

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

**Neuropsychological Profiles of Adolescents
With a History of Childhood Trauma**

Michaela Ashley-Cooper – ASHMIC005

A dissertation submitted in fulfilment of the requirements for the award of the degree of Master of Social Science (MSocSc) in Psychology

Faculty of Humanities
University of Cape Town

2010

COMPULSORY DECLARATION

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature:

Date:

ACKNOWLEDGEMENTS

Dr. Kevin G. F. Thomas: Supervisor of this project. I cannot thank him enough for his time, effort, and his guidance on this project. His hard work is truly appreciated.

Dr. Jonathan Ipser: Researcher with the Cross University Brain Behaviour Initiative (CUBBI) and director of the Brain Behaviour Initiative (BBI) at UCT. I would like to thank Jonathan for the use of the Affective Go/NoGo test that he developed, and for his great help with setting it up and analyzing the data.

Ms. Kathleen Dey: Counselling Coordinator for Rape Crisis. Her help with recruitment and organizing the conducting of the interviews for this project was invaluable, and more than appreciated. Thanks must also go to Ms. Shiralee McDonald (Locum counselling coordinator for Rape Crisis), Joyce Doni (counselling coordinator for the Rape Crisis Centre in Khayelitsha), and all the wonderful staff at the Rape Crisis Centers; their selfless help with recruitment for this project went above what could be expected.

Scholarships: Harry Crossley Foundation PG scholarship, KW Johnston Bequest, Sylvia Gavron Psychology scholarship, Ernst & Ethel Eriksen Trust, and University Research Scholarship. This project would in no way have been possible without the financial assistance provided by these scholarships, my sincere thanks go to all these funders.

TABLE OF CONTENTS

ACKNOWLEDGEMENT	2
TABLE OF CONTENTS	3
LIST OF FIGURES	8
LIST OF TABLES	10
ABSTRACT	12
INTRODUCTION	13
The Neuropsychology of Post-traumatic Stress Disorder (PTSD)	16
Low IQ as a Risk Factor for PTSD	20
Neurological Correlates of PTSD Symptoms	22
Problem 1: Not Using Theory as a Basis for Choice of Test	24
<i>Long-Term Neurobiological Effects of Childhood Trauma</i>	24
<i>The Physiological Stress Response</i>	24
<i>The Hippocampus</i>	27
<i>The Prefrontal Cortex</i>	27
<i>From Neurobiology and Neurophysiology to Neuropsychology</i>	27
<i>Problem 1: Summary and Conclusion</i>	28
Problem 2: Lack of Appropriate Control Groups	29
<i>Problem 2: Summary and Conclusion</i>	30
Problem 3: Not Assessing both Traumatic Even Characteristics and Post-traumatic Reactions	30
<i>Problem 3: Summary and Conclusion</i>	31
Other Specific Areas of Interest in the Current Study	31
<i>Spatial Navigation</i>	32
<i>Information-Processing Biases</i>	33

<i>Sex Differences in Cognitive Performance, Spatial Navigation, and Stress</i>	37
Conclusion	38
Specific Objective and Hypotheses	39
DESIGN AND METHODOLOGY	42
Participants	42
Materials and Apparatus	45
<i>Demographic Questionnaire</i>	45
<i>Screening Questionnaire</i>	45
<i>Clinical Instruments</i>	46
<i>Psychiatric Interview</i>	46
<i>Posttraumatic Stress Diagnostic Scale</i>	46
<i>Childhood Trauma Questionnaire - Short Form</i>	47
<i>Positive and Negative Affect Schedule</i>	47
<i>Connor-Davidson Resiliency Scale</i>	47
<i>Life Events Questionnaire</i>	48
<i>State-Trait Anxiety Inventory</i>	48
<i>Neuropsychological Battery</i>	49
<i>Wechsler Abbreviated Scale of Intelligence</i>	49
<i>NEPSY-II Inhibition subtest</i>	49
<i>Children's Memory Scale</i>	49
<i>Cambridge Neuropsychological Test Automated Battery</i>	50
<i>Computer-Generated Arena</i>	56
<i>Object Recognition Task</i>	58
<i>Arena Reconstitution Task</i>	59
Procedure	61
Ethical Consideration	63
Data analysis	65
Statistical Procedures	68
<i>Between-Group Comparison of Demographic and Clinical Characteristics</i>	68
<i>Neuropsychological Test Battery Performance</i>	69

<i>Data Sets With Repeated Measures</i>	69
RESULTS	70
Socio-Demographic Characteristics of the Sample	70
<i>Age</i>	71
<i>Race</i>	71
<i>Home Language</i>	71
<i>Years of Education</i>	71
<i>IQ data</i>	72
Clinical Characteristics of the Sample	73
Other Characteristics of the Sample	76
<i>Trauma-Related Characteristics of the Sample</i>	79
Testing Hypothesis 1 (1a and 1b)	80
<i>Descriptive Statistics of Domain Scores</i>	87
<i>Testing Hypothesis 1: Regression Results</i>	88
<i>Testing Hypothesis 1: Overall Regression Models</i>	89
<i>Testing Hypothesis 1: Post-hoc Regression Results for Individual Test Outcome Measures in Inhibition</i>	92
<i>Testing Hypothesis 1: Regression Models for Outcome Measures in Inhibition</i>	94
<i>Hypothesis 1: Summary and Conclusion</i>	95
Testing Hypothesis 2 (2a and 2b)	95
<i>Testing Hypothesis 2: CG Arena, Visible Target Trials</i>	96
<i>Testing Hypothesis 2: CG Arena, Invisible Target Trials</i>	100
<i>Testing Hypothesis 2: CG Arena, Probe Trial</i>	104
<i>Hypothesis 2: Summary and Conclusion</i>	108
Testing Hypothesis 3 (3a and 3b)	108
<i>Affective Go/NoGo Task: Emotional Information Processing Biases</i>	109
<i>Testing Hypothesis 3: Affective Go/NoGo Task, NoGo Data</i>	109
<i>Testing Hypothesis 3: Affective Go/NoGo Task, Go Data</i>	112
<i>Testing Hypothesis 3: Affective Go/NoGo Task, Beta</i>	114

<i>Hypothesis 3: Summary and Conclusion</i>	116
Testing Hypothesis 4	116
<i>Testing Hypothesis 4: Multiple Regression of Trauma and PTSD data</i>	116
<i>Testing Hypothesis 4: Two Domains Significantly Affected by One of the Step 2 Variables</i>	118
<i>Hypothesis 4: Summary and Conclusion</i>	120
Other Analyses	121
<i>Testing Effects of Personal Characteristics and Traumatic Events</i>	121
<i>The Three Domains Significantly Affected by One of the Step 2 to 6 Variables</i>	122
<i>Other analyses: Summary and Conclusion</i>	126
DISCUSSION	127
Hypothesis 1: The Effects of Trauma and PTSD on Performance in the Test Battery	128
<i>Cognitive Domains Affected</i>	128
<i>Cognitive Domains Not Affected</i>	130
<i>The Role of IQ in PTSD and Trauma Exposure</i>	132
Hypothesis 2: The Effects of Trauma and PTSD on Performance of Spatial Navigation Tasks	133
<i>Testing Hypothesis 2: CG Arena, Visible Target Trials</i>	134
<i>Testing Hypothesis 2: CG Arena, Invisible Target Trials</i>	134
<i>Testing Hypothesis 2: CG Arena, Probe Trial</i>	135
Hypothesis 3: The Effects of Trauma and PTSD on Information-Processing Bias	135
Hypothesis 4: The Effects of Trauma Characteristics and Trauma Responses on Performance	136
Other Analyses of Interest: The Role of Other Factors in Test Performance	136
Sex Differences in the Effects of Trauma Exposure and PTSD on Test Performance	137
Possible Reasons for Inconsistent Findings	140
<i>Age differences in the Research</i>	142

Important Findings	142
Other Interesting Findings	143
<i>Clinical Characteristics of the Trauma and PTSD Adolescents</i>	143
<i>Other Characteristics of the Whole Sample</i>	143
Limitations and Directions for Future Research	144
Conclusion and Implications for Treatment	145
REFERENCES	147
APPENDIX A: Demographic Questionnaire	173
APPENDIX B: Screening Questionnaire	175
APPENDIX C: Screening Questionnaire (Rationale and Results)	178
APPENDIX D: Life Events Questionnaire	179
APPENDIX E: Parent/Guardian's Informed Consent Document	181
(for Participants from Treatment Centers and the Boys School)	
APPENDIX F: Assent Form for Participants at the Treatment Centers	185
APPENDIX G: Parent/Guardian's Informed Consent Document	187
(for Participants from the Co-ed School)	
APPENDIX H: Example of Assessment Report	190
APPENDIX I: ART Scoring Sheet	192
APPENDIX J: Post-hoc Results from the CG Arena: Invisible Trials	193
APPENDIX K: Post-hoc Results from the CG Arena: Probe Trial	194
APPENDIX L: Separate Regression Results for the Factor: Perpetrator	195

LIST OF FIGURES

Figure 1. Traumatic events experienced by participants in final sample.	43
Figure 2. Flow chart of final sample	44
Figure 3. Screen shot of the SSP tasks	51
Figure 4. Screen shot of the SOC task	52
Figure 5. Screen shot of the IST task	53
Figure 6. Screen shot of the IED task	54
Figure 7. Screen shot of the CGT task.	54
Figure 8. Screen shot of the PAL task	55
Figure 9. Screen shot of the SRM task	56
Figure 10. Facing the North wall, with the invisible target marked	57
Figure 11. Facing the corner of the East wall and the South wall	58
Figure 12. Facing the corner of the West wall and the South wall	58
Figure 13. ORT target and distractor pictures (displayed separately as 8 screens)	59
Figure 14. ART Stimulus Sheet	60
Figure 15. Number of participants who met diagnoses of grouped disorders	76

Figure 16. Visible trials: length to target	99
Figure 17. Length to visible target: main effect of group membership	100
Figure 18. Length to invisible target: main effect of group membership.	103
Figure 19. Length to invisible target: interaction effect between group membership and sex.	104
Figure 20. Probe Trial: time spent in each quadrant.	106
Figure 21. Time spent in the northwest quadrant: interaction effect between group membership and sex.	107
Figure 22. Average NoGo error rates across groups for each emotional condition	111
Figure 23. Average reaction time for each target emotion across the three groups	113
Figure 24. AGNG Beta: interaction between group membership and sex	115

LIST OF TABLES

Table 1. <i>Neuropsychological Test Battery used in the Study</i>	61
Table 2. <i>Tests Administered in Each Session</i>	63
Table 3. <i>Socio-Demographic Characteristics of the Current Sample</i>	70
Table 4. <i>IQ Results for each Group Broken Down by Sex</i>	72
Table 5. <i>Psychiatric Disorder Comparisons Across Groups</i>	74
Table 6. <i>Characteristics of the Sample as Assessed by the Paper-and-Pencil Questionnaires</i>	77
Table 7. <i>Levene's Test Results for the Paper-and-Pencil Questionnaires</i>	78
Table 8. <i>Trauma-related Characteristics of the Sample as Assessed by the MINI KID 5.0 and the PDS</i>	79
Table 9. <i>Descriptive Statistics for Composite Domain Categories</i>	82
Table 10. <i>Testing Hypothesis 1: Regression Analysis Results of Significant Neuropsychological Domain score.</i>	89
Table 11. <i>Testing Hypothesis 1: Primary Regression Model Results for each Domain</i>	91
Table 12. <i>Testing Hypothesis 1: Post-hoc Regression Analysis Results for Significant Individual Outcome Measure.</i>	93

Table 13. <i>Testing Hypothesis 1: Post hoc Regression Model Results for each Outcome Measure in Inhibition</i>	94
Table 14. <i>Testing Hypothesis 2: CG Arena Data for Visible Target Trials for Each Group</i>	97
Table 15. <i>Testing Hypothesis 2: CG Arena Data for Invisible Target Trials for Each Group</i>	101
Table 16. <i>Testing Hypothesis 2: CG Arena Data for the Probe Trial for each Group</i>	105
Table 17. <i>Testing Hypothesis 3: AGNG No-Go Data</i>	110
Table 18. <i>Testing Hypothesis 3: AGNG Go-Target Data</i>	112
Table 19. <i>Testing Hypothesis 3: AGNG Response Bias Data</i>	114
Table 20. <i>Testing Hypothesis 4: Trauma and PTSD Regression R^2 Results for each Neurocognitive Domain</i>	120
Table 21. <i>Testing Effects of Personal Characteristics and Traumatic Events: Regression R^2 Results for each Domain</i>	124
Table 22. <i>Testing Effects of Personal Characteristics and Traumatic Events: Regression Results for Steps 2, 3, 4, 5, and 6</i>	125

ABSTRACT

Traumatic events experienced in childhood, such as physical and sexual abuse, can lead to multiple long-term effects on later cognitive functioning. Empirical research has shown that specific brain regions are affected by traumatic stress, including the hippocampus and the prefrontal cortex. It follows that the cognitive abilities subserved by these regions, including spatial navigation, new learning, and executive functioning, are negatively affected.

Furthermore, in some cases, this exposure may lead to posttraumatic stress disorder (PTSD), a disorder which brings with it, its own set of neural abnormalities and their corresponding neuropsychological deficits. However, very few studies have looked at the effects of trauma exposure and PTSD on *adolescents'* performance on a comprehensive neuropsychological test battery. Furthermore, due to methodological problems in the research that has been done, findings have, thus far, been conflicting and thus inconclusive. The aim of the current study was therefore, to provide a thorough investigation of how trauma exposure and PTSD effect adolescents' cognitive functioning (looking specifically at those tasks subserved by the hippocampus and the prefrontal cortex), correcting for the methodological flaws seen in the research thus far.

The study compared the performance of three groups of South African adolescents, consisting of 16 adolescents who had been exposed to a trauma and had a diagnosis of PTSD, 16 adolescents who had been exposed to a trauma but did not have PTSD, and 17 adolescents who served as matched controls. Results showed that participants in the trauma group performed significantly poorer in the domain of Inhibition (compared to controls), and that participants in PTSD group performed significantly poorer in the domain of Spatial navigation (compared to controls; this effect was however, only seen in females). Moreover, the results showed that number of symptoms experienced by trauma victims is significantly associated with performance in some domains (namely Processing speed and Decision-making/impulsivity). The findings of the current study suggest that the differences in performance reflect impaired neural functioning which is due to both trauma exposure as well as the individual's posttraumatic responses.

Keywords: Posttraumatic Stress Disorder; adolescents; trauma; prefrontal cortex; hippocampus; cognitive functioning

INTRODUCTION

Adverse events during early childhood, such as physical and sexual abuse, maltreatment, and neglect, often have long-term effects on later functioning. These effects, the consequences of early adversity, can often be seen in adolescents' performance on tests of neuropsychological function.

For instance, Beers and De Bellis (2002) found that children and adolescents with maltreatment-related posttraumatic stress disorder (PTSD; $n = 14$, mean age = 11.38 years, $SD = 2.60$) exhibited significant deficits in two domains of neuropsychological functioning (attention, and problem-solving and abstract reasoning) compared to a control group who had not experienced any known trauma ($n = 15$, mean age = 12.17, $SD = 1.75$). Their findings also suggested relative deficits in the PTSD group in the cognitive domains of verbal learning and memory and of visual-spatial functioning (however, these results did not remain significant after statistical corrections).

The Beers and De Bellis study is the seminal work in this field in that it is one of the very few investigations that reports the administration of a comprehensive battery of neuropsychological tests to children and adolescents with a history of early adversity, thus examining the wide-ranging effects of childhood trauma on adolescent neuropsychological functioning. Other studies have provided some support for the data reported in that study. For instance, research with adult samples has consistently indicated that, with regard to the domains of attention, problem-solving, abstract reasoning, and visual-spatial functioning, individuals with PTSD perform more poorly on neuropsychological tests than do non-trauma controls (see, e.g., reviews by Anda et al., 2006; Beitchman et al., 1992; De Bellis, 2005; Pine, 2003; Teicher et al., 2003; Twamley, Allard, et al., 2009; Watts-English, Fortson, Gibler, Hooper, & De Bellis, 2006). Furthermore, numerous studies show that children who have experienced abuse and neglect present with deficits on standardized measures of cognitive and academic abilities when compared to demographically-matched samples (Hoffman-Plotkin & Twentyman, 1984; Holt, Finkelhor, & Kantor, 2006; Shonk & Cicchetti, 2001; Veltman & Browne, 2001; Wodarski, Kurtz, Gaudin, & Howing, 1990; Zolotor et al., 1999).

Other studies in the field have, however, introduced conflicting evidence as to the specific neuropsychological deficits present in adolescents with a history of early childhood trauma. The domain of memory is particularly salient here. Beers and De Bellis (2002) found significant between-group differences on only one of six measures of memory, suggesting that memory impairments in their PTSD group were confined to a relatively circumscribed set of memory processes. In contrast, Moradi, Neshat-Doost, Taghavi, Yule, and Dalgleish (1999) found that, relative to an age-matched control group with no history of trauma exposure and no past or current emotional disorder ($n = 22$, mean age = 14.33, $SD = 1.46$), children with PTSD ($n = 18$, mean age = 14.28, $SD = 2.04$) exhibited significant deficits in *overall* everyday memory performance, as assessed by the Rivermead Behavioural Memory Test (Wilson, Cockburn, & Baddeley, 1985).

The discrepancies in the findings of these two studies, where one found only circumscribed memory deficits in their adolescent PTSD group and the other found general and wide-spread memory deficits in a similar group, are instructive in highlighting problematic issues in evaluating findings in this literature. This discrepancy could, for instance, be due to measurement differences (the two sets of investigators did not use common tests), relatively small sample sizes, trauma severity, severity of PTSD symptoms, or differences in the ages of the children in the two samples (those in the Beers and De Bellis study were, on average, almost 2 full years younger than those in the Moradi, Neshat-Doost, et al. study, a fact that might have developmental significance).

Thus, the problematic issues in evaluating research in this field centre largely around methodological issues. Although there are a limited number of comprehensive studies on the neuropsychological effects of trauma on adolescent functioning, even within this small number there are numerous methodological problems. These are problems that are present in almost all research investigating the long-term neuropsychological effects of trauma (Moore & Zoellner, 2007). The three most important of these problems in terms of their relevance to the current study are detailed below.

The first problem, which is clearly illustrated by the Beers and De Bellis (2002) study, is that many researchers tend to simply administer a large battery of neuropsychological tests without any theoretical basis for their choice of test. Otherwise stated, they do not explicitly predict, based on psychological, neurobiological, neuropsychological, or any other theoretical framework, *which* cognitive functions will be affected by early adversity and *why* those functions will be affected.

The second problem is evident in both Beers and De Bellis (2002) and in Moradi, Neshat-Doost, et al. (1999), and, in fact, throughout trauma research. This problem involves the absence of appropriate control groups. In studies that examine the effect of trauma by examining groups with PTSD, control groups typically consist of non-traumatized healthy controls. The absence of a control group with a history of trauma exposure, but without a PTSD diagnosis, leads to inconclusive results: This control group is needed in order to clearly differentiate between the effects of exposure to trauma as opposed to the effects of the post-traumatic reactions (i.e., the symptoms of PTSD). Without this control group, the reasons for the presented deficits are uncertain, at best.

The third problem is that of flaws in the measurement of the potentially traumatic event. Many studies in this field (see, e.g., Brewin, Reynolds, & Tata, 1999; Brewin, Watson, McCarthy, Hyman, & Dayson, 1998; Kuyken & Brewin, 1995) assess the impact of the traumatic event solely by the characteristics of that event (e.g., severity, rate of occurrence, type of event). Other studies, however, assess the impact of the event by measuring individuals' responses to it (see, e.g., Burnside, Startup, Byatt, Rollinson, & Hill, 2004; Peeters, Wessel, Merckelbach, & Boon-Vermeeren, 2002; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001). Moore and Zoellner (2007) show that, at least with regard to autobiographical memory processes, the reaction of individuals to a traumatic event is more important to predicting outcomes than are the actual characteristics of the event itself. To this point, however, very few studies in this field have assessed both traumatic event characteristics *and* post-traumatic reactions, although it seems clear that there are sound reasons to make both part of the research design.

Before each of these problems can be discussed in detail, a thorough examination of the neuropsychology of PTSD and the neurological correlates of PTSD symptomology is necessary.

The Neuropsychology of Post-traumatic Stress Disorder (PTSD)

A DSM-IV-TR (Diagnostic and Statistical Manual, Fourth Edition, Text Revision; American Psychiatric Association [APA], 2000) diagnosis of PTSD necessitates that a person has been exposed to a significant threat, and that that exposure resulted in feelings of fear, helplessness, or horror. The diagnostic criteria also suggest, and empirical research confirms, that there are three main groups of symptoms: re-experiencing the trauma, avoidance of trauma-related external and internal cues, and hyperarousal/hypervigilance (Brandes et al., 2002; Nemeroff et al., 2006).

Exposure to traumatic stress can lead to a prolonged physiological response. In some cases this develops into PTSD. Indeed, empirical research has also shown that PTSD is associated with the dysregulation of the neurobiological stress systems in the human brain. Furthermore, empirical research has shown that structural alterations in at least three brain regions are associated with PTSD. These brain regions include the amygdala and, not surprisingly, the hippocampus and the medial prefrontal cortex (PFC) (Vasterling & Brewin, 2005). In PTSD, there is a reduction in the size of the hippocampus, and a reduction in responsivity of the PFC.

With regard to the hippocampus, numerous neuroimaging studies confirm that, in samples of adults diagnosed with PTSD, the volume of this region appears to be decreased (see, e.g., Bremner et al. 1995; Gurvits et al., 1996; Stein et al., 1997). Two meta-analyses on this topic confirmed that adult PTSD patients do exhibit smaller volumes, in both hippocampi, compared to adults without PTSD (Bremner, 2005; Smith, 2005).

It logically follows that this association between PTSD and reduced hippocampal volume might lead to impairments in cognitive processes dependent on the integrity of the hippocampal formation. Much research has therefore been conducted on exploring the effects PTSD has on hippocampal-dependent cognition in adults. For instance, Bremner et al. (1995) found that combat veterans with PTSD displayed significant deficits in verbal declarative memory compared to a control group of comparison subjects without PTSD. More specifically, these

deficits were on tests of immediate and delayed recall and retention; all of these processes are dependent on the left hemisphere hippocampus (Bell et al., 2002). Numerous similar studies showing PTSD-associated deficits in verbal learning and memory have been published over the past two decades (see, e.g., Bremner et al., 1993; Bremner et al., 1999; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; but see Brandes et al. (2002) and Stein et al. (1999, 2002) for studies reporting no memory deficits in adults with PTSD). Vasterling and Brewin (2005, p. 183) summarized this literature by saying that “initial acquisition [of verbal information] is the most pervasively impaired aspect of memory dysfunction associated with PTSD.”

The association between a diagnosis of PTSD and impaired performance on neurocognitive tests dependent on the integrity of the right hemisphere hippocampus (e.g., tests of spatial navigation and spatial learning and memory; Baddeley, Kopelman, & Wilson, 2002; Maguire, 1999) has not been explored in very many investigations. Even in those few investigations, however, equivocal data have been reported. For instance, some researchers have found no differences between adults with PTSD and non-traumatized comparison samples with regard to performance on spatially-based tasks (see, e.g., Gilbertson et al. 2001; Gurvits, Carson, et al., 2002; Sullivan et al., 2003), whereas others have found clear between-group differences in performance on such tasks (see, e.g., Emdad & Sondergaard, 2006; Gurvits, Lasko, et al., 2002).

Although much research has been conducted on the neuropsychological effects of PTSD on adult survivors of trauma, very few neuropsychological studies have focused on PTSD in children and adolescents. Perhaps of most interest with regard to the latter is that, unlike in adults, there is no pattern of hippocampal volume reduction in children with PTSD (Carrion et al., 2001; De Bellis et al., 1999; De Bellis, Keshavan et al., 2002). This discrepancy between the findings of adult and child neuroimaging studies of PTSD raises the potentially interesting question of how adolescent hippocampal functioning is affected by PTSD.

Although neuroimaging research has not found a reduction in hippocampal volume in children with PTSD, some neuroimaging studies have found that there are profound structural alterations in the PFC (e.g., a reduction in volume; Vasterling & Brewin, 2005), and in other brain regions, in children with PTSD (Anderson, 2002; Carrion et al. 2001; De Bellis, et al., 2002). The

expected deficits associated with such alterations in the PFC have been confirmed by empirical research into the matter (see, e.g., Bremner, 1999; Shin et al., 1999). For instance, Richert, Carrion, Karchemskiy, and Reiss (2005) showed that all regions of the PFC were significantly altered in children and adolescents with PTSD, that functional impairment (e.g., school performance and social functioning) followed specifically from these alterations, and that the cognitive-emotional functions dependent on the dorsolateral PFC were the most affected.

Although most studies exploring executive function deficits in PTSD have found marked differences amongst patients compared to healthy controls (see, e.g., Beckman, Crawford, & Feldman, 1998; Gilbertsen et al., 2001), many studies have found no such differences (Stein, Hanna et al., 1999; Twamley, Hami, & Stein, 2004). The reason for these different findings across studies is probably due, at least in part, to the fact that the term 'executive function' encompasses numerous distinct cognitive skills, such as response inhibition, attention, problem-solving, impulsivity, processing speed, working memory and information-processing. Therefore, when considering the research done on executive functioning in adults with PTSD, it is more useful to consider the effects of the psychiatric disorder on *specific* abilities that fall under this umbrella term.

Research has found specific deficits in the following executive abilities in PTSD populations: mental flexibility (Kanagaratnam & Abjorsen, 2007); working memory and attention (Brandes et al., 2002; Gilbertson et al., 2001; Samuelson et al., 2006; Vasterling et al., 2002); processing speed (Brandes et al., 2002; Twamley, Allard, et al., 2009); inhibition (Stein, Kennedy, & Twamley, 2002)

On the other hand, research has also suggested that there are no significant differences between PTSD patients and control participants on executive abilities such as attention (Golier et al., 1997; Yehuda, Keefe et al., 1995); response inhibition (Kanagaratnam & Abjorsen, 2007); switching within inhibition (Twamley, Allard, et al., 2009); set-shifting (Sutker, Vasterling, Brailey, & Allain, 1995); and abstraction (Twamley, Allard, et al., 2009).

This brief review of the research done on executive functioning in PTSD highlights that there are notoriously mixed results in this field of study. The possible reasons for these conflicting results (reviewed in Danckwerts & Leathem; 2003) revolve around issues such as overgeneralizing the results from specific samples to more general populations, the use of markedly different neuropsychological tests across studies, and the use of neuropsychological tests that cannot be extrapolated to everyday situations (i.e do not test for the deficits that exhibit in daily life, and moreover that do not measure cognition in a way that the results can be applied to daily life.)

Although many studies, like those of Richert et al. (2005), De Bellis et al. (2002) and Carrion et al. (2001), only compared a PTSD group to a non-traumatized group, the pattern of results showing PTSD-associated alterations in PFC structure has been replicated in a study comparing a group of adults with PTSD to a group of adults who had experienced trauma but who had no PTSD diagnoses (Woodward et al., 2006). Similarly, in a study comparing a PTSD group, a traumatized non-PTSD group, and a non-traumatized non-PTSD healthy control group, Leskin and White (2007) found that adults with PTSD showed particular executive function deficits over and above the deficits exhibited by the adults who had experienced trauma but had no PTSD diagnosis.

Although the association of PTSD with reduced volume in certain brain regions and with a pattern of cognitive deficits consistent with damage to those regions is undeniable given the weight of the literature, reasons for *why* people with a history of trauma show reductions in brain volume in certain regions are not completely agreed upon. Many researchers (e.g., Bremner, 1999; Watts-English et al., 2006) assert that exposure to traumatic levels of stress leads to a reduction in growth, and perhaps even neural cell loss, in certain brain regions (putative mechanisms for this causal chain are discussed in later sections.) These researchers usually base their theories on empirical work that looks at exposure to traumatic events, rather than at populations with PTSD diagnoses.

There is, however, an alternate theory proposing to explain the link between regional reductions in brain volume and a history of childhood trauma and/or a PTSD diagnosis. Proponents of this alternate theory hold that reduced size of these specific brain regions *precedes* any traumatic

exposure (and therefore also, of course, precedes any PTSD diagnosis), and indeed is a marker for vulnerability to psychopathological post-traumatic reactions. Empirical support for this theoretical proposition emerges from studies such as that of Gilbertson et al. (2002), who found that individuals with reduced hippocampal volume were more prone to developing PTSD (i.e., a smaller hippocampus appeared to constitute a vulnerability for the disorder among those exposed to trauma). In this study, Gilbertson et al. (2002) compared monozygotic twins with discordant histories of trauma exposure, and found that disorder severity in PTSD patients was negatively correlated with the hippocampal volume of both the patient and the patient's trauma-unexposed identical co-twin. Furthermore, severe PTSD twin pairs (both trauma-exposed and non-exposed) had significantly smaller hippocampi than non-PTSD twin pairs. Similar findings on vulnerability have been found in Holocaust survivors (Yehuda, Schmeidler, Wainberg, Binder-Brynes, & Duvdevani, 1998) and in children and adolescents (Silva, Alpert, Munoz, Singh, Matzner, & Dummit, 2000).

Silva et al. (2000) examined the interactions between factors such as demographic characteristics, such as age and gender, and clinical antecedents (other diagnoses) and the development of PTSD. They found that, not surprisingly, pre-existing levels of anxiety is a precipitating factor in the development of PTSD, as well as having a lower IQ.

Low IQ as a Risk Factor for PTSD

An area of particular importance in understanding vulnerability to PTSD is the research done on the relationship between IQ and PTSD. As suggested by the findings of Silva et al. (2000), numerous research studies, from independent laboratories, have reported that lower IQ is a significant risk factor for developing PTSD (Leskin & White, 2007; McNally, 2003); this is true both in adults (Buckley, Blanchard, & Neill, 2000; Kaplan et al., 2002; Macklin et al., 1998), and in children and adolescents (Koenen et al., 2006; Saltzman et al., 2006; Silva et al., 2000). In particular, longitudinal prospective studies have found that trauma victims who develop a PTSD diagnosis had lower IQ scores prior to the traumatic event compared to those victims who did not develop a PTSD diagnosis.

In the longitudinal study by Koenen et al. (2006), a community sample of 1037 adults (from a birth cohort) were assessed for PTSD and the effects that previous childhood factors (including antisocial behaviour, poverty and IQ) have on the development of this disorder were examined. The participants were assessed at age 26 and at age 32 again. Using this data and childhood data from the cohort study, the researchers found that low IQ in childhood (assessed by IQ tests administered at age 5) significantly predicted PTSD in adulthood.

Conversely, some research has suggested that trauma and PTSD may in fact lead to deficits in performance on IQ tests, both in adults (Brandes et al., 2002; Vasterling, Brailey, Constans, Borges, & Sutker, 1997), and in children and adolescents (Jenkins, Langlais, Delis, & Cohen, 2000; Saigh, Yasik, Oberfield, Halamandaris, & Bremner, 2006). Based on the literature discussed on the neuropsychological effects of stress, trauma, and PTSD, it is not far-fetched to suggest that performance on IQ tests should also be negatively affected. This argument is plausible because IQ tests rely, at least to some degree, on intact hippocampal and PFC functioning (including intact memory and attentional capacities; Mills-Schumann et al., 2007). These results are, however, not conclusive, and therefore, in this study, IQ will act as an independent variable/covariate, and is not assessed as an outcome measure.

Although the foundation of this study, based on sound theory and substantial empirical research, identifies the neuroanatomical abnormalities of the prefrontal cortex and the hippocampus as the reason for the neuropsychological deficits exhibited associated with PTSD, many researchers have suggested that it is the symptoms of PTSD which are responsible. Along with other researchers, Moradi, Neshat-Doost, et al., (1999) suggest that the presence of the PTSD symptoms of intrusion, avoidance and hyperarousal may interfere with a child's ability to perform. This is possibly due to these symptoms disrupting their ability to maintain attention.

This theory however, is *not* contradictory to the theory set out in this study. Explorations of the neurological correlates of PTSD are thus explored in order to explain how these theories may *both* in fact be true.

Neurological Correlates of PTSD Symptoms.

The prolonged physiological response to traumatic stress, described in more detail later, leads to prolonged hyperarousal in multiple structures, including the hippocampus, the PFC, and the amygdala. This hyperarousal (and the resulting brain structure alterations) is suggested by some to be the basis of the way in which PTSD symptoms manifest at a behavioural level (Weber & Reynolds, 2004). The potential role of each of the three structures mentioned above in the development of PTSD symptoms is detailed below.

As discussed more thoroughly later, exposure to stress increases the release of dopamine, which affects PFC structural integrity and functionality. These stress-related alterations in dopamine release and the consequent brain structure changes are thought to be associated with the manifestation of the hypervigilance- and paranoia-related symptoms of PTSD (Weber & Reynolds, 2004). It has been suggested that the influx of dopamine, initially over-stimulating the PFC, leads to a non-responsiveness in the PFC, and this hypoactivity is associated with PTSD symptoms severity. Indeed in a meta-analysis on the structural brain abnormalities exhibited in PTSD Etkin & Wager, (2007) found that the ventromedial PFC, a structure linked to the experience and regulation of emotion, is consistently hypoactive in PTSD patients, and that this hypoactivation is a significant predictor of symptom severity

Furthermore, this excessive hyperarousal following traumatic stress has been shown to effect elements of executive functioning subserved by the PFC, such as attention (Kanagaratnam & Asbjormsen, 2007). This breakdown in functioning leads to a decrease in the ability to suppress involuntary thoughts and direct attention. It is this inability which researchers have suggested lead to the intrusive symptoms of PTSD (Kanagaratnam & Asbjormsen, 2007).

In terms of the hippocampus, along with the cognitive impairments resulting from excessive stress related alterations to this region, research has suggested that PTSD symptoms are associated with damage to the hippocampus. More specifically, neuroimaging research has found that hippocampal *volume* is inversely related to dissociative and depressive symptoms in PTSD (Bremner, Vythilingam, et al., 2003) reexperiencing symptoms in PTSD (Lindauer, et al., 2004) as well as PTSD symptom severity (Karl et al., 2006). It is suggested that the role of the

hippocampus in regulating the hypothalamic-pituitary-adrenal (HPA) axis response is responsible, at least in part, for the some symptoms experienced (such as dissociation; Etkin and Wager, 2007).

Along with the PFC and the hippocampus, the amygdala is also affected by traumatic stress (Etkin and Wager, 2007; Shin, Rauch & Pitman, 2006). Although the functioning of this structure is not assessed in this study, the effects of amygdala dysfunction are of interest in terms of understanding the symptoms of PTSD.

Although no reduction in size of the amygdala has been found in neuroimaging studies, hyperresponsiveness of the amygdala in PTSD patients has been found (Pissiota, Frans, Fernandez, von Knorring, Fischer, and Fredrikson, 2002; Protopopescu, Pan, Tuescher, Cloitre, Goldstein, Engelien et al., 2005; Shin, Orr, Carson, Rauch, Macklin, Lasko, et al., 2004).

As part of the limbic system, the amygdala is involved in the expression of emotion, particularly fear, emotional memory, and it is involved in autonomic reactions (such as increased blood pressure, increased heart rate, and the startle response). Theoretically dysfunction of this structure should lead to deregulation in these processes.

Indeed, empirical research has shown that a positive correlation exists between amygdala activation and PTSD symptom severity (Protopopescu et al., 2005; Shin, Orr, et al., 2004) and self reported anxiety (Fredrikson and Furmark, 2003; Pissiota, et al., 2002), suggesting that the hyperactivity of this structure plays a significant role in the symptoms of PTSD (particularly hyperarousal, and emotional intrusive thoughts).

It is suggested that it is a combination of both the biological alterations in these structures and the resulting affective and behavioral symptoms which are responsible for these deficits.

Now that an understanding of the neuropsychology of PTSD has been established, the reasoning behind the urgent need to address the three methodological problems mentioned earlier (namely, the utilization of neuropsychological theory in choice of tests, the use of appropriate control

groups, and the assessment of both traumatic event characteristics and post-trauma reactions) in the design of studies in this field is separately detailed below.

Problem 1: Not Using Theory as a Basis for Choice of Tests

In attempting to examine the overall cognitive deficits due to trauma exposure and PTSD, many studies merely test as many cognitive domains as possible. With no theoretical basis for test choice, the areas of that are responsible for the functional impairments remain unclear.

Conversely, basing test choice on neurobiological and neuropsychological theory allows for the clear examination of cognitive functioning, with direct indications to the brain regions that are responsible for these deficits. The following section attempts to outline the theory used in the current study, thereby addressing this need.

Long-Term Neurobiological Effects of Childhood Trauma

Research into the neurological effects of early adversity has shown that childhood exposure to traumatic events, such as sexual abuse, maltreatment, and neglect, leads to significant changes in brain functioning and development (Adamec, Blundell, & Burton, 2005). For the sake of clarity, the neurostructural and neurochemical effects of early adversity will be discussed separately from the cognitive impairments that might result from these brain abnormalities.

Childhood and adolescence are periods of significant development in the brain. This development includes selective elimination of neurons (Sowell, Trauner, Gamst, & Jernigan, 2002) and formation of discrete brain structures (Paus, Collins, Evans, Leonard, Pike, & Zijdenbos, 2001). These stages are thus critical to the development of the adult brain, and are subsequently particularly sensitive to insult. Any physical or psychological trauma experienced during these stages therefore has significant potential to disrupt typical neurodevelopment processes, thus leading to long-term negative outcomes (Watts-English et al., 2006).

The Physiological Stress Response. Trauma results in the human brain being subjected to high and unusually prolonged levels of stress hormones (glucocorticoids, the adrenal steroids secreted during times of stress; in humans, the major mediator of the physiological effects of stress is the glucocorticoid cortisol), as well as higher levels of catecholamines (neurotransmitters such as

epinephrine, norepinephrine, metanephrine, and dopamine). Studies investigating the effects of stress hormones on specific brain structures have suggested that damage to these regions is associated with direct exposure to glucocorticoids (Sapolsky, Uno, Rebert, & Finch, 1990).

The physiological stress response largely centres around the release of glucocorticoids, which is regulated by the hypothalamic-pituitary-adrenal (HPA) axis. This is a closed-loop neurocircuit controlled by a regulatory set of afferents, mostly the neurons in the paraventricular region of the hypothalamus. When the brain recognizes a stressful event or stimulus, these neurons secrete corticotrophin-releasing hormone (CRF). CRF stimulates the anterior pituitary gland to release adrenocorticotrophic hormone (ACTH), which then stimulates the adrenal gland to secrete glucocorticoids. The secretion of glucocorticoids regulates the entire HPA axis by providing negative feedback to stop further CRF and ACTH release (Bowman, 2005). Under traumatic levels of stress, this negative feedback is halted, meaning that glucocorticoids continue to be released unchecked.

Research into the neurophysiology of the stress response has accurately described the impact of stress hormones (such as cortisol) on particular parts of the brain. Researchers have identified three brain structures that are most affected by stress: the hippocampus, the PFC, and the amygdala (Kudielka & Kirschbaum, 2005). These regions show particular tendencies toward plasticity (i.e. they are particularly vulnerable to alterations; Teicher, Ito, Glod, Schiffer, & Gelbard, 1996), and so any adverse effects on them during key neurodevelopmental stages may have grave long-term consequences.

The human response to stress is, however, not confined to one system; it involves multiple networks. Along with the HPA-axis system, researchers have also identified some other neurotransmitter systems that play an important role in physiological responses to stressful events, including: the noradrenergic system, the serotonergic system, and the dopaminergic system (Vasterling & Brewin, 2005).

The noradrenergic system is involved in attention to selected stimuli, vigilance, alertness, and the cardiovascular response to stressful or life-threatening stimuli (the flight or fight response;

Aston-Jones, Raikowski, Kubiak, & Alexinsky, 1994). This system is responsible for creating a fear response, and for increasing the release of norepinephrine when a stressor is encountered. This neurotransmitter is released in the hypothalamus, and has effects on the amygdala, the hippocampus, and the PFC (Zigmond, Finlay, & Sved, 1995). Under traumatic stress, too much norepinephrine is produced, causing dysfunction in these regions.

Serotonergic systems are present in multiple brain regions, including the amygdala, the hippocampus, and the PFC (Nestler, Hyman, & Malenka, 2001). Under traumatic stress, there is excessive release of serotonin, once again causing dysfunction in these regions (Bremner et al., 2003).

The dopaminergic system is involved in selecting information to be processed, and is involved in general emotional responses. Its function also includes mediating basic behaviours such as locomotor activity, sexual activity, and appetite (Pani, Porcella & Gessa, 2000). This system is responsible in increasing the release of dopamine when a stressor is encountered. It has been suggested that this system attempts to protect against stress, as it is released in the Medial prefrontal cortex (MPFC), and this activity in the MPFC suppresses further dopamine release in the limbic system (Pani, Porcella & Gessa, 2000). However, under traumatic stress, too much dopamine is produced, causing dysfunction in these PFC specifically (Weber & Reynolds, 2004).

When traumatic stress occurs during *childhood*, neural development is highly likely to be adversely affected (Watts-English et al., 2006). For instance, traumatic levels of stress experienced due to child maltreatment have been associated with dysfunction in the neurobiological systems that are directly involved with brain maturation and associated cognitive and emotional/behavioral regulation (De Bellis, 2005).

In terms of the current study, only the cognitive effects of stress on the hippocampus and the PFC is of interest, and therefore only the hippocampus and the PFC will be discussed in terms of how stress affects these regions.

