Differentiating the Effects of Posttraumatic Stress Disorder and Sexual Abuse on Neurocognitive Performance in Traumatized South African Adolescents

- MASTER THESIS -

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Abstract

Development of posttraumatic stress disorder (PTSD) is a common sequela after experiencing a traumatic event. Despite clinical symptoms like increased arousal and re-experiencing, neuropsychological impairments are commonly observed in adult PTSD population. Moreover, an aggravation of this phenomenon through childhood sexual abuse (CSA) has been suggested. However, attempts to generalize these results to children and adolescents lead to inconsistent results.

The present study assessed the impact of both PTSD diagnosis and CSA experience on various cognitive functions in traumatized South African adolescents while hypothesizing lower performance in those participants with CSA and PTSD. In a cross-sectional design, 105 adolescents were tested with a broad neuropsychological battery including memory, executive functioning, and language assessments.

Results of variance analyses did not confirm our expectations and revealed PTSD and CSA related impairments only in the domain of verbal memory; however, the experience of CSA often inverted the pattern of lower scores associated with PTSD. Although we did not find correlations between neuropsychological performance and CSA/PTSD severity, the scores were able to predict the severity in regression analyses.

This study highlights the impact of PTSD and CSA on neurocognition. However, the discrepancies of our findings in contrast to studies in adult populations are not understood yet. Acknowledging methodological limitations, we discuss the implications of the current findings. As the effects of CSA and PTSD appear to be stronger than the impact of trauma alone, more studies in this area are crucial to expand our understanding of long-term consequences of trauma, CSA, and PTSD.

Key words: Posttraumatic stress disorder (PTSD), childhood sexual abuse (CSA), neurocognitive functioning, adolescents

Zusammenfassung

Viele Menschen, die ein traumatisches Ereignis erleben, entwickeln in der Folge eine Posttraumatische Belastungsstörung (PTBS). Neben klinischen Symptomen berichten Studien, dass sich häufig neuropsychologische Beeinträchtigungen in erwachsenen PTBS Patienten beobachten lassen - ein Phänomen, das ebenso nach sexuellen Missbrauchs während der Kindheit (SMK) auftritt. Jedoch scheinen sich diese Befunde nicht ohne Einschränkungen auf Kinder und Adoleszente übertragen zu lassen.

Die vorliegende Studie hatte das Ziel, den Einfluss von PTBS Diagnose sowie des Erlebens von SMK auf verschiedene neurokognitive Funktionen zu untersuchen um so die Hypothese zu testen, dass PTBS und SMK mit verschlechterten Leistungen einhergeht. In einem querschnittlichen Design wurden 105 südafrikanische Adoleszente mit einer breiten neurokognitiven Batterie (Gedächtnis, exekutive Funktionen und Sprachfähigkeit) untersucht.

Jedoch konnten die Ergebnisse der ANOVA unsere Annahmen nicht bestätigen und zeigten lediglich PTBS und SMK assoziierte Performanzdefizite im verbalen Gedächtnis, bei dem die Erfahrung von SMK das Muster von schlechterer Leistung in der PTBS Gruppe umkehrte. Obwohl die Korrelationsanalysen keine Ergebnisse zeigten, deuten die Regressionen daraufhin, dass sich die PTBS- und SMK-Schwere durch die Ergebnisse in den neuropsychologischen Tests vorhersagen lässt.

Diese Studie beleuchtet den Einfluss von PTBS und SMK auf Neurokognition, aber mehr Forschung ist notwendig um zu verstehen, wie diese Ergebnisse – die sowohl von unseren Hypothesen als auch der Forschung im Bereich von Erwachsenen widersprechen – zu verstehen und erklären sind. Neben methodischen Schwächen diskutieren wir mögliche Implikationen dieser Ergebnisse. Da die Effekte von PTBS und KMS sich von den Effekten bloßer Traumatisierung unterscheiden, wären mehr Studien in diesem Bereich wünschenswert um die Langzeitfolgen von Trauma, KMS und PTBS zu verstehen.

Schlüsselwörter: Posttraumatische Belastungsstörung (PTBS), sexueller Missbrauch während der Kindheit (SMK), neurokognitive Funktionen, Adoleszente

Contents

List of Abbreviations

ACTH	Adrenocorticotropic hormone
ANOVA	Analysis of Variance
ANS	Autonomic nervous system
B	Standardized regression coefficient
BDI	Beck Depression Inventory
β	Unstandardized regression coefficient
CD-RISC	Connor-Davidson Resilience Scale
CEA	Childhood Emotional Abuse
CEN	Childhood Emotional Neglect
COWAT	Controlled Word Association Test
CPA	Childhood Physical Abuse
CPC	Childhood Post Traumatic Stress Disorder Checklist (CPC)
CPN	Childhood Physical Neglect
CRH	Corticotrophin-releasing hormone
CSA	Childhood sexual abuse
CSA-	CSA experience negative
CSA+	CSA experience positive
CTQ	Childhood Trauma Questionnaire
DRT	Dual Representation Theory
DSM	Diagnostic and Statistical Manual of Mental Disorders
E.g.	For instance
EF	Executive functions
fMRI	Functional magnet resonance imaging
GC	Glucocorticoids
HPA	Hypothalamic-pituitary-adrenal axis
I.e.	That is
IQ	Intelligence
K-SADS-PL	Kiddle-Schedule of Affective Disorders and Schizophrenia - Present and Lifetime Version
LEQ	Life Events Questionnaire
M	Mean
MRI	Magnet resonance imaging

N.s.	Not significant
NCC	Number of categories completed
P.	Page
Pp.	Pages
PET	Positron Emission Tomography
PFC	Prefrontal cortex
PTSD	Posttraumatic Stress Disorder
PTSD-	PTSD diagnosis negative
PTSD+	PTSD diagnosis positive
RAVLT	Rey Auditory Verbal Learning Test
RCFT	Rey-Oesterrieth Complex Figure Test
SAM	Situationally accessible memory
SD	Standard Deviation
SE	Standard error
SSAIS-R	Senior South African Individual Scale-Revised
TC1	Trials to complete category 1
TMT	Trial Making Test
VAM	Verbally accessible memory
WCST	Wisconsin Card Sorting Test
WM	Working Memory
WMS-R	Wechsler Memory Scale-Revised

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Introduction

> "Dementors are among the foulest creatures that walk this earth. They infest the darkest, filthiest places, they glory in decay and despair, they drain peace, hope, and happiness out of the air around them. […] Go too near a dementor and every good feeling, every happy memory, will be sucked out of you. […] You'll be left with nothing but the worst experiences of your life." (Rowling, 1999, p. 187)

Within the famous novels written by J. K. Rowling, dementors are the wizarding prison guards who punish their prisoners by forcing them to re-experience their worst memories and feelings. Upon meeting these dementors, the main character Harry Potter is forced to hear the last moments in his mother's life – her crying and screaming – before she is murdered. Persons with psychological knowledge might associate the description of these beings and their impact on humans with a psychiatric disorder first mentioned in the Diagnostic and Statistical Manual of Mental Disorders (DSM, American Psychiatric Association) in 1980 – the Posttraumatic Stress Disorder (PTSD). Before PTSD found its way into the DSM-III, the psychopathology was known under different names specially deriving from wars, e.g. soldier's heart, shell shock, and combat fatigue (Pitman, Rasmusson, Koenen, & Shin, 2012). However, all constructs had similar symptoms, including re-experiencing, avoidance, and arousal. PTSD was the first disorder requiring a specific causing event conceptualized as "an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone" (A criterion, DSM-III). The concept was based on the assumption that traumatic events (in contrast to other stressful events) are linked etiologically to a specific syndrome (Breslau, 2009).

The authors of the DSM-III were convinced that the disorder is only present in extreme stressors and therefore seldom, but epidemiological data suggest a different pattern. The lifetime prevalence to experience a traumatic event according to the DSM is nearly 90 percent (Breslau et al., 1998), and lifetime prevalence rates for PTSD differ between countries and studies from one to eight percent (Breslau, 2009; Kessler, Sonnega, Bromet, Huhges, & Nelson, 1995), with a lifetime prevalence of 1.3% in Germany (Perkonigg, Kessler, Storz, & Wittchen, 2000). Studies examining PTSD prevalence in South Africa, however, report higher levels (Ensink, Robertson, Zissis, & Leger, 1997; Peltzer, 1998, 1999; Seedat, van Nood, Vythilingum, Stein, & Kamlner, 2010; Suliman, Kaminer, Seedat, & Stein, 2005; Ward, Flisher, Zissis, Muller, & Lombard, 2001). For instance, a study from 2004 (Seedat, Nyamai,

Njenga, Vythilingum, & Stein, 2004) collected data in nine schools in Cape Town asking grade 10 students to complete anonymous self-report questionnaires. More than 80% of 1140 adolescents reported exposure to severe trauma with 22% showing current full-symptom PTSD and 12% current partial-symptom PTSD, respectively (Seedat et al., 2004). Reasons for the fluctuation in prevalence rates most likely lay in differences in social general framework and other circumstances since these determine the frequency of traumatic events. This could provide an explanation for the differences in PTSD prevalence between South Africa and Western countries. According to Seedat and colleagues (2004), 44% of adolescents were exposed to three or more traumatic events and those meeting the diagnosis for PTSD endorsed more traumas than people with partial-syndrome or without PTSD. Additionally, the type of trauma plays a role for different prevalence due to a "conditional probability" for developing PTSD depending on the type of trauma (Hidalgo & Davidson, 2000) and varying between the sexes (Nemeroff et al., 2006). Sexual and physical assaults as well as serious accidents are associated with the highest risk for a subsequent PTSD symptomatology (Seedat et al., 2004).

Trauma exposure and PTSD symptomatology can have severe consequences. Besides the emotional and behavioural problems referred to in DSM criteria (see Appendix), cognitive impairments are often observed in adults suffering from PTSD and can be found in several domains including attention, memory, and cognitive functioning (Scott et al., 2015). Literature about cognitive functioning in traumatized adolescents, however, is sparse and often does not include severe and chronically traumatized adolescents (Schoeman, Carey, & Seedat, 2009). Since neuropsychological performance is often linked to academic achievement, impairments in cognitive domains play an important role in neurobehavioral difficulties (Kavanaugh, Dupont-Frechette, Jerskey, & Holler, 2016). making it crucial to expand knowledge in this field. Studies in the field of adolescent PTSD and neurocognitive impairments could have theoretical and practical implications. On one hand, assessing a broad domain of cognitive functions with standardized neuropsychological tests might offer insight into the underlying neurobiological processes affected by PTSD symptomatology. It is also important for understanding the correlates and mechanisms of PTSD. On the other hand, it enables the identification of adolescents in need for interventions improving cognitive functions. Studies investigating scholastic impairments in PTSD patients show lower performances in the domains of vocabulary, reading, spelling, mathematics, languages, and science compared to both healthy and traumatized controls (Saigh, Mroueh, & Bremner, 1997). Thus, further research is needed to identify affected neurocognitive domains. In addition, a better understanding of the relationship between history of traumatic events,

PTSD, and neurocognition could help development and implementation of interventions that aim to improve cognitive functioning. Moreover, identifying fundamental cognitive deficits associated with PTSD may not only support everyday academic and social functioning of patients but further support treatment objectivities to reduce PTSD symptomatology (Carrion, Wong, & Kletter, 2013).

Therefore, this study aimed to study neurocognition in traumatized South African adolescents by quantifying the impairments in dependency of PTSD diagnosis and history of sexual abuse in a cross-sectional design. Firstly, the theoretical background for this research question will be reviewed. After illustrating the methods and study design used to answer the research question, results will be displayed and finally discussed in the last section with a focus on possible implications as well as limitations.

Theoretical Background

Stress Effects on Memory

Confrontation with stress prompts the brain to activate two biological stress systems, namely the autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis (Joëls & Baram, 2009). If limbic and prefrontal structures appraise a situation as threatening, the hypothalamus sends signals to the adrenal medulla via the sympathetic nervous system. Within seconds the adrenal medulla releases catecholamines causing, for example, an increase in heart rate and blood pressure. In addition to the activation of the ANS, the hypothalamus releases the corticotropin-releasing hormone (CRH), which in turn prompts the pituitary to unleash the adrenocorticotropic hormone (ACTH). ATCH reaches the adrenal cortex via the blood stream, where it induces the release of glucocorticoids (GC, i.e. cortisol in humans). This system is known as HPA axis.

Albeit being less intense, everyday stress is similar to physiological responses during a traumatic event; hence, studying the impact of acute stress on healthy individuals can provide important insights on trauma memory (Merz, Elzinga, & Schwabe, 2016). Stress has differential effects on learning depending on the temporal relationship between stressor and memory processes (Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012; Schwabe & Wolf, 2013). Stress during or immediately after learning facilitates memory performance making it more rigid and inflexible ("habit learning"; Schwabe & Wolf, 2013). Besides literature suggesting emotionally arousing events to be better remembered (Cahill & McGaugh, 1998) – especially when participants are stressed (Cahill, Gorski, & Le, 2003; Smeets, Otgaar, Candel, & Wolf, 2008; Zoladz et al., 2011) – the release of catecholamines and cortisol during the traumatic event is assumed to lead to an "overconsolidation" (Pitman, 1989) of trauma memory, in turn causing the development of PTSD symptoms.

Dual Representation Theory (DRT)

Psychological theories of PTSD try to explain why some people develop a psychiatric disorder subsequent to the experience of a traumatic event while others do not. Of note, the symptoms itself are not the defining aspect of PTSD, rather it is their frequency, longevity, and the associated impairment (Brewin, 2007). To explain the development of PTSD, the Dual Representation Theory (DRT; Brewin, Dalgleish, & Joseph, 1996) puts emphasis on certain memory types and assumes that these different types of memory can explain the

complex phenomenology of PTSD. It postulates that trauma memory can be stored in two different representation formats: verbally accessible memory (VAM) and situationally accessible memory (SAM). VAM is conscious, hippocampus-dependent, and deliberately retrievable from the store of autobiographic experiences; whereas SAM is non-conscious, triggered by external cues, and not discriminating between present and past. SAM is supposed to contain information obtained from more extensive, lower level perceptual processing of the trauma and the person's bodily response to it. SAM can be evoked by stimuli similar to the traumatic event supporting trauma-related dreams and flashbacks.

The amydgala is the key structure for processing fear memory and receives threat information from the sense organs via separate pathways (LeDoux, 1994). With the thalamus being the starting point, sensory information is either directly sent to the amygdala using rapid, subcortical pathways or is processed in cortical regions and hippocampus independently projecting to the amygdala. During traumatic events, encoding of VAM may be highly selective because of anxiety processes that focus attention and decrease short-term memory capacity. According to the model, that is why VAM contains only some of the sensory features and emotional or physiological responses. Deficits regarding trauma memory occur in the VAM as opposed to the SAM, which is not hippocampus dependent (Brewin, 2001). Because of the stress-induced release of GC during trauma and their effects on hippocampus, it was hypothesized that VAM shows degrees of disorganisation, degradation, and incompleteness with memory deficits centering on "hot spots" (Brewin, 2001). The thalamo-amygdala route, however, is less sophisticated and needs fewer resources for processing trauma features. Hence, SAM corresponds to a superficial encoding of lower-level sensory features with a relationship between stimuli and primary emotions.

The processing of flashbacks, which are supported by SAM and common in the aftermaths of a trauma, has a critical role in the development of PTSD. If the person focuses attention on the content of flashbacks, it is possible to recode additional sensory information associated with periods of intense emotion into the incomplete VAM. By integrating into VAM, the information acquires a temporal location in the past, is deliberately retrievable and no longer triggered by external cues, thus inhibiting symptoms of re-experiencing. PTSD patients, however, fail to create complete VAM representations, resulting in a considerable amount of trauma features only encoded via SAM and vulnerable for re-activation by external cues. VAM representation remains impoverished when a person's behaviour is marked by avoidance. This is in line with the mnemonic model of PTSD (Rubin, Berntsen, & Bohni, 2008) which claims that explicit memory of the event rather than the event itself causes the

symptomatology. The unsuccessful adaption can result in two different outcomes (Brewin et al., 1996): chronic emotional processing and premature inhibition of processing. Chronic emotional processing is marked by permanent preoccupation with the consequences of trauma and intrusive memories as well as chronic heightened arousal and attention/memory biases. Further, secondary reactions like depressive mood or anxiety are likely. On the other hand, premature inhibition of processing is characterized by attentional biases favouring threat information, impaired memory of the traumatic event, phobic avoidance of trauma-related situations, and evidence for somatization.

Besides evidence deriving from studies using experimental traumatization (see e.g. Hellawell & Brewin, 2002, 2004; Holmes, Brewin, & Hennessy, 2004), there are connections between DRT and neuroscientific findings (Brewin, 2001). For instance, research showing that stress alters the recruitment of multiple memory systems (Schwabe, 2013) supports presumptions of DRT. Multiple memory systems are associated with different brain regions (Squire & Zola-Morgan, 1991). Neuroimaging studies found evidence for a stress induced shift from hippocampus-dependent to hippocampus-independent learning by influencing the connectivity between brain regions (Schwabe, Tegenthoff, Höffken, & Wolf, 2013; Vogel et al., 2015). Additionally, there is evidence for the assumptions of a bad functioning hippocampus-dependent VAM when looking at neurobiological findings.

Neurobiological Findings in PTSD

Adult PTSD population.

Multiple interconnected neurobiological systems are involved in the (de-)activation of stress response and hence, in the development and maintenance of PTSD. If the HPA system does not function properly or if it is overactivated, a posttraumatic stress reaction may occur, resulting in a constant state of hyperarousal. Certain alterations were found in adult patients with PTSD, including neuroendocrinological systems (Herane Vives et al., 2015; Meewisse, Reitsma, De Vries, Gersons, & Olff, 2007; Morris, Compas, & Garber, 2012; Wahbeh & Oken, 2013) and structural/functional neuroanatomy (e.g. Kühn & Gallinat, 2013; Patel, Spreng, Shin, & Girard, 2012). These alterations can be linked to specific symptoms (for a review see e.g. Heim & Nemeroff, 2009; Pitman et al., 2012; Sherin & Nemeroff, 2011). Research findings show decreases in cortisol concentrations compared to controls (for a recent overview see Lehrner, Daskalakis, & Yehuda, 2016). This hypocorticolism is owed to an increased sensitivity of the HPA to negative GC feedback (hyper-responsive GC receptors)

and seems to be related to higher responses to stress, abnormal stress encoding, and fear conditioning (Sherin & Nemeroff, 2011).

Further, overactive cortisol segregation due to chronic stress contributes to neuronal cell death, decreased neuronal dendritic branching as well as inhibition of neurogenesis in brain regions with high GC receptor density (Sapolsky, 2000; Sapolsky, Krey, & McEwen, 1985; Sapolsky, Uno, Rebert, & Finch, 1990). Atrophy in hippocampal and frontal regions have been found in adult PTSD patients compared to healthy controls. Reduced hippocampus volume (Karl et al., 2006; Kitayama, Vaccarino, Kutner, Weiss, & Bremner, 2005; Smith, 2005; Woon & Hedges, 2008) is associated with poorer control of stress responses, declarative memory, and extinction (Sherin & Nemeroff, 2011). The association of hippocampus and memory, therefore, is in line with the predictions made by DRT. This further interacts with aberrations in the HPA axis since the hippocampus is the major brain region inhibiting stress response. Volume decline in prefrontal regions (Karl et al., 2006; Kühn & Gallinat, 2013) could be associated with impairments in higher cognitive and neuropsychological functioning. These structural alterations in neuroanatomy could lead to dysfunctional circuits in PTSD patients down regulating the adaption to stress. Such dysfunctional circuits could also be observed on a functional level: findings of a meta-analysis of fMRI studies of PTSD support the assumptions of a hyperactive amydgala and hypoactive prefrontal regions (Patel et al., 2012).

Adolescent PTSD population.

To understand the neurobiological and neuropsychological sequelae of trauma exposure in children and adolescents, postnatal development needs to be considered since the associated plasticity has far-reaching implications either as a protective or a risk factor for the development of PTSD (Macdonald, Vasterling, & Rasmusson, 2012). Neurodevelopmental processes during childhood can be severely disrupted/altered by trauma experiences, thus resulting in environmentally induced complex developmental disorders as hypothesized in developmental traumatology frameworks (De Bellis, 2001; De Bellis & Zisk, 2014). Several studies provide support for this assumption; for instance by reporting smaller intracranial volume in children with PTSD compared to control participants (Carrion et al., 2001; De Bellis et al., 1999; Teicher et al., 1997) with smaller volumes being related to earlier onset of trauma (De Bellis, Keshavan, Frustaci, et al., 2002). In particular, reduced prefrontal volumes have been observed (Carrion et al., 2009; Carrion, Weems, Richert, Hoffman, & Reiss, 2010). Besides the detection of structural changes in prefrontal cortex (PFC) (Carrion et al., 2001; Carrion, Weems, et al., 2010; Richert, Carrion, Karchemskiy, & Reiss, 2006), a functional

MRI study could demonstrate decreased prefrontal activation in an inhibition task (Carrion, Garrett, Menon, Weems, & Reiss, 2008). Impaired structure and function of the PFC may contribute to cognitive impairments observed in children and adolescents with PTSD, especially in executive functioning since the PFC supports cognitive control (Casey, Tottenham, & Fossella, 2002).

