and young people has been neglected and is well overdue if we are to take this disorder seriously and understand, treat, and prevent the morbidity of this disorder in our valuable young Australians.

The aims of this study are primarily exploratory. This study aims to help fill the void in the current PTSD literature by exploring the frequency, age at onset, duration, precipitating events and comorbidity of PTSD in an Australian community sample of children and young people to help shed more light on the presentation of this disorder among this younger population. A number of hypotheses can be inferred from previous adult and child PTSD literature. Firstly, the prevalence of PTSD amongst children and young people is unlikely to be rare and is hypothesised to be consistent with the findings from the NSMHW2 (ABS, 2007) which found a 12-month prevalence rate of 6.4%. Secondly, it is hypothesised that the likelihood of developing PTSD will increase with age. Thirdly, consistent with adult findings, it is hypothesised that PTSD will be often chronic (exceeding 6 months in duration) and highly comorbid with depression, anxiety, behavioural disorders, alcohol and substance use disorders, and suicidality.

The Mater University Study of Pregnancy (MUSP) 15-year and 20-year follow-up data set was used to explore PTSD symptoms from birth, throughout childhood, and to 20 years of age amongst young Australians. The MUSP is an ongoing study of the health of mothers and their children that began during 1981 and 1984 when over 8000 pregnant women were invited to join the study during their first clinic visit at the

Mater Mothers Hospital in Brisbane, Australia. Further details about the project have been reported in previous publications (Keeping et al., 1989). The subject pool used in the current study was selected from the larger cohort with the intention of including children of mothers with varying severity and duration of depressive symptoms, in addition to children of mothers with no depression. The current study is over-sampled for children of mothers with elevated and/or persisting levels of depressive symptoms either in the prenatal period or within the first five years of the child's life. As discussed in chapter one, a family history of depression is associated with PTSD development. This study provides the unique opportunity to explore the differences in PTSD amongst offspring of mothers with and without depressive symptoms during the early years of child development. This study would therefore expect to find a higher prevalence of PTSD symptoms amongst offspring of depressed mothers compared to offspring of mothers without depression.

In summary, the following hypotheses were made:

1. The prevalence of PTSD amongst children and young people is unlikely to be rare and is hypothesised to be consistent with the findings from the NSMHW2 (ABS, 2007) which found a 12-month prevalence rate of 6.4%.
2. The likelihood of developing PTSD will increase with age.
3. PTSD will be often chronic (exceeding 6 months in duration) and highly comorbid with depression, anxiety, behavioural disorders, alcohol and substance use disorders, and suicidality.
4. There will be a higher prevalence of PTSD symptoms amongst offspring of depressed mothers compared with offspring of mothers without depression.

Methodology

Background to Methodology

The Mater-University Study of Pregnancy (MUSP) is a collaborative project between the Mater Misericordiae Hospital, Brisbane, Australia and the Departments of Social and Preventive Medicine and Anthropology and Sociology at The University of Queensland, Australia. The 15-year and 20 year follow-ups were a collaborative project with Professor P. Brennan from Emory University, Atlanta, Georgia and Professor C. Hammen from University of California, Los Angeles, U.S.A.

The original aim of the MUSP was to investigate the child's physical, cognitive, and psychological health as a function of pregnancy and obstetric conditions, birth weight, and psychosocial conditions and to predict health, development, and behaviour at age five years. Follow-up of a selected sub-sample of children aged 15 years was undertaken to assess the impact of the severity and timing of maternal depression on child outcome. The data have been used in this study to explore PTSD in the children and young people, and the premorbid risk factors (including maternal depression) for the development of PTSD after age 15 years.

Presented below is information about the research participants, data collection from the 15-year and 20-year follow-ups, ethical considerations, research measures used to assess the variables, and the analytic strategy.

Participants

Pregnant mothers were initially enrolled in the MUSP study when they presented to the Mater Hospital at their first antenatal clinic visit. Fieldwork for the MUSP study commenced for the first phase in January 1981. At this time, the Mater Hospital was only one of two major teaching hospitals in greater Brisbane and serviced the southern part of the city. The mean gestation at entry to the study was about 18 weeks. A total of 8,556 consecutive women attending their first obstetrical visit at the Mater Hospital between 1981 and 1984 were invited to participate in the study. Prior to being discharged from the hospital, a post-birth phase of data collection was undertaken. For those mothers who agreed to participate by completing both phases, 7,223 live singleton children were discharged. This is referred to as the birth cohort. Some 98 mothers refused the initial phase, 710 did not deliver a live child at the public hospital (including 169 miscarriages and those who chose to use other facilities), 59 mothers had multiple births and were excluded from this part of the study, 312 did not complete the post-birth phase, 99 children died during or post delivery and 55 children were adopted prior to discharge and were excluded from the study.

The mothers in the MUSP study completed the Bedford-Foulds Delusions-Symptoms-States Inventory (DSSI) (Bedford & Foulds, 1977) to assess depressive symptoms and provided information about their infants at four assessment periods; at their first antenatal clinic visit, when their child was born (3-4 days after delivery), when their child was six months old, and when their child was five years old. Sample selection at the 15-year follow-up was based on mothers' depression scores on the DSSI completed on those four occasions between pregnancy and child's age five

years. Principal Investigators aimed to obtain a sample of women with the greatest differences in maternal depression to assess the impact of the severity and timing of maternal depression on child outcomes. Using the DSSI results and sampling algorithms, women with varied frequency and severity of depression histories were selected, along with a non-depressed sample. Specifically, women were targeted who reported severe depression at two or more times (12%); severe depression only once (35%); moderate depression two or more times but never severe (23%); and low or no depression at all assessment periods (30%). Thus, from a total of 5,277 13-year-old adolescents retained from the original study (68%), 991 families were targeted for inclusion in the age 15 cohort. A final sample of 816 families was located and consented to participate (82% of the 991 selected for follow-up). Of those not included, 68 families could not be located, 103 declined participation in this round of research, three included a child with a visual or hearing impairment that precluded participation, and one child had died. Children in this sample were not significantly different from the original birth cohort in terms of gender, income or mother's education measured in years.

The 15-year sample consisted of 414 boys and 402 girls, mean age 15 years, 2 months (range: 14-16 years, 95.4% were within two months of age 15, $SD = 0.29$). The overall sample included 92% Caucasians; median family income was in the level of Australian middle and working-class socio-economic status, median mothers education was Grade 10, and the mothers median age at follow-up was 41 years.

Fieldwork for the 20-year follow-up commenced in February of 2001. The 15-year sample was contacted at approximately age 20 years and invited to participate in

the Mater- University Study of Pregnancy 20-year follow-up. Of the original 816 youth, 706 (87%) were interviewed in the age 20 follow-up (2 were deceased, 51 refused, and 52 could not be located or scheduled for appointments; 5 participants did not complete diagnostic interviews). Those who were included were marginally more likely to have mothers who were depressed [$\chi^2(1) = 3.56, p = 0.059$]. Males were less likely to participate in the follow-up than females [$\chi^2(1) = 12.63, p = .006$].

Participants did not differ from non-participants on their own prior depression status, [$\chi^2(1) = 1.32, p = .25$], or on any prior diagnosis. Nonparticipants also came from families with lower income, $t(783) = 2.11, p < .05$. The current study consisted of the 706 young people (343 males and 363 females) and their mothers who completed both parts of the study and provided outcome data at both the 15-year and 20-year follow up. Participants in the age 20 study did not differ from the original cohort in family income, maternal education, and gender of the target child. The overall sample was 91.2% Caucasian, 8.8% minority (Asian, Pacific Islander, and Aboriginal). Median family income was middle and lower middle classes.

Procedure

Interviews for the 15-year and 20-year follow-up were conducted with the young person and mother within the family home. Masters-level and PhD-level psychologists were employed to undertake the fieldwork in the 15-year and 20-year follow-ups and a team of two interviewers conducted confidential interviews separately and privately, and were blind to diagnostic status or history. The young person and mothers were administered a structured diagnostic interview and were

asked about their family history and the young person's psychological and social functioning. Participants also completed a battery of questionnaires. The young person and mother provided written and informed consent and were paid for their participation, which lasted approximately 3.5 hrs for each interview.

Interviewers were clinically trained post-graduate psychology students from two leading Brisbane universities, The University of Queensland and Griffith University. The team of interviewers was trained to proficiency by principal investigators to conduct diagnostic and life-stress interviews and was closely supervised by means of audiotape and periodic visits by the investigators. The author of this thesis was one interviewer amongst the team and personally completed 198 mother or young person interviews during the three year period of the 20-year follow-up. Samples of interviews were systematically reviewed at six-month intervals over the three-year course of data collection to prevent drift.

Ethical Considerations

The MUSP longitudinal study was conducted under the NHMRC guidelines. Ethical approval was obtained for the 15-year and 20-year follow-ups from Emory University Human Investigations Committee, University of California Institutional Review Board, The Mater Children's Hospital Research Ethics Committee, and The Behavioural and Social Sciences Ethical Review Committee of The University of Queensland. Data for this research came from the MUSP longitudinal study and no additional collection of data or contact with families was required. The Human Experimentation Ethical Review Committee has approved that data previously

collected or in records does not require review, provided appropriate gatekeeper approval has been obtained and personal information purged from the data set. The author of this current study was one of the project interviewers for the duration of the 20-year data collection period (three years) and received approval from the Principal Investigators to access the project data for the purposes of completing this dissertation (refer to Appendix A). Thus, given that prior ethical approval has been sought, no new data was collected, and approval from the principal investigators had been obtained, this study was deemed outside the scope of the University's animal and human ethics research arrangements, and as such did not require University ethical review as confirmed by completion of the Griffith University Research Ethics Scope Checker (refer to Appendix B).

Informed Consent

Informed consent forms were provided and verbally explained by interviewers and signed by participating parents and young people prior to commencement of interviews. Consent forms contained a confidentiality statement in relation to risk of harm to self or others, and harm to children (refer to Appendix C). The projects psychiatric consultant was Dr William Bor at the Mater Misericordiae Hospital, South Brisbane. All interviewers were provided with Dr Bor's direct mobile number to be used in the event of serious concern or disclosure of significant risk of harm. Information was provided about local community agencies, reputable psychologists and psychiatrists as appropriate. When suitable, referrals were made to these services.

Measures

Psychological disorders. The presence of current or lifetime PTSD, depression, anxiety, substance use, and behavioural disorders in the young person at age 15 years was determined using the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Epidemiologic version revised for DSM-IV (K-SADS-E) (Orvaschel, 1995), administered separately to the parent and young person and blind to mothers' depression history. The K-SADS-E (Chambers, Puig-Antich, & Hirsch, 1985) is a semi-structured child and adolescent psychiatric interview designed for use with children aged 9 years and over (Ambrosini, 2000; Rapoport & Ismond, 1996). Administration of the K-SADS-E interview followed the prescribed format. The interview was first administered to the parent alone, and the same clinician then administered the interview to the child alone. The diagnostic status of the young person at the 15-year follow-up, as well as lifetime incidence of disorders (including date of onset and recovery), was determined by combining information from the K-SADS interviews with the young person and mother.

The Structured Clinical Interview for DSM-IV (SCID-I) (First, Spitzer, Gibbon, & Williams, 1995) was administered at age 20 years to ascertain current and past diagnoses of PTSD, depression and other DSM-IV clinical diagnoses. The presence of current or past diagnoses were ascertained blind to the young person's previous diagnoses at the 15-year follow-up. The instrument is a semi-structured interview administered by trained clinical interviewers and covers current, past and lifetime disorders (including date of onset and recovery). The interviewer formed diagnostic decisions in consultation with the clinical rating team. The current analyses

merged diagnoses from the 15-year follow-up with those from the 20-year follow-up to ascertain all lifetime diagnoses prior to age 20 years. As accurately as possible, date of onset of disorder was recorded for all diagnoses.

A blind review team, based in the U.S.A., analysed audiotapes of diagnostic interviews and determined reliability using weighted kappa (based on no symptoms, symptoms, diagnosis) for diagnostic categories (including depressive disorders, anxiety disorders, substance use and behavioural disorders) for both current and past disorders. There was high agreement between the teams ranging from 0.67 to 1.0 for diagnoses. The kappa for anxiety diagnoses were 0.94 for current diagnoses and 0.89 for past diagnoses.

Previous researchers in the field of PTSD have highlighted that partial PTSD symptoms are common and may be significantly impairing even when full criteria is not met (Giaconia et al., 1995; Goenjian et al., 1995). Furthermore, it is common for studies investigating PTSD to include subclinical and clinical PTSD symptoms. Given the difficulty in diagnosing PTSD in young people, the high prevalence of subclinical features (partial symptoms that do not reach full DSM-IV diagnostic criteria), and the impairing nature of subclinical features, subclinical PTSD in addition to clinical PTSD was relevant to the current study. For the purposes of the current study, three categories of diagnostic outcome for young people were used: Clinical PTSD, Subclinical PTSD and No PTSD. Young people with a lifetime diagnosis of Clinical PTSD met the strict standards of full DSM-IV diagnostic criteria for the PTSD diagnosis, including significant impairment. The subclinical PTSD group included those who met two or more of the PTSD diagnostic criteria, but who fell short of

meeting the strict full DSM-IV diagnostic criteria for PTSD. Some participants reported meeting three criteria but at the end of PTSD specific questioning denied significant impairment in daily functioning. In summary, the subclinical PTSD group comprised of young people who met two diagnostic criteria, and those meeting three criteria but without evidence of impairment. Young people without any lifetime PTSD diagnoses and no subclinical PTSD symptoms (as defined above), but in many cases alternative diagnoses, fell into the No PTSD category.

Suicidal ideation. Young person wellbeing and social functioning was measured at age 15-years and at age 20-years using a semi-structured interview (Hammen, 1991) developed from earlier versions of interviews for chronic strain and functioning for adults and children. At the 15-year and 20-year interviews the young person was asked whether they had previously considered or attempted suicide (refer to Appendix D). Those who had attempted suicide were asked for additional detail about method, intention and number of attempts. Those who had considered suicide were asked for additional information to determine suicidal ideation compared with thoughts of self harm that were not necessarily suicidal by nature. Those with thoughts of death, dying and/or a desire to end one's life were identified as having suicidal ideation. The variable used in the current study was categorical and included three categories: no suicidal ideation or attempt, suicidal ideation without attempt, and suicide attempt (including multiple attempts).

Analytic Strategy

A series of ANOVAS and chi-squares were used to analyse the data. All statistical tests were two-tailed and α levels set at .05 for main effects. For some analyses, Clinical PTSD and Subclinical PTSD were collapsed into one PTSD group and diagnostic categories for each analysis are therefore reported throughout the results. Where small cell sizes negated the use of chi-square analyses T-tests were conducted. All tables are presented with sample sizes.

Summary

In summary, data from this study come from the MUSP longitudinal data and the age 15-year and age 20-year follow-ups. The total sample size comprised of the 706 young people ($n=362$ females, 344 males) who provided data at both the 15-year and 20-year follow-ups. This study used the clinical and subclinical PTSD diagnoses, in addition to major depression, anxiety, substance use and behavioural diagnoses to explore the frequency, duration, triggering events, and psychological comorbidity of PTSD in children and young people. The next section presents initial descriptive analyses.

Results

Sample Characteristics

Young person sample characteristics are presented in Table 1 including gender, ethnicity, mother's highest educational attainment, and family income at the age 15-year follow-up. Table 1 illustrates these demographics for the sample as a whole and those with PTSD, subclinical PTSD, and no PTSD. A total of 45 (6.4%) young people received a lifetime diagnosis of clinical PTSD, and 30 (4.2%) had subclinical PTSD. Combined, 10.6% of the young people were diagnosed with symptoms of PTSD (subclinical or clinical). A total of 631 of the 706 young people had no PTSD diagnosis. Given the over-sampling of mother's depression as measured by the DSSI at four time periods prior to child's age 5 (see participants above), all sample characteristics are reported with mothers' depression.

Gender, Ethnicity, Education and Income

From Table 1, it is evident that PTSD was more common among females than males. Eighty-two percent of young people with a lifetime diagnosis of PTSD, and 67% with a lifetime diagnosis of subclinical PTSD were female. The sample was largely Caucasian, with 8.8% Indigenous Australian. Eleven percent of young people with PTSD were Indigenous, and 3% with subclinical PTSD were Indigenous. Education was based upon mother's educational level at the age 15 follow-up and all levels were represented: 16.5% completed Junior School, 63.2% completed Secondary School (Yr 10 or Year 12) and 20.2% had completed Tertiary (including TAFE). Over

Table 1

Young person characteristics (n=706)

Characteristics	Cohort (n = 706)	PTSD (n = 45)	Subclinical PTSD (n= 30)	No PTSD (n= 631)
Male	48.7% (344)	17.8% (8)	33.3% (10)	51.7% (326)
Female	51.3% (362)	82.2% (37)	66.7% (20)	48.3% (305)
Mean age at 15-year follow-up	15.2 ± 0.3	15.1 ± 0.2	15.2 ± 0.4	15.2 ± 0.3
Caucasian	91.2% (644)	88.9% (40)	96.7% (29)	91.1% (575)
Indigenous	8.8% (62)	11.1% (5)	3.3% (1)	8.9% (56)
Mothers education				
Junior school	16.5% (116)	15.6% (7)	13.8% (4)	16.7% (105)
Secondary school	63.2% (444)	73.3% (33)	69% (20)	62.3% (391)
Tertiary	20.2% (142)	11.1% (5)	17.2% (5)	21% (132)
Family income				
Low	33.6% (224)	41.9% (18)	42.9% (12)	32.6% (194)
Medium	55.7% (371)	46.5% (20)	53.6% (15)	56.5% (336)
High	10.7% (71)	11.6% (5)	3.6% (1)	10.9% (65)
Mothers depression on DSSI				
Depressed	67.7% (478)	64.4% (29)	70% (21)	67.8% (428)
Not Depressed	32.3% (228)	35.6% (16)	30% (9)	32.2% (203)

half of the sample (56%) were middle-income earners, with 33.6% low income earners, and 10.7% high-income earners. Chi square analyses found no association between PTSD, and ethnicity, mother's educational level and family income.

Mothers' Depression

Principal investigators were interested in the impact of maternal depression on child outcome and the study cohort was over-sampled for mothers' depression based upon scores on the DSSI administered at the first antenatal visit and at an additional three points after birth and between the child's age 5 years. Almost one third of the sample had mothers who did not experience depressive symptoms during the first five years (MUSP control group) and the remaining two-thirds of the sample had mothers with varying levels of depressive symptoms (moderate or severe scores on the DSSI) across one or more of the four assessment points. Therefore, categories of depressed and not depressed were formed based upon continuous variables of DSSI scores. Whilst suggestive of diagnostic categories, the two groups do not necessarily reflect depression diagnoses. Because this sample was selected to include about 70% of mothers with varying levels of self reported depression symptoms at one or more time-points on the DSSI during the first five years of the child's life, critics may argue that the results of this study on PTSD cannot be generalized to a normal community sample. To transparently explore and report the impact of early maternal depressive symptoms, and the subject selection, all variables are reported with mother's depression group. Refer to Table 2 for sample characteristics of young people with depressed mothers and Table 3 for young people without depressed mothers. Chi-

square analyses were conducted to examine differences across variables based upon mothers' depression.