The Hippocampus. High levels of cortisol release, such as those seen under conditions of traumatic stress, disrupt the functioning of the hippocampus, a structure in the brain necessary for spatial navigation, new learning, the formation of new memories, and the memory processes of consolidation and retrieval (Payne & Nadel, 2004; Squire, 1992).

The reason for this specific disruption is because the hippocampus contains a high concentration of corticosteroid receptors, and, as mentioned above, is a major influence on the HPA axis by way of negative feedback (Ruel & de Kloet, 1995; Wright, 2006). When these receptor sites are filled due to unchecked cortisol release, hippocampal functioning is impaired. Empirical studies have confirmed that even acute stressful experiences increase cortisol levels and consequently are associated with impaired performance on hippocampal-dependent cognitive tasks (Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Payne, Nadel, Allen, Thomas, & Jacobs, 2002).

The Prefrontal Cortex. As mentioned in the discussions on the noradrenergic and the serotonergic systems, functioning in the PFC has been found to be significantly affected by the flooding of both norepinephrine and serotonin as a consequence of stress. Furthermore, in terms of the HPA-axis system, an increase in the release of glucocorticoids has been shown to have specific effects on the functionality of the PFC (Roosendaal, Jayme, & McGaugh, 2004).

The PFC is thus particularly susceptible to the adverse effects of stress. It has been suggested that the reason for this vulnerability is due to the fact that this region is not fully myelinated until almost 30 years of age (Teicher et al, 1997; until the neurons are completely myelinated the brain remains particularly plastic). Another reason suggested for this specific disruption is because the PFC contains a disproportionately high concentration of dopamine receptors (Donnelly et al., 1999). When these receptor sites are filled due to excess dopamine release, PFC functioning is impaired (Weber & Reynolds, 2004).

From Neurobiology and Neurophysiology to Neuropsychology

The brief review above makes it clear that exposure to traumatic events and consequent abnormal hormonal and neurotransmitter action can lead to dysregulated stress systems,

impairments in brain development, and discrete effects on various brain regions. Research done on stress in rodents, non-human primates, and humans suggests that early exposure to traumatic stress leads to long-term changes in many brain circuits and systems, including the amygdala, the cerebellum, and, most pertinently for the current study, the hippocampus and the PFC (Anderson et al., 2002; Gilbertson et al., 2002; Ito et al., 1993; Pine, 2003; Roche, Mangaoang, Commins, & O'Mara, 2005; Sapolsky et al., 1990; Stein, Koverola, Hanna, Tochia, & McClarty, 1997; Teicher et al., 1993).

The PFC is said to be generally involved in what neuropsychologists term the executive functions (Anda et al., 2006). More precise theoretical perspectives suggest, however, that three anatomically separate regions underlie their own set of cognitive functions: the dorsolateral regions subserve the maintenance of information and attention, the ventral regions are involved in response inhibition, and the medial regions are implicated in motivation, affect regulation, and the monitoring of performance or affective state (Pine, 2003). Interestingly in terms of the population studied in the current research, the dorsolateral region of the PFC appears to remain underdeveloped well into adolescence (Nelson et al., 2002; Lewis, 1997; McGlashan & Hoffman, 2000).

As noted above, increased levels of stress hormones damage the hippocampus. Stress-related damage to hippocampal neurons results in impaired learning and memory for both verbal and spatial information (Edwards, Harkins, Wright, & Menn, 1990; Sapolsky, 2000; Sapolsky et al., 1990; Simantov et al., 1996; Smythies, 1997).

Problem 1: Summary and Conclusion

The combined data from neurobiological, neurophysiological, neurochemical, and neuropsychological research on the long-term effects of early adversity show, quite conclusively, that discrete brain regions are affected. Those brain regions include the hippocampus and the prefrontal cortex. Cognitive functioning dependent on the latter includes impulse control and risk-taking in decision making, working memory capacity, strategy use in problem-solving, information-processing biases and inhibitory control for positive and negative stimuli,

impulsivity and decision-making, response inhibition, and judgement. Cognitive functioning dependent on the former involves new learning, spatial cognition, and episodic memory.

Therefore, in order to overcome the problem of not using theory in the choice of test batteries, studies in this field should employ neuropsychological tests that tap into those cognitive processes subserved by the discrete brain regions identified above. Furthermore, neuropsychological data focused on those brain regions are required for populations, such as adolescents with a history of trauma exposure, that have been relatively understudied by researchers.

Problem 2: Lack of Appropriate Control Groups

As noted earlier, this methodological problem, although simply remedied, is one that if not taken into account leads to major problems in arriving at easily interpretable and possibly conclusive results. It also raises the very important issue of differentiating between the neuropsychological effects of trauma exposure and the neuropsychological effects of psychopathological post-traumatic reactions, such as occur in PTSD.

As discussed, not all victims of trauma develop PTSD; mere exposure to trauma is, therefore not the same as actually having a PTSD diagnosis. A diagnosis of PTSD brings with it a range of behavioural, emotional, and, as highlighted earlier, significant neuropsychological and neurological abnormalities (Beckman et al., 1998; Bell et al., 2002; Bremner et al., 2005; Maguire, Burgess, & O'Keefe, 1999). It is thus of most importance that researchers attempting to explore the effects of PTSD on cognitive functioning, include three groups in their studies. These are, namely: a trauma-exposed non-PTSD group, a trauma-exposed PTSD-present group, and lastly, a healthy control group. In so doing, researchers can clearly differentiate between the effects of exposure to trauma as opposed to the effects of the post-traumatic reactions (i.e., the symptoms of PTSD). If these three groups are not included, the results of the studies cannot say whether it is the trauma itself which is responsible for significant deficits in performance, or whether it is the presence of a PTSD diagnosis that is responsible.

In order to illustrate this point more clearly, a brief discussion of two studies that did not include these groups is looked at. One study (by Moradi, Taghavi Neshat-Doost, Yule, & Dalgleish, 1999) assessed performance on the Stroop colour-naming task in 23 children and adolescents with PTSD, and compared their performance to a group of 23 demographically-matched healthy controls. These controls had no history of trauma exposure. The results showed that performance on this task was significantly contingent on having a PTSD diagnosis. The fact that there was no trauma-exposed non-PTSD group means that the deficits seen could have been due to the exposure to trauma, and not PTSD. Similarly, in an adult study on the effects of PTSD on cognitive functioning, Twamley, Allard, et al. (2009) assessed 75 women on comprehensive neuropsychological test battery. Of these women, 55 had a diagnosis of PTSD (related in to intimate partner violence), and their performance was compared to a group of 20 demographically-matched healthy women, none of whom had experienced a traumatic event. The results showed that the PTSD group performed significantly worse on speeded tasks compared to the controls. Once again, the fact that there was no trauma-exposed non-PTSD group means that the poor performance exhibited on these tasks could have been due to the exposure to trauma, and not a diagnosis of PTSD.

Problem 2: Summary and Conclusion. Research on PTSD has shown that this disorder brings with it its own set of neurological and neuropsychological consequences. Thus, in order to assess the effects of trauma on functioning, studies need to incorporate three groups (trauma-exposed PTSD, trauma-exposed non-PTSD, non-traumatized non-PTSD controls). If this is done the cognitive effects due to trauma exposure itself and those due solely to a PTSD diagnosis can be clearly assessed.

Problem 3: Not Assessing both Traumatic Event Characteristics and Post-traumatic Reactions

As noted earlier, this methodological problem, although easy to remedy, is vital in the understanding of how factors in the experience of trauma, impact on the individual, and on their cognitive abilities. Most studies in the field of trauma tend to look solely at the one group of factors in the assessment of a traumatic event, either they look at the characteristics of the trauma (such as the type of event, whether the perpetrator was known to the victim or not, and rate of

occurrence), or they look at the trauma victims' reactions to the trauma (such as the amount of PTSD symptoms they experience, the severity of the symptoms they experience, etc.). Most studies in this field fail to look at both groups of factors in their assessment of the effects of trauma.

The few studies that have assessed both groups of factors in their assessment have shown that both the trauma characteristics and the post-traumatic responses provide important information, and both make for significant predictors of future outcomes.

Moore and Zoellner (2007) clearly showed, in their review of 24 studies looking at the effects of trauma on autobiographical memory, that many studies in this field found that the characteristics of the trauma were significant predictors of outcome measures (Henderson, Hargreaves, Gregory & Williams, 2002; Nilsson-Ihrfelt, et al., 2004; Raes, Hermans, Williams, Eelen, 2005).

Similarly many of the studies that assessed post-traumatic responses found that it was these responses that were most significant in predicting cognitive deficits (Stokes, Dritschel, & Bekerain, 2004).

Problem 3: Summary and Conclusion

This review highlights that it necessary to include both the characteristics of the trauma as well as post-traumatic responses in any study on the effects of trauma. In so doing a thorough understanding of all possible factors in the effects of trauma and PTSD is thoroughly examined.

Other Specific Areas of Interest in the Current Study

The following section attempts to highlight the nuances present in particular areas of research, areas which are specifically assessed in the current study. This includes a brief exploration into the fields of spatial navigation and information-processing biases (and the effects stress, trauma and PTSD have on these cognitive domains), and is followed by a brief look at sex differences in various cognitive abilities. Furthermore the review of sex differences attempts to highlight how males and females respond to stress differently, and what impact this may have on the cognitive functions explored in the current study

Spatial Navigation

One of the cognitive abilities of particular interest in the current study is spatial navigation. Although spatial navigation has traditionally been considered to be a hippocampus-dependent ability, there are in fact at least two kinds of navigation, and only one of these is dependent on optimal functioning of the hippocampal formation. Researchers have distinguished these two forms of navigation empirically; one involves following a route, almost unconsciously, by using landmarks and knowing the routes that connect them. This form of navigation is typically known as *route following* or *landmark-guided* navigation (Thorndyke & Hayes-Roth, 1982). The second kind of navigation involves a more conscious process of constructing a cognitive map of the environment using knowledge of the spatial relationships between locations in an environment. This form of navigation is typically known as *wayfinding* or *cognitive map-guided* navigation (Maguire, Burgess, & O'Keefe, 1999; O'Keefe & Nadel, 1978).

Empirical research has demonstrated that these two forms of spatial navigation are facilitated by different areas of the brain (Bohbot, Iaria, & Petrides, 2004; Etchamendy & Bohbot 2007; O'Keefe & Nadel, 1978; Rosenbaum, Ziegler, Winocur, Grady, & Moscovitch, 2004). Multiple neuroimaging studies have shown that it is in fact only wayfinding, or cognitive map-guided navigation, that is hippocampus-dependent (Hartley, Maguire, Spiers, & Burgess, 2003; Maguire et al., 1999). For instance, Kumaran and Maguire (2005) compared brain activation patterns while healthy adults completed two different navigation-type tasks in a functional magnetic resonance imaging (fMRI) paradigm. One task involved navigating through a spatial domain (the city in which they lived), whereas the other task involved navigating through a non-spatial domain (their social network). The results showed that the right hemisphere hippocampus was only activated and engaged for spatial domain navigation.

Similarly, Bohbot et al (2004) compared brain activation while healthy adults completed two tasks each requiring the use of a different navigation strategy. These navigation strategies each depended on a different memory system. The spatial strategy involved using multiple landmarks that were available in the virtual environment. The response strategy involved responding to left or right turns starting from a specific starting point in the virtual environment. The researchers

showed that the response strategy was associated with sustained activity in the caudate nucleus, whereas the spatial strategy was associated with sustained activity in the hippocampus.

In a neuroimaging study particularly relevant to the current concerns, Astur et al. (2006) used an fMRI paradigm to compare the performance of individuals with and without PTSD on a spatial navigation task. Results showed that the hippocampus was indeed activated in this task (Astur et al., 2006). Although there were no significant between-group differences (i.e. the PTSD and the control group performed statistically similarly), it was demonstrated that degree of hippocampal activation on this task was a good predictor of PTSD severity.

Another line of research that has established the hippocampal-dependent nature of wayfinding relates to studies that have induced acute psychosocial stress in normal healthy adults. Schwabe et al. (2007) compared the performance of 88 healthy participants on a task that involved learning particular kinds of strategies in order to successfully navigate in a virtual environment. Half the participants were exposed to an acute psychosocial stressor (the Trier Social Stress Test (TSST); Kirschbaum, Pirke, & Hellhammer, 1993) and the other half were not. The results showed that stressed individuals used significantly different strategies for learning; they relied less on spatial (hippocampal-based) strategies, and more on stimulus-response (non-hippocampal) strategies. The authors interpreted these data as indicating that the hippocampus is disrupted by acute stress and therefore could not be used in order to navigate through the space successfully.

Similarly, Thomas, Laurance, Nadel, and Jacobs (2010) compared the spatial navigation performance of stressed individuals (with stress once again induced using the TSST) to non-stressed individuals. They demonstrated that acute stress disrupted cognitive map-guided navigation, but did not disrupt landmark-guided navigation.

Information-Processing Biases

Another area of cognition that is of interest in the current research is that of information-processing bias. This term refers to the possible biases people exhibit when processing information. For instance, one individual may remember information that is considered negative

or sad far better than they remember positive or happy information, whereas another individual may remember positive information far better than negative information. Similarly, when attempting to maintain attention, one individual may be far more distracted by the face of someone who is angry than by someone who is happy, whereas another individual may be more distracted by a sad face. In this study, information-processing bias is examined specifically relative to emotional stimuli (such as happy or sad faces or positive or negative words). Emotional information-processing bias is a form of response inhibition for emotional stimuli, and is dependent on the functioning of the PFC.

Functional MRI studies have suggested that these biases are due to the increased neural firing caused by certain stimuli (such as happy or positive stimuli) and not by others (such as sad or negative stimuli) (Leppanen, 2006). More specifically, in one study, attending to certain emotional stimuli was associated with enhanced neural response in two medial PFC regions: the subgenual cingulate (when attending to either positive or negative stimuli) and the ventral anterior cingulate (when attending to positive stimuli only; Elliott, Rubinsztein, Sahakian, & Dolan, 2000).

A large body of research has shown that specific psychiatric diagnoses are associated with specific patterns of information-processing biases. That is to say, people with certain disorders, such as PTSD, show a specific bias towards certain types of information (such as threatening stimuli), compared to matched healthy controls (who usually show a bias towards positive stimuli and away from threatening stimuli; Dalgleish, Moradi, Taghavi, Neshat-Doost, & Yule, 2001; Moradi, Taghavi, et al., 1999). The differences exhibited are seen across various cognitive processes (such as on tasks of memory, attention, etc.) and across multiple forms of stimuli (such as words, pictures, faces, etc.). Studies in this field look specifically at the effects that different emotional conditions (such as happy stimuli or sad stimuli) have on information processing, thereby assessing possible biases for emotional conditions.

Specifically, empirical studies have shown that, when presented with various emotionally-laden stimuli, depressed children, adolescents, and adults perform significantly differently to those with PTSD and to healthy subjects (Dalgleish et al., 2003). Even more specifically, people with a

diagnosis of major depressive disorder show a greater bias in favour of negative or depression-related stimuli (such as sad faces); that is, they show greater accuracy for remembering and noticing these kinds of faces compared to positive or neutral stimuli (such as happy or neutral faces). (For a comprehensive review of this literature see Williams, Watts, MacLeod, & Mathews, 1997). Similarly, people with a diagnosis of PTSD generally show a greater bias in favour of threat-related stimuli (such as angry words) (Dalgleish et al., 2001; Moradi, Taghavi, et al., 1999).

In the study by Dalgleish et al. (2001), the performance of 24 children with PTSD was compared to that of 24 healthy controls on a task of visual attention for emotional material. The stimulus material consisted of 48 words, a third of which were related to physical threat (such as “explosion”), a third of which were related to social threat (such as “rejected”), and a third of which were related to depression-related words (such as “sad”). The results showed that the children and adolescents with PTSD exhibited a bias toward social threat words (that is, they took quicker to notice these words and spent longer attending to these words), as well as bias away from (i.e., an avoidance of) depression-related words (that is, they took longer to notice these words and spent less time attending to these words).

The interpretation of data from these studies is not clear-cut, however, as one has to consider the effects of age and co-morbidity of disorders on these biases (see, e.g., Neshat-Doost et al., 2003). For instance, some studies have found that age has a significant effect on biases exhibited (where older children exhibited a less pronounced set of biases than younger children; Dalgleish et al., 2001). Other studies have found that comorbid disorders tend to confound findings (where biases presented could be due to the presence of another disorder) (Neshat-Doost et al., 2003).

Recent studies have shown that tasks that employ emotional faces as stimuli provide more consistent patterns of bias, particularly with regard to the biases exhibited by children with anxiety disorders (Heim-Dreger, Kohlmann, Eschenbeck, & Burkhardt, 2006; Waters, Mogg, Bradley, & Pine, 2008). Of particular importance for the current study is that empirical research has shown that PTSD patients exhibit a bias for threat-related faces (e.g., those showing angry and fearful expressions), that is they tend to respond quicker to threat-related faces, and are more accurate on recognizing these faces. Moreover, if these faces act as distractors in a task, PTSD

patients will perform worse on the task (i.e. be less accurate) than if the distractors are happy faces (Ladouceur et al., 2006).

In order to understand the biases exhibited by individuals with PTSD, it is crucial to understand the biases exhibited by normal health controls. As mentioned earlier, empirical research has shown that healthy controls show a reliable and statistically significant bias for happy faces. That is, on tasks where individuals are asked to attend to faces, and press a key when they see a certain emotion, healthy controls show faster response times for happy faces (as opposed to other emotions) and they make more mistakes when attempting to inhibit a response to happy faces (Schultz et al., 2007). Furthermore, they respond slower to neutral faces when these are set amongst happy faces as opposed to when these neutral faces are set amongst threat-related faces (i.e., angry or scared faces.)

Studies done on patients with anxiety disorders have found that these individuals respond in a significantly different way to controls, exhibiting a specific and different set of biases. For instance, Ladouceur et al. (2006) found that a clinical group of individuals who were diagnosed with at least one anxiety disorder were biased toward threat-related distractors. This means they responded more slowly, on a task that asked them to attend to neutral faces which were set amongst distractor faces of displaying emotions. The participants responded slower to neutral faces when the neutral faces were set amongst threat-related distractor faces as opposed to when the neutral faces were set amongst happy distractor faces. In other words, the participants showed a bias toward being drawn toward the threat-related distractors.

Of further interest here are findings of sex differences in these information-processing bias studies. Studies with children and adolescents that have included biological sex as a factor in their design and/or statistical analyses have shown that girls tend to be more prone to these biases; boys respond in a statistically similar manner to each other, regardless of diagnoses or lack of diagnosis (see, e.g., Waters & Valvoi, 2009).

Sex Differences in Cognitive Performance, Spatial Navigation, and Stress.

There is great debate over whether there are actual, clinically significant differences in general cognitive performance across the sexes (see, e.g., Baenninger & Newcombe, 1995)). However, empirical research has generally shown that there are some marked differences in certain domains, with males, in general, performing better on tests of visuospatial ability and females, in general, performing better on tests of verbal ability (see, e.g., Hyde, Fennerma, & Lamon, 1990; Johnson & Bouchard, 2007).

Of importance for the current research are the sex differences seen in the human response to stress. Both animal and human studies have shown that there are distinct differences in the way males and females respond to environmental stress. Even though both males and females show an increase in cortisol levels when they experience a stressor, Kudielka, Buske-Kirschbaum, Hellhammer, and Kirschbaum (2004) showed that, when exposed to a stressful task, females had a greater increase in heart rate than their male counterparts.

The neurological mechanisms that underpin these sex differences are not understood fully. However, more and more empirical research has suggested that cognitive and motor skills and the response to stress are, at least partially, mediated by the effects of circulating sex hormones on the brain, particularly the organisational or activational effects of oestrogen (Hampson, Finestone, & Levy, 2005; Kimura, 2004; Kimura & Hampson, 1994). With regard to the sex differences seen in the response to stress, the effects of these sex hormones on the HPA axis is of most importance (Kudielka & Kirschbaum, 2005). Some recent studies have found that there are significant differences between the sexes in terms of the patterns of hormonal response to activation of the HPA axis (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kudielka, Buske-Kirschbaum, et al., 2004). Although there have been some contradictory findings, most studies have found higher cortisol responses in men than in women after acute real world psychological stress as well as stress induced in controlled laboratory settings (such as Stroud, Salovey, & Epel, 2002; for a review see Kudielka & Kirschbaum, 2005). Furthermore, some research has shown that this stress exposure facilitates fear conditioning in males and inhibits fear conditioning in females (Jackson, Payne, Nadel, & Jacobs, 2005).

Not surprisingly, empirical studies have shown that these sex differences in neurophysiological and neurochemical responses to environmental stress are associated with sex differences in cognitive performance following stress. Wolf, Schommer, Hellhammer, McEwan, & Kirschbaum. (2001) showed that, although both males and females exhibited an increase in cortisol following exposure to the TSST, this increase in cortisol led to the recall of fewer words that has been previously learned *only* in males; no such effect was seen in females. In other words, in this study females were not as vulnerable to the effects of stress on verbal learning and memory performance as males were. However, the effects of stress across the sexes in the field of spatial navigation provides evidence of the opposite pattern.

Indeed, the research on sex difference in spatial navigation provides some interesting results. Although animal research has also shown that female rats are more resistant to stress-induced impairment on tests of spatial abilities than males (Bowman, 2005), human research has shown the opposite pattern. For instance, Thomas et al. (2010) found that, after TSST exposure, spatial navigation (specifically cognitive map-based navigation) was impaired by stress in females only; male performance on a cognitive map-guided navigation task was not affected by stress. The same effect was not seen in landmark-guided navigation (*viz.*, neither male nor female performance was affected on that task), suggesting it was the cognitive map-building abilities (*i.e.* the hippocampus-dependent abilities) of females that were affected.

Conclusion

Although research into the neuropsychology of childhood trauma and PTSD is burdened with inconclusive and conflicting results, overall, the research on cognitive functioning in PTSD provides some interesting findings. These findings include that both hippocampus- and PFC-dependent tasks are particularly affected by trauma and PTSD, with widespread deficits exhibited in domains ranging from response inhibition and information-processing bias to working memory and spatial navigation. Although these deficits may be subtle and thus difficult to detect using standard clinical neuropsychological tests, the effects of these deficits may still be significantly disruptive to the adolescent victim of childhood trauma (Leskin & White, 2007). The subtlety of these deficits adds to the rationale for careful choices in test batteries (choosing tests based on carefully formulated theory), and the use of multiple comparison groups.

Furthermore, although the current study does not attempt to resolve the debate between the theoretical positions on vulnerability, it is hoped that the data gathered from this study might inform the debate, and that this study will set the stage for in-depth neuroimaging investigations of adolescents with a history of trauma exposure and, perhaps, for longitudinal investigations of children and adolescents from populations at high risk for trauma exposure.

Specific Objectives and Hypotheses

The preceding review highlights the fact that research in this field is fraught with methodological flaws, unanswered questions, and conflicting results. The current study attempts to remedy these methodological problems, thereby answering some unanswered questions, and providing clear information to help clarify previously conflicting results.

To remedy the methodological flaws inherent in the extant literature, I used sound theory to inform and direct test choice; included three groups in the study (a trauma-exposed-PTSD group, a trauma-exposed-non-PTSD group, and a healthy control group); and measured both the characteristics of the trauma (e.g., how long ago the trauma occurred, the type of trauma experienced) as well as the individual's response to the trauma (e.g., symptoms experienced, rate of occurrence of symptoms, etc.).

By remedying the methodological flaws in this way, this study attempted to address the unanswered questions by measuring specific cognitive processes, focussing specifically on the effects the experience of childhood trauma, and the effects PTSD, has on these processes in adolescents. By answering these questions, I thereby hope to provide some clarity regarding previously equivocal results. In so doing, I attempted to describe (a) the neuropsychological deficits exhibited by adolescents who have experienced childhood trauma, and (b) the distinction, if any, between the neuropsychological profile of adolescents with a trauma history but no PTSD diagnosis and those with a trauma history and a PTSD diagnosis. Furthermore, this study attempted to provide an indicator of test performance that can be used to classify victims of trauma and maltreatment from a group of typically-developing adolescents with no trauma history.

The following specific hypotheses, all of which emerged from the literature reviewed above, were tested:

1. On the general neuropsychological test battery:
 - a. adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls on hippocampal-dependent and PFC-dependent cognitive tasks, and
 - b. of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly on the test battery than will those without such a diagnosis.
2. On the specific test of Spatial navigation:
 - a. adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls on hippocampal-dependent cognitive tasks, and
 - b. of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly on the test battery than will those without such a diagnosis.
3. On the specific test of information-processing bias, adolescents with a diagnosis of PTSD will show a bias toward threat-related stimuli (i.e., they will show faster reaction times on tasks where such stimuli are targets, but will show slower reaction times on tasks where such stimuli are distracters); participants in the control group, in contrast, will show a bias toward positive-valence stimuli (i.e., they will show faster reaction times on tasks where such stimuli are targets, but will show slower reaction times on tasks where such stimuli are distracters).
4. In the group of adolescents who have experienced childhood trauma, poorer performance on the general neuropsychological test battery will be positively correlated with (a) a longer time since the traumatic experience (Olf, Langeland, & Gersons, 2005); (b) a greater number of post-traumatic symptoms (e.g., more avoidance, more numbing, more hyperarousal, more hypervigilance); (c) a higher level of symptom severity; and (d) a greater level of overall functional impairment.

Furthermore, on many of the tests, there will be marked sex differences in the effects that trauma exposure and PTSD has on performance. (For example, on tasks of spatial navigation,

females in the childhood trauma group (both PTSD and non-PTSD) will perform more poorly than both male and female controls. Further, girls in the PTSD group will show the poorest performance, whereas a group membership effect will not be seen in males. Otherwise stated, relative performance on the spatial navigation tasks might be captured as PTSD girls < trauma girls < control girls = PTSD boys = trauma boys = control boys.)

University of Cape Town

DESIGN AND METHODS

Participants

The sample consisted of 49 adolescents between the ages of 12 and 16 years. Thirty-two of these participants had been exposed to at least one traumatic event (e.g., sexual abuse, emotional, physical, or verbal abuse, general maltreatment, neglect, the witnessing of violence) during childhood (including adolescence). One of the inclusion criteria for the study was that this trauma could have taken place anytime during the adolescent's life, but had to have occurred at least 3 months prior to testing.

The issue of defining what constitutes exposure to traumatic events (and indeed, what constitutes a traumatic event) is particularly important for research studies of this kind. In this study, I define a "traumatic event" using the definition of a Criterion A event given in the Diagnostic and Statistical Manual of Mental Disorders (4th edition, text revision (DSM-IV-TR); American Psychiatric Association, 2000). Both Criterion A1 (individual must have experienced, witnessed, or been confronted by the threat of death or serious injury, or a threat to the physical integrity, of oneself or others) and Criterion A2 (during the event, the individual must have experienced intense fear, helplessness, or horror) were assessed, although the latter is not necessary for categorization of the event as a trauma.

The 32 participants with a history of traumatic exposure were recruited using two different strategies. The first strategy was recruitment via treatment centres. Thirteen of the participants were recruited from two different centres, namely, the Rape Crisis Centre and the Trauma Centre, both based in the Western Cape. The second strategy for recruitment involved working with local schools (a boys school, and a co-ed school) in the Western Cape. These strategies are detailed in the Procedure section.

My recruitment and screening procedures ensured that, of the 32 participants with a history of traumatic exposure, 16 had a current diagnosis of PTSD and 16 did not. Given the base rate of PTSD in the South African adolescent population, I had no difficulty recruiting the 16 adolescents with this diagnosis (Carey, Stein, Zungu-Dirwayi, & Seedat, 2003; Seedat, Nyamai,

Njenga, Vythilingum, & Stein, 2004). The traumatic events experienced by the participants in the final sample are presented in Figure 1.

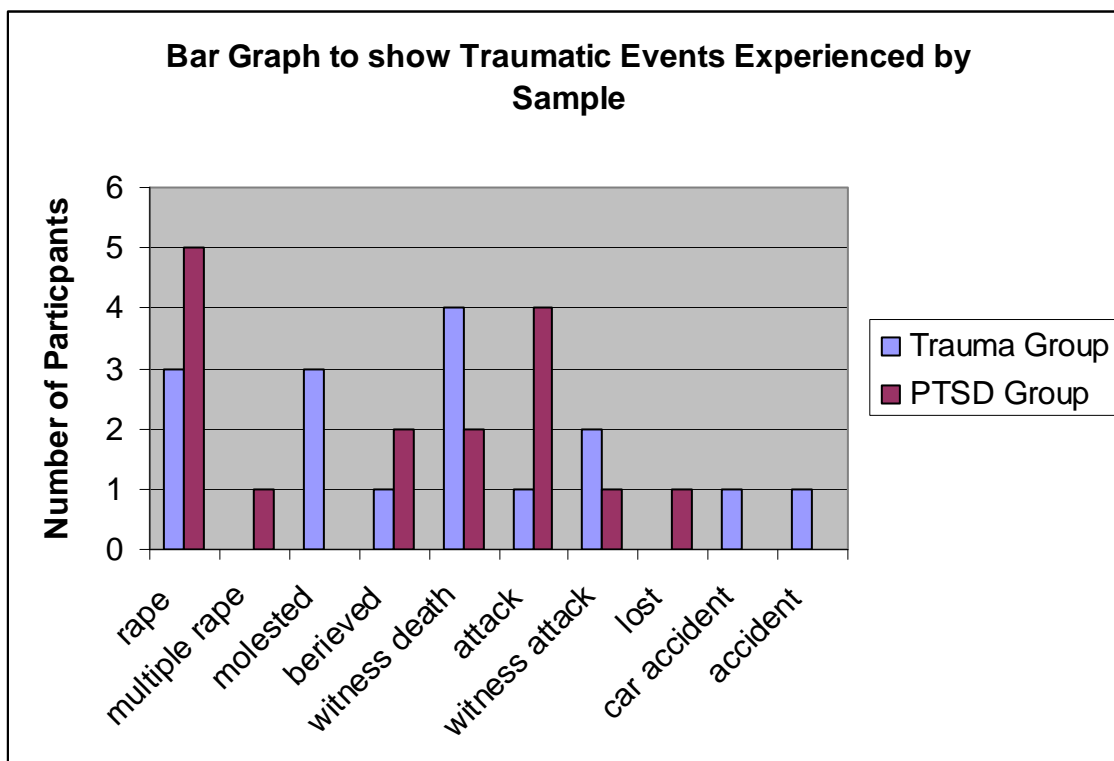


Figure 1. Traumatic events experienced by participants in final sample.

I recruited a control group ($n = 17$) consisting of adolescents with no history of childhood trauma exposure. These participants were recruited from the local public schools (a boys school and a co-ed school). Ethics clearance for doing research in these schools was obtained from the Western Cape Education Department.

Groups were matched, as far as possible, on the following socio-demographic variables: age, socio-economic status, years of education, race, sex, and home language. Multiple exclusion criteria were applied to all groups, including: a history, or a current diagnosis, of any psychotic disorders, a history or current diagnosis of major depressive disorder or dysthymia (for participants in the healthy control group), a history of head injury or any other neurological problem, and a history of alcohol and substance abuse. These exclusion criteria were employed because each may affect brain structure and function and thus confound results (Kyte, Goodyer,

& Sahakian, 2005; Smith, 2005). It is important to note that due the high co-morbidity rate of anxiety disorders with PTSD, co-morbid anxiety disorders were not included as exclusion criteria. The flow chart in Figure 2. illustrates how the final sample was determined.

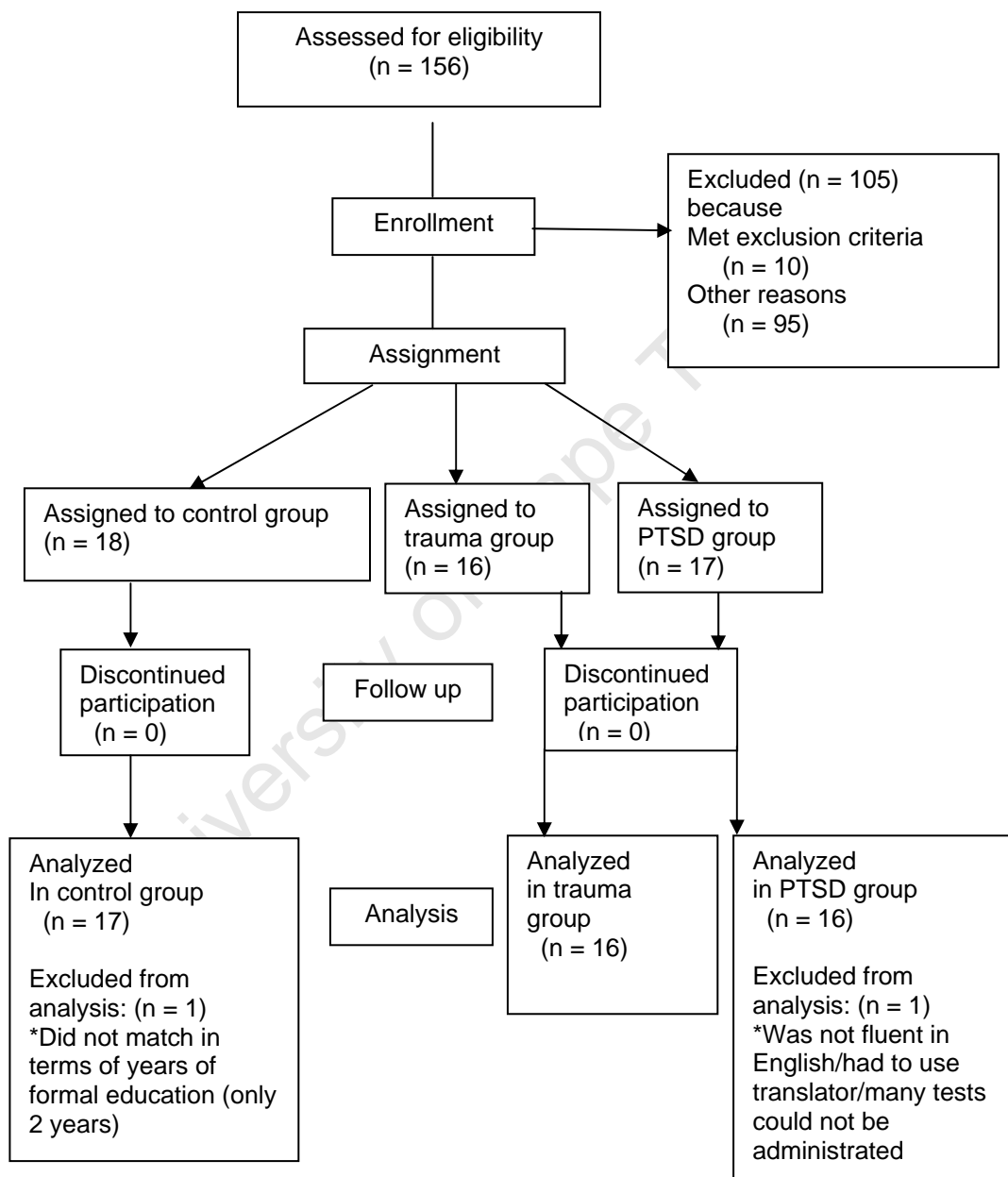


Figure 2. Flow chart of final sample

Note. In the 105 participants excluded from enrollment in the study, 95 were excluded for ‘other reasons’. The reasons for these exclusions were, firstly, time constraints with regard to the amount of time each school allowed for testing, and secondly, the need to have approximately the same number of participants in each group, therefore some people could not be enrolled as they were already too many in the group they would belong to.

Materials and Apparatus

Participants were administered a clinical interview, several paper-and-pencil questionnaires, and a battery of neuropsychological tests. The clinical interview was designed to assess whether the participant was currently experiencing, or had in the past experienced, any psychiatric disorders, including PTSD, depression, and substance abuse. The questionnaires were used to gather details regarding the childhood trauma and the participant's response to it, as well as to assess the participant's current emotional state and aspects of his/her personality. The neuropsychological test battery assessed general intellectual functioning and cognitive functions subserved primarily by either the hippocampus or the prefrontal cortex. Each of the instruments used in this study is detailed below.

Demographic Questionnaire

A self-report questionnaire created specifically for this study assessed demographic variables of interest (e.g., age, socioeconomic status (SES), and years of education). These variables were used in order to match the participants in the PTSD group with participants in the trauma and control groups. The questionnaire is presented in Appendix A.

Screening Questionnaire

This questionnaire was also created specifically for this study. The purpose of this instrument was to assess potential participants for inclusion and exclusion criteria for the current study, as well as to ascertain which group the adolescent might be suitable for (control, trauma or PTSD). It therefore assessed various important demographic variables (e.g., age, sex, home-language, race), and asked about history of and current alcohol and drug use and abuse (as noted above, such use/abuse was an important exclusion criterion). Lastly, and most importantly, the questionnaire included an assessment of trauma exposure and symptoms experienced following the traumatic experience. This part of the questionnaire included all of the items from the PTSD section of the MINI KID 5.0 (Sheehan et al., 1998). By exploring trauma exposure and a possible PTSD diagnosis, this section of the screening questionnaire allowed the researcher to ascertain which group the potential participant might be suitable for. Further rationale for the use of this questionnaire is presented in Appendix C. The actual questionnaire is presented in Appendix B.

Clinical Instruments

Psychiatric Interview. The Mini International Neuropsychiatric Interview For Children and Adolescents, Version 5 (MINI KID 5.0; Sheehan et al., 1998) is a comprehensive, abbreviated structured psychiatric interview that takes approximately 25 minutes to administer. It assesses the major DSM-IV-TR Axis I disorders including depression, substance abuse, and PTSD. This is based on the adult version of the MINI (Sheehan et al., 1998). Numerous psychometric studies have established that this interview is a reliable and sensitive as a measure of psychopathology, with good predictive value. For instance, a large-scale study conducted by Sheehan et al. (1998) found that the MINI produced the same diagnoses as the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1996) 85-95% of the time. Similarly, Sheehan, Sheehan et al. (2010) found that the MINI KID 5.0 was a reliable and valid clinical interview in the diagnosing of disorders in children and adolescents. The MINI has been used extensively in psychological research in South African (see, e.g., Kaminer, Stein, Mbanga, & Zungu-Dirwayi, 2001; Van der Ryst, Strydom, Scott, Boshoff, Joubert, & Els, 2002).

Posttraumatic Stress Diagnostic Scale (PDS). The PDS (Foa, 1995) is a comprehensive questionnaire that assesses PTSD symptomatology in detail. This 48-item questionnaire not only assesses which symptoms are experienced, but also assesses how often these symptoms are experienced. The questionnaire is divided into 4 parts. The first part assesses what traumatic events the participant has experienced. The second part assesses which of these experienced events bothers the participant the most, and asks when that event occurred. It also asks whether the participant was injured as a result of the event, felt helpless or terrified during the event, and whether he/she thought someone's life was in danger during the event. The third part assesses 17 symptoms, each of which is rated on 4-point Likert-type scale ('not at all or only one time', 'once a week or less/once in a while', '2 to 4 times a week/half the time', and '5 or more times a week/almost all the time'). This part of the instrument also assesses the amount of time the symptoms have been occurring, as well as how long after the event the symptoms began occurring. Lastly, the fourth part of the questionnaire assesses which areas of the participant's life are affected by these symptoms (e.g., work, relationships with friends and family, fun and leisure activities, and general satisfaction with life). Overall, then, the PDS gives important

information about PTSD-related level of impairment, number of symptoms, and symptom severity.

Childhood Trauma Questionnaire – Short Form (CTQ-SF). The CTQ-SF was developed and validated as a screening measure for histories of maltreatment for both clinical and nonclinical groups (Bernstein et al., 2003). It is a 28-item version of the original 70-item CTQ, which assesses childhood abuse and neglect (Bernstein et al., 1994). Items on the CTQ-SF are rated on a 5-point Likert-type scale. The responses range from ‘never true’ to ‘very often true’. Five types of maltreatment (factors) are measured, each represented by 5 items. The five factors are physical abuse, sexual abuse (sexual contact or conduct between a child and an older adult), emotional abuse, physical neglect, and emotional neglect. The CTQ-SF is scored by adding the raw scores for each factor, thus producing an index of trauma severity ranging from 5 to 25. The questionnaire also includes a 3-item minimization/denial scale that detects underreporting. The CTQ-SF is frequently used in trauma research in South Africa (see, e.g., Lochner et al., 2004).

Positive and Negative Affect Schedule (PANAS). The PANAS (Watson, Clarke, & Tellegen, 1988) is a short questionnaire that measures the participant’s tendencies to experience negative or positive emotions. It involves two 10-item mood scales, one each for positive and negative affect. The PANAS has demonstrated high internal consistency and stability over a 2-month time period (Watson et al., 1988). In the current study, this instrument was used to provide a measure of the approach avoidance/withdrawal tendencies of the participant. The PANAS is widely accepted as a good measure of positive and negative affect, and thus is used internationally and in South African research (see, e.g., Strümpfer, Viviers, & Gouws, 1998; Voogt et al., 2004).

Connor-Davidson Resiliency Scale (CD-RISC). The CD-RISC (Connor & Davidson, 2003) was developed in order to provide a measure of stress-coping ability and an individual’s ability to thrive in the face of adversity (Campbell-Sills & Stein, 2007). It is a 25-item self-report measure, with each item rated on a 5-point Likert-type scale. Each item provides a statement, such as, ‘Good or bad, I believe that most things happen for a reason’ or, ‘I feel in control of my

life'; responses range from 'not true at all' to 'true nearly all the time'. With good internal consistency and construct validity, this scale is widely-used internationally as an efficient measurement of resilience. For instance, a recent study used the CD-RISC to collect resiliency data from individuals in 12 countries, including South Africa (Davidson et al., 2006).