Although reduced hippocampal volume is often found in adults suffering from PTSD, this could not be replicated in children and adolescents, neither cross-sectionally (De Bellis et al., 1999; De Bellis, Keshavan, Shifflett, et al., 2002) nor longitudinally (De Bellis, Hall, Boring, Frustaci, & Moritz, 2001). Carrion and colleagues reported smaller hippocampal volume in children with PTSD compared to control subjects, but this effect did not remain significant after controlling for total brain volume (Carrion et al., 2001). However, severity of PTSD could predict hippocampal volume loss in adolescents over an ensuing 12-18 month interval (Carrion, Weems, & Reiss, 2007). In their meta analysis of hippocampal volume loss in children and adults with maltreatment-related PTSD, Woon and Hedges (2008) found smaller hippocampi in adults, but not in children, thus leading to conclusion of maltreatment-related volume changes not being apparent until adulthood. Therefore, overexposure to cortisol as reported by Carrion and colleagues (2002) may damage the hippocampus, but effects seem to not emerge until later development (Carrion et al., 2013), making hippocampal volume decline eventually dependent on duration and chronicity of PTSD symptoms. This is in line with a MRI study done in adults demonstrating a negative correlation between hippocampus volume and PTSD duration (Felmingham et al., 2009). Moreover, PTSD has high rates of comorbid disorders (American Psychiatric Association, 2013). Hence, psychiatric comorbidities like alcohol and substance dependence, often observed in adults with PTSD, could account for volume decline of the hippocampus (De Bellis, Spratt, & Hooper, 2011). The comorbidity between addictive disorders and PTSD in adolescents, however, is smaller providing another explanation for the inconsistent findings between adolescents and adults. Finally, capacity for neurogenesis in the hippocampus and developmental brain volume increases (Fuchs & Gould, 2000) are probably masking any effects in hippocampal volume in paediatric patients with PTSD (De Bellis et al., 2011). In absence of consistent hippocampal atrophy, however, activation deficits of the hippocampus were found during memory retrieval (Carrion, Haas, Garrett, Song, & Reiss, 2010) with higher symptom scores being negatively correlated to activation during retrieval. This functional alteration was associated with decreased retrieval accuracy.

To sum up, "PTSD has become one of the better understood psychiatric disorders from a biological standpoint" (Pitman et al., 2012, p. 782) and volumetric and functional abnormalities referred to above may be associated with cognitive impairments found in patients with PTSD, indicating differences in brain responses (Carrion et al., 2013). However, results deriving from preliminary literature in the field on neurobiological changes caused by PTSD reveal patterns that are different in children and adolescents compared to adults. The next section aims at answering the question of whether such inconsistencies are also found in memory and executive functioning, respectively.

Neurocognitive Deficits in PTSD

Memory impairments in adults.

The Oxford Dictionary of Psychology describes memory as the "psychological function of preserving information, involving the processes of encoding, storage, and retrieval" ("Memory," 2014). One of the first studies targeting the question if memory differences can be observed when comparing PTSD patients with healthy or traumatized controls in performances on neuropsychological tests was conducted by Klonoff and colleagues in 1976 (Klonoff, McDougall, Clark, Kramer, & Horgan, 1976), even before PTSD was considered as a diagnosis in DSM-III (American Psychiatric Association, 1980). They compared two groups of war prisoners (low vs. high stress) in their performances in neurocognitive tests and found several differences indicating more problems in the high stress group. They interpreted these results within the hypothesis of a "survivor syndrome or war neurosis" (Klonoff et al., 1976, p. 251). Although many of the following studies were conducted using veterans as participants (e.g. Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Vasterling et al., 2002), the results of cognitive impairment could also be replicated in other (civilian) populations, including rape victims (Jenkins, Langlais, Delis, & Cohen, 1998), sufferers of childhood abuse (Bremner et al., 1995; Bremner, Vermetten, Afzal, & Vythilingam, 2004), traffic accident victims (Bae, Hyun, & Lee, 2014) as well as holocaust survivors (Golier et al., 2002).

Brewin and colleagues (Brewin, Kleiner, Vasterling, & Field, 2007) reviewed 27 studies investigating memory functioning in PTSD adults and reported a mean effect $d = .200$ [95%CI .170 - .229] indicating a small to moderate effect (Cohen, 1992) for impaired memory performance in PTSD when compared to controls with higher effect sizes for verbal than for visual material, but without significant differences for retention period. Memory disturbances in immediate rather than delayed recall suggest dysfunction in attention or

strategic processes, whereas the reverse pattern can be considered as difficulty in retaining newly learned information (Brewin et al., 2007). Since they did not find significant differences, it remains unclear whether PTSD patients suffer from acquisition impairment versus reduced ability to consolidate memory. It is believed that decrements in long-term memory (delayed recall) are due to deficits in encoding (Gilbertson et al., 2001; Jelinek et al., 2006; Johnsen & Asbjørnsen, 2009). This points to the suggestion that the memory deficits on both domains are associated with attentional dysfunction. Further, the meta-analysis found larger effect sizes when using a non-traumatized (healthy) control group compared to a traumatized (trauma-exposed) control group. The variance of effect sizes due to the type of control group allows for the assumption that experiencing a traumatic event may have an effect on memory performance independent of diagnosis, but cannot account on its own for the observed difficulties in memory.

A second meta-analysis conducted by Johnsen and Asbjørnsen (2008) came to similar conclusions. They report a moderate effect size d = .74 [95%CI .58 - .90] for impaired verbal memory based on 28 studies differing in dependency of the chosen control group. Similar to the results reported by Brewin and colleagues (Brewin et al., 2007), the type of trauma had an effect on impairments within civilian samples showing smaller effect sizes than military samples. They attribute this to the fact that military samples mostly contain Vietnam veterans who suffer from chronic PTSD and hence the memory impairment could be related to illness duration.

Because of different mnemonic disturbances in PTSD patients, it was suggested to characterize it as a disorder of memory (Brewin, 2011; Rubin et al., 2008). For instance, the occurrence of intrusive memories can be seen as a deficit in episodic memory (Schönfeld, Boos, & Müller, 2011). These findings are in line with the DRT, postulating that re-experiencing symptoms are caused by a well-functioning image-based memory system (SAM) that cannot be inhibited by a badly functioning verbal system (VAM). Brewin and colleagues (2007) assumed that these symptoms can be measured using non-autobiographical memory tests and hypothesized that patients with PTSD should show impaired verbal but not visuospatial memory. The finding of larger impairments in verbal memory compared to visual memory supports the predictions of the DRT to some extent (Brewin, 2007). Nevertheless, a decrease in non-verbal memory function was observed, too.

Memory impairments in adolescents.

Examining memory disturbances in children and adolescents with PTSD, Moradi and colleagues (Moradi, Doost, Taghavi, Yule, & Dalgleish, 1999) reported poorer overall

everyday memory performance in PTSD patients compared to healthy controls and to the norm of the used neuropsychological test. Expanding their results, they asked patients with PTSD and healthy controls to learn 60 words – of which 36 were negative – and found an overall poorer memory performance in the PTSD population. However, the patients also recalled more negative words compared to neutral and positive words than the control group (Moradi, Taghavi, Neshat-Doost, Yule, & Dalgleish, 2000).

Further, Yasik and colleagues (Yasik, Saigh, Oberfield, & Halamandaris, 2007) demonstrated group effects for verbal memory indices with worse performance in PTSD patients compared to both healthy and trauma-exposed controls. Another study found worse memory performance in children with neglect related PTSD compared to healthy and neglected children without PTSD (De Bellis et al., 2009). In a similar vein, Ahmed and colleagues (Ahmed, Spottiswoode, Carey, Stein, & Seedat, 2012) observed group differences between PTSD patients and trauma exposed control participants in measurement of verbal and visual memory, but not all of them reached statistical significance. A study contrasting South African trauma-exposed adolescents to a PTSD population reported significant group differences for the visual but not for verbal subtests of the Wechsler Memory Scale-Revised (WMS-R) (Schoeman et al., 2009). Further, they report an effect on the total number of words learned in the Rey Auditory Verbal Learning Test (RAVLT).

When requiring participants to learn a word list provided in the California Verbal Learning Test, the majority of studies could not find an effect of PTSD diagnosis (Barrera, Calderón, & Bell, 2013; Beers & De Bellis, 2002; De Bellis, Woolley, & Hooper, 2013). Likewise, when controlling for initial learning, effects of diagnosis on delayed recall trials disappeared (Samuelson, Krueger, Burnett, & Wilson, 2010). How can these non-significant findings be explained? Samuelson and colleagues (2010), in discussing their data, highlighted that memory problems in PTSD might be related to learning rather than retrieval. Memory impairments, however, were not attributable to deficits in attention since the group differences in words learned (trials 1 to trial 5) remained significant even after controlling for performance in trial 1 – an indicator for initial attention.

Attention and executive functioning in adults.

Executive functions (EF) or "control of complex goal-directed behaviour" (Aupperle, Melrose, Stein, & Paulus, 2012, p. 686) can be divided in different concepts including attention/working memory, inhibition as well as flexibility/switching and planning/problem solving. Attention ("Attention," 2014) can be defined as "sustained concentration on a specific stimulus, sensation, idea, thought, or activity, enabling one to use information

processing systems with limited capacity to handle vast amounts of information available from the sense organs and memory stores", whereas working memory (WM) is a "temporary store for recently active items that are currently occupying consciousness and that can be manipulated and moved in and out of short-term memory" ("Working memory," 2014). On the other hand, inhibition refers to the prevention of automatic responses to maintain goal-directed behaviour. Flexibility/switching displays the ability to switch between different tasks or strategies and planning/problem solving can be described as the ability to develop and implement strategic behaviours to obtain a future goal (Aupperle et al., 2012).

Problems in concentration are also commonly reported by PTSD patients to constitute a DSM diagnostic criteria (American Psychiatric Association, 2013). In fact, deficits in attention and WM in adult patients with PTSD have been found using different paradigms (Gil, Calev, Greenberg, & Kugelmass, 1990; Gilbertson et al., 2001; Jelinek et al., 2006; Sutker, Vasterling, Brailey, & Allain, 1995; Uddo, Vasterling, Brailey, & Sutker, 1993; Vasterling et al., 2002; Vasterling, Brailey, Constans, & Sutker, 1998). Smaller scores in tasks like digit span (Jenkins, Langlais, Delis, & Cohen, 2000; Vasterling et al., 2002) reflect problems in the ability to maintain and manipulate information in order to perform cognitive tasks, whereas the Stroop test measures inhibition. In contrast to healthy controls, both traumatized participants with and without PTSD performed worse on colour and inference trials (Stein, Kennedy, & Twamley, 2002). Additionally, patients with PTSD showed a specific emotional Stroop effect compared to trauma exposed controls: they were slower in naming the colour of words related to their trauma compared to emotional and neutral words (McNally, Kaspi, Riemann, & Zeitlin, 1990).

Besides this strategic processing of threat stimuli (Buckley, Blanchard, & Neill, 2000), PTSD is associated with impairments in cognitive flexibility and switching (Twamley et al., 2009; Uddo et al., 1993). When using the Trial Making Test Part B (TMT B) – a measurement for the ability to shift between different tasks – some studies observed increased completion time in PTSD patients (e.g. Beckham, Crawford, & Feldman, 1998; Gilbertson et al., 2001; Jenkins et al., 2000; Stein et al., 2002), while others did not (e.g. Koenen et al., 2001; Twamley, Hami, & Stein, 2004). Due to the time limit within the TMT B, it can be argued that this test measures switching of attention, whereas the Wisconsin Card Sorting Test (WCST) measures switching of categories (Aupperle et al., 2012). Most studies failed to observe PTSD related deficits in the WCST (Gilbertson et al., 2006; Kanagaratnam & Asbjørnsen, 2007; Vasterling et al., 2002, 1998; but see also Gilbertson et al., 2001). In a meta-analysis, Polak and colleagues (Polak, Witteveen, Reitsma, & Olff, 2012) included 18

studies and found that people with PTSD performed worse on a variety of EF tasks compared to controls. The type of control group again influenced this finding, but – contrary to the authors' expectations – the effects were larger when comparing PTSD patients to trauma-exposed controls than when comparing them to healthy controls. The pooled mean differences in PTSD patients versus healthy controls were only significant for TMT B and Stroop task, whereas the comparison to trauma-exposed controls lead to significant effect sizes for TMT B, WCST, and digit span total.

These findings indicate impairments in a wide range of executive functioning in adult PTSD patients compared to trauma-exposed controls with impaired EF not being related to trauma exposure per se. The observed differences might rather be attributable to differences in efficiency of coping strategies (Polak et al., 2012). Further, these deficits are in line with prefrontal atrophy (Karl et al., 2006; Kühn & Gallinat, 2013) and hypoactivation (Patel et al., 2012) observed in patients with PTSD. Additionally, as previously noted, PTSD can be regarded as a memory disorder (Brewin, 2011; Rubin et al., 2008). As applied to memory, executive functions inhibit the entry of irrelevant or unwanted information into consciousness (Brewin & Andrews, 1998). Therefore, impairments in EF may be related to symptoms of re-experiences in PTSD.

Attention, executive functioning, and language in adolescents.

Based on results of neuroimaging research, Beers and De Bellis (Beers & De Bellis, 2002) were one of the first examining cognitive functioning in adolescents with PTSD using a battery of neuropsychological instruments. Although they observed poorer performance in four of six cognitive domains, only the deficits in attention and abstract reasoning/executive functioning reached significance. Compared to sociodemographically matched healthy controls, PTSD subjects completed fewer categories on the WCST and named fewer animals in Controlled Word Association Test (COWAT) – a measurement of verbal fluency and semantic organization. Besides, PTSD populations showed a lower colour word ratio in the Stroop task. Barrera and colleagues (Barrera et al., 2013) reported PTSD patients making more errors in the Stroop task in contrast to healthy controls. Similar to adults, a trauma-specific Stroop effect could be demonstrated investigating children and adolescents (Moradi, Taghavi, Neshat Doost, Yule, & Dalgleish, 1999), indicating a trauma-related attentional bias. Additionally, visual attention per se was reported to be impaired in association with PTSD as well as executive functioning (De Bellis et al., 2009). Using a complex neuropsychological test battery, De Bellis and colleagues (De Bellis et al., 2013) compared children and adolescents with maltreatment-related PTSD to maltreated participants without PTSD and

healthy controls and found evidence for trauma-associated impairments in the domains attention, language, visuospatial functioning, and EF. Further, there were PTSD associated performance deficits in visuospatial domains and executive functioning. In a similar study, Kavanaugh and Holler (Kavanaugh & Holler, 2014) observed poorer scores in both trauma groups in domains of problem solving and language compared to healthy controls. The PTSD patients further differed from healthy controls in flexibility and WM, as well as from both control groups (healthy and traumatized) in measures of EF.

However, it should be noted that not all studies could find significant effects. For instance, Yang and colleagues (Yang et al., 2014) assessed children and adolescents diagnosed with PTSD following the Sichuan earthquake in China and compared them to adolescents who experienced the same earthquake, but did not develop PTSD symptomatology in a longitudinal design (four and eight months after the earthquake). They failed to find significant group differences in any of the neuropsychological tests (Rey Oesterrieth Complex Figure Test [RCFT], Digit Span paradigm, Stroop Test, TMT, and COWAT) neither at the first nor the second time point.

Summary.

Investigating adults suffering from PTSD studies found cognitive distortions in several domains, including memory, attention, and executive functions. A very recent meta-analysis (Scott et al., 2015) was conducted with the aim to examine the profile and magnitude of cognitive deficits associated with PTSD in a broad range of cognitive domains including verbal and visual learning/memory, speed of information processing, attention/working memory, executive functioning, language, and visuospatial ability. The overall effect size was $d = -.49$ [95%CI –.452 to –.528], ranging from $d = -.29$ to $-.62$ across the specific domains and indicating general cognitive impairment in PTSD. The authors concluded that the pattern of reported deficits is consistent with dysfunction in fronto-limbic networks implicated in the pathophysiology of PTSD.

Researchers studying memory found the medial temporal lobe as being the core structure for explicit memory functioning (Squire & Zola-Morgan, 1991). As noted earlier, hippocampal volume is significantly reduced in patients with PTSD (Karl et al., 2006; Kitayama et al., 2005; Kühn & Gallinat, 2013; O'Doherty, Chitty, Saddiqui, Bennett, & Lagopoulos, 2015; Smith, 2005) which could be associated with findings of impaired memory functioning in adult patients with PTSD. Regarding children and adolescents, however, there is no evidence for hippocampal atrophy (Carrion et al., 2001, 2007, De Bellis et al., 1999, 2001; De Bellis, Keshavan, Shifflett, et al., 2002; Woon & Hedges, 2008). This is in line with

the inconsistencies observed regarding memory functioning in youths with PTSD. Although disturbances in memory lead to the highest effect sizes in adults, the results could not always be replicated when regarding children and adolescents with PTSD leading to the presumption of different mechanisms in the development and maintenance of PTSD in children and adults. However, there are in general fewer studies investigating neurocognition in adolescents suffering from PTSD and results from preliminary research in children and adolescents lead to conflicting results, especially when investigating memory function. This suggests different patterns of neurobiological and neurocognitive changes in children and adolescents after experiencing a traumatic event. Further, there is little known about moderator variables explaining the inconsistencies across studies in children and adolescents. Experiencing sexual abuse is such a potential moderator.

Effects of Childhood Sexual Abuse

Childhood sexual abuse (CSA) is defined as sexual contact or conduct between a child younger than 18 years of age and an adult or older person including assault if child resists, or inappropriate touching or foundling with sexual intent (Bernstein et al., 2003; Irigaray et al., 2013). Together with physical/emotional abuse and neglect, it represents a form of childhood maltreatment. Although there is a lack of consensus regarding prevalence rates for childhood maltreatment in general and sexual abuse specifically (Briere & Elliott, 2003; Gorey, Gorey, & Leslie, 1997), different rates for women and men are commonly reported (MacMillan et al., 1997; Perkonigg et al., 2000). Reported incidence rates of PTSD resulting from sexual abuse range from 42 to 90% (Dubner & Motta, 1999; Lipschitz, Winegar, Hartnick, Foote, & Southwick, 1999; McLeer, Callaghan, Henry, & Wallen, 1994). Besides being risk factors for later psychopathology (Beitchman et al., 1992; De Bellis et al., 2011), sexual abuse and childhood maltreatment have been identified to affect academic performance (Boden, Horwood, & Fergusson, 2007; Buckle, Lancaster, Powell, & Higgins, 2005; Coohey, Renner, Hua, Zhang, & Whitney, 2011; Eckenrode, Laird, & Doris, 1993; Jones, Trudinger, & Crawford, 2004; Kendall-Tackett & Eckenrode, 1996; Nolin & Ethier, 2007; Slade & Wissow, 2007). For instance, Mills and colleagues (Mills et al., 2011) reported an association between maltreatment defined as at least one notification to child-protective authority and poorer performance in reading and mathematics scores. The performance was independently affected by both neglect and abuse.

There is an amount of research suggesting that maltreatment may have negative outcomes on cognitive functioning (Augusti & Melinder, 2013; Cowell, Cicchetti, Rogosch,

& Toth, 2015; De Bellis et al., 2009; DePrince, Weinzierl, & Combs, 2009; Fishbein et al., 2009; Kavanaugh & Holler, 2014, 2015; Kavanaugh, Holler, & Selke, 2015; Kirke-Smith, Henry, & Messer, 2014; Palmer et al., 1997; Perna & Kiefner, 2013; Spann et al., 2012; Vasilevski & Tucker, 2015). Several studies examined the role of childhood sexual abuse on cognitive functioning in adults. Victims of childhood physical and sexually abuse diagnosed with PTSD performed worse in measurements of verbal, but not visual memory in contrast to healthy controls (Bremner et al., 1995). Extending their results by using a healthy as well as a traumatized control group to be compared to victims of CSA with PTSD, Bremner and colleagues (Bremner et al., 2004) were able to replicate the observation of impaired verbal rather than non-verbal memory. Further studies support the assumption of a negative relationship between CSA and verbal as well as visual memory (Bremner et al., 1997; Savitz, van der Merwe, Stein, Solms, & Ramesar, 2007).