Although this sample is over represented for maternal depression prior to child's age 5, there were no statistically significant differences in mother's depression for offspring PTSD [$\chi^2 (2) = 0.295, p = 0.863$], mother's ethnicity [$\chi^2 (1) = 0.331, p = 0.565$], and mother's education [$\chi^2 (2) = 1.81, p = 0.404$]. There were, however, significant differences in family income between families with depressed and non-depressed mothers [$\chi^2 (2) = 22.765, p = 0.000$]. Families of depressed mothers were more likely to have lower incomes than families without depressed mothers. Overall, chi square analyses suggest that mother's depression scores on the DSSI prior to the child's age 5, and which formed the basis of the selection of the sample, was not significantly associated with onset of PTSD symptoms in this child cohort.

Given that latter analyses also examine comorbidity of other psychological diagnoses with PTSD, analyses were conducted to test for associations between mother's depression scores on the DSSI and other lifetime diagnoses for young people. In addition to having no association with PTSD amongst offspring, there was no significant difference between mothers' depression status and child status for major depression [$\chi^2 (1) = 2.374, p = 0.123$], substance use disorders combined [$\chi^2 (1) = 2.011, p = 0.156$], alcohol use disorders [$\chi^2 (1) = .687, p = 0.407$], cannabis use disorders [$\chi^2 (1) = 3.244, p = 0.072$], behavioural disorders [$\chi^2 (1) = .056, p = 0.814$], and suicidal behaviours [$\chi^2 (2) = 1.579, p = 0.454$]. However, there was a significant difference for other anxiety disorders [$\chi^2 (1) = 8.342, p = 0.004$] with depressed mothers more likely to have children with other anxiety disorders.

Table 2

Sample characteristics for young people with depressed mothers on the DSSI prior to age 5 years (n = 478).

Characteristics	Cohort	PTSD	Subclinical	No PTSD
	(n = 478)	(n = 29)	(n = 21)	(n = 428)
Male	47.1% (225)	20.7% (6)	33.3% (7)	49.5% (212)
Female	52.9% (253)	79.3% (23)	66.7% (14)	50.5% (216)
Caucasian	90.8% (434)	89.7% (26)	95.2% (20)	90.7% (388)
Indigenous	9.2% (44)	10.3% (3)	4.8% (1)	9.3% (40)
Mothers Education				
Junior School	19.1% (91)	20.7% (6)	9.5% (2)	19.5% (83)
Senior School	61.6% (293)	62.1% (18)	71.4% (15)	61% (260)
Tertiary	19.3% (92)	17.2% (5)	19% (4)	19.5% (83)
Family income				
Low	38.7% (174)	50% (14)	47.6% (10)	37.4% (150)
Medium	51.8% (233)	46.4% (14)	47.6% (10)	52.4% (210)
High	9.6% (43)	3.6% (1)	4.8% (1)	10.2% (41)

Table 3

Sample characteristics for young people without depressed mothers on the DSSI prior to age 5 years (n = 228).

	Cohort	PTSD	Subclinical PTSD	No PTSD
Characteristics	(n = 228)	(n = 16)	(n = 9)	(n = 203)
Male	52.2% (119)	12.5% (2)	33.3% (3)	66.2% (114)
Female	47.8% (109)	87.5% (14)	66.7% (6)	43.8% (89)
Caucasian	92.1% (210)	87.5% (14)	100% (9)	92.1% (187)
Indigenous	7.9% (18)	12.5% (2)	0% (0)	7.1% (16)
Mothers education				
Junior school	11.1% (25)	6.3% (1)	25% (2)	10.9% (22)
Senior school	66.8% (151)	93.8% (15)	62.50% (5)	64.9% (131)
Tertiary	22.1% (50)	0% (0)	12.5% (1)	24.3% (49)
Family income				
Low	23.1% (50)	26.7% (4)	28.6% (2)	22.7% (44)
Medium	63.9% (138)	46.7% (7)	71.4% (5)	64.9% (126)
High	13% (28)	26.7% (4)	0% (0)	12.4% (24)

Age of Onset

Ninety-two percent of participants with PTSD provided a specific date of onset ($n = 69$), the minimum age being 6 years old and the maximum age being 21 years old (mean = 15 years and 4 months, $SD = 3$ years and 6 months). There was no significant difference in mean age of onset between clinical and subclinical PTSD ($F(1,67) = 2.714, p = .104$). Combining PTSD and subclinical PTSD, young people with PTSD symptoms were most likely to have an age of onset after age 15 ($n = 45, 60\%$). Almost one-third ($n = 23; 30.7\%$) of the young people with PTSD symptoms had an age of onset during the adolescent years of 10 to 15 years old, and 9.3% ($n = 7$) had an early onset under 10 years of age. This equates to 6.4%, 3.25%, and 0.9% of the entire sample for these age groups respectively. There was no significant difference in age of onset between males and females ($F(1,67) = 0.690, p = .409$) or between mothers' depression group ($F(1,67) = 0.905, p = .345$).

PTSD Chronicity

The estimated duration of clinical and subclinical PTSD symptoms ranged from 1 month to 10 years, with a mean duration of 1 year and 11 months ($SD = 2$ years and 7 months). More than one-third (36%) of young people with clinical or subclinical PTSD had continuing PTSD symptoms at the time of the 20-year interviews. Therefore, these figures underestimate the chronicity of symptomatology. No significant difference was observed in mean duration of PTSD episode between clinical PTSD and subclinical PTSD ($F(1,67) = 0.010, p = .919$). Similarly, no significant difference was observed in mean duration of symptoms between males and

females ($F(1,67) = 0.887, p = .350$) or between mothers' depression group ($F(1,67) = 2.184, p = .144$).

Sixty-one percent ($n = 42$) of young people experienced chronic PTSD and subclinical PTSD symptoms that exceeded 6 months in duration (as per current DSM-IV-TR diagnostic criteria for chronic PTSD). Eighteen percent ($n = 13$) had PTSD symptoms exceeding 3 years in duration. Again, there was no statistically significant difference in chronicity of symptoms between clinical and subclinical PTSD [$\chi^2(1) = 2.34, p = .126$]. However, there was a statistically significant difference between genders on chronicity over 6 months, with females more likely than males to experience chronic symptoms [$\chi^2(1) = 4.78, p = .029$]. Over two-thirds ($n = 36; 68\%$) of the females with PTSD had chronic symptoms exceeding 6 months in duration, compared with one-third of the males ($n = 6; 37.5\%$). Females with a duration of symptoms over 6 months represented just over half (52.2%) of the total sample of children and young people with PTSD.

Recurrence

Recurrence of PTSD (i.e. relapse) was significantly more likely for females than males ($F(1,704) = 9.75, p = .002$). Ten young people (13%), all females, reported more than one episode of PTSD or subclinical PTSD. No significant association was found for recurrence and mothers' depression group ($F(1,67) = 0.225, p = .636$).

Triggering Events

Table 4 outlines the triggering events for PTSD in this sample. By far the most common triggering event was a reported sexual assault of young people and included rape. Witnessing severe injury or death to another person (including witnessing domestic violence) was the second most common reported precipitating traumatic event closely followed by accidental injury (including vehicle accidents). Finally, physical assault (including being held at gunpoint) and learning of a traumatic event experienced by someone else were less common. Whilst it appeared that males were more likely than females to report accidental injury (which included vehicle accidents) as the triggering event, this relationship was not significant [$\chi^2 (1) = 3.356, p = .067$]. There was one significant gender difference observed for triggering events. More females than males reported 'sexual assault' as the triggering event [$\chi^2 (1) = 16.86, p = 0.000$]. Sample sizes were too small to conduct analyses on mothers' depression and triggering events.

Diagnostic Co-Morbidity

Table 5 illustrates the various psychological disorders experienced by the sample. Chi square analyses were conducted to examine differences between three outcome groups; young people with PTSD, subclinical PTSD, and young people without PTSD. As presented in Table 5, a diagnosis of PTSD and subclinical PTSD was highly comorbid with a lifetime diagnosis of major depression. Compared to young people with no PTSD, those with clinical PTSD and subclinical PTSD had higher rates of lifetime

Table 4

Frequency of triggering events by gender for PTSD and subclinical PTSD (n = 75).

Triggering event	Gender		
	Total (n = 75)	Males (n = 18)	Females (n = 57)
Sexual assault	37.3% (28)	16.6% (3)	43.9% (25)
Mother depressed	24% (18)	11.1% (2)	28.1% (16)
Mother not depressed	13.3% (10)	5.5% (1)	15.8% (9)
Witnessed death or serious injury	20.0% (15)	22.2% (4)	19.3% (11)
Mother depressed	14.7% (11)	22.2% (4)	12.3% (7)
Mother not depressed	5.3% (4)	0% (0)	70% (4)
Accidental injury	18.7% (14)	33.3% (6)	14.0% (8)
Mother depressed	13.3% (10)	16.7% (3)	12.3% (7)
Mother not depressed	5.3% (4)	16.7% (3)	1.7% (1)
Physical assault	13.3% (10)	16.7% (3)	12.3% (7)
Mother depressed	5.3% (4)	11.1% (2)	3.5% (2)
Mother not depressed	8% (6)	5.5% (1)	8.8% (5)
Learned of traumatic event experienced by another person	10.7% (8)	11.1% (2)	10.5% (6)
Mother depressed	9.3% (7)	11.1% (2)	8.8% (5)
Mother not depressed	1.3% (1)	0% (0)	1.7% (1)

major depression [$\chi^2(2) = 52.716, p = .000$]. Furthermore, a diagnosis of major depression, or anxiety disorder was more common among young people with clinical PTSD than young people with subclinical PTSD [$\chi^2(1) = 6.520, p = .011$; $\chi^2(1) = 11.343, p = .001$ respectively]. Compared to young people with no PTSD and subclinical PTSD, those with clinical PTSD had higher rates of other lifetime anxiety disorders [$\chi^2(2) = 27.329, p = .000$]. There were similar rates of lifetime anxiety disorders between young people with subclinical PTSD and with no PTSD.

Similarly, lifetime diagnoses of substance use disorders (combined alcohol, cannabis, amphetamines, opioids, sedatives, ecstasy, and polysubstance) were also highly comorbid with PTSD diagnoses. Compared to young people with no PTSD, young people with clinical PTSD and subclinical PTSD had higher rates of any lifetime substance use disorder [$\chi^2(2) = 13.445, p = .001$]. No significant difference in lifetime substance disorders was observed between clinical and subclinical PTSD [$\chi^2(1) = 1.768, p = .184$]. There was no statistically significant difference between groups for alcohol use disorders [$\chi^2(2) = 4.931, p = .085$], possibly due to the high levels of alcohol use diagnoses in the total sample of young people ($n = 197, 27.9\%$). Cannabis use disorders were also common across the total sample of young people ($n = 159, 22.5\%$), and the association with PTSD was approaching significance [$\chi^2(2) = 5.940, p = .051$].

Clinical PTSD and subclinical PTSD were collapsed together (combined), due to low numbers, to allow statistical analysis of other specific substance use disorders. All other comorbidity analyses analysed clinical and subclinical PTSD separately. Young people with clinical or subclinical PTSD (combined) had higher rates of

Table 5

Comorbid diagnoses across subgroups (n = 706).

Comorbid diagnoses	PTSD (n= 45)	Subclinical PTSD (n= 30)	No PTSD (n = 631)
Major depression	75.5% (34)	46.7% (14)	26.1% (165)
Mother depressed	46.6% (21)	36.7% (11)	19.2% (121)
Mother not depressed	28.9% (13)	10% (3)	6.9% (44)
Anxiety disorders	75.6% (34)	36.7% (11)	36.3% (229)
Mother depressed	51.1% (23)	30% (9)	27.1% (171)
Mother not depressed	24.4% (11)	6.7% (2)	9.2% (58)
Substance use disorder	62.2% (28)	46.7% (14)	35.8% (226)
Mother depressed	42.2% (19)	26.7% (8)	72.1% (163)
Mother not depressed	20% (9)	20% (6)	27.9% (63)
Cannabis use disorder	35.6% (16)	30% (9)	21.2% (134)
Mother depressed	26.7% (12)	13.3% (4)	75.4 (101)
Mother not depressed	8.9% (4)	16.7% (5)	24.6% (33)
Alcohol use disorder	40% (18)	36.7% (11)	26.6% (168)
Mother depressed	26.7% (12)	23.3% (7)	70.8% (119)
Mother not depressed	13.3% (6)	13.3% (4)	29.2% (49)
Amphetamine use disorder	20% (9)	6.7% (2)	6.2% (39)
Mother depressed	13.3% (6)	6.7% (2)	74.4% (29)
Mother not depressed	6.7% (3)	0% (0)	25.6% (10)
Ecstasy use disorder	4.4% (2)	6.7% (2)	1.3% (8)
Mother depressed	0% (0)	6.7% (2)	75% (6)
Mother not depressed	4.4% (2)	0% (0)	25% (2)
Opioid use disorder	6.7% (3)	0% (0)	1.3% (8)
Mother depressed	6.7% (3)	0% (0)	1.0% (6)
Mother not depressed	0% (0)	0% (0)	0.3% (2)
Hallucinogen use disorder	2.2% (1)	0% (0)	0.5% (3)
Mother depressed	2.2% (1)	0% (0)	0.3% (2)
Mother not depressed	0% (0)	0% (0)	0.2% (1)

	PTSD (<i>n</i> = 45)	Subclinical PTSD (<i>n</i> = 30)	No PTSD (<i>n</i> = 631)
Comorbid diagnoses			
Behavioural disorders	8.9% (4)	13.3% (4)	4.4% (28)
Mother depressed	8.9% (4)	3.3% (1)	3% (19)
Mother not depressed	0% (0)	10% (3)	1.4% (9)
Suicide attempt	17.8% (8)	0% (0)	2.5% (16)
Mother depressed	15.6% (7)	0% (0)	1.6% (10)
Mother not depressed	2.2% (1)	0% (0)	0.9% (6)

lifetime amphetamine use disorders [$\chi^2(1) = 7.335, p = .007$], and ecstasy use disorders ($F(1,704) = 6.675, p = .010$) compared to those with no PTSD. No significant differences were observed for lifetime opioid use disorder ($F(1,704) = 3.268, p = .071$) or hallucinogen disorder ($F(1,704) = .874, p = .350$).

Behavioural Disorders (oppositional defiant disorder and conduct disorder) were also commonly comorbid with a diagnosis of clinical PTSD and subclinical PTSD. Compared to young people with no PTSD, those with clinical PTSD and subclinical PTSD had higher rates of behavioural disorders [$\chi^2(2) = 6.110, p = .000$]. No significant difference in behavioural disorders was observed between clinical and subclinical PTSD [$\chi^2(1) = 0.071, p = .541$].

High rates of suicide attempts were found among young people with PTSD. A total of 19% ($n = 8$) of participants with clinically significant PTSD diagnoses were found to have attempted suicide at least once and a further 19% ($n = 8$) had considered suicide but had not attempted. None of the participants with subclinical PTSD had

attempted suicide, but 20% reported suicidal ideation. Additional analyses were conducted to determine the role of major depression in the suicidal behaviours of young people with PTSD. Young people with a lifetime diagnosis of clinical PTSD were more likely to have attempted suicide than those with a lifetime diagnosis of depression ($n = 19, 9.6\%$), and no PTSD ($n = 5, 1.1\%$), and this relationship was statistically significant [$\chi^2(2) = 41.271, p = .000$]. However, all of those with PTSD who had attempted suicide ($n = 8$) also had a lifetime depression diagnosis. Thus, 25% of young people with comorbid lifetime diagnoses of clinical PTSD and major depression had attempted suicide ($n = 8$), compared with 7.7% of participants with major depression and without PTSD ($n = 10$). None of the participants with PTSD and without depression attempted suicide. It would seem then, that comorbid lifetime depression and PTSD accounted for the higher rates of suicide attempt.

Discussion

Studies that follow people, particularly children, as they grow and develop are the best source of information about the prevalence, causes of continuity and discontinuity and presentation of psychological disorders. This study aimed to explore the frequency, duration, precipitating events and psychological comorbidity of PTSD in an Australian longitudinal sample of 706 community dwelling children and young people. The aim was to enhance our awareness and understanding of PTSD among this younger population. This study also aimed to examine the relevance of early maternal depressive symptoms on PTSD symptoms amongst offspring.

Unlike the findings from the Australian National Survey of Mental Health and Wellbeing (ABS, 2007), lifetime PTSD amongst children and young people was not the most commonly diagnosed anxiety disorder, and was not more common than depression, which was diagnosed in 30% of the sample. The estimated 12 month incidence of clinical PTSD amongst Australian adults aged 16 to 85 years old is 6.4% (ABS, 2007). The American adult prevalence of PTSD is approximately 8% (APA, 1994) and the American child estimate is between 3.5% (Cuffe et al., 1998) and 6.3% (Giaconia et al., 1995). Supporting the first hypothesis, the lifetime prevalence of clinical PTSD in this sample of young people was 6.4%, a figure mirroring previous adult rates in this country and closer to American child estimates. A further 4.2% experienced subclinical symptoms of PTSD. This brought the total prevalence of lifetime PTSD symptoms to a considerable 10.6%, suggesting that PTSD is not uncommon in Australian children and young people and is certainly worthy of more attention than it has received.

In this sample of children and young people, PTSD was significantly more common among females than males. Over 80% of the children and young people with PTSD, and close to 70% of the children with subclinical PTSD were female. The most common triggering event for PTSD symptoms in this sample was sexual assault, which precipitated 37% of clinical and subclinical PTSD cases. Significantly more females than males reported sexual assault as the triggering event, which may explain the gender differences in this sample. Unfortunately, this study was unable to analyse the prevalence of PTSD symptoms among all males and females who had ever experienced sexual assault to explore these gender differences in more depth.