Life Events Questionnaire (LEQ). The LEQ (originally developed by Masten, Neemann, & Andenas, 1994) gives a measure of the perceived everyday stress the participant may be facing. It does not assess traumatic events, but rather assesses minor changes in one's life, or minor events that can lead to a build up of stress experienced by the participant. The original version of the LEQ has been shown to be a reliable and valid measure of perceived everyday stress (Masten, Miliotis, Graham-Bermann, Ramirez, & Neemann, 1993), used both in South Africa and abroad (Masten, Neemann, & Andenas, 1994). In the version of the LEQ used in this study, there were 23 items, each of which the participant had to indicate whether he/she had ever experienced. The participant then had to indicate whether the event occurred in the last 6 months or before that. Furthermore, for each item, the participant had to indicate what impact the event had on them ('no impact', 'some impact', or 'significant impact'). This version of the LEQ is presented in Appendix D.

State-Trait Anxiety Inventory (STAI). This questionnaire (developed by Spielberger, Gorsuch, Lushene, Vagg, and Jacobs, 1983) is an anxiety scale and assesses the participants' generalized anxiety at the present moment and in general. This does not refer to the anxiety felt over a specific traumatic event or events, but the general anxiety the participant may feel day to day about their life as a whole. The STAI consists of two separate parts. The STAI Form Y-1 (STAI 1) measures the individual's anxiety in the specific moment (state anxiety), whereas STAI Form Y-2 (STAI 2) gives a measure of general levels of anxiety (trait anxiety). Each form includes 20 statements, each of which are measured on a Likert-type scale ranging from "not at all" or "almost never" to "very much so" or "almost always". Items are included that are reverse scored so as to reduce response sets. This questionnaire has been shown to have good psychometric properties, with high levels of reliability, internal consistency, and validity (Spielberger & Vagg, 1984).

Neuropsychological Battery

Wechsler Abbreviated Scale of Intelligence (WASI). The WASI (The Psychological Corporation, 1999) is a short, four-subtest version of the Wechsler Adult Intelligence Scale (WAIS). Using the Vocabulary, Similarities, Block Design and Matrix Reasoning subtests, this instrument provides a reliable and valid estimate of WAIS Verbal, Performance and Full Scale IQ scores. The WASI takes approximately 30 minutes to complete and can be used with subjects from 6-89 years. It is used extensively in research that requires an overall IQ measure (see, e.g., Saltzman, Weems, & Carrion, 2006), and is currently being used in South Africa to test local adolescents with traumatic brain injuries (Schrieff & Thomas, 2008).

NEPSY-II Inhibition Subtest. The NEPSY-II (Korkman, Kirk, & Kemp, 2007) is a valid and reliable test battery, shown to be particularly sensitive to subtle deficits in children's neuropsychological performances, and appropriate for children aged 5 to 16 years. The Inhibition subtest assesses the ability to inhibit automatic responses in favour of new responses, as well as the ability to switch between different response types. The subtest is separated into 3 sections, with each section using 2 worksheets: the squares and circles sheet (the shape sheet) and the arrows sheet. In the first section, called 'Naming', the examinee has to look at the shape sheet and simply state whether each shape is a circle or a square. Then, looking at the arrows sheet, the examinee must simply state whether each arrow is pointing up or down. In the second section of the subtest, called 'Inhibition', the examinee must look at the shape sheet and each time he/she sees a circle say "square" and vice-versa.. Then, looking at the arrows sheet, the examinee must look at each arrow and say "up" if the arrow is pointing down, and must say "down" if the arrow is pointing up (i.e. say the opposite direction of the arrow). In the third section of the subtest, called 'Switching', the examinee has to look at the shape sheet and must say the correct name for the shape when its colour is black, but must say the wrong name (i.e. "square" if it is a circle, "circle" if it is a square) if its colour is white. Then, looking at the arrows sheet, the examinee must say the correct direction of the arrow when it is coloured black, but must say the opposite direction when it is white.

Children's Memory Scale (CMS). The CMS (Cohen, 1997) is an extensive neuropsychological battery that provides measures of different aspects of memory for individuals

aged 5 to 16 years. It is currently being successfully used in a study testing local children and adolescents with traumatic brain injuries (Schrieff & Thomas, 2008). In the current study, only the *Word Pairs* and *Digit Span* subtests were used. Each of which is detailed below.

Word Pairs measures immediate and delayed recall and recognition for verbal information (cognitive processes subserved by the hippocampus). In this subtest the administrator reads a list of fourteen word pairs, (for e.g. “football, pencil”; “nurse, fire”; “leaf, school”) to the examinee. The administrator then says one word from each pair, and the examinee is required to say which word went in that word pair. If the examinee gives the wrong answer, the administrator repeats the correct word pair. This procedure is done three times. The examinee is then asked to recall as many of the fourteen word pairs as they can without the administrator giving the first word. After a 30 minute delay, the examinee is asked once again to recall as many of the fourteen word pairs as they can without the administrator giving the first word. This is followed with the administrator reading a list of forty-two word pairs, and after each pair the examinee must say if they recognize this pair as one of the original fourteen or not.

Digit Span measures working memory (a cognitive process subserved by the prefrontal cortex). In the *Forward* subtest the administrator reads a series of numbers to the examinee and they must in turn say the series back to the administrator. The series starts with two numbers and systematically increases to nine numbers, with two trials for each series. The task is discontinued if they examinee gets both trials wrong for a series. In the *Backward* subtest the procedure is exactly the same, except the examinee has to repeat the series in the reverse order to what the administrator read.

Cambridge Neuropsychological Test Automated Battery (CANTAB). The computerized neuropsychological tests in the CANTAB (Fray, Robbins, & Sahakian, 1996) are currently used in more than 50 countries, with a bibliography of over 500 peer-reviewed journal papers. These extensively validated tests allow for quick and accurate assessment of many cognitive domains, with excellent sensitivity. Furthermore, as no reading ability or verbal responses are required, the CANTAB tests are well suited for cross-cultural administration and for the assessment of young children (Cambridge Cognition, 2006). All CANTAB tests are administered on a touch-screen

computer, and are presented in the form of games that the examinee must complete. Most importantly, the battery was developed with the aid of functional neuroimaging techniques; therefore, the neuroanatomical regions associated with performance on each subtest within the battery have been well defined.

The CANTAB battery used in the current study consisted of six tests assessing prefrontal cortex functioning: the Spatial Span (SSP) test (both forwards and backwards), the Stockings of Cambridge (SOC) test, the Information Sampling Task (IST), the Intra/Extradimensional Set Shift (IED) test, and the Cambridge Gambling Task (CGT). Details for each of these tests is presented below.

The Spatial Span (SSP) subtest, a measure of working memory capacity, is essentially a visual/spatial digit span test. The computer screen displays 9 white blocks, and these change colour in a random order. In the forwards SSP test, the participant must remember this sequence and then, after a signal (a beep), must touch the blocks in the same order. The testing starts with a 2-block sequence; with every correct trial, the sequence increases in length by 1 block, up to a 9-block sequence. For each sequence length, the examinee is allowed three trials to produce the correct sequence right; if he/she cannot do so within those three trials, the test is discontinued. The backwards SSP test follows the same procedure, except that the examinee must touch the blocks in the reverse order from that presented on the screen. Figure 3 represents a screen shot from the SSP task.

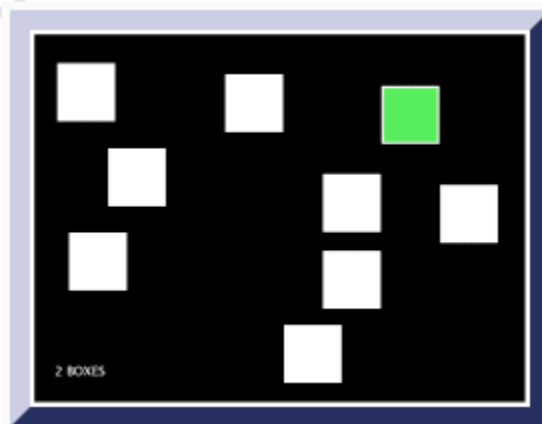


Figure 3. Screen shot of the SSP tasks

The Stockings of Cambridge (SOC) subtest, a measure of problem solving, is essentially similar to the classic Tower of London test (Shallice, 1982). In the SOC, the computer screen is divided into two, with a picture at the top and the bottom of the screen of colourful balls in three stockings hanging from a bar. The examinee has to make the bottom half of the computer screen match the top half of the screen by moving the balls from one stocking to another. In order to move a ball the examinee simply touches the ball they want to move, and then touches the stocking they want to move the ball to. Each of the 20 problems within this subtest can be solved in a particular minimum number of moves; the closer the examinee gets to this number on each problem, the more successful his/her performance is judged to be. Figure 4 represents a screen shot from the SOC task

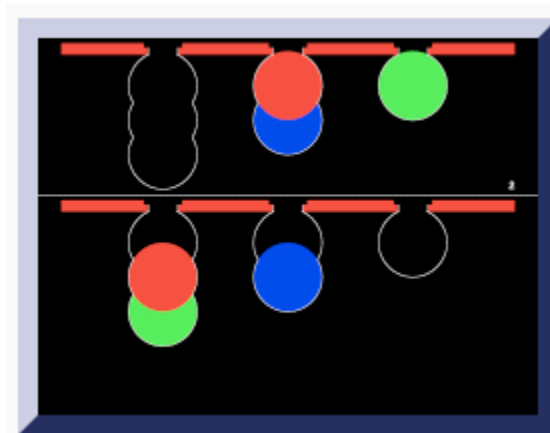


Figure 4. Screen shot of the SOC task

In the *Information Sampling Task (IST)*, a measure of impulsivity and decision making, the examinee is presented with 25 grey boxes on the computer screen. When a box is touched it opens up to show which colour it is (1 of 2 colours, for e.g. pink or blue). Once the examinee has opened as many boxes as he/she wants to, he/she must decide which colour there are more of, out of all the 25 boxes. The examinee is given 100 credits to start with. In the fixed-winnings condition, a correct guess yields 100 credits, regardless of how many boxes have been opened; an incorrect guess, in contrast, loses 100 credits. In the decreasing-winnings condition, the more boxes the examinee opens the fewer credits he/she can win. A wrong guess, however, still leads to a loss of 100 points, no matter how many boxes have been opened. Each condition consists of 10 trials. Figure 5 represents a screen shot from the IST task

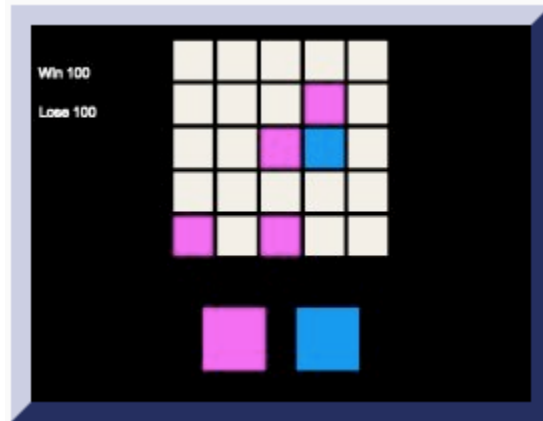


Figure 5. Screen shot of the IST task

In the *Intra-Extra Dimensional Set Shift (IED)* subtest, a measure of rule acquisition and attentional set-shifting, the computer screen displays 4 boxes. Two of these boxes have one of two purple patterns in them. The examinee must learn a rule in order to guess which pattern is correct (for example, the square is correct). In the very first trial the participant can only guess, but once he/she has made a decision (by touching a pattern) the software indicates whether the decision was right or wrong. From this information, the examinee can eventually acquire the rule. The rule is changed 8 times, therefore this task involves 9 stages. During stage 3, white lines are introduced into the patterns without warning. The first 7 stages (pre-set-shift stages) are quite simple, as the rule involves changing from the one purple pattern to other. In the last 2 stages (set-shift stages), the rule involves changing from the purple patterns to the white lines, and changing from the one white line to the other. Figure 6 represents a screen shot from the IED task

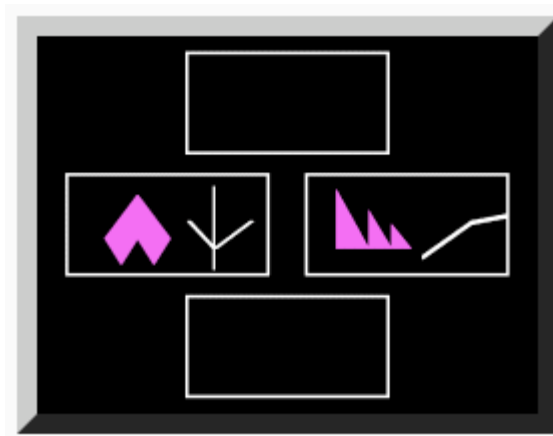


Figure 6. Screen shot of the IED task

In the *Cambridge Gambling Task (CGT)*, a measure of impulse control and risk-taking in decision-making, the computer displays a row of 10 boxes at the top of the screen. Some of these boxes are red and some are blue. The examinee must guess which colour box a yellow token would be in. Once he/she has chosen red or blue (by touching the name of the colour at the bottom of the screen), a series of bets is offered by the computer software. The examinee starts off with a pot of 100 credits ; in the ascending condition the series of bets starts small and then increases (from 5% to 25% to 50 % to 75% to 95% of their pot). The examinee simply touches the number he/she wants to bet. In the descending condition, the series of bets starts large and then decreases (from 95% to 75% to 50% to 25% to 5% of their pot). If the participant loses so much credit that the pot is 1 or less, the stage is stopped and the next stage commences. Each condition has 4 stages, and each stage includes 9 trials. Figure 7 represents a screen shot from the CGT task

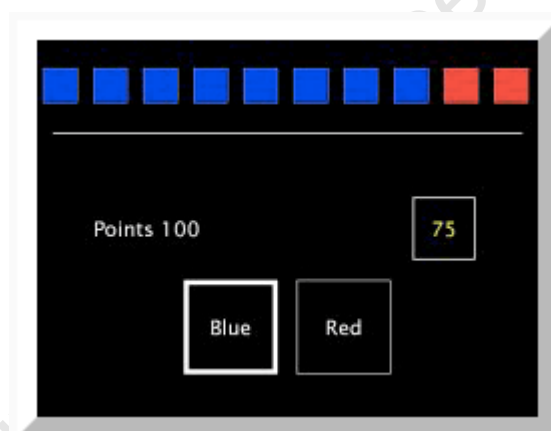


Figure 7. Screen shot of the CGT task.

The current CANTAB battery also included two tests assessing hippocampal-dependent memory and learning: the Paired Associates Learning (PAL) subtest and the Spatial Recognition Memory (SRM) subtest. Details of these tests are presented below.

In the *Paired Associates Learning (PAL)* subtest, a measure of episodic memory and learning, six blocks are immediately presented to the examinee on the computer screen. These then open up one at a time, in a random order, to reveal a pattern inside the box. After all 6 boxes have opened and closed, a pattern is presented in the middle of the closed boxes. The participant must

then touch the box that contained the target pattern. Testing involves 8 stages: the first two feature one target pattern, the third and fourth feature two target patterns, the fifth and sixth feature three target patterns, the seventh features six target patterns, and the last stage features 8 boxes and 8 target patterns. The participant must complete each stage successfully, no matter how many trials it takes. Figure 8 represents a screen shot from the PAL task

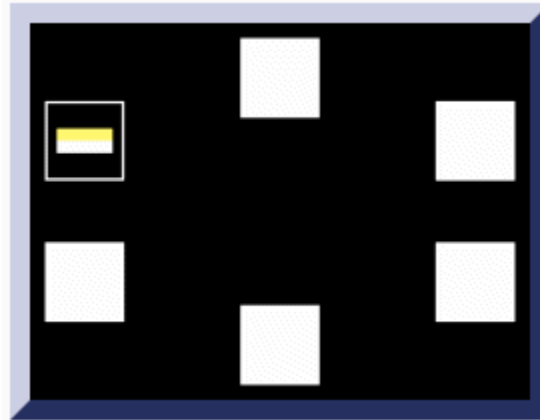


Figure 8. Screen shot of the PAL task

In the *Spatial Recognition Memory (SRM)* task, a measure of spatial recognition memory, the computer screen displays a blank screen, which is followed by the presentation of five blocks, one at a time. After the presentation, a series of two block pairs appears and the examinee must touch the block that is in the same place as one of the five blocks previously shown (five pairs are shown). This task involves four stages; each stage involves the viewing of five blocks which must be remembered, followed by five pairs of blocks. The examinee must recognize which of the pair is in the correct place and touch that block. Figure 9 represents a screen shot from the SRM task



Figure 9. Screen shot of the SRM task

Computer-Generated Arena. The CG Arena is a desktop-based, non-immersive, virtual environment (VE) spatial navigation task (Jacobs, Laurance, & Thomas, 1997; Jacobs, Thomas, Laurance, & Nadel, 1998). Numerous studies have shown the CG Arena tasks to be reliable and valid measures of spatial navigation (see, e.g., Thomas, Hsu, Laurance, Nadel, & Jacobs, 2001). Furthermore, optimal performance on the CG Arena, which requires a wayfinding/cognitive map-guided form of navigation, has been demonstrated to be heavily dependent upon hippocampal functioning (Frakey et al., 2005).

The CG Arena task that the participants performed in the current study involved them viewing, from a first-person perspective, a computer screen that displayed a square room, inside of which was a circular arena. On the walls of the room was a set of pictures that the participant needed to use as landmarks or distal cues for navigation. The participant was instructed to navigate through the room, using a joystick, in order to search for a target located on the arena floor.

Representations of the room can be seen in Figures 9, 10, and 11.

This task was performed under two conditions. In the first series of 5 trials, the target was visible and the participant could find it simply by scanning the environment. In the subsequent series of 8 trials, the target was invisible until the participant found its location and stepped on it. In this condition, the target was always in the same place in the room, meaning that the participant needed to use the distal cues on the walls, and the spatial relationships among them, to construct

a cognitive map of the environment to successfully locate and re-locate the target across trials (Roche, Managaoang, Commins, & O'Mara, 2005; Thomas et al., 2001).

Following this series of invisible target trials, the participants were administered a “probe trial,” where they were instructed to once again search for the invisible target; unbeknownst to them, however, the target was removed from the room on this trial. This trial assessed persistence of search and cognitive mapping ability; those individuals who successfully created a mental map of the environment should persist longer in their search of the target’s former location (Morris, 1984). Figure 10, 11 and 12 represent screen shot of the CG Arena.



Figure 10. Facing the North wall, with the invisible target marked.



Figure 11. Facing the corner of the East wall and the South wall.



Figure 12. Facing the corner of the West wall and the South wall.

Finally, the participant was administered two tasks that are addenda to the CG Arena: the Object Recognition Test (ORT) and the Arena Reconstitution Task (ART).

Object Recognition Task (ORT). The ORT is a forced-choice test assessing recognition memory. Specifically, the test assesses recognition memory for the pictures that were on the

walls of the room in which the invisible target was located. This test is also a computer-based test (developed with E-Prime version 1.0; Psychology Software Tools, 2007). The participant is given the instructions, told to look at the screen, and indicate readiness to proceed by pressing the space bar. A series of eight screens are then displayed. Each screen has two pictures on it. One of these pictures was on the wall in the invisible-target room, and one is a distractor. Each photograph is numbered (as “1” or “2”). The participant must then indicate, by pressing the appropriate number key, which picture is recognized as being one of the landmarks from the invisible-target room. The eight screens that were displayed are shown in Figure 13.

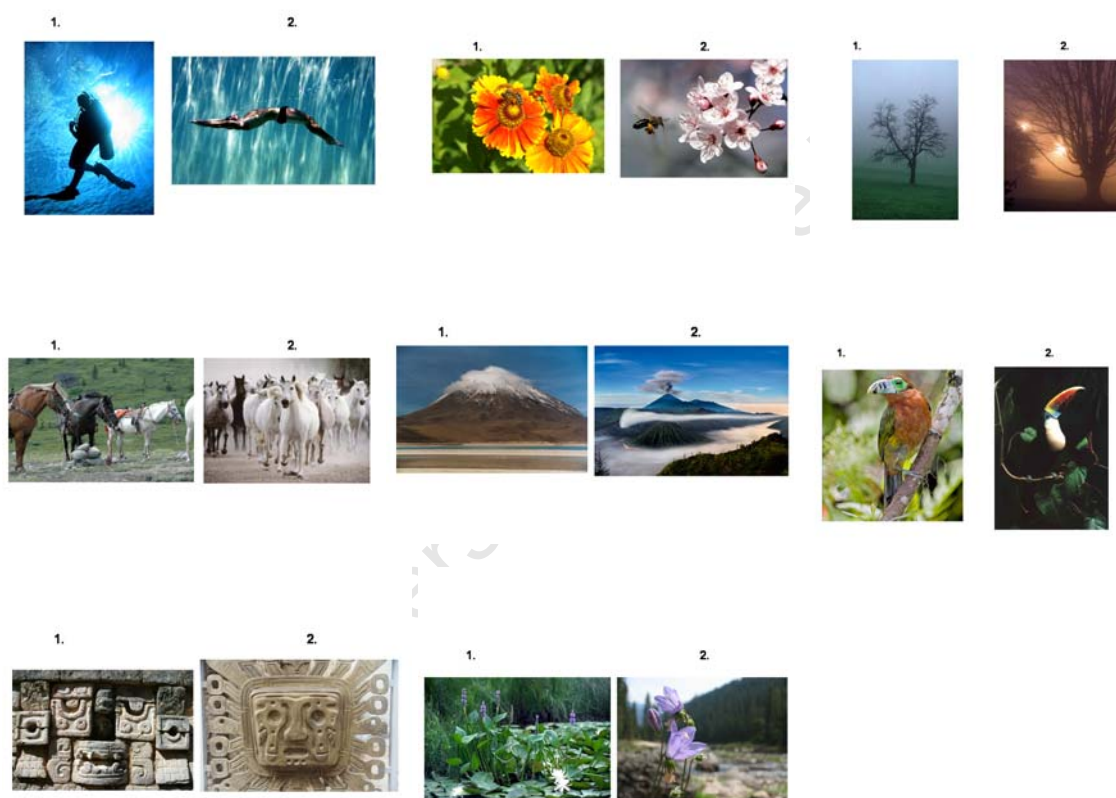


Figure 13. ORT target and distractor pictures (displayed separately as 8 screens)

Arena Reconstitution Task (ART). The ART is a visuoconstructional task that assesses memory for the layout of the distal cues in the CG experimental rooms; it thus measures the quality of the participant’s cognitive map of the CG virtual room. The task thus gives a measure of cognitive mapping ability. The participant is presented with a stimulus sheet (as seen in the

Figure 14) and is given eight small laminated pictures. He/she is then instructed to recreate the layout of the room. When this task is completed, the participant is instructed to mark (by writing an X on the sheet) in which square the invisible target was located, in relation to the pictures he/she placed in the room.

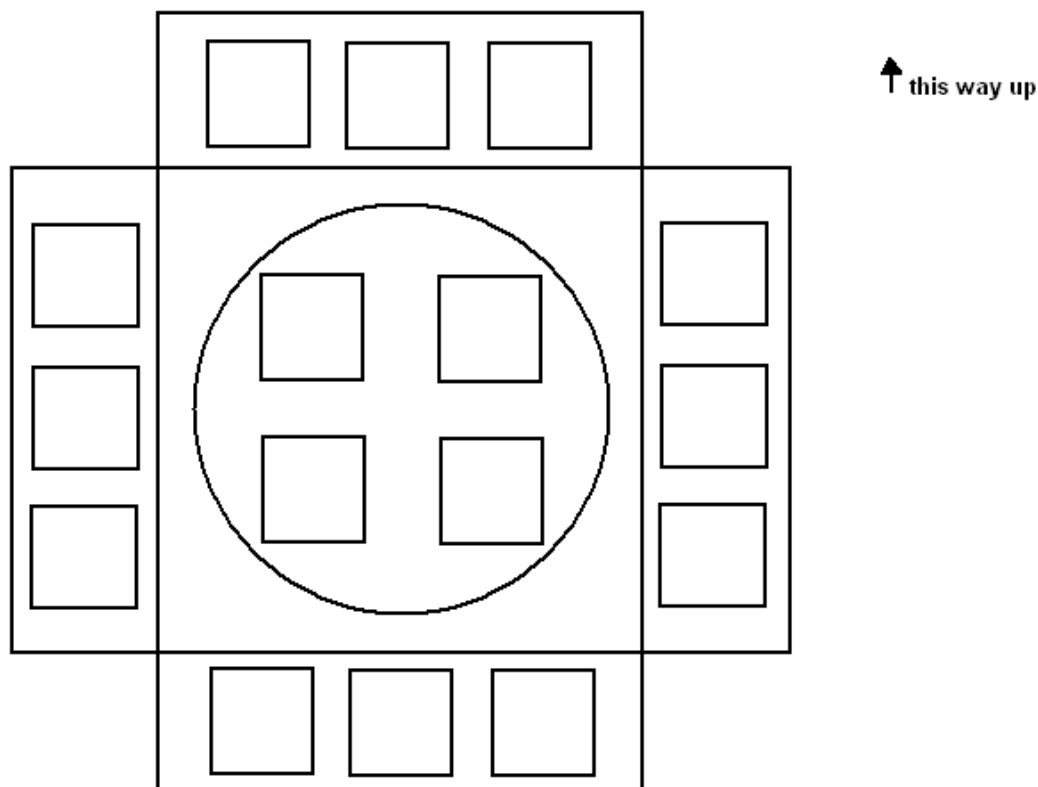


Figure 14. ART Stimulus Sheet

Affective Go/No-go (AGNG) test. The AGNG test used in this study (developed by Ipser, 2008) is a computer-based test, designed to assess response inhibition and affective bias, where the participant is instructed to watch a series of faces, each showing either a happy, fearful, or neutral (where the face shows no emotion) expression. The test consists of three blocks of 64 trials each (with the presentation of one face constituting a trial). Each face is shown for 500 milliseconds, with an interval of 500 milliseconds. At the beginning of each block, the participant is told to look for a particular emotion (the 'target' emotion; this differs from block to block), and to hit the 'g' key on the computer keyboard when it is presented. Participants are also instructed to not hit the key when any of the other emotions (distractor emotions) are presented. Each block then consists of 48 'target' faces and 16 'distractor' faces. In the current study, the

first block's target emotion was 'fearful' and the distractors were 'neutral'. In the second block, the target emotion was 'neutral' and the distractors were 'fearful'. In the third block, the target emotion was 'fearful' and the distractors were 'happy'. AGNG tasks such as this one have been shown to be valid tests of the constructs of response inhibition and affective bias (Schulz et al., 2007; Water & Valvoi, 2009).

Table 1 provides a summary of the specific neurocognitive functions assessed by each test in the neuropsychological test battery employed here.

Table 1
Neuropsychological Test Battery used in the Study

Brain Region/Test	Cognitive Domain Assessed by Test	Example of Study in Which Test is Used
Prefrontal cortex		
Intra/Extradimensional Set Shift (IES)	Rule acquisition and attentional set-shifting	Randall et al. (2004)
Spatial Span (SSP)	Working memory capacity	Sweeney et al. (2000)
Stockings of Cambridge (SOC)	Spatial planning and motor control	Bedard et al. (2004)
Cambridge Gambling Task (CGT)	Impulse control & risk-taking in decision making	Whitlow et al. (2004)
Information Sampling Task (IST)	Impulsivity and decision making	De Luca et al. (2003)
Affective Go/No Go (AGNG)	Information processing biases & inhibitory control	Murphy et al. (2004)
NEPSY-II: Inhibition subtest	Response inhibition	Lehto et al. (2003)
CMS: Digit Span	Working memory	Schrieff & Thomas (2008)
Hippocampus		
Paired Associates Learning (PAL)	Episodic memory and learning	Coull et al. (2006)
Spatial Recognition Memory (SRM)	Spatial recognition memory	Luciana & Nelson (2002)
CG Arena	Spatial navigation	Thomas et al. (2001)
CMS: Word Pairs	Verbal memory	Schrieff & Thomas (2008)

Procedure

As stated earlier, two recruitment strategies were employed. Each strategy involved quite different processes, which insured that all the participants could volunteer for this study in as informed a way as possible, and thus each will be discussed separately.

The first strategy involved recruitment through treatment centres. This recruitment involved working with the counsellors at the clinics to find individuals who would be suitable participants in terms of age, when the traumatic event occurred, emotional stability, etc. The counsellors then telephoned the identified individual, briefly explained what this study was about and asked if the

individual would feel comfortable with a researcher telephoning to discuss the study. The researcher then contacted all the individuals who agreed to accept this telephone call. At the outset of the call, both the adolescent and their parent or guardian were given an explanation of the study procedures. For those who continued to be interested in participation, the researcher arranged with the individual's parent or guardian to meet at a suitable time at the treatment centre where the adolescent had received treatment.

At this meeting, the parent/guardian read through and signed the parent consent form (Appendix E). These consent forms included the primary researcher's contact details, in case the parent or guardian wanted to make contact at a later point. The adolescent was then given the assent form (Appendix F). Once the adolescent had received written informed consent from their parents/guardians, and had signed the assent form, he/she was enrolled in the study. The study protocols were then administered at the treatment centre.

The second strategy for recruitment involved working with the headmaster and class teachers of two schools in the Western Cape. Suitable classes (in terms of appropriate age group) were identified and then addressed as a group. The adolescents were told about the study and given parental consent forms (seen in Appendix G for the co-ed school, and Appendix E for the boys school). Those adolescents who brought these forms back were then administered the screening questionnaire. This questionnaire included an assent form (see Appendix B). From this questionnaire, possible participants were approached and those who assented to take part in the actual interviews were enrolled in the study.

In the course of this initial screening process, 126 adolescents filled out screening questionnaires. Due to time constraints (in terms of the amount of time the schools allowed the researcher to conduct interviews) of these possible candidates, 21 adolescents were enrolled in the study. (The screening process itself provided some important results; these can be seen in Appendix C)

For all participants, regardless of the manner in which they had been recruited, the actual study protocols were identical. The protocol consisted of two sessions: one session that included the psychiatric interview as well as some testing, and one session that comprised the majority of the

neuropsychological battery. Table 2 shows the instruments administered in each session, and the order of administration. Each session lasted approximately 120 minutes. Both sessions included hippocampal-dependent and prefrontal cortex-dependent tests.

Table 2
Tests Administered in Each Session

Session #1	Session #2
MINI Psychiatric Interview	Affective Go/No Go
LEQ	CG Arena
STAI	CANTAB Spatial Span Test (forwards)
CTQ-SF	CANTAB Spatial Span Test (reverse)
CD-RISC	CANTAB Spatial Recognition Memory
PANAS	CANTAB Paired Associates Learning
WASI	CANTAB Stockings of Cambridge
CMS Word Pairs 1	CANTAB Information Sampling Task
NEPSY-II: Response Inhibition	CANTAB Intra/Extradimensional Set Shift
CMS Digit Span	CANTAB Cambridge Gambling Task
CMS Word Pairs 2	

Note. Abbreviation are as follows: MINI KID 5.0 (MINI); Life Events Questionnaire (LEQ); State-Trait Anxiety Inventory (STAI); Childhood Trauma Questionnaire- Short Form (CTQ-SF); Connor-Davidson Resiliency Scale (CD-RISC); Positive and Negative Affect Schedule (PANAS); Wechsler Abbreviated Scale of Intelligence (WASI); and Children's Memory Scale (CMS).

The testing was spread over two sessions (on two separate days) so as to avoid participants suffering from fatigue or boredom, which could have affected their performance. Both sessions were administered within a maximum of 3 weeks of each other. This was done so that the information from the interview session was still valid in terms of drawing conclusions about correlations with this information and performance on the neuropsychological tests.

Ethical Considerations

It is important to note that in order to administer the MINIKID 5.0 correctly, the primary researcher was trained by a clinical psychologist (the supervisor). The diagnoses obtained through the MINIKID 5.0 were formulated by following the structured format of this interview; each diagnostic section includes critical criteria which, if met, indicate the presence of that diagnosis. The diagnoses derived from the MINIKID 5.0 were assessed for the purpose of this research study only, and were not reported back to participants or parents/ guardians.

All the participants who were interviewed at a treatment centre were reimbursed for travel expenses by the researchers. Furthermore, all the participants (except for those from the co-ed school) received a total of R100; R50 after the first session, and R50 after the last session. The co-ed school did not want their pupils who participated to receive monetary compensation for participation. Instead, these participants were entered into a raffle and the winner was drawn after all the interviews were conducted.

It must be noted that certain further precautions were taken due to the possible vulnerability of those participants who had experienced a traumatic event and/or who were experiencing some form of psychopathology. This vulnerability might have become manifest particularly during the initial interview session, where participants were faced with relatively specific questions about previous exposure to traumatic events. Thus, all participants were verbally assured at the beginning of each session that they were able to withdraw from the study at any point without penalty, and that that they did not have to give more details than they were comfortable with. Furthermore, for all the participants, a registered clinical psychologist was made available to them if they were in any way distressed by the study procedures. For the participants tested at the treatment centres, counsellors were available at any time if needed. At the conclusion of the study procedures, participants were fully debriefed and provided with information on trauma counselling centres and trauma counsellors.

After all the interviews and testing were complete, a feedback report was created for each participant. These reports consisted of information about the range of the participant's performance on the cognitive processes tested. For an example of one of these reports, see Appendix H.

All study protocols were approved by the Research Ethics Committee of UCT Faculty of Health Sciences, by the Research Ethics Committee of the UCT Department of Psychology, and by the relevant bodies at the treatment centres and the schools involved in the study.

Data Analysis

In order to understand the nature of particular outcome variables, as well as some of the independent variables used in the statistical analyses that follow, a brief explanation of the scoring of many the tests is essential. (The tests that used standard scoring are not discussed here; rather, instructions for their scoring and interpretation of their scores can be found in the relevant test manuals.)

The Life Events Questionnaire (a measure of everyday stress; see Appendix D) was scored as follows: No points were given to an event if the participant indicated it had not occurred or it had occurred but had had “no impact” on him/her; 1 point was given to an event that the participant indicated had occurred and which he/she indicated had had “some impact” on him/her; and, lastly, 2 points were given to an event if the participant indicated it had occurred and had had “significant impact” on him/her.

In order to score the PANAS, the self-reported item scores related to positive emotions were added together, and the self-reported item scores related to negative emotions were added together.

All the computer-based tasks (viz., the CG Arena tasks, the Affective Go No-Go task, and the tasks in the CANTAB battery) generate distinct data files for each participant.

More specifically, the CG Arena software produces two distinct data files for each participant, one for the 5 trials of the visible target condition and one for 9 trials of the invisible target condition (including the probe trial). These data files include, for each individual trial, information on how long it took the participant to get to the target (in seconds), how long the path was that the participant took to get to the target, and how long (in seconds) the participant spent on each quadrant of the room. (To see more details on the information presented in these data files, see Thomas et al., 2001.)

The software used in the CG Arena ORT task (E-prime version 1.0) produces a data file for each participant that includes information on the recognition rates, the number of times the participant

correctly recognized a picture (true hits), and the number of times the participant mistakenly picked the wrong picture (false alarms).

These recognition rates were used to calculate a d-prime (d') score for each participant. The d' statistic is a measure of recognition sensitivity proposed by Signal Detection Theory (SDT). SDT is used to explore the decision-making process a person goes through when attempting to decide between various options (such as picking which picture is recognized as one that was on the walls in the arena). The d' statistic is calculated by subtracting the standard score of the true hit rate (H) from the standard score of the false alarm rate (FA). The formula is thus $d' = z(\text{FA}) - z(\text{H})$, where H and FA correspond to the right-tail probabilities on the normal distribution. A larger d' score indicates greater accuracy, where the participant rarely guessed. A smaller d' score (of 0 or less) indicates that the participant guessed often. The d' statistic is a superior way to judge performance on recognition tasks; if one only looked at the true hit rate, for instance, it would be impossible to know how much of the participant's performance was due to guessing.

In order to score the ART, I used the method shown in the score sheet reproduced in Appendix I. As can be seen, on this sheet points are awarded for particular aspects of the reconstruction. For example, 2 points are awarded for having the correct layout of the room (i.e., a set of 3 pictures on opposite walls, and 1 picture on the each of the other walls), 1 point is awarded for having the correct picture directly south of the target, 1 point is awarded for having the three correct pictures directly north of the target, and so on, resulting in a final score out of 35.

The AGNG software (E-prime version 1.0) produces a data file for each participant that includes information about the number of times the participant correctly hit a target, mistakenly missed a target, correctly did not hit a distractor, and mistakenly hit a distractor. Information on the distractors can also be broken down by emotion, so that the three distractor emotions' data (fear, happy, and neutral) can be analyzed separately. Lastly, the average reaction time for each time the participant hit a key is recorded. This information can also be broken down into key strokes corresponding to the two target emotions (fear and neutral). These data were used to calculate the d' statistic (as discussed earlier), as well as the β statistic. The β statistic, also proposed by SDT, examines any possible response bias (i.e., the tendency to favour a particular response).

Response bias (β) is calculated independently of recognition sensitivity, and is determined by looking specifically at the ratio between ‘no’ responses (in this case, not hitting the target key) and ‘yes’ responses (in this case, hitting the target key). This statistic is calculated for correct target hits (true hits) and incorrect target hits (false alarms). The extent to which one response is more probable than the other is thus assessed. A (natural log) β score of 0 shows no bias. A β score of less than zero indicates a bias towards responding ‘yes’ to all trials (i.e., always hitting the target key). A β score of more than zero indicates a bias towards responding ‘no’ to all trials (i.e., always not hitting the target key).

The CANTAB software produces a comprehensive data file for each participant that includes detailed information on each on the eight tests in the battery. For the Spatial Span tests (SSP forwards and backwards), information is included on how many trials the participant successfully completed, how many trials he/she made a mistake on, and how far the participant progressed in the test (the maximum possible span is 9). For the Spatial Recognition Memory (SRM) task, information is included on how many trials the participant got right (out of 20), as well as the reaction times for each trial. The latter were used to calculate the average reaction time for a correct decision and the average reaction time for an incorrect decision. For the Paired Associates Learning (PAL) task, information is included on how many trials the participant successfully completed, and on how many trials he/she made a mistake. For the Stockings of Cambridge (SOC) task, information is included on the time it took for the participant to complete each problem, and on the number of problems (out of 12) that were solved in the minimum number of moves. For the Information Sampling Task (IST), information is included on the time it took for the participant to choose a colour for each trial, the number of boxes opened on each trial, and the number of trials the participant completed successfully (out of 10 trials for the fixed-winnings condition and 10 trials for decreasing-winnings condition). For the Intra-Extra Dimensional Set Shift (IED) task, information is included on the number of stages successfully completed (out of a possible 9), and the total number of errors made (which could be broken down into two parts, seven pre set-shift stages, and two stages that involved a set-shift). Lastly, for the Cambridge Gambling Task (CGT), information is included on the amount of time it took the participant to pick a bet, the amount of time it took him/her to pick a colour, the percentage

of their points staked in the chosen bet, and the number of times the participant picked the colour which actually had 40% or less chance of winning.

Lastly, in terms of the NEPSY-II, along with the Naming, Inhibition, and Switching data, two further abilities are derived from combinations of the three sections, namely 'Inhibitory control' (made up from the 'Naming' and the 'Inhibition' data) and 'Cognitive flexibility' (made up from the 'Inhibition' and the 'Switching' data). The scores are derived from both accuracy and the time taken to completion.

These outcome measures were used as the dependent variables in the statistical analyses that follow.

Statistical Procedures

All the statistical analyses begin with an exploration of descriptive statistics. This exploration gave an initial picture of the performance of all the participants, and of possible differences between the three groups. Furthermore, these explorations allowed for the testing of assumptions that must be upheld for further inferential statistical analysis.

Between-Group Comparison of Demographic and Clinical Characteristics

In order to determine whether the sample was properly matched on all demographic variables across the three groups, one-way analyses of variance (ANOVAs) were conducted on all continuous variables, and chi-square (χ^2) analyses were conducted on all categorical variables. In order to explore possible differences in the clinical characteristics of the sample (i.e., disorders presented), chi-square (χ^2) analyses were conducted. In order to explore the personal characteristics and trauma characteristics of the sample, a series of one-way ANOVAs, and a series of 1-tailed t tests were conducted.

The series of statistical analyses that follow were performed in order to assess the differences between the three groups in terms of any predicted functional differences in hippocampal and prefrontal cortex regions.

Neuropsychological Test Battery Performance

In order to assess the relationship between group membership and performance on the complete test battery, after controlling for Full Scale IQ scores and sex differences, a series of hierarchical multiple regression analyses were conducted. In addition, these analyses attempted to ascertain whether group membership can be predicted by test performance (following Nolin & Ethier, 2007).

Data Sets With Repeated Measures

In order to assess the relationship between group membership and performance on the CG Arena and possible AGNG information processing biases properly, repeated-measures and factorial ANOVAs were conducted, thereby taking into account the effects of multiple trials on performance.

All statistical analyses used an alpha level of $p = 0.05$ for the threshold of statistical significance. Effect size estimates are reported, where appropriate, as these estimates allow for assessment of the real-world significance of group differences.

All statistical analyses were conducted using the software package Statistica version 8 (Statsoft, 2008).

RESULTS

Socio-Demographic Characteristics of the Sample

The sample ($N = 49$) was reasonably well matched across groups in terms of the demographic variables assessed. The breakdown of these variables for each group can be seen in Table 3.

Table 3
Socio-Demographic Characteristics of the Current Sample.