When comparing participants who had experienced CSA to those who did not, Navalta and colleagues (Navalta, Polcari, Webster, Boghossian, & Teicher, 2006) could not demonstrate group effects; however, correlational analyses suggested strong relationships between duration of abuse and impairments in attention, inhibition, and memory. This is in line with suggestions made by Stein and colleagues (Stein, Hanna, Vaerum, & Koverola, 1999). In discussing their non-significant group differences between sexually abused women and matched non-victimized controls, they highlighted that differences in memory performance might not have been detectable given the young age of the participants. Further, small sample sizes might contribute to the lack of significant findings as suggested by Bremner and colleagues. Contrasting patients with CSA-related PTSD to healthy controls revealed group differences in blood flow (measured with PET) when retrieving learned words in the absence of performance deficits (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Staib, et al., 2003). In a similar way, smaller bilateral hippocampal volume and failure of hippocampus activation has been reported in victims of CSA with PTSD compared to victims of CSA without PTSD as well as healthy controls (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Nazeer, et al., 2003); however, there was no evidence for differences in memory performance. The authors discussed that studies focusing on neuropsychological assessment have larger sample sizes and, hence can detect smaller effects with larger power.

To provide more insights into neurocognitive sequelae of sexual abuse in adolescents, Porter and colleagues (Porter, Lawson, & Bigler, 2005) tested the hypothesis of group differences between sexually abused and matched control participants in domains of

intelligence and memory. When controlling for socioeconomic status, they found significant differences in verbal and performance IQ as well as for the indices of verbal memory, attention concentration, sequential recall, and free recall. However, when controlling for full scale IQ, there were no differences observable for memory measurements. To expand the literature, Barrera and colleagues (Barrera et al., 2013) compared the neuropsychological performance in children who have experienced sexual abuse with and without PTSD to participants without a history of CSA. Although assuming a poorer performance in the CSA with PTSD population compared to both control groups, they failed to verify this hypothesis and therefore, compared participants with sexual abuse experience to those without. They found effects in the Stroop task despite the absence of effects on all other measurements (TMT, CVLT, RCF, WCST). Using a logistic regression model containing all neuropsychological tests, CSA status was predictable; however, only the Stroop task made a significant contribution to the model. While these results may lead to the conclusion of EF being not affected by CSA, other studies report moderate negative correlations between domains of cognitive flexibility/set shifting, problem solving/planning, language skills, and sexual abuse (Kavanaugh et al., 2015). More specifically, association of impairments in language and memory with sexual abuse have been observed (De Bellis et al., 2013). Concomitant with this, Revington and colleagues (Revington, Martin, & Seedat, 2011) found significant correlations between physical and sexual abuse and neuropsychological performance reflecting the influence of abuse experiences on cognitive domains.

The Present Research

To sum up, inconsistencies are reported in the literature on neurocognitive dysfunction in adolescent PTSD which seem to be related to the presence of sexual abuse (Kavanaugh et al., 2016). In their review on the changes in neurodevelopment associated with CSA, De Bellis and colleagues (De Bellis et al., 2011) argued that – although any severe trauma can result in PTSD – trauma experiences of interpersonal origins (e.g. rape, abuse) may override resilience factors, increasing the risk of PTSD and its associated impairments in the victims. Further support for this assumption derives from prevalence studies reporting the highest conditional likelihood for developing PTSD after interpersonal and sexual trauma (Nemeroff et al., 2006; Perkonigg et al., 2000).

However, systematic research thereon is still scarce (Barrera et al., 2013) and it remains debatable to what extent sexual abuse and PTSD have distinct neuropsychological effects. Therefore, the present study was conducted to determine the effects of a PTSD

diagnosis and CSA experience on neurocognition. In the light of this, adolescents with a history of sexual abuse and a diagnosis of PTSD (PTSD+/CSA+) were compared to PTSD patients without a history of CSA (PTSD+/CSA-) as well as victims of CSA without PTSD (PTSD-/CSA+) and traumatized adolescents without PTSD diagnoses or a history of sexual abuse (PTSD-/CSA-) using a neuropsychological test battery to assess a broad domain of cognitive functioning including memory, attention, executive functioning, visuo-spatial abilities, and language. Owing to the impact of both PTSD and CSA, it was hypothesized that CSA and PTSD should both be associated with negative impacts on performance with sexually abused children with PTSD showing poorer neuropsychological performance than the other three groups. Apart from group differences, this study aimed to quantify the effects of PTSD symptoms and extent of CSA on cognitive functioning to test the prediction that greater severity of CSA and PTSD symptomatology would be related to poorer outcomes across all neuropsychological domains. Finally, we were interested in whether it is possible to predict CSA and PTSD scores based on neuropsychological scores.

Methods

Sample

Participants were recruited within the project "A comparison of Neuropsychological Functioning and Structural MRI in Traumatized Adolescents with and without PTSD" under the leadership of Soraya Seedat and Paul Carey. Using the Bathuthuzele Youth Stress Clinic, based at the MRC Unit for Anxiety and Stress Disorders, Department of Psychiatry, University of Stellenbosch (South Africa), a sample of 105 11- to 19-year olds were recruited between 2004 and 2010. The Bathuthuzele Youth Stress Clinic offers specialized services to youths affected by violence and extreme trauma. Participants were included if they had experienced at least one qualifying traumatic event, had at least 6 years of formal education and were proficient in Afrikaans and/or English. Participants could not participate if any of the following criteria were met: known mental retardation, use of sedative psychotropic medication, visual or auditory impairment, traumatic brain injury with loss of consciousness, major medical illness that precluded participation in diagnostic/neuropsychological assessment, or use of illicit substances.

Measurements

Diagnostic interviews and self-report questionnaires.

To determine the presence or absence of psychiatric diagnosis in general and PTSD in particular, a clinician-administered semi structured diagnostic interview was conducted. The *Kiddle-Schedule of Affective Disorders and Schizophrenia-Present and Lifetime version* (K-SADS-PL; Kaufman et al., 1997) assesses current and lifetime history of psychiatric disorders in children and adolescents based on DSM-IV diagnostic criteria. It further obtains severity ratings of symptomatology and covers depressive disorders, bipolar disorders, psychosis, panic disorder, separation anxiety disorder, avoidant disorder/social phobia, agoraphobia/specific disorders, overanxious/generalized anxiety, obsessive compulsive disorders, enuresis, encopresis, anorexia nervosa, bulimia nervosa, attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorders, tic disorder, cigarette use, alcohol and substance abuse, and PTSD. The K-SADS-PL has shown high reliability and validity (Kaufman et al., 1997).

The K-SADS-PL also provides a trauma checklist to assess if one of the following traumatic experiences has occurred to the participants: car accident, other accident, fire,

witness of a disaster, witness of a violent crime, victim of violent crime, confronted with traumatic news, witness to domestic violence, physical abuse, sexual abuse, or other events.

To assess exposure to non-PTSD life events during the past year, the *Life Events Questionnaire for Adolescents* (LEQ-A; Masten, Neemann, & Andenas, 1994) was used. It contains 51 items representing negative and positive life events (e.g. "My parents divorced" or "I had at least one outstanding personal achievement this past year") that do not qualify as traumatic events. Respondents were required to indicate ("yes" or "no") if the event has happened to them.

The *Childhood Trauma Questionnaire* (CTQ; Bernstein et al., 2003; Bernstein & Fink, 1998) was developed to assess the extent of different types of childhood abuse and neglect during childhood and adolescence. The retrospective self-report measure consists of 28 items that can be divided into five categories, namely emotional abuse (CEA "People in my family said hurtful or insulting things to me"), physical abuse (CPA, e.g. "Got hit so hard that I had to see a doctor or go to the hospital") and sexual abuse (CSA, e.g. "Someone molested me") as well as emotional (CEN, e.g. "People in my family looked out for each other" reversed item) and physical neglect (CPN, e.g. "I didn't have enough to eat"). Each of these subscales are represented by five items ranging from "never true" (1) to "very often true" (5) resulting in subscale scores ranging from 5 to 25. Proposed cut-off scores allow a distinction for each type of trauma as being "none (or minimal)", "low (to moderate)", "moderate (to severe)", or "severe (to extreme)". There are three additional items measuring tendencies to minimalize or deny abuse. The internal reliability as measured as Cronbach's alpha was 0.768 for the total score and ranged from 0.284 (CPN) to 0.896 (CSA) within the five abuse and neglect domains.

Current PTSD symptoms during the last month were evaluated using the *Childhood Post Traumatic Stress Disorder Checklist* (CPC; Amaya-Jackson et al., 1995 as cited in Boyes, Cluver, & Gardner, 2012; Frank-Schultz, Naidoo, Cloete, & Seedat, 2012). Based on the DSM-IV criteria, participants were asked to rate each of the 17 PTSD symptoms on a 4-point Likert scale with answers ranging from "not at all" (0) to "all of the time" (3). An example item is, "When something reminds you of what happened, do you get tense or upset?" The most upsetting event reported in the K-SADS-PL was used as index event. The 28 items are summarized to a total score. The questionnaire demonstrated high internal consistency in this study sample (Cronbach's alpha = 0.933).

The *Beck Depression Inventory* (BDI; Beck, Steer, & Brown, 1996) is a self-report questionnaire reflecting severity of depressive symptoms like avolition or sadness during the last two weeks. Calculations revealed a Cronbach's alpha = 0.884.

The *Connor-Davidson Resilience Scale* (CD-RISC; Connor & Davidson, 2003) is a 25-item self-report questionnaire used to measure resilience with reference to the past month. Each of the items (e.g. "Able to adopt to change") is rated on a 5-point Likert scale ranging from "rarely true" (0) to "true nearly all of the time" (4). Higher sum scores reflect greater resilience. The internal consistency was high (Cronbach's alpha = 0.903).

Neuropsychological assessments.

The *Senior South African Individual Scale-Revised* (SSAIS-R; van Eeden, 1991) was constructed as a measurement of general intelligence being usable for English- and Afrikaans-speaking white, coloured and Indian pupils (van Eeden & Visser, 1992) and consists of 12 subtests. These tests can be differentiated into the categories verbal and non-verbal. The verbal category consists of modules regarding vocabulary, comprehension, similarities, number problems, story memory, and memory for digits. Pattern completion, block designs, missing parts, form board, and Coding can be classified as non-verbal. Three subscales were administered: Memory for Digits, Block Designs, and Missing Parts. In the Memory for Digits subtest, the examiner reads out a series of digits (up to eight) and the participant is asked to repeat them either forwards (same sequence) or backwards (reverse sequence). This measures WM, auditory sequencing, and auditory attention (Cockcroft, 2013). The Block Design test requires the re-creation of a model demonstrated either on cards or concrete (a tester builds a model and leaves it on the table or tester demonstrates the items with the blocks). Therefore, four or nine plastic cubes are used. The fifteen items evaluate non-verbal problem solving, visuospatial analysis and synthesis, perceptual organisation, visuomotor coordination, and attention (Cockcroft, 2013). Twenty pictures are presented in the Missing Parts test and participants have to identify the essential part missing (e.g. sleeve of the dress). This determines the ability to distinguish between essential and non-essential information as well as the ability to understand the whole in relation to its parts, visual perception, and long-term visual memory (Cockcroft, 2013).

The *Rey Auditory Verbal Learning Test* (RAVLT; Rey, 1941) assesses auditory memory, immediate recall memory, repetitive verbal learning, influence of interference, and recognition memory. A list containing 15 nouns (list A, e.g. "coffee") is read to the participants and they are asked to recall as many words as possible. This procedure is repeated over five different trials (Trial 1-5) followed by 15 additional nouns (list B, e.g. "cloud") that

are presented to the participants. Again, participants are asked to recall them (Interference). Immediately after the recall of list B, participants shall repeat list A again (Trial 6) and second time 30 minutes later (Trial 7). Subsequent to this, a recognition trial with 30 words containing the 15 words from list A is applied.

During *Rey Oesterreith Complex Figure Test* (RCFT; Osterreith, 1944; Rey, 1941; Spreen & Strauss, 2006), participants are asked to copy the default figure with its 18 features as detailed as possible on a blank piece of paper. When copied correctly, the participant can receive two points for each feature resulting in 36 as a maximum number of points. Participants were given 2 points when the item is placed and reproduced correctly; 1 point when the item is reproduced incompletely, placed incorrectly, or presented some distortion; 0.5 point is attributed when the item was placed or reproduced poorly. A zero score is given when the item is absent or not recognized. After finishing the copy trial and without former annunciation, the participant shall reproduce the copy from memory on a new blank piece of paper (immediate recall), which is repeated 30 minutes later (delayed recall). Besides visual memory, the RCFT measures visuospatial construction.

To further evaluate memory functioning, the three subscales of the *Wechsler Memory Scale-Revised* (WMS-R; Wechsler, 1987) were used: Verbal Paired Associates, Visual Reproduction, and Logical Memory. After initial presentation of the eight word pairs (e.g. metal – iron, Crush – dark) the test requires participants to complement the pairs. Therefore, the first word is given. In contrast to the manual guidelines only the first three trials are administered and there was no delayed recall. The sum score of the three trails is supposed to reflect auditory learning as well as immediate memory. In the Logical Memory test, two stories are read to the participants. Besides the immediate re-narration (verbal memory), it includes further a delayed recall as well as yes-no-questions to measure recognition. To gain further insight into immediate and delayed visual memory, the Visual Reproduction subtest was used. In total, five patterns are presented for 10 seconds each and shall be reproduced from memory immediately afterwards. Long-term memory is operationalized by a delayed recall 30 minutes later as well as by a recognition task where participants have to choose the known pattern out of six patterns. In the current study, recognition trials were not administered, neither in the Logical Memory nor in the Visual Reproduction subtest.

In addition to memory, language and executive functioning was assessed. The *Controlled Word Association Test* (COWAT; Benton, 1969) instructs the participants to say as many words as possible, beginning with the first letter F within 60 seconds. This is followed by the same procedure for the first letters A and S. Besides this phonemic condition,

a semantic one can be applied where participants again have 60 seconds to name various animals. Taken together, the test is typically viewed as assessment of verbal fluency.

The *Trail Making Test* (TMT; Reitan, 1955) is an instrument to measure attention, motor speed and visuomotor tracking (TMT A) as well as cognitive flexibility and shifting (TMT B). The task for the participants is to connect encircled numbers in ascending order (i.e. 1-2-3-…; TMT A) or to join encircled numbers and encircled letters in ascending, alternating order (i.e. 1-A-2-B-3-C-…; TMT B) until completed while the experimenter stops time.

As a last measure of EF, especially abstract reasoning, problem solving, and cognitive flexibility, participants had to perform the *Wisconsin Card Sorting Test* (WCST; Heaton, Chalune, Talley, Kay, & Curtis, 1993). Mainly, the task requires sorting 128 cards to four category cards. The cards vary according to colour, number and geometric shape. The examiner creates sorting rules according to the three dimensions. When assigning a card to a category, the participant uses the provided feedback ("correct" or "incorrect"). After ten consecutive correct matches, the experimenter changes the sorting rule without informing of the switch. The number of trials in which participants persist with responding to a discarded category is measured as perseverative errors. Additional important parameters are the number of categories achieved as well as failure to maintain set.

Procedure

Schools, clinics, and nongovernmental organizations working with community development and trauma were informed about the study through recruitment visits, phone calls, emails, and the distribution of pamphlets and asked to refer willing participants who had experienced trauma with emotional and/or behavioural problems secondary to traumatic exposure. Referring organizations were asked to complete a short referral form indicating trauma exposure, current symptoms, duration of the problem, and the contact details of the potential participant's parent or guardian. Referred traumatized adolescents were telephonically contacted and screened for their potential eligibility to participate in this study. Those meeting inclusion and exclusion criteria were invited to attend an appointment at the clinic. When not eligible, alternative mental health resources, where participants could receive treatment, were suggested.

Participants aged 18 and older obtained written informed consent, whereas participants under 18 years provided informed assent and a parent or legal guardian provided informed consent. All participants were evaluated over three sessions. The first assessment comprised a diagnostic interview during which a trained clinical psychologist evaluated participants with

the K-SADS-PL followed by completion of self-report measures of trauma exposure and psychopathology. Efforts were made to minimize missing data by checking completed questionnaires by a member of the research team and participants were encouraged to complete all items. The second visit entailed neuropsychological tests administered by a trained rater blind for presence or absence of PTSD diagnosis and scoring followed standardized protocols. The neuropsychological assessments started with RAVLT (trials 1 to 5 as well as inference list and trial 6) and were followed by RCFT (copy and immediate recall condition). Afterwards, both subtests of COWAT and TMT were administered as well as the WMS Verbal Paired Associates. Subsequently, participants were asked to recall the words learnt during the five RAVLT trials as well as to distinguish them from new words (recognition trial) and to draw the RCFT (delayed recall) again. This was followed by the WMS-R Logical Memory and Visual Reproduction tasks. To induce a temporal delay between the two recall conditions, SSAIS-R tasks were used: Memory for digits (first forward condition), Block Design Test, and Missing Parts. After testing the delayed recall of Logical Memory and Visual Reproduction subtests, the last test administered was the WCST. As a whole, the neuropsychological assessment lasted two to three hours depending on how many breaks the test subject took. The third appointment was a structural MRI scan carried out at the Tygerberg Hospital in Cape Town. Further, samples of salivary cortisol were collected during the final assessment as well as a blood sample. Results of the latter two parts of the study are published elsewhere (Ahmed et al., 2012). Participants who were in need of treatment due to the K-SADS-PL results were referred as appropriate. All services were offered free of charge and participants were reimbursed for their travel expenses (40 Rand per study visit).

Design and Ethical Statement

The study followed a cross-sectional, correlational design with PTSD diagnosis/symptoms and experience/severity of childhood sexual abuse as independent variables and performance on neurocognitive tests as dependent variables. The assignment of participants to the groups was based on pre-existing characteristics and not matched based on demographic variables. Ethical approval to conduct the study was obtained from the Western Cape Department of Education as well as from the Health Research Ethics Committee at Stellenbosch University.

Statistical Analyses

Data were analysed using IBM SPSS Statistics 23 and R 3.3.2. The group categorisation of participants was based on the K-SADS-PL: participants with a full- or subsyndromal PTSD diagnosis were assigned to the PTSD+ group, whereas the remaining participants were categorized as PTSD-. Previous studies demonstrated that there is no difference between partial and full PTSD in terms of functional impairment (Carrion et al., 2002; Samuelson et al., 2010). To verify this assumption in the current sample, two-sample t-test on the outcomes of cognitive tests were performed.

Based on their report of experiencing sexual abuse within the diagnostic interview, participants were further classified according to their history of being sexually abused (CSA+ or CSA-) resulting in four groups: those who neither were sexually abused nor had PTSD symptomatology (CSA-/PTSD-), those who were sexually abused without PTSD symptomatology (CSA+/PTSD-), those who were not sexually abused but had PTSD symptomatology (CSA-/PTSD+), and those who were sexually abused and had PTSD symptomology (CSA+/PTSD+).

The resulting four groups were compared on background characteristics, trauma history related variables, and comorbid diagnoses using two-tailed *t*-tests or chi-square tests of association, depending on scaling of the variables. Performances on neuropsychological tests were analysed using a between-group ANOVA with PTSD diagnosis (PTSD+ vs PTSD-) and experience of sexual abuse (CSA+ vs CSA-) as between-subject factors, controlling for age, gender, age at index trauma, months between index trauma and assessment, chronic trauma experience in childhood as well as number of experienced traumatic events (sum of Trauma Checklist) and LEQ scores. Critical parameters for each test are displayed in table 1. Significant interaction effects were further pursued by appropriate post hoc tests that were corrected for multiple comparisons, if required. Critical *p*-values were set to $p < 0.05$. All reported *p*-values are two-tailed.

Further, relationship between neuropsychological performance and clinical characteristics (severity of PTSD and CSA, stressful life events, and experienced trauma) were examined using Spearman correlation coefficients. Due to directed hypothesis that higher scores in the questionnaires are associated with lower neurocognitive performance, all significance tests were one-sided and p was set at < 0.05. In order to identify the variables that were most predictive of CPC and CSA scores, all clinical, neuropsychological, and confounding variables were entered into a best subsets regression analysis. This method compares all

possible models using a specified set of predictors and displays the best-fitting models possible allowing for a choice of the optimal model.