Previous authors have argued that PTSD in children is rare (Ford, Goodman, & Melzer, 2003; Koenen et al., 2007). Ford and colleagues found prevalence rates of less than 0.5% amongst a sample of over 10,000 British children under 15 years of age (Ford et al., 2003). The assertion that PTSD is rare in childhood may lead to the exclusion of younger children from PTSD research, and the assumption that risk factors were measured prior to PTSD onset based solely upon the age of the child at the time of measurement rather than a thorough assessment of whether the child already presented with PTSD symptoms prior to measurement. The findings of Ford et al (2003) are clearly not supported by the results of this Australian study, which found that 4.2% of the total sample developed PTSD symptoms (clinical and subclinical combined) prior to age 15 years, and 1.7% had full clinical PTSD diagnoses prior to age 15 years. Therefore, these findings are three times higher than the prevalence rates of PTSD in young children that are often quoted in the literature, and used to justify the omission of young children from PTSD research. Consistent with the findings of this study, increasingly researchers are recognising that PTSD is not rare even in pre-school aged children (Scheeringa, Zeanah, & Cohen, 2010). In support of the second hypothesis, the prevalence of PTSD was found to increase with age in this study, however, about 40% of children with either clinical or subclinical PTSD developed symptoms before age 15 years. About 10% of children with PTSD symptoms developed symptoms in early childhood (0-10 years), followed by about 30% in middle childhood (10 to 15 years), and finally about 60% developed symptoms in later adolescence and early adulthood (15 to 20 years old). There was no difference in age of onset between males and females. Therefore, whilst the prevalence of PTSD

increased with age and was more likely to develop in later adolescence, the view that PTSD is rare in childhood was not supported. It is argued that this view must be updated if we are to give this disorder the serious attention it deserves.

Interestingly, there was no relationship with the mothers' early depressive symptoms measured by the DSSI and the prevalence, and age of onset of PTSD in this sample. Whilst this sample was over-selected for mothers with self-reported depressive symptoms on the DSSI prior to child age 5 years, this factor was not associated with PTSD symptoms in this sample. Mothers' early depressive symptoms were associated with other anxiety disorders (combined generalised anxiety disorder, separation anxiety disorder, and phobias), but not PTSD specifically. The finding that early maternal depression was not associated with PTSD was unexpected, but there are a couple of possible explanations. Whilst apparently inconsistent with previous risk factor research, the differing results may be accounted for by differing methodologies. The current study used the DSSI questionnaire to assess mother's depressive symptoms during pregnancy and prior to the child reaching age 5 years. The DSSI is a measure of depressive symptoms, not clinical diagnoses of depressive disorders. Different results may have been obtained with the use of diagnostic interviews capturing clinical diagnoses of major depression rather than the broad range of depressive symptoms captured with the use of the questionnaire measure used in this study. Secondly, mother's early depressive symptoms were analysed. The measurement of depressive symptoms closer in proximity to the child's onset of PTSD symptoms may have yielded different results. Thirdly, consistent with a growing number of studies of multiple risk factor models of the effects of maternal depression,

the effects of mother's depression may be largely mediated by other factors (Hammen, Brennan, & Shih, 2004). Early maternal depression symptoms alone, in the absence of other risks or adversities, may not be associated with child PTSD in this sample and may instead be mediated by variables with which maternal depression may be strongly associated. For example, using this very data sample, Hammen and colleagues (2004) found that maternal depression in the absence of family conflict was not associated with depression in offspring. Similarly, other factors may mediate the relationship between maternal depression and PTSD in offspring. Given the, albeit surprising, lack of association between maternal depression symptoms and offspring PTSD, it can therefore be argued that the PTSD results obtained in this study may indeed be generalised to a normal community sample of Australian children and young people.

The third hypothesis in relation to the chronicity and comorbidity of PTSD in children and young people was also supported. Consistent with adult findings (Kessler et al., 1995), this study also found that PTSD in children and young people was often chronic. It should also be noted that a large proportion of young people with PTSD (clinical or subclinical) (36%) had ongoing symptoms at the time of participation, therefore, these results underestimate the chronicity of PTSD, with the true duration unknown at this time. In this context, the mean duration of PTSD was close to two years, and did not statistically differ between clinical and subclinical diagnoses. The DSM-IV (APA, 1994) defines PTSD as 'chronic' if symptoms exceed 6 months in duration. According to this definition, over 60% of children and young people with PTSD experienced chronic PTSD symptoms, making chronicity the norm rather than the exception. Females were more likely than males to experience chronic PTSD

symptoms. Thus, not only were females more likely to be diagnosed with PTSD, they were also more likely to experience chronic symptoms exceeding 6-months in duration, and to experience relapse, suggesting a poorer prognosis for females with PTSD generally. These findings raise questions about the usefulness of the DSM-IV-TR definition of chronicity for PTSD, given that by this definition, most children, young people and adults with PTSD seem to experience chronic symptoms.

Consistent with previous international research (Dislavet et al., 2007; Goenjian et al., 1995; McCloskey & Walker, 2000; Perkonigg et al., 2000; Ulzen & Hamilton, 2003; Weber & Reynolds, 2004), PTSD among Australian children and young people was also highly comorbid with lifetime diagnoses of major depression (76%), other anxiety disorders (76%), and substance use disorders (62%), and to a lesser degree behavioural disorders (9%). Therefore, although PTSD is often considered to be an internalising disorder, it is highly comorbid with both internalising and externalising symptoms. All four diagnoses (major depression, other anxiety disorders, substance use disorders and behavioural disorders) were significantly more common amongst those with clinical PTSD than those with no PTSD. Interestingly, compared with children and young people with no PTSD, those with subclinical PTSD were also significantly more likely to have lifetime diagnoses of major depression (47%), substance use disorders (47%), and behavioural disorders (13%), but not other anxiety disorders (37%). Therefore, it would seem that a lifetime comorbidity of PTSD symptoms with other anxiety disorders may be associated with the development of more severe clinical features of PTSD among children and young people. Having a

lifetime diagnosis of another anxiety disorder seemed to discriminate between whether the PTSD symptoms were clinical or subclinical.

The high comorbidity of PTSD with substance use disorders amongst this young population is quite disturbing, especially given that a comorbid substance use problem is known to lead to a poorer response to PTSD treatments (Back et al., 2005). Also of significant concern was the high prevalence of alcohol and cannabis use disorders in the sample (28% and 22.5% respectively). According to DSM-IV diagnostic criteria, approximately one in five young Australians in this sample had a lifetime alcohol use disorder, and one in four had a lifetime cannabis use disorder prior to age 20 years. Furthermore, maternal depression symptoms did not account for this finding, suggesting an accepted culture of alcohol abuse amongst Australian young people. Although lifetime comorbidity rates of alcohol use disorders were high amongst those with PTSD (40%) they were not statistically different from those with subclinical PTSD and no PTSD. Lifetime cannabis use disorders were also highly comorbid with PTSD (36%), but the difference in cannabis use disorders between PTSD and no PTSD groups was only borderline in significance ($p = 0.051$) and is worthy of further research. However, whilst the numbers were small, there was a statistically significant difference between groups of young people in their use of amphetamines and ecstasy. One in five (20%) young people with PTSD also had a lifetime amphetamine use diagnosis, compared with 6.7% of young people with subclinical PTSD and 6.2% with no PTSD. A diagnosis of ecstasy abuse was made for 5.3% of young people with either clinical or subclinical PTSD, compared with 1.3% of young people with no PTSD. As discussed in Chapter 1, alcohol and substance use

disorders often develop after PTSD onset, supporting a self medication hypothesis. Unfortunately, this study did not examine the temporal sequencing of psychological diagnoses, and therefore conclusions cannot be drawn regarding whether these disorders were precipitants or antecedents to PTSD and trauma exposure.

The high rate of suicide attempts among young people with PTSD was of great concern. Consistent with previous research with adult samples (Davidson et al., 1991; Kotler et al., 2001; TARRIER & GREGG, 2004), almost one in five (19%) young people with PTSD had attempted suicide at least once. An additional 19% had contemplated suicide but had never attempted. Overall, young people with lifetime diagnoses of both major depression and PTSD were at greater risk of suicide. One in four (25%) young people with lifetime diagnoses of both depression and PTSD had attempted suicide and analyses found that comorbid depression and PTSD accounted for the higher rates of suicide attempts. Therefore, it may be concluded that amongst young people, comorbid depression and PTSD may significantly increase the risk of suicide attempt compared with depression or PTSD alone. This is an issue that is yet to be seriously incorporated into standard assessment practices.

The strengths and limitations of this study, together with future implications will be discussed in Chapter 4 in conjunction with those from Study 2. In conclusion of this study however, the results suggest that PTSD was not uncommon amongst community dwelling Australian children and young people. Whilst the likelihood of developing PTSD was found to increase with age, a substantial proportion of this sample developed PTSD prior to reaching age 15 years. PTSD was significantly more common amongst females, who were more likely to report sexual assault as a

triggering event for PTSD symptoms. Overall, the prognosis for PTSD was poorer for females, who were more likely to experience chronic (exceeding six months duration) and recurrent PTSD symptoms. Consistent with adult comorbidity studies, PTSD in children and young people was also found to often be chronic and highly comorbid with lifetime diagnoses of major depression, other anxiety disorders, and substance use disorders. Behavioural disorders were also more common amongst children and young people with PTSD. This study found significant rates of suicide attempts (25%) amongst young people with comorbid major depression and PTSD. These are all characteristics pointing toward PTSD as a serious, chronic, and debilitating disorder. Effective service planning in Australia depends in the first instance, on accurate estimates of the prevalence of PTSD and an understanding of the course, prognosis, psychological comorbidity, and suicide risk associated with this disorder. Without this awareness, prevention, detection, and treatment attempts remain haphazard, inadequate and untargeted, a situation that is unacceptable if we are to relieve the current distress associated with PTSD, improve future functioning, and prevent the intergenerational transmission of disadvantage and psychological dysfunction.

CHAPTER 3

Study Two: Predictors of PTSD in Young People

Post traumatic Stress Disorder (PTSD) is a serious, often chronic, psychological disorder that causes considerable impairment in functioning for children, young people, and adults alike. An understanding of PTSD in childhood and adolescence is extremely important for early prevention, detection, and treatment to not only relieve the experience of distress and assist a return to a normal developmental trajectory, but to improve functioning in later adulthood and prevent the long term intergenerational deleterious effects of psychological dysfunction. Risk factor studies have been fundamental to current understanding of the diagnosis of Posttraumatic Stress Disorder (PTSD) and the large variation in individual responses to trauma. Studies that examine risk factors for PTSD in children and young people are vital to targeted and informed prevention and treatment efforts. An understanding of the salient risk factors is important in order to design effective prevention and treatment initiatives that help to safeguard children and young people against the negative impact of PTSD. As outlined in detail in Chapter 1, a range of potential individual and environmental risk factors for PTSD have been implicated. Many previous studies are limited by methodological problems. Significant limitations include retrospective methodological designs that may confuse cause and effect, use of self-report questionnaires to measure symptoms, use of individuals with PTSD as primary informants regarding familial psychopathology, the generalisation of results

from adult samples to younger members of the community, and the glaring lack of Australian risk factor studies.

Prospective longitudinal studies designed to identify risk factors for PTSD prior to traumatic exposure are scarce, partly because identification of future exposure to potentially traumatic experiences that would qualify for a PTSD diagnosis is very difficult but also because longitudinal studies are difficult to execute, are expensive, and are labour-intensive. Whilst some traumatic events may be easily identifiable (e.g. natural disasters, public incidents of violence, hospitalisation after accidents and injuries), many traumatic events experienced by community members occur in private and may not lead them to attend hospitals or welfare services where they can be more readily identified. As a result, whilst there has been a dramatic rise in the number of published longitudinal studies in recent years, much of the available literature measures risk factors prior to PTSD onset, but targets individuals or communities who have recently been exposed to a traumatic event but have not yet had time to develop a diagnosis of PTSD (DSM-IV-TR diagnostic criteria requires symptom duration of at least one month). Most of the studies that are truly prospective in design and measure factors prior to trauma exposure tend to use adult combat veteran samples as these men and women were usually assessed prior to entry into the defence forces. Prospective longitudinal designs are still needed to measure factors before exposure to trauma and onset of PTSD and to add credibility and accuracy to the potential risk factors.

Childhood risk factors are considered to play an important role in the development of PTSD and recent researchers (Koenen et al., 2007; Pynoos et al., 1999) have argued that PTSD may have developmental origins. However, there have

been very few genuine prospective longitudinal risk factor studies using a community population and examining childhood risk factors. This study addresses current gaps in the PTSD literature by using formal DSM-IV-TR diagnostic criteria and semi-structured interviewing procedures for measuring diagnoses. Furthermore, this study includes family member informants in a prospective longitudinal design to examine childhood risk factors for the development of PTSD in community dwelling Australian young people. The same Australian prospective birth cohort from Study 1 was used to examine premorbid risk factors for the development of PTSD in young people after age 15 years. There are two parts to this study. Part A will examine which risk factors differentiate these young people with PTSD from those without PTSD. All predictor variables in Part A were measured prior to age 15 and prior to onset of PTSD symptoms. Given that major depression is also well-known to be a diagnostic sequelae of trauma, Part B will examine which risk factors differentiate young people with PTSD from young people with major depression. All predictor variables in Part B were measured leading up to the onset of PTSD or major depression and therefore included factors before and after age 15 and closer in proximity to diagnostic onset. Examining the risk factors for PTSD symptoms when compared with an alternative diagnosis (such as in Part B), had the unique benefit of allowing for the inclusion of risk factors leading up to onset of diagnosis, rather than simply prior to a specific age cut-off. Therefore, Part B of this study has the added strength of examining risk factors in closer proximity to the onset of PTSD symptoms, as well as allowing for a comparison of risk factors for these two serious forms of psychological disorder among this young

population. To the best of this author's knowledge, no other published researcher has yet compared risk factors for PTSD with those for major depression.

A large set of potential individual and environmental childhood risk factors were selected from the literature discussed in detail in Chapter 1. Specifically, it was hypothesised that the following within individual variables would differentiate young people with PTSD from those with no PTSD in Part A, and from those with major depression in Part B: female gender; presence of major depressive disorder, anxiety disorders, substance use disorders and behavioural disorders; high internalising symptoms; high externalising symptoms; and low intelligence. It was also hypothesised that the following environmental variables would differentiate young people with PTSD from those with no PTSD (Part A) and major depression (Part B): the accumulation of premorbid stressful life events; a history of having been physically or sexually abused; poor social support and social integration; presence of mother's major depressive disorder, anxiety disorder, or comorbid depression and anxiety disorder; and the mother's history of having been physically or sexually abused. In light of the impracticalities of designing prevention and treatment efforts targeting a large number of risk factors, it would seem that programs would be best informed by the identification of the most salient predictors of PTSD. Therefore, variables were tested for their unique contribution to PTSD symptoms, when compared with a control group without PTSD in Part A, and a control group with Major Depression diagnoses in Part B. The inclusion of many different risk factors into one analysis to identify the most significant risk factors is the next logical step in understanding PTSD etiology. Therefore, all significant risk factors were examined as a group to determine which

significantly predicted PTSD in young people when compared with no PTSD (Part A) and Major Depression (Part B).

Methodology

Procedure

The background and procedure of this study are identical to those of Study 1.

Participants – Part A

Participants in Part A of the present study were a subsample of those participating in Study 1. Specifically, Part A comprised of PTSD ($n = 48$) and No PTSD ($n = 631$) categories. Young people who experienced onset of clinical PTSD ($n=32$) or subclinical PTSD ($n=16$) after age 15 were collapsed into one group. Young people diagnosed with PTSD ($n=13$) or subclinical PTSD ($n=14$) prior to age 15 who had no new onset of PTSD disorder after age 15 were excluded, to allow for investigation of premorbid predictive factors. The control group consisted of all other young people in the study without lifetime diagnoses of PTSD (No PTSD group; $n = 631$). Those without PTSD included young people with and without other diagnoses. The total sample size for analyses in Part A was 679 young people and their mothers. Premorbid diagnoses for the young people were measured at the 15-year follow-up, and thus include only diagnoses with an onset prior to age 15.

Sample characteristics for Part A participants are presented in Table 6 and include gender, ethnicity, mother's highest educational attainment, and family income

Table 6

Part A: Sample characteristics across subgroups (n=679)

	Cohort (n = 679)	PTSD (n = 48)	No PTSD (n= 631)
Characteristics			
Male	49.5% (336)	20.8% (10)	51.7% (326)
Female	50.5% (343)	79.2% (38)	48.3% (305)
Mean age at 15-year follow-up	15.2 ± 0.3	15.1 ± 0.2	15.2 ± 0.3
Caucasian	91.0% (618)	89.6% (43)	91.1% (575)
Aboriginal	9.0% (61)	10.4% (5)	8.9% (56)
Mothers education			
Junior school	16% (108)	6.4% (3)	16.7% (105)
Senior completion	63.6% (429)	80.9% (38)	62.3% (391)
Tertiary	20.4% (138)	12.8% (6)	21% (132)
Family income			
Low	33.5% (213)	42.2% (19)	32.6% (194)
Medium	55.8% (357)	46.7% (21)	56.5% (336)
High	10.9% (70)	11.1% (5)	10.9% (65)

at the age 15 follow-up. Descriptive analyses and unadjusted binomial logistic regression analyses found no association between ethnicity, mother's highest educational level and family income with the outcome variable (PTSD and No PTSD). A significant difference between PTSD and No PTSD groups was observed for gender [$\chi^2(1) = 16.962, p = .000$] with the majority of young people with PTSD being female ($n = 38; 79\%$), caucasian ($n = 43; 89.6\%$), and from families of low to medium income. Over half had mothers who had not completed Year 12 senior schooling ($n = 27; 57.4\%$).

Participants – Part B

Participants in Part B of the present study were also a subsample of those participating in Study 1. Specifically, part B compared risk factors for young people with PTSD ($n = 48$) with young people with Major Depression ($n = 142$). The PTSD group was identical to those in Part A and therefore, some also had comorbid major depression. The Major Depression group was comprised of all young people who experienced a major depressive episode (without PTSD) after age 15 years. Young people diagnosed with either PTSD or Major Depression prior to age 15 who had no new onset of PTSD or major depression after age 15 were excluded, to allow for investigation of genuine premorbid factors. All other young people with no diagnosis or who had other DSM-IV-TR diagnoses (but not PTSD and Major Depression) after age 15 were excluded from analyses. The total sample size for analyses in Part B was 190 young people and their mothers.

Dates of onset of premorbid diagnoses became key to examining temporal sequencing of the various diagnoses. For each individual, onset dates for all relevant diagnoses were compared in detail to determine conditions premorbid to onset of PTSD and depression after age 15 years. Therefore, unlike Part A that only examined premorbid diagnoses up to age 15 years, Part B examined all relevant diagnoses prior to the date of onset of PTSD or major depression for each individual up to age 20 years. This allowed for better examination of substance use as a risk factor, which most often commenced after age 15 years, and also allowed for the inclusion of other psychological diagnoses in closer proximity to onset of PTSD or Major Depression. In addition, dates of onset for mother's diagnoses were compared in thorough detail with the onset of PTSD and major depression in young people to determine temporal sequencing.