	Sample ($N = 49$) $M (SD)$, % or ratio	Control Group ($n = 17$) $M (SD)$, % or ratio	Trauma Group ($n = 16$) $M (SD)$, % or ratio	PTSD Group ($n = 16$) $M (SD)$, % or ratio
Sex				
Female:Male	30:19	10:7	10:6	10:6
Ethnicity				
% Coloured	80	88	75	75
% Black	20	12	25	25
Home Language				
% Xhosa	18	6	25	25
% Afrikaans & English	20	24	6	31
% English	61	71	69	44
Age				
Age (in years)	15.498 (1.134)	15.401 (1.050)	15.313 (1.419)	15.786 (0.888)
Years of Education	9.184 (0.993)	9.176 (1.131)	9.125 (0.683)	9.250 (0.683)

Note. For all of the variables not presented as percentages or ratios, means are presented with their standard deviations in parentheses. All the participants were from low socioeconomic status backgrounds, and therefore this variable is not included in the table. This was determined by both the socio-economic data on the communities the participants were from and the school they attended, and was confirmed by data in the demographic questionnaire.

Age

In order to assess for possible differences between the average age of the three groups, a one-way ANOVA was performed, with group membership as the independent variable (a between-subjects factor). The assumptions of normality of the distribution and homogeneity of variances (as shown by Levene's test, $F(2, 46) = 0.316, p = 0.731$) were upheld. The ANOVA showed that the factor of group membership was not statistically significant, $F(2, 46) = 0.786, p = 0.461$. The three groups were thus successfully matched for age.

Race

In order to assess for possible differences between the distribution of participants of different races across the three groups, a set of chi-square analyses was performed. The analyses showed that there were similar numbers of participants of different races in each of the three groups (Coloured: $\chi^2 = 0.462, p = 0.794$; black: $\chi^2 = 0.800, p = 0.670$). The three groups were thus successfully matched for race of participants.

Home Language

In order to assess for possible differences between the distribution of participants with different home languages across the three groups, a set of chi-square analyses was performed. The analyses showed that there were similar numbers of participants with different home languages in each of the three groups (English: $\chi^2 = 1.400, p = 0.497$; English and Afrikaans: $\chi^2 = 2.600, p = 0.273$; Xhosa: $\chi^2 = 2.000, p = 0.368$). The three groups were thus successfully matched for home language.

Years of Education

In order to assess for possible differences between the average years of education within the three groups, a one-way ANOVA was performed, with group membership as the independent variable. The assumptions of normality of the distribution and homogeneity of variances (as shown by Levene's test, $F(2, 46) = 1.411, p = 0.254$) were upheld. The ANOVA showed that the factor of group membership was not significant, $F(2, 46) = 0.062, p = 0.940$. The three groups were thus successfully matched for years of education.

IQ Data

For the sake of clarity, the data on the IQ results is presented separately, in Table 4. These IQ scores are an important factor in later data analysis and therefore closer inspection of these results is warranted.

Table 4
IQ Results for each Group Broken Down by Sex

	Full Scale IQ	Verbal IQ	Performance IQ
Control group	94.59 (11.28)	99.65 (10.93)	89.71 (13.48)
Girls ($n = 10$)	96.60 (10.77)	103.30 (11.60)	88.90 (10.19)
Boys ($n = 7$)	91.71 (12.19)	94.43 (7.93)	90.86 (18.06)
Trauma group	91.00 (16.49)	95.94 (18.19)	85.88 (14.90)
Girls ($n = 10$)	87.80 (18.91)	93.90 (20.86)	81.50 (16.99)
Boys ($n = 6$)	96.33 (10.82)	99.33 (13.68)	93.17 (6.68)
PTSD group	82.13 (14.63)	87.38 (17.48)	79.44 (12.51)
Girls ($n = 10$)	77.30 (11.73)	83.50 (16.52)	74.70 (11.13)
Boys ($n = 6$)	91.50 (13.23)	95.67 (14.85)	87.33 (11.25)
All participants	89.51 (14.59)	94.65 (15.90)	85.10 (14.04)
Girls ($n = 30$)	87.23 (15.94)	93.57 (18.14)	81.70 (13.97)
Boys ($n = 19$)	93.11 (11.65)	96.37 (11.78)	90.47 (12.73)

Note. The IQ results presented are the average scores, with standard deviations in parentheses.

A one-way ANOVA was performed on the IQ scores, with group membership being the between-subjects factor. The assumption of normality of the data distribution was upheld for all three IQ measures. Levene's tests showed that the assumption of homogeneity of variances was also upheld for all three measures (Full Scale IQ, $F(2, 46) = 1.047, p = 0.359$; Verbal IQ, $F(2, 46) = 2.485, p = 0.094$; Performance IQ, $F(2, 46) = 0.435, p = 0.650$).

The ANOVA showed that there was no significant effect of group membership on Full Scale IQ, $F(2, 46) = 3.156, p = 0.052$, Verbal IQ, $F(2, 46) = 2.397, p = 0.102$, or Performance IQ, $F(2, 46) = 2.366, p = 0.105$. These results therefore indicate that the three groups were successfully matched for IQ.

Clinical Characteristics of the Sample

Of most interest in terms of the clinical characteristics of the sample are the disorders present in the current participants (co-morbid disorders in the case of the PTSD participants). Chi-square analysis (performed on all the disorder frequency data) showed that the prevalence of all disorders is contingent on group membership, Pearson $\chi^2 = 83.38$, $p < 0.010$, Cramer's $V = 0.489$. The prevalence of disorders in the sample is presented in Table 5.

University of Cape Town

Table 5
Psychiatric Disorder Comparisons Across Groups

Disorders	Number of children with disorder		
	Control	Trauma	PTSD
Depression (current)	0	2	8
Depression (recurrent)	0	2	6
Suicide Risk (low)	6	5	4
Suicide Risk (moderate)	0	0	6
Suicide Risk (high)	0	0	2
Dysthymia (current)	0	0	6
Hypomania (current)	2	4	3
Hypomania (past)	3	7	3
Mania (current)	1	0	4
Mania (past)	2	0	5
Panic Disorder (current)	1	0	8
Panic Disorder (limited symptom attacks lifetime)	1	2	0
Panic Disorder (lifetime)	2	1	9
Agoraphobia	3	6	8
Separation Anxiety Disorder	4	2	12
Social Phobia (generalized)	0	0	1
Social Phobia (non-generalized)	0	0	1
Specific phobia (current)	3	1	3
Obsessive Compulsive Disorder (current)	0	1	10
Tourette's	0	0	1
Motor Tic	1	0	0
Vocal Tic	0	0	0
Transient Tic Disorder (current)	0	0	0
ADHD Combined	0	0	1
ADHD Inattentive	0	0	1
ADHD Hyperactivity/Impulsive	0	0	0
Conduct Disorder (current)	1	0	1
Oppositional Defiant Disorder	0	0	3
Mood Disorder with Psychotic Features (current)	0	0	4
Mood Disorders with Psychotic Features (lifetime)	0	0	3
Anorexia Nervosa	0	0	0
Bulimia Nervosa	0	0	1
Generalized Anxiety Disorder	0	0	0
Adjustment Disorders	0	0	0
Pervasive Development Disorder	0	0	0

Note. These are the disorders as categorized by the MINI KID 5.0. Because the presence of alcohol abuse and dependence, substance abuse, and psychotic disorders were all exclusion criteria, there were no participants included in this study that met those diagnoses.

The individual disorders listed in Table 3 were grouped into categories in order to test which groups of disorders were statistically significantly different across the three groups. Only those

disorders that had more than one participant who met the diagnosis were included in these groupings. The disorder groups were: Depressive Disorders (consisting of current depression, recurrent depression, any suicide risk, and dysthymia), Mania/Hypomania (current or past mania and current or past hypomania), Anxiety Disorders (any panic disorder, agoraphobia, separation anxiety disorder, any social phobia, and specific phobia), Obsessive Compulsive Disorder (OCD), Attention Deficit Hyperactivity Disorders (all forms of ADHD), Social Behavioural Disorders (conduct disorder and oppositional defiant disorder), and Mood Disorders (consisting of mood disorders with psychotic features, both current and lifetime).

Separate chi-square analyses were performed for each disorder group. The results showed that the presence of the following groups of disorders were significantly contingent on group membership; Depressive Disorders, $\chi^2 = 25.83, p < 0.0001$; Anxiety Disorders, $\chi^2 = 24.82, p < 0.0001$; OCD, $\chi^2 = 16.55, p < 0.0001$; and Mood Disorders, $\chi^2 = 14.00, p < 0.0009$. In all these groupings, the PTSD group accounted for the most number of individuals. In Depressive Disorders and OCD the trauma group accounted for the next largest number of individuals. In Anxiety Disorders the control group accounted for the next largest number of individuals after the PTSD group. And lastly, in Mood Disorders, no other groups accounted for individuals in these disorders. The presence of Mania/Hypomania and ADHD was not significantly contingent on group membership, $\chi^2 = 0.68, p = 0.710$, and $\chi^2 = 4.00, p = 0.135$, respectively. Furthermore, the presence of any of the group of Social Behavioural Disorders approached statistical significance with regard to contingency on group membership, $\chi^2 = 5.20, p = 0.074$. In this grouping, the PTSD group accounted for the largest number of individuals, the control group for only one individual, and there were no individuals from the trauma group. These grouped frequencies are presented graphically in Figure 15.

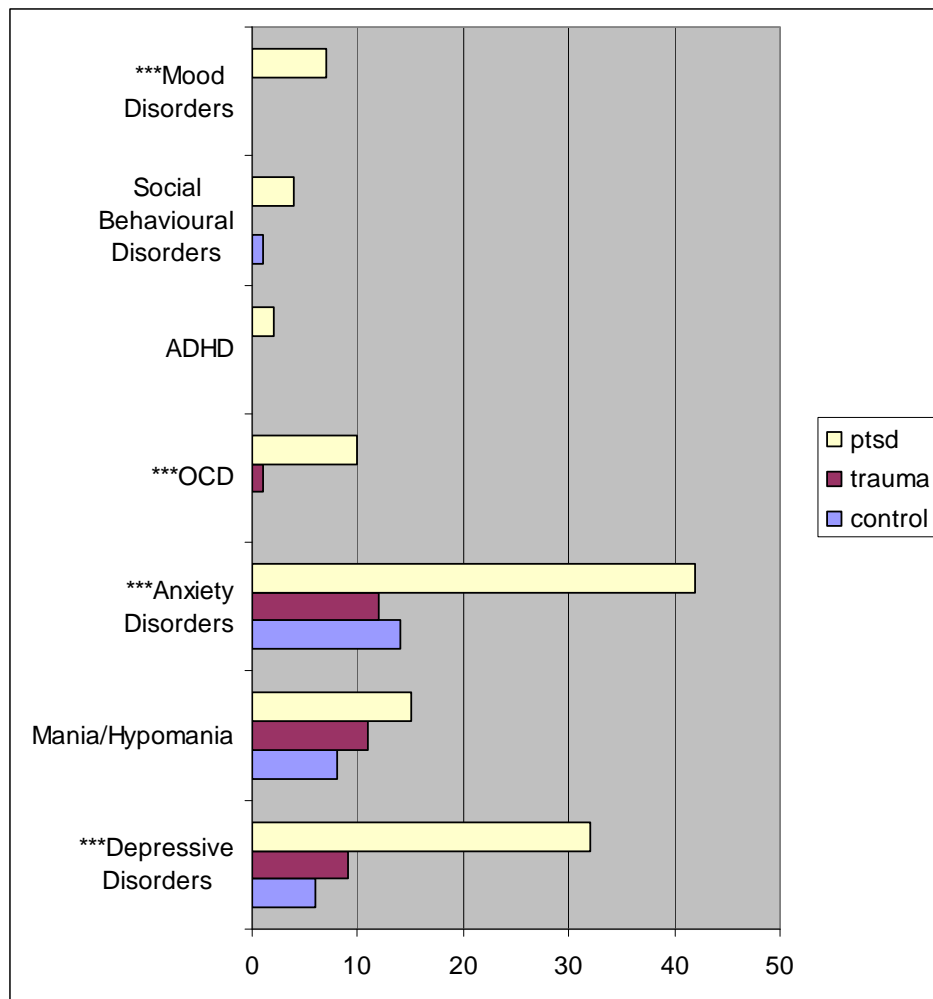


Figure 15. Number of participants who met diagnoses of grouped disorders

Note. Significant group differences: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Other Characteristics of the Sample

A set of comparisons was performed on the data collected via the paper-and-pencil questionnaires in order to assess differences between the three groups in terms of personal characteristics and attributes, as well as life events experienced. The personal characteristics and attributes consisted of: overall trait-like and current state anxiety (as measured by the STAI), perceived everyday stress (as measured by the LEQ), approach avoidance/withdrawal tendencies (as measured by the PANAS), and resiliency (as measured by the CD-RISC). The average scores across the three groups for these measures can be seen in Table 6.

Table 6
Characteristics of the Sample as Assessed by the Paper-and-Pencil Questionnaires

Measure	Possible Range	Control (<i>n</i> = 17)	Trauma (<i>n</i> = 16)	PTSD (<i>n</i> = 16)
LEQ				
< 6 months	0 - 46	2.76 (3.01)	1.75 (1.98)	4.19 (3.73)
> 6 months	0 - 46	1.59 (1.54)	1.81 (2.40)	2.69 (3.46)
CTQ-SF				
Emotional abuse*	5 - 25	7.53 (2.92)	7.06 (2.54)	10.75 (6.42)
Physical abuse*	5 - 25	5.59 (2.43)	5.31 (0.87)	7.50 (3.54)
Sexual abuse	5 - 25	5.00 (0.00)	6.25 (5.00)	5.50 (2.00)
Emotional neglect	5 - 25	7.06 (2.49)	6.69 (2.33)	7.69 (4.36)
Physical neglect	5 - 25	5.35 (0.70)	6.56 (2.16)	6.44 (1.71)
Minimization	0 - 3	1.12 (0.78)	1.44 (1.15)	1.25 (0.93)
STAI				
State**	20 - 80	35.18 (9.62)	31.69 (12.11)	43.87 (9.07)
Trait**	20 - 80	39.65 (8.36)	36.31 (9.93)	53.00 (13.01)
PANAS				
Positive	0 - 50	38.29 (5.06)	40.19 (5.53)	35.00 (8.41)
Negative*	0 - 50	20.71 (7.74)	22.19 (6.97)	28.50 (10.60)
CD-RISC	0 - 100	80.06 (7.54)	81.13 (20.46)	69.00 (21.47)

Note. * $p < 0.05$, ** $p < 0.001$. The questionnaire measure results presented for each group are the average scores, with standard deviations in parentheses. The possible score ranges are the score ranges for each questionnaire measure. For example, in the CTQ-SF, a participant answering *never* for all questions would have a score of 5 (i.e., 5 is the minimum for this measure). Controls' CTQ-SF scores could be higher than 5 as they might have experienced some of these events, even though further investigation revealed that the experience could not be classified as a DSM-IV-TR traumatic event (further details on this point are in the Participants sub-section of the Design and Methods section).

A series of one-way ANOVAs was performed on these data, with group membership always being the between-subjects factor. The assumption of normality of the distribution of the data was upheld for all the measures. Levene's test for homogeneity of variances showed that this assumption was violated for many of the measures, however. Nonetheless, due to the fact that (a) there was an approximately equal number of participants in each group, and (b) ANOVA is relatively "robust with respect to violations" (Howell, 2004, p.359), the analyses were carried out despite this violation. The results of the Levene's tests are shown in Table 7.

Table 7
Levene's Test Results for the Paper-and-Pencil Questionnaires

Measure	Levene's <i>F</i>	Levene's <i>p</i>
LEQ		
< 6 months	3.435	0.041*
> 6 months	3.138	0.053
CTQ-SF		
Emotional abuse	4.171	0.022*
Physical abuse	2.632	0.083
Sexual abuse	1.125	0.333
Emotional neglect	3.862	0.028*
Physical neglect	15.620	0.000**
Minimization	12.165	0.000**
STAI		
State	0.733	0.486
Trait	0.897	0.415
PANAS		
Positive	1.819	0.174
Negative	2.100	0.134
CD-RISC	3.295	0.046*

Note. * $p < 0.05$, ** $p < 0.001$. ($df = 30$)

The set of ANOVAs showed that there were significant between-group differences on the following measures: STAI - State, $F(2, 48) = 5.915$, $p < 0.005$; STAI - Trait, $F(2, 48) = 11.230$, $p < 0.0001$; PANAS - Negative, $F(2, 48) = 3.803$, $p < 0.030$; CTQ-SF Emotional Abuse, $F(2, 48) = 3.504$, $p < 0.038$; and CTQ-SF Physical Abuse, $F(2, 48) = 3.581$, $p < 0.036$.

Post-hoc analyses (using Tukey's HSD test) indicated that the significant differences could be attributed to characteristics of the PTSD participants. Specifically, on the STAI -State measure, participants in the PTSD group scored significantly higher than those in the Trauma group ($p < 0.005$); furthermore, the difference between the PTSD group and the Control group approached statistical significance ($p = 0.0507$). Similarly, on the STAI - Trait measure, participants in the PTSD group scored statistically significantly higher than those in both the Control group ($p < 0.002$) and the Trauma group ($p < 0.0003$). On the PANAS - Negative measure, participants in the PTSD group scored statistically significantly higher than those in the Trauma group ($p < 0.032$). On the CTQ-SF Emotional Abuse index, participants in the PTSD group scored significantly higher than those in the Trauma group ($p < 0.0499$). Finally, on the CTQ-SF

Physical Abuse index, participants in the PTSD group scored significantly higher than those in the Trauma group ($p < 0.047$).

Trauma-Related Characteristics of the Sample

Another set of comparisons was performed on the data collected regarding the trauma-related characteristics of the sample. These analyses sought to assess differences between participants in the Trauma and the PTSD groups in terms of post-traumatic symptoms experienced (as assessed by the MINI KID 5.0 and the PDS) and in terms of the number of months since the trauma. The average scores for these measures can be seen in Table 8.

Table 8

Trauma-related Characteristics of the Sample as Assessed by the MINI KID 5.0 and the PDS

Measure	Range	Group		Levene's Test	
		Trauma ($n = 16$)	PTSD ($n = 16$)	F	p
MINI KID 5.0					
Q4 score**	0 - 7	1.81 (1.05)	5.00 (1.41)	3.387	0.076
Q5 score**	0 - 5	1.13 (1.09)	3.94 (1.29)	2.745	0.108
PDS					
Level of impairment**	0 - 9	0.13 (0.34)	6.19 (3.35)	150.497	0.0001*
Number of symptoms**	0 - 17	2.06 (3.40)	12.75 (3.36)	0.332	0.569
Symptom severity**	0 - 51	3.50 (6.31)	27.38 (10.75)	4.680	0.039*
No. months since trauma	3 - 203	46.63 (41.02)	37.94 (47.68)	0.661	0.423

Note. * $p < 0.05$, ** $p < 0.001$. The questionnaire measure results presented for each group are the average scores, with standard deviations in parentheses. The possible score ranges are the score ranges for each questionnaire measure. For the “Number of months since trauma”, as the trauma had to happen at least 3 months prior to participation in the study, the minimum for this measure is 3. The maximum number of months since trauma is based on the maximum age limit of the participants. The results of Levene’s tests presented are the F scores as well as their respective p values ($df = 30$).

As there were only two groups in these analyses, a series of one-tailed t -tests was performed on the data. Where the assumption of homogeneity of variances was not upheld (as seen in the Levene’s test results), separate estimates of variance were used. Where this assumption was upheld, pooled estimates of variance were used.

As expected (because it was these measures that directly informed group membership), the two groups were statistically significantly different on all the MINI KID 5.0 and PDS outcome measures: MINI KID 5.0 question 4 score, $t(30) = 5.694$, $p < 0.0001$; MINI KID 5.0 question 5

score, $t(30) = 5.725, p < 0.0001$; PDS level of impairment, $t(15.278) = 6.661, p < 0.0001$; PDS number of symptoms, $t(30) = 7.337, p < 0.0001$, PDS symptom severity, $t(22.67) = 6.840, p < 0.0001$. For all these results, the PTSD group had significantly higher scores than the trauma group. There was no statistically significant between-group difference in terms of number of months since trauma, $t(30) = -0.582, p = 0.565$.

Testing Hypothesis 1

The following analyses tested hypothesis 1, namely: on the general neuropsychological test battery: (1a.) adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls on hippocampal-dependent and PFC-dependent cognitive tasks, and (1b.) of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly on the test battery than will those without such a diagnosis.

In order to test these hypotheses, a set of multiple hierarchical regression analyses were carried out. In order to conduct these analyses domains were created from the relevant outcome measures. These procedures are detailed below.

Due to the vast number of variables assessed in this study, a hybrid method of grouping the outcome measures was employed. This method (as described by Medina et al., 2007) involves grouping variables based on both (a) the theoretical categories of cognitive domains (Lezak et al., 2004) and (b) results of reliability analyses using Cronbach's alpha (α). This method is employed so that each final domain category is made up of variables that are both statistically significantly correlated as well as theoretically associated.

In order to decide which outcome measures went together in each cognitive domain, the measures were initially grouped together based on the theory of what each outcome measure assessed. These groupings were then statically assessed for internal consistency. In order to do this, outcome measure scores and grouped domain category scores needed to be calculated. In order to calculate the final domain category scores, each outcome measure score was converted

into a Z score based on the entire sample's scores ($N = 49$). These Z scores were then averaged for all the tests in each category to give the final composite Z score for each domain category.

These final composite Z scores were then assessed for internal consistency by reliability analysis (i.e., by computing Cronbach's α coefficients). The final composite scores were reassessed if the α coefficient was too low. This method resulted in nine composite domain categories: 1) Working Memory; 2) Verbal Memory; 3) Visual Memory; 4) Inhibition; 5) Impulsivity and Decision-Making; 6) Spatial Navigation Abilities; 7) Problem-Solving Abilities; 8) Rule Acquisition and Attentional Set-Shift Abilities; and 9) Processing Speed. These domains have Cronbach's α coefficients ranging between 0.524 and 0.908. These coefficients are within the acceptable standard for α coefficients in reliability testing (Finchilescu, 2002). The various domains with their respective descriptive statistics can be seen in Table 9.

Table 9
Descriptive Statistics for Composite Domain Categories.

	Cronbach's alpha (α)	Control (<i>n</i> = 17)		Trauma (<i>n</i> = 16)		PTSD (<i>n</i> = 16)	
		M (SD)	Range	M (SD)	Range	M (SD)	Range
Working Memory (domain z scores)	0.719	0.23 (0.67)	-0.91 to 1.35	-0.11 (0.67)	-1.33 to 0.83	-0.15 (0.55)	-0.90 to 1.04
CMS Numbers:							
Forwards - RS		11.00 (2.09)	8 - 15	9.94 (2.38)	7 - 15	9.63 (1.89)	7 - 13
Backwards - RS		6.71 (1.83)	4 - 9	5.56 (2.16)	2 - 9	5.00 (1.59)	3 - 9
CANTAB SSP:							
Forwards - RS		6.41 (1.62)	3 - 9	5.94 (1.81)	3 - 9	5.81 (1.33)	3 - 9
Forwards - SRate		0.74 (0.09)	0.59 - 0.90	0.71 (0.12)	0.44 - 0.92	0.71 (0.10)	0.53 - 0.90
Backwards - RS		5.82 (1.94)	3 - 9	5.56 (1.26)	4 - 8	5.40 (1.72)	3 - 9
Backwards - SRate		0.69 (0.11)	0.46 - 0.86	0.67 (0.09)	0.50 - 0.81	0.71 (0.11)	0.41 - 0.84
Verbal Memory (domain z scores)	0.788	0.02 (0.61)	-1.28 - 0.94	0.13 (0.97)	-1.71 - 1.50	-0.15 (0.77)	-1.48 - 1.01
CMS Word Pairs :							
Learning-SS		7.12 (2.29)	3 - 10	7.94 (4.58)	1 - 15	6.25 (3.24)	2 - 12
Immediate recall-SS		7.88 (2.89)	2 - 11	8.56 (5.06)	2 - 16	7.56 (3.16)	2 - 13
Delayed recall-SS		9.41 (3.02)	3 - 15	8.88 (4.19)	3 - 17	9.25 (3.13)	3 - 15
Delayed recognition-SS		9.94 (2.63)	3 - 11	10.44 (2.06)	3 - 12	9.06 (3.395)	1 - 11
Visual Memory (domain z scores)	0.625	0.27 (0.57)	-0.69 - 1.36	-0.12 (0.64)	-1.45 - 0.91	-0.17 (0.78)	-1.61 - 1.67
CANTAB SRM							
SRate		0.83 (0.12)	0.55 - 1.00	0.75 (0.14)	0.45 - 0.95	0.80 (0.14)	0.45 - 0.95
CANTAB PAL							
SRate		0.90 (0.09)	0.72 - 1.00	0.85 (0.10)	0.59 - 1	0.84 (0.08)	0.71 - 1.00
CG ARENA ORT							
d prime (accuracy)		1.41 (0.78)	-0.95 - 2.17	1.48 (0.55)	0.45 - 2.17	1.15 (0.69)	-0.45 - 2.17
CG ARENA ART							
SRate		0.40 (0.17)	0.22 - 0.84	0.30 (0.15)	0.00 - 0.59	0.30 (0.24)	0.03 - 1.00
Spatial Navigation Abilities (domain z scores)	0.857	0.31 (0.29)	-0.64 - 0.61	-0.05 (0.70)	-1.56 - 0.58	-0.29 (0.93)	-2.54 - 0.53
CG ARENA							

	Cronbach's alpha (α)	Control ($n = 17$)		Trauma ($n = 16$)		PTSD ($n = 16$)	
		M (SD)	Range	M (SD)	Range	M (SD)	Range
Invisible Trial 2		100.22 (67.51)	51.21 - 274.32	147.29 (89.71)	56.55 - 395.57	157.94 (128.26)	51.70 - 471.37
Invisible Trial 3		76.90 (55.71)	50.44 - 278.88	77.46 (42.40)	48.02 - 206.78	104.17 (84.71)	49.95 - 336.867
Invisible Trial 4		41.43 (30.63)	21.63 - 143.36	67.65 (60.28)	23.08 - 182.65	186.74 (218.77)	25.51 - 789.02
Invisible Trial 5		71.05 (19.03)	51.21 - 113.70	114.83 (96.83)	49.76 - 375.81	130.24 (119.05)	50.73 - 528.62
Invisible Trial 6		61.71 (15.59)	49.47 - 104.28	103.08 (93.99)	49.47 - 350.58	118.72 (114.29)	50.44 - 419.09
Invisible Trial 7		39.95 (30.19)	21.63 - 144.82	118.48 (184.710)	21.14 - 591.09	92.26 (71.70)	23.57 - 258.31
Invisible Trial 8		56.30 (51.30)	21.34 - 176.33	50.55 (30.73)	22.80 - 112.04	113.17 (207.24)	28.13 - 744.71
Probe Trial		44.34 (9.85)	23.50 - 54.28	38.58 (11.09)	11.09 - 52.05	37.74 (13.92)	7.94 - 54.44
Problem-Solving (domain z scores)	-	0.32 (0.73)	-1.34 - 1.54	-0.22 (1.29)	-2.49 - 1.54	-0.12 (0.89)	-1.92 - 1.54
CANTAB SOC							
SRate		0.74 (0.11)	0.50 - 0.92	0.66 (0.19)	0.33 - 0.92	0.68 (0.13)	0.42 - 0.92
Inhibition (domain z scores)	0.686	0.29 (0.58)	-0.58 - 1.36	-0.12 (0.48)	-1.26 - 0.65	-0.19 (0.47)	-1.00 - 0.71
NEPSY-II							
Naming (SS)		6.88 (3.20)	2 - 12	5.31 (3.44)	1 - 13	5.19 (3.02)	2 - 10
Inhibition (SS)		8.47 (2.96)	3 - 14	6.44 (2.97)	2 - 12	6.75 (2.60)	2 - 11
Switching (SS)		8.47 (4.13)	1 - 15	5.88 (3.22)	1 - 12	5.38 (2.39)	2 - 10
Inhibitory control (SS)		10.29 (3.24)	5 - 17	8.25 (3.87)	3 - 15	8.81 (2.46)	5 - 15
Cognitive flexibility (SS)		8.94 (4.02)	2 - 15	6.81 (3.37)	2 - 14	6.63 (2.36)	3 - 10
AGNG							
Go Target SRate		0.83 (0.10)	0.63 - 0.95	0.85 (0.09)	0.62 - 0.96	0.78 (0.16)	0.40 - 0.99
NoGO SRate		0.69 (0.13)	0.46 - 0.90	0.63 (0.15)	0.28 - 0.79	0.66 (0.17)	0.46 - 0.90
Reaction time (neutral target)		445.12 (46.19)	367.23 - 540.26	448.90 (59.12)	354.61 - 530.03	460.40 (95.76)	290.17 - 688.05
d prime		1.54 (0.55)	0.78 - 2.60	1.43 (0.53)	0.60 - 2.28	1.35 (0.45)	0.48 - 2.11

	Cronbach's alpha (α)	Control ($n = 17$)		Trauma ($n = 16$)		PTSD ($n = 16$)	
		M (SD)	Range	M (SD)	Range	M (SD)	Range
Rule Acquisition and Attentional Set-Shift (domain z scores)	0.906	0.24 (0.72)	-1.26 - 0.73	-0.16 (0.82)	-1.57 - 0.75	-0.09 (0.10)	-2.66 - 0.79
CANTAB IED							
Stages completed		8.71 (0.69)	7 - 9	8.50 (0.82)	7 - 9	8.50 (0.89)	7 - 9
Total errors		18.94 (16.69)	9 - 59	27.94 (18.24)	8 - 61	26.50 (22.55)	8 - 74
Total errors (Adjusted)		9.59 (9.99)	2 - 29	16.63 (11.95)	1 - 41	13.81 (11.82)	1 - 42
Pre-shift errors (stage1-6)		1.18 (3.34)	0 - 14	0.94 (1.84)	0 - 6	2.63 (5.62)	0 - 23
Post-shift errors (stage7-8)		8.41 (9.25)	1 - 29	15.69 (10.92)	0 - 37	11.19 (9.25)	1 - 28
Decision Making/Impulsivity (domain z scores)	0.524	-0.07 (0.35)	-0.66 - 0.39	0.13 (0.33)	-0.32 - 0.78	-0.06 (0.35)	-0.69 - 0.70
CANTAB IST (fixed)							
SRate		0.84(0.13)	0.50 - 1.00	0.85 (1.21)	0.60 - 1.00	0.86 (0.10)	0.7 - 1.00
Decision latency		13497.80 (4419.43)	5525.10 - 19686.50	14128.59 (4436.64)	8014.20 - 22653.60	13114.51 (4370.59)	6518.30 - 21528.60
# of boxes opened		14.94 (3.76)	7.60 - 20.70	16.03 (3.61)	9.90 - 25.00	16.22 (4.63)	9.20 - 24.80
CANTAB IST (descending)							
Success rate		0.69 (0.16)	0.40 - 1.00	0.71 (0.11)	0.50 - 0.90	0.68 (0.13)	0.40 - 0.80
Decision latency		10225.31 (3594.87)	5009.60 - 16934.80	11379.01 (4148.80)	6487.80 - 19997.80	9405.63 (4219.90)	4815.30 - 20622.40
# of boxes opened		8.02 (2.90)	4.30 - 15.00	8.11 (2.90)	2.50 - 14.50	7.93 (3.84)	2.80 - 17.50
CANTAB CGT (ascending)							
Choice latency		2970.25 (918.01)	1627.67 - 4871.61	3256.48 (1246.61)	1974.31 - 6145.19	3542.84 (1463.48)	1841.19 - 7989.47
Bet latency		1004.98 (257.30)	606.25 - 1511.31	1181.86 (366.45)	665.58 - 1954.90	1090.34 (338.22)	427.00 - 1677.44
% staked		22.63 (14.11)	5.00 - 52.78	26.11 (17.58)	7.78 - 73.89	26.21 (15.15)	5.56 - 48.47

	Cronbach's alpha (α)	Control ($n = 17$)		Trauma ($n = 16$)		PTSD ($n = 16$)	
		M (SD)	Range	M (SD)	Range	M (SD)	Range
# of times picked the min colour		2.00 (2.08)	0.00 - 7.00	1.84 (1.69)	0.00 - 5.50	2.88 (1.57)	0.50 - 6.00
CANTAB CGT (descending)							
Choice latency		2348.97 (1033.60)	1146.25 – 5424.89	2348.73 (988.58)	1254.78 - 5059.68	2795.67 (1579.19)	1505.97 - 8215.19
Bet latency		905.03 (257.23)	491.56 - 1472.72	1209.59 (368.54)	538.58 - 1873.66	988.09 (291.58)	543.77 - 1602.94
% staked		81.42 (13.17)	54.03 - 95.00	81.34 (21.03)	15.83 - 94.43	84.15 (11.78)	60.69 - 95.00
# of times picked the min colour		0.91 (1.56)	0.00 - 6.50	0.56 (0.70)	0.00 - 2.50	1.41 (1.36)	0.00 - 4.00
Processing Speed (domain z scores)	0.545	0.04 (0.53)	-0.68 - 0.93	-0.11 (0.55)	-1.03 - 0.77	0.06 (0.49)	-1.21 - 0.57
CG ARENA ORT							
Reaction time		2466.55 (564.70)	1707.13 - 3652.86	2996.20 (1011.80)	1873.88 - 4839.00	2499.16 (810.88)	1578.50 - 4395.33
CANTAB SRM							
Reaction time		2551.69 (743.75)	1422.53 - 4353.53	2545.10 (811.62)	1376.74 - 4307.94	2471.52 (895.55)	1344.16 - 4382.56
CANTAB SOC							
Reaction time		11848.91 (8208.16)	2151.00 - 28825.75	9704.56 (6904.70)	1437.50 - 20982.25	7039.31 (8082.75)	1124.00 - 34785.50
CANTAB IST (fixed)							
Decision latency		13497.80 (4419.43)	5525.10 - 19686.50	14128.59 (4436.64)	8014.20 - 22653.60	13114.51 (4370.59)	6518.30 - 21528.60
CANTAB IST (descending)							
Decision latency		10225.31 (3594.87)	5009.60 - 16934.80	11379.01 (4148.80)	6487.80 - 19997.80	9405.63 (4219.90)	4815.30 - 20622.40
CANTAB CGT (ascending)							
Choice latency		2970.25 (918.01)	1627.67 - 4871.61	3256.48 (1246.61)	1974.31 - 6145.19	3542.84 (1463.48)	1841.19 - 7989.47

	Cronbach's alpha (α)	Control ($n = 17$)		Trauma ($n = 16$)		PTSD ($n = 16$)	
		M (SD)	Range	M (SD)	Range	M (SD)	Range
CANTAB CGT (descending) Choice latency		2348.97 (1033.60)	1146.25 - 5424.89	2348.73 (988.58)	1254.78 - 5059.68	2795.67 (1579.19)	1505.97 to 8215.19

Note. Data presented are Z scores (converted Z scores based on the whole sample, $N = 49$) for composite domain categories, and scaled scores (SS) or raw scores (RS) for individual test measures, unless otherwise stated (e.g., sometimes success rate (SRate) is presented). The raw and scaled scores are presented with the average performance of each group's participants and the standard deviation of their performance in parentheses. These raw and scales scored are provided for descriptive purposes only; the Z scores were used in the statistical analyses (unless otherwise stated). Cronbach's standardized α is reported as a measure of composite domain reliability. The CG Arena invisible trial data presented are path lengths to the target; the probe trial data presented are the times spent in the target quadrant. The CANTAB SOC measures includes the number of problems the participant solved in the minimum amount of moves indicated. The Processing speed measures report the average reaction time for all *correct* decisions for the ORT and for SRM, and the average reaction time for *all* decisions in the SOC task. The CANTAB IED total errors adjusted is the adjusted score of the raw total error score; if a participant failed to complete the test (i.e., did not reach the 9th stage), 25 errors were added to his/her score for each stage not completed (this is because each stage has 50 trails and the participant could get 50% right due to chance). All z scores were formatted so that all measures were scored in the same direction (with the higher the score, the better the participant's performance.)

Using these z scores, a series of multiple hierarchical regression analyses were performed on the data. More specifically, a separate regression analysis was performed on each of the domain z scores. Multiple regression was conducted in order to determine whether group membership predicted performance within each domain. All assumptions for regression analysis (normality of data, linearity, and homoscedasticity) were met for each domain.

For each regression analysis, a domain z score was the outcome variable and group membership, Full scale IQ scores, and sex were hierarchically entered as predictor variables. Full Scale IQ score and sex were entered at the first 'block' on Step 1 and group membership was entered on Step 2. Full Scale IQ and sex were entered first as they are predictors known to affect cognitive performance, whereas group membership is an unknown predictor we wanted to assess, and therefore was entered second (as suggested by Field, 2009). If results showed that group membership significantly predicted performance on a certain domain, post-hoc multiple regression analyses were performed on each of the individual outcome measures of the tests that made up that domain. As with the initial multiple regression analyses, the post-hoc analyses were also performed on the outcome measure z scores.

Descriptive Statistics for Domain Scores

The mean domain scores presented in Table 10 show that, for the majority of the domains, participants in the PTSD group performed the worst out of the three groups, with participants in the control group performing the best. These domains were: Working Memory, Visual Memory, Spatial Navigation, and Inhibition. In two of the other domains (Problem-Solving and Rule Acquisition), participants in the control group still performed the best, but in these cases those in the trauma group performed worst. Curiously, in the Verbal Memory domain, participants in the PTSD group performed the worst, but those in the trauma group performed the best. Lastly, in the domains of Decision-Making/Impulsivity and Processing Speed (domains which can be seen as theoretically linked, and which actually have four outcome measures which are the same), the patterns

of performance were quite different. For the Impulsivity domain, the control participants performed the worst, meaning they were the most impulsive, and the trauma participants performed the best, i.e. were the least impulsive. (This pattern is however made up of two very different patterns for the boys and the girls when viewed separately, an effect which is discussed later). For the Processing Speed domain, the trauma participants were the slowest and the PTSD participants were the quickest.

Testing Hypothesis 1: Regression Results

Although the overall regression models for many of the domains were significant, the regression analyses indicated that group membership specifically was statistically significantly associated with performance in only one of the neuropsychological domains: after controlling for Full Scale IQ and the effects of sex differences, group membership was significantly related to a decreased performance on tests in the Inhibition domain (control vs. trauma: $\beta = -0.290$, $p < 0.031$). The results further showed that the sex of the participant alone was not a significant predictor of performance in that domain, $\beta = -0.052$, $p = 0.653$, but that Full Scale IQ score was a significant predictor, $\beta = 0.576$, $p < 0.0001$ (i.e. participants with higher Full Scale IQ scores performed better on tests in this domain). These results are presented in Table 11.

In this domain, Full Scale IQ score and sex of the participant together accounted for 37 % of the variability in performance. Group membership alone (control vs. trauma and control vs. PTSD) accounted for 7 % of the variability in performance in Inhibition. The overall regression model for Inhibition (with all three independent variables) explained 44 % of the variability in the data, and was a statistically significant model, $F(4,44) = 8.549$, $p < 0.0001$. The regression results for each step are also presented in Table 10.

Table 10
Testing Hypothesis 1: Regression Analysis Results of Significant Neuropsychological Domain score.

	Inhibition		
	β	t	p value
Step 1.			
Constant		0.283	0.779
Sex	-0.052	-0.436	0.665
IQ: Full Scale	0.618	5.188	0.000***
Step 2.			
Constant		0.320	0.751
Sex	-0.052	-0.453	0.653
IQ: Full Scale	0.576	4.676	0.000***
Group: control vs. trauma	-0.290	-2.229	0.031*
Group: control vs. PTSD	-0.191	-1.390	0.172

Note. Δ = change; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Furthermore, for Step 1: $R^2 = 0.37$; ΔR^2 for Step 2. = 0.07; For Step 2. $R^2 = 0.44$
 F to enter/remove = 2.543 ($p = 0.900$)

The decrements in performance associated with group membership (as seen in the step 2 control vs. trauma factor) in the domain of Inhibition is associated with a relatively impaired ability to inhibit one's automatic responses in favour of new responses, as well as a relatively impaired ability to switch between different response types (i.e. a relatively impaired ability to respond when one should and not respond when one shouldn't, and to be able to switch between these response types).

Testing Hypothesis 1: Overall Regression Models

The overall regression models (with all three variables: Full Scale IQ, sex, and group membership) were statistically significant for the domains of: Working Memory, $F(4,44) = 15.096$, $p < 0.0001$ (Full Scale IQ and sex were significant predictors); Verbal

Memory, $F(4,44) = 7.471, p < 0.0001$ (Full Scale IQ was a significant predictor); Visual Memory, $F(4,44) = 15.659, p < 0.0001$ (Full Scale IQ and sex were significant predictors); Spatial Navigation, $F(4,44) = 14.402, p < 0.0001$ (Full Scale IQ and sex were significant predictors); Rule Acquisition, $F(4,44) = 5.530, p < 0.0001$ (Full Scale IQ was a significant predictor); and Inhibition, $F(4,44) = 8.549, p < 0.0001$ (Full Scale IQ and group membership were significant predictors, as discussed).

Only the domains of Problem-Solving, Processing Speed, and Decision-Making/Impulsivity did not have statistically significant models (i.e., none of the independent variables significantly predicted the scores in these domains).

More details about these regression models are presented in Table 11.

Table 11

Testing Hypothesis 1: Primary Regression Model Results for each Domain.