Table 1. Parameters analysed

Test	Critical parameters	Comment	Domain
Rey Auditory Verbal Learning Test (RAVLT)			
	Trial 1	Number of items recalled	Attention
	Trial 6	Number of items recalled	Verbal memory
	Trial 7	Number of items recalled	Verbal memory
	Total Words	Σ Items recalled (Trial 1 to 5)	Verbal memory
	Words Learned	Trial 5 – Trial 1	Verbal memory
	Interference	(Trial 6 / Trial 5) * 100	Inference effects
	Recognition	True Positives – False Negatives	Verbal memory
Rey Oesterreith Complex Figure Test (RCFT)			
	Copy	Score in Copy Condition	Attention/visuospatial
	Immediate Recall	Score in immediate recall	Visual memory
	Delayed Recall	Score in delayed recall	Visual memory
Controlled Word Association Test (COWAT)			
	FAS	Words named	Verbal fluency
	Animals	Animals named	Verbal fluency
Trail-Making Test (TMT)			
	Trial A	Seconds to complete Trial A	Attention
	Trial B	Seconds to complete Trial B	Switching
Wechsler Memory Scale-Revised (WMS-R)			
	Paired Associates	Completed pairs	Verbal memory
	Logical Immediate	Recalled details immediate recall	Verbal memory
	Logical Delayed	Recalled details delayed recall	Verbal memory
	Visual Immediate	Correct patterns immediate recall	Visual memory
	Visual Delayed	Correct Patterns delayed recall	Visual memory
Senior South African Individual Scale-Revised (SSAIS-R)			
	Digit Span Forwards	Memory for Digits forwards	Working memory
	Digit Span Backwards	Memory for Digits backwards	Working memory
	Digit Span Total	Σ (Forwards + Backwards)	Working memory
	Block Design	Re-created models	Planning
	Missing Parts	Identified missing parts	Visual perception
Wisconsin Card Sorting Test (WCST)			
	Correct Trials	Number of correct trials	Executive functions
	Error Trials	Number of error trials	Executive functions
	Perseverative Errors	Number of perseverative errors	Shifting
	NCC	Number of categories completed	Problem solving
	TC1	Trials to complete category 1	Abstract thinking

Results

Study Participants

Within the study protocol, n = 131 participants were assessed. Due to different reasons, n = 26 were excluded: n = 10 participants did not meet the age range for adolescence (World Health Organization, 2011), n = 2 participants were excluded because of comorbid alcohol dependence, n = 13 had inconsistent data sets with regard to questionnaires and neuropsychological assessments, and n = 1 was excluded since the language of assessment was Xhosa. The final study sample analysed consisted of n = 105 adolescents aged between 11 and 19 (M = 15.42, SD = 1.98, range = [11.17, 18.83]), with 47 males and 58 females. According to the K-SADS-PL interview, 52 participants had a full or subsyndromal PTSD diagnosis (PTSD+) and 53 did not have PTSD (PTSD-), respectively. Participants also suffered from additional comorbid diagnoses including Major Depression (n = 21), Dysthymia (n = 4), Depressive Disorder Not Otherwise Specified (n = 2), Adjustment Disorder with Depressed Mood (n = 6), Panic Disorder (n = 3), Separation Anxiety Disorder (n = 5), Simple Phobia (n = 1), Social Phobia (n = 2), Overanxious Disorder (n = 1), General Anxiety Disorder (n =4), Acute Stress Disorder (n =5), Attention Deficit Disorder (n = 6), Conduct Disorder (n = 6), Oppositional Defiant Disorder (n = 5), Adjustment Disorder with Disturbed Conduct (n = 1), and Adjustment Disorder with Mixed Mood and Conduct (n = 2). Based on their report of experiencing sexual abuse, they were either classified as sexually abused (n = 34; CSA+) or as not sexually abused (n = 71; CSA-). There was no significant association between the experience of sexual abuse and PTSD diagnosis ($\chi^2(1) = 0.235$, p = 0.628).

Table 2. Sociodemographic sample characteristics (categorical data)

Measure	CSA-/ PTSD- (n = 37)	CSA+/ PTSD- (n = 16)	CSA- /PTSD+ (n = 34)	CSA+/ PTSD+ (n = 18)	χ^2	p
Gender					12.43	0.006**
Female	15	12	16	15		
Male	22	4	18	3		
Ethnicity					2.381	0.469
White	2	3	3	2		
Non-White	25	13	31	16		
Level of education					3.399	0.346
Grade 4-7	10	5	6	2		

Grade 8-12	26	10	28	16		
Language of Assessment					3.600	0.324
Afrikaans	14	6	12	11		
English	22	10	22	7		
Type of trauma as index event					96.250	0.000***
Car accident	1	1	2	0		
Other accident	0	0	0	0		
Fire	0	0	0	0		
Witness of disaster	0	0	0	0		
Witness of violent crime	2	1	9	0		
Victim of violent crime	5	0	12	0		
Confronted traumatic news	16	2	6	2		
Witness to domestic violence	7	0	5	0		
Physical abuse	5	3	0	2		
Sexual abuse	0	9	0	14		
Other	1	0	0	0		
Index trauma					0.492	0.921
Acute	29	13	27	13		
Chronic	8	3	7	5		
Chronic trauma experience in Childhood					1.377	0.711
Yes	9	4	10	7		
No	28	12	24	11		
Additional Diagnoses					9.509	0.023*
Yes	14	3	18	12		
No	23	13	16	6		
Depressive Diagnoses					13.621	0.004**
Yes	9	0	15	9		
No	28	16	19	9		

Note. $N = 105$, $p = p$-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, χ^2 = Chi Square value.

Table 2 summarizes categorical demographic characteristics for each group, whereas Table 3 displays interval scaled demographic and questionnaire data. The four groups differed significantly in age, gender, type of trauma as index event, additional and depressive diagnoses. Moreover, there were significant group effects in questionnaire sum scores of CTQ, CTQ-CSA subscale, CPC, and LEQ as well as trend in number of traumas experienced.

Table 3. Sociodemographic sample characteristics (interval data)

Measure	CSA-/ PTSD- ($n = 37$)	CSA+/ PTSD- ($n = 16$)	CSA-/ PTSD+ ($n = 34$)	CSA+/ PTSD+ ($n = 18$)	*F*	*p*-Value	post-hoc comparisons
Age					3.644	0.015*	1 < 3
	14.66 ±1.89	15.29 ±2.24	16.03 ±1.94	15.95 ±1.52			

Number of additional diagnoses					3.42	0.020*	*n.s.*
	0.49 ±0.80	0.31 ±0.70	0.91 ±1.00	1.11 ±1.13			
Age at index trauma					0.72	0.542	
	12.19 ±4.18	12.81 ±4.07	13.29 ±3.16	13.50 ±3.57			
Months between index trauma and assessment					0.151	0.929	
	27.43 ±32.93	24.06 ±28.90	24.29 ±34.08	21.78 ±20.40			
Number of experienced trauma (Checklist)					2.509	0.063^{+}	
	2.68 ±1.18	3.00 ±1.26	3.44 ±1.35	3.39 ±1.33			
LEQ					3.079	0.031*	1 < 2
	10.68 ±4.81	15.38 ±6.62	11.59 ±4.53	11.61 ±5.94			
CPC					16.04	0.000***	1 < 3,4
	22.62 ±14.93	31.50 ±18.73	36.09 ±14.16	51.56 ±11.40			2 < 4, 3 < 4
CTQ					3.755	0.013*	*n.s.*
	64.05 ±10.62	73.44 ±14.53	64.41 ±11.80	72.39 ±11.80			
CSA					12.57	0.000***	1 < 2, 4
	5.65 ±1.62	10.88 ±6.97	5.88 ±3.22	11.56 ±6.39			2 > 3, 3 < 4

Note. Data represents means ± standard deviation, 1 = CSA-/PTSD-, 2 = CSA+/PTSD-, 3 = CSA-/PTSD+, 4 = CSA+/PTSD+; LEQ = Life Events Questionnaire, CPC = Child PTSD Checklist, CTQ = Childhood Trauma Questionnaire, CSA = Childhood Sexual Abuse Subscale; $N = 105$, $p = p$-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, F = F-value, post-hoc comparisons bonferroni-corrected.

Group Comparisons

To answer the question whether there are significant group differences in the performances on neuropsychological tests, 2 x 2 ANOVAs with PTSD diagnosis (PTSD+ vs. PTSD-) and experience of sexual abuse (CSA+ vs. CSA-) as between-subject factors were conducted. Variables relevant for trauma exposure (LEQ score, number of experienced traumas, age at index trauma, and time between trauma and assessment) as well as age and gender were included as covariates in subsequent ANOVA analyses. The adjusted means and standard errors within each group are reported in Table 4. Both BDI ($n = 76$) and CD-RISC ($n = 63$) were not administered to all participants and thus, could not be included as control variables.

After defining linear ANOVA model for each dependent variable, data was explored to check whether the assumptions were met for calculating a factorial ANOVA. Therefore, each model was plotted and Shapiro-Wilk normality tests were performed to check for normal

distribution of errors within each of the four groups separately. Significant results ($p < 0.05$) indicate non-normally distributed errors in at least one of the groups and were found for: RAVLT trial 7 (CSA-/PTSD+), RAVLT words learned (CSA+/PTSD-), RAVLT recognition (all groups), RCFT copy (all except CSA+/PTSD-), TMT A (CSA+/PTSD-, CSA-/PTSD+), TMT B (CSA-/PTSD-), WMS-R paired associates (CSA-/PTSD+), WMS-R immediate logical memory (CSA-/PTSD+), WMS-R delayed logical memory (CSA+/PTSD-), WMS-R delayed visual reproduction (CSA-/PTSD-, CSA+/PTSD+), SSAIS-R digit span forwards (CSA-/PTSD+), SSAIS-R block design (CSA+/PTSD+), WCST correct trials (CSA-/PTSD-, CSA+/PTSD-), WCST perseverative errors (CSA-/PTSD+), and trials to complete first category (CSA-/PTSD-, CSA+/PTSD+). The assumption of variance homogeneity across groups was assessed using Levene's test leading to significant p-values < 0.05 for the dependent variables RAVLT Recognition and RCFT Copy.

Due to the fact that WCST and the WMS-R Visual Reproduction subscale were not part of the initial study protocol but added later, $n = 29$ participants were not tested on these measurements. Of the remaining $n = 76$ participants, $n = 23$ were CSA-/PTSD, $n = 13$ were CSA+/PTSD-, $n = 27$ were CSA-/PTSD+, and $n = 13$ were CSA+/PTSD+, respectively. There was no significant association between group membership and completeness of neuropsychological data ($\chi^2(3) = 3.403$, $p = 0.334$).

Post-hoc power calculations using the software G-Power 3.1.9.2 (Erdfelder, Faul, & Buchner, 1996; Faul, Erdfelder, Lang, & Buchner, 2007) were performed on both sample sizes to determine the power to detect effects. Following Cohen's classification (Cohen, 1992), an effect of $f = 0.40$ (partial $\eta^2 = 0.14$) is large, whereas $f = 0.25$ (partial $\eta^2 = 0.07$) represents a medium effect, and $f = 0.10$ (partial $\eta^2 = 0.01$) a small effect, respectively. The sample size of $n = 105$ [$n = 76$)] had a power (1-β) of 0.77 [0.57] to detect large effect. For medium effects, the power (1-β) was 0.322 [0.233], whereas small effects could only be found with a power (1-β) of 0.08 [0.07].

To ensure that adolescents with partial/subsyndromal PTSD were not significantly different from the adolescents with full syndromal PTSD, a series of t-tests were conducted comparing the two groups (full and partial PTSD) on all outcome measures as well as demographic characteristics (see appendix, Table 2 and 3). There were significant differences between the full and partial PTSD groups on age ($t = 4.146$, $p = 0.047$), age at index trauma ($t = 5.810$, $p = 0.020$), and CPC scores ($t = 9.878$, $p = 0.003$) as well as in level of education ($\chi^2(2) = 6.082$, $p = 0.014$ and acuteness of index trauma ($\chi^2(2) = 4.222$, $p = 0.039$. Regarding neuropsychological performance (see appendix, Table 4), there were significant differences in

TMT A (t = -3.118, p = 0.003) and TMT B (t = -2.213, p = 0.030) with the partial PTSD group showing a better performance.

Table 4. Adjusted means ± standard errors for performance on neuropsychological tests

Measure	CSA-/ PTSD- (n = 37, [a] n = 23)	CSA+/ PTSD- (n = 16, [a] n = 13)	CSA-/ PTSD+ (n = 34, [a] n = 34)	CSA+/ PTSD+ (n = 18, [a] n = 13)
Rey Auditory Verbal Learning Test (RAVLT)				
Trial 1	6.56 ±0.33	5.45 ±0.49	5.94 ±0.33	6.90 ±0.45
Trial 6	11.25 ±0.52	8.13 ±0.78	10.83 ±0.52	10.42 ±0.72
Trial 7	10.79 ±0.55	8.92 ±0.83	10.12 ±0.56	10.32 ±0.76
Total Words	51.32 ±1.63	45.91 ±2.45	47.29 ±1.65	50.48 ±2.27
Words Learned	5.96 ±0.41	5.32 ±0.62	5.69 ±0.42	4.94 ±0.57
Interference	89.34 ±2.95	73.83 ±4.43	92.56 ±2.99	88.59 ±4.10
Recognition	13.13 ±0.63	11.34 ±0.95	12.25 ±0.64	10.84 ±0.88
Rey Osterrieth Complex Figure Test (RCFT)				
Copy	33.59 ±0.66	33.32 ±1.00	33.85 ±0.67	30.67 ±0.92
Immediate	21.47 ±1.18	19.30 ±1.77	19.69 ±1.19	19.39 ±1.64
Delayed	21.45 ±1.21	19.51 ±1.82	19.74 ±1.22	19.50 ±1.68
Controlled Word Association Test (COWAT)				
Animals	14.77 ±0.63	13.59 ±0.94	13.14 ±0.64	13.14 ±0.87
FAS	25.19 ±1.50	24.68 ±2.26	21.61 ±1.52	20.70 ±2.09
Trial Making Test (TMT)				
Trial A	44.00 ±4.33	54.60 ±6.51	44.63 ±4.39	47.77 ±6.03
Trial B	98.44 ±8.21	104.51 ±12.36	96.18 ±8.33	123.91 ±11.44
Wechsler Memory Scale-Revised (WMS-R)				
Paired Associates	18.69 ±0.67	17.51 ±1.02	19.73 ±0.68	18.52 ±0.94
Logical Immediate	19.95 ±1.24	15.23 ±1.87	16.15 ±1.26	16.46 ±1.73
Logical Delayed	15.03 ±1.16	10.59 ±1.74	12.49 ±1.17	13.21 ±1.61
Visual Immediate [a]	32.62 ±1.17	28.84 ±1.54	30.67 ±1.03	31.44 ±1.49
Visual Delayed [a]	26.97 ±1.75	23.79 ±2.30	26.77 ±1.54	25.12 ±2.24
Senior South African Individual Scale-Revised (SSAIS-R)				
Digit Span F	8.69 ±0.41	8.14 ±0.61	8.90 ±0.41	8.09 ±0.56
Digit Span B	4.74 ±0.31	4.59 ±0.47	3.91 ±0.32	4.07 ±0.44
Digit Span T	13.44 ±13.44	12.73 ±0.89	12.81 ±0.60	12.15 ±0.82
Block Design	19.64 ±1.35	18.70 ±2.03	20.81 ±1.37	19.26 ±1.88
Missing Parts	14.60 ±0.64	13.96 ±0.96	13.85 ±0.65	13.46 ±0.89
Wisconsin Card Sorting Test (WCST)				
Correct Trials [a]	74.87 ±2.76	68.89 ±3.64	69.10 ±2.43	74.89 ±74.89
Error Trials [a]	41.49 ±4.63	52.24 ±6.11	42.83 ±4.08	43.24 ±5.92
Perseverative Error [a]	19.98 ±3.51	29.36 ±4.63	23.53 ±3.10	21.49 ±4.49
NCC [a]	4.97 ±0.33	4.34 ±0.44	4.52 ±0.30	4.63 ±0.43
TC1 [a]	17.12 ±4.44	13.95 ±5.86	20.47 ±3.92	20.01 ±5.68

Note. N = 105, [a] N = 76 (not administered to all participants), F = Forward, B = Backwards, T = Total, NCC = Number of categories completed, TC1 = Trials to complete category 1

Rey Auditory Verbal Learning Test (RAVLT).

A between subject ANOVA on words remembered in RAVLT Trial 1 revealed a trend for an effect of sexual abuse ($F(10, 94) = 3.423$, $p = 0.067$) showing that participants with CSA recalled fewer words than those without in the absence of an effect of PTSD diagnosis ($F(10, 94) = 1.737$, $p = 0.191$). As illustrated in Figure 1, there was a significant interaction effect of CSA and PTSD ($F(10, 94) = 6.708$, $p = 0.011$, partial $\eta^2 = 0.067$). This interaction effect showed the following pattern: if participants did not experience CSA (CSA-), the scores in the PTSD+ were lower than in the PTSD-. The inverted pattern occurred for sexually abused participants: the scores in the PTSD- were lower than in PTSD+. However, using Tukey's Honest Significance Difference method, there were no significant differences between the groups after adjustment for multiple comparisons (all $p \geq 0.130$). The ANOVA conducted on the performance on RAVLT Trial 6 (Figure 1) showed a non-significant effect of PTSD ($F(10, 94) = 0.313$, $p = 0.577$). There was a significant main effect of CSA ($F(10, 94) = 10.679$, $p = 0.002$, partial $\eta^2 = 0.10$): participants who experienced sexual abuse showed lower scores. This main effect, however, was qualified by a significant interaction between PTSD and CSA ($F(10, 94) = 4.561$, $p = 0.036$, partial $\eta^2 = 0.046$). Post-hoc procedures revealed that this interaction effect was driven by significant differences between CSA+/PTSD- and CSA-/PTSD- as well as between CSA+/PTSD- and CSA+/PTSD+ with CSA+/PTSD- performing worse in both cases. The ANOVA conducted on performance in Trial 7 (Figure 2) showed a trend for a significant main effect of CSA ($F(10, 94) = 3.413$, $p = 0.068$). There was no significant main effect for PTSD diagnosis ($F(10, 94) = 0.698$, $p = 0.406$) nor an interaction effect between PTSD and CSA ($F(10, 94) = 2.357$, $p = 0.128$). However, descriptive data showed that although PTSD was associated with performance decreases in CSA-, the opposite pattern emerged in CSA+ were PTSD+ was associated with higher performance than PTSD+.

Regarding the number of words learned in trial 1 to trial 5 (Total Words, Figure 1), analyses revealed main effects on trend level for both PTSD ($F(10, 94) = 2.859$, $p = 0.094$) and CSA ($F(10, 94) = 3.225$, $p = 0.076$). Lower scores were observed in patients with PTSD diagnosis and experience of CSA, respectively. Additionally, there was a significant interaction effect ($F(10, 94) = 4.587$, $p = 0.035$, partial $\eta^2 = 0.047$), but post-hoc tests showed no significant group differences after p value adjustment for multiple comparisons (all $p \geq 0.279$). On a descriptive level, there were lower scores within the PTSD+ compared to PTSD- when the participant did not experience CSA. However, the pattern was the opposite in the CSA+ group: lower scores in PTSD- compared to PTSD+. Although participants with PTSD

learned fewer words than those without PTSD and although participants with CSA performed worse than participants without CSA, the ANOVA conducted on Words Learned showed that there was no effect of PTSD ($F(10, 94) = 0.196$, $p = 0.659$) or CSA ($F(10, 94) = 0.703$, $p = 0.404$) or their interaction ($F(10, 94) = 0.013$, $p = 0.0.911$). When looking at Interference score (i.e. performance on Trial 6 in relation to Trial 5, Figure 1), there was a significant main effect of sexual abuse with sexually abused participants performing worse ($F(10, 94) = 8.128$, $p = 0.005$, partial $\eta^2 = 0.080$) in the absence of a main effect of PTSD ($F(10, 94) = 0.561$, $p = 0.456$) and an interaction effect ($F(10, 94) = 2.535$, $p = 0.115$). There was no main effect of neither PTSD ($F(10, 94) = 0.909$, $p = 0.343$), nor CSA ($F(10, 94) = 2.373$, $p = 0.127$) nor an interaction effect ($F(10, 94) = 0.061$, $p = 0.805$) found when analysing recognition performance, but descriptive data revealed lower scores in PTSD+ than in PTSD-. The same is true for CSA: experience of CSA decreased the performance.