Measures

Premorbid Diagnoses. As outlined in Chapter 2, the presence of current or lifetime PTSD, depression, anxiety, substance use, and behavioural disorders in the young person at age 15 years was determined using the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Epidemiologic version revised for DSM-IV (K-SADS-E) (Orvaschel, 1995). See Chapter 2 for more detail.

Internalising and externalising symptoms. Pre-morbid internalising was assessed using the Child Behaviour Checklist (CBCL; administered to the father) (Achenbach, 1991b), the Youth Self Report (YSR) (Achenbach, 1991e) and the Teacher Report Form (TRF) (Achenbach, 1991d) administered at the 15-year follow-up. Ratings from the mother were excluded in the current study because of the potential problem of maternal rating bias due to maternal depression (Berg-Nielsen, Vika, & Dahl, 2003; Najman et al., 2000).

The Child Behaviour Checklist (CBCL) (Achenbach, 1991b) was developed as a parent report tool to measure child competencies and behaviour problems from ages four to 18 years. The CBCL consists of 20 social competence and 113 behavioural problem items and generates broad-band (i.e., Externalising, Internalising) and narrow-band behavioural scales (i.e., Social Withdrawal, Somatic Complaints, Anxious/Depressed behaviour, Social Problems, Thought Problems, Attention Problems, Delinquent Behaviour, and Aggressive Behaviour). On all three measures (CBCL, YSR, and TRF), emotional-behavioural problem items are rated on a three-point likert scale indicating if the problem is “very/often true” (2), “somewhat or sometimes true” (1) or “not true” (0). All forms have parallel Internalising Behaviours (fearful, shy, anxious, and inhibited), Externalising (aggressive, antisocial, and undercontrolled), and Total Problems scales. The Internalizing scale is constructed from the sum of the Withdrawn, Somatic Complaints, and Anxious/Depressed scales and is made up of 31 items. The Internalizing scale reflects the internal stress. The Externalizing scale is constructed from the sum of the scores of the Delinquent and Aggressive Behaviour scales and is made up of 32 (CBCL) and 31 (YSR) items. The Externalising scale

reflects conflict with other people and their expectations. Raw scores can be converted to age-standardized scores that can be compared with scores obtained from normative samples of children within the same broad age range. For Total Problems, Externalizing Problems, and Internalizing Problems, T scores less than 60 are considered in the normal range, 60-63 represent borderline scores, and scores greater than 63 are in the clinical range.

The YSR is the youth report counterpart of the CBCL and is a 112 item, self-report measure, that also has competence and problem items which generally parallel those of the CBCL. Young people rate themselves for how true each item is currently, or was within the past six months.

The TRF obtains teachers' reports of children's academic performance, adaptive functioning, and emotional-behavioural problems. The first section of the TRF examines relevant background information, ratings of academic performance, and ratings of four aspects of adaptive functioning. The remaining 112 items comprise a problem behaviour checklist that is a compliment measure to the CBCL and YSR.

Reliability and validity of the CBCL, YSR, and TRF are excellent, however, cross-informant agreement on the CBCL, Teacher Report Form (TRF) and Youth Self Report (YSR) varies according to the groups of informants being compared (Achenbach, 1991a, 1991e; Achenbach, Dumenci, & Rescorla, 2002; Bird & Gould, 1995; Essau, Muris, & Ederer, 2003; Rosenblatt & Rosenblatt, 2002). Concordance (kappa) between raters has been found to be high between mothers and fathers ratings (Internalising $k = .66$; Externalising $k = .80$; Total Problem scale $k = .76$) and moderate between parent and

child ratings (Internalising $k = .40$; Externalising $k = .44$; Total Problem scale $k = .41$) (Rosenblatt & Rosenblatt, 2002). In addition, classification of children into normal, borderline and clinical groups has shown significant agreement between all raters on most scales (Achenbach, 1991a).

In the current study, the internalising and externalising scales were constructed by using the questionnaire results collected from the young person, father and teacher. Normalised T-scores were assigned to the internalising and externalising raw scores and a single internalising and externalising T-score was calculated from the averaged T-scores of all three individual respondents. The internalising T-scores ranged from 31 to 91 (mean = 50.1, SD = 7.98) and the externalising T-scores ranged from 32 to 83.3 (mean = 53.4, SD = 8.27) with higher scores indicating greater internalising or externalising symptoms.

Intelligence. The measure of intelligence was derived from selected sections of the Wechsler Intelligence Scale for Children, Third Edition (WISC-III) (Wechsler, 1991) administered when the young person was 15 years of age. The subtests included were Vocabulary (a measure of verbal intelligence) and Digit Span (a measure of Working Memory). WISC-III protocols were administered and subtests were scored according to standard manualised procedures. The WISC-III has been found to be a highly reliable assessment of intelligence (Wechsler, 1991). Scores were converted to age scaled scores, which on the WISC-III, have a mean score of 10 and a standard deviation of 3. Age scaled scores were used in all analyses. For the purposes of this study, the Vocabulary scaled score and the Digit Span scaled score were summed to

create an intelligence composite score. Intelligence composite scores ranged from 3 to 32 (mean = 17.91, SD = 4.77) with higher scores indicating higher intelligence.

Young person abuse history. At the 15-year follow-up and at the conclusion of comprehensive diagnostic interviewing, the young person and the mother were asked in separate interviews, whether the young person had ever experienced sexual or physical abuse. As discussed above, all interviewers were clinically trained and able to help the mother and young person feel relaxed and comfortable throughout the interview process, to facilitate disclosure of personal information. See Chapter Two for further detail about informed consent and availability of a psychiatric consultant.

Mother and young person interview responses were compiled and the young person was identified as having experienced either physical or sexual abuse if the mother or young person had disclosed this information. A dichotomous variable was formed based upon whether the young person had or had not experienced physical or sexual abuse prior to age 15 years.

Stressful life events. A semi-structured interview procedure developed by one of the principal investigators, Hammen (1991), and modelled after the contextual threat assessment of stressful life events (Brown & Harris, 1978) was administered at age 15 years (refer to Appendix E) and at age 20 years (refer to Appendix F). The stressful life events (episodic stress) interview has been used extensively by Hammen and colleagues (Hammen, 1991) across varying age groups and mood disorders. This interview procedure was developed to overcome many of the limitations of questionnaire checklists and self-report measures that can be biased by the distorted perceptions of the salience of events as a result of the individual's mood. At the age 15

follow-up, the interview probed for the occurrence of stressful life events in the past year. The interviewer inquired about various domains of the person's life, and carefully collected details about the events that occurred during the prescribed period, including the specific dates of events. The interviewer prepared a narrative of each event and probed to obtain information about the event including duration, whether the event was expected, whether the person had previously encountered a similar event, the resources available to cope with the event, the consequences of the event and other relevant details. The interview approach aimed to obtain sufficient information surrounding the event to objectively (as much as is possible) characterise the impact on the person's life. The interviewer omitted details about the individual's actual reaction to the event and presented it to a rating team that was blind to the young person's family status and actual reactions to the event. The team rated each event on a 5-point scale of severity, indicating how much impact the event would have on a typical person under similar conditions (1= no stress or negative impact, 5 = severe stress or negative impact). Interrater reliabilities based upon independent ratings by Australian and U.S. teams for 89 cases at the 15 year follow-up and 130 cases at the 20 year follow-up yielded intraclass correlations of 0.92 and 0.95 respectively. A Spearman rho correlation was conducted. Team ratings and subjective offspring ratings of level of stress and impact for life events were highly correlated ($\rho r = .922$; $p = .000$). In Part A of this study, the stressful life events rating consisted of the sum of all objective team ratings (including those with an impact of 1, i.e. only mildly stressful) for events occurring during the one-year period preceding the 15-year follow-up. In Part B, the stressful life events rating consisted of the sum of objective team ratings for stressful

life events rated moderate (3) to severe (5) in the 12-month period prior to onset of PTSD or depression. The benefit of this change for Part B was that events in Part B were closer in proximity to the PTSD and Major Depression diagnoses rather than simply using a 15 Year cut-off. Another advantage to Part B is that only stressful events rated moderately stressful and above were used, ruling out events elicited as a result of anxious tendencies.

Social support and social integration. Social relationships were measured at age 15 years using a semi-structured interview (Hammen, 1991) developed from earlier measures of chronic strain and functioning for adults and children (refer to Appendix G). The version used in the current study inquired about conditions in four key social domains over the past six months: social life, close friendships, romantic relationships, and relations with family members and includes aspects of both social integration and social support. Interviewers questioned each area with the young person using standard general questions followed by more specific queries where needed. Interviewers scored each domain on a 5-point scale with behaviourally specific anchors (1 represented superior functioning, 5 represented severe difficulties).

The independent team rating of audiotaped interviews ($n = 88-96$) found interclass correlations between .55 (romantic life) and .84 (relationship with family members), with a mean interclass correlation of .70. The four interpersonal relationships scales were summed to form an overall social support score. Overall social support and social integration scores ranged from 5 to 20, with lower scores indicating stronger social integration and social support and higher score indicating poorer social integration and social support.

Maternal diagnoses: depression, anxiety, and comorbid depression and anxiety. In the current study, the Structured Clinical Interview for the DSM-IV (SCID-I) (First et al., 1995) was administered at the 15-year and 20-year follow-ups and was used to ascertain mother's current and lifetime diagnoses of major depressive disorders and anxiety disorders. Interviews were conducted in the privacy of the mother's home and mothers were paid for their participation. Diagnoses were made in consultation with the clinical rating team and interviewers were blind to the mother's scores on previous depression questionnaires, and to previous diagnoses. An independent rating team based in the U.S.A. yielded weighted kappa values of .87 for current diagnoses of depression at the 15-year follow-up and 1.00 at the 20-year follow-up. The kappa values for past depressive diagnoses at the 15 and 20-year follow-ups were .84 and .79 respectively. In the current study, the mother sample included 346 (49%) women with a lifetime diagnosis of Major Depressive Disorder (at least one episode), 370 (52.4%) with a lifetime diagnosis of an anxiety disorder, and 235 (33.3%) women with lifetime diagnoses of both anxiety and major depression disorders.

Maternal abuse history. At the 15-year follow-up and at the conclusion of comprehensive diagnostic interviewing, the mother was asked whether she had ever experienced sexual or physical abuse. Mothers were identified as having experienced either physical or sexual abuse if they so disclosed. A dichotomous variable was formed based upon whether the mother had or had not experienced physical or sexual abuse prior to the 15-year follow-up.

Analytic Strategy

This study assessed the relationship between the independent variables and PTSD symptoms using binomial logistic regression. The model was developed as follows. Crude odds ratios with 95% confidence intervals were calculated by entering the variables separately, with “No PTSD” as the reference category in Part A and “Major Depression” as the reference category in Part B, to estimate the crude association between PTSD symptoms and various factors hypothesised to influence PTSD diagnoses. All variables found to be significantly associated with PTSD in the crude analyses, were then included simultaneously in the first step in the full model. Any variables that were no longer significantly related to PTSD were removed from the model, one at a time (beginning with the least significant), and the impact on the remaining variables assessed. If no changes to the odds ratios of the other variables beyond 10% were observed, then the variable was not included in the final model. Thus, the final model comprises all variables that were significantly associated with PTSD after adjusting for relevant variables, including identified confounders (those that could not be removed as doing so caused a change to odds ratio’s greater than 10%). All statistical tests were two-tailed and α levels set at .05 for main effects and .10 for interactions. All tables are presented with sample sizes.

Results

Part A: PTSD compared with no PTSD

Descriptive characteristics of within individual and environmental predictor variables are presented in Table 7 for the Part A participants. For the within individual variables,

chi-square analyses demonstrated that prior to age 15 years, significantly more young people with PTSD experienced diagnoses of major depression [$\chi^2(1) = 13.662, p = .000$], anxiety [$\chi^2(1) = 7.408, p = .006$], behavioural disorders [$\chi^2(1) = 6.096, p = .014$] and physical or sexual abuse [$\chi^2(1) = 4.690, p = .030$] compared to young people without PTSD, but there was no difference between groups on premorbid substance use disorders [$\chi^2(1) = 2.102, p = .147$] or IQ composite scores ($F(1,630) = 1.630, p = .202$). Between group ANOVA's demonstrate that those in the PTSD group reported significantly higher prior internalising scores ($F(1,677) = 7.292, p = .007$) and externalising scores ($F(1,677) = 10.126, p = .002$) than young people without PTSD.

For the environmental variables, young people with PTSD were found to have had significantly more stressful life events ($F(1,677) = 23.002, p = .000$) and poorer social support ($F(1,676) = 7.776, p = .005$), and were more likely to have had maternal major depression [$\chi^2(1) = 5.521, p = .019$], maternal anxiety disorder [$\chi^2(1) = 5.837, p = .016$], maternal comorbid depression and anxiety [$\chi^2(1) = 6.636, p = .010$], and a maternal history of physical or sexual abuse [$\chi^2(1) = 10.376, p = .001$], when compared with young people with No PTSD.

Table 7

Part A: Predictor variable characteristics across subgroups (n = 679)

Predictors	Cohort (n = 679)	PTSD (n=48)	No PTSD (n = 631)
Within Individual Variables			
Female gender	50.5% (343)	79.2% (38)	48.3% (305)
Premorbid depression	8.5% (58)	22.9% (11)	7.4% (47)
Premorbid anxiety	11.0% (75)	22.9% (11)	10.1% (64)
Premorbid substance use disorders	1.6% (11)	4.2% (2)	1.9% (9)
Premorbid behavioural disorder	5.0% (34)	12.5% (6)	4.4% (28)
Internalising T-score (mean \pm SD)	50.0 (7.8)	52.9 (9.1)	49.7 (7.7)
Externalising T-score (mean \pm SD)	53.3 (8.2)	56.9 (7.7)	53.0 (8.2)
IQ composite scores (mean \pm SD)	18.0 (4.7)	18.8 (4.8)	17.9 (4.7)
Young person abuse history	3.1% (21)	8.3% (4)	2.7% (17)
Environmental Variables			
Maternal depression	34.5% (234)	50.0% (24)	33.3% (210)
Maternal anxiety	18.3% (124)	31.3% (15)	17.3% (109)
Maternal depression and anxiety	11.5% (78)	22.9% (11)	10.6% (67)
Maternal abuse history	23.4% (158)	42.6% (20)	21.9% (138)
Stressful life events (mean \pm SD)	5.9 (3.9)	8.5 (4.6)	5.7 (3.8)
Social support(mean \pm SD)	9.0 (1.2)	9.4 (1.6)	8.9 (1.2)

Unadjusted analyses. The relationship between the independent variables and PTSD was assessed using binomial logistic regression. The crude (unadjusted) association between PTSD and predictor variables was first examined by entering the variables separately, with “No PTSD” as the reference category. The results of these unadjusted logistic regression analyses are presented in Table 8. When entered separately, all variables except IQ and substance use disorders significantly predicted PTSD group membership, when compared with young people without PTSD. The strongest predictors of PTSD group membership in the unadjusted analyses were female gender, and premorbid depression diagnoses, premorbid behavioural diagnoses, and the young person’s history of having been sexually or physically abused.

Adjusted Analyses. All within individual and environmental variables significant at the univariate (unadjusted) level were selected for inclusion in the multivariate model. IQ and substance use were not significant at the univariate level, and were removed from further analyses. All other 13 variables were entered simultaneously, with “No PTSD” as the reference category. The following variables were *no longer* statistically significant in predicting PTSD or depression in the full model: maternal depression, maternal anxiety, comorbid maternal depression and anxiety, social support, young person abuse history, and premorbid behavioural disorders. These non-significant variables were removed from the model, one at a time (beginning with the least significant), and the impact on the remaining variables was assessed. All non-significant variables were removed from the model without any significant change to the odds ratio’s (OR’s) (any changes to OR were less than 10%).

Table 8

Part A: Unadjusted predictors of PTSD (n =679).

Predictors	PTSD (n=48)	
	Unadjusted OR (95%CI)	<i>P</i> - value
Female gender	4.06 (2.0-8.3)	.000*
Premorbid depression	3.69 (1.8-7.7)	.000*
Premorbid anxiety	2.63 (1.3-5.4)	.008*
Premorbid substance use disorders	3.01 (0.6-14.3)	.167
Premorbid behavioural diagnosis	3.08 (1.2-7.8)	.019*
Internalising	1.05 (1.0 – 1.1)	.008*
Externalising	1.06 (1.0-1.1)	.002*
IQ	1.04 (1.0 – 1.1)	.202
Young person abuse history	3.27 (1.1-10.1)	.040*
Stressful life events	1.17 (1.1-1.2)	.000*
Social support	1.33 (1.1-1.6)	.006*
Maternal depression	2.01 (1.1-3.6)	.021*
Maternal anxiety	2.18 (1.1-4.1)	.018*
Maternal depression and anxiety	2.50 (1.2-5.1)	.012*
Maternal abuse history	2.64 (1.4-4.8)	.002*

a. From a series of binomial logistic regressions estimating 2 categories of outcome: PTSD and No PTSD (as reference).

b. OR, Odds ratio; CI, confidence interval.

c. $p < .05$

Although premorbid behavioural disorders and young person abuse history were two of the strongest predictors at the univariate level, at the multivariate level, removal of these two variables resulted in a change of less than 10% to the OR. Therefore, they were removed without making any significant change to the final model.

Results of adjusted binomial logistic regression analyses are presented in Table 9. Gender, premorbid anxiety diagnoses, stressful life events, and maternal abuse history remained in the final model and were significantly associated with PTSD. As is evident from Table 9 and the odds ratio's, female gender was the strongest predictor of PTSD in young people in the final model followed by maternal abuse history. Externalising approached significance level ($p = .056$), but as this was only borderline significant, externalising symptoms was removed from the final model. The overall model was found to be significant [$\chi^2(4) = 46.133, p = .000$]. Approximately 16.6% of the variance in the outcome groups was explained by the model (Nagelkerke R-Squared = .166) but as this is a logistic regression, this is an approximation of OLS R² superscript, and does not reflect the actual percent of the variance explained. Further analyses for interactions found no significant interactions between variables in the full model.

Table 9

Part A: Adjusted model of predictors of PTSD (n = 679).