	Working memory	Verbal memory	Visual memory	Spatial navigation	Rule acquisition	Inhibition	Problem-solving	Processing speed	Decision-making/Impulsivity
β : control vs.trauma.	-0.162	0.142	-0.183	-0.154	0.158	-0.290	-0.228	-0.104	0.254
β : control vs. PTSD	-0.017	0.131	-0.035	-0.147	0.039	-0.191	-0.126	0.128	-0.034
Model F(4,44)	15.096	7.461	15.659	14.402	5.530	8.549	2.158	1.227	1.388
Model p -level	0.000	0.000	0.000	0.000	0.000	0.000	0.090	0.313	0.254
Step 1 R^2	0.555	0.386	0.559	0.545	0.303	0.372	0.125	0.063	0.039
ΔR^2	0.024	0.018	0.028	0.022	0.032	0.065	0.039	0.037	0.073
Step 2 R^2	0.578	0.404	0.587	0.567	0.335	0.437	0.164	0.100	0.112
F to ent/rem	1.242	0.674	1.514	1.121	1.049	2.543	1.033	0.920	1.819
Step 2. p -level	0.299	0.515	0.231	0.335	0.359	0.090	0.364	0.406	0.174

The change in R^2 at step 2 (group membership) is of particular interest, as these values speak to practical significance. The increases in multiple R^2 values range from 0.018 (Verbal memory), to 0.065 (Inhibition), to 0.073 (Impulsivity, although group membership as a predictor was not significant in this domain).

Furthermore, the beta values (which are standardized correlation coefficients), show that when Full Scale IQ scores and sex are held constant, the trauma and PTSD groups both perform worse than the control group on five of the domains.

Testing Hypothesis 1: Post-hoc Regression Results for Individual Test Outcome Measures in Inhibition

Post-hoc multiple regression analysis was performed on the individual test outcome measures (specifically, the z-scores) that made up the domain of Inhibition (the only domain in the primary regression analyses in which group membership was a statistically significant predictor). The individual outcome measures that comprised the Inhibition domain were from the Nepsy-II and the AGNG task. The individual outcome measures from the Nepsy-II Inhibition subtest were: naming, inhibition, switching, inhibitory control, and cognitive flexibility (all scaled score). The dependent measures from the AGNG were: Go Target success rate, No-Go success rate, reaction time (for neutral targets), and d' scores).

Although the overall regression models for many of the outcome measures were significant, the regression analyses indicated that, group membership was statistically significantly associated with only one of the outcome measures: the measure of switching on the NEPSY-II Inhibition subtest. After controlling for Full Scale IQ and the effects of sex, group membership was significantly related to a decreased performance in switching ability (control vs. trauma: $\beta = -0.282$, $p < 0.038$). These results are shown in Table 12. The results showed that the sex of the participant alone was not a significant predictor, $\beta = -0.019$, $p = 0.872$, but that the Full Scale IQ score was a significant predictor, $\beta = 0.555$, $p < 0.0001$ (i.e. participants with higher Full Scale IQ scores performed better on this test).

On the measure of switching in the NEPSY-II Inhibition subtest, Full Scale IQ score and the sex of the participant together accounted for 36% of the variability in performance. Group membership alone (control vs. trauma and control vs. PTSD) accounted for 6% of the variability in performance in the Inhibition domain. The overall regression model for switching (with all three independent variables) explained 42% of the variability in the data, and was a statistically significant model, $F(4,44) = 7.927, p < 0.0001$. These results are reported in full in Table 12.

Table 12

Testing Hypothesis 1: Post-hoc Regression Analysis Results for Significant Individual Outcome Measure.

	Switching		
	β	t	p value
Step 1			
Constant		-3.613	0.0009***
Sex	-0.019	-0.162	0.872
IQ: Full Scale	0.555	4.434	0.000***
Step 2			
Constant		0.320	0.751
Sex	-0.052	-0.453	0.653
IQ: Full Scale	0.576	4.676	0.000***
Group: control vs. trauma	-0.282	-2.135	0.038*
Group: control vs. PTSD	-0.198	-1.415	0.164

Note. Δ = change; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Furthermore, for Step 1: $R^2 = 0.36$; ΔR^2 for Step 2. = 0.06; For Step 2. $R^2 = 0.42$
 F to enter/remove = 2.37 ($p = 0.105$)

Testing Hypothesis 1: Regression Models for Outcome Measures in Inhibition

The overall regression models (with all three variables: Full Scale IQ, sex, and group membership) were significant for the outcome measures: naming, $F(4,44) = 3.009, p < 0.028$ (Full Scale IQ was a significant predictor); inhibition, $F(4,44) = 4.479, p < 0.004$ (Full Scale IQ was a significant predictor); switching, $F(4,44) = 7.927, p < 0.0001$ (Full Scale IQ and group membership was a significant predictor, as discussed); cognitive flexibility, $F(4,44) = 4.475, p < 0.004$ (Full Scale IQ was a significant predictor); AGNG go target success rate, $F(4,44) = 3.042, p < 0.027$ (Full Scale IQ was a significant predictor); and lastly, AGNG go neutral ave RT, $F(4,44) = 4.446, p < 0.004$ (Full Scale IQ and sex were significant predictors).

The outcome measures of inhibitory control, AGNG NoGo success rate, and AGNG d prime did not have statistically significant models (i.e. none of the independent variables significantly predicted the scores in these measures). This information is seen in table form in Table 13.

Table 13

Testing Hypothesis 1: Post hoc Regression Model Results for each Outcome Measure in Inhibition

Outcome Measure	$F(4, 44)$	p	Step 1 R^2	ΔR^2	Step 2 R^2
NEPSY-II naming	3.009	0.028	0.190	0.025	0.215
NEPSY-II inhibition	4.479	0.004	0.227	0.062	0.289
NEPSY-II switching*	7.927	0.000	0.356	0.063	0.419
NEPSY-II inhibitory control	2.057	0.103	0.100	0.057	0.158
NEPSY-II cognitive flexibility	4.475	0.004	0.496	0.043	0.289
AGNG Go target success rate	3.042	0.027	0.196	0.021	0.217
AGNG NoGo success rate	1.849	0.137	0.106	0.037	0.144
AGNG Go neutral ave RT	4.446	0.004	0.290	0.002	0.293
AGNG d prime	1.007	0.414	0.078	0.006	0.084

Note. * Group membership significant in this outcome measure.

The change in R^2 at step 2 (group membership) is, once again, of interest. The increases in multiple R^2 values range from as little at 0.002 (AGNG Go neutral ave RT), to 0.063 (switching). These results suggest that the real world significance of these effects are in fact quite small.

Lastly, a regression equation was constructed using sex of the participant, Full Scale IQ score, and switching scores as the predictor variables, to predict group membership. The final equation was as follows: $\text{Group} = 0.126 - 0.024 (\text{gender}) + 0.005 (\text{Full Scale IQ score}) - 0.192 \text{ switching score (on the Nepsy-II Inhibition subtest)}$. (In this equation female: 1, male: 2; trauma exposed: 1, non-trauma control: 0.)

Hypothesis 1: Summary and Conclusion

The analyses done in order to test hypothesis 1, showed that trauma was indeed a significant factor on performance in at least one cognitive domain, namely Inhibition. This confirms hypothesis 1a (adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls). The patterns of performance suggest that with great a sample size this hypothesis could have been confirmed on many more domains (such as Processing speed). Hypothesis 1b (of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly) could not be confirmed, although the patterns of performance suggest that with greater sample sizes this hypothesis could have been confirmed in many of the domains (such as on Working memory, Visual memory. Verbal memory and Spatial navigation).

Testing Hypothesis 2

The following analyses tested hypothesis 2, namely: on the specific test of Spatial navigation: (2a) adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls on hippocampal-dependent cognitive tasks, and (2b) of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly on the test battery than will those without such a diagnosis.

In order to test this hypothesis, a set of repeated-measures ANOVAs were carried on the CG Arena visible and invisible trail data. Following this, a factorial ANOVA was carried out on the CG Arena probe trail data.

Testing Hypothesis 2: CG Arena, Visible Target Trials

Data from the visible target trials were analyzed by examining the path length each participant took from starting position to the visible target in the virtual room. An examination of those data, conducted prior to the inferential statistical analysis, showed that the assumption of sphericity (homogeneity of variances) of the data was violated. Some of the participants data was therefore dropped by random selection in order to have equal cell sizes in each group for both girls and boys (the final sample for this analysis was $N = 36$). This step ensured that the repeated-measures analysis could be conducted effectively, even without the assumption of sphericity being met. The descriptive statistics for each modified group across the five visible target trials are presented in Table 14.

Table 14

Testing Hypothesis 2: CG Arena Data for Visible Target Trials for Each Group.

	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
Control group	35.08 (11.70)	33.43 (14.22)	35.98 (14.61)	63.66 (13.33)	60.10 (11.69)
Girls ($n = 6$)	42.77 (11.13)	43.05 (14.62)	45.39 (14.35)	73.44 (12.01)	66.89 (13.36)
Boys ($n = 6$)	27.40 (5.98)	23.81 (2.99)	26.57 (7.16)	53.88 (4.11)	53.30 (3.36)
Trauma group	28.97 (17.51)	34.21 (22.87)	36.53 (33.27)	63.40 (22.36)	64.91 (26.44)
Girls ($n = 6$)	33.79 (24.57)	42.93 (30.99)	45.07 (46.78)	72.32 (29.94)	74.44 (36.09)
Boys ($n = 6$)	24.15 (3.84)	25.50 (2.82)	27.99 (8.47)	55.48 (3.52)	55.38 (4.23)
PTSD group	32.58 (11.62)	38.27 (14.41)	36.83 (14.54)	66.60 (11.99)	66.96 (16.83)
Girls ($n = 6$)	41.73 (9.06)	43.80 (16.55)	47.76 (12.87)	75.89 (9.17)	78.64 (17.03)
Boys ($n = 6$)	23.44 (2.00)	32.74 (10.46)	25.90 (3.54)	57.31 (5.01)	55.28 (2.36)
All participants	32.21 (13.72)	35.30 (17.26)	36.45 (21.94)	64.55 (16.13)	63.99 (18.98)
Girls ($n = 18$)	39.43 (16.07)	43.26 (20.64)	46.07 (27.47)	73.88 (18.25)	73.32 (23.36)
Boys ($n = 18$)	25.00 (4.38)	27.35 (7.28)	26.82 (6.38)	55.22 (4.29)	54.66 (3.35)

Note. The data presented here are average length of the path taken from the start position in the room to the visible target, with standard deviations in parentheses.

This task is designed to train participants for the next stage of the CG Arena (i.e., for the task presented by the eight invisible target trials), so that previous computer game-playing and joystick experience is not a factor on those subsequent trials. One would expect a large variation in the first trials due to this prior experience, with the variance becoming smaller towards the last trials. This pattern of data is expected because one would assume that, across trials, all participants would become better at navigating through the room to the target, but that no participant would ever find the target in fewer than the path length representing the most direct straight-line route from start position to target; therefore, there should be a steadily decreasing decrease in variance from the first to the last visible target trial.

However, as one can see from the table above, the variation fluctuates greatly, and shows no pattern in reduction from the first to the last trial. This could be due to the simple fact that the target was in a different location in each trial, and in some later trials, the target was placed so that it actually took longer to find it.

In order to analyze the data ANOVA with repeated measures was performed. This analysis included two between-subjects factors (group membership and sex) and one within-subjects factor (trials, which had five levels). Although the assumptions of normality of data distribution and independence of observations were upheld, sphericity (the assumption of homogenous variances) was violated, as indicated by Mauchly's test, $\chi^2(9) = 19.62, p < 0.02$. Greenhouse-Geisser estimates for sphericity were therefore used to correct degrees of freedom ($\epsilon = 0.80$).

In terms of the within-subjects effects, there was a significant main effect of trials on the dependent variable, length to target, $F(4, 120) = 170.69, p < 0.0001$. This means that the length it took for the participants to find the target depended, in a statistically significant manner, on the trial they were completing. None of the within-subjects interaction effects were significant: trials x group, $F(8, 120) = 1.10, p = 0.369$; trials x sex, $F(4, 120) = 0.71, p = 0.587$; trials x group x sex, $F(8, 120) = 0.817, p = 0.589$.

In terms of the between-subjects effects, there was a significant main effect of sex, $F(1, 30) = 12.03, p < 0.002$. As indicated in Figure 16, this analysis suggests that the girls took significantly longer, on average, to find the visible target. Furthermore, the variance for the girls' data is far greater than the variance for the boys' data. This difference in variance was found to be significant using Mauchly's test of sphericity, Wilks lambda = 0.534, $\chi^2(9) = 20.34, p < 0.016$.

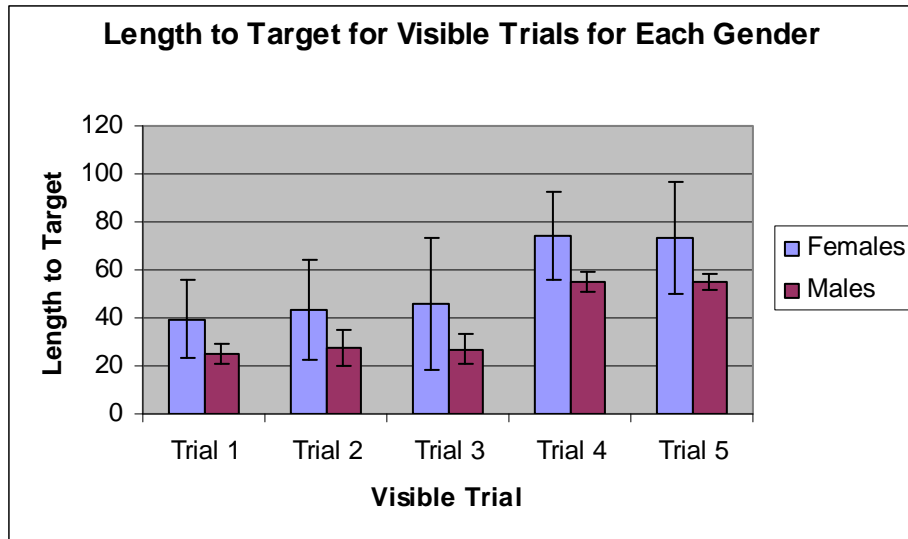


Figure 16. Visible trials: length to target. Error bars represent 95% confidence intervals.

Most importantly, the main effect of group membership (which is graphically presented in Figure 17) was not statistically significant, $F(2, 30) = 0.122, p = 0.886$. The between-subjects interaction (group x sex) effect was also not statistically significant, $F(2, 30) = 0.020, p = 0.981$. As success of this task is not hippocampus-dependent, this non-significant result is expected.

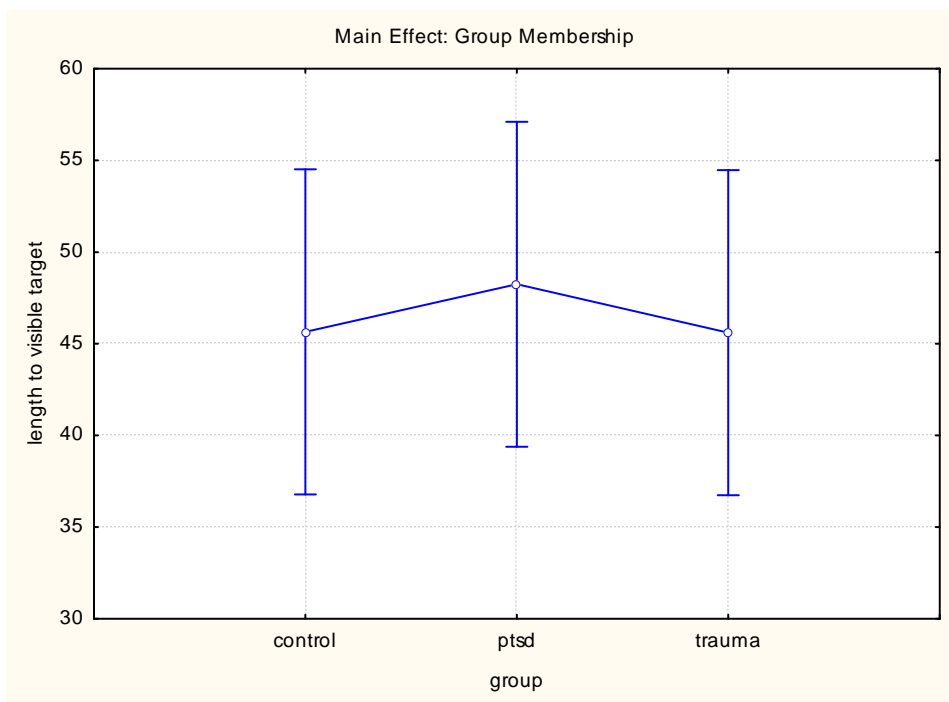


Figure 17. Length to visible target: main effect of group membership.

Testing Hypothesis 2: CG Arena, Invisible Target Trials

Data from the set of CG Arena invisible trials were also analyzed by examining the length of each participant's path, on each trial, from start position to the invisible target in the virtual room. Table 15 shows descriptive statistics for performance of participants in the three groups across the eight invisible-target trials.

Table 15

Testing Hypothesis 2: CG Arena Data for Invisible Target Trials for Each Group.

	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5	Trial 6	Trial 7	Trial 8
Control	149.98 (128.80)	100.21 (67.51)	76.90 (55.71)	41.43 (30.63)	71.05 (19.03)	15.59 (61.71)	39.95 (30.19)	56.30 (51.30)
Girls (<i>n</i> =10)	207.49 (132.56)	108.68 (84.48)	84.37 (69.50)	51.91 (36.91)	74.36 (22.72)	18.78 (66.52)	49.95 (36.61)	71.61 (59.39)
Boys (<i>n</i> =7)	67.83 (67.01)	88.12 (34.05)	66.24 (28.38)	26.48 (3.88)	66.32 (12.14)	5.01 (54.83)	25.65 (3.90)	34.43 (27.89)
Trauma	278.70 (238.00)	147.29 (89.71)	77.46 (42.40)	67.65 (60.28)	114.83 (96.83)	93.99 (103.08)	118.48 (184.71)	47.39 (32.27)
Girls (<i>n</i> =10)	266.56 (244.68)	169.17 (104.46)	84.24 (50.66)	72.24 (62.32)	132.05 (116.17)	114.76 (122.55)	141.04 (218.13)	50.79 (37.72)
Boys (<i>n</i> =6)	298.95 (247.75)	110.82 (44.12)	66.17 (23.00)	60.00 (61.64)	86.13 (47.50)	27.83 (70.63)	80.88 (118.29)	41.72 (22.37)
PTSD	196.33 (254.98)	157.94 (128.26)	104.17 (84.54)	186.74 (218.77)	130.24 (119.05)	114.29 (118.72)	92.26 (71.70)	113.17 (207.24)
Girls (<i>n</i> =10)	180.46 (234.06)	183.22 (135.76)	127.34 (100.35)	270.61 (241.08)	170.86 (136.57)	136.30 (151.02)	117.61 (79.94)	160.17 (254.94)
Boys (<i>n</i> =6)	222.78 (308.36)	115.82 (113.00)	65.56 (21.19)	46.95 (38.07)	62.53 (12.02)	13.53 (64.88)	50.01 (22.23)	34.85 (8.92)
All	207.15 (215.52)	134.43 (99.14)	85.99 (63.19)	97.44 (143.07)	104.67 (90.18)	86.74 (93.83)	82.67 (116.95)	71.96 (124.41)
Girls (<i>n</i> =30)	218.17 (205.85)	153.69 (111.37)	98.65 (76.46)	131.59 (172.43)	125.76 (108.46)	106.00 (113.36)	102.87 (136.78)	94.19 (155.03)
Boys (<i>n</i> =19)	189.75 (234.69)	104.04 (68.07)	66.00 (23.25)	43.53 (40.87)	71.38 (28.72)	17.91 (62.99)	50.78 (67.65)	36.86 (20.78)

Note. The Invisible Target trial data presented are average length of the path taken from the start position in the room to the visible target, with standard deviations in parentheses.

This task is designed to test the spatial abilities of the participants as it requires them to build a cognitive map of the virtual environment in order to re-locate the invisible target (which, recall, is always in the same location) on trials 2 through 8. On trial 1, the participants obviously do not know where the target is, and have to find it by searching the environment.

In order to analyze the data, repeated-measures ANOVA was performed. This analysis included two between-subjects factors (group membership and sex) and one within-subjects factor (trials, which had eight levels). The assumptions of normality of data distribution and independence of observations were upheld, as was the assumption of sphericity, as indicated by Mauchly's test, $\chi^2(27) = 41.11, p = 0.055$.

In terms of the within-subjects effects, there was a significant main effect of trials on the dependent variable, path length to target, $F(7, 301) = 7.86, p < 0.0001$). This statistic indicates that the length of the participants' path from starting position to target varied systematically depending on the trial they were completing. In other words, the number of steps it took for the participants' to find the target significantly depended on the trial they were on. This result was expected because, as noted above, on the first invisible-target trial the participants had to find the target by simply walking around the environment until they happened upon it. On subsequent trials, because they had been told that the target would be in the same place on all trials, they therefore had gained information on where to look, and thus were expected to take significantly shorter path lengths to the target. Post-hoc comparisons (using Tukey's HSD test) showed that the statistically significant F -value reported above was indeed a product of first-trial performance compared to subsequent trials performance. Specifically, trial 1 compared to subsequent trials resulted in all significant p values; trial 2: $p < 0.025$; trial 3: $p < 0.0001$; trial 4: $p < 0.0001$; trial 5: $p < 0.00015$; trial 6: $p < 0.0001$, trial 7: $p < 0.0001$, trial 8: $p < 0.0001$. No other significant differences were seen across any other trials. All the results of this post-hoc analysis can be seen in Appendix J.

None of the within-subjects interaction effects were significant: trials x sex, $F(7, 301) = 0.346, p = 0.932$); trials x group, $F(14, 301) = 1.194, p = 0.279$; trials x group x sex, $F(14, 301) = 1.167, p = 0.300$.

In terms of the between-subjects effects, there was a significant main effect of sex, $F(1, 43) = 7.74, p < 0.008$: Girls took significantly longer, overall, to find the target; means = 129.783 (standard deviation: 141.343, standard error: 11.228) vs. 78.167 (standard deviation: 101.388, standard error: 14.147).

Of primary importance here is the main effect of group membership, which the analysis showed to be statistically significant, $F(2, 43) = 3.579, p < 0.036$. Post-hoc analyses (using Tukey's HSD test) indicated statistically significant differences between the

control and the PTSD group, with participants in the latter group taking significantly longer paths to reach the target than those in the former; means = 137.45 (standard deviation: 162.275, standard error: 15.879) vs. 74.69 (standard deviation: 68.045, standard error: 15.154), $p < 0.015$. This effect, across all 8 trials, is presented graphically in Figure 18.

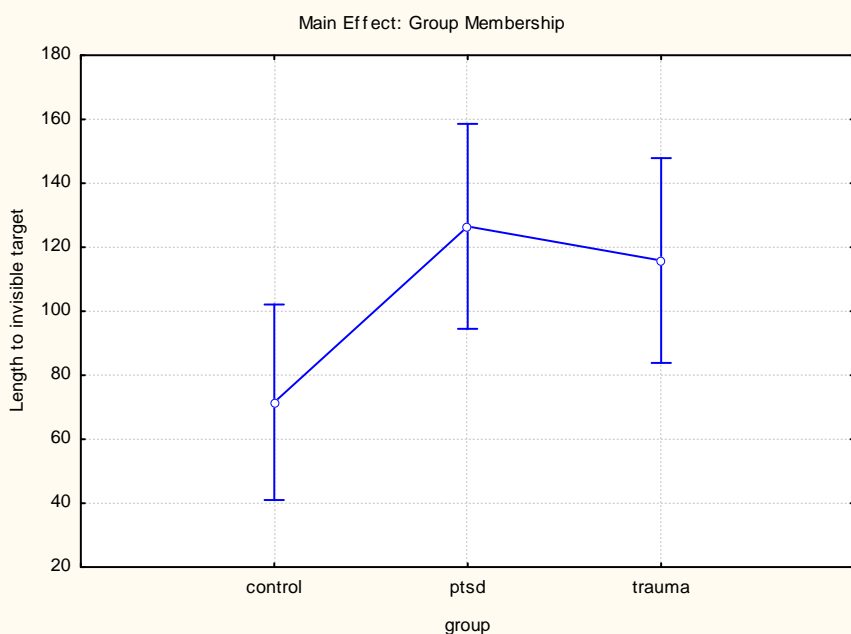


Figure 18. Length to invisible target: main effect of group membership. Error bars represent 95% confidence intervals.

Although the interaction effect of group x sex was not statistically significant, $F(2, 43) = 1.042$, $p = 0.362$, this interaction is also presented in Figure 19 in order to show the group effect when broken down by sex. This figure illustrates that the main group effect is due to the girls' performances.

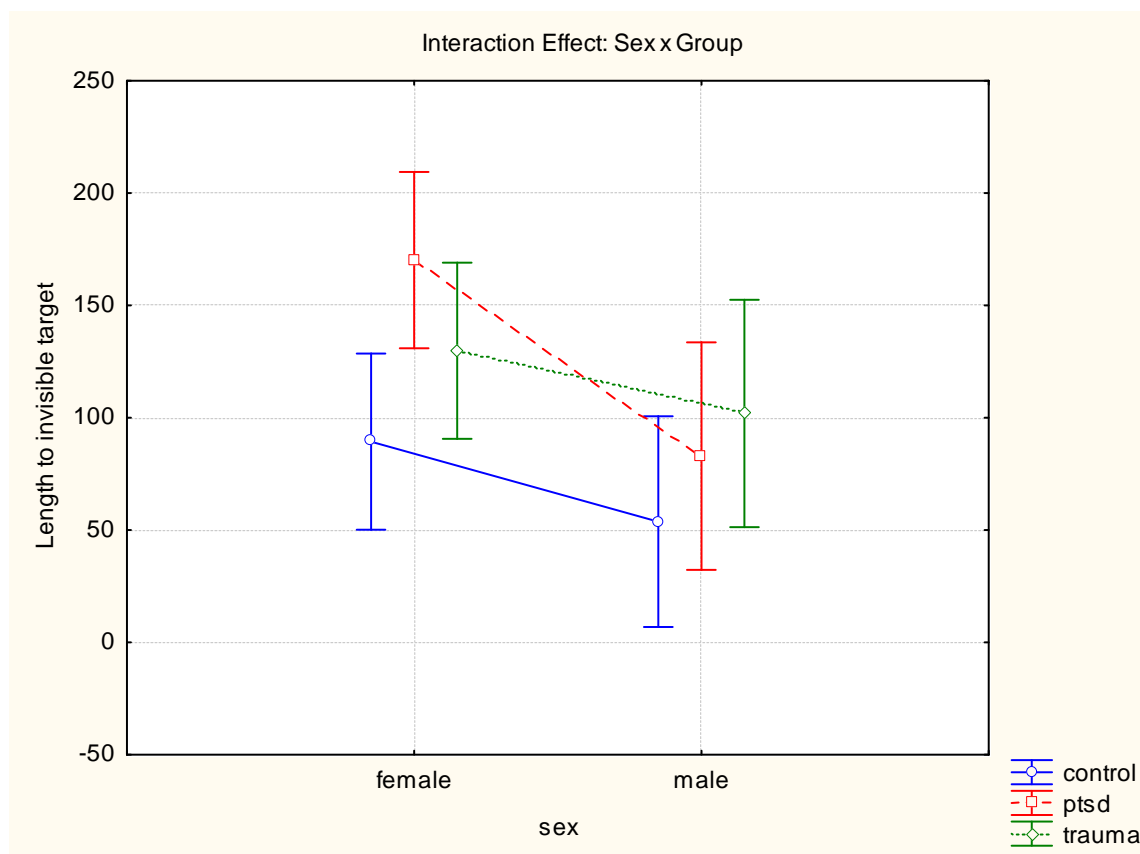


Figure 19. Length to invisible target: interaction effect between group membership and sex.

Testing Hypothesis 2: CG Arena, Probe Trial

As in previous CG Arena studies (Thomas, Laurance, Nadel, and Jacobs, 2010), this trial was used to measure the participant's persistence of search. In other words, through the set of invisible-target trials the participant was told the target was always in the same place. On this trial (which immediately followed the set of invisible-target trials and was formally identical to them), the target was, unbeknownst to the participant, removed from the room. The participant should therefore spend most of his/her time searching for the target in the quadrant of the room where the target was formerly located (i.e., the 'target quadrant'). The dependent variable for this analysis, therefore, is time spent in the northwest quadrant (i.e., dwell time). One of the hypotheses that is being tested here is that there will be marked sex differences in performance (where a group membership effect may only be seen in females; with trauma participants performing worse than

controls, and the PTSD participants performing most poorly out of the three groups.) Table 16 shows descriptive statistics for performance of participants in the three groups for the probe trial. Figure 20 shows the average performance per group across all quadrants.

Table 16

Testing Hypothesis 2: CG Arena Data for the Probe Trial for each Group.

	The Probe Trial (Average time spent in the northwest quadrant)
Control group	44.34 (9.85)
Girls ($n = 10$)	46.52 (9.07)
Boys ($n = 7$)	41.22 (10.76)
Trauma group	38.58 (11.09)
Girls ($n = 10$)	37.59 (10.73)
Boys ($n = 6$)	40.23 (12.51)
PTSD group	37.74 (13.92)
Girls ($n = 10$)	31.96 (13.42)
Boys ($n = 6$)	47.37 (8.91)
All participants	40.30 (11.84)
Girls ($n = 30$)	38.69 (12.42)
Boys ($n = 19$)	42.85 (10.69)

Note. The Probe trial data presented are average time spent in the target quadrant, with standard deviations in parentheses.

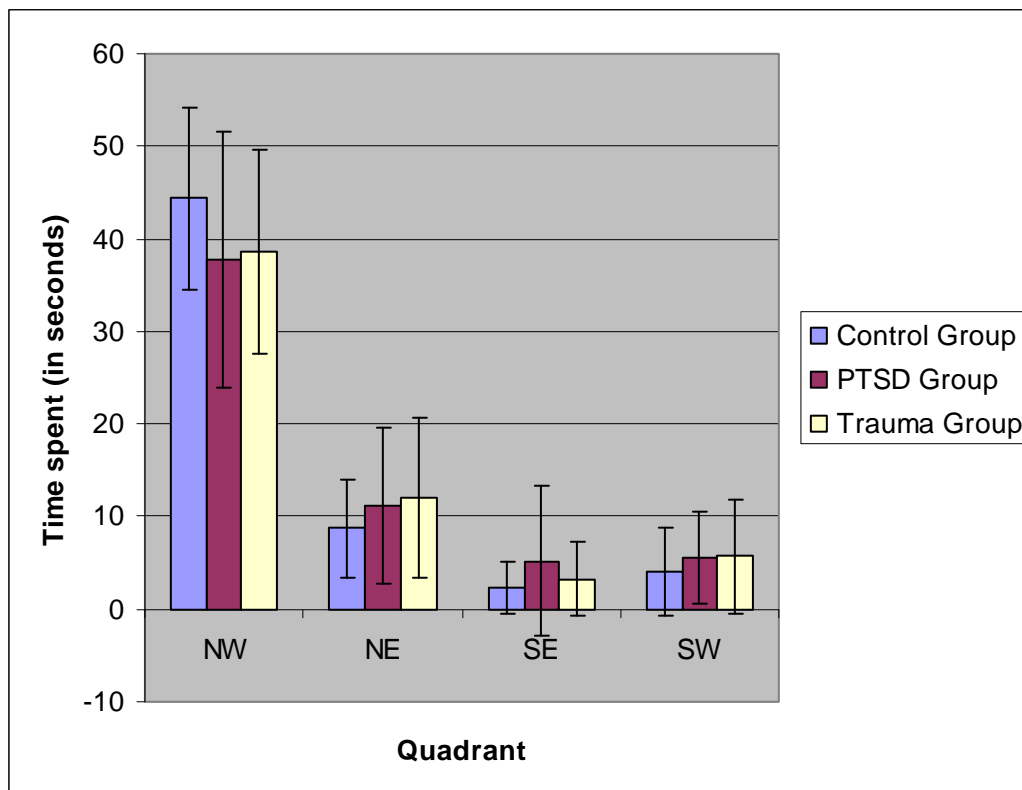


Figure 20. Probe Trial: time spent in each quadrant. Error bars represent 95% confidence intervals.

Figure 20 shows that, on average, participants in all three groups spent the most time in the northwest (target) quadrant. Further, it appears that participants in the control group spent the most time in this quadrant, followed by those in the trauma group and then those in the PTSD group.

To confirm these impressions statistically, I performed a two-way factorial ANOVA on the time spent in the northwest quadrant data. This analysis included two between-subjects factors (group membership and sex). The assumption of homogeneity of variances was not violated, Levene's test $F(5, 43) = 0.69, p = 0.63$.

The main effect of group membership was not statistically significant, $F(2, 43) = 0.93, p = 0.403$. The main effect of sex was also not statistically significant, $F(1, 43) = 1.71, p = 0.198$. Of interest, however, is that the interaction effect of sex x group was statistically

significant, $F(2, 43) = 3.47$, $p < 0.040$, Adjusted $R^2 = 0.021$. The exploration of this interaction effect is particularly interesting, and is presented visually in Figure 21.



Figure 21. Time spent in the northwest quadrant: interaction effect between group membership and sex. Error bars represent 95% confidence intervals.

In breaking down the data by group and sex, I found particularly interesting results. The girls' average performance across the three groups followed the same pattern seen in the main effect of group membership, however the boys' average performance across the three groups is quite different. As the figure shows, boys in the control and trauma groups spent relatively similar amounts of time in the target quadrant, whereas those in the PTSD group spent more time in the target quadrant; this result was not expected, but (as post-hoc analyses showed) the between-groups comparison was also not statistically significant.

Post-hoc analysis (using Tukey's HSD test) of the interaction effect indicated that there was a between-group difference approaching significance when comparing male and female PTSD participants, with the former spending substantially longer times in the target quadrant than the latter (means 47.37 (8.91) vs. 31.96 (13.42), $p < 0.097$). There

were also a difference approaching significance between the female control group and the female PTSD group; means 46.52 (9.07) vs. 31.86 (13.42), $p < 0.055$. All the results of this post-hoc analysis can be seen in Appendix K.

Adjusted R^2 suggests that 13% of the variability in this data is explained by group x sex (multiple $R^2 = 22\%$). Furthermore 2% of the variability in this data is explained by just group membership. These results show that the real world effect of group membership is quite small. These results show that the girls in the sample were affected by PTSD, whereas the boys were not. This confirms our predictions on the sex differences that would be presented in Spatial Navigation.

Hypothesis 2: Summary and Conclusion

The analyses done in order to test hypothesis 2, showed that having a diagnosis of PTSD was indeed a significant factor on performance Spatial navigation. The analyses tended to support hypothesis 2b (those adolescents with a PTSD diagnosis will perform most poorly). Hypothesis 2a (adolescents who have experienced childhood trauma will, regardless of whether they are carrying a PTSD diagnosis or not, perform more poorly than non-trauma controls) could not be confirmed, as the trauma group's performance was not statistically different from the control group. Interestingly, the predictions on sex differences in this cognitive domain proved correct (i.e. an group effect was seen in the girls performance, whereas it was not seen in the boys performance.)

Testing Hypothesis 3

The following analyses tested hypothesis 3, namely, on the specific test of information-processing bias, adolescents with a diagnosis of PTSD will show a bias toward threat-related stimuli (i.e., they will show faster reaction times on tasks where such stimuli are targets, but will show slower reaction times on tasks where such stimuli are distracters); participants in the control group, in contrast, will show a bias toward positive-valence stimuli (i.e., they will show faster reaction times on tasks where such stimuli are targets, but will show slower reaction times on tasks where such stimuli are distracters). It is important to note that there is no prediction here about the performance of the trauma

group. The performance of this group is included in order to get a picture of their performance compared to the PTSD and the control group, not to test a specific hypothesis.

In order to test this hypothesis, a set of repeated-measures ANOVAs were carried on the Affective Go/No-Go task data, specifically the NoGo data and GoTarget data. Following this, a factorial ANOVA was carried out on the AGNG response bias (β) data.

Affective Go/No-Go Task: Emotional Information-Processing Biases

This task is designed to assess inhibition as well as possible biases that participants exhibit for processing information for different emotions. The analysis that follows looks specifically at the data related to information-processing biases (the inhibition data were analyzed as part of regression analyses described earlier).

The information-processing biases data from the AGNG were analyzed by looking six outcome measures: the average No-Go error rates for each of the three emotional conditions; the average reaction times for each of the two target emotions; and the Beta coefficients (which, as noted earlier, assess possible response bias; that is, they measure the tendency to either hit a target key too often or not often enough). Due to incomplete data sets and corrections made to maintain statistical assumptions, some of the participants' data had to be removed before the analyses described below could be completed. This strategy resulted in different sample sizes for each analysis, and therefore the descriptive statistics for analysis are presented in separate tables.

Testing Hypothesis 3: Affective Go/NoGo Task, No-Go Data

Table 17 shows the No-Go performance of participants in the three groups ($N = 47$), separated by sex. The three No-Go conditions were: *Fear* (fearful distractors with neutral targets); *Happy* (happy distractors with fearful targets); and *Neutral* (neutral distractors with fearful targets).

Table 17
Testing Hypothesis 3: AGNG NoGo Data

	Fear	Happy	Neutral
Control	0.34 (0.13)	0.31 (0.16)	0.29 (0.18)
Girls (<i>n</i> =10)	0.36 (0.12)	0.29 (0.13)	0.25 (0.18)
Boys (<i>n</i> =7)	0.31 (0.14)	0.34 (0.21)	0.35 (0.19)
Trauma	0.32 (0.14)	0.36 (0.10)	0.32 (0.21)
Girls (<i>n</i> =9)	0.31 (0.13)	0.38 (0.10)	0.28 (0.23)
Boys (<i>n</i> =5)	0.34 (0.18)	0.34 (0.11)	0.39 (0.17)
PTSD	0.32 (0.19)	0.39 (0.20)	0.30 (0.23)
Girls (<i>n</i> =10)	0.29 (0.22)	0.33 (0.18)	0.23 (0.20)
Boys (<i>n</i> =6)	0.38 (0.13)	0.51 (0.17)	0.44 (0.23)
All	0.33 (0.15)	0.35 (0.16)	0.30 (0.20)
Girls (<i>n</i> =29)	0.32 (0.16)	0.33 (0.14)	0.25 (0.20)
Boys (<i>n</i> =18)	0.34 (0.14)	0.39 (0.18)	0.39 (0.19)

Note. Data presented are average error rates for each emotional condition, with standard deviations in parentheses.

To analyze these data, repeated-measures ANOVA was performed. This analysis included two between-subjects factors (group membership and sex) and one within-subjects factor (emotional condition, which had three levels). The assumptions of normality and independence of observations were upheld, as was that of sphericity (the assumption of homogenous variances), as indicated by Mauchly's test, $\chi^2(2) = 2.70, p < 0.259$.

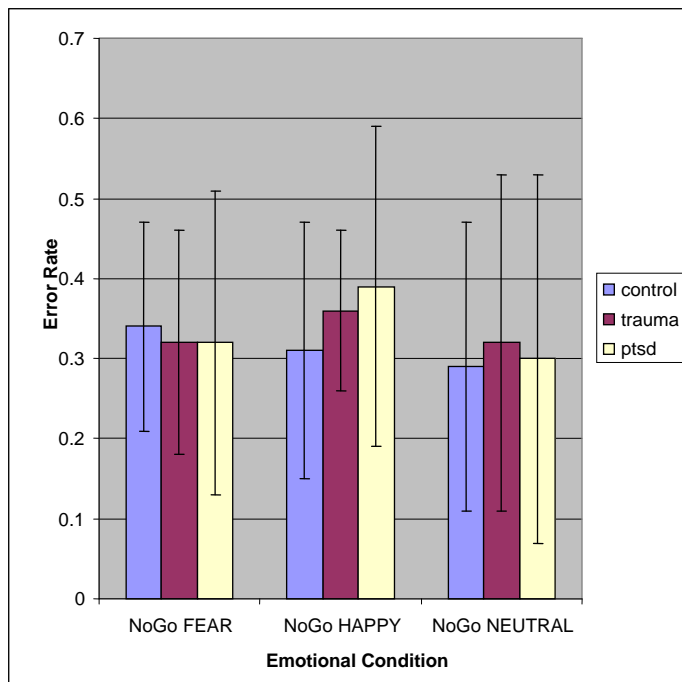


Figure 22. Average NoGo error rates across groups for each emotional condition. Error bars represent 95% confidence intervals.

Figure 22 shows average No-Go performance broken down by group and by emotional condition. Although it appears that participants in the three groups performed in quite a different pattern across the three emotional conditions, the statistical analysis showed that none of the main effects or the interaction effects were significant: emotional condition, $F(2, 41) = 1.19, p = 0.31$; sex, $F(1, 41) = 3.36, p = 0.07$; group, $F(2, 41) = 0.40, p = 0.67$; sex x group, $F(2, 41) = 1.05, p = 0.36$; emotional condition x sex, $F(2, 41) = 2.28, p = 0.11$; emotional condition x group, $F(4, 41) = 0.73, p = 0.58$; emotional condition x group x sex, $F(4, 41) = 0.44, p = 0.78$. There were, therefore, no statistically significant differences in the patterns of performances across groups for the No-Go conditions. This means that the three groups responded in much the same way no matter what emotion the distractor faces were. There was no significant response biases across the different emotions.

Testing Hypothesis 3: Affective Go/NoGo Task, Go Data

An examination of the data, conducted prior to the analysis, showed that, for these data, the assumption of sphericity of data was not upheld. Some of the participants' data was

therefore dropped by random selection, in order to have equal cell sizes in each group for both girls and boys. This step ensured that the repeated-measures analysis could be conducted effectively, even without the assumption sphericity being met.