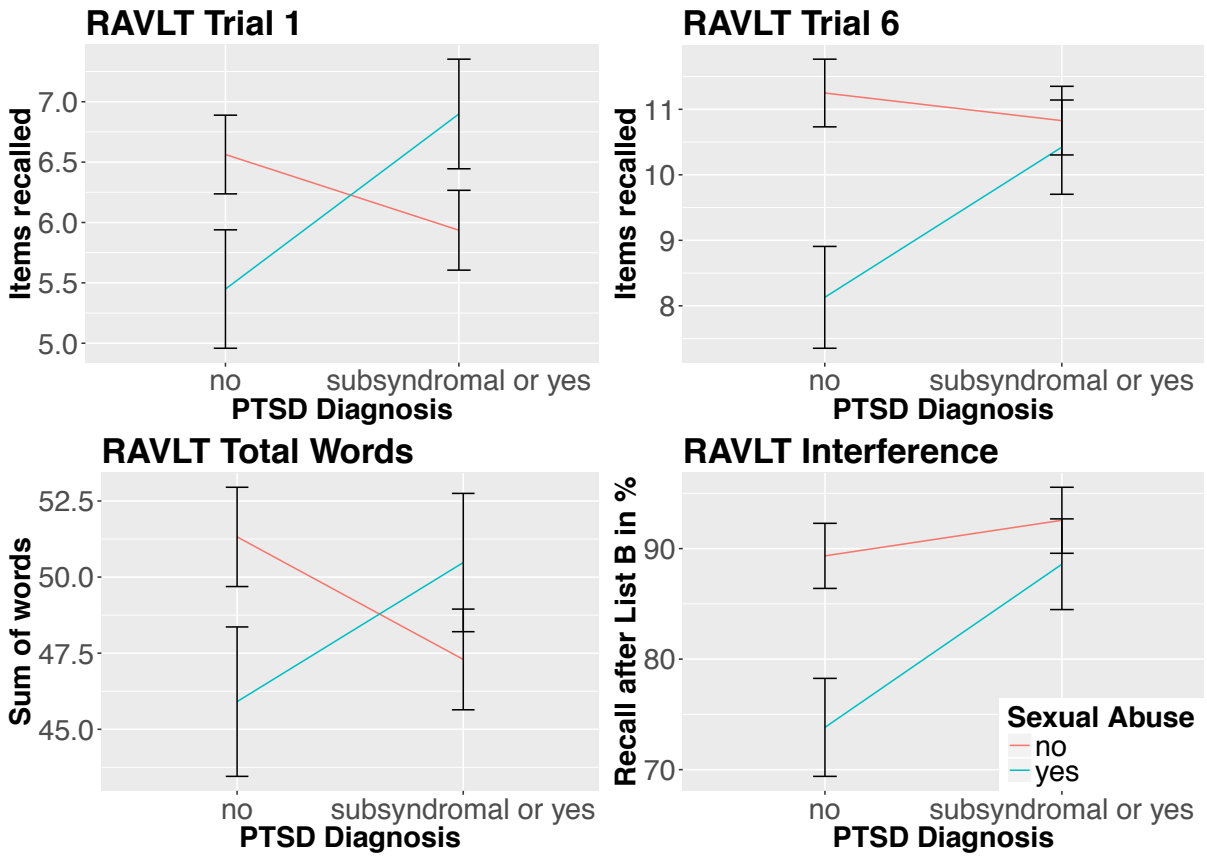

Figure 1. Significant interaction effects found in Rey Auditory Learning Test (RAVLT). Error bars represent standard errors

Rey Oesterrieth Complex Figure Test (RCFT).

There were no main effects of PTSD ($F(10, 94) = 0.071$, $p = 0.791$) or CSA ($F(10, 94) = 0.052$, $p = 0.820$) on the score in the RCFT copy condition. However, analyses revealed a trend for an interaction effect between CSA and PTSD ($F(10, 94) = 3.190$, $p = 0.077$) with

lower scores in the CSA+/PTSD+ group (Figure 2). Participants within the CSA+ group showed diminished performance in recall conditions compared to those in the CSA- group. Diagnosis of PTSD affected the performance in the CSA- group negatively, but not in the CSA group. However, none of the effects reached significance for immediate or delayed recall. (all $p \geq 0.301$).

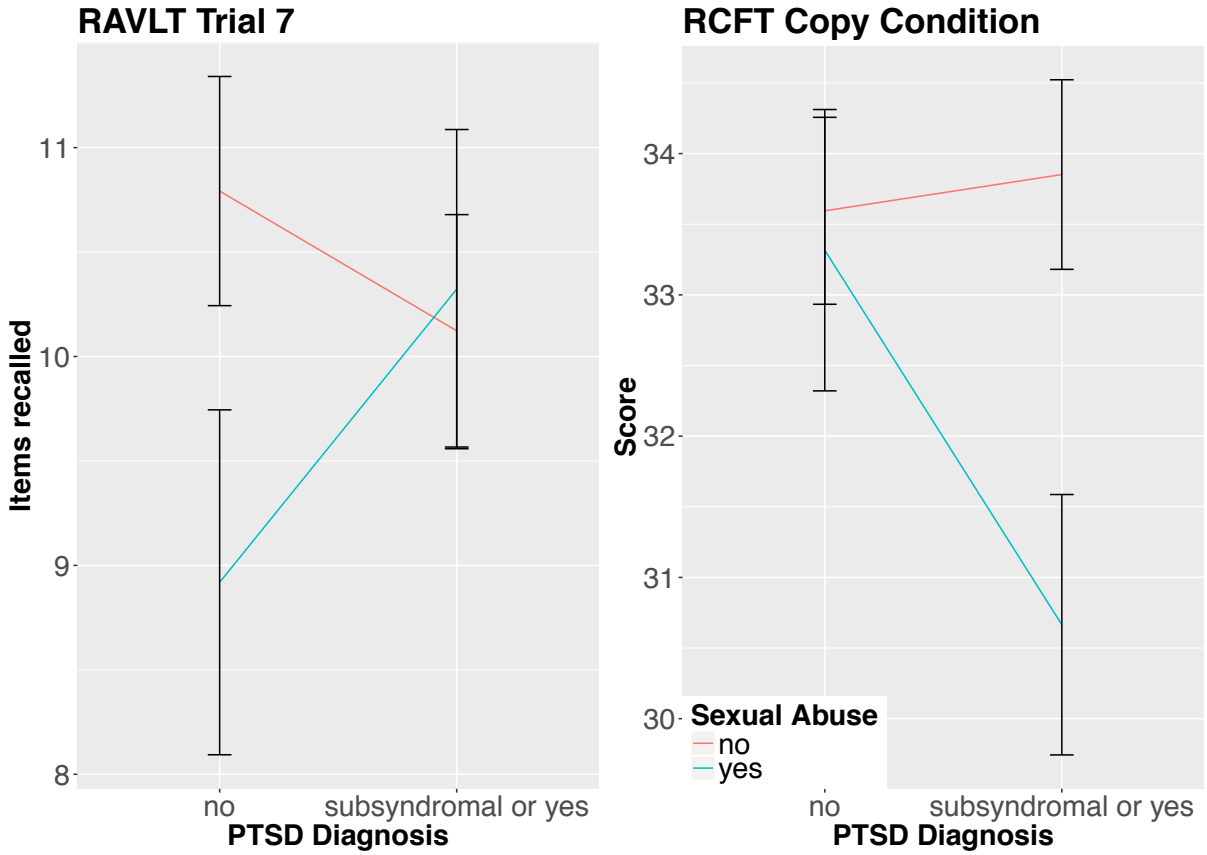

Figure 2. Trend effects observed in Rey Auditory Verbal Learning Test (RAVLT) and Rey Oesterrieth Complex Figure Test (RCFT). Error bars represent standard errors.

Controlled Word Association Test and Trial Making Test.

The ANOVA on words mentioned FAS condition revealed no significant effects (all $p \geq .106$) although scores were lower in the PTSD+ group compared to PTSD- and in CSA+ compared to CSA-. In the Animals condition, data indicated a trend for a significant main effect of PTSD ($F(10, 94) = 3.181$, $p = 0.078$) with lower scores within the PTSD group (Figure 4). There was no main effect of CSA ($F(10, 94) = 1.040$, $p = 0.310$) nor an interaction effect ($F(10, 94) = 0.591$, $p = 0.444$), but participants in the CSA+/PTSD- group named fewer animals than in the CSA-/PTSD- group.

Further, there were no effects observable when analysing the reaction times in TMT Trial A and B (all $p \geq 0.188$), although participants who experienced CSA had higher reaction times than those who did not. To further examine whether the observed differences between

full and partial PTSD in TMT scores had an effect on the results, a 2 x 2 ANOVA on TMT A and TMT B was conducted using PTSD (full syndromal vs. subsyndromal and no diagnosis) and CSA as between-group factors, controlling for the confounding variables. Results revealed that there was neither a main nor an interaction effect for both TMT A and TMT B observable (all $p \geq 0.129$).

Wechsler Memory Scale-Revised.

The between-subject ANOVA revealed no main or interaction effects on the scores in the Paired Associate subtest (all $p \geq 0.295$). However, the scores within the task were lower within the CSA+ participants compared to CSA-. PTSD+ was associated with higher scores compared to PTSD-.

In the immediate recall condition of the Logical Memory subtest (Figure 3), however, there was a significant effect of PTSD ($F(10, 94) = 4.383$, $p = 0.039$, partial $\eta^2 = 0.045$) as well as of CSA ($F(10, 94) = 4.211$, $p = 0.043$, partial $\eta^2 = 0.043$). As expected, the PTSD+ had lower scores than the PTSD-. In a similar way, sexually abused participants performed worse. The interaction effect, however, was non-significant ($F(10, 94) = 2.706$, $p = 0.103$). In the delayed recall condition, there was no main effect of PTSD ($F(10, 94) = 2.265$, $p = 0.136$), but a main effect of CSA ($F(10, 94) = 4.318$, $p = 0.040$, partial $\eta^2 = 0.044$) with lower score in CSA+ group. Further, the interaction effect was significant on trend level ($F(10, 94) = 3.284$, $p = 0.072$). Scores in both immediate and delayed recall condition followed the pattern of PTSD associated decrease in CSA-, but a PTSD associated increase in CSA+.

Analyses of the Visual Reproduction subtest immediate condition showed no main effect of PTSD ($F(10, 94) = 1.469$, $p = 0.230$), but a trend ($F(10, 65) = 3.591$, $p = 0.063$) for a significant main effect of CSA (lower scores in CSA+) as well as a trend for an interaction effect ($F(10, 65) = 2.856$, $p = 0.096$). While there was lower performance associated with PTSD diagnosis in CSA-, the performance was better in PTSD patients in CSA+ compared to those without PTSD symptomatology. Similarly, the descriptive mean scores in the delayed recall condition did not differ in terms of PTSD diagnosis when looking at CSA-, but if participants experienced CSA and had PTSD, their performance was better compared to those without PTSD. However, no effects in the delayed recall condition reached significance (all $p \geq 0.290$).

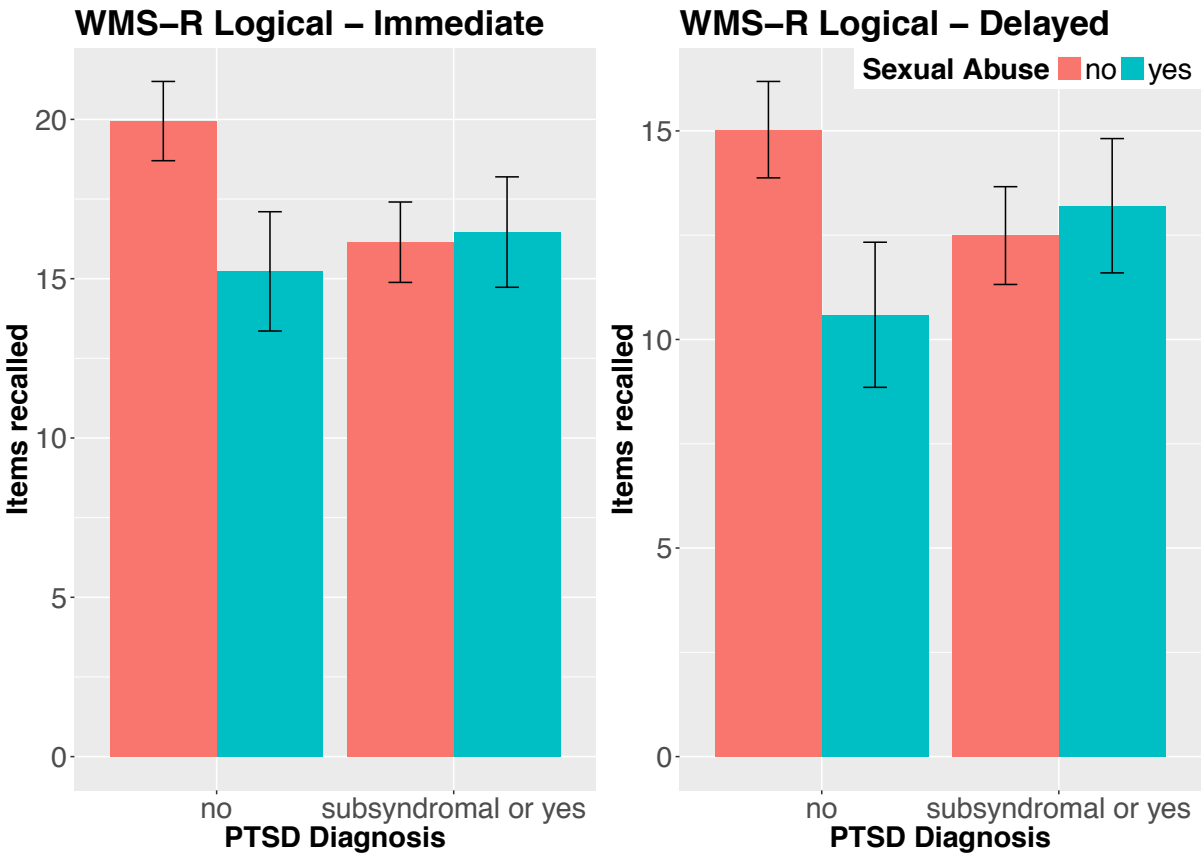

Figure 3. Main Effects found in Logical Memory Subtest of Wechsler Memory Scale-Revised (WMS-R). Error bars represent standard errors.

Senior South African Individual Scale-Revised.

Looking at the descriptive statistics of the Memory of Digits subtest revealed lower digit span forwards and total scores if participants experienced CSA. The between-subject ANOVA on Memory for Digits did not yield to significant main and interaction effects when regarding digit span forward (all $p \geq 0.462$) and digit span total (all > 0.466). There was, however, a trend for a significant main effect of PTSD on digit span backwards ($F(10, 94) = 3.364$, $p = 0.070$) with lower scores in PTSD+ group (Figure 4). Apart from that, there was no significant main effect of CSA ($F(10, 94) = 0.0745$, $p = 0.785$) nor an interaction effect between the two factors ($F(10, 94) = 0.171$, $p = 0.680$). Participants with PTSD had higher scores in the Block Design subtest, whereas they had lower scores in the Missing Parts subtest. While there was decreased performance in CSA+ compared to CSA- in the adjusted means, however, no significant main or interaction effects emerged (all $p \geq 0.419$) when analysing the Block Design and Missing Parts subtest.

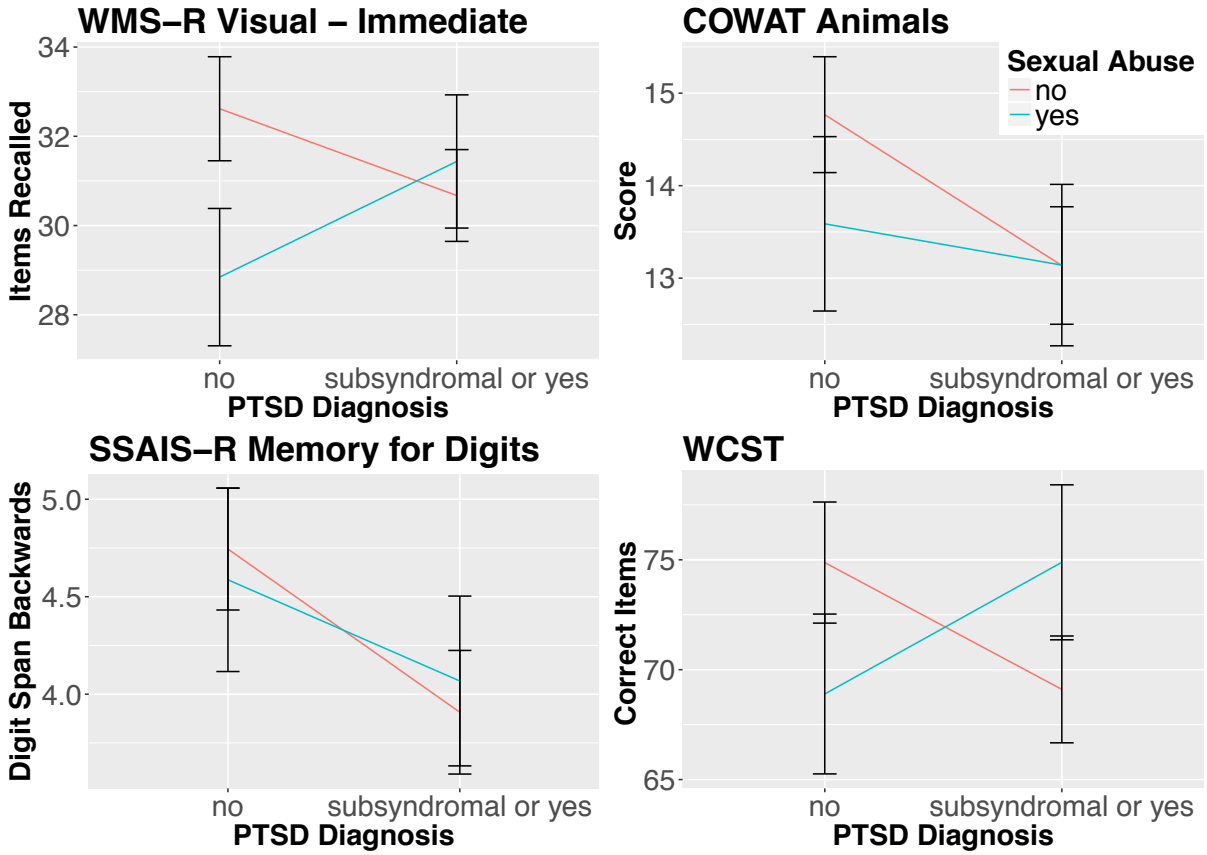

Figure 4. Trend effects observed in Wechsler Memory Scale-Revised (WMS-R), Controlled Word Association Test (COWAT), Senior South Afircan Individual Scale-Revised (SSAIS-R), and Wisconsin Card Sorting Test (WCST). Error bars represent standard errors.

Wisconsin Card Sorting Test.

Adjusted means for number of correct trials within the WCST were lower in those participants who did were not sexually abused but had PTSD compared to those who did not experience CSA but did not have PTSD symptomatology. The inverse pattern was observed in those participants who were sexually abused: their score were lower if they had no PTSD compared to those who had PTSD. Similar descriptive patterns were found with regard to number of errors, perseverative errors, and categories completed. On the other hand, sexually abused participants needed fewer trials to complete the first category compared to those who did not experience CSA, while diagnosis of PTSD was associated with an increased number of trials.

Between-subject ANOVAs carried out on the scores of the WCST yielded the following results. There were no main effects of neither PTSD ($F(10, 65) = 2.318$, $p = 0.133$) nor CSA ($F(10, 65) = 1.1615$, $p = 0.208$) on the number of correct trials, but data showed a trend (Figure 4) for a significant interaction effect ($F(10, 65) = 3.439$, $p = 0.068$). However, there were no additional main or interaction effects on the outcomes number of errors (all $p \geq$

0.179), preservative errors (all $p \geq 0.122$), number of trials to complete the first category (TC1, $p \geq 0.584$) or number of categories completed (NCC, all $p \geq 0.277$).

Correlations

To check whether there is a relationship between neuropsychological performance and scores in CPC and CTQ-CSA subscale as well as with the number of life and traumatic events, correlational analyses were conducted. Results of Shapiro-Wilk normality tests indicated a violation of assumption of normality on CPC sum scores ($W = 0.974$, $p = 0.040$), CTQ-CSA sum scores ($W = 0.576$, $p < 0.001$), LEQ sum scores ($W = 0.958$ $p = 0.002$), and sum scores of trauma checklist ($W = 0.932$, $p < 0.001$), respectively. Therefore, the non-parametric Spearman's correlation coefficient was used. As indicated in Table 5, there were significant correlations between CPC and the performance in the Animals subtest, the TMT and the Logical Memory subscale. Regarding CSA, a significant correlation with number of trials to complete the first category was observed. However, due to multiple testing the critical p-value was bonferroni-adjusted to $\alpha = 0.05/29 = 0.0017$ and no significant results remained.