Predictors	PTSD (n=48)	
	Adjusted OR (95%CI)	<i>P</i> - value
Female gender	3.75 (1.7-8.0)	.001*
Premorbid anxiety disorder	2.35 (1.1–5.1)	.031*
Stressful life events	1.13 (1.1-1.2)	.001*
Maternal abuse history	2.52 (1.3-4.8)	.004*

- a. Adjusted odds ratio's are presented.
- b. Premorbid depression, premorbid behavioural diagnosis, internalising, young person abuse history, social support, maternal depression, maternal anxiety, and maternal comorbid depression and anxiety variables were excluded from analysis as they were non-significant and removal resulted in a change of less than 10% to OR.
- c. Externalising was approaching significance at OR 1.04 (1.0-1.1), $p = .056$, but was removed from the final model.
- d. No PTSD as reference category.
- e. OR, Odds ratio; CI, confidence interval.
- f. * $p < .05$

Part B: PTSD compared with major depression

Descriptive characteristics of predictor variables are presented in Table 10. For the within-individual variables, chi-square analyses demonstrated that significantly more young people with PTSD were female [$\chi^2(1) = 7.074, p = .008$] and had premorbid diagnoses of depression [$\chi^2(1) = 8.982, p = .003$], and substance use disorders [$\chi^2(1) = 6.297, p = .012$], compared to young people with depression (and without PTSD). ANOVA and chi-square analyses demonstrated that there were no differences between groups in internalising and externalising behaviour, IQ, abuse history, and premorbid anxiety.

For the environmental variables, ANOVA demonstrated that young people with PTSD had significantly higher scores for stressful life events ($F(1,188) = 32.976, p = .000$) during the 12 months prior to diagnosis, than young people with depression. There was no significant difference between groups on social support. Chi-square analyses demonstrated that significantly more young people with PTSD had mothers who had experienced diagnoses of major depression [$\chi^2(1) = 5.536, p = .019$], anxiety [$\chi^2(1) = 6.717, p = .010$], and comorbid depression and anxiety [$\chi^2(1) = 5.644, p = .018$], compared to young people with depression. More young people with PTSD had mothers who had previously been abused, and this result was approaching significance [$\chi^2(1) = 3.744, p = .053$].

ANOVA demonstrated no significant difference between PTSD and major depression groups on mean age of onset of PTSD and depression (17.5 ± 1.7 and 17.8 ± 1.7 respectively). Therefore, significant differences in premorbid variables cannot be attributed to variations in age across diagnoses.

Table 10

Part B: Predictor variable characteristics across sample subgroups (PTSD and depression) (n = 190).

Predictors	Cohort (n = 190)	PTSD (n=48)	Depression (n=142)
Gender			
Male	36.8% (70)	20.8% (10)	42.3% (60)
Female	63.2% (120)	79.2% (38)	57.7% (82)
Premorbid depression	23.7% (45)	39.6% (19)	18.3% (26)
Premorbid anxiety	48.9% (93)	47.9% (23)	49.3% (70)
Premorbid substance use disorder	29.5% (56)	43.8% (21)	24.6% (35)
Internalising T-score	53.19 (8.11)	52.89 (9.15)	53.28 (7.77)
Externalising T-score	55.62 (8.28)	56.90 (7.71)	55.19 (8.45)
IQ composite scores	18.01 (4.90)	18.81 (4.82)	17.74 (4.91)
Young person abuse history	5.8% (11)	8.3% (4)	4.9% (7)
Stressful life events (mean \pm SD)	2.39 (2.96)	4.35 (3.7)	1.73 (2.3)
Social support (mean \pm SD)	9.25 (1.30)	9.44 (1.62)	9.19 (1.17)
Premorbid maternal depression	45.8% (87)	60.4% (29)	40.8% (58)
Premorbid maternal anxiety	54.7% (104)	70.8% (34)	49.3% (70)
Premorbid maternal depression and anxiety	35.8% (68)	70.8% (24)	31.0% (44)
Maternal abuse history	31.1% (59)	41.7% (20)	27.5% (39)

Unadjusted Analyses. The relationship between the independent variables and clinical diagnoses of PTSD and Depression was assessed using binomial logistic regression. The crude (unadjusted) association between PTSD and predictor variables was calculated by entering the variables separately, with “Depression” as the reference category. The results of unadjusted logistic regression analyses are presented in Table 11.

When entered separately, maternal depression, maternal anxiety, comorbid maternal depression and anxiety, stressful life events, gender, premorbid depression, and premorbid substance use disorder variables significantly predicted young person’s PTSD when compared with young people with depression. Odds ratios for significant associations ranged from 2.92 (95% CI = 1.4 – 6.0, $p = .003$) (strongest) for premorbid depression, to 1.36 (95% CI = 1.2 – 1.5, $p = .000$) (weakest) for stressful life events.

Adjusted Analyses. Results of adjusted binomial logistic regression analyses are presented in Table 12. The seven variables significant at the univariate level (gender, premorbid depression, premorbid substance use disorders, stressful life events, maternal depression, maternal anxiety, and maternal comorbid depression and anxiety) were selected to include in the multivariate model. The variables were entered simultaneously, with “Depression” as the reference category. Maternal premorbid depression, and maternal premorbid comorbid depression and anxiety were no longer significant in the full model and were removed from the model one at a time (beginning with the least significant) and the impact on the remaining variables was assessed. Neither of the non-significant variables could be removed without changing the odds ratio’s (OR) of other variables by 10%.

Table 11

Part B: Unadjusted predictors of PTSD compared with Major Depression (n = 190).

Predictors	PTSD (n=48)	
	Unadjusted OR (95%CI)	P value
Female Gender	2.78 (1.3-6.0)	.009*
Premorbid depression	2.92 (1.4-6.0)	.003*
Premorbid anxiety	0.95 (0.5-1.8)	.869
Premorbid substance use disorder	2.38 (1.2-4.7)	.013*
Internalising	0.99 (0.9 – 1.0)	.773
Externalising	1.02 (1.0-1.1)	.217
IQ	1.05 (1.0 – 1.1)	.193
Young person abuse history	1.74 (0.5-6.2)	.394
Stressful life events	1.36 (1.2-1.5)	.000*
Social support	1.15 (0.9-1.5)	.255
Maternal depression	2.21 (1.1-4.3)	.020*
Maternal anxiety	2.50 (1.2-5.0)	.011*
Maternal depression and anxiety	2.23 (1.1-4.3)	.019*
Maternal abuse history	1.96 (1.0-3.9)	.055

a. From a series of binomial logistic regressions estimating 2 categories of outcome:

PTSD and Major Depression (as reference).

b. OR, Odds ratio; CI, confidence interval.

c. * $p < .05$

Therefore, these variables remained in the full model but were considered confounding variables. The overall model was found to be significant [$\chi^2 (7) = 53.102, p = .000$]. Approximately 36% of the variance in the outcome groups was explained by the model (Nagelkerke R-Squared = .360). As this is a logistic regression, this is an approximation of OLS R² superscript, and does not reflect the actual percent of the variance explained. Female gender, premorbid depression, premorbid substance use disorder, stressful life events, and maternal premorbid anxiety were found to significantly predict PTSD when compared with depression in young people. As is evident from Table 12 and the odds ratio's, maternal anxiety was the strongest predictor of PTSD in young people when compared with depression, followed by gender, premorbid depression, premorbid substance use and finally stressful life events. Analyses found no significant interactions between variables in the full model.

Table 12

Part B: Adjusted model of predictors of PTSD compared with depression (n = 190).

Predictors	PTSD (n=48)	
	Adjusted OR	p
	(95%CI)	
Female gender	2.92 (1.2-7.2)	.021*
Premorbid major depression	2.86 (1.2-6.9)	.018*
Premorbid substance use diagnosis	2.32 (1.0-5.4)	.051*
Stressful life events	1.37 (1.2-1.6)	.000*
Maternal premorbid anxiety	3.33 (1.06–10.5)	.040*

- a. Adjusted odds ratio's are presented. In addition to the variables listed in the table, analyses are adjusted for relevant confounding variables: premorbid maternal depression, and premorbid maternal comorbid anxiety and depression as removal of these variables resulted in a change of more than 10% to OR
- b. Depression as reference category.
- c. OR, Odds ratio; CI, confidence interval.
- d. * p<0.05

Discussion

This study used a prospective longitudinal design to determine risk factors for the development of PTSD in 679 Australian young people aged 15 to 20 years old. The selection of potential risk factors was informed by two meta-analyses of risk factors for PTSD for adults (Brewin et al., 2000; Ozer et al., 2003) and another longitudinal study of risk factors for children (Koenen et al., 2007). This study tested the hypothesis that childhood factors prior to age 15 contribute to the development of PTSD in young people between the ages of 15 and 20 years old. A number of within individual and environmental childhood variables were examined. In Part A, risk factors were measured at or before the 15-year follow-up, prior to the development of PTSD. Young people with PTSD were compared to those with no PTSD to differentiate those that predicted PTSD when compared with all other young people with and without other psychological diagnoses to compare with a normal population control group. All young people with a history of PTSD prior to age 15 years were excluded from the original sample of 706 young people. Therefore, unlike many risk factors studies in the current literature, it can be confidently asserted that the risk factors were antecedent to onset of PTSD symptoms. Part B directly compared risk factors for major depression with risk factors for PTSD and had the important advantage of allowing for the inclusion of premorbid diagnoses and prior stressful events with closer proximity to the onset of these two disorders rather than being restricted to using a broadband 15 year age cut-off. In addition, major depression is also a common psychological sequelae to stress and trauma and is also commonly

comorbid with PTSD. Therefore, Part B aimed to differentiate the risk factors for PTSD from those with major depression without PTSD.

Findings indicated that both within individual and environmental factors play a role in the development of PTSD in this sample of young people. All within individual factors, except IQ and premorbid substance use, and all environmental factors in this study significantly predicted PTSD when analysed separately. Of the within individual potential predictors, gender, premorbid depression, premorbid anxiety, premorbid behavioural diagnosis, internalising, externalising and having an abuse history, were all significantly associated with increased risk of PTSD between the ages of 15 and 20 years old when analysed separately. When analysed separately, a number of within individual predictors were not only significant, but were strongly associated with the development of PTSD. Prior to age 15 years, young people with PTSD symptomatology were three and a half times more likely to have a history of major depression, three times more likely to have a behavioural disorder, and two times more likely to have a prior anxiety disorder, when compared with young people without PTSD symptomatology. Thus, previous psychopathology prior to age 15 placed young people at heightened risk for later PTSD. Both internalising and externalising were significantly associated with PTSD but the strength of prediction was weak.

When entered separately, a number of environmental variables also showed strong associations with PTSD. Young people with a childhood history of having experienced abuse were three times more likely to develop PTSD after age 15 years old suggesting that an earlier interpersonal trauma history places young people at increased risk for PTSD after age 15 years. Relapse from an earlier history of

childhood PTSD does not account for this finding, as all those with PTSD symptomatology prior to age 15 were excluded from this study. Furthermore, a delayed onset of PTSD symptoms does not account for this finding either, as the triggering event was collected at interview and none of the subjects reported early childhood abuse as the triggering trauma for the PTSD symptoms analysed in this study. Therefore, when analysed separately, a history of having experienced abuse in childhood was associated with the development of PTSD symptoms after age 15 years following a separate traumatic event. Similarly, when analysed separately, young people with a mother who had experienced physical or sexual abuse prior to the young person reaching age 15 years were two and a half times more likely to develop PTSD symptoms after age 15 years. Therefore, it would seem that an earlier personal or maternal trauma history in the form of interpersonal abuse, is inclined to contribute to a vulnerability for PTSD symptom onset for an alternative trauma among young people.

Contrary to hypotheses, a history of substance use did not significantly predict PTSD in young people when compared with young people without PTSD. This study hypothesised that the young sample and younger legal drinking age in Australia (age 18 years old compared with age 21 years in the US) would lead to a finding that alcohol and substance use disorders pose a risk for the development of PTSD amongst young people in the 15 to 20-year age bracket. This hypothesis was not supported by the findings in Part A. Therefore, results in Part A were consistent with most other previous research which supports the self-medication hypothesis and has concluded that PTSD is the primary disorder that temporally precedes alcohol dependence and

use of other substances (Jacobsen et al., 2001). There are a number of possible explanations for this finding, the first being that substance use disorders do not increase the risk of developing PTSD symptoms in young people. However, despite the previous international research, I remain reluctant to confidently draw this conclusion given the small number of young people in this sample ($n = 11$) with a substance use disorder prior to age 15 years. In this sample, most young people to receive a diagnosis of a substance use disorder (drugs or alcohol) did so after age 15 years, however, the cut-off age for premorbid diagnoses in Part A was 15 years old. In fact, a total of 44% of young people with PTSD symptoms had a premorbid substance use disorder (alcohol or drugs) however, using an age cut-off excluded most of these premorbid substance diagnoses. Study 1 (discussed in Chapter 2) also showed that compared to young people with no PTSD, those with PTSD symptoms and diagnoses had higher rates of substance use disorders (although alcohol misuse was high across all groups). Therefore, methodological challenges associated with temporal sequencing may account for the non-significant findings. What can be concluded from this study is that substance use disorders that manifested prior to age 15 years, did not significantly increase the risk for PTSD in young people. However, it remains unclear whether substance use disorders after age 15 and closer in proximity to PTSD onset pose a PTSD risk for Australian young people. Given the younger age of the sample, and that substance use disorders at this age is less common, a larger sample of Australian young people with substance use problems would be needed to temporally sequence these two highly comorbid disorders and draw stronger and more convincing conclusions.

Intelligence was the only other premorbid variable in this study found to be unrelated to PTSD in this sample of young people when analysed separately. A number of other studies (Brewin et al., 2000; Koenen et al., 2007; McNally & Shin, 1995) have found a small but reliable relationship with lower IQ and the development of PTSD. This study did not find a relationship between IQ and PTSD, and this finding may be due to methodological differences. In this study, results from only two subscales of the Wechsler Intelligence Scale for Children – Third Edition (WISC-III) were used in a combined and continuous variable. The use of the Vocabulary and Digit Span subscales as an indicator of IQ is questionable and the Fullscale IQ or a categorical variable, may have led to different results supportive of previous literature linking lower IQ with PTSD. Therefore, the absence of a relationship of PTSD with IQ in this study should be interpreted with caution.

The full model examines all significant predictors together, to determine those that predict PTSD symptoms taking into consideration all significant factors. Although most variables significantly predicted PTSD in young people when entered separately, in the full model only four variables (female gender, premorbid anxiety, stressful events, and maternal abuse history) remained independently predictive of PTSD symptoms when compared with young people without PTSD symptoms. All other predictor variables could be removed without significant changes to the odds ratio's, indicating that they did not confound the results. Consistent with previous research among adult populations, female gender was robustly associated with PTSD in young people, with females being 3.75 times more likely than males to develop PTSD symptoms. Unfortunately, there are currently no epidemiological studies of Australian

children reporting results for PTSD with which to compare this finding. Interestingly, early Australian adult epidemiological studies still frequently referenced in the literature did not find a gender difference between rates of PTSD amongst men and women (McLennan, 1997). The finding of this current study is more consistent with recent adult epidemiological studies which have found a significant gender difference (ABS, 2007), however, the strength of the finding in this study is higher than would have been expected. As discussed in Chapter 1, one of the most consistent findings in the epidemiology of PTSD is the higher risk of this disorder amongst women, and the findings of this current study supports this phenomenon for Australian young people. The exact mechanism for this increased risk of PTSD for females is currently unclear, but may be related to a combination of factors including greater exposure to sexual assault and interpersonal trauma which carries a higher probability for PTSD, a greater vulnerability to stress (Frans et al., 2005), stronger cognitive perceptions of threat and loss of control (Olf et al., 2007), and current diagnostic criteria which centres around subjective cognitive and emotional responses to traumatic events that may be more likely to be reported by females. Posttraumatic symptomatology amongst males may be characterised more by behavioural and substance use features and may be difficult to capture using current DSM-IV-TR diagnostic criteria.

It is not at all surprising that a premorbid anxiety diagnosis was found to increase the likelihood of developing PTSD after age 15 years. As may be expected, young people with PTSD symptoms after age 15 years were 2.35 times more likely to have a pre-existing anxiety sensitivity, as evidence by a premorbid anxiety disorder, when compared with young people without PTSD. Consistent with the American

research by Breslau and colleagues (2006), this current study found that childhood anxiety diagnoses increased the risk for later development of PTSD symptoms amongst young people. However, unfortunately, Breslau et al (2006) were unable to analyse premorbid childhood major depression. As discussed in Chapter 1, most PTSD risk factor studies examining premorbid anxiety and depression have often concluded that both anxiety and depression increase the risk for PTSD. However, these studies have often combined symptoms of both anxiety and depression into one predictor variable (Kleim et al., 2007; Ozer et al., 2003), have used cross-sectional or retrospective methodological designs (Breslau, Davis, Peterson et al., 1997; Bromet et al., 1998; Fullerton et al., 2000; Zatzick et al., 2007), or have used questionnaire measures to assess anxiety sensitivity or traits rather than diagnostic interview (Bryant & Panasetis, 2001; Lang et al., 2002; Lee et al., 1995; Lonigan et al., 1994; Shnurr et al., 1993; Storr et al., 2007) and have often administered these questionnaires after exposure to trauma (when findings may be biased by the effects of trauma exposure). An interesting finding of this current prospective longitudinal study using diagnostic interviews from multiple informants is that when analysed together with other predictors, unlike premorbid anxiety, a history of major depression did not significantly predict PTSD symptoms in young people. Therefore, when analysed together with other predictor variables, anxiety and not depression significantly increased the risk of PTSD amongst this Australian cohort of young people.

This study found that young people with a greater accumulation of premorbid stressful life events were significantly more likely to develop PTSD than those with fewer and/or less stressful life events. Overall, young people who develop PTSD after

age 15 years, have generally reported more stressful lives prior to age 15 years, supporting a dose of exposure theory or alternatively a 'kindling effect', whereby an accumulation of stress increases the risk for the development of PTSD symptomatology. Although this association was not strong (OR = 1.13) this finding is consistent with that of Lawyer et al (2006) who found that even after controlling for exposure related variables, the accumulation of life stressors 12-months prior to the September 11 terrorist attacks significantly predicted PTSD amongst New York City residents. Similarly, Currier and colleagues (2009) found that the accumulation of stressful life events (both related and unrelated to cancer treatment) predicted the intensity of trauma responses among children with cancer. Therefore, the accumulation of multiple stressors prior to trauma exposure would appear to combine or cumulate to lead to more severe psychological outcomes for young people.

As discussed by Monroe and Harness (2005), there is a current theoretical question as to whether people become desensitised, or alternatively, overly sensitised to stress over time (Monroe & Harkness, 2005). Research has found a robust and causal association between stressful life events and depression (Hammen, 2005) and the findings of this current study would be consistent with the 'kindling effect' or the 'stress sensitivity model' for depression. The kindling hypothesis was initially posed by Post and colleagues (Post, 1992; Post, Rubinow, & Ballenger, 1984) and a growing interest has followed in the hypothesis that recurrent episodes of mood disorders require less external stress as the person becomes more and more sensitised ('kindled') to the negative effects of stressful life events (Hammen, 2005). Neurobiological changes associated with repeated stressors and repeated episodes of mood disorder

diminish the association between stressful life events and repeated episodes of depression. Findings from this study suggest that this theory could potentially be extended to the development of PTSD. The association of PTSD symptoms with premorbid anxiety diagnoses and a premorbid accumulation of stressful life events would also suggest that young people may become more sensitive to stress following an accumulation of stressful life events, making them more vulnerable to the deleterious effects of trauma exposure.