Table 18 shows the Go performance of participants in the three groups ($N = 36$), separated by sex. The two Go-Target conditions were: *Fear* (fearful targets with both neutral and happy distractors); and *Neutral* (neutral targets with fearful distractors).

Table 18

Testing Hypothesis 3: AGNG Go-Target Data

	Fear	Neutral
Control	436.51 (53.66)	438.90 (45.64)
Girls ($n=6$)	451.91 (71.10)	456.62 (50.27)
Boys ($n=6$)	421.12 (26.67)	421.18 (36.08)
Trauma	433.06 (47.57)	450.08 (63.48)
Girls ($n=6$)	453.73 (52.80)	479.49 (59.19)
Boys ($n=6$)	412.40 (34.16)	420.66 (57.32)
PTSD	440.34 (73.75)	436.05 (92.40)
Girls ($n=6$)	471.10 (91.05)	477.01 (109.51)
Boys ($n=6$)	409.58 (37.47)	395.09 (52.59)
All	436.64 (57.75)	441.68 (68.13)
Girls ($n=18$)	458.91 (69.45)	471.04 (73.57)
Boys ($n=18$)	414.36 (31.48)	412.31 (48.16)

Note. Data presented are average reaction time for each target emotion, with standard deviations in parentheses.

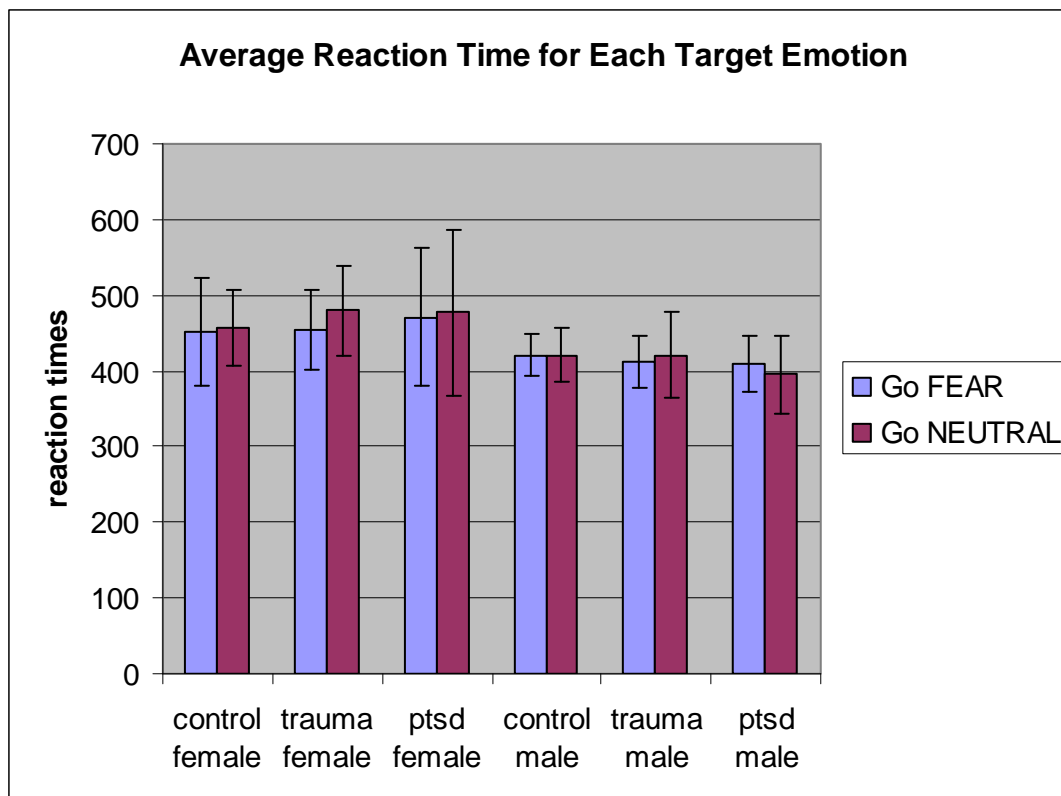


Figure 23. Average reaction time for each target emotion across the three groups. Error bars represent 95% confidence intervals.

Figure 23 shows average Go-Target performance broken down by group, sex, and emotional condition. In order to analyze this data set, repeated-measures ANOVA performed. This analysis included two between-subjects factors (group membership and sex) and one within-subjects factor (Go-Target emotional condition, which had two levels, fear and neutral). The assumptions of normality and independence of observations were upheld, but, as discussed earlier, the assumption of sphericity was not upheld for this data set.

As Figure 23 shows, on average, participants in all of the sub-groups (except for the control boys sub-group) performed slightly differently on each Go-Target condition. However, the statistical analysis showed that only sex had a significant effect on reaction time, $F(1,30) = 7.05$, $p < 0.013$, with the boys performing particularly faster than the girls, regardless of emotional condition). All the other effects were not statistically significant: emotional condition, $F(1, 30) = 0.70$, $p = 0.41$; group, $F(2, 30) = 0.02$, $p =$

0.98; sex x group, $F(2,30) = 0.33$, $p = 0.72$; emotional condition x sex, $F(1,30) = 1.39$, $p = 0.25$; emotional condition x group, $F(2, 30) = 1.09$, $p = 0.35$; emotional condition x group x sex, $F(2, 30) = 0.16$, $p = 0.85$. . This means that the three groups responded in much the same way no matter what emotion the target faces were. There was no significant response biases, in terms of response time for the different target emotions.

Testing Hypothesis 3: Affective Go/NoGo Task, Beta

The last set of AGNG data that were analyzed involved the Beta coefficient, which, as described above, measures response bias. Table 19 shows the average Beta coefficient (β) for the entire sample ($N = 49$) and for each group, separated by sex.

Table 19
Testing Hypothesis 3: AGNG Response Bias Data

	Beta (β)
Control	-0.38 (0.45)
Girls ($n=10$)	-0.42 (0.45)
Boys ($n=7$)	-0.32 (0.48)
Trauma	-0.50 (0.37)
Girls ($n=10$)	-0.53 (0.43)
Boys ($n=6$)	-0.45 (0.26)
PTSD	-0.33 (0.77)
Girls ($n=10$)	0.04 (0.50)
Boys ($n=6$)	-0.95 (0.77)
All	-0.40 (0.55)
Girls ($n=30$)	-0.30 (0.51)
Boys ($n=19$)	-0.56 (0.58)

Note. Data presented are average Beta coefficients, with standard deviations in parentheses.

To investigate the presence of between-group differences and interaction effects, these data were analyzed by means of a two-way factorial ANOVA. This analysis included two between-subjects factors (group membership and sex). The assumptions of normality and of homogeneity of variances were not violated for this data set (Levene's test, $F(5,43) = 0.77$, $p = 0.58$).

The analysis showed that there were no statistically significant main effects (group, $F(2, 43) = 0.25, p = 0.78$; sex, $F(1, 43) = 3.51, p = 0.07$). There was, however, a statistically significant group x sex interaction effect, $F(2, 43) = 6.00, p < 0.005$. This interaction is presented graphically in Figure 24.

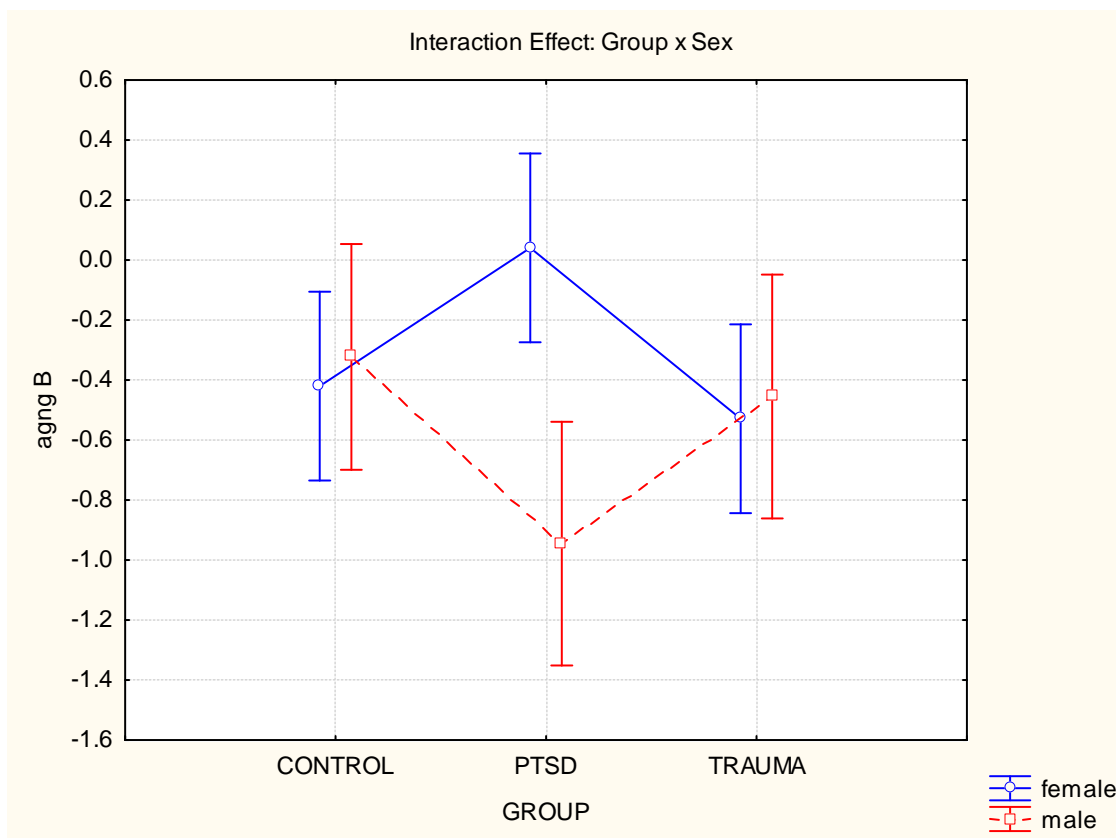


Figure 24. AGNG Beta: interaction between group membership and sex. Error bars represent 95% confidence intervals.

Post-hoc analysis of the interaction effect indicated significant differences between participants in the boy girl PTSD groups ($p < 0.005$), with boys being far more biased toward hitting the target key often ($\beta = -0.95$); girls showed a smaller bias, which was interestingly in the opposite direction ($\beta = 0.04$). This means that the boys in the PTSD group were most biased in their responses, as they tended to just hit the response key as much as possible. Conversely, the girls in the PTSD group were least biased, as they did only sometimes hit the response key too little.

There were no other statistically significant between-group difference, i.e. there were no sex differences across the groups (control, trauma, and PTSD).

Hypothesis 3: Summary and Conclusion

The analyses done in order to test hypothesis 3 (on the specific test of information-processing bias, adolescents with a diagnosis of PTSD will show a bias toward threat-related stimuli, whereas participants in the control group, in contrast, will show a bias toward positive-valenced stimuli), showed that having a diagnosis of PTSD did not affect information-processing biases. The analyses therefore did not support the hypothesis.

Testing Hypothesis 4

The analyses that follow tested hypothesis 4, namely: in the group of adolescents who have experienced childhood trauma, poorer performance on the general neuropsychological test battery will be positively correlated with (a) a longer time since the traumatic experience; (b) a greater number of post-traumatic symptoms (e.g., more avoidance, more numbing, more hyperarousal, more hypervigilance); (c) a higher level of symptom severity; and (d) a greater level of overall functional impairment.

In order to test this hypothesis, a set multiple hierarchical regression analyses were carried out on the trauma and PTSD participants data from the MINI KID 5.0 and the PDS.

Testing Hypothesis 4: Multiple Regression of Trauma and PTSD Data

A set of hierarchical multiple regression analyses was performed using the neuropsychological domain z score data from the trauma and the PTSD participants ($n = 32$) as outcome variables, and characteristics of the trauma and participant's response to the trauma (as measured by the MINI KID 5.0 and the PDS) as predictor variables. These analyses were conducted, therefore, in order to assess the possible effects of the characteristics of the trauma (namely, how many months ago the trauma occurred, and

whether the perpetrator was a stranger, if applicable), and the participant's response to the trauma (such as the number of symptoms experienced and the severity of the symptoms) on the participants' neuropsychological test performance. This analysis did not include group membership as a predictor variable, as it is the data from the participant's response to the trauma that directly determined group membership. Once again, Full Scale IQ and the sex of the participant were entered at the first step of the regression; all the other variables were entered at the second step. This strategy was used in order to ascertain the effects of these variables after controlling for the effects of participant Full Scale IQ score and sex.

The independent variable of perpetrator (i.e., whether the perpetrator was a stranger or was known to the participant as the time of the trauma) was only applicable to twenty of the participants. Therefore, a separate set of regression analyses were performed to investigate whether this factor was statistically significantly associated with fluctuations in neuropsychological test performance. The results showed that this variable was not a significant predictor for performance any of the domains (p values ranged from 0.41 to 0.92, multiple R^2 ranged from 0.0002% to 0.02%, and β scores ranged from -0.0169 to 0.161.). The full set of results can be seen in Appendix L.

The final independent variables in the primary analysis on trauma characteristics and responses were:

Step 1: participant sex and Full Scale IQ score;

Step 2: Number of months since trauma; avoidance and numbing symptom score (derived from score on MINI KID 5.0, PTSD section, question 4); hyperarousal/ hypervigilance symptom score (derived from score on MINI KID 5.0, PTSD section, question 5); level of impairment, number of symptoms, and symptom severity (all as measured by the PDS).

The results showed that after controlling for the effects of sex and Full Scale IQ, two domains were significantly affected by at least one of the other factors (i.e., those added in at step 2).

Testing Hypothesis 4: Two Domains Significantly Affected by one of the Step 2 Variables

In the domain of *Decision-making/impulsivity*, MINI KID Question 5 scores were significantly associated with performance, $\beta = 1.009$, $p < 0.013$. All the step 2 variables together (separate from Full Scale IQ score and sex) accounted for 31 % of the variability of performance in *Decision-making/impulsivity*. The overall regression model (with all independent variables) was not statistically significant fit, $F(8, 23) = 1.521$, $p = 0.204$.

In the domain of *Processing speed*, MINI KID question 5 scores were significantly associated with performance, $\beta = -1.320$, $p < 0.003$. All the step 2 variables together (separate from Full Scale IQ score and sex) accounted for 32 % of the variability of performance in *Processing speed*. The overall regression model (with all independent variables) was however, not a statistically significant fit, $F(8, 23) = 1.921$, $p = 0.105$.

Seven of the domains were not significantly associated with any one of the factors entered at step 2 of the analysis (i.e., with any one of the trauma characteristics/response to trauma variables):

- (1) In the domain of *Visual memory*, only Full Scale IQ score was a significant factor; however, 12 % of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for *Visual memory* was statistically significant, $F(8, 23) = 5.740$, $p < 0.0001$.
- (2) In the domain of *Working memory*, only Full Scale IQ score was a significant factor, and only 4% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for *Working memory* was statistically significant, $F(8,23) = 4.418$, $p < 0.003$.
- (3) In the domain of *Verbal memory*, only Full Scale IQ score was a significant factor, although 14% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for *Verbal memory* was statistically significant, $F(8,23) = 4.310$, $p < 0.003$.

- (4) In the domain of *Spatial navigation*, only Full Scale IQ score was a significant factor, although 11% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for Spatial navigation was statistically significant, $F(8,23) = 6.984, p < 0.0001$.
- (5) In the domain of *Inhibition*, only Full Scale IQ score was a significant factor, although 15% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for Inhibition was statistically significant, $F(8,23) = 2.557, p < 0.037$.
- (6) In the domain of *Rule acquisition*, only Full Scale
- (7) Full Scale IQ score was a significant factor, although 13% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for Rule acquisition approached statistical significance, $F(8,23) = 2.327, p < 0.054$.
- (8) In the domain of *Problem-solving*, there were no significant factors, and only 7% of the variability in test performance in this domain was explained by trauma characteristics/response to trauma variables factors alone. The overall model for problem-solving was not statistically significant, $F(8,23) = 0.801, p < 0.608$.

The regression R^2 results are presented in Table 20.

Table 20
Testing Hypothesis 4: Trauma and PTSD Regression R² Results for each Neurocognitive Domain.

	Step 1 R ²	ΔR^2	Step 2 R ²	F to enter/remove	p-level
Working memory	0.549	0.042	0.591	0.392	0.876
Verbal memory	0.458	0.141	0.600	1.354	0.274
Visual memory	0.545	0.122	0.666	1.396	0.258
Spatial navigation	0.603	0.105	0.708	1.383	0.263
Rule acquisition	0.319	0.129	0.447	0.891	0.517
Inhibition	0.319	0.152	0.471	1.097	0.394
Problem-solving	0.144	0.074	0.218	0.363	0.895
Processing speed	0.079	0.321	0.400	2.055	0.099
Decision-making/Impulsivity	0.039	0.307	0.346	1.799	0.144

Lastly, it must be noted that although “months since trauma” was not a significant factor in any of the domains, the β coefficients of this factor in most of the domains suggested that the length of time since trauma was negatively correlated with performance, Working memory: $\beta = -0.108$, $p = 0.560$, Verbal memory: $\beta = -0.235$, $p = 0.206$, Spatial navigation: $\beta = -0.065$, $p = 0.676$; Rule acquisition: $\beta = -0.023$, $p = 0.916$; Inhibition: $\beta = -0.031$, $p = 0.576$; Problem-solving: $\beta = -0.202$, $p = 0.433$; Processing speed: $\beta = -0.074$, $p = 0.740$; Impulsivity: $\beta = -0.131$, $p = 0.576$. This suggests that the more time that had passed since the trauma the poorer performance in these domains. (Only Visual memory resulted in a positive correlation, $\beta = 0.106$, $p = 0.526$.)

Hypothesis 4: Summary and Conclusion

The analyses done in order to test hypothesis 4 (namely, that poorer performance on the general neuropsychological test battery would be positively correlated with increased severity and number of symptoms as well as level of impairment and length since trauma), confirmed that specifically, number of symptoms were significant predictors on two domains (Processing speed and Decision-making/impulsivity). Moreover, the effect sizes indicate that all the factors assessed accounted for a good portion of the variability of the participants' performance

Other Analyses

Testing Effects of Personal Characteristics and Traumatic Events

A last set of hierarchical multiple regression analyses was performed in order to assess the possible associations between the participants' personal characteristics and traits, as well as the effects of traumatic events, as reported by CTQ-SF, on the neuropsychological test performance. The personal characteristics and traits measured here were: general anxiety (as measured by the STAI Trait), perceived everyday stress (as measured by the LEQ), approach avoidance/withdrawal tendencies (as measured by the PANAS), and resiliency (as measured by the CD-RISC). As these variables are significantly correlated with group membership, both theoretically and statistically (correlations ranged from -0.01 to 0.52), these regression analyses did not include group membership as a factor.

The independent variables in this analysis, in the order in which they were entered into the regression equation, were:

Step 1: participant sex and Full Scale IQ score;

Step 2: LEQ score (past 6 months) and LEQ score (more than 6 months ago);

Step 3: CTQ-SF Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect index scores

Step 4: PANAS positive score, PANAS negative score

Step 5: STAI Trait score

Step 6: CD-RISC score

In the analyses, Full Scale IQ and sex of the participant were entered on the first step of the regression, and the other independent variables (personal characteristics and traits) were entered on the next steps. This strategy was adopted in order to ascertain the effects of these latter variables after controlling for the effects of Full Scale IQ and sex. The personal characteristics and trait variables were grouped together and ordered in the hierarchy in this way in order to assess the specific R^2 associated with each set.

The regression results showed that after controlling for the effects of sex of the participant and Full Scale IQ scores, three domains were significantly affected by at least one of the other factors (i.e., the personality characteristics and trait variables included at steps 2, 3, 4, 5 and/or 6 of the analysis).

The Three Domains Significantly Affected by One of the Step 2 to 6 Variables

In the domain of *Spatial navigation*, STAI Trait scores were significantly associated with test performance, $\beta = 0.490$, $p < 0.037$. All of the personal characteristics and trait variables together (steps 2 to 6), separate from Full Scale IQ and sex, accounted for 12% of the variability in performance on Spatial navigation tasks. The overall regression model (i.e., that including all independent variables) was a statistically significant fit, $F(13, 35) = 5.3203$, $p < 0.0004$. (It is worth noting here that Full Scale IQ score was, by itself, also a significant predictor).

In the domain of *Problem-solving*, PANAS negative scores (avoidance/withdrawal tendencies) were significantly associated with test performance, $\beta = -0.147$, $p < 0.016$. All of the personal characteristics and trait variables together (steps 2 to 6), separate from Full Scale IQ and sex, accounted for 37% of the variability in performance on Problem-solving tasks. The overall regression model (i.e., that including all independent variables) was a statistically significant fit, $F(13, 35) = 2.626$, $p < 0.012$. (Again, it is worth noting here that the sex of the participant was, by itself, also a significant predictor).

In the domain of *Rule acquisition*, PANAS positive scores (approach tendencies) were significantly associated with test performance, $\beta = 0.411$, $p < 0.032$. All of the personal characteristics and trait variables together (steps 2 to 6), separate from Full Scale IQ and sex, accounted for 18% of the variability in performance on Rule-acquisition tasks. The overall regression model (i.e., that including all independent variables) was a statistically significant fit, $F(13, 35) = 2.516$, $p < 0.015$. (Once again, it is worth noting here that Full Scale IQ was, by itself, also a significant predictor).

Six of the domains were not significantly associated with any one of the personal characteristics and trait factors:

(1) *Working memory* was only significantly predicted by Full Scale IQ scores and the sex of the participant, and only 4% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Working memory was statistically significant, $F(13,35) = 3.886, p < 0.0007$.

(2) *Verbal memory* was only significantly predicted by Full Scale IQ scores, although 18% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Verbal memory was statistically significant, $F(13,35) = 3.444, p < 0.002$.

(3) *Visual memory* was only significantly predicted by Full Scale IQ scores, although 12% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Visual memory was statistically significant, $F(13,35) = 5.610, p < 0.00002$.

(4) *Inhibition* was only significantly predicted by Full Scale IQ scores, although 12% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Inhibition was significant, $F(13,35) = 2.602, p < 0.012$.

(5) *Processing speed* was not significantly predicted by any of the factors, and only 6% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Processing speed was not statistically significant, $F(13,35) = 0.359, p = 0.974$.

(6) *Decision-making/impulsivity* was not significantly predicted by any of the factors, although 15% of the variability in test performance in this domain was explained by the personal characteristics and trait factors together (separate from Full Scale IQ and sex). The overall model for Decision-making/impulsivity was not statistically significant, $F(13,35) = 0.632, p = 0.811$.

The effect sizes associated with the statistics reported in these analyses are presented in Table 21. The change in R^2 at each step (and the corresponding F to enter/remove and p values) in the regression analyses is reported in Table 22.

Table 21

Testing Effects of Personal Characteristics and Traumatic Events: Regression R^2 Results for each Domain

	Step 1 R^2 (IQ and sex)	Overall ΔR^2 (Steps 2 to 6)	Final R^2
Working memory	0.555	0.036	0.591
Verbal memory	0.386	0.176	0.561
Visual memory	0.559	0.117	0.676
Spatial navigation	0.545	0.119	0.664
Rule acquisition	0.303	0.180	0.483
Inhibition	0.372	0.119	0.491
Problem-solving	0.125	0.369	0.494
Processing speed	0.063	0.055	0.118
Decision-making/Impulsivity	0.039	0.151	0.190

Table 22

Testing Effects of Personal Characteristics and Traumatic Events: Regression Results for Steps 2, 3, 4, 5, and 6

	Working memory	Verbal memory	Visual memory	Spatial navigation	Rule acquisition	Inhibition	Problem solving	Processing speed	Decision- making/ Impulsivity
Step 2 ΔR^2	0.004	0.033	0.041	0.032	0.031	0.023	0.090	0.014	0.089
<i>F</i> to enter/ remove	0.175	1.249	2.274	1.656	1.022	0.830	2.532	0.330	2.236
<i>p</i> -level	0.840	0.297	0.115	0.203	0.368	0.443	0.091	0.721	0.119
Step 3 ΔR^2	0.016	0.069	0.034	0.035	0.042	0.045	0.098	0.035	0.037
<i>F</i> to enter/ remove	0.286	1.056	0.725	0.714	0.530	0.633	1.108	0.306	0.342
<i>p</i> -level	0.918	0.399	0.609	0.617	0.752	0.676	0.372	0.907	0.884
Step 4 ΔR^2	0.010	0.045	0.012	0.007	0.060	0.012	0.104	0.002	0.022
<i>F</i> to enter/ remove	0.434	1.782	0.642	0.317	1.973	0.414	3.286	0.041	0.499
<i>p</i> -level	0.651	1.183	0.532	0.731	0.153	0.664	0.049*	0.960	0.611
Step 5 R^2	0.004	0.006	0.028	0.040	0.041	0.004	0.020	0.004	<0.001
<i>F</i> to enter/ remove	0.338	0.465	3.143	4.227	2.791	0.250	1.295	0.178	0.001
<i>p</i> -level	0.565	0.500	0.085	0.047*	0.103	0.620	0.263	0.676	0.982
Step 6 ΔR^2	0.003	0.022	0.001	0.005	0.006	0.035	0.057	<0.001	0.004
<i>F</i> to enter/ remove	0.277	1.743	0.083	0.536	0.426	2.409	3.953	0.0002	0.174
<i>p</i> -level	0.602	0.195	0.775	0.469	0.518	0.130	0.055	0.990	0.679

Note. Step 2: LEQ scores; Step 3: CTQ-SF scores; Step 4: PANAS scores; Step 5: STAI Trait scores, and Step 6: CD-RISC scores.

* $p < 0.05$

As shown in Table 22, only two of the steps (from step 2 to 6) in two domains added statistically significantly to the fit of the model. These significant steps were in the domains of:

- (1) *Problem-solving*: Step 4 (PANAS scores) was a statistically significant step: change in $R^2 = 0.104$, F to enter/remove = 3.286, $p < 0.049$; and
- (2) *Spatial navigation*: Step 5 (STAI Trait scores) was a statistically significant step: change in $R^2 = 0.040$, F to enter/remove = 4.227, $p < 0.047$.

Other Analyses: Summary and Conclusion

The analyses done in order to test the effects of personal characteristics and traumatic events (as assessed by the CTQ-SF), showed that higher general anxiety was associated with poorer performance on Spatial navigation tasks, higher withdrawal tendencies was associated with poorer Problem-solving abilities, and lastly that higher approach tendencies was associated with better performance on Rule-acquisition tasks.

DISCUSSION

This study aimed to explore the effects that childhood trauma has on adolescents' neuropsychological functioning, and to explore whether the diagnosis of PTSD allows for unique predictions about that domain of functioning, over and above predictions made on the basis of trauma exposure alone. In so doing, the research explored the effects that the characteristics of the trauma, the individual's response to the trauma, and the personal characteristics of the individual, have on neuropsychological functioning in adolescents. Additionally, this study sought to provide a profile of test performance that can be used to distinguish victims of trauma. The neuropsychological profile of trauma victims will be discussed by exploring the performance exhibited by participants in the three groups, with the eventual aim of identifying a subset of tests that can be used to distinguish those exposed to trauma from those with no such exposure. The statistical analyses provided some important information which will be discussed in terms of these aims. Furthermore, the discussion of data obtained in the current study will attempt to provide some clarity with regard to the discrepancies and gaps seen in the previously published literature.

There were four specific hypotheses that were investigated in the current study:

Hypothesis 1: on the general neuropsychological test battery: adolescents who have experienced childhood trauma will perform more poorly than non-trauma controls, and of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly: *Hypothesis 2:* on the specific test of Spatial navigation: adolescents who have experienced childhood trauma will perform more poorly than non-trauma controls, and of those adolescents who have experienced childhood trauma, those with a PTSD diagnosis will perform more poorly: *Hypothesis 3:* On the specific test of information-processing bias, adolescents with a diagnosis of PTSD will show a bias toward threat-related stimuli whereas participants in the control group will show a bias toward positive-valence stimuli: *Hypothesis 4:* In the group of adolescents who have experienced childhood trauma, poorer performance on the general neuropsychological test battery will be positively correlated with increased severity and number of symptoms as well as level of impairment and length since trauma.

Furthermore, it is predicted than on many of the tests, there will be marked sex differences in the effects that trauma exposure and PTSD has on performance.

Hypothesis 1: The Effects of Trauma and PTSD on Performance in the Test Battery

The various statistical analyses conducted here showed that group membership had a significant effect on performance in only one of the composite cognitive domains: Inhibition (a prefrontal cortex-dependent task).

Cognitive Domains Affected

Regression analyses demonstrated that group membership was associated with poorer performance on the tests in Inhibition. In neuropsychological terms, inhibition is a cognitive ability that is generally included under the rubric of executive functioning; in neural terms, the cognitive processes that describe inhibition are subserved by the prefrontal cortex.

Within the group of tests included in the domain of Inhibition, there were particularly strong between-group differences on the switching task in the Inhibition subtest of the NEPSY-II. Specifically, participants in both the PTSD group and in the trauma group performed considerably more poorly than those in the control group. However, in both the overall domain of Inhibition and the individual test of switching, this *significant* effect was specifically seen in the performance of the trauma group (the control vs. trauma factor).

(As one of the aims of this study was to provide a profile to distinguish victims of trauma from people who had not experienced trauma based on neuropsychological test performance, it is important to note that these results suggest that the test of switching would provide the most reliable prediction. The equation for this prediction is seen in the in the testing of Hypothesis 2: post-hoc regression analysis of individual outcome measures of Inhibition section).

The results of our analyses on Inhibition, namely a significant group membership factor for the trauma participants, confirmed the *a priori* hypothesis that participants in the trauma group would perform more poorly than those in the control group on tasks dependent on prefrontal cortex function. The data did not confirm the hypotheses stating that participants in the PTSD group would perform significantly more poorly than those in the trauma group, or more poorly than all other participants.

The association between trauma exposure and poorer performance on tests of Inhibition, in previously published literature is not clear; the few studies in this research area that have examined performance on tests of Inhibition have reported some conflicting results. For example, Braunstein-Bercovitz, Dimentman-Ashkenazi, and Lubow (2001) found significant differences in response inhibition performance of people experiencing stress versus that of controls, whereas Kanagaratnam and Abjorsen (2007) found no such differences. Both of those studies, however, were conducted with adult participants.

The results of the overall primary regression models suggested that the trauma group did worse than the PTSD group (as seen in the standardized β coefficients, and in the fact that the performance of PTSD participants was not significantly different to that of control participants, as seen in the non-significant PTSD group factor). However the overall regression models controlled for Full Scale IQ scores and sex of the participant and consequently the final results suggest a different pattern of performance compared to the raw data.

If one looks solely at the average Inhibition domain scores (as well as the average switching scores), as seen in Table 9, the PTSD group did in fact perform worse than the trauma group. This pattern of performance lends support for the *a priori* hypothesis which predicted that participants with PTSD participants would perform worse than all other participants, and suggests that with a larger sample this hypothesis would be confirmed. This discussion applies to both the overall domain of Inhibition as well as to the individual outcome measure of switching on the Nepsy-II Inhibition subtest.

Of most importance in assessing the effects of group membership on performance, are the effect sizes of group membership in the analyses, which provides a measure of practical significance. Group membership (both group membership factors together) accounted for 6.5% of the variability in Inhibition; a relatively modest proportion of the variance is thus explained by group membership. Similarly, group membership accounted for 6.3% of the variability in switching task.

It is important to note, that although not many domains in the analyses returned significant group membership results, Inhibition, and specifically cognitive flexibility (an ability which the Nepsy-II Inhibition subtest measures), is considered by some researchers to be the core feature of intact executive function (Goldstein, 1990). Furthermore, is important to note that response inhibition is subserved by the ventral region of the PFC. These results of this study therefore suggest that it is this region in the PFC that is most affected by trauma.

This association between a history of trauma exposure and poorer performance on tests of executive functioning (such as the test of Inhibition) is consistent with previous research on neuropsychological deficits in adults with a history of trauma exposure (e.g., Roozendaal, Jayme, & McGaugh, 2004; Weber and Reynolds, 2004), and provides supports for the theoretical framework built upon data from neuroimaging studies examining the effects of stress on the PFC (Roozendaal, Jayme, & McGaugh, 2004). Furthermore, the current data in this regard are consistent with those from similar research done on adolescent samples: Beers and De Bellis (2002) found deficits in executive functioning (although the deficits found were specifically in Attention, Semantic organization, and Problem-solving). The current study could not, however, replicate the exact findings of Beers and De Bellis (2002).

Cognitive Domains Not Affected

Although Semantic organization was not assessed in this study, Problem-solving *was* assessed in this study. In the domain of Problem-solving the control participants did perform best, with the trauma participants performing most poorly (as seen in the average

Problem-solving success rates and domain scores, as well as the Beta standardized coefficients for the group membership factors in the regression results). These results suggest that a larger sample size would lead to significant findings in this domain, however the PTSD group did not perform the worst, as was the case in the Beer and De Bellis (2002) study. These data provides support for the *a priori* hypothesis stating that the PTSD group would perform most poorly.

Attention was also assessed in this study, in the pre-shift errors (errors made in the pre-shift stages of the CANTAB Intra-Extra Dimensional Set Shift task) in the domain of rule acquisition and attentional set shift. For this individual outcome measure the PTSD participants performed the most poorly (making the most errors) which again suggests that a larger sample size would lead to confirmation of the *a priori* hypothesis, and is consistent with the PTSD literature, Notably, the trauma participants performed the best in this task, which is directly contradicting the *a priori* hypothesis which stated that the trauma participants would perform significantly worse than the controls.

Similarly, this study could not replicate the findings of Moradi et al. (1999) of overall memory deficits associated with PTSD in adolescents. In fact, this study did not find significant differences for any of the measures of memory assessed in the test battery. The pattern of performance however, showed that the PTSD group performed the worst out of the three groups, on all three measures of memory that were assessed, namely: Working memory, Verbal memory, and Visual memory. This leads to the conclusion that a larger sample size may had lead to significant findings, similar to those found by Moradi, Neshat-Doost et al. (1999), and tends to support the *a prior* hypothesis regarding the PTSD participant's performance

The trauma group performed worse than the controls on measures of Working and Visual memory, but *better* than controls on measures of Verbal memory. *This* finding, as well as the results of the attention task (and seen in the domain of Decision-making/impulsivity), where the trauma participants show superior performance, is particularly interesting.

Hypothesis 1, based on the theory of the effects of traumatic stress on the prefrontal cortex and the hippocampus, and based on adult research on the neuropsychological effects of PTSD and trauma, proposed that group membership would affect *all* measures of prefrontal cortex and hippocampus functioning. However, as can be seen in the preceding discussion, this was not found in this study (only Inhibition was statistically significantly affected). Along with the domains already discussed, the analyses of the domains of Rule acquisition, Processing-speed, and Decision-making/impulsivity also reflected that group membership was not a significant factor in these domains. The analysis in the domain of Spatial navigation also reflected that, group membership was not a significant factor in this domain. This composite domain was made up of only the CG Arena invisible trails and the probe trail data, therefore a significant group membership result was expected, as seen in Hypothesis 2. Indeed the effect of group membership was evident, however the effect was too small for significant findings. These significant findings of group membership are however seen in a more thorough exploration of the CG Arena data, which is explored later in the discussion of the testing of Hypothesis 2.

The fact that group membership was not significantly associated with poorer performance on most of the composite cognitive domains is further reinforced by the particularly small effect sizes of group membership in the regression analysis testing Hypothesis 1.

The Role of IQ in PTSD and Trauma Exposure

In this study the role of IQ was particularly important. In the regression analysis, Full Scale IQ score was a significant factor in the majority of the cognitive domain assessed (all except Problem-solving, Processing speed and Decision-making/impulsivity.) In these significant domains, Full Scale IQ score, along with sex of the participant, produced particularly large effect sizes (ranging from 30% to 56%). These findings show the magnitude of the role of Full Scale IQ in neuropsychological test performance.

In the testing of Hypothesis 1, in the regression analysis, Full Scale IQ scores were controlled for. After controlling for these scores, the group membership effect in the

domain of Inhibition remained significant, however, group membership in the domain of Spatial navigation was not significant. As will be discussed shortly in the discussion of the testing of Hypothesis 2, group membership did in fact have a significant effect in many of the outcome measures in the ANOVA analyses of Spatial navigation (where participants in the PTSD group performed most poorly compared to controls). These analyses did not control for Full Scale IQ scores. This implies that a primary reason why the participants in PTSD group did most poorly is due to the fact that their Full Scale IQ scores were lower than the control group. However, as discussed in the introduction, research assessing the relationship between IQ and PTSD has found that lower IQ may in fact be a symptom of PTSD (Jenkins et al., 2000; Saigh et al., 2006). Furthermore lower IQ may be a preceding risk factor for PTSD (Koenen et al., 2006; McNally, 2003; Saltzman, Weems, and Carrion, 2006). These studies suggest that the confounding results of IQ may be misleading.

Hypothesis 2: The Effects of Trauma and PTSD on Performance of Spatial Navigation Tasks

The seminal study in the field of the effects of trauma (specifically PTSD) on adolescents, conducted by Beers and De Bellis (2002) found deficits in adolescents' visual-spatial abilities. Notably, this was assessed by considerably different tests to the ones employed in the current study (e.g., they used the Judgment of Line Orientation test; Spreen & Strauss, 1998). Their results were, however, not significant after corrections were applied for multiple comparisons. Although these previous results were not significant (also seen in the testing of hypothesis 1), the following analyses measured Spatial navigation in a far more detailed way, taking into account many complexities which were not done in the previous study, and in so doing discovered some important results.

Although not all the results from the ANOVA analyses of the CG Arena data were statistically significant, a thorough discussion is necessary to understand the overall picture of how group membership affected Spatial navigation in this sample.

Testing Hypothesis 2: CG Arena, Visible Target Trials

Analyses of the visible-target trials data analysis showed that there was no group membership effect on these data (i.e. participants in the control group, the trauma group, and the PTSD group all performed in a statistically similar manner). These data confirmed *a priori* predictions: The visible-target trials task does not require any kind of cognitive mapping ability for successful completion, and therefore those participants with putative hippocampal dysfunction should perform just as well as those with intact hippocampal functioning (i.e., there should be no effect of group membership on the ability to complete visible-target trials successfully). These findings are consistent with previous research conducted on Spatial navigation in the CG Arena (e.g., Thomas et al., 2010).

Testing Hypothesis 2: CG Arena, Invisible Target Trials

Analyses of the invisible-target data showed that there was a significant group membership effect on this task. On these trials, participants in the PTSD group performed significantly more poorly than did those in the control group (i.e., they took a much longer path length to reach the target). The performance of participants in the trauma group was not statistically significantly different from that of participants in either of the other two groups, although they did perform slightly better than those in the PTSD group and slightly worse than those in the control group). These data, then, confirmed the *a priori* hypothesis that participants in the PTSD group would perform most poorly on the invisible-target trials due to the cognitive mapping demands of the task and their putative hippocampal dysfunction. The data did not confirm the hypotheses stating that participants in the PTSD group would perform significantly more poorly than those in the trauma group, and that those in the trauma group would perform significantly more poorly than those in the control group. Nonetheless, the pattern of performance suggested confirmation of this hypotheses might be possible with a larger sample. The finding of a statistically significant group membership effect on a cognitive map-based spatial navigation task, at least with respect to the comparison of PTSD with control participants, is consistent with previous research (e.g., Hartley, Maguire, Spiers, & Burgess, 2003; Schwabe et al., 2007; Thomas et al., 2010).

Testing Hypothesis 2: CG Arena, Probe Trial

Analyses of the probe trial data showed that group membership did not have a statistically significant effect on this measure of persistence of search and robustness of cognitive mapping. The trend in performance (the controls spent the most time in the target quadrant and the PTSD group spent the least time in the target quadrant, out of the three groups) is in the correct direction, following the *a priori* hypotheses. It is possible, therefore that with more power (i.e., a larger sample this finding would reach statistical significance). Although the findings were not significant, the trend exhibited here, suggesting a group membership effect on a cognitive map-based spatial navigation task, at least with respect to the comparison of PTSD with control participants, is consistent previous research (e.g., Hartley, Maguire, Spiers, & Burgess, 2003; Schwabe et al., 2007; Thomas et al., 2010).

Hypothesis 3: The Effects of Trauma and PTSD on Information-Processing Bias

The various statistical analyses conducted here showed that group membership did not have a significant effect on biases for emotional information-processing.

Analyses of the AGNG task showed that there was no significant group membership effect on the NoGo error rates, the Go reaction times or the Response bias data (as measured by the β statistic). These findings therefore did not support the *a priori* hypothesis that individuals with PTSD will present a significantly different set biases than control participants (where the individuals with PTSD will be biased towards threat-related stimuli, in this case angry faces, and that controls will be biased towards positively-valenced stimuli, in this case happy faces). Analyses of the data obtained on these measures, showed that the patterns of performance were in fact opposite to the *a priori* predictions made based on the literature (Ladouceur et al., 2006; Schultz et al., 2006; Waters & Valvoi, 2009).

Hypothesis 4: The Effects of Trauma Characteristics and Trauma Responses on Performance

The various statistical analyses conducted here on the trauma and PTSD data showed that PTSD symptom clusters (specifically avoidance and numbing), was significantly associated with poorer performance on two domains, namely: Decision-making/impulsivity and Processing speed.

In Decision-making/impulsivity, higher avoidance/numbing symptoms were significantly associated with higher scores on this domain. This means that participants with higher symptom scores were in fact less impulsive, and made less risky decisions. In Processing speed, avoidance/numbing symptom scores were significantly associated with lower scores on this domain. This means that participants with higher symptom scores were in fact slower in processing information.