Table 5. Correlations between neuropsychological tests and questionnaire sum scores

	CPC		CSA		LEQ		TraumaCL	
Measure	Rho	*p*	Rho	*p*	Rho	*p*	Rho	*p*
Rey Auditory Verbal Learning Test (RAVLT, *n* = 105)								
Trial 1	-0.037	0.355	0.125	0.898	0.107	0.861	0.025	0.598
Trial 6	0.040	0.658	0.020	0.580	0.111	0.871	0.018	0.574
Trial 7	0.047	0.683	0.106	0.858	0.144	0.929	0.194	0.977
Total Words	0.044	0.674	0.181	0.967	0.144	0.929	0.110	0.868
Words Learned	0.022	0.590	0.075	0.776	-0.005	0.478	0.126	0.901
Interference	0.080	0.790	-0.063	0.261	0.133	0.912	-0.046	0.320
Recognition	-0.044	0.327	0.007	0.530	0.132	0.911	0.044	0.672
Rey Osterrieth Complex Figure Test (RCFT, *n* = 105)								
Copy	0.072	0.767	0.006	0.524	-0.145	0.070^{+}	0.071	0.765
Immediate Recall	0.041	0.661	0.040	0.656	0.027	0.607	0.169	0.957
Delayed Recall	0.038	0.651	0.048	0.687	0.127	0.902	0.148	0.934
Controlled Word Association Test (COWAT, *n* = 105)								
FAS	-0.052	0.300	0.062	0.735	0.163	0.952	0.257	0.996
Animals	-0.172	0.039*	-0.011	0.455	0.123	0.894	0.129	0.905
Trial Making Test (TMT, *n* = 105)								
Trial A	0.273	0.002**	0.155	0.058^{+}	-0.020	0.581	-0.092	0.825
Trial B	0.169	0.042*	0.151	0.063^{+}	-0.050	0.693	0.012	0.450
Wechsler Memory Scale-Revised (WMS-R, [a] *n* = 105, [b] *n* = 76)								
Paired Associates [a]	0.047	0.683	-0.040	0.344	0.029	0.616	0.096	0.835
Logical Immediate [a]	-0.174	0.038*	-0.039	0.345	0.018	0.573	0.065	0.745

Logical Delayed [a]	-0.198	0.021*	-0.121	0.109	-0.010	0.458	0.068	0.754
Visual Immediate [b]	-0.084	0.234	-0.086	0.230	0.138	0.883	0.088	0.774
Visual Delayed [b]	0.030	0.600	-0.074	0.262	0.172	0.931	0.247	0.984
Senior South African Individual Scale-Revised (SSAIS-R, n = 105)								
Digit Span F	-0.086	0.191	-0.079	0.212	-0.072	0.232	0.020	0.580
Digit Span B	-0.072	0.234	0.026	0.602	0.043	0.667	0.053	0.705
Digit Span T	-0.103	0.148	-0.057	0.282	-0.027	0.393	0.058	0.720
Block Design	0.079	0.790	-0.012	0.450	0.038	0.649	0.094	0.829
Missing Parts	-0.004	0.483	0.019	0.577	0.009	0.535	0.174	0.962
Wisconsin Card Sorting Test (WCST, n = 76)								
Correct Trials	0.116	0.842	0.091	0.782	0.065	0.713	0.272	0.991
Error Trials	-0.093	0.789	0.028	0.405	0.033	0.388	-0.262	0.989
Perseverative Errors	-0.079	0.750	-0.016	0.555	0.073	0.266	-0.253	0.986
NCC	0.090	0.780	0.031	0.606	0.090	0.781	0.295	0.995
TC1	0.126	0.139	0.218	0.029*	0.066	0.286	-0.024	0.583

Note. Rho = Spearman's ρ (correlation coefficient), CPC = Child PTSD Checklist, CSA = Childhood Sexual Abuse subscale, LEQ = Life Event Questionnaire, TraumaCL = K-SADS-PL Trauma Checklist, F = Forward, B = Backwards, T = Total, NCC = Number of categories completed, TC1 = Trials to complete category 1, p = p-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***

Regression

All clinical and confounding variables (age, gender, age at index trauma, months between trauma and assessment, chronic trauma experience in childhood, LEQ score, Trauma Checklist sum score, number of additional diagnoses and presence of a depressive diagnosis) as well as all neuropsychological scores were included in best subsets regression models to determine those that are consistently predictive for CPC and CTQ-CSA score. Due to linear dependencies with digit span forward and digit span backwards, the total digit span was not included in the model.

The model for predicting the CSA subscale of CTQ contained 37 regressors. Due to missing data, n = 29 observations were deleted, resulting in n = 76 participants included in the analysis. This model could account for 49% of variance observed within the data, but did not reach significance (R^2= 0.491, F(37,38) = 0.9921, p = 0.509). The best subset procedure (see appendix, Figure 1) revealed the highest values for R^2 when the 13 predictors presented in Table 6 were included. This model was significant and explained approximately 40% of variance (R^2= 0.410, adjusted R^2= 0.287, F(13,62) = 3.317, p = 0.001). Further, the use of all regressors could not explain more variance (F(24,38) = 0.253, p = 0.999).

This final model was used to check whether the assumptions for computing a multiple regression were met. After plotting the model (see appendix, Figure 2), the Shapiro-Wilk normality test (W = 0.896, $p < 0.001$) verified non-normal distribution of errors within the

model. To test for the assumption of no multicollinearity, VIF and tolerance statistics were used. The smallest tolerance value was 0.211 (M = 0.626) and the highest VIF value observed was 4.486 (M = 1.941). To assess the assumption of independence, the Durbin-Watson test indicated that the errors are independent (DW = 1.9597, p = 0.396).

Table 6. Best subsets regression results

	β	SE	B	T	*p*-Value	
			Model CSA			
(Intercept)	16.411	7.342		2.235	0.029	*
Age	0.433	0.278	0.175	1.557	0.125	
Gender	2.517	1.009	0.263	2.495	0.015	*
Depressive Diagnoses	-2.844	1.150	-0.267	-2.475	0.016	*
RCFT Delayed	0.173	0.085	0.251	2.040	0.046	*
FAS	-0.135	0.062	-0.248	-2.186	0.033	*
TMT A	0.048	0.021	0.270	2.346	0.022	*
Logical Delayed	0.102	0.084	0.136	1.217	0.228	
Visual Delayed	-0.243	0.079	-0.377	-3.069	0.003	**
Digit Span Backwards	0.517	0.326	0.183	-3.069	0.118	
Block Design	0.111	0.089	0.168	1.254	0.215	
Correct Trials WCST	0.086	0.047	0.237	1.832	0.072	+
Error Trials WCST	0.089	0.046	0.401	1.941	0.057	+
NCC WCST	0.921	0.644	0.303	1.429	0.158	
			Model CPC			
(Intercept)	-34.169	23.086		-1.480	0.144	
Age	2.184	1.143	0.237	1.911	0.061	+
Gender	8.901	3.764	0.250	2.365	0.021	*
Age at index trauma	0.786	0.786	0.157	1.311	0.194	
LEQ	0.784	0.328	0.237	2.393	0.020	*
Additional Diagnoses	8.334	3.666	0.233	2.273	0.026	*
TMT A	0.077	0.072	0.115	1.059	0.293	
Logical Immediate	-0.517	0.287	-0.189	-1.808	0.075	+
Visual Immediate	-0.641	0.470	-0.182	-1.363	0.178	
Visual Delayed	0.374	0.314	0.157	1.192	0.238	
Correct Trials WCST	0.259	0.156	0.191	1.665	0.101	
TC1 WCST	0.167	0.105	0.187	1.597	0.115	

Note. N = 76, β = unstandardized regression coefficient, SE = unstandardized standard error, B = standardized regression coefficient, T = t-value, p = p-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, CPC = Child PTSD Checklist, CSA = Childhood Sexual Abuse subscale, LEQ = Life Event Questionnaire, TraumaCL = K-SADS-PL Trauma Checklist, RCFT = Rey Oesterrieth Complex Figure Test, TMT = Trial Making Test, WCST = Wisconsin Card Sorting Test, NCC = Number of categories completed, TC1 = Trials to complete category 1

Following the same procedure for predicting CPC scores, the initial models contained 37 predictors and could explain nearly 50% of variance in the dataset (R^2= 0.497, F(37,38) = 1.016, p = 0.480). The final model, resulting from best subset regression (see appendix, Figure 3), retrained 11 predictors as displayed in Table 6 and could account for a significant proportion of variance in CPC scores (R^2= 0.412, adjusted R^2= 0.311, F(11,64) = 4.075, p = 0.0001). There was no significant change in variance explained compared to using all predictors (F(26,38) = 0.249, p = 0.999).

Checking the assumptions regarding the distribution of errors within the model, neither the plot (see appendix, Figure 4) nor the result of Shapiro-Wilk normality tests (W = 0.985, p = 0.521) indicated a violation of the assumptions of normally distributed errors. Further, the Durbin-Watson tests showed that the errors are independent (DW = 1.9597, p = 0.396). Finally, multicollinearity was objected by looking at lowest tolerance (0.517, M = 1.442) and highest VIF value (1.932, M = 0.719).

Explorative Analyses

To evaluate the influence of comorbid diagnoses on the results, the best subset regression was repeated using only those participants without any diagnoses in addition to PTSD (n = 58). The starting model was therefore reduced and contained all neuropsychological test scores except total digit span and clinical/confounding variables (age, gender, age at index trauma, months between trauma and assessment, chronic trauma experience in childhood, LEQ score, and Trauma Checklist sum score). Due to missing data, n = 15 observations were deleted, resulting in n = 43 included in the explorative analysis.

Using all 35 predictors, the model (R^2= 0.754, F(35,7) = 0.6132, p = 0.841) could explain approximately 75% of the variances in CSA subscale scores. The final model contained 13 regressors and is shown in Table 7. The overall model was significant and could explain 66% of variance (R^2= 0.668, adjusted R^2= 0.519, F(13,29) = 4.075, p < 0.001). All predictors could not explain more variance than a model using 13 predictors (F(22,4) = 0.112, p = 0.999).

All predictors could take account for 87% of variance in CPC scores observed, but the model did not reach significance (R^2= 0.878, F(35,7) = 1.442, p = 0.323). After the best subset method, 13 predictors were retained in the model (Table 7) explaining 76 % of variance (R^2= 0.767, adjusted R^2= 0.663, F(13,29) = 7.35, p < 0.001). Likewise to the CSA model, the final model could explain as much variance as the model using all predictors (F(22,4) = 0.290, p = 0.988).

Table 7. Explorative best subsets regression results

	β	SE	B	T	*p*-Value	
	Model CSA					
(Intercept)	-36.068	12.389		-2.911	0.007	**
Gender	2.896	1.426	0.272	2.045	0.050	+
Months lapsed	-0.033	0.023	-0.200	-1.416	0.167	
TraumaCL	-1.478	0.713	-0.352	-2.070	0.047	*
RCFT Copy	0.591	0.277	0.302	2.131	0.042	*
RCFT Immediate	-0.847	0.313	-1.163	-2.706	0.011	*
RCFT Delayed	0.949	0.314	1.274	3.021	0.005	**
FAS	0.185	0.094	0.316	1.957	0.060	+
TMT B	0.059	0.015	0.589	3.950	0.000	***
Logical Delayed	0.459	0.113	0.558	4.049	0.000	***
Digit Span Backwards	-0.606	0.566	-0.157	-1.071	0.293	
Perseverative Errors	0.220	0.072	0.769	3.060	0.005	**
NCC WCST	1.626	0.800	0.525	2.033	0.051	+
TC1 WCST	-0.072	0.041	-0.338	-1.756	0.090	+
	Model CPC					
(Intercept)	-97.381	25.544		-3.812	0.001	**
Gender	4.970	3.6154	0.143	1.275	0.179	
Age at index trauma	2.955	0.924	0.600	3.197	0.003	**
Months lapsed	0.281	0.110	0.525	2.560	0.016	*
LEQ	2.382	0.365	0.713	6.524	0.000	***
TraumaCL	-2.778	1.907	-0.203	-1.457	0.156	
RAVLT Recognition	-1.346	0.631	-0.262	-2.135	0.041	*
RCFT	1.197	0.653	0.187	1.834	0.077	+
RCFT Immediate	2.239	0.794	0.940	2.820	0.009	**
RCFT Delayed	-2.2387	0.775	-0.933	-2.931	0.007	**
FAS	-0.289	0.235	-0.151	-1.230	0.229	
Logical Delayed	-0.935	0.293	-0.348	-3.188	0.003	**
Correct Trials WCST	0.636	0.1821	0.526	3.494	0.002	**
Error Trials WCST	0.222	0.1194	0.286	1.862	0.073	+

Note. $N = 43$, β = unstandardized regression coefficient, SE = unstandardized standard error, B = standardized regression coefficient, T = *t*-value, *p* = *p*-value, $p < 0.10$+, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, CPC = Child PTSD Checklist, CSA = Childhood Sexual Abuse subscale, LEQ = Life Event Questionnaire, TraumaCL = K-SADS-PL Trauma Checklist, RAVLT = Rey Auditory Verbal Learning Test, RCFT = Rey Oesterrieth Complex Figure Test, TMT = Trial Making Test, WCST = Wisconsin Card Sorting Test, NCC = Number of categories completed, TC1 = Trials to complete category 1

Discussion

Summary of Results and Their Interpretation

The aim of the present study was to clarify whether there are PTSD- and CSA-related neurocognitive deficits observable in a sample of traumatized South African youths. Therefore, more then 100 adolescents were assessed using a broad neuropsychological test battery as well as a diagnostic interview and questionnaires in a cross-sectional design. It was hypothesized that the CSA+/PTSD+ group has lower scores compared to the other groups. Using different measurements of memory, attention, and executive functioning, we found results contrary to our expectation that the interaction of experiencing CSA and having a PTSD would lead to impaired performance. Although some of the test scores showed a significant interaction effect, none of the post-hoc tests supported the assumption.

Memory.

Regarding verbal memory, results indicated a significant interaction effect on scores in the first and sixth trial as well as the total number of words learned within the RAVLT. While expecting that these interactions were driven by worse performance in CSA+/PTSD, the data showed an opposite effect: If participants did not experience CSA, the expected pattern of lower scores within the PTSD group emerged. When looking at sexually abused participants, however, the pattern inverted, resulting in higher scores in those who had PTSD compared to those without. Interestingly, there was a main effect of CSA on the interference parameter showing that participants with CSA experience remembered fewer items in the sixth trial relative to the fifth trial, which can be attributed to the inferring influence of list B. This result could be interpreted in the light of CSA victims getting distracted more easily – a result which is in line with others reporting reduced ability for attention and inhibition associated with CSA (Barrera et al., 2013; Porter et al., 2005). Barrera and colleagues argue that such CSA associated impairments might be a risk factor for general psychopathology after CSA because they are related to cognitive inhibition (Barrera et al., 2013).

In the lack of significant findings within Paired Associates WMS-R subtest, we found evidence for PTSD and CSA related impairments in the Logical Memory subtest, but – again – not the expected interaction of both. Correlation analyses on the Logical Memory subtest scores revealed negative association of symptom severity as measured by CPC and scores in both immediate and delayed recall of small to medium magnitude. Although the results did not remain significant after adjustment of the critical p value, this finding is in accordance to

previous reports of memory impairments associated with PTSD. However, we could not detect a negative correlation between memory performance and CSA as it was reported in other studies (De Bellis et al., 2013).

These results add to the literature in several ways. Although verbal memory deficits in PTSD are consistently reported in adults (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008; Scott et al., 2015), some studies in children and adolescents led to group effects (De Bellis et al., 2009, 2013) while others did not (Beers & De Bellis, 2002). In light of absent hippocampal atrophy in children with PSTD (Woon & Hedges, 2008), our observation of PTSD related verbal memory impairment is of further interest because it can be interpreted as functional hippocampal impairment, which is often reported in adult population.

Lower performance observed in those participants who experienced CSA is in line with adult studies suggesting that CSA is related to memory impairments (Bremner et al., 1995, 1997, 2004; Savitz et al., 2007) and adds literature to this topic for adolescent populations. For instance, when looking at the effect of CSA on cognitive functioning, Barrera and colleagues (2013) were unable to detect differences with respect to PTSD diagnosis when looking at sexually abused children. When comparing all sexually abused children with control participants, no effects of verbal or visual learning emerged. In a similar way, Porter et al. (Porter et al., 2005) reported that they observed differences in attention and recall indices between sexually abused and comparison participants, but effects disappeared after controlling for socioeconomic status and IQ. Further, performances of sexually abused children were within average range. In addition to this, there were no differences between children diagnosed with PTSD and the control participants (Porter et al., 2005).

Interestingly, it seems that the experience of sexual abuse might be able to explain some of these inconsistencies as suggested by Kavanaugh and colleagues (Kavanaugh & Holler, 2015). For instance, we found that PTSD is associated with performance decrements in total number of words within RAVLT in those participants without the experience of CSA; however, when looking at the CSA positive individuals, the pattern inverted and participants without PTSD performed worse than those with PTSD. Despite hypothesizing that participants in the CSA+/PTSD+ group would have lower scores than the other groups, the CSA+/PTSD- group often had the lowest scores. Although some researcher argue that sexual abuse is related to general cognitive impairments (Jones et al., 2004; Sadeh, Hayden, McGuire, Sachs, & Civita, 1994), being diagnosed with PTSD additionally influences performance. How can the finding be explained that victims of CSA and PTSD diagnosis perform better than those without PTSD diagnosis? This could be interpreted in the light of

CSA increasing the risk of general psychopathology (Beitchman et al., 1992; De Bellis et al., 2011) and hence, having a negative impact on cognitive functioning even in the absence of formal diagnoses (e.g. Buckle et al., 2005). Thinking this further, one could argue that South African adolescents with CSA experience but without PTSD diagnoses might have lesser access to the public health system compared to adolescents with PTSD due to limited resources. The lack of supportive interventions in that particular group could account for the observed lower scores. However, this group did not differ from the others in most sociodemographical and trauma related aspects and future studies should address the question whether they differed in terms of therapy experiences or access to it.

With respect to performance on tests of visual memory, we found a trend effect for CSA related lower performance in the immediate recall condition within the Visual Reproduction WMS-R subtest further influenced by a marginal significant interaction effect. When looking at the descriptive statistics, this interaction might have been driven by differences between CSA positives and negatives in the non-PTSD diagnosis group with the lowest scores appearing in the CSA+/PTSD- group. However, no effects emerged in the delayed recall condition. As a second measurement of visual memory, the RCFT was used. When looking at both immediate and delayed recall condition, there were no effects of both CSA and PTSD as reported by others (Barrera et al., 2013; Beers & De Bellis, 2002; Yang et al., 2014), although descriptive statistics suggested better performance in the CSA-/PTSD- compared to all other groups. In the copy condition the interaction effect reached marginal significance with worse performance in the CSA+/PTSD+ group compared to the other three groups. This is congruent to the results of other studies reporting significantly worse performance in adolescent PTSD (Beers & De Bellis, 2002; De Bellis et al., 2013) or maltreatment populations in this subtest (Kavanaugh & Holler, 2015). However, this parameter is associated with attention and planning rather than memory, leading to the conclusion that there were no effects on visual memory at all.

The lack of findings regarding visual memory is in accordance with studies in adults showing that CSA is associated with verbal memory decline rather than visual memory decline (Bremner et al., 1995, 2004). In terms of PTSD, meta-analytic evidence suggest that deficits in visual memory have smaller effect sizes than differences in verbal memory (Brewin et al., 2007; Scott et al., 2015) which is further in line with predictions made by DRT. As stated in previous sections, one core assumption of the DRT postulates that symptoms of re-experiencing are caused by a well functioning image-based memory system (SAM) that is insufficiently inhibited by the impaired verbal system. Our results indeed

suggest that PTSD is associated with impaired verbal memory in the absence of decrements in visual memory. This can be interpreted as preliminary indication that the DRT is not only able to explain development of PTSD in adults, but might also be valid in some points with regards to adolescents.

Attention, executive functioning, and language.

Besides memory, we also assessed EF and language fluency; however, we did not find any effects in the hypothesized way. Although we were not able to detect any group effects on the reaction time to complete the TMT trials, we found small to moderate correlations with CSA and CPC scores. As expected, higher scores in severity of CSA and PTSD were associated with longer reaction times in both trials, but the results did not remain significant after alpha error adjustment. We further observed a marginal significant main effect of PTSD on digit span backwards within the SSAIS-R Memory for Digits subtest, indicating a trend for worse WM associated with PTSD. Analysing the WCST – a test associated with shifting and problem solving – a trend for an interaction effect on the number of correct trials was revealed. In participants without CSA experience, PTSD diagnosis was associated with a smaller number of correct trials; however, this pattern was inverted in sexually abused participants: participants without PTSD diagnosis had less correct trials compared to those with PTSD. Interestingly, CSA severity was positively associated with the number of trials to complete the first category, but again the correlation did not survive bonferroni adjustment. Nevertheless, this could be an indicator for impaired problem solving abilities associated with the experience of sexual abuse.