The exact mechanism for this vulnerability is currently unknown, and consistent with current cognitive conceptual models of PTSD (Ehlers & Clark, 2000) it could be hypothesised that those who experience multiple stressors and/or trauma's prior to late adolescence may be at increased likelihood of processing the later traumatic event and/or it's aftermath in a in a way that produces a sense of current and ongoing threat. Perhaps due to the experiences of a accumulation of stress and trauma, they are less likely to view the trauma as a time-limited event and consequently are at increased risk of forming negative appraisals of the external environment (e.g. the world is an unsafe place, I attract disaster, bad things always happen to me) or, their internal world (e.g. lack of belief in themselves as a competent and capable person), and responding with dysfunctional coping strategies (e.g. avoidance) that have the paradoxical impact of enhancing PTSD symptoms. Alternatively, perhaps the explanation is more simple and given that these individuals have reported generally more stressful lives, they may simply have been more likely to experience a more severe traumatic event. Thus, an alternative explanation is that they are more likely to have been exposed to trauma when compared to those who have reported less stressful

lives. However, this study (in addition to other risk factor studies) would suggest that environmental factors combine with within individual factors leading to an individual vulnerability and that environmental factors alone are not sufficient in explaining the risk for PTSD. Further research is needed to understand the specific processes and skills deficits that may make some young people more vulnerable to stressful events, including exposure to trauma.

An interesting finding in this study is that the accumulation of stressful life events was a significant predictor of PTSD among young people in the full model and an earlier history of physical or sexual abuse (generally a PTSD qualifying event) was no longer significant. This would suggest that the accumulation of stressful events over a protracted period and in closer proximity to trauma exposure is a better predictor of PTSD than a history of having experienced the earlier trauma of abuse (although both are significant predictors when analysed separately). This finding has significant clinical relevance in helping to identify children and young people most at risk of developing PTSD following exposure to trauma, and indicates the necessity in assessing for previous stressful life events and previous anxiety diagnoses. Information about the accumulation of stressful events is also easier and less invasive to extract during interview and assessment than information about abuse experiences, and could be readily integrated into initial medical and psychological interviews to help identify those at increased risk for PTSD following trauma. Caution in interpreting this finding is however advised as it is possible that this finding reflects a recency effect, whereby young people have simply had more time to recover from the earlier trauma when compared with the more recent stressful experiences.

Another somewhat surprising finding was that maternal depression and anxiety were no longer associated with PTSD amongst offspring in the full model, although, a maternal history of trauma in the form of sexual or physical abuse remained significant. Young people with PTSD symptoms were two-and-a-half times more likely than those without PTSD symptoms to have mothers with a history of having experienced physical or sexual abuse such as childhood abuse or abusive intimate relationships prior to the young person turning age 15 years. Thus, prior maternal abuse history (with or without PTSD or depressive features) confers a significant risk for the development of PTSD among 15 to 20 year old offspring. In the full model, this risk could not be explained by maternal anxiety or depression diagnoses.

One possible explanation is that the young people were also exposed to abuse by the same perpetrator as their mother. However, the young person's own history of abuse was no longer significant in the full model, so the results do not support this explanation. Alternatively, perhaps the young person with PTSD either personally witnessed or was vicariously exposed to their mother's traumatic event or events by way of personal disclosure. Whilst this may have conferred a risk for the development of PTSD among offspring through indirect mechanisms such as modelling of cognitions, beliefs and behaviours that may increase the risk for anxiety disorders, it is unlikely that the mother's trauma directly contributed to PTSD among offspring as the abuse perpetrated to the mother was identified as the triggering event for only one of the 48 young people in this sample with PTSD symptoms. All other young people with PTSD symptoms after exposure to domestic violence had been excluded due to development of symptoms prior to 15 years of age.

The results of this study therefore offer support to the notion of an intergenerational transmission of trauma, a cumulative impact of trauma that is intergenerational. The concept of transgenerational trauma has been used to explain the intergenerational impact of earlier trauma for cultural groups such as Indigenous Australians (Atkinson, 2002; Ober et al., 2000), and Native and African Americans (Dass-Brailsford, 2007), and the patterns of traumatisation that are repeated from one generation to another (Frazier et al., 2009). Similarly, other researchers have found that a paternal history of war trauma has a deleterious effect on offspring independent of the role of parental psychopathology (Rosenheck & Fontana, 1998). The findings from this current study are consistent with this previous intergenerational research and recent arguments posed by transgenerational theorists, who purport that a maternal abuse history is not only a marker for exposure to trauma amongst offspring, but also contributes indirectly by contributing to emotional vulnerabilities amongst offspring rendering them at increased risk of psychopathology following traumatic exposure (Bert et al., 2009). The findings from this current study extend this theory of intergenerational trauma to Australian community dwelling young people.

The mechanism for this intergenerational impact is currently unclear. Perhaps the intergenerational risk is transmitted through a 'kindling effect', whereby offspring of mothers who have experienced the trauma of physical or sexual abuse are more sensitive to the deleterious effects of stress and trauma (i.e. are less resilient to the effects of trauma due to a pre-existing sense of hyper-arousal and a lack of safety) and are more likely themselves to develop PTSD symptoms after exposure to their own trauma. In addition, perhaps intergenerational stress can be partially explained by the

stress generation hypothesis (Hammen, 2005), whereby offspring of mother's who may be locked in highly stressful family and interpersonal environments with a high propensity for exposure to potentially traumatic events are themselves at increased risk of experiencing traumatic events due to a more stressful environment or behaviours influencing their environmental choices. This, combined with the individual's stress sensitivity as evidenced by premorbid anxiety, would seem to place young people in this sample at heightened risk for the development of PTSD symptoms. Future research aimed at exploring the mechanism by which significant predictors increase the risk for PTSD is needed.

The aim of Part B was to directly compare risk factors for the development of PTSD symptoms with the risk factors for the development of major depression in young people, in light of the fact that major depression is also a well-known psychological sequelae of trauma exposure. To date, it is unclear why some people develop PTSD after trauma whilst some others develop major depression. This study was interested in exploring differences in risk factors across the two diagnoses. Findings of the current study may be used to help determine young people at risk for PTSD and depression, and to help understand the differences between the two diagnoses. Findings of this study are not exact though. Most of the young people with PTSD had lifetime co-morbid diagnoses of major depression. The high comorbidity of these two disorders made it impossible to analyse those with PTSD only due to insufficient numbers. To explore the differences in risk factors with more accuracy, three groups would be needed including a group of young people with PTSD and no

depression, a group of young people with major depression and no PTSD, and a group of young people with comorbid PTSD and major depression.

When entered separately, a total of seven within individual and environmental variables predicted PTSD symptom when compared with depression. In the full model, a total of five variables remained significant. Female gender continued to predict PTSD symptoms when compared with depression and overall, those with PTSD were almost three times more likely to be female. Therefore, whilst we know that depression is also more common amongst females than males, this gender difference was stronger amongst those with PTSD symptoms in this sample. This finding suggests that a combination of factors may contribute to females in a community sample being more vulnerable to PTSD symptoms and that emotional vulnerability cannot account for this difference entirely.

Stressful life events in the 12-month period leading up to diagnosis also remained significant in the full model, consistent with the findings from Part A, indicating that those who develop PTSD experience a greater accumulation of stress in the period leading up to diagnosis. As previously discussed, this may be due to a more stressful environment, or to individual choices and decisions that contribute to an accumulation of stress over time. Whilst not surprising, this finding is nonetheless important in indicating that the 'dose of exposure' theory may in part explain why some people develop PTSD whilst others develop depression following stressful and traumatic events. However, this alone did not predict PTSD symptoms when compared with depression, and other within individual and environmental variables also remained significant in the full model, even when stressful events were taken into

consideration. It is important to note, that female gender and stressful life events consistently differentiated young people with PTSD symptoms from those without PTSD in Part A, and from those with major depression in Part B.

Other significant findings were quite different to the findings of Part A, which compared predictors for PTSD symptoms with no PTSD symptoms. The three significant predictor variables that uniquely predicted PTSD symptoms when compared with depression included premorbid major depression, premorbid substance use, and maternal premorbid anxiety. Surprisingly, young people with PTSD symptoms were 2.86 times more likely to have a premorbid depression diagnosis than young people with major depression. Thus, pre-existing depression is more likely to predict PTSD symptoms than it is to predict depression amongst young people in this sample shedding light on the temporal sequencing of these two highly comorbid diagnoses and suggesting that a history of depression plays a significant role in the development of PTSD in young people and is not solely a consequence of trauma and distressing PTSD symptoms. It is interesting that premorbid depression did not differentiate PTSD symptoms from No PTSD in Part A. This may be due to the differing methodologies in the two studies. Part B allowed for the inclusion of diagnoses after age 15 years but prior to onset of PTSD or depression, therefore including diagnoses with closer proximity than Part A could allow.

As noted above, premorbid substance use significantly differentiated between young people with PTSD symptoms and young people with depression. Given that Part B, unlike Part A, allowed for thorough exploration of temporal sequencing of all substance use diagnoses leading up to the time of onset of disorder, the findings whilst

only borderline in significance ($p = .051$), suggest that pre-existing substance use disorders may indeed play an important role in the development of PTSD symptoms amongst young Australians. The odds ratio ($OR = 2.32$) would indicate that those with PTSD symptoms are over twice two times more likely to have a premorbid substance use diagnosis than young people with depression. The finding however, is not nearly as strong as was hypothesised.

The other variable that significantly predicted PTSD symptom when compared with depression was maternal premorbid anxiety. It is interesting that mother's anxiety and not the young person's own anxiety (as in Part A) predicted PTSD symptoms when compared with young people with depression. This indicates that once again, the mother's history is significantly associated with the development of PTSD amongst offspring and should be investigated during a thorough psychological assessment. This study does not examine the exact mechanism for this intergenerational transmission of risk, but it could be hypothesised that anxious tendencies may be both biologically inherited and also behaviourally modelled, leaving offspring at increased risk for PTSD through biological and emotional and social learning mechanisms. In addition, mothers with a history of anxiety disorder may react with a heightened level of fear and distress following offspring exposure to trauma, interacting with the young person's own distress to escalate and maintain rather than sooth symptoms of hyperarousal amongst young people. Maternal anxiety did not differentiate PTSD symptoms from no PTSD in Part A possibly due to the inclusion of young people with other anxiety disorders in the no PTSD category. Part B focussed more specifically on PTSD symptoms compared with depression rather than the diverse group of young

people analysed in the part A control group. This study has significant strengths and limitations which will be discussed in Chapter 4.

In summary, findings from this study support the growing view that PTSD has developmental origins and has demonstrated that the developmental conditions of childhood increase the risk of developing PTSD symptoms. In part A, all predictor variables except IQ and premorbid substance use differentiated young people with PTSD symptoms from those with no PTSD when analysed separately. When analysed together, in the full model, the following four variables predicted PTSD symptoms compared with young people with no PTSD: female gender; premorbid anxiety disorder; stressful life events; and maternal abuse history. In part B, seven variables differentiated young people with PTSD symptoms from young people with depression when analysed separately. Those variables were: female gender; premorbid depression; premorbid substance use disorder; stressful life events; maternal depression; maternal anxiety; and maternal comorbid depression and anxiety. When these seven variables were analysed together in the full model, five variables significantly predicted PTSD symptoms in young people when compared with major depression. The significant variables in the full model were: female gender; premorbid major depression; premorbid substance use diagnosis (borderline result); maternal premorbid anxiety; and, stressful life events. Findings indicate that more attention should be given to developmental conditions, including premorbid diagnoses, the accumulation of stressful life events, and the impact of intergenerational trauma and anxiety, particularly those experienced by mothers who are often the primary emotional caregivers. This study suggests that the theories used to explain the increased

sensitivity to depression through a 'kindling effect' may also apply to anxiety and PTSD whereby individuals become increasingly sensitised to the deleterious effects of stress and trauma. Furthermore, this study suggests that this kindling effect may be intergenerational, whereby mother's exposure to trauma may negatively impact on young people by either increasing the likelihood of exposure to trauma (above and beyond early child abuse) or increasing the vulnerability for PTSD following exposure to trauma. Additional research may lead to a more thorough understanding of the exact mechanism of this intergenerational transmission of risk and better prevention of trauma exposure and PTSD onset among young people.

CHAPTER 4

Discussion

Summary

In Australia, an overwhelming one in five young people experience mental health problems, and this prevalence appears to be on the rise. Highlighting the importance of adolescence as a particularly critical developmental period, it is now known that most adults with psychological disorders begin experiencing symptoms prior to 24 years of age. Unfortunately, despite the often devastating sequelae of PTSD and current research indicating that PTSD is the most common anxiety disorder amongst Australians aged 16 to 85 years old, relatively little is known about PTSD in young people. An understanding of PTSD in children and adolescents is important for prevention, detection and treatment to alleviate distressing symptoms, improve later adult functioning, and prevent negative intergenerational effects of psychological dysfunction. To date, research examining PTSD amongst young Australians has been relatively neglected and is of utmost importance for the wellbeing of our valuable young Australians.

This thesis consisted of two studies. Study 1 aimed to enhance our awareness and understanding of PTSD amongst Australian young people. This study also aimed to examine the relevance of early maternal depressive symptoms on PTSD symptoms amongst offspring. The frequency, duration, precipitating events and psychological comorbidity of PTSD in a large longitudinal Australian community sample of 706

children (followed through childhood to age 20-years) was examined to help fill the considerable gap in the literature examining the topography of PTSD in young Australians. In summary, this study demonstrated that PTSD was not uncommon among Australian children and young people with 6.4% of this large young sample meeting full DSM-IV-TR diagnostic criteria, and a further 4.2% with significant subthreshold PTSD symptoms. This finding alone is further evidence for the need for more awareness, research, and treatment facilities targeting PTSD among non-adult Australian populations. PTSD was also found to be significantly more common amongst females and results suggested a poorer prognosis for females generally. Overall, females were: more likely to report sexual assault as the triggering event; to experience chronic symptoms exceeding six months in duration; and to experience relapse. Most children and young people with PTSD in this study experienced chronic symptoms exceeding six months in duration, raising questions about the usefulness of the DSM-IV-TR definition of chronicity for PTSD.

Although previous authors have argued that PTSD in children is rare (Ford, Goodman, & Melzer, 2003; Koenen et al., 2007), almost 10% of those with PTSD in this sample had an onset prior to 10 years of age, demonstrating that young children are also afflicted. The prevalence of PTSD symptoms was found to increase with age and about 40% of children with PTSD symptoms developed symptoms before age 15 years. The mean age of onset in this sample was 15 years and four months. Once established, it would seem that PTSD among children and young people is often chronic, and is highly comorbid with major depression, other anxiety disorders, substance use disorders, behavioural disorders, and suicide attempts. The lifetime

comorbidity of PTSD symptoms with other anxiety disorders appeared to be associated with the development of more severe clinical features of PTSD among children and young people, and discriminated between whether symptoms were clinical or subclinical.

A disturbing finding was that almost one in five young people with PTSD had attempted suicide at least once, and an additional 19% had contemplated suicide but had not attempted. Study 1 found that amongst young people, a lifetime comorbidity of depression and PTSD increased the risk of suicide attempt compared with PTSD or depression alone. These findings underscore the enormous turmoil experienced by young people with PTSD. The findings of this study would indicate that PTSD is a serious, chronic and debilitating disorder that has a considerable negative impact and may result in a new, unfavourable developmental path for children and young people.

This study was also interested in the impact of maternal depression on child outcome. The study cohort was oversampled for mothers' depression based upon questionnaire results on the DSSI administered at the first antenatal visit and at an additional three points after birth and between the child's age 5 years. Although the sample is over represented for maternal depression, mother's depression scores on the DSSI prior to the child's age 5 was not significantly associated with PTSD symptoms in this child cohort. Study 2 used a different methodology for assessing maternal depression diagnoses, including clinical depressive episodes after the child's age 5. Clinical ratings of maternal major depressive episodes were based upon the Structured Clinical Interview for the DSM-IV (SCID-I). Whilst premorbid maternal depression was associated with child PTSD in Part A and Part B when analysed separately,

mother's depression did not predict PTSD symptoms in either full model when all other significant variables were considered. Given that maternal depression was not associated with child PTSD in Study 1 or 2, it is concluded that the results of this research may be generalisable to a normal sample.

The ability to provide early treatment and effective intervention depends on identifying individuals who are most at risk as soon as possible. Study 2 seeks to address current gaps in the PTSD literature by using a prospective longitudinal design to examine a large set of 15 potential within individual and environmental risk factors for the development of PTSD in community dwelling Australian young people. The study was divided into two parts. It was hypothesised that a range of potential risk factors would differentiate young people with PTSD symptoms from those with no PTSD in Part A and from young people with major depression in Part B. The following within individual potential predictors were examined: female gender; presence of premorbid major depression, anxiety disorders, substance use disorders, and behavioural disorders; higher internalising symptoms; higher externalising symptoms; and lower intelligence. The following environmental potential predictors were examined: the accumulation of stressful life events; a history of having been physically or sexually abused; poor social support and social integration; the presence of mother's major depression, anxiety, or comorbid depression and anxiety; and the mother's history of having been physically or sexually abused. Results of Study 2 indicated that both within individual and environmental factors played a role in the development of PTSD symptoms in this sample of Australian young people. Findings would suggest that it is possible to determine those most at risk of developing PTSD

symptoms. In the full model, four variables remained independently predictive of PTSD symptoms in young people within the 15 to 20 year age bracket when compared with young people with no PTSD. Those significant variables were female gender, a premorbid anxiety diagnosis, the accumulation of stressful life events prior to age 15 years, and having a mother with a history of having been physically or sexually abused. As would be expected, young people with PTSD symptoms were 2.35 times more likely to have a pre-existing anxiety condition when compared with young people without PTSD. Most previous research has combined symptoms of anxiety and depression into one predictor variable rather than analysing the two disorders separately. A unique finding of this study is that pre-existing anxiety disorders (prior to age 15 years) and not depression (prior to age 15 years) significantly increased the risk of PTSD symptoms amongst this Australian cohort of young people, when compared with all other young people without PTSD.