The factor of ‘months since trauma’ was not significantly associated with performance in any of the domains. However, as mentioned in the results, the regression results suggested that the length of time since trauma was *negatively* associated with performance of eight of the domains (all but Visual Memory). This means that the longer time that passed since the trauma, the more poorly individuals did in these domains. This provides support for the a priori hypothesis.

The effect sizes in this analysis go further to suggest the role that PTSD symptoms have on performance, ranging from 4% for Working memory to 32% for Processing speed. These effect sizes must be read with caution, however, as they include the effect of the trauma characteristic, ‘months since trauma.’

Other Analyses of Interest: The Role of Other Factors in Test Performance

The secondary regression analyses, which assessed whether perceived everyday stress (as assessed by the LEQ) and general anxiety (as assessed by the STAI 2 trait questionnaire) had an impact on functioning, showed that Spatial navigation was associated with higher general anxiety levels. However, these findings could be due to the fact that the PTSD

participants had significantly higher general anxiety levels than all the other participants, and therefore it could have the presence of PTSD that led to both the increased anxiety levels as well as the decreased performance on this domain. The results of the analyses suggested that perceived everyday stress and stress felt on the day of testing had no impact on test performance.

These analyses also assessed whether personal characteristics and attributes such as approach avoidance/withdrawal tendencies (as assessed by the PANAS) and resiliency (as assessed by the CD resiliency questionnaire) had an effect on performance. The results showed that more avoidance/withdrawal tendencies were significantly associated with poorer performance on the domain of Problem-solving, and that less approach tendencies were significantly associated with poorer performance on the domain of Rule Acquisition. Once again, these findings could be due to the fact that the PTSD participants had significantly higher avoidance/withdrawal tendencies and also lower approach tendencies (although *these* differences were not significant), and therefore it was the presence of PTSD that led to both the tendencies, as well as the decreased performance on these domains. The results suggested that resiliency had no impact on test performance.

Sex Differences in the Effects of Trauma Exposure and PTSD on Test Performance

The role that the sex of the participant plays in test performance, looking particularly at the interaction between sex and group membership is of interest in this study. This is looked at generally under the prediction that suggests that in many domains there will be marked sex differences in the effects that trauma exposure and PTSD has on performance.

Although sex of the participant was a significant predictor in many of the domain results in the primary regression (specifically: Working memory, Visual memory, and Spatial navigation), it is the interaction between a significant group membership factor and sex that is of importance in this study. As discussed, only Inhibition showed a significant group membership effect, but sex of the participants was not a significant predictor in this

domain. This means that on the general test battery there were no significant interactions between group membership and sex of the participant.

This however, is quite different in terms of the analyses done in order to test hypothesis 2. The analyses indicated some important sex differences, each of which is detailed below.

Analysis of the visible trial data showed that there was a significant sex difference on this task (with girls performing significantly more poorly than boys). This is however not a hippocampus-dependent task and therefore the sex differences exhibited are not discussed further.

Analysis of the invisible-target trial data showed that, as with the visible trials, there were significant sex differences (with girls performing far slower than boys). More importantly, these results showed that, although there were no interaction effects between the sex of the participant and group membership, the average performances of each group when broken down by sex, showed that the girls' performance was the reason for the initial significant group effect; the males' performance showed quite a different pattern, with the trauma group performing the worst. Due to the non-significant interaction effects, these results do not statistically confirm the hypotheses relating to sex differences in Spatial navigation, namely that the trauma exposed girls (and particularly the girls in the PTSD group) will perform more poorly than controls, however these results do suggest support for these hypotheses. These results, as well as the pattern of the boys' performance do, however, confirm the hypothesis that the group membership effect is not seen in boys, as suggested by research conducted by Thomas et al. (2010).

The data from Probe trial showed that there was a significant interaction effect between sex of the participant and group membership. Of interest here is that girls in the PTSD group performed more poorly than all of the other participants. Additionally boys in the PTSD group actually performed *better* than all other participants. Although these results only approached statistical significance, they tend to provide support for the a priori

hypotheses regarding sex differences in the effect of group membership on cognitive map-based spatial navigation tasks. Moreover, these results are consistent with the previous results reported by members of our laboratory (e.g., Thomas, 2010), most notably Attwood (2008), who reported that males who were stressed (using the TSST) performed better than all other participants, compared to non-stressed males and both stressed and non-stressed females

The results of the sex differences presented raise an important point of discussion. The research conducted on Spatial navigation, has shown that this ability is significantly affected by stress. This research includes studies done on war-veterans with PTSD, the majority of which are male. However, the research done on the effects of stress using the CG Arena specifically, has shown that stress may not have a detrimental effect on males' Spatial navigation abilities (Thomas et al., 2007) and may even increase performance (Attwood, 2008). The discrepancies within the research could be due to the fact that the one involves long-term traumatic stress, and the other involves current stress (which is obviously not traumatic). On the other hand the discrepancies could be due to measurement issues. Our results however tend to support the CG Arena research on current stress, which lends support to the latter reasoning.

Lastly, the analyses done in order to test hypothesis 3, indicated that there was a significant interaction between group membership and sex of the participant on response bias (β) exhibited in the sample (as suggested by Waters & Valvoi, 2009). Our findings were, however, not consistent with their findings. Specifically, whereas Waters and Valvoi (2009) suggest that females in the clinical group are most affected on tasks of response bias, and males in the clinical group are not affected, the results in the current study showed that the PTSD boys were most biased in responding (compared to all other groups), and statistically significantly different to only the PTSD girls, who were least biased in responding (compared to the other groups).

Possible Reasons for Inconsistent Findings

The possible reasons for a lack of many significant findings in this study is vital in understanding the real relationship between trauma, PTSD and cognitive performance. Based on the results, it could be assumed that trauma exposure and PTSD do not affect many areas of cognition, however, as discussed previously, the study of executive functioning in PTSD and trauma exposure is particularly known for producing elusive results (Danckwerts & Leathem, 2003). These deficits may be too subtle and therefore hard to detect in neuropsychological testing, but as Leskin and White (2007) point out, the effects of possible deficits may still be disruptive to the adolescent victims in their daily lives.

In the test battery, the PTSD participants did not perform the best out of the three groups on *any* of the cognitive abilities, except for Processing speed. However, as discussed, the trauma group performed the best in many of the cognitive abilities (namely, Verbal memory, and Decision-making/impulsivity). This finding is inconsistent with the research done both in adolescent and adult trauma research.

One possible reason for these results is that the trauma group experienced vastly different traumatic events, and although all these events met the criteria for a traumatic event (discussed in the methods section), many of the participants in this group did not feel any lasting effects from the trauma (i.e., they were not traumatized). On the other hand, some of these participants were very close to having a diagnosis of PTSD (however did not meet the exact criteria). Therefore, although many of these participants would have exhibited neuropsychological deficits, due to the trauma they experienced, there were many that would not have been affected in the same way, and thus remain high functioning in terms of the abilities assessed in this study. (This theory is reinforced by the fact that the victims of traumatic events which would seem more severe (such as rape), exhibited more deficits in more domains than other participants).

The possible reasons for the smaller amount of significant results on fewer domains found in the current study compared to Beers and De Bellis (2002) specifically, remain

unclear, however it may be attributed to two possible reasons. These are: firstly, the current study used notably different neuropsychological tests to assess cognitive performance, and secondly, the sample in the current study was from a markedly different population, and therefore culture may be playing an important role in mediating the effects of traumatic stress, trauma in general and PTSD specifically.

With regard to the use of notably different neuropsychological tests, the current study attempted to use tests which were not only of high reliability and validity, but also attempted to use tests which could detect subtle deficits and were well suited for cross-cultural administration (such as those tests in the CANTAB battery; Cambridge Cognition, 2006). Furthermore, as the current study was so strongly based on the theory of the various brain regions affected, it was also important to use tests which had concrete research to elucidate which regions subserved performance on each test, such as the CG Arena (Hartley, Maguire, Spiers, & Burgess, 2003; O'Keefe & Nadel, 1978), and the CANTAB test battery (Cambridge Cognition, 2006). For these reasons it was necessary to use quite different tests to those used in the seminal study.

With regard to the effect culture may have played in the current study, it is vital to highlight that the sample in the current study were all from poor black and coloured communities in the Western Cape, all of a low socio-economic status, and many of whom had received less than optimal education. This is quite different to the American sample assessed in Beers and De Bellis (2002). Indeed research has suggested that culture can play an important role in the experience and appraisal of traumatic events (Aranda & Knight, 1997) and in the functional impairments associated with psychiatric disorders such as PTSD (Canino, Costello, & Angold, 1999). Similarly, researchers have found that culture plays a significant role in manifestation of PTSD symptoms (Norris, Perilla, Ibanez, & Murphy, 2004). Another possible reason for the conflicting results is that Beers and De Bellis (2002) studied maltreatment-related PTSD, whereas the current research studied various types of PTSD. A final possible reason for the conflicting results is seen in the age differences between the two studies.

Age differences in the Research

The average age of the sample in Beers and De Bellis (2002) was PTSD: 11.38 years old and control: 12.17 years old, whereas the average age of the sample in this study was PTSD: 15.79, trauma: 15.31, and control: 15.40. As discussed Beers and De Bellis (2002) only found significant group differences on measures of prefrontal cortex functioning (attention, abstract reasoning and problem solving). They did not find significant group differences on measures of hippocampal functioning. This is consistent with the literature that suggests there is no reduction in the size of the hippocampus in children and adolescents with PTSD (Carrion et al., 2001; De Bellis, 1999, 2002), a reduction which is seen in adult samples with PTSD (Bremner, 2005; Smith, 2005). It therefore follows that the present study, which had a notably older sample, found deficits in hippocampus-dependent tasks whereas Beers and De Bellis did not.

Conversely, the research on executive functioning on adult PTSD samples (such as Beckham, Crawford, & Feldman, 1998; Gilbertsen, Gurvits, Lasko, Orr, & Pitman, 2001) implies that, based on the age of our sample, we should have found significantly more deficits in prefrontal-cortex dependent tasks as well, and this was not the case; neurological research has shown that the prefrontal cortex is not fully developed in adolescence (Lewis, 1997; McGlashan & Hoffman, 2000), therefore it is possible that most adolescents struggle with prefrontal cortex dependent tasks, and therefore only in adult samples are statistically significant differences found.

Important Findings

One of the most important aims of the current study was to explore whether the diagnosis of PTSD allows for unique predictions about certain domains in cognitive functioning, over and above predictions made based on trauma exposure alone. By including three groups in the study, the effects of experiencing trauma alone, separate to having a PTSD diagnosis are thus clearly differentiated.

Both Beers and De Bellis (2002) and Moradi, Neshat-Doost, et al. (1999) only had a PTSD group and a control group and therefore uncertainty over the cause of group

differences surround their research (i.e. were group differences due to experiencing a traumatic event, or was it due to having the disorder of PTSD?) Our study provided some important information in this regard.

In the testing of Hypothesis 1, analyses showed that (specifically, in the domain of Inhibition), it was the trauma participants who were significantly different to the controls (the PTSD group were not significantly different to the controls). This implicates experiencing trauma alone as a factor for neuropsychological deficits.

Furthermore, in the testing of Hypothesis 2, assessing Spatial navigation, it was the PTSD participants who were significantly difference to the controls (the trauma group were not significantly different to the controls). This implicates the diagnosis of PTSD alone as a factor for neuropsychological deficits.

These findings show that, like the research done with adults in this field, both PTSD and the experiencing of trauma are significant factors in adolescents neuropsychological functioning.

Other Interesting Findings

Clinical Characteristics of the Trauma and PTSD Adolescents

Analyses on the clinical characteristics of the sample, as assessed by the MINI KID 5.0, showed that, as expected, the prevalence of having a disorder is contingent on group membership. This effect was specifically seen in Depressive disorders; Anxiety disorders, Obsessive Compulsive Disorder, and Mood disorders. A point of interest here is the association with OCD, as intrusive thoughts are symptoms of both disorders.

Other Characteristics of the Whole Sample

The effects of group membership on other characteristics, namely: general anxiety, perceived everyday stress, approach avoidance/withdrawal tendencies, and resiliency provided some interesting results. The PTSD group scored significantly higher on measures of general anxiety, both in state anxiety and trait anxiety (compared to both

control and trauma participants), and on measures of avoidance/withdrawal tendencies (compared to trauma participants). Lastly, the PTSD group had significantly higher physical and emotional abuse scores (compared to trauma participants). Whether or not the increased general anxiety reported and the increased avoidance/withdrawal tendencies exhibited in the PTSD group is a predicting factor for PTSD or a result of PTSD was not determined in this study.

Limitations and Directions for Future Research

The current study had a number of limitations, all of which should be addressed in future research. Firstly, a potential problem for the interpretation of the current data is that the traumatic events experienced by participants in the sample, although all meeting the criteria for a DSM-IV traumatic event, were of ranging severity. It is possible, therefore, that many of these participants (particularly those in the trauma non-PTSD group) were not significantly affected by the event. Along with using standard definitions of exposure to trauma, one way to address this concern in future research would be to assess the level of severity of the traumatic events experienced and only include participants where the traumatic events experienced are of similar severity.

A second potential limitation of the current study relates to sample size. Although the sample size used here was larger than that in many of the previously published studies in this field (and, significantly, was larger than both Beers and De Bellis (2002), and Moradi, Neshat-Doost, et al. (1999)), many of the trends in the data suggest that with a larger sample many more statistically significant results would have been found. Future studies should attempt to use larger samples so as to clarify, for instance, whether the patterns of test performance seen in the current study were due to chance or to actual between-group differences.

A third potential limitation of the current study is that, due to resource constraints and despite the fact that the study was conducted in a multicultural context, I could not recruit a sample large enough to address questions about cross-cultural differences and SES differences in the neuropsychological sequelae of trauma in adolescents. A large body of

literature has established that neuropsychological test performance is heavily affected by culture and SES (such as in Aranda & Knight, 1997). The question of whether culture and/or SES has an influence on the individual's response to trauma, and whether that in turn leads to differing neuropsychological outcomes, is an interesting question that future research ought to address.

A fourth potential limitation of the current study is that, due to resource constraints, the current study examined a cross-sectional view of individuals with PTSD and trauma exposure, and therefore questions about vulnerabilities to PTSD in adolescents could not be answered. Future studies should attempt to explore longitudinally, possible premorbid factors that are associated with PTSD (such as IQ, anxiety levels, withdrawal tendencies), in order to assess whether or not these factors are a result of a PTSD diagnosis or indicate a predisposition or vulnerability to developing a PTSD diagnosis.

A fifth limitation of the current study is that the current study did not differentiate between a single trauma and recurrent/chronic trauma and thus did not explore the possible differences in effect. Future studies should make this differentiation and include this factor in their analyses in order to fully understand how multiple/chronic trauma impacts on the individual as opposed to a single traumatic experience.

A final potential limitation of the current study is that, due to resource constraints, the current study did not administer functional magnetic resonance imaging (fMRI) as a technique to explore neurological abnormalities associated with trauma and PTSD, but rather relied on previous neurological research for these correlates. Future studies should attempt to include studies using functional magnetic resonance imaging (fMRI) in order to examine the neural activation in the brain regions of adolescent trauma victims and adolescents with PTSD. This would provide a clear indication of how these adolescents differ in neural activation compared to healthy controls that have not experienced trauma.

Conclusion and Implications for Treatment

Overall, the data presented in this thesis suggest that both prefrontal cortex and hippo-

campal functioning are impaired in adolescents with a history of childhood trauma. Moreover, the data suggest that the response to trauma significantly affects later neuropsychological functioning. Taken together, the data therefore imply that although there is a biological basis for the neuropsychological deficits exhibited in victims of trauma and PTSD, the way in which the individual responds to that trauma plays an important role in subsequent cognitive outcome. This suggests that treatment with children and adolescents should focus on helping the individual cope with and manage their responses to traumatic events. In so doing the possible negative effects of trauma are minimized.

University of Cape Town

REFERENCES

- Adamec, R. E., Blundell, J., & Burton, P. (2005). Relationship of the predatory attack experience to neural plasticity, PCREB expression and neuroendocrine response. *Neuroscience & Biobehavioral Reviews*, *30*, 356-375.
- American Psychiatric Association, (1994). Diagnostic and Statistical Manual of Mental Disorders (4th edition) (DSM-IV). Washington, DC: APA.
- American Psychiatric Association. (2000). Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision). Washington, DC: APA.
- Anda, R. F., Felitti, V. J. Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., et al. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry Clinical Neurosciences*, *256*, 174-186.
- Anderson, C. M., Teicher, M. H., Polcari, A., & Renshaw, P. I. (2002). Abnormal T2 relaxation time in the cerebellar vermis of adults sexually abused in childhood: potential role of the vermis in stress-enhanced risk for drug abuse. *Psychoneuroendocrinology*, *27*, 231-244.
- Aranda, M. P. & Knight, B. G. (1997). The influence of ethnicity and culture on the caregiver stress and coping process: A sociocultural review and analysis. *The Gerontologist*, *37*, 342-354.
- Aston-Jones, G., Raikowski, J., Kubiak, P., & Alexinsky, T. (1994). Locus coeruleus neurons in monkeys are selectively activated by attended cues in a vigilance task. *Journal of Neuroscience*, *14*, 4467-4480.

Astur, R. S., St. Germain, S. A., Tolin, D., Ford, J., Russell, D., & Stevens, M. (2006). Hippocampus function predicts severity of post-traumatic stress disorder. *CyberPsychology & Behavior*, *9*, 234-240.

Attwood, C. B. (2008). *The Impact of Acute Psychological Stress on Spatial Cognition* (Unpublished master's thesis). University of Cape Town, South Africa.

Baddeley, A. D., Kopelman, M. D., & Wilson, B. A. (Eds.). (2002). *The handbook of memory disorders*. New York: Wiley.

Baenninger, M. & Newcombe, N. (1995). Environmental input to the development of sex-related differences in spatial and mathematical ability. *Learning and Individual Differences*, *7*, 363-379.

Beckham, J. C., Crawford, A. L., & Feldman, M. E. (1998). Trail Making test performance in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of Traumatic Stress*, *11*, 811-819.

Bedard, A., Martinussen, R., Ickowicz, A., & Tannock, R. (2004). Methylphenidate improves visual-spatial memory in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*, 260-268.

Beers, S. R., & De Bellis, M. D. (2002). Neuropsychological function in children with maltreatment-related post-traumatic stress disorder. *American Journal of Psychiatry*, *159*, 483-489.

Beitchman, J. H., Zucker, K. J., Hood, J. E., daCosta, G. A., Akman, D., & Cassavia, E. (1992). A review of the long-term effects of child sexual abuse. *Child Abuse and Neglect*, *16*, 101-118.

- Bell, B., Hermann, B., Seidenberg, M., Davies, K., Cariski, D., Rosenbek, J., et al. (2002). Ipsilateral reorganization of language in early onset left temporal lobe epilepsy. *Epilepsy & Behaviour, 3*, 158-164.
- Bernstein, D. P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., Sapareto, E., & Ruggiero, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry, 151*, 1132-1136.
- Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., & Ahluvalia, et al., (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse and Neglect, 27*, 169-190.
- Bohbot, V. D., Iaria, G., & Petrides, M. (2004). Hippocampal function and spatial memory: Evidence from functional neuroimaging in healthy participants and performance of patients with medial temporal lobe resections. *Neuropsychology, 18*, 418-425.
- Bowman, R. E. (2005). Stress-induced changes in spatial memory are sexually differentiated and vary across the lifespan. *Journal of Neuroendocrinology, 17*, 526-535.
- Brandes, D., Ben-Schacher, G., Gilboa, A., Bonne, O., Freedman, S., & Shalev, A. Y. (2002). PTSD symptoms and cognitive performance in recent trauma survivors. *Psychiatric Research, 110*, 231-238.
- Braunstein-Bercovitz, H., Dimentman-Ashkenazi, I., & Lubow, R. E. (2001). Stress affects the selection of relevant from irrelevant stimuli. *Emotion, 1*, 182-192.
- Bremner, J. D. (1999) Does stress damage the brain? *Biological Psychiatry, 45*, 707-805.

Bremner, J. D., Narayan, M., Staib, L.H., Southwick, S.M., McGlashan, T., & Charney, D.S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry*, *156*, 1787-1795.

Bremner, J. D., Noriyuki, K., Vaccarino, V., Kutner, M., & Weiss, P. (2005). MRI measurement of hippocampal volume in PTSD: A meta-analysis. *Journal of Affective Disorders*, *88*, 79-86.

Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., et al. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, *152*, 973-81.

Bremner, J. D., Scott, T. M., Delaney, R. C., Southwick, S. M., Mason, J. W., Johnson, D. R. (1993). Deficits in short-term memory in posttraumatic stress disorder. *American Journal of Psychiatry*, *150*, 1015-1019.

Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S.M., McGlashan, T., Nazeer, A., et al. (2003). MRI and PET study of deficits in hippocampal structure and function in women with childhood sexual abuse and posttraumatic stress disorder. *American Journal of Psychiatry*, *160*, 924-932.

Brewin, C. R., Reynolds, M., & Tata, P. (1999). Autobiographical memory processes and the course of depression. *Journal of Abnormal Psychology*, *108*, 511-517.

Brewin, C. R., Watson, M., McCarthy, S., Hyman, P., & Dayson, D. (1998). Intrusive memories and depression in cancer patients. *Behaviour Research and Therapy*, *36*, 1131-1142.

Buckley, T. C., Blanchard, E., B., & Neill, W., T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review*, *28*, 1041-1065.

Burnside, E., Startup, M., Byatt, M., Rollinson, L., & Hill, J. (2004). The role of overgeneral autobiographical memory in the development of adult depression following childhood trauma. *British Journal of Clinical Psychology, 43*, 365–376.

Campbell-Sills, L. & Stein, M. B. (2007). Psychometric analysis and refinement of the Connor-Davidson Resilience Scale (CD-RISC): Validation of a 10-item measure of resilience. *Journal of Traumatic Stress, 20*, 1019-1028.

Canino, G., Costello, E. J., & Angold, A. (1999). Assessing functional impairment and social adaptation for child mental health services research: A review of measures. *Mental Health Services Research, 1*, 93-108.

Carey, P. D., Stein, D. J., Zungu-Dirwayi, N., & Seedat, S. (2003). Trauma and posttraumatic stress disorder in an urban Xhosa primary care population: prevalence, comorbidity, and service use patterns. *Journal of Nervous and Mental Disorders, 191*, 230-236.

Carrion, V.G., Weems, C.F., Eliez, S., Patwardhan, A., Brown, W., Ray, R.D., et al. (2001). Attenuation of frontal asymmetry in pediatric posttraumatic stress disorder. *Biological Psychiatry, 50*, 943-951.

Cambridge Cognition. (2006). The Cambridge Neuropsychological Testing Automated Battery (CANTAB) subject bibliography. Retrieved from <http://www.camcog.com>.

Cohen, M. J. (1997). *Children's Memory Scale*. San Antonio, Texas: The Psychological Corporation.

Connor, K. M., & Davidson, J. R. T. (2003). Development of a new resilience scale: the Connor-Davidson Resilience Scale (CD-RISC). *Depression & Anxiety, 18*, 76-82.

Coull, J. T., Middleton, H. C., Robbins, T. W., & Sahakian, B. J. (2006). Clonidine and diazepam have differential effects on tests of attention and learning.

Psychopharmacology, *120*, 322-332.

Dalgleish, T., Moradi, A., Taghavi, R., Neshat-Doost, H., & Yule, W. (2001). An experimental investigation of hypervigilance for threat in children and adolescents with post-traumatic stress disorder. *Psychological Medicine*, *31*, 541–547.

Dalgleish, T., Taghavi, R., Neshat-Doost, H., Moradi, A., Canterbury, R., & Yule, W. (2003). Patterns of processing bias for emotional information across clinical disorders: a comparison of attention, memory, and prospective cognition in children and adolescents with depression, generalized anxiety, and posttraumatic stress disorder. *Journal of Clinical Child and Adolescent Psychology*, *32*, 10-21.

Danckwerts, A., & Leathem, J. (2003). Questioning the link between PTSD and cognitive dysfunction. *Neuropsychology Review*, *13*, 221-235.

Davidson, J., Baldwin, D., Stein, D. J., Kuper, E., Benattia, I., Ahmed, et al. (2006). Treatment of posttraumatic stress disorder with venlafaxine extended release: a 6-month randomized controlled trial. *Archives of General Psychiatry*, *63*, 1158-1165.

De Bellis, M. D. (2005). The psychobiology of neglect. *Child Maltreatment*, *10*, 150-172.

De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., Jenkins, F.J., Ryan, N.D. (1999). Developmental traumatology. Part I: Biological stress systems. *Biological Psychiatry*, *15*, 1259-1270.

De Bellis, M.D., Keshavan, M.S., Shifflett, H., Iyengar, S., Beers, S.R., Hall, J., Moritz, G. (2002) Brain structures in pediatric maltreatment-related posttraumatic stress disorder: a sociodemographically matched study. *Biological Psychiatry*, *52*, 1066-1078.

De Luca, C. R., Wood, S. J., Anderson, V., Buchanan, J. A., Proffitt, T. M., Mahony, K., et al. (2003). Normative data from the Cantab. I: Development of executive function over the lifespan. *Journal of Clinical & Experimental Neuropsychology*, 25, 242-254.

Edwards, E., Harkins, K., Wright, G., & Menn, F. (1990). Effects of bilateral adrenalectomy on the induction of learned helplessness. *Behavioral Neuropsychopharmacology*, 3, 109-114.

Elliot, R., Rubinsztein, J. S., Sahakian, B. J., & Dolan, R. J. (2000) *Neuroreport*, 11, 1739-1744.

Emdad, R. & Sondergraad, H.P. (2006). Short Communication: Visuoconstructional ability in PTSD patients compared to a control group with the same ethnic background. *Stress and Health*, 22, 35-43.

E-Prime. (2007) Psychology Software Tools. Pittsburgh: Psychology Software Tools, Inc.

Etchamendy, N. and Bohbot, V.D. (2007) Spontaneous navigational strategies and performance in the virtual town. *Hippocampus*, 17, 595-599/

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *The American Journal of Psychiatry*, 164, 1476-1488.

Finchilescu, G. (2002). Measurements. In C. Tredoux & K. Durrheim (Eds.), *Numbers, hypotheses & conclusions: A course in statistics for the social sciences*. (201-229). Cape Town: UCT Press.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structures Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV)*. Washington, D.C.: American Psychiatric Press, Inc.

Foa, E. B. (1995). *Posttraumatic Stress Diagnostic Scale*. USA: NCS Pearson, Inc.

Frakey, L. L., Shrikisoon, A., Thomas, K. G. F., Jacobs, W. J., & Bauer, R. M. (2005). Identifying deficits in spatial abilities following right medial temporal lesions. *Journal of the International Neuropsychological Society*, *11*, 77-78.

Fray, P. J., Robbins, T. W., Sahakian, B. J. (1996). Neuropsychiatric applications of CANTAB. *International journal of geriatric psychiatry*, *11*, 329-336.

Fredrikson, M., & Furmark, T. (2003). Amygdaloid regional cerebral blood flow and subjective fear during symptom provocation in anxiety disorders. *Annals New York Academy of Sciences*, *985*, 341-347.

Gilbertson, M.W., Gurvits, T.V., Lasko, N.B. Orr, S.P., & Pitman, R.K. (2001). Multivariate assessment of explicit memory function in combat veterans with posttraumatic stress disorder. *Traumatic stress*, *14*, 413-420.

Gilbertson, M.W., Shenton, M.E., Ciszewski, A., Kasai, K., Lasko, N.B. Orr, S.P., et al. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Natural Neuroscience*, *5*, 1242-1247.

Goldstein, G. (1990). Neuropsychological heterogeneity in schizophrenia: a consideration of abstraction and problem-solving abilities. *Archives of Clinical Neuropsychology*, *5*, 251-264.

Golier, J., Yehuda, R., Cornblatt, B., Harvey, P., Gerber, D., & Levengood, R. (1997). Sustained attention in combat-related posttraumatic stress disorder. *Integrative Physiological Behavioral Sciences*, 32, 52–61.

Gurvits, T.V., Shenton, M.E, Hokama, H., Ohta, H., Lasko, N.B., Gilbertson, M.W., et al. (1996). Magnetic Resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry*, 40, 1091-1099.

Gurvitz, T. V., Carson, M. A., Metzger, L., Croteau, H. B., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2002). Absence of selected neurological soft signs in Vietnam nurse veterans with posttraumatic stress disorder. *Archives of General Psychiatry*, 110, 81-85.

Gurvitz, T. V., Lasko, N. B., Repak, A. L., Metzger, L., Orr, S. P., & Pitman, R. K. (2002). Performance on visuospatial copying tasks in individuals with chronic posttraumatic stress disorder. *Psychiatry Research*, 112, 263-268.

Hampson, E., Finestone, J. M., & Levy, N. (2005). Menstrual cycle effects on perceptual closure mediate changes in performance on a fragmented objects test of implicit memory. *Brain and Cognition*, 57, 107-110.

Hartley, T., Maguire, E. A., Spiers, H. J., & Burgess, N. (2003). The well-worn route and the path less travelled: distinct neural bases of route following and wayfinding in humans. *Neuron*, 37, 877-888.

Heim-Dreger, U., Kohlmann, C., Eschenbeck, H., & Burkhardt, U. (2006). Attentional biases for threatening faces in children: vigilant and avoidant processes. *Emotion*, 6, 320-325.

Henderson, D., Hargreaves, I., Gregory, S., & Williams, J. M. G. (2002). Autobiographical memory and emotion in a nonclinical sample of women with and

without a reported history of childhood sexual abuse. *British Journal of Clinical Psychology*, 41, 129-141

Hoffman-Plotkin, D., & Twentyman, C. (1984). A multimodal assessment of behavioral and cognitive deficits in abused and neglected preschoolers. *Child Development*, 55, 794-802.

Holt, M. K., Finkelhor, D., & Kantor, G. K. (2006). Multiple victimization experiences of urban elementary school students: Associations with psychosocial functioning and academic performance. *Child Abuse & Neglect*, 31, 503-515.

Howell, D. C. (2004). *Fundamental Statistics for the Behavioural Sciences* (5th edition). USA: Thomson Wadsworth.

Hyde, J. S., Fennema, E., & Lamon, S. J. (1990). Gender differences in mathematics performance: A meta-analysis. *Psychological Bulletin*, 107, 139-155.

Jackson, E. D., Payne, J. D., Nadel, L., & Jacobs, W.J. (2005). Stress differentially modulates fear conditioning in healthy men and women. *Biological Psychiatry*, 59, 516-522.

Jacobs, W. J., Laurance, H. E., & Thomas, K. G. F. (1997). Place learning in virtual space I: Acquisition, overshadowing, and transfer. *Learning and Motivation*, 28, 521-541.

Jacobs, W. J., Thomas, K. G. F., Laurance, H. E., & Nadel, L. (1998). Place learning in virtual space II: Topographical relations as one dimension of stimulus control. *Learning and Motivation*, 29, 288-308.

Jenkins, M., Langlais, P. J., Delis, D., & Cohen, R. A. (2000). Attentional dysfunction associated with posttraumatic stress disorder among rape victims. *The Clinical Neuropsychologist*, 14, 7-12.

Johnson, W., & Bouchard, T. J. (2007). Sex differences in mental abilities: g masks the dimension on which they lie. *Intelligence*, 35, 23-39.

Kaminer, D., Stein, D. J., Mbanga, I., & Zungu-Dirwayi, N. (2001). The Truth and Reconciliation Commission in South Africa: relation to psychiatric status and forgiveness among survivors of human rights abuses. *The British Journal of Psychiatry*, 178, 373-377.

Kanagaratnam, P., & Asbjornsen, A. E. (2007). Executive deficits in chronic PTSD related to political violence. *Journal of Anxiety Disorders*, 21, 510-525.

Kaplan, Z., Weiser, M., Reichenberg, A., Rabinowitz, J., Caspi, A., Bodner, E., et al. (2002). Motivation to serve in the military influences vulnerability to future posttraumatic stress disorder. *Psychiatry Research*, 109, 45-49.

Kimura, D., & Hampson, E. (1994). Cognitive pattern in men and women is influenced by fluctuations in sex hormones. *Current directions in Psychological science*, 3, 57-61.

Kimura, D. (2004). Human sex differences. *Sexuality, Evolution & Gender*, 6, 45-53.

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61, 154-162.

Kirschbaum, C., Wolf, O. T., May, M., Wippich, W., & Hellhammer, D. H. (1996). Stress and treatment-induced elevation of cortisol levels associated with impaired declarative memory in healthy adults. *Life Science*, 58, 1475-1483.

Kirschbaum, C., Pirke, K.M., and Hellhammer, D.H. 1993. The 'Trier Social Stress Test'- A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76–81.

Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., and Caspi, A. (2006). Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychological Medicine*, 37, 181-192.

Korkman, M., Kirk, U., & Kemp, S. (2007) *Nepsy – Second Edition (Nepsy-II)*. USA: Pearson Education Ltd.

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). Differential heart rate reactivity and recovery after psychosocial stress (TSST) in healthy children, younger adults, and elderly adults: The impact of age and gender. *International Journal of Behavioral Medicine*, 2, 116-121.

Kudielka, B. M. & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: a review. *Biological Psychology*, 69, 113-132.

Kuyken, W., & Brewin, C. R. (1995). Autobiographical memory functioning in depression and reports of early abuse. *Journal of Abnormal Psychology*, 104, 585–591.

Kyte, Z. A., Goodyer, I. M., & Sahakian, B. J. (2005). Selected executive skills in adolescents with recent first episode major depression. *Journal of Child Psychology and Psychiatry*, 46, 995-1005.

Ladouceur, C. D., Dahl, R. E., Williamson, D. E., Birmaher, B., Axelson, D. A., Ryan, N. D., et al. (2006). Processing emotional facial expressions influences performance on a Go/No Go task in pediatric anxiety and depression. *Journal of Child Psychology and Psychiatry*, 47, 1107-1115.

Lehto, J. E., Juujarvi, P., & Pulkkinen, L. (2003). Dimensions of executive functioning: Evidence from children. *British Journal of Developmental Psychology, 21*, 59-80.

Leppanen, J. M. (2006). Emotional information processing in mood disorders: a review of behavioral and neuroimaging findings. *Current Opinion in Psychiatry, 19*, 34-39.

Leskin, L. P., & White, P. M. (2007). Attentional networks reveal executive function deficits in posttraumatic stress disorder. *Neuropsychology, 21*, 275-284.

Lewis, D.A. (1997). Development of the prefrontal cortex during adolescence: Insights into vulnerable neural circuits in schizophrenia. *Neuropsychopharmacology, 16*, 385-398.

Lezak, M., Howieson, D., & Loring, D. (2004). *Neuropsychological assessment*. New York: Oxford University Press.

Lindauer, R.J., Vlioger, E.J., Jalink, M., Olf, M., Carlier, I.V., Majoie, C.B., den Heeten, G.J., & Gersons, B.P. (2004). Smaller hippocampal volume in Dutch police officers with posttraumatic stress disorder. *Biological Psychiatry, 56*, 356-363.

Lochner, C., Seedat, S., Hemmings, S. M. J., Kinnear, C. J., Corfield, V.A., Niehaus, D.J.H., et al. (2004). Dissociative experiences in obsessive-compulsive disorder and trichotillomania: Clinical and genetic findings. *Comprehensive Psychiatry, 45*, 384-391.

Luciana, M., & Nelson, C. A. (2002). Assessment of neuropsychological function in children using the Cambridge Neuropsychological Testing Automated Battery (CANTAB): Performance in 4 to 12 year-olds. *Developmental Neuropsychology, 22*, 595-623.

Macklin, M. L., Metzger, L. J., Litz, B. T., McNally, R. J., Lasko, N. B., Orr, S. P., et al. (1998). Lower pre-combat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting Clinical Psychology, 66*, 323-326.

Maguire, E. A., Burgess, N., & O'Keefe, J. (1999). Human spatial navigation: Cognitive maps, sexual dimorphism, and neural substrates. *Current Opinion in Neurobiology*, 9, 171-177.

Masten A. S., Miliotis, D., Graham-Bermann, S. A., Ramirez, M. L., & Neeman, J. (1993). Children in homeless families: Risks to mental health and development. *Journal of Consulting and Clinical Psychology*, 61, 335-343.

Masten, A.S., Neemann, J., and Andenas, S. (1994). Life events and adjustment in adolescents: The significance of event independence, desirability and chronicity. *Journal of Research on Adolescence*, 4, 71-97.

McClure, E. B. (2000). A meta-analytic review of sex differences in facial expression processing and their development in infants, children, and adolescents. *Psychological Bulletin*, 126, 424-453.

McGlashan, T.H., Hoffman, R.E. (2000). Schizophrenia as a disorder of developmentally reduced synaptic connectivity. *Archives of General Psychiatry*, 57, 637-648.

McNally, R. J. (2003). Psychological mechanisms in acute response to trauma. *Society of Biological Psychiatry*, 53, 779-788.

Medina, K.L., Hanson, K.L., Schweinsburg, A.D., Cohen-Zion, M., Nagel, B.J., & Tapert, S.F. (2007). Neuropsychological functioning in adolescent marijuana users: subtle deficits detectable after a month of abstinence. *Journal of the International Neuropsychological Society*. 13, 807-820.

Mills-Schumann, C., Hamstra, J., Goodlin-Jones, B. L., Kwon, H., Reiss, A. L., & Amaral, D. G. (2007). Hippocampal size positively correlates with verbal IQ in male children. *Hippocampus*, 17, 486-493.

- Moore, S. S., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: An evaluative review. *Psychological Bulletin*, *133*, 419-437.
- Moradi, A. R., Neshat-Doost, H. T., Taghavi, M. R., Yule, W., & Dagleiash, T. (1999). Everyday memory deficits in children and adolescents with PTSD: performance on the Rivermead behavioural memory test. *Journal of Child Psychology and Psychiatry*, *40*, 357-361.
- Moradi, A., Taghavi, R., Neshat-Doost, H., Yule, W., & Dagleish, T. (1999). The performance of children and adolescents with PTSD on the Stroop colour naming task. *Psychological Medicine*, *29*, 415-419.
- Morris, R. (1984). Developments of a water-maze procedure for studying spatial learning in the rat. *Journal of Neuroscience Methods*, *11*, 47-60.
- Murphy, F. C., Smith, K. A., Cowen, P. J., Robbins, T. W., & Sahakian, B. J. (2004). The effects of tryptophan depletion on cognition and affective processing in healthy volunteers. *Psychopharmacology*, *163*, 42-53.
- Nelson, E. C., Heath, A. C., Madden, P.A., Cooper, M. L., DinWiddie, S. H., Bucholz, K. K., et al. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: results from a twin study. *Archives of General Psychiatry*, *59*, 139-145.
- Nemeroff, C. B., Bremner, J. D., Foa, E. B., Mayberg, H. S., North, C. S., & Stein, M. B. (2006). Posttraumatic stress disorder: A state-of-the-science review. *Journal of Psychiatric Research*, *40*, 1-21.

Neshat-Doost, H., Moradi, A., Taghavi, R., Yule, W. & Dalgleish, T. (2000). Lack of attentional bias for emotional information in clinically depressed children and adolescents on the dot-probe task. *Journal of Child Psychology and Psychiatry*, 41, 363-368.

Nestler, E. J., Hyman, S. E., & Malenka, R. C. (2001). *Molecular neuropharmacology: a foundation for clinical neuroscience*. New York: McGraw-Hill.

Nilsson-Ihrfelt, E., Fjällskog, M., Liss, A., Jakobsson, O., Blomqvist, C., Andersson, G. (2004). Autobiographical memories in patients treated for breast cancer. *Journal of Psychosomatic Research*, 57, 363-366.

Nolin, P., & Ethier, L. (2007). Using neuropsychological profiles to classify neglected children with and without physical abuse. *Child Abuse & Neglect*, 31, 631-643.

Norris, F. H., Perilla, J. L., Ibanez, G. E., & Murphy, A., D. (2004). Sex differences in symptoms of Posttraumatic stress: Does culture play a role? *Journal of traumatic stress*, 14, 7-28.

O'Keefe J., & Nadel, L. (1978). *The Hippocampus as a Cognitive Map*. Oxford: Clarendon.

Olf, M., Langeland, W., & Gersons, B. P. R. (2005). The psychobiology of PTSD: coping with trauma. *Psychoneuroendocrinology*, 30, 974-982.

Pani, L., Porcella, A., & Gessa, G. L. (2000). The role of stress in the pathophysiology of the dopaminergic system. *Molecular Psychiatry*, 5, 14-21.

Paus, T., Collins, D. L., Evans, A. C., Leonard, G., Pike, B., & Zijdenbos, A. (2001). Maturation of white matter in the human brain: A review of magnetic resonance studies. *Brain Research Bulletin*, 54, 255-266.

Payne, J. D., & Nadel, L. (2004). Sleep, dreams, and memory consolidation: The role of the stress hormone cortisol. *Learning and Memory, 11*, 671-678.

Payne, J. D., Nadel, L., Allen, J. J. B., Thomas, K. G. F., & Jacobs, W. J. (2002). The effects of experimentally-induced stress on false recognition. *Memory, 10*, 1-6.

Peeters, F., Wessel, I., Merckelbach, H., & Boon-Vermeeren, M. (2002). Autobiographical memory specificity and the course of major depressive disorder. *Comprehensive Psychiatry, 43*, 344-350.

Pine, D. S. (2003). Developmental psychobiology and response to threats: relevance to trauma in children and adolescents. *Society of Biological Psychiatry, 53*, 796-808.