The lack of findings in the domain of EF contrasts to what is suggested by studies in adults (Polak et al., 2012; Scott et al., 2015) and neurobiological studies, highlighting both structural and functional impairments in frontal regions (Carrion et al., 2009, 2008; Carrion, Weems, et al., 2010). However, it could be argued that EF and the PFC are still maturing during late childhood (Blakemore & Choudhury, 2006), therefore possibly masking the effects of PTSD and CSA until late adulthood. A recent review (Kavanaugh et al., 2016) concluded that attention and EF are one of the most studied neuropsychological domains following maltreatment with only one study not finding maltreatment-related weakness in EF (Mezzacappa, Kindlon, & Earls, 2001). For instance, when comparing maltreated and non-maltreated adolescents, Kavanaugh and colleagues (2015) found worse performance in several domains of executive functions, including cognitive flexibility and shifting, WM, as well as problem solving and planning. However, the effects disappeared when controlling for PTSD, suggesting that although maltreatment is related to impairments, the diagnosis of

PTSD was associated with the significant differences. Furthermore, PTSD was negatively associated with executive and language measures. With regard to sexual abuse, the authors reported negative correlations of small to medium effect sizes between sexual abuse and executive functioning and language. They assessed problem solving using the WCST and planning with the RCFT copy condition and the TMT-B score representing a measurement of cognitive flexibility and shifting. Although these and other studies have reported PTSD and abuse related impairments in EF domains, we predominantly did not find this to be the case. There are several aspects that should be taken into account when discussing these differences: Kavanaugh and colleagues (2015) assessed an inpatient sample in which the control group was not maltreated (i.e. non-traumatized). This is in line with other studies who found group effects of trauma and PTSD when comparing the traumatized participants with a healthy control group (Beers & De Bellis, 2002; De Bellis et al., 2009, 2013). Therefore, the lack of significant findings regarding EF might be due to the fact that group differences are very small when comparing trauma-exposed individuals without PTSD to trauma-exposed individuals suffering from PTSD – a suggestion also made by Yang and colleagues (Yang et al., 2014) who were not able to detect any group differences in youth witnessing an earthquake.

Finally, in terms of language, we found a trend for worse performance in PTSD patients with respect to the COWAT Animals condition. The performance was also negatively related to CPC score with a small to moderate effect size before bonferroni adjustment. While some studies were able to detect group effects (De Bellis et al., 2009, 2013; Kavanaugh & Holler, 2014), they only emerged when comparing trauma-exposed adolescents to healthy controls, whereas post-hoc procedures revealed no differences in terms of PTSD diagnosis in the two trauma-exposed groups. In a similar way, a number of studies could not detect language related group effects of PTSD (Beers & De Bellis, 2002) or abuse (Nolin & Ethier, 2007; Vasilevski & Tucker, 2015). Although previous studies report a relationship between sexual abuse and language weakness (De Bellis et al., 2013), we were not able to replicate this in the present sample.

Prediction of severity.

To answer the question whether the results of neuropsychological tests can be used to predict PTSD and CSA severity, all subset regression analyses were performed. Regarding CTQ-CSA sub score, the final model contained 13 predictors with scores in WMS-R visual reproduction delayed recall condition, TMT trial A, COWAT FAS scores, and RCFT delayed recall scores showing beta weights significant different from zero. Eleven predictors showed

the highest predictability of CPC scores, but although the predictors contained neuropsychological test, none of them had a beta weight significantly different from zero. However, both models were able to explain around 40% of variance within the CSA and CPC scores, respectively. These results are in accordance with other studies reporting that neuropsychological test scores can predict CSA (Barrera et al., 2013) and PTSD symptomatology (De Bellis, Hooper, Woolley, & Shenk, 2010; Suliman, Stein, & Seedat, 2014).

Due to the fact that depressive and additional diagnoses contributed significantly to the models, analyses were repeated only including participants without any comorbid disorders. This influenced and changed the models. With respect to CSA sub scores, the best model included eleven predictors, amongst them all three RCFT sub scores, scores of TMT trial B, WMS-R Logical Memory Delayed condition, and number of preservative errors in WCST being significant. Excluding participants with comorbid disorders further influenced the model for predicting CPC scores with RAVLT Recognition index, immediate and delayed RCFT recall score, WMS-R Logical Memory Delayed Recall and number of correct WCST trial being significant predictors. When only including participants without comorbid disorders, the models could predict approximately 65% and 75% of variance, respectively. This indicates that high comorbidity rates observed in the present sample influenced the results of all subset regression by inducing more bias. Unfortunately, it is not clear how participants with comorbid disorders might have further influenced the results of the between group ANOVAs. If we were to exclude all participants with comorbid disorders, the group size would become too small for calculating ANOVAs including all covariates.

Generalizability and Limitations

In the interpretation of results, it is worth bearing in mind some limitations. Certain issues need to be addressed to answer the question of how far the reported results can be generalized and for what kind of inference population they can be generalized. It is evident that the current sample represents a specific population and it is an open question to what extent the results can be generalized to other populations in Africa or even populations with a separate cultural background (i.e. Western Europe). Further, the data was acquired within an accumulating sample restricting the external validity. Due to the method of recruitment using schools and nongovernmental organizations, only those adolescents in contact with these referring institutions could be assessed. It could be argued that these adolescents might differ from those who are not enrolled in these programs in terms of e.g. help-seeking behaviours.

Assuming that the participants included in the current study possibly had better access to consultant services and could therefore be recruited, leads to the question whether these participants differ from those adolescents without any connections to such support-providing organizations in terms of cognitive functioning. Future studies should address this topic by putting emphasis on recruiting a representative sample.

As indicated in the exploratory analyses, excluding participants with comorbid disorders influences the results of the best subset regression and it seems reasonable that these participants might also have biased the group comparisons. However, it is debatable whether participants with comorbid disorders should be excluded or not. Excluding them increases the internal validity of the results by making it possible to draw conclusions about whether CSA and PTSD are associated with cognitive impairments. In the present sample, it cannot be ruled out that other (comorbid) disorders might have covered group differences associated with PTSD and CSA. For instance, depression is also associated with cognitive impairments (Austin, Mitchell, & Goodwin, 2013), but it was not possible to examine the influence of depressive symptoms in the present study because the BDI was not administered to all participants with the same being true for resilience. On the other hand, excluding all participants with comorbid disorders would diminish the external validity of the results due to high incidence rate of comorbidities in PTSD patients (American Psychiatric Association, 2013).

Further, the present data does not offer insight regarding the question if previous or concurrent treatment may influence the results. As mentioned before, the data showed the unexpected pattern that scores in the CSA+/PTSD- often tend to be lower than in the other groups, even if not significant in all groups. To test the post-hoc explanation that this could be associated with a lack of treatment and supportive interventions, future studies should include therapy experiences. For instance, some studies found evidence that cognitive-behavioural therapies not only decrease clinical symptoms but also help to restore neurocognitive functioning (Walter, Palmieri, & Gunstad, 2010).

Another potentially confounded variable that could not be considered in the present analyses is intelligence, however, it is very likely that intelligence plays a role in PTSD. For example, Vasterling et al. (2002) reported that IQ was negatively correlated with PTSD severity, even after controlling for trauma exposure. The effect for lower IQ as a predictor of PTSD is thereby $r = .18$ (Brewin, Andrews, & Valentine, 2000). The finding of lower IQ in adult PTSD patients compared to both healthy (Gil et al., 1990; Jelinek et al., 2006) and traumatized controls (Boscarino, 2006; Brandes et al., 2002; Pitman et al., 2006; Vasterling,

Brailey, Constans, Borges, & Sutker, 1997), leads to the question whether low IQ is a risk factor or high IQ is a protective factor (McNally, 2006). In a longitudinal study, Breslau and colleagues found that IQ over 115 measured in childhood was protective against a PTSD symptomatology in adolescence (Breslau, Lucia, & Alvarado, 2006). Similar associations were found for adults (Betts, Williams, Najman, Bor, & Alati, 2012; Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). Together with findings of intelligence being related to performances on neuropsychological tests (Ardila, Pineda, & Rosselli, 2000; Bolla‐Wilson & Bleecker, 1986), this stresses the necessity of including IQ measurements in future studies. Due to the fact that IQ was not included in the current analyses, we cannot exclude the possibility that the observed differences in memory domains are mainly or partially driven by differences in IQ. Therefore, future studies should pay attention to the influence of this potential confounder.

Further methodological limitations lay in the lack of a healthy control group that might account for the lack of significant findings in some tests. Previous studies were often not able to detect differences between trauma exposed control participants without PTSD diagnosis and PTSD patients, neither in adults (e.g. Stein et al., 2002) nor in adolescents (e.g. De Bellis et al., 2009, 2013). Meta-analytic results point to the assumption that while group differences between healthy controls and PTSD patients are large, the differences to trauma-exposed controls are only modest (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008), therefore making it necessary to include large group sizes to be able to detect small and medium differences with sufficient power. As the post-hoc power analyses revealed the sample size had sufficient power to detect large effects, but WCST and WMS-R Visual Reproduction subtest were not administered to all participants, thus lowering the power to detect large effects on these tests below the acceptable threshold. The power to detect moderate or small effects was only 0.32 or 0.08, respectively; therefore, the results of the current study cannot give definitive insight to the question whether there are small or moderate group differences between trauma-exposed and PTSD adolescents.

Due to the cross-sectional, observational design used in the current study, it is not possible to make causal interferences regarding the question whether PTSD symptoms lead to neuropsychological impairments or if neuropsychological impairments facilitate the development of PTSD symptoms. Much of our knowledge of risk factors for the development of PTSD derives from cross-sectional study designs where participants are asked to give a retrospective report about pretrauma risk factors with some studies even assessing change in these pre-trauma risk factors over time. However, participants might be influenced by recall

biases making prospective longitudinal studies necessary (DiGangi et al., 2013). Such prospective longitudinal designs are characterised by assessing participants both prior to and after the experience of traumatic events to draw conclusions about whether, for example, pre-trauma cognitive functioning can predict later development of PTSD symptoms. For instance, a ground-breaking study conducted by Parslow and Jorm (2007) found group differences in neuropsychological performance between PTSD patients and traumatized controls not only after, but also prior to trauma exposure. These results have been expanded for other neuropsychological tests (Marx, Doron-Lamarca, Proctor, & Vasterling, 2009) as well as for IQ (Macklin et al., 1998). Further, the assumption of neuropsychological impairments being risk factors for the development of PTSD supports findings of a relationship of childhood IQ and later PTSD symptomatology (Betts et al., 2012; Breslau et al., 2006; Koenen et al., 2007). Further evidence for a causing rather than a consequent role of cognitive impairments in PTSD derives from twin studies (Gilbertson et al., 2002, 2006) leading to the conclusion of a familial vulnerability regarding neurocognitive performance since the decreased cognitive functioning as well as smaller hippocampal volume was also observable in the monozygotic PTSD patient's co-twin who was not exposed to combat traumas.

At the same time, attention should be drawn to the materials used in the current study. Due to the fact that some information is not accessible for external observation (e.g. thoughts and feelings), questionnaires are a crucial tool in psychological research in general and psychiatric research in particular. Although relying on standardized and established neuropsychological tests and questionnaires, their application as outcome measurement is associated with certain disadvantages (Milne, 1999). To enable participants to give reliable self-disclosure, introspective insight is necessary. Further, perception of the self needs to be unbiased and complex response processes as required when answering questionnaires need cognitive resources. Moreover, diagnostic features associated with PTSD might have induced additional biases. For instance, concentration problems in patients might have diminished their resources and therefore their ability to answer the questionnaires. This can particularly affect retrospective information (i.e. CTQ). Apart from that, response tendencies or social desirability could have affected the questionnaire data. On the other hand, neuropsychological tests seem to provide more objective behavioural data; however, it is debatable whether they are suitable for the purpose of the current study. Neuropsychological tests were initially developed to assess impairments in brain functioning after stroke or traumatic brain injury (Reitan, 1955). It remains an open question whether or not the neurobiological alterations associated with trauma and PTSD are severe enough to be detected with these measurements,

especially without using a healthy control population. Furthermore, the results of other studies suggest that performances of PTSD patients usually fall within an average range (e.g. Breslau et al., 2006). In a similar way, it can be problematic to use neuropsychological tests for participants of differing age since age affects neurocognitive performance (Gur et al., 2012). Unfortunately, not all tests were normalised for the age range of this study sample, thus making it necessary to use row scores and include age as a covariate. However, it is not clear whether the applied measurements are actually comparable in an adolescent sample, especially regarding the fact that there were significant group differences in age with participants in CSA-/PTSD- group being younger than those in CSA-/PTSD+. Assuming that adolescents' performance increases with age, any effects could have been masked by effects of PTSD. Hence, future studies should put more emphasis to the selection of measurements that are normalised accordingly.

There is further support for the assumption that the chosen measurements may not have been suitable for our sample and research question. On a statistical level, testing the assumptions of the performed tests revealed problems mostly regarding the normal distribution of either the data itself or the errors of the models. In particular, CSA scores were a cause for concern. Although both regression and ANOVA represent quite robust methods of data analysis (Harwell, Rubinstein, Hayes, & Olds, 1992), it would be desirable to replicate the findings in future studies. Additionally, there were discrepancies between R^2 and adjusted R^2 indices. The adjusted R^2 provides information about the loss of predictive power, i.e. how well a model generalizes, and should be very close to the value R^2 (Field, Miles, & Field, 2012, p. 1059). However, the observed shrinkage between R^2 and adjusted R^2 means that the models would account for less variance if they were based on a population as opposed to the sample. This again stresses the question regarding the inference populations and how much the results can be generalized.

Starting Points for Further Research

Although this study adds to the literature of CSA and PTSD in adolescents, it is nevertheless a research area with only preliminary results and knowledge. Therefore, future studies are required; particularly in respect to the limitations discussed above. To sum up, it would be desirable to replicate the reported results with a larger sample size to rule out whether the lack of findings in domains of visual memory and EF is due to low power or whether traumatized South African adolescents in fact do not differ from South African adolescent PTSD patients. Further, it would be of interest if these traumatized participants differ from healthy controls as

suggested by other studies (De Bellis et al., 2009, 2013; Kavanaugh & Holler, 2014). When including non-traumatized control participants, it might be worth paying attention to other measurements and confounding variables as well. For instance, paradigms like No-Go task as measurements of inhibitory control might be less prone to ceiling effects than neuropsychological tests and could be used in conjunction with neuroimaging techniques. This could enhance our understanding of dysfunctional brain circuits associated with PTSD and CSA in adolescents. Comparing this data with existing data of adult PTSD patients may give further insight into how effects of early and late traumatization are different on brain level, leading to starting points for more efficient pharmacological interventions. Additionally, future studies should answer the question whether the observed effects in verbal memory generalize to other samples, nationalities and cultures.

Besides addressing the limitations, replicating the results of the current study can be useful in terms of secondary research. Although there are numerous meta-analyses of cognitive impairments in adult PTSD patients (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008; Polak et al., 2012; Scott et al., 2015), we are not aware of any meta-analyses targeting children and adolescents with PTSD. However, in regards to the conflicting results in the literature – especially in terms of memory – it seems to be crucial to test whether there are neurocognitive impairments associated with adolescent PTSD. Further, a meta-analytic approach can be helpful in clarifying the role of CSA as well as determining the effect size of cognitive impairments in traumatized adolescents.

Additionally, future research is needed to answer the question of whether neuropsychological impairments are either a cause or a consequence – also known as the "chicken and egg problem" (Jelinek et al., 2006). Twin and prospective studies (Gilbertson et al., 2002, 2006; Macklin et al., 1998; Marx et al., 2009; Parslow & Jorm, 2007) suggest that "these categories, long considered [as] aspects of post-trauma psychopathology, were actually present before index trauma" (DiGangi et al., 2013, p. 740). However, it cannot be fully dismissed that the trauma itself has certain effects on cognitive functioning. Vasterling and Brailey (2005) argue that a "downward spiral" (p. 192) may exist in which impaired neurocognitive functioning leads to an increased vulnerability for PTSD and the disorder itself, causing more cognitive dysfunction. This facilitates the development of further symptoms. Due to a lack of research examining adolescents, more studies are required to clarify whether the assumption of a "downward spiral" deriving from adult samples can be transferred to adolescents.

Theoretical, Clinical and Practical Implications

Despite having limitations, there are several implications that can be derived from the results of the present study. Although we found PTSD and CSA related impairments in verbal memory, there were no group effects with respect to visual memory and EF. This lack of findings lead to the conclusion that – although impairments in all these domains were found in adults (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008; Polak et al., 2012; Scott et al., 2015) – it is not possible to generalize the findings in adults to adolescents without restrictions. Instead, it might be more reasonable to assume that certain impairments do not emerge until adulthood (Karl et al., 2006). In line with suggestions made by Stein and colleagues (Stein et al., 1999) who attributed their lack of findings to the young age of study participants. Cognitive impairments could further be associated with illness duration and chronicity (Felmingham et al., 2009; Johnsen & Asbjørnsen, 2008; Navalta et al., 2006) and cannot be found in small adolescent sample sizes.

Moreover, studies of prevalence rate of trauma and PTSD conducted in South African samples (e.g. Seedat et al., 2004; Suliman et al., 2005) indicate high exposure rates, thus leading to the conclusion that South African youths differ in some regards from e.g. Western youths. The fact that participants were experiencing multiple traumas might be one factor that could explain the differences to other studies. This further indicates that cultural and socioeconomic frameworks can influence whether the PTSD experience per se is associated with neurocognitive decline when compared to traumatized controls. Exposure to multiple traumas as often observed in South African adolescents (e.g. Seedat et al., 2004) might have led to a greater trauma load concealing the additional effects of PTSD and CSA itself.

However, the presence of PTSD symptoms and not mere exposure per se can lead to effects on verbal memory. Interestingly, this is only true for the WMS-R Logical Memory subtest immediate recall condition while the experience of CSA was additionally associated with performance declines in the RAVLT. Using a detailed neuropsychological test battery was one of the strengths of this study because it offers insight into complex neurobiological alterations associated with both PTSD and CSA. For instance, CSA+/PTSD+ showed a similar performance to CSA-/PTSD- in the first trial of the RAVLT – a measurement of verbal short-term memory and auditory attention, whereas the CSA+/PTSD+ group performance in the RCFT copy condition was worse than in the other three groups. The copy condition is associated with visuospatial planning and attention. This is just one example to illustrate how complex the relationship between sexual abuse in childhood, PTSD, and neurocognitive functioning can be. Although we were expecting to find the lowest

performance to appear in CSA+/PTSD+ group, this was only the case in a minority of cases. However, another interesting pattern was observed, namely that the CSA+/PTSD- group often had performance deficits compared to the other groups suggesting that participants had higher functionality when suffering from PTSD symptoms compared to not having PTSD symptoms in conjunction with CSA. On a similar note, Buckle and colleagues (Buckle et al., 2005) report a positive association between CSA and academic achievements; whereas physical abuse is negatively correlated. However, this might be only true for some tasks because in other aspects of memory (e.g. verbal) CSA experience was associated with diminished performance.

The fact that reductions in cognitive functioning in domains of attention and memory were associated with CSA raises the question whether being sexually abused itself has a potential negative impact on neurocognition. PTSD is only one possible sequelae of CSA; therefore it might be that the participants in the CSA+/PTSD- were suffering from other comorbid disorders hampering their performance. Moreover, observed effects in domains of memory and attention can also be seen as reflecting general risk factors for any psychopathology. However, not every victim of CSA develops subsequent disorders, but reduced performance might still be present, stressing the need for interventions for CSA survivors regardless of PTSD diagnosis.

When considering all of the inconsistent results reported in separate studies using different neuropsychological tests, parameters, and participants; we are led to the question of whether or not these tests have sufficient sensitivity to be used to investigate PTSD patients. One could argue that impairments associated with PTSD are too small to be detected with tests designed to assess impairments after e.g. brain injuries. For instance, when comparing trauma exposed participants to PTSD patients, Karl and colleagues (Karl et al., 2006) only observed small to moderate hippocampal atrophy and it might be debatable whether this dysfunction is best measured using neuropsychological tests. Due to the fact that studies often observed PTSD patients' performance in these tests within a normal range (e.g. Breslau et al., 2006), it might be useful to develop neuropsychological assessments specific for PTSD patients and trauma survivors. For instance, experimental paradigms with a high number of stimuli might prevent ceiling effects and therefore, induce more variance in the findings. However, it must be kept in mind that neuropsychological tests have the advantage of being usable for individual assessment and evaluation, thus making them valuable to use in studies as well. It has been recommended to receive neurodevelopmental assessment following trauma exposure (National Scientific Council on the Developing Child, 2014).

Despite having found dichotomous group effects of PTSD and CSA on cognitive functioning, the results of the regression analyses indicate that there is also a continuous relationship between neuropsychological impairments and PTSD and CSA severity. Especially when only looking at those participants without additional diagnoses, measurements of memory and EF could predict PTSD symptoms as well as sexual abuse severity. Although regression analyses in cross-sectional data cannot give insight into causal relationships between the variables, the finding of neuropsychological results being predictive supports the assumption that effective PTSD treatments should also target neurocognitive functioning. Further, performances in such tasks could be used as an objective indicator of therapy success. As stated in DRT, PTSD symptoms arise due to a hampered VAM system; therefore, improvements in symptoms should be accompanied by improvements in measurements of verbal memory. It would be interesting in the future to test whether e.g. memory training on its own can improve symptom severity and if so, who may benefit from such interventions. The fact that we found PTSD and CSA associated impairments lead to the conclusion that these participants might profit most; however due to the lack of a non-traumatized control group, the current study offers no insight to the question of whether all four groups show performance decline. Admitting this to be true, interventions are needed that are easily accessible to traumatized adolescents. Promising approaches derive from the work of Emily Holmes. She found first evidence that playing the videogame Tetris might prevent the development of PTSD after experiencing a trauma (Holmes, James, Coode-Bate, & Deeprose, 2009). Although being limited to a short time window, this research is promising by possibly offering the opportunity of a stepped-care intervention while avoiding the risk of stereotypes.

Another suggestion made by the DRT is that there are no group differences in visual memory. Although we could not find main effects of PTSD on visual scores, the immediate and delayed RCFT recall condition contributed significantly to the model explaining CTQ-CSA and CPC scores. While the immediate condition predicted CSA severity negatively, the beta weight was positive for the delayed recall condition; and most interestingly this pattern was inverted when looking at CPC scores. This suggests that the role of visual memory in CSA and PTSD might be more multifaceted than previously assumed, hence the need for more attention in future studies.

Concluding Remarks

Evidence from previous studies in both adults and youths suggested that PTSD and CSA would have aversive effects on performance in neuropsychological tests. Despite shortcomings, the present study provides an important contribution to research in this area by showing that the experience of CSA might explain some of the inconsistencies observed in previous studies. By using a broad neuropsychological test battery, it further highlights the complex sequelaes of CSA experience and PTSD on cognitive functioning. The lack of findings in some areas might be due to methodological issues (i.e. lacking a sufficient sample size). We were able to find CSA and PTSD associated impairments in the domains of attention and verbal memory, even whilst using a traumatized control group offering a target subject for treatment interventions. This allows the conclusion that both PTSD and CSA have notable and unique impacts on neurocognition and differ from the effects of mere traumatization. However, in terms of identifying who is in need for interventions, the inclusion of a healthy control group is indispensable. There is yet still an amount of open questions remaining to be studied and researched on neurocognitive deficits in traumatized youths; therefore further future research is required to expand and extend our knowledge.

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Appendix

A Tables

B Figures

C Eidesstattliche Erklärung

D Veröffentlichung der Abschlussarbeit im Bibliothekssystem

Table 1. Diagnostic Criteria (American Psychiatric Association, 2013, pp. 271 - 274)

Posttraumatic Stress Disorder (309.81)

A Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:
   1. Direct experiencing
   2. Witnessing, in person, the event(s) as it occurred to others
   3. Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.
   4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains; police officers repeatedly exposed to details of child abuse).
      Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:
   1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).
      Note: In children older than 6 years, repetitive play may occur in which terms or aspects of the traumatic event(s) are expressed.
   2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).
      Note: In children, there may be frightening dreams without recognizable content.
   3. Dissociative reactions (e.g. flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.)
   4. **Note**: In children, trauma-specific enactment may occur in play.
   5. Intense and prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
   6. Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

C Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:
   1. Avoidance of or efforts to avoid distressing memory, thoughts, or feelings about or closely associated with traumatic event(s).
   2. Avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

D Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:
   1. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).
   2. Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g. “I am bad”, “No one can be trusted”, “The world is completely dangerous”, “My whole nervous system is permanently ruined”.)
   3. Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others.
   4. Persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame).
   5. Markedly diminished interest or participation in significant activities.
   6. Feelings of detachment or estrangement from others.

7. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

E Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:
1. Irritable behaviour and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects.
2. Reckless or self-destructive behaviour.
3. Hypervigilance.
4. Exaggerated startle response.
5. Problems with concentration.
6. Sleep disturbances (e.g., difficulty falling or staying asleep or restless sleep).

F Duration of the disturbance (Criteria B, C, D, and E) is more than 1 month.

G The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H The disturbance is not attributable to the physiological effects of a substance (e.g. medication, alcohol) or another medical condition.

Specify whether:

With dissociative Symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and in addition, in response to the stressor, the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalisation**: Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).
2. **Derealisation**: Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts, behaviour during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Specify if:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Table 2. Sociodemographic characteristics (categorical data)

Measure	Diagnosis subsyndromal (n = 14)	Diagnosis definitive (n = 38)	χ^2	p
Gender			0.736	0.391
Female	7	24		
Male	7	14		
Ethnicity			0.481	0.488
White	2	3		
Non-White	12	35		
Level of education			6.082	0.014*
Grade 4-7	5	3		
Grade 8-12	9	35		
Language of Assessment			0.259	0.611
Afrikaans	7	16		
English	7	22		
Index trauma			*Not computable*	
Car accident	0	2		
Other accident	0	0		
Fire	0	0		
Witness of disaster	0	0		
Witness of violent crime	3	6		
Victim of violent crime	2	10		
Confronted traumatic news	1	7		
Witness to domestic violence	4	1		
Physical abuse	2	0		
Sexual abuse	2	12		
Other	0	0		
Index trauma			4.222	0.039*
Acute	8	32		
Chronic	6	6		
Chronic trauma experience in Childhood			0.899	0.343
Yes	6	11		
No	8	27		
Additional diagnoses			3.791	0.052
Yes	5	25		
No	9	13		
Depressive Diagnoses			2.383	0.123
Yes	4	20		
No	10	18		

Note. N = 52, p = p-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, χ^2 = Chi Square value

Appendix

Table 3. Sociodemographic characteristics (interval data)

Measure	Diagnosis subsyndromal (n = 14)	Diagnosis full syndromal (n = 38)	t	p
Age			4.146	0.047*
	15.20 ±1.89	16.30 ±1.68		
Number of additional diagnoses			3.100	0.084
	0.57 ±0.85	1.13 ±1.07		
Age at index trauma			5.810	0.020*
	11.64 ±3.30	14.00 ±3.07		
Months between index trauma and assessment			2.009	0.163
	33.00 ±48.64	19.89 ±18.72		
Number of experienced trauma (Checklist)			0.200	0.657
	3.29 ±1.27	3.47 ±1.37		
LEQ			0.956	0.333
	12.71 ±4.75	11.18 ±5.09		
CPC			9.878	0.003**
	31.43 ±16.50	45.13 ±12.93		
CTQ			0.989	0.325
	64.14 ±13.10	68.29 ±13.42		
CSA			1.399	0.243
	6.43 ±3.01	8.37 ±5.83		

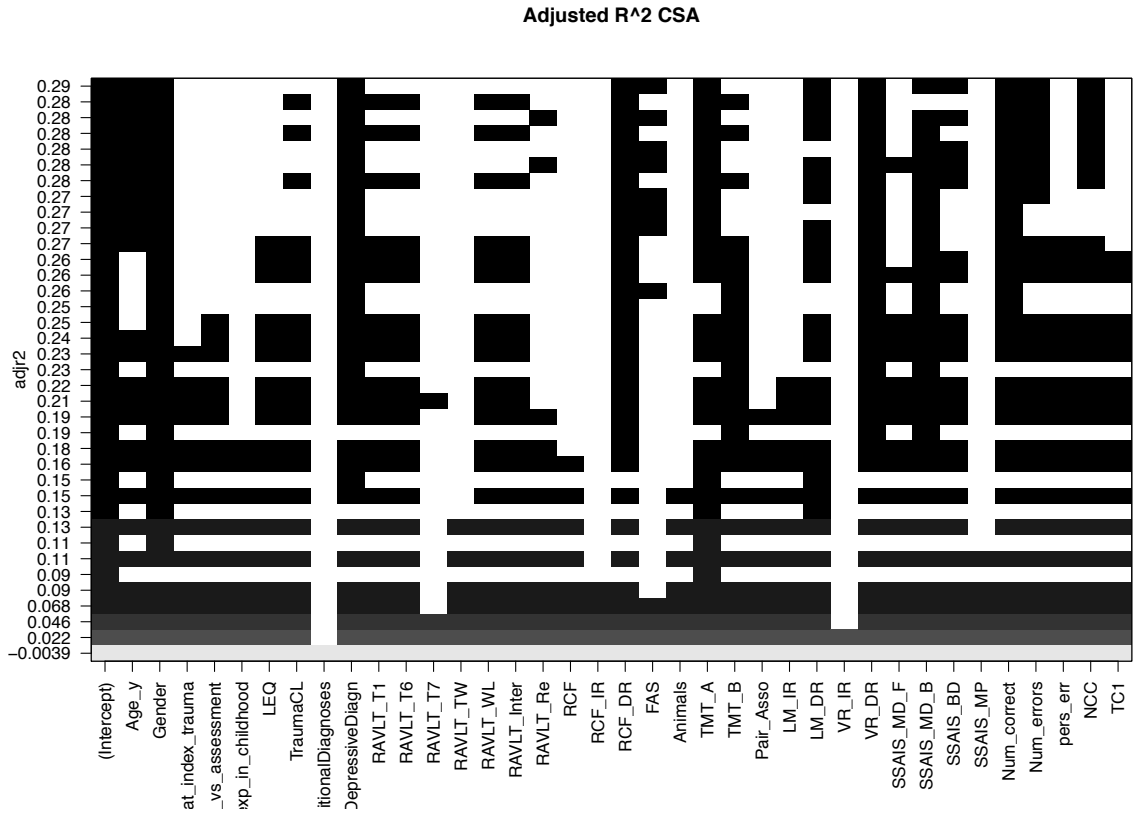
Note. N = 52, p = p-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, t = t-value.

Table 4. Welch two sample t-test on neuropsychological performance

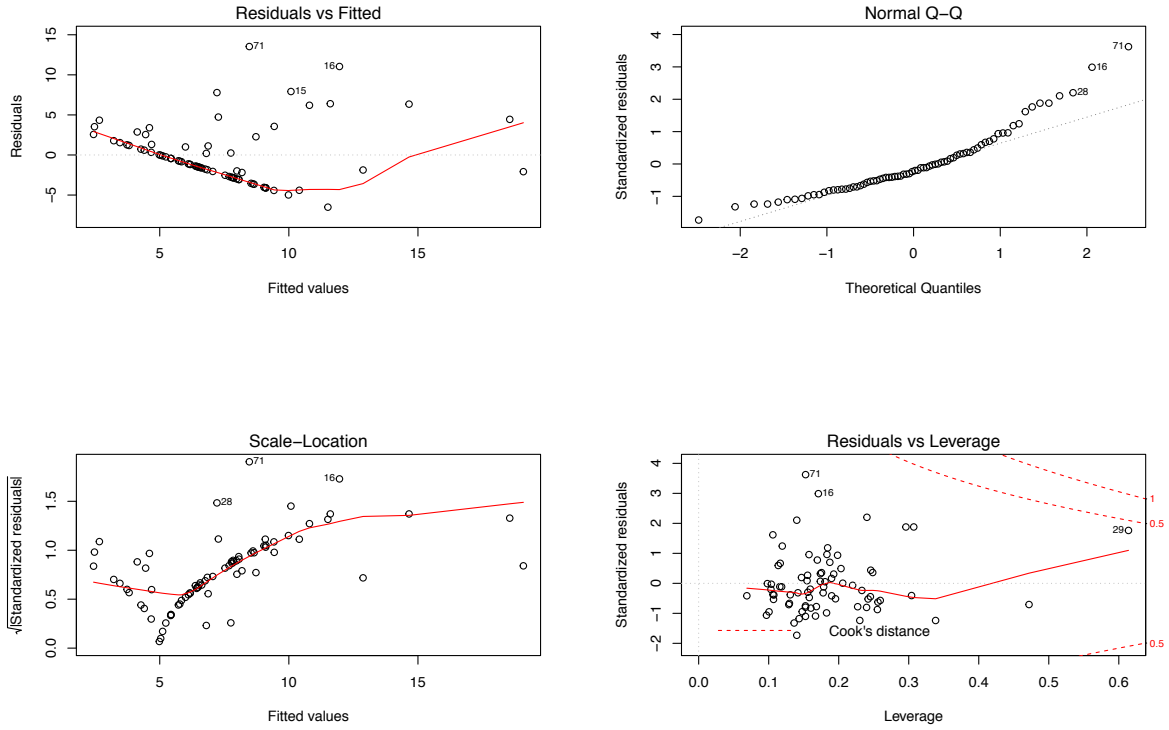
Measure	Diagnosis subsyndromal (n = 14, [a] n = 12)	Diagnosis full syndromal (n = 38, [a] n = 28)	t	p
Rey Auditory Verbal Learning Test (RAVLT)				
Trial 1	6.36 ±1.82	6.13 ±2.07	0.381	0.706
Trial 6	11.00 ±2.51	10.47 ±2.75	0.653	0.520
Trial 7	11.00 ±2.88	9.97 ±3.16	1.109	0.278
Total Words	49.57 ±9.80	48.16 ±9.76	0.462	0.648
Words Learned	5.71 ±2.58	5.50 ±2.11	0.278	0.784
Interference	91.98 ±17.41	89.23 ±16.23	0.513	0.613
Recognition	12.71 ±2.20	11.24 ±4.64	1.547	0.129
Rey Oesterrieth Complex Figure Test (RCFT)				
Copy	32.54 ±3.34	33.21 ±5.27	-0.547	0.588
Immediate Recall	17.64 ±6.26	20.83 ±6.54	-1.608	0.121
Delayed Recall	19.18 ±6.56	20.20 ±6.96	-0.489	0.630
Controlled Word Association Test (COWAT)				
Animals	13.43 ±3.01	13.08 ±3.57	0.3532	0.727
FAS	22.29 ±10.71	22.08 ±8.49	0.0651	0.949
Trial Making Test (TMT)				
Trial A	36.26 ±11.48	50.05 ±19.66	-3.118	0.003**
Trial B	88.92 ±30.33	113.36 ±46.27	-2.213	0.030*
Wechsler Memory Scale-Revised (WMS-R)				
Paired Associates	19.43 ±4.18	19.21 ±3.87	0.170	0.867
Logical Immediate	17.36 ±7.31	16.11 ±6.51	0.564	0.579
Logical Delayed	13.79 ±5.67	12.45 ±6.44	0.727	0.474
Visual Immediate [a]	31.67 ±3.77	30.54 ±5.31	0.760	0.451
Visual Delayed [a]	26.92 ±6.22	26.32 ±6.59	0.273	0.788
Senior South African Individual Scale-Revised (SSAIS-R)				
Digit Span Forwards	8.43 ±3.06	8.68 ±2.29	-0.285	0.779
Digit Span Backwards	4.07 ±1.44	4.08 ±2.05	-0.015	0.988
Digit Span Total	12.50 ±3.98	12.76 ±3.51	-0.219	0.8294
Block Design	19.57 ±7.61	20.92 ±7.63	-0.567	0.576
Missing Parts	15.14 ±3.03	13.79 ±3.40	1.380	0.180
Wisconsin Card Sorting Test (WCST)				
Correct Trials [a]	71.75 ±11.46	72.21 ±13.66	-0.111	0.913
Error Trials [a]	40.92 ±21.70	41.11 ±23.06	-0.025	0.980
Perseverative Errors [a]	24.00 ±15.80	20.64 ±17.13	0.600	0.554
NCC [a]	4.33 ±1.78	4.82 ±1.56	-0.825	0.420
TC1 [a]	15.83 ±9.82	19.82 ±23.13	0.766	0.449

Note. N = 52, [a] N = 40, p = p-value, $p < 0.10^{+}$, $p < 0.05$*, $p < 0.01$**, $p < 0.001$***, t = t-value.

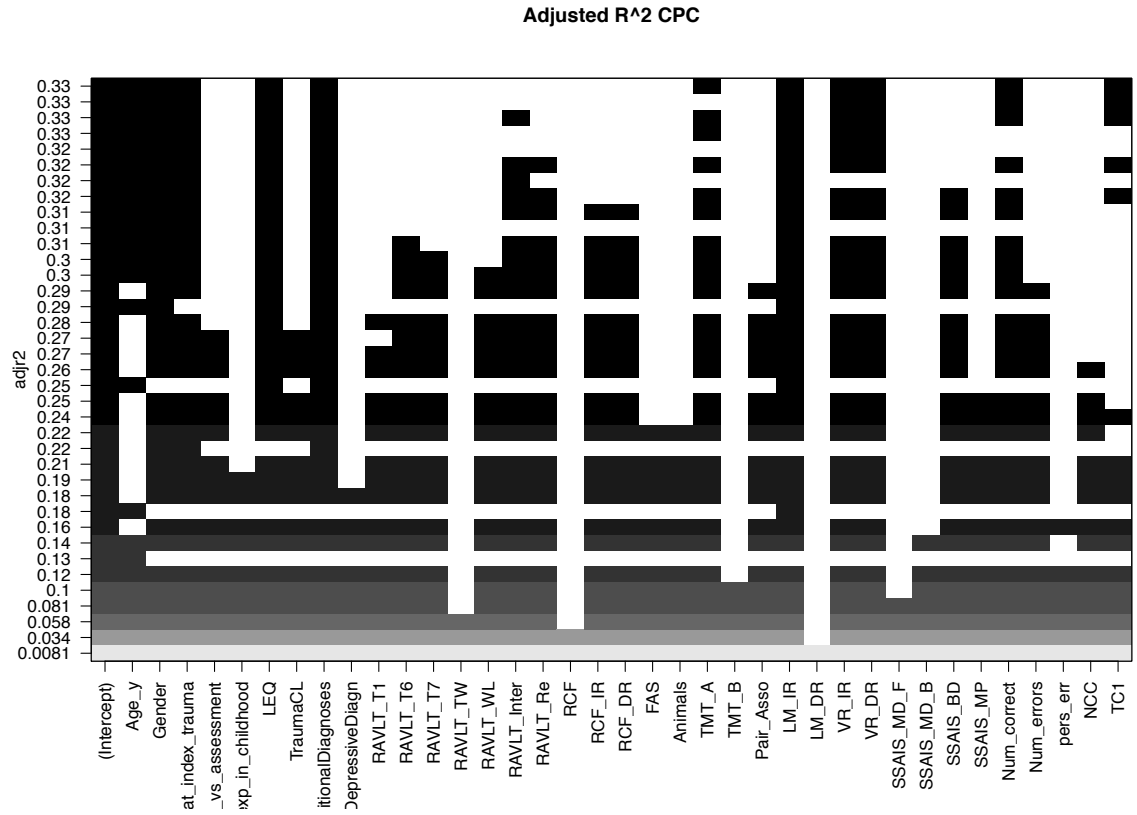
Appendix



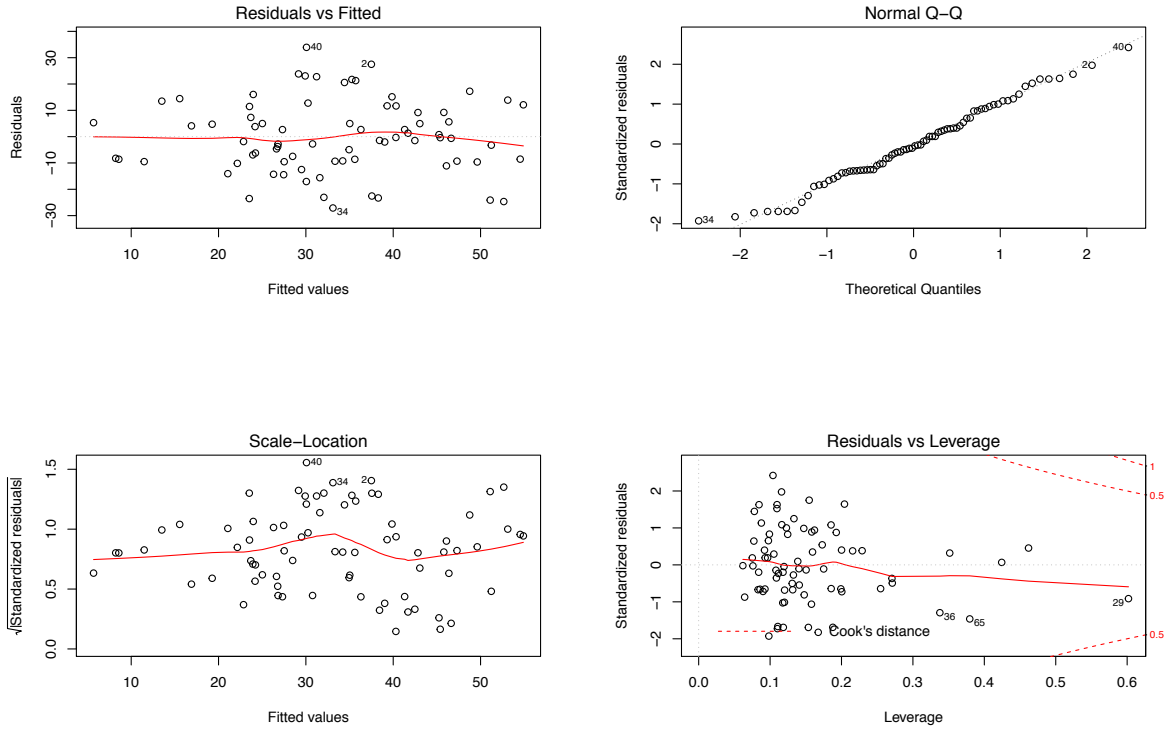
Appendix Figure 