It was surprising to find that maternal abuse history remained significant in the final model, and that this could not be explained by maternal diagnoses of anxiety or depression, or the young person's direct exposure to abuse or domestic violence. The results of this study offer support to the notion of transgenerational trauma suggesting that maternal abuse history may be a marker for exposure to trauma amongst offspring, and indirectly contribute to emotional vulnerabilities among offspring rendering them at increased risk for PTSD symptoms following trauma.

Female gender was also robustly associated with PTSD symptoms when compared with young people with major depression. Although it is well known that female gender increases the risk for diagnoses of major depression, female gender

remained significantly predictive of PTSD symptoms amongst young people when compared with those with depression. This would suggest that female gender plays an even greater role in the development of PTSD symptoms than with the development of depression in young people. Similarly, the accumulation of stressful life events prior to diagnostic onset also differentiated PTSD symptoms from depression in young people. Thus, findings from both Part A and Part B suggest that young people may become more sensitive to stress following an accumulation of stressful life events, making them more vulnerable to the deleterious effects of trauma exposure and increasing the risk for the development of PTSD symptoms. An interesting finding of both Part A and Part B, is that whilst the accumulation of stressful life events significantly predicted PTSD symptoms among young people in the full models, an earlier history of physical or sexual abuse was no longer significant. This finding has significant clinical relevance, emphasising the importance of gathering information about the accumulation of stressful events (rather than more intrusive questioning about abuse history) at initial medical and psychological interviews to help identify those most at risk for PTSD symptoms following trauma.

An additional three variables differentiated young people with PTSD symptoms from young people with depression in the full model. These variables were different to those that were significant in Part A and were: premorbid depression, premorbid substance use disorders and having a mother with premorbid anxiety. Somewhat contradictory to Part A, Part B found that young person premorbid depression and not premorbid anxiety significantly predicted PTSD symptoms when compared with young people with depression. Thus depression may play a significant

role in the development of PTSD after age 15 years and the high comorbidity of these two disorders is not solely a consequence of trauma and distressing PTSD symptoms. It would seem that although early depression (prior to age 15 years) was not a risk factor for PTSD in young people when compared with those without PTSD, depression closer in proximity to the PTSD onset (including depression after age 15 years) was significantly associated with the development of PTSD symptoms when compared with young people with depression. This differing result would suggest that either depression closer in proximity to PTSD onset confers a significant risk for PTSD symptoms, or, that premorbid depression differentiates PTSD symptoms in young people from depression but not from all other young people without PTSD. Similarly, premorbid substance abuse was also significantly (borderline significance) associated with PTSD symptoms when compared with young people with depression. Given that so few young people had substance use diagnoses prior to age 15 years, it would seem that this differing result may be due to the inclusion of diagnoses closer in proximity to onset of PTSD symptoms in Study B. Finally, maternal anxiety also differentiated PTSD symptoms from depression in young people, once again underscoring the important role of mothers' psychological functioning.

In summary, interventions aimed at preventing exposure to trauma and an early response aimed at targeting these individuals in the aftermath of trauma exposure, may help prevent the chronic impairment often associated with PTSD. Findings from this study help to identify those individuals most at risk for PTSD symptoms. Furthermore, these results would also suggest that a thorough family history should be gathered at

assessment and parental inclusion in treatment should be seriously considered, even for adolescents.

Strengths and Limitations

This research has some significant and unique strengths. First and foremost this study is the first Australian prospective longitudinal study of risk factors for PTSD amongst a non-clinical population of young people. Prospective longitudinal studies that follow children throughout their lifespan are the best source of information about pre-existing factors that contribute to risk or resilience to trauma and psychological problems. They are the gold standard in psychological research and are the envy of many researchers, but are expensive, time consuming, difficult to execute, and are highly labour intensive. Most previous research examining risk factors for PTSD is limited by a number of methodological problems, not the least of which being retrospective methodological designs that may confuse cause and effect. Without a doubt, a major strength of this thesis is the prospective longitudinal design. All risk factors were measured prior to onset of PTSD and data was meticulously sorted to analyse young person and mother premorbid diagnoses with as much accuracy and certainty as possible. All young people with diagnoses of PTSD prior to age 15 were removed from analyses to allow for the clean examination of genuine prospective analyses.

Another significant strength of this study is that it utilises an Australian sample of children and young people. American risk factor studies dominate the literature and there is a glaring lack of Australian studies. This research adds significantly to the

current literature by utilising a large, young Australian birth cohort. Much PTSD research with children and young people is limited by small sample sizes but this research utilised a large sample of 706 Australian young people. Furthermore, there is often a generalisation of results from PTSD studies of adult samples to young people. This study specifically examined PTSD in children and young people to provide a more accurate and thorough understanding of the prevalence, course, chronicity, psychological comorbidity, and risk factors of PTSD amongst community dwelling young Australians.

In addition, diagnoses were formed from thorough diagnostic interviews rather than questionnaire results, and utilised multiple informants (including mothers, fathers and teachers), enhancing the validity of the findings. The collection of thorough and clear lifetime diagnoses allowed for the separate examination of depression, anxiety, behavioural disorders and substance use disorders as risk factors for PTSD, unlike other studies that have combined depression and anxiety symptoms. Maternal diagnoses were obtained from thorough diagnostic interviews with the mothers, rather than utilising those with PTSD as primary informants for familial diagnoses. Overall, this study has a large sample size, good design, strong methodology and has drawn conclusions based upon rigorous statistical analyses.

Whilst this research provides important evidence for the understanding of PTSD amongst children and young people and the illumination of risk factors, several limitations require mention. Firstly, the development of PTSD is confounded with exposure to a traumatic event. Some young people are exposed to trauma and others are not, but only those exposed can develop PTSD symptoms. However, factors that

lead to trauma exposure may be different to factors that increase the risk of developing PTSD symptoms after exposure. Some studies have aimed to identify factors that contribute to risk at both points to help inform interventions that prevent trauma exposure and those that may prevent the development of PTSD after exposure. Unfortunately, this study did not reliably document trauma exposure among all participants, including those without PTSD diagnoses, and therefore could not separate the degree to which the factors predicted trauma exposure compared with risk of PTSD symptoms after exposure. Therefore, the factors reported herein to predict PTSD, may also be predictors of trauma exposure. Further Australian research that reliably untangles the risk factors for trauma exposure and the risk factors for PTSD onset after trauma exposure amongst young people is required.

This study also utilised a birth cohort that was over-sampled for mothers' depression symptoms as self-reported in questionnaire prior to the young person reaching five years of age. Whilst these questionnaire results were not statistically associated with PTSD symptoms in this sample, it should still be recognised that the sample is skewed for maternal depression and may not be truly representative of a community sample. In light of this, caution is advised when applying these results to a general population.

Another potential limitation is that PTSD and subclinical PTSD symptoms were merged into the one outcome variable in Study 2. Given that PTSD occurs in four to eight percent of the normal population, an extremely large sample size would be required to run statistical analyses to longitudinally assess PTSD risk factors. Therefore, in this study clinical and subclinical symptoms needed to be combined. This

practice is not uncommon in the PTSD literature, particularly in light of the theoretical arguments surrounding the rigidity of the current PTSD diagnostic criteria, the limited suitability to child populations, and the fact that PTSD is significantly impairing even when it presents at a subclinical level.

This research used a young sample that was followed longitudinally from birth to age 20 years. Ideally, this research would have examined PTSD across the entire lifespan without being limited by an age cut-off. This 20 year cut-off has significant relevance to the chronicity data reported in Study 1, where more than one third of young people with PTSD had continuing symptoms at the time of the 20-year interviews. Therefore, although Study 1 reported a mean duration of PTSD symptoms of 1 year and 11 months, these statistics underestimate the chronicity of PTSD symptoms among young people. Only ongoing longitudinal follow-up throughout the lifespan can accurately shed light upon the true chronicity of this disorder.

Intellectual disadvantage has been found to be a small but reliable predictor of PTSD in adult populations. Unfortunately, the measure of intelligence used in this research was derived from only the Vocabulary and Digit Span subtests of the Wechsler Intelligence Scale for Children, Third Edition (WISC-III) (Wechsler, 1991). The rationale behind selecting these two subtests is unclear, but costs and lengthy assessment prohibited the gathering of Full Scale IQ scores in this study. Although this study found no significant relationship between intelligence and PTSD, this finding must be interpreted with caution given the limited measures used.

Whether treatment was received by the young person during the course of the study was not reliably assessed and therefore, the impact on findings is unknown. It

would be expected that effective treatment would impact positively on the course, duration and comorbidity of PTSD in children and young people. However, it is impossible to know what percentage of young people accessed treatment, the type and timing of treatment interventions, and exactly what impact this had on the results. It is even more difficult to speculate on the possible impact on risk factor findings in Study 2. Future research examining the influence of treatment received by parents and young people, and the association with specific risk factors would be a useful addition to PTSD risk factor research.

Areas for Future Research

There are many areas for further research in the field of PTSD amongst younger samples. Firstly, much of the current research examining premorbid factors in the development of PTSD (including this current research) remains pathology driven. Therefore, further research is required to examine resilience and protective factors in young people to help develop strategies for fostering well-being to not only prevent deleterious outcomes, but also help our next generation of young people to meet their full potential and thrive, even after facing trauma and adversity.

This study focused primarily on the years between birth and 20 years of age. Conclusions could only be drawn upon those early years. Further research is needed to follow these young people throughout adulthood to fully understand the long term implications and sequelae of PTSD symptoms throughout the lifespan. It is also likely that risk factors for PTSD symptoms differ across different age brackets. A thorough

examination of the risks across different developmental periods would be useful to our understanding of those most at risk at different ages of trauma exposure.

A number of maternal variables were examined as potential environmental predictors of PTSD among young people. In both Part A and Part B maternal factors were found to be significant predictors of PTSD symptoms among young people. This then begs the question of the role that paternal premorbid diagnoses and abuse history have on the development of PTSD amongst offspring. Future research including paternal data is needed given that this current research demonstrates the important role that parents play in the development of PTSD, even among older adolescents. In addition, further research into the role of pre-existing parental and attachment relationships within families and the development of PTSD amongst child offspring is an important area for future research.

This research drew some inconclusive results on the role of premorbid substance use disorders in the development of PTSD symptoms amongst young Australians. A history of substance use disorder was not significantly associated with PTSD symptoms in the full model when compared with young people with no PTSD. This could be partly explained by methodological problems in using an age cut-off of 15 years when comparing across groups of young people with PTSD symptoms and those without PTSD symptoms, as most young people with substance use disorders developed problems with substances after age 15 years. Part B in study 2 used a control group of young people with Depression and therefore allowed for examination of substance use disorders after age 15 years and in closer proximity to onset of diagnoses. In this study, premorbid substance use was found to be borderline in

significance as a risk factor for PTSD symptoms. Future research is needed to unravel the role of alcohol and substance use disorders as risk factors for PTSD amongst young Australians when compared with young people without PTSD, rather than just compared with those with an alternative psychological disorder. This research would require either a larger sample size than this current study or a matched control group allowing for inclusion of diagnoses past age 15 years and closer in proximity to PTSD onset. Given the alarmingly high rates of alcohol and substance use disorders in this Australian sample and the Australian legal drinking age of 18 years old, a more accurate assessment of alcohol and substance use as a risk for trauma exposure and PTSD onset among young people is certainly needed before it can be confidently concluded that the self medication hypothesis explains the high comorbidity of these disorders. Given the cultural differences, it can be considered inadequate and neglectful to generalise American findings to the young Australian population.

Similarly, young person premorbid depression and maternal premorbid anxiety did not differentiate between young people with PTSD and those without PTSD, but did significantly differentiate between PTSD and depression in young people. Further research is required to determine whether this difference is due to the inclusion of disorders with closer proximity to PTSD onset, or whether this difference is specific to PTSD and depression diagnoses.

Whilst this research identified a number of within individual and environmental risk factors for PTSD symptoms, this research can only hypothesise mechanisms by which these factors affect children and young people. Further research is needed to understand the specific processes and skills deficits or strengths that may make some

young people more vulnerable or more resilient to trauma and PTSD. This study has some very interesting and surprising findings, such as the significance of the mother's abuse history independent of the child's own history of abuse. The findings of this research set the scene for further research into the mechanisms by which these risks confer to children and young people.

Concluding Remarks

Although PTSD is one of the most common psychological disorders following trauma, relatively little is known about the prevalence, nature, course, and risk factors for PTSD among children and young people. Given that most existing research has examined adults and our American counterparts, this thesis aimed to improve our knowledge and understanding of the epidemiology and risk factors of PTSD in an Australian sample of community dwelling young people. An understanding of the typical presentation of PTSD and identifying those individuals most at risk in this younger age group can help guide targeted prevention and treatment efforts and circumvent the entrenchment of severe disorder, and the onset of secondary comorbid disorders, leading to better outcomes and quality of life for children and young people. With this knowledge, clinicians may be alert to early detection, prevention, and treatment of PTSD.

This study highlights the impairing and chronic nature of PTSD in children and young people. However, in Australia there is a tremendous need for services for young people with non-psychotic disorders whose symptoms may tend to be less clinically severe, undetected as trauma responses, or mislabelled as behavioural. The limitations

of existing service models, particularly for children who have experienced abuse or trauma, needs to be addressed. Existing child and youth mental health services, be they within schools or the broader community are often overstretched and under resourced, resulting in young people accessing treatment belatedly and often in the context of extreme crisis (including in the event of self harm or suicide attempts). Furthermore, community mental health services are usually divided into paediatric and adult. This division of services that occurs with a cut-off at age 18 years potentially disrupts treatment consistency for young people, who struggle to have their needs met in either the paediatric or adult sector. Therefore, at the pivotal time when specialist mental health services are most needed to circumvent a chronic and negative life trajectory, they are often inaccessible or unsuitable in design and culture to young people. Studies such as this will help to demonstrate the need for additional therapeutic resources and preventative strategies that are targeted and aimed at minimising the harm of trauma.

Findings from this research indicate that PTSD is a serious, chronic and debilitating disorder that is more common amongst young people than many researchers have previously suggested and is yet to be truly understood and appreciated by researchers and clinicians alike. Current awareness, prevention, detection and treatment efforts for PTSD in young Australians are haphazard, untargeted and grossly inadequate. This situation is especially unacceptable given the high rates of suicide attempts found in this study. It is anticipated that the results of this research can add substantially to our knowledge and understanding of the impact of this disorder amongst our valuable Australian children and young people. Effective service planning depends on a fuller understanding of the epidemiology and risks if

interventions are to successfully relieve the distress associated with PTSD, improve the lifelong trajectory of those young Australians afflicted by this severe disorder and prevent the intergenerational transmission of psychological dysfunction and disadvantage.

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Appendix A

PERMISSION TO ACCESS THE PROJECT DATA

Page 1 of 1

naomi beutel

From: "Hammen, Constance" <hammen@psych.ucla.edu>
To: "naomi beutel" <naomibeutel@aapt.net.au>
Sent: Friday, 7 August 2009 1:09 AM
Subject: RE: Thesis

Hi Naomi,

This message confirms that Patricia Brennan and I, principal investigators of the Age 15 and Age 20 follow-ups of the sample, gave you permission to analyze data for your doctoral thesis. Your project fell within the scope of the original aims of the study (predictors of youth outcomes) and was covered in the individual participant consent forms. The original projects were approved by the ethics and institutional review boards of all participating facilities, including the University of Queensland, Emory University, UCLA, and Mater Hospital. We are very grateful for your important contributions to the age 20 project as an interviewer, and your thesis is also an important scientific contribution to our efforts. We wish you all the best as you finalize your thesis.

Connie Hammen

Constance Hammen, Ph.D.
Distinguished Professor, Departments of Psychology
and Psychiatry and Biobehavioral Sciences

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From: naomi beutel [mailto:naomibeutel@aapt.net.au]
Sent: Tuesday, August 04, 2009 12:16 AM
To: Hammen, Constance
Subject: Thesis

Dear Connie,

It has been some time since you have last heard from me! I hope that everything is well for you. I am finally completing the Phd of Clinical Psychology (well at least hope to) at Griffith University and as previously discussed, I am using the MUSP 15-year and 20-year data as agreed. Please find attached a copy of my write-up, as it presently stands. I would appreciate any feedback that you have, but please remember that your brain is bigger than mine! ha ha

I have also misplaced any written permission that I have to use this data. Can you please confirm in writing that I do have permission to use this data set?

Kind regards,

Naomi Beutel
Interviewer at the MUSP 20-Year follow-up.

25/08/2009

Appendix B

GRIFFITH UNIVERSITY RESEARCH ETHICS SCOPE CHECKER REPORT

 **Griffith** UNIVERSITY

Griffith University Animal Ethics Committee / Griffith University Human Research Ethics Committee

Project Title
Predictors of Posttraumatic Stress Disorder

Applicant
Naomi Beutel

Completed the Griffith University Research Ethics Scope Checker on 03 August 2009. In completing the checker they indicated:

- 1. About or involving humans? Yes**
- 2. Archival research. Yes**
2a) Personal information. No
- 3. Administrative or service delivery. No**
- 4. Quality assurance or audit. No**
- 5. Exercise or test for teaching purposes No**
- 6. Routine experiment or procedure for teaching purposes. No**
- 7. Work/ data collection by student only for teaching/ learning. No**

On this basis the described activity is outside the scope of the University's animal ethics and human research ethics arrangements, and as such does not require University ethical review.

This is a service maintained by the Office of Research on behalf of AEC and HREC.

Appendix C

INFORMED CONSENT FORMS

MATER AND UNIVERSITY STUDY OF PREGNANCY

AGE 20 FOLLOW-UP (M20)



ID _____

THE UNIVERSITY OF QUEENSLAND
 C/- School of Social Sciences
 Brisbane Qld 4072 Australia
 Inquiries: Principal Research Officer – Robyne Le Brocq
 Telephone (07) 3365 4554
 Mobile 0413 554 261

INFORMED CONSENT FORM: YOUTH

Introduction: You are being asked to volunteer for a follow-up of the Study of Pregnancy by the Mater Mother's -University Research Unit. The current phase of this research project is taking place under the joint cooperation of the University of Queensland, University of California, Los Angeles (UCLA), and Emory University, Atlanta, Georgia, USA.

This project continues to study the health and general well-being of mothers and their children with the aim of improving the quality of care received from medical and social agencies. The current follow-up will examine the health, development, and behavior of the youth in this project. A primary focus of this follow-up is the young adults' psychological well-being and social relationships, including specific questions about stressful events, moods and emotional functioning. We are interested in how youth and parents' emotional experiences and adjustment during children's early years relate to these adulthood outcomes in the children. Approximately 700 youth will be asked to participate in this phase of the study.

Procedure: This research involves a personal interview with you which will take approximately three hours for completion. The interview may be audiotaped, and you will have the right to review, edit or erase in whole or in part, your recording. The interview questions will be very similar to those that you answered during the last follow-up, when you were 15 years old. They include questionnaires and interviews about your health, mood, behavior, stresses and recent life events, and relationships with friends, family, and romantic partners, as well as work and school history, pregnancies, previous psychiatric treatment and use of social services and welfare agencies, exposure to violence in relationships, criminal activities, and drug use. We will also be contacting your mother and asking her to talk about your current behavior and relationship functioning. Finally, we will ask for your permission to contact one of your peers (or if you are in a romantic relationship, your partner) and ask him or her to complete questionnaires about your relationship and current behavior.

Risks: This study involves minimal risk. The interview will include questions about aspects of your behavior and health status. Some of these questions might be embarrassing or seem personal to you, and discussing personal information may be upsetting or distressing.

Benefits: You might find it helpful to discuss these matters with a trained and interested interviewer. If your responses to the interview indicate a need for treatment services, we will provide you with an appropriate referral list. You may not benefit personally from this study, but the knowledge that will be gained may benefit others.

Confidentiality: All information concerning you will be kept private. No one who knows your identity will see your answers except the person who conducts the interview. We will not inform your mother or your friend of your responses, and you will not be informed of their responses to interview questions. The data for this study will be used by researchers at the University of Queensland, UCLA, and Emory University.

PRINCIPAL INVESTIGATORS

Prof. Patty Brennan	Emory University, Atlanta Georgia, USA	Dr Michael O'Callaghan	Mater Misericordiae Hospital, South Brisbane
Prof. Connie Hammen	UCLA, California, USA	Dr William Bor	Mater Misericordiae Hospital, South Brisbane
Prof. Jake Najman	The University of Queensland, Brisbane	Prof. Gail Williams	The University of Queensland, Brisbane
Ms Margaret Anderson	The University of Queensland, Brisbane		

All data will be identified with a coded ID number, not by names. The code linking names and numbers will be kept in a locked filing cabinet in Brisbane. The audiotapes will be used for coding purposes only, and will be deleted upon the completion and data analyses of the study. When we present the results of this study in a journal or meeting, we will write in such a way that you cannot be recognized.

The only exceptions to the rule of confidentiality are cases in which: (1) the interviewer finds that a child has been hurt or harmed, or (2) you are at risk of harming yourself or others. In these instances, this information must be reported to our project's psychiatric consultant as well as the appropriate social service agency. Australian confidentiality and privacy laws do not provide protection in cases where researchers are subpoenaed to release their data in relation to criminal investigations. We can assure you, however, that in over 20 years of experience with many studies in Australia and the United States, the research investigators on this project have never received such a subpoena and have never been asked to turn their data over to the police or court personnel.

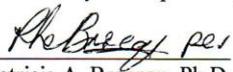
Compensation and Costs: You will be provided with monetary compensation for your time and efforts of participation in the amount of \$50.

Voluntary Participation: You are free to choose whether or not to complete your interview, answer particular questions, or withdraw from the study without loss of any benefits of participation.

Contacts: This study has been cleared by one of the ethics committees of the University of Queensland in accordance with the National Health and Medical Research Council's guidelines. The study has also been cleared by the ethics committee of the Mater Hospital. The interviewer will be happy to answer any questions that you might have about taking part in this study. If complaints or problems concerning this research project should arise, they should be reported to Dr. Jake Najman at the University of Queensland (phone 33653344), or Dr. Patricia Brennan at Emory University (phone 1-404-727-7458) or you may contact Dr. James Keller, Chairman of the Emory University Human Investigations Committee regarding your rights as a research subject (phone 1-404-727-5646). If you would like to speak to an officer of the University not involved in the study, you may contact the Ethics Officer on 3365 3924.

New Findings: If we learn anything new during the course of this research study that we believe is important to you, we will tell you about it. We will give you a copy of this consent form to keep. Feedback about the results of the study will only be available through publications in academic journals.

Thank you in advance for your effort and honesty in completing the interview today. Once again, many thanks for your help in the past and we hope that you will be able to help again.


 Patricia A. Brennan, Ph.D.
 Principal Investigator

20/03/01
 Date

Your signature below indicates that you consent voluntarily to be in this study.

Youth's Name _____ Youth's Signature _____ Date _____

Witness Name _____ Witness Signature _____ Date _____

Appendix D

SUICIDAL BEHAVIOUR

SUICIDAL BEHAVIOUR OR PLAN

Did ... ever have a specific plan to kill her/himself, that s/he didn't follow, carry out or try?

Has ... ever tried to kill her/himself or done anything that could have killed her/him?

IF NOT, STOP HERE

How many times? Other details?

	PAST	CURRENT
SUICIDAL PLAN		
DISCRETE GESTURES OR ATTEMPTS		
SUICIDAL INTENT		
<i>Question seriousness</i>		
ACTUAL THREAT TO LIFE OR PHYSICAL CONDITION FOLLOWING ATTEMPT		
IMPAIRED CONSCIOUSNESS DURING RESCUE		
RECEIVING TREATMENT		
TREATMENT REQUIRED		

IF SUICIDAL BEHAVIOUR OCCURRED DURING EPISODES OF ILLNESS RECORD UNDER THE FOLLOWING DIAGNOSTIC CATEGORIES. INDICATE ALL THAT APPLY.

- Depression _____
- Mania _____
- Schizophrenia _____
- Conduct Disorder _____
- Alcoholism _____
- Substance Abuse _____
- Schizo-Affective Disorder _____
- Other: _____

Appendix E

STRESSFUL LIFE EVENTS 15-YEAR INTERVIEW

EPISODIC STRESS INTERVIEW

EVENT CODE: _____ ID #: _____
 Interpersonal? _____ Date: _____
 Conflict? _____ Interviewer: _____
 1 = yes 0 = no

DATE OF OCCURRENCE: _____

DESCRIPTION OF EVENT: (Probe: what happened, expected or not, previous experience with event, desirability of event, resources-tangible or emotional support, coping ability, particular circumstances of subjects life that may modify event's impact and consequences, how event changed life of subject)

Duration: _____

A. Subjective Rating of Negative Impact/Stress:

1	2	3	4	5
none	mild	mod.	marked	severe

B. Team Objective Rating of Negative Impact/Stress:

1	2	3	4	5
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C. Team Rating of Independence:

1	2	3	4	5
almost completely independent		mixed		almost completely dependent

Appendix F

STRESSFUL LIFE EVENTS 20-YEAR INTERVIEW

MAJOR NEGATIVE EVENTS BETWEEN AGE 15-19

INSTRUCTIONS: Read each item, use 4-year timeframe, write narratives only for those thought to be level-3 or higher in severity. Do not be limited to examples—they are for illustration only. Events that happen to other people must have some direct impact on the youth's life—not just emotional impact.

1. Major academic failure [quit school, expelled or flunked out, failure to be admitted to hoped for program or institution]
2. Severe financial difficulties [severely threatens lifestyle, major obligation beyond typical]
3. Severe illness or injury of close family member, close friend, or self [either life-threatening or has significant health, financial, or care implications]
4. Death of close family member or close friend
5. Assault or rape [including date rape]
6. Became pregnant or fathered pregnancy
7. Birth of child
8. Victim of serious crime
9. Legal problem, committed crime, jail [do not include traffic violations unless significant negative consequences]
10. Major argument with close family member [involves violence, leads to prolonged rupture of relationship]
11. Marital separation or divorce [could also include break off of engagement, end of serious committed relationship, split with father of child if marriage had been expected]
12. Other _____
13. Other _____

Appendix G

SOCIAL SUPPORT AND SOCIAL INTEGRATION INTERVIEW

Question over the last 6 months

Close Friendships

Closeness, conflicts, trust, availability, reciprocity, dependability, contact.

Number of close friends (excluding siblings, aunts and uncles, spouse's siblings, co-workers unless there is contact out of work, opposite sex if dating and old friends with no recent contact)

- 1 9 or more
- 2 5 to 8
- 3 2 to 4
- 4 1
- 5 No close friends

Reticence (talking about feelings with friends and satisfaction with this)

- 1 Reasonably open with at least 1 person
- 2 Mildly reticent
- 3 Moderately reticent or occasionally unable to discuss
- 4 Usually unable to discuss feelings
- 5 Unable to discuss feelings at any time

Social Life

Social group, number of friends, activities, conflict, popularity

Number of social interactions (entertaining or visiting friends, weddings, parties, club meetings, etc)

- 1 Twice a week or more
- 2 once or twice a week
- 3 once a fortnight
- 4 once a month
- 5 none to once every 2 months

Activities

- 1 Well developed, specific interests or activities which the subject participates in more than once a week
- 2 Definite interests or activities to which the subject devotes regular but less frequent time
- 3 Some specific interests but these are sporadic
- 4 Some interests but these are superficial or indiscriminating (e.g watching TV with little regard to programs)
- 5 Absence of any interests or activities

Friction

- 1 Smooth relationships or no visible annoyance
- 2 Not provocative but overt difficulty with sensitive situations
- 3 Rather uneasy tense relationships or one major incident
- 4 Moderate friction or friction with many people
- 5 Many furious clashes or is deliberately avoided by all others

Have any of your friends offended you or hurt your feelings in the past 6 months? Tell me what happened. How long did it take you to get over this? Do you act the same now toward that person as you did before they offended you?

- 1 Behaviour reasonable or unaffected, or subject does not remember taking offence
- 2 Behaviour affected but returns to normal within hours
- 3 Behaviour affected but recovers in days
- 4 Behaviour altered requiring a week or more to recover
- 5 Behaviour altered toward others as well as individual, behaviour has not recovered in a month

Have you felt ill at ease, tense or shy when you have been with people during the past 6 months? Did you feel anxious to get away or to be alone with people? Did you avoid being with people because you felt uncomfortable? Rate subject's feelings. Do not rate unless there were several people present or if subject has not been in a social situation.

- 1 Enjoys company
- 2 Occasionally uncomfortable but can relax
- 3 Often distressed but can enjoy company at times
- 4 Mostly distressed
- 5 Always very distressed in company

Have you felt lonely and wished for companionship these last 6 months? Have you felt this way when you were around people too? Do not include fear of being alone or "cosmic" loneliness.

- 1 Has not felt isolated
- 2 Feels a little more isolated or isolated occasionally
- 3 Feels moderately isolated or isolated often (i.e every weekend)
- 4 Feels a great need for people
- 5 Feels totally alone or feels lonely everyday

Boredom: *Have you felt bored in your free time in the last 6 months? Did you stay bored very long or could you find something to do?* Do not rate boredom at work.

- 1 Not usually bored
- 2 Occasionally bored but able to find activity or pass time
- 3 Frequently bored
- 4 Bored most of free time
- 5 Feels bored everyday

EPISODIC STRESSORS FOR FRIENDSHIPS IN LAST 12 MONTHS!

Romantic Relationships

Are they in a romantic relationship? 1=NO 2=YES

Duration of relationship (yrs & mths)

Have they been in a romantic relationship in the past 6 months? 1=NO 2=YES

Duration of relationship (yrs & mths)

In a relationship
 Conflict, sexual, reciprocal, problems, time spent together, partner problems. R1

No partner or brief uncommitted relationship
 Possibilities, contentment, dating-how often, different people. Sexual relations, Time since last partner. Pressure R2

UNMARRIED/NOT LIVING TOGETHER

Contact with girlfriend/boyfriend (Face to face contact)

- 1 More than twice weekly
- 2 Once or twice weekly
- 3 Once every two weeks
- 4 Once a month or less
- 5 Not at all in the past 6 months
- 6 No girlfriend/boyfriend

How much interest in spending time with girlfriend/boyfriend during past 6 months? Did you enjoy it or would you have been interested in it?

- 1 Pleasurable or interested
- 2 Usually pleasurable or interested
- 3 Variable or some interest
- 4 Little or no interest
- 5 Active dislike

FOR COUPLES LIVING TOGETHER OR MARRIED OR IN COMMITTED RELATIONSHIP

Friction

- 1 Smooth, warm relationship
- 2 Few tensions and disagreements
- 3 Moderate friction or coolness
- 4 Marked friction
- 5 Constant friction, marriage/partnership may be breaking

History of partner abuse (by partner)?

- 1 No
- 2 Yes, currently
- 3 Yes, current and past
- 4 Yes, in past

History of partner abuse (by subject)?

- 1 No
- 2 Yes, currently
- 3 Yes, current and past
- 4 Yes, in past

Reticence (talking about feelings and problems with partner)

- 1 Confides freely
- 2 Keeps back only a little
- 3 Moderate disability in communication
- 4 Marked disability
- 5 Completely unable to express themselves

FOR COUPLES LIVING TOGETHER OR MARRIED OR IN COMMITTED RELATIONSHIP

Domineering Behaviour: Who has been making most of the decision at home in the last 6 months? What decisions have you been making? Do you take your partner's wishes into consideration? Even when they're not there?

- 1 Non-domineering
- 2 Mildly domineering
- 3 Moderately domineering
- 4 Little consideration given to partner's wishes
- 5 Tyrannical

Submissiveness: If you and your partner have a disagreement on something, who usually gets their way? Who usually goes along? Have you been pressured or bullied by your partner during the past 6 months? Could you give me an example?

- 1 Can be firm when necessary
- 2 Firm enough except on unimportant issues
- 3 Cannot assert self against partner's firm decisions
- 4 Cannot assert self against partner's minor opposition
- 5 Cannot assert opinion even if invited to do so

During the past 6 months, have you had to depend on your partner to help you? What kinds of things have you needed help with? Do you lean on them for emotional support when you are upset?

- 1 Reasonably independent
- 2 Dependent in some ways
- 3 Moderately dependent
- 4 Markedly dependent
- 5 Depends on partner in least things, cannot care for self

What have your feelings been toward your partner during the past 6 months? Have you felt affection? Have you disliked them? Did you love them even when you were not getting along or did you sometimes wonder?

- 1 Consistent feelings of affection
- 2 Mostly feels affection but some misgivings
- 3 Markedly ambivalent feelings
- 4 Mostly negative feelings
- 5 Consistent dislike or detestation

**FOR ALL
EPISODIC STRESSORS FOR ROMANTIC RELATIONSHIPS IN LAST 12 MONTHS!**

EPISODIC STRESSORS FOR RELATIONSHIPS/DATING IN LAST 12 MONTHS!

Family Relationships

Closeness, trust, availability, acceptance, conflict, dependability, contact



Friction

- 1 Harmonious family relations
- 2 Fairly harmonious family relations
- 3 Indifferent or a few disagreements or one major argument
- 4 Moderate friction involving more than one person
- 5 Very discordant family relations

Reticent (Talk about feelings and problems openly)

- 1 Reasonably open with at least one person
- 2 Mildly reticent
- 3 Moderately reticent or occasionally unable to discuss
- 4 Usually unable to discuss feelings
- 5 Unable to discuss feelings at any time

Have you made an effort to keep in touch with family members or have you waited for them to contact you over the last 6 months? Who usually arranges getting together? Is there anyone in the family you have avoided seeing?

- 1 Initiates some contacts regularly
- 2 Initiates some contacts
- 3 Relies on family to initiate contacts
- 4 Avoids family contacts
- 5 No contact with family at all

Do you depend on your family for help or advice? for babysitting? for financial help? When you go visiting or go out is it usually with family or friends?

- 1 Quite independent
- 2 A few dependent relationships
- 3 Mostly dependent but has other resources
- 4 Almost totally dependent
- 5 Completely dependent

Did you do things just to make your family angry or annoyed or just to go against their wishes? Did you want to make them angry but didn't do it?

- 1 Feels no urge to defy family
- 2 A little inhibited by need to defy family
- 3 Some decisions and values determined solely by need to defy
- 4 Many important decisions/values determined solely by need to defy
- 5 Goes out of way to defy family continuously

Have you worried about things happening to members of your outside family during the past 6 months? What kinds of things have you been worrying about?

- 1 Shows reasonable concern
- 2 Frequently uneasy
- 3 Worries a fair amount
- 4 Very often worried
- 5 Crippled by unreasonable fears

Over the past 6 months, have you been feeling that you have let your relatives down at any time? How did you let them down? Have you felt guilty?

- 1 No guilt
- 2 Some slight misgivings
- 3 Moderately guilty
- 4 Very ashamed of their behaviour
- 5 Constant distressing feelings of guilt

Have you been feeling that your relatives have let you down at any time? How did they let you down? Have you felt bitter?

- 1 Reasonably satisfied with family
- 2 Appreciative but some grievances
- 3 Disappointment but some appreciation
- 4 Mostly bitter or disillusioned
- 5 Consumed by bitterness or resentment

IF CHILDREN

What kinds of things have you been doing with the children during the past 6 months?

- 1 Active involvement in children's lives
- 2 Good interest, knows children's lives well
- 3 Moderate interest
- 4 Little interest
- 5 Disinterest, totally uninvolved

Have you been able to talk with your children? Do they come to you with problems?
Consider what is appropriate for the child's age.

- 1 Communicates easily
- 2 Most times can communicate
- 3 Fair communication
- 4 Rarely able to talk
- 5 Never able to talk

Friction: How much friction has there been between you and the children? Have you had to discipline them much over the past 6 months? Do you tend to snap at them when you are tired or upset?

- 1 Smooth relationships
- 2 A little friction or tension
- 3 Moderate friction
- 4 Marked friction
- 5 Constant state of friction or children are intimidated and avoid parent totally

IF CHILDREN

What have your feelings been toward the children during the past 6 months? Have you felt affection for them? Did you dislike them? Did you wish sometimes that they weren't around or that they didn't live with you?

- 1 Consistently felt affection
- 2 Mostly loves the children
- 3 Moderate disaffection
- 4 Marked lack of love
- 5 Absolute lack of love and affection, dislikes children

MARRIED, LIVING TOGETHER, COMMITTED RELATIONSHIP OR CHILDREN

Have you worried about things happening to your partner, ex-spouse, or children during the past 6 months? What kinds of things have you been worrying about?

- 1 Shows reasonable concern
- 2 Frequently uneasy
- 3 Worries a fair amount
- 4 Very often worried
- 5 Crippled by unreasonable fears

Have you been feeling that you have let your partner, children, or ex-spouse down at any time in the past 6 months? How did you let them down? Have you felt guilty?

- 1 No guilt
- 2 Some slight misgivings
- 3 Moderately guilty
- 4 Very ashamed of their behaviour
- 5 Constant distressing feelings of guilt

In the past 6 months, have you been feeling that your partner, children, or ex-spouse have let you down at any time? How did they let you down? Have you felt bitter?

- 1 Reasonably satisfied with family
- 2 Appreciative but some grievances
- 3 Disappointment but some appreciation
- 4 Mostly bitter or disillusioned
- 5 Consumed by bitterness or resentment

EPISODIC STRESSORS FOR FAMILY RELATIONSHIPS IN LAST 12 MONTHS!