Pissiota, A., Frans, O., Fernandez, M., von Knorring, L., Fischer, H., & Fredrikson, H. (2002). Neurofunctional correlates of posttraumatic stress disorder: a PET symptom provocation study. *European Archives of Psychiatry and Clinical Neuroscience, 252*, 68-75.

Protopopescu, X. H., Pan, O., Tuescher, M., Cloitre, M., Goldstein, W., Engelien, J., et al. (2005). Differential time courses and specificity of amygdala activity in posttraumatic stress disorder subjects and normal control subjects. *Biological Psychiatry, 57*, 464-473.

Raes, F., Hermans, D., Williams, J. M. G., & Eelen, P. (2005). Autobiographical memory specificity and emotional abuse. *British Journal of Clinical Psychology, 44*, 133-138.

Stokes, D. J., Dritschel, B. H., & Bekerian, D. A. (2004). The effect of burn injury on adolescents' autobiographical memory. *Behaviour Research and Therapy, 42*, 1357-1365.

Randall, D. C., Fleck, N. L., Shneerson, J. M., & File, S. E. (2004). The cognitive-enhancing properties of modafinil are limited in non-sleep-deprived middle-aged volunteers. *Pharmacology Biochemistry and Behaviour, 77*, 547-555.

- Richert, K. A., Carrion, V. G., Karchemskiy, A., & Reiss, A. L. (2005). Regional differences of the prefrontal cortex in pediatric PTSD: an MRI study. *Depression and Anxiety, 23*, 17-25.
- Roche, R. A. P., Mangaoang, M. A., Commins, S., & O'Mara, S. M (2005). Hippocampal contributions to neurocognitive mapping in humans: A new model. *Hippocampus, 15*, 622-641.
- Rosenbaum, R. S., Ziegler, M., Winocur, G., Grady, C. L., & Moscovitch, M. (2004), "I have often walked down this street before": fmri studies on the hippocampus and other structures during mental navigation of an old environment. *Hippocampus, 14*, 826-835.
- Roosendaal, B., Jayme, R., & McGaugh, J. L. (2004). The Basolateral Amygdala Interacts with the Medial Prefrontal Cortex in Regulating Glucocorticoid Effects on Working Memory Impairment. *Journal of Neuroscience, 24*, 1385-1392.
- Ruel, J. M., & de Kloet, E. R. (1995). Two receptors systems for corticosterone in rat brain: microdistribution and differential occupation. *Endocrinology, 117*, 2505-2511.
- Saigh, P. A., Yasik, A. E., Oberfield, R. A., Halamandaris, P. V., Bremner, J. D. (2006). The intellectual performance of traumatized children and adolescents with or without posttraumatic stress disorder. *Journal of Abnormal Psychology, 115*, 332-340.
- Saltzman, K. M., Weems, C. F., & Carrion, V., G. (2006). IQ and posttraumatic stress symptoms in children exposed to interpersonal violence. *Child Psychiatry and Human Development, 36*, 261-272.
- Samuelson, K. W., Neylan, T. C., Merzler, T. J., Lenoci, M., Rothlind, J., Henn-Haase, C., et al. (2006). Neuropsychological functioning in posttraumatic stress disorder and alcohol abuse. *Neuropsychology, 20*, 716-726.

Sapolsky, R. M. (2000). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry*, *57*, 925-935.

Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *Journal of Neuroscience*, *10*, 2897-2902.

Schrieff, L., & Thomas, K. G. F. (2008, January). Establishing a neuropsychological rehabilitation service for children with traumatic brain injury. Paper presented at the annual convention of the South African Clinical Neuropsychological Association, Durban.

Schultz, K. P., Fan, J., Magidina, O., Marks, D. J., Hahn, B., & Halperin, J. M., (2007). Does the emotional go/no-go task really measure behavioral inhibition?: Convergence with measures on a non-emotional analog. *Archives of Clinical Neuropsychology*, *22*, 151-160.

Schwabe, L., Oitzl, M. S., Philippson, C., Richter, S., Bohringer, A., Wippich, W., et al. (2007). Stress modulates the use of spatial versus stimulus-response learning strategies in humans. *Learning & Memory*, *14*, 109-116.

Seedat, S., Nyamai, C., Njenga, B., Vythilingum, B., & Stein, D. J. (2004). Trauma exposure and post-traumatic stress symptoms in urban African schools: Survey in CapeTown and Nairobi. *British Journal of Psychiatry*, *184*, 169-175.

Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London: Biological Sciences*, *298*, 199-209.

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., et al. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development

and validation of a structured psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59, 22-33.

Sheehan, D. V., Sheehan, K. H., Shytle, R. D., Janavs, J., Bannon, Y., Rogers, J. E., Milo, K. M., Stock, S. L., Wilkinson, B. (2010). Reliability and validity of the Mini International Neuropsychiatric Interview for Children and Adolescents (MINI-KID). *Journal of Clinical Psychiatry*, 71, 313-26.

Shin, L.H., McNally, R.J., Kosslyn, S.M., Thompson, W.L., Rauch, S.L., Alpert, N.M., Metzger, L.J., Lasko, N.B., Orr, S.P., & Pitman, R.K. (1999). Regional cerebral blood flow during script-driven Imagery in childhood sexual abuse-related PTSD: a PET investigation. *American Journal of Psychiatry*, 156, 575-584.

Shin, L. M., Orr, S. P., Carson, M. A., Rauch, S. L., Macklin, M. L., Lasko, N. B., et al. (2004). Regional cerebral blood flow in amygdala and medial prefrontal cortex during traumatic imagery in male and female Vietnam veterans with PTSD. *Archives of General Psychiatry*, 61, 168-176

Shin, L. M., Rauch, S. L., & Pitman, R., K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals New York Academy of Sciences*, 1071, 67-79.

Shonk, S., & Cicchetti, D. (2001). Maltreatment, competency deficits, and risk for academic and behavioral maladjustment. *Developmental Psychology*, 37, 3-17.

Silva, R. R., Alpert, M., Munoz, D. M., Singh, M. D., Matzner, F., & Dummit, S. (2000). Stress and vulnerability to posttraumatic stress disorder in children and adolescents. *American Journal of Psychiatry*, 157, 1229-1235.

Simantov, R., Blinder, E., Ratovitski, T., Tauber, M., Gabbay, M., & Porat, S. (1996). Dopamine induced apoptosis in human neuronal cells: Inhibition by nucleic acids antisense to the dopamine transporter. *Neuroscience*, 74, 39-50.

Smith, M.E. (2005): Bilateral hippocampal volume reduction in adults with PTSD: A meta-analysis of structural MRI studies. *Hippocampus*, 15, 798-807.

Smythies, J. R. (1997). Oxidative reactions and schizophrenia: A review discussion. *Schizophrenia Research*, 24, 357-364.

Sowell, E. R., Trauner, D. A., Gamst, A., & Jernigan, T. L. (2002). Development of cortical and subcortical brain structures in childhood and adolescence: A structural MRI study. *Developmental Medicine & Child Neurology*, 44, 4-16.

Spielberger, C. D., Gorsuch, R. L., Lushene, R. D., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the state-trait anxiety inventory*. Palo Alto: Consulting Psychologists Press.

Spielberger C. D., & Vagg P. R. (1984). Psychometric properties of the STAI: a reply to Ramanaiah, Franzen, and Schill. *Journal of Personality Assessment*, 48, 95–97.

Spreen, O., & Strauss, E. (1998). *A Compendium of Neuropsychological Tests*. (2nd ed). New York: Oxford University Press.

Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and human. *Psychological Review*, 99, 195-231.

StatSoft, Inc. (2008). STATISTICA (data analysis software system), version 8.0. www.statsoft.com.

Stein, M. B., Hanna, C., Vaerum, V., & Koverola, C. (1999). Memory function in adult women traumatized by childhood sexual abuse. *Journal of Traumatic Stress*, 12, 527-534.

Stein, M. B., Kennedy, C. M. & Twamley, E. W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry*, 52, 1079-1088.

Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, 27, 951-9.

Stokes, D. J., Dritschel, B. H., Bekerian, D. A. (2004). The effect of burn injury on adolescents' autobiographical memory. *Behaviour Research and Therapy*, 42, 1357-1365.

Stroud, L. R., Salovey, P., Epel, E. S. (2002). Sex differences in stress responses: social rejection versus achievement stress. *Biological Psychiatry*, 52, 318-327.

Strümpfer, D. J. W., Viviers, M. R., Gouws, J. F. (1998). Item-phrasing in Antonovsky's sense of coherence scale related to negative and positive affectivity. *Personality and Individual Differences*, 24, 669-675.

Sullivan, K., Kregel, M., Proctos, S. P., Devine, S., Heeren, T., & White, R. F. (2003). Cognitive functioning in treatment-seeking Gulf war veterans: Pyridostigmine bromide use and PTSD. *Journal of Psychopathology and Behavioural Assessment*, 25, 95-103.

Sutker, P. B., Vasterling, J. J., Brailey, K., & Allain, A. N. (1995). Memory, attention, and executive deficits in POW survivors: contributing biological and psychological factors. *Neuropsychology*, 9, 118-25.

Sweeney, J. A., Kmiec, J. A., & Kupfer, D. J. (2000). Neuropsychologic impairments in bipolar and unipolar mood disorders on the CANTAB neurocognitive battery. *Biological Psychiatry*, 48, 674-684.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C., Navalta, C. P., & Kim, D. M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neuroscience and Biobehavioral Reviews*, 27, 33-44.

Teicher, M. H., Ito, Y., Glod, C. A., Anderson, S.L., Dumont, N., & Ackerman, E. (1997). Preliminary evidence for abnormal cortical development in physically and sexually abused children using EEG coherence and MRI. *Annals New York Academy of Sciences*, 821, 160-175.

Teicher, M. H., Ito, Y., Glod, C. A., Schiffer, F., and Gelbard, H. A. (1996). Neurophysiological mechanisms of stress response in children. In Pfeffer, C. (ed.). (1996). *Severe Stress and Mental Disturbance in Children*. Washington, DC: American Psychiatric Press.

The Psychological Corporation. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, Texas: Harcourt Brace & Company.

Thomas, K. G. F., Hsu, M., Laurance, H. E., Nadel, L., & Jacobs, W. J. (2001). Place learning in virtual space III: Investigation of spatial navigation training procedures and their application to fMRI and clinical neuropsychology. *Behavior Research Methods, Instruments, & Computers*, 33, 21-37.

Thomas, K. G. F., Laurance, H. E., Nadel, L., & Jacobs, W. J. (2010). Stress-induced impairment of spatial navigation in females. *South African Journal of Psychology*, 40, 32-43.

Thorndyke, P. W., & Hayes-Roth, B. (1982). Differences in spatial knowledge acquired from maps and navigation. *Cognitive Psychology*, 14, 560-589.

Twamley, E. W., Allard, C. B., Thorp, S. R., Norman, S. B., Cissell, S. H., Berardi, K. H., et al. (2009). Cognitive impairment and functioning in ptsd. *Journal of the International Neuropsychological Society*, 15, 879-887.

Twamley, E.W., Hami, S., & Stein, M.B. (2004). Neuropsychological function in college students with and without posttraumatic stress disorder. *Psychiatry Research*, 126, 265-274.

Van der Ryst, E., Strydom, W., Scott, C., Boshoff, W., Joubert, G., & Els, C. (2002). A high rate of psychiatric comorbidity in South African HIV/AIDS patients. *International Conference on AIDS*, 12, 571-572.

Vasterling, J. J., Brailey, K., Constans, J. I., Borges, A., & Sutker, P. B. (1997). Assessment of intellectual resources in Gulf War veterans: Relationship to PTSD. *Assessment*, 4, 51-59.

Vasterling, J. J., & Brewin, C. R. (2005). *Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives*. New York: Guilford.

Vasterling, J. J., Duke, L. M., Brailey, K., Constans, J. I., Allain, A. N., Jr., & Sutker, P. B. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology*, 16, 5-14.

Veltman, M., & Browne, K. (2001). Three decades of child maltreatment research. *Trauma, Violence and Abuse*, 2, 215-239.

Voogt, E., Van Der Heide, A., Van Leeuwen, A. F., Visser, A. P., Cleiren, M. P., Passchier, J. Et al. (2004). Positive and negative affect after diagnosis of advanced cancer. *Psychooncology*, 14, 262-273.

- Waters, A. M., Mogg, K., Bradley, B. P., & Pine, D. S. (2008). Attentional bias for emotional faces in children with generalized anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 435-442.
- Waters, A. M. & Valvoi, J, S. (2009). Attentional bias for emotional faces in paediatric anxiety disorders: An investigation using the emotional go/no go task. *Journal of Behavior Therapy and Experimental Psychiatry, 40*, 306-316.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.
- Watts-English, T., Fortson, B. L., Gibler, N., Hooper, S. R., & De Bellis, M. D. (2006). The psychobiology of maltreatment in Childhood. *Journal of Social Issues, 62*, 717-736.
- Weber, D. A, & Reynolds, C. R. (2004). Clinical perspectives on neurobiological effects of psychological trauma. *Neuropsychology Review, 12*, 115-129.
- Wessel, I., Meeren, M., Peeters, F., Arntz, A., & Merckelbach, H. (2001). Correlates of autobiographical memory specificity: The role of depression, anxiety, and childhood trauma. *Behaviour Research and Therapy, 39*, 409-421.
- Whitlow, C. T., Liguori, A., Livengood, B., Hart, S. L., Mussat-Whitlow, B. J., Lamborn, C. M., et al. (2004). Long-term heavy marijuana users make costly decisions on a gambling task. *Drug and Alcohol Dependence, 76*, 107-111.
- Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (2nd edition). Chichester, England: Wiley.
- Wilson, B. A., Cockburn, J., & Baddeley, A. D. (1985). *The Rivermead Behavioural Memory Test*. Bury St Edmunds: Thames Valley Test Company.

Wodarski, J. S., Kurtz, P. D., Gaudin, J. M., & Howing, P. T. (1990). Maltreatment and the school-age child: Major academic, socioemotional, and adaptive outcomes. *Social Work, 35*, 506-513.

Wolf, O. T., Schommer, N. C., Hellhammer, D. H., McEwen, B. S., & Kirschbaum, C. (2001). The relationship between stress induced cortisol levels and memory differs between men and women. *Psychoneuroendocrinology, 26*, 711-720.

Woodward, S. H., Kaloupek, D. G., Streeter, C. C., Martinez, C., Schaer, M., & Eliez, S. (2006). Decreased anterior cingulate volume in combatrelated PTSD. *Biological Psychiatry, 59*, 582-587.

Wright, R. L., Lightner, E. N., Harman, J. S., Meijer, O. C., & Conrad, C. D. (2006). Attenuating corticosterone levels on the day of memory assessment prevents chronic stress-induced impairments in spatial memory. *European Journal of Neuroscience, 24*, 595-605.

Yehuda, R., Keefe, R. S., Harvey, P. D., Levengood, R. A., Gerber, D. K., Geni, J., et al. (1995). Learning and memory in combat veterans with posttraumatic stress disorder. *American Journal of Psychiatry, 152*, 137-139.

Yehuda, R., Schmeidler, J., Wainberg, M., Binder-Brynes, K., Duvdevani, B. A. (1998). Vulnerability to Posttraumatic Stress Disorder in Adult Offspring of Holocaust Survivors. *American Journal of Psychiatry, 155*, 1163-1171.

Zigmond, M. J., Finlay, J. M., & Sved, A. F. (1995). Neurochemical studies of central noradrenergic responses to acute and chronic stress. In Vasterling, J. J., & Brewin, C.

Zolotor, A., Kotch, J., Dufort, V., Winsor, J., Catellier, D., & Bou-Saada, I. (1999). School performance in a longitudinal cohort of children at risk for maltreatment. *Maternal and Child Health, 3*, 19-7.

APPENDIX A

Demographic Questionnaire

DEMOGRAPHIC QUESTIONNAIRE

GENERAL INFORMATION

Full name:	
Telephone:	Home: () Cell:
How would you describe your ethnicity / race?	1. Black 2. Coloured 3. White 4. Asian 5. Other(specify):
Home Language:	
Full name (Child):	
Gender:	M F
Date of Birth:	
Grade:	
Address: area of residence:	

HOUSEHOLD INCOME: (Please circle appropriate number)

Household income per year:	1. R0 2. R1 – R5 000 3. R5001 – R25 000 4. R25 001 – R100 000 5. R100 001+
----------------------------	--

PARENTAL EDUCATION: (Please circle appropriate number)

	Biological mother	Biological father	Guardian
Highest level of education reached? Mark one response for each person as follows:			
1. No formal education (never went to school)	1.	1.	1.
2. Grades 1-6 / Sub A-Std 4 - didn't complete primary school	2.	2.	2.
3. Grade 7 / Std 5 - completed primary school	3.	3.	3.
4. Grades 8-11 / Stds 6-9 -didn't complete high school	4.	4.	4.
5. Grade 12 / Std 10 - completed senior school	5.	5.	5.
6. 13+ years - completed university / technikon / college	6.	6.	6.
7. Don't know	7.	7.	7.

MATERIAL AND FINANCIAL RESOURCES (ASSET INDEX): (Please circle appropriate number)

Which of the following items, in working order, does your household have?

Items	Yes	No
1. A refrigerator or freezer	1.	1.
2. A vacuum cleaner or polisher	2.	2.
3. A television	3.	3.
4. A hi-fi or music center (radio excluded)	4.	4.
5. A microwave oven	5.	5.
6. A washing machine	6.	6.
7. A video cassette recorder or dvd player	7.	7.

Which of the following do you have in your home?

Items	Yes	No
1. Running water	1.	1.
2. A domestic servant	2.	2.
3. At least one car	3.	3.
4. A flush toilet	4.	4.
5. A built-in kitchen sink	5.	5.
6. An electric stove or hotplate	6.	6.
7. A working telephone	7.	7.

	Yes	No
1. Do you have a computer at home	1.	1.
2. Do you have a computer at school	2.	2.

APPENDIX B

Screening Questionnaire

research questionnaire



university of cape town

We're talking with teenagers from the Western Cape to gather information about their daily lives, how they have been feeling recently. We also want to see how those teenagers perform on certain tests of memory and thinking. The information gathered will be used to create a thesis to be given to the University of Cape Town as part of a Masters degree.

Some people will be asked to continue with this study by participating in 2 more sessions. However, filling out this questionnaire does not mean you have to continue.

In this questionnaire, you will be asked personal questions. I would really appreciate it if you would answer the questions honestly and openly. Your answers are very important to us.

All the answers you give in this questionnaire will be kept **confidential**. That means they will be private between you and me. No one else will see your name or your answers to this questionnaire.

Thank you for your time and honesty with this questionnaire.

Please indicate that you understand and would like to participate in this research by signing in the space provided.

Date: _____

Name/Agreement: _____

SECTION 1

GENERAL INFORMATION

Full name:	
How would you describe your ethnicity/ race?	1. Black 2. Coloured 3. White 4. Other(specify):
Home Language:	
Gender:	Male Female
Date of Birth:	
Grade:	
What area do you live in:	

SECTION 2.

During the past month, how often did you drink alcohol?

Not once
 Every day
 Several times per week
 Once a week
 Once a month

Have you been drunk in the past month?

Yes
 No

On a day or evening when you drink, how many do you usually have?

Have you ever used any drug to make yourself feel high?
 Tick any you have:

..... not once dagga mandrax
 glue heroin cocaine
 crack petrol ecstasy
 acid tik other

In the past month, how many times did you take these drugs?



ape Town

SECTION 3.

3.1. Has anything really awful happened to you? Like being in a really bad accident, a fire, or a natural disaster? Like seeing someone get killed or hurt really bad? Like being attacked by someone? Or has something else really awful happened to you? NO YES

**IF YOU CIRCLED YES TO THIS QUESTION CONTINUE WITH THIS SECTION
IF YOU CIRCLED NO TO THIS QUESTION YOU CAN SKIP TO THE END.**

3.2. Did you respond with intense fear, feel helpless or horrified? NO YES

3.3 **In the past month**, has this awful thing come back to you in some way? Like dreaming about it or having a strong memory of it or feeling it in your body? NO YES

3.4. **In the past month:**

a Have you tried not to think about this awful thing? NO YES

b Have you tried to stay away from things that might remind you of it? NO YES

c Have you had trouble remembering some important part of what happened? NO YES

d Have you been much less interested in your hobbies or your friends? NO YES

e Have you felt cut off from other people? NO YES

f Have you noticed that you don't have strong feelings about things? NO YES

g Have you felt that your life will be shortened or that you will die sooner than other people? NO YES

3.5. **In the past month:**

a Have you had trouble sleeping? NO YES

b Have you been moody or angry for no reason? NO YES

c Have you had trouble paying attention? NO YES

d Were you nervous or "jumpy"? NO YES

e Would you jump when you heard noises? NO YES

f. Would you jump when you saw something out of the corner of your eye? NO YES

3.6. **In the past month:**

a. Have these problems upset you a lot?

b. Have these problems caused you to have problems at school, at home, or with your friends?

IF YES TO ANY, CIRCLE YES NO YES

Thank you again for completing this questionnaire.
Your time and honesty is really appreciated.

APPENDIX C

Screening Questionnaire: Rationale and Results

Further Rationale

One very important reason for wanting to recruit a select number of participants for each group from the start stemmed from the legal obligations set in place to report first time confessions of suspected abuse (now further enforced by the Sexual Offences Act 2008). This legal obligation was spelt out in the assent forms (APPENDIX #). The screening questionnaire was thus initially implemented so there would not be too many people in the trauma and PTSD groups, thereby controlling for a possible situation where there were unmanageable numbers of legal issues. However, while conducting the interviews, it came to light that there were no traumatic events that went unreported, as all of the traumatic events that took place, and were discussed in the interviews, had been reported to the police where necessary.

Findings

Interestingly, the screening process itself provided some important results. Out of the 126 adolescents who completed the screening questionnaires, only 57 self-reported having not experienced a traumatic event (i.e., 69 self-report having experienced a traumatic event). Of these 69 adolescents, 39 met the criteria for PTSD (as assessed by the MINIKid 5.0). This is a staggering 31% of *all* the adolescents assessed (39 out of 126 adolescents).

APPENDIX D

Life Events Questionnaire

INITIALS _____

STUDY # _____

DATE _____

Have any of the following life events or problems happened to you during the last 6 months? How about more than 6 months ago? If so, please also rate the impact on you.

	Did this occur in past 6 months? (circle correct answer)	Impact (circle correct answer)	Did this occur more than 6 months ago? (circle correct answer)	Impact (circle correct answer)
You broke off a steady relationship.	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You had a serious problem with a close friend, neighbor or relative.	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You were fired from your job.	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You moved to a new school.	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
Your began to see your mother less	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
Your began to see your father less	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
There has been a change (up or down) in the family's financial status	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You have a new brother or sister in the family	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
Another adult has come to live with the family	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi

You began a relationship	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
One of your close friends moved away	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A sibling of yours moved away	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A pet of yours died	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
Your parents began to argue or fight more	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
Your parents separated or divorced	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You began to argue more with your parents	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
One of your parents has lost their job	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A parent had problems with the police and received a jail sentence	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
The family was forced to move from where you were staying	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A sibling has become involved in drugs	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
One of your parents has been seriously ill and had to be Hospitalized	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A brother or sister has been ill and had to be hospitalized	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
You got a new stepparent	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi
A sibling has become involved in drugs	Yes / No	None/Some/Significant	Yes / No	None/Some/Signi

APPENDIX E

Parent/Guardian's Informed Consent Document (for Participants from Treatment Centers and the Boys School)

UNIVERSITY OF CAPE TOWN DEPARTMENT OF PSYCHOLOGY

Informed Consent to Allow Participation in Research and Permission for Collection, Use, and Disclosure of Cognitive Performance and Other Personal Data

You are being asked to allow your child to take part in a research study. This form provides you with information about the study and seeks your permission for the collection, use and disclosure of your child's test performance data, as well as other information necessary for the study. The Principal Investigator (the person in charge of this research) will also describe this study to your child and answer all of their questions. Your child's participation is entirely voluntary. Before you decide whether or not they can take part, read the information below and ask questions about anything you do not understand. By allowing participation in this study you and your child will not be penalized or lose any benefits to which you would otherwise be entitled.

1. Name of Participant ("Study Subject")

2. Title of Research Study

Neuropsychological Profiles of Adolescents with a History of Childhood Trauma

3. Principal Investigator and Telephone Number(s)

Kevin G. F. Thomas, Ph.D.
Senior Lecturer
Department of Psychology
University of Cape Town
021-650-4608

Michaela Ashley-Cooper
Masters student
Department of Psychology
University of Cape Town
0832473954

4. What is the purpose of this research study?

The main purpose of this research is to describe how adolescents, from a South African population, with a history of childhood trauma, perform on the particular battery of neuropsychological tests. Specifically, we plan to compare the performance of adolescents with a history of trauma, with the performance of adolescents with no such history. Furthermore we plan to assess the effect of a posttraumatic stress diagnosis has on performance in these tests.

5. What will be done if your child takes part in this research study?

In this study, a series of questionnaires and cognitive tests will be administered. The questionnaires assess your child's current psychological functioning, and ask about the trauma they have experienced. These questions are not detailed questions, your child simply answers yes or no to the questions; they do not need to give any details. The cognitive tests measure certain aspects of your child's memory and thinking skills.

6. If you choose to allow your child to participate in this study, how long will they be expected to participate in the research?

The experiment consists of two sessions. The first should not last longer than 120 minutes. The second session will also take approximately 120 minutes. If at any time during the sessions your child finds any of the procedures uncomfortable, they will be free to stop participating without penalty.

7. How many people are expected to participate in the research?

100

8. What are the possible discomforts and risks?

There are no known risks associated with participation in this study. A possible discomfort your child may experience is slight fatigue. If they become tired during any of the tests, or questionnaires, they can take a break. They will be allowed to take breaks whenever they want to. During the questionnaire session, sensitive questions may be asked; however, your child will be assured that he/she only needs to answer what they feel comfortable with. Furthermore, all participants will be informed that a registered clinical psychologist will be available to them if they are in any way distressed by the study procedures. At the conclusion of the study procedures, all participants will be fully debriefed and provided with a list of trauma counselling centres and trauma counsellors.

If you wish to discuss the information above or any discomforts you may experience, you may ask questions now or call the Principal Investigator listed on the front page of this form.

9. What are the possible benefits to your child? Will you or your child receive compensation for taking part in this research study?

Your child will receive monetary compensation for their involvement in this study. This will include R50 for the first session, and R50 for the second session.

10. What are the possible benefits to others?

Information from this study will improve our understanding of how trauma affects functioning in later life. The research may help to identify people who have experienced various forms of trauma.

11. If you choose to allow your child to take part in this research study, will it cost you

anything?

Allowing your child to participate in this study will not cost you anything. The research will be conducted at the centre your child is currently attending, and all travelling costs will be reimbursed.

12. Can your child withdraw from this research study?

You are free to withdraw your consent and to stop your child participating in this research study at any time. If you do withdraw your consent, there will be no penalty.

If you have any questions about your child's rights or welfare as a research participant, you may contact Professor Marc Blockman on 021 4066496.

13. If your child withdraws, can information about your child still be used and/or collected?

Information already collected may be used.

14. Once personal and performance information is collected, how will it be kept secret (confidential) in order to protect you and your child's privacy?

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people have the right to review these research records. These people include the researchers for this study and certain University of Cape Town officials. Your child's research records will not be released without your permission unless required by law or a court order.

15. What information about your child may be collected, used and shared with others?

This information gathered will be records of your child's performance on cognitive tests, as well as information on their history and current psychological functioning. However their name does not appear on any of the data.

16. How will the researcher(s) benefit from your child being in the study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigator and others attached to this research project may benefit if the results of this study are presented at scientific meetings or in scientific journals.

17. Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; and how the participant's performance and other data will be collected, used, and shared with others:

Signature of Person Obtaining Consent and Permission

Date

You have been informed about this study's purpose, procedures, possible benefits, and risks; and how your performance and other data will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You agree to allow participation in this study. You hereby give permission for the collection, use and sharing of your child's performance and other data. By signing this form, you are not waiving any of your legal rights.

Signature of Person Consenting and Giving Permission

Date

Name of Child

Age

Please indicate below if you would like to be notified of future research projects conducted by our research group:

_____ (initial) Yes, I would like to be added to your research participation pool and be notified of research projects in which I might participate in the future.

Method of contact:

Phone number: _____

E-mail address: _____

Mailing address: _____



APPENDIX F

Assent Form for Participants at the Treatment Centers

Assent Form for Participation in Study

Hello! My name is Michaela Ashley-Cooper. I'm here for a study on behalf of the University of Cape Town.

We're talking with adolescents from the Western Cape to gather information about their daily lives, how they have been feeling recently, and performance on certain tests. The information gathered will be used to create a thesis to be given to the University of Cape Town, as part of a Masters degree.

Firstly, we would like to ask you some questions in an interview that will take a little less than 2 hours. This interview will discuss experiences you had in your childhood and will look at how you are feeling currently. You will then do 1 more session, also lasting less than 2 hours, involving tests that assess certain things. Most of the tests will be like computer games. The interview and tests will be done just with me. I would really appreciate it if you would answer the questions honestly and openly, so that we can find out how you really feel, and what you really think. Your answers are very important to us. You will be given R50 for the first session, and R50 for the last session. Whoever brings you to the centre will be reimbursed for travel costs.

Some of these questions may talk about things that people can find quite personal, or may be difficult to answer. If any of the questions make you feel uncomfortable or you don't want to answer them, you do not have to. If any of the questions upset you, or if you would like to talk to someone about the feelings you experienced during the interview, please let me know and I, or another responsible adult, will be happy to take that time with you.

If you decide to participate in this study, you will have the chance to provide important information to this research. Even though this isn't a quick process, your thoughts and opinions are very valuable.

Remember, you do not have to talk about anything you don't want to.

If you agree to take part in this study, the things you tell me will be confidential. That means they will be private between you and me. I want to let you know, though, that it is my responsibility to make sure that you are safe. That means if you tell me you are being hurt by another person, you are hurting yourself, or you are planning to hurt another person, I will have to let another responsible adult

know so that, depending on your situation, the right actions can be taken to make sure that you are safe.

If you have any questions about what you have read or if you think of any questions in the future, you can reach me, Michaela, at 083 247 3954.

Would you like to participate in the research? Please indicate here with your name or a mark if you would like to participate.

Date: _____

Child's Name/Agreement: _____

Researcher's Signature: _____

University of Cape Town

APPENDIX G

Parent/Guardian's Informed Consent Document (for Participants from the Co-ed School)

UNIVERSITY OF CAPE TOWN DEPARTMENT OF PSYCHOLOGY

You are being asked to allow your child to take part in a research study. This form provides you with information about the study and seeks your permission for the collection, use and disclosure of your child's test performance data, as well as other information necessary for the study. The Principal Investigator (the person in charge of this research) will also describe this study to your child and answer all of their questions. Your child's participation is entirely voluntary. Before you decide whether or not they can take part, read the information below and ask questions about anything you do not understand. By allowing participation in this study you and your child will not be penalized or lose any benefits to which you would otherwise be entitled.

18. Name of Child

19. Title of Research Study

Neuropsychological Profiles of Adolescents with a History of Childhood Trauma

20. Principal Investigator and Telephone Number(s)

Kevin G. F. Thomas, Ph.D.
Senior Lecturer
Department of Psychology
University of Cape Town
021-650-4608

Michaela Ashley-Cooper
Masters student
Department of Psychology
University of Cape Town
0832473954

21. What is the purpose of this research study?

The main purpose of this research is to describe how South African adolescents with a history of childhood trauma perform on a set of tests of learning, memory, attention, and problem-solving. Specifically, we plan to compare the performance of adolescents with a history of trauma, with the performance of adolescents with no such history.

22. What will be done if your child takes part in this research study?

Firstly, your child will fill out some questionnaires. After this, your child will take some cognitive tests will be administered. The questionnaires assess your child's current psychological functioning, and ask about the trauma they have experienced. The tests measure certain aspects of your child's memory and thinking skills.

23. If you choose to allow your child to participate in this study, how long will they be expected to participate in the research?

The experiment consists of two sessions. Both sessions should not last longer than 90 minutes. If at any time during the sessions your child finds any of the procedures uncomfortable, he or she will be free to stop participating without penalty. Both of these sessions will be done during school time.

24. What are the possible discomforts and risks?

There are no known risks associated with participation in this study. A possible discomfort your child may experience is slight fatigue. If your child becomes tired at any point during the study, he or she will be allowed to take a break. Your child will be allowed to take as many breaks as he/she needs. During the questionnaire session, sensitive questions may be asked; however, your child will be assured that he/she only needs to answer questions he/she feels comfortable with. Furthermore, a registered clinical psychologist will be available to your child if he/she is in any way distressed by the study procedures. At the conclusion of the study procedures, your child will be fully debriefed and provided with a list of trauma counselling centres and trauma counsellors if needed. If you wish to discuss the information above or any discomforts you may experience, you may ask questions now or call the Principal Investigator listed above. It is important to note that if any child reveals abuse or any traumatic events they have experienced that is not known by parents and/or the relative authorities, our legal obligation is to report this information.

25. What are the benefits of this research?

Information from this study will improve our understanding of how childhood trauma affects memory and thinking in later life.

26. If you choose to allow your child to take part in this research study, will it cost you anything?

Allowing your child to participate in this study will not cost you anything.

27. Can your child withdraw from this research study?

You are free to withdraw your consent and to stop your child participating in this research study at any time. If you do withdraw your consent, there will be no penalty. If you have any questions about your child's rights or welfare as a research participant, you may contact Professor Marc Blockman, the head of the UCT Faculty of Health Sciences Research Ethics Committee, on 021-406-6496.

28. If your child withdraws, can information about your child still be used and/or collected?

Information already collected may be used.

29. Once personal and performance information is collected, how will it be kept secret (confidential) in order to protect you and your child's privacy?

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people have the right to review these research records. These people include the researchers for this study and certain University of Cape Town officials. Your child's research records will not be released without your permission unless required by law or a court order.

30. What information about your child may be collected, used and shared with others?

This information gathered will be records of your child's performance on cognitive tests, as well as information on their history and current psychological functioning.

31. How will the researcher(s) benefit from your child being in the study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigator and others attached to this research project may benefit if the results of this study are presented at scientific meetings or in scientific journals.

32. Signatures

You have been informed about this study's purpose, procedures, possible benefits, and risks; and how your child's performance and other data will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You agree to allow participation in this study. You hereby give permission for the collection, use and sharing of your child's performance and other data. By signing this form, you are not waiving any of your legal rights.

Signature of Person Consenting and Giving Permission

Date

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; and how the participant's performance and other data will be collected, used, and shared with others:

Signed by candidate

Signature Removed

Signature of Researcher (Person Obtaining Consent and Permission)

THANK YOU SO MUCH FOR YOUR TIME AND FOR CONSIDERING THIS RESEARCH

APPENDIX H**Example of Assessment Report****UNIVERSITY OF CAPE TOWN**

Department of Psychology

14 June 2009

RE: Tandi Surname

Dear Parent/Guardian:

Thank you very much for consenting to Tandi's participation in our research study. She was selected as a participant and assented to participate. During the two testing sessions I administered various cognitive tests to Tandi. She completed all of them, including tests of memory, attention, learning, spatial navigation, planning, decision-making, judgment, and problem-solving. Because this is a research study and not an individual clinical assessment, the feedback I can give you about results of these tests can only be stated in terms of the range in which your child performed.

These ranges are determined by the population in which the test was developed (and most of the tests we used were developed in the United States or United Kingdom). Therefore, if the table below indicates that the child performed in the "average" range, then what we are saying is that, on the test in question, the child's performance was the same as that of an average child of the same age in the US or UK.

Results for test battery:

Test of:	Range of Performance
Verbal memory - Immediate recall	Below Average
Verbal memory - Delayed recall	Low Average
Verbal memory - Delayed recognition	Average
Visual working memory capacity-forward	High Average to Superior
Visual working memory capacity-backward	High Average to Superior
Visual episodic memory	High Average
Visual-Spatial recognition memory	Low Average
Planning	Low Average
Rule acquisition	Average
Inhibition	Average
Inhibitory Control & Cognitive Flexibility	Below Average
Verbal learning	Below Average
Visual learning	High Average
Attention	Average

Overall, Tandi showed a varied performance in the test battery, with some areas of significant strength (e.g., on tests of visual working memory where pictures were presented to her and she had to remember the order they were presented in), and some areas of weakness (e.g., on tests of verbal working memory and verbal learning, where a list of words are to be remembered and recalled). It is notable that in terms of both memory and learning, Tandi appeared to learn better when information was presented visually (e.g., in the form of pictures) as opposed to orally (i.e., when it was read out to her).

If you have any questions or concerns about the study or about our findings, you may contact us at the numbers below. Also, if you would like a copy of the final research report we are creating, please e-mail Ms. Ashley-Cooper.

Michaela Ashley-Cooper
M.Soc.Sci Candidate
Department of Psychology
University of Cape Town
0832473954
ashmic005@uct.ac.za

Kevin G. F. Thomas, Ph.D.
Senior Lecturer and Supervisor
Department of Psychology
University of Cape Town
021-650-4608
kevin.thomas@uct.ac.za

APPENDIX I

ART Scoring Sheet

Subject no.: _____

- | | |
|--|-------|
| 1) Correct layout of the room (i.e. 1, 3, 1, 3 picture layout) (2) | _____ |
| 2) Target situated in the corner between Items 8 & 1 (2) | _____ |
| 3) Item 1 alone on a wall (2) | _____ |
| 4) Item 1 east of target (2) | _____ |
| 5) Item 8 north of target (2) | _____ |
| 6) Item 8 north of target in correct position (2) | _____ |
| 7) Item 7 north of target (2) | _____ |
| 8) Item 7 north of target in correct position (2) | _____ |
| 9) Item 6 north of target (2) | _____ |
| 10) Item 6 north of target in correct position (2) | _____ |
| 11) Items 6,7,8 on the same wall (2) | _____ |
| 12) Items 6,7,8 on the same wall north of target (2) | _____ |
| 13) Item 4 alone on a wall (1) | _____ |
| 14) Item 4 west of target (1) | _____ |
| 15) Item 5 south of target (1) | _____ |
| 16) Item 5 south of target in correct position (1) | _____ |
| 17) Item 2 south of target (1) | _____ |
| 18) Item 2 south of target in the correct position (1) | _____ |
| 19) Item 3 south of target (1) | _____ |
| 20) Item 3 south of target in the correct position (1) | _____ |
| 21) Items 5,2,3 on the same wall (1) | _____ |
| 22) Items 5,2,3 on the same wall south of the target (1) | _____ |
| 23) Entire reconstruction correct (1) | _____ |

TOTAL _____

Out of 35.

APPENDIX J

Post-hoc Results from the CG Arena: Invisible Trials

Invisible Trials: Main Effect of ‘Trials’

Table A1

Post-hoc analysis results using Tukey’s HSD test: main effect of ‘trials’ on invisible trials data in the CG Arena.

	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5	Trial 6	Trial 7	Trial 8
Trial 1		0.025	0.0001	0.0001	0.00015	0.0001	0.0001	0.0001
Trial 2	0.025		0.371	0.716	0.887	0.608	0.284	0.096
Trial 3	0.0001	0.371		1.000	0.991	1.0000	1.000	0.999
Trial 4	0.0001	0.716	1.000		1.000	1.000	0.998	0.948
Trial 5	0.00015	0.887	0.991	1.000		1.000	0.977	0.826
Trial 6	0.0001	0.608	1.000	1.000	1.000		1.000	0.977
Trial 7	0.0001	0.284	1.000	0.998	0.977	1.000		1.000
Trial 8	0.0001	0.096	0.999	0.948	0.826	0.977	1.000	

Note. The data presented are p values for comparing differences across trials on average length to target (for the whole sample, regardless of sex or group).

APPENDIX K

Post-hoc Results from the CG Arena: Probe Trial

Probe Trial: Interaction Effect of Sex x Group

Table A2

Post-hoc analysis results using Tukey's HSD test: interaction effect of sex x group membership on probe trial data in the CG Arena.

	Control Girls	PTSD Girls	Trauma Girls	Control Boys	PTSD Boys	Trauma Boys
Control Girls		0.055	0.474	0.924	1.000	0.879
PTSD Girls	0.055		0.863	0.541	0.097	0.699
Trauma Girls	0.474	0.863		0.985	0.533	0.997
Control Boys	0.924	0.541	0.985		0.916	1.000
PTSD Boys	1.000	0.097	0.533	0.916		0.872
Trauma Boys	0.879	0.699	0.997	1.000	0.872	

Note. The data presented are p values for comparing differences across the three groups and across the different sexes on time spent in the target quadrant.

APPENDIX L

Separate Regression Results for the Factor: Perpetrator

Table A3

Separate Regression Results of Factor: Perpetrator

	Model <i>F</i> (1, 20)	Model <i>p</i> value	Multiple <i>R</i> ²	β	β <i>t</i> (20)	β <i>p</i> value
Working memory	0.414	0.527	0.020	0.142	0.644	0.527
Verbal memory	0.164	0.690	0.008	-0.090	-0.405	0.690
Visual memory	0.064	0.803	0.003	-0.057	-0.253	0.803
Spatial navigation	0.006	0.940	0.0003	-0.017	-0.076	0.940
Rule acquisition	0.074	0.788	0.004	0.061	0.273	0.788
Inhibition	0.050	0.826	0.003	-0.050	-0.222	0.826
Problem solving	0.707	0.410	0.034	0.184	0.841	0.410
Processing speed	0.493	0.491	0.024	-0.155	-0.702	0.491
Decision- making/Impulsivity	0.529	0.475	0.026	0.161	0.727	0.475

Note. In this regression the factor of perpetrator is measured as either known or stranger; known was inputted at 101, and stranger was inputted at 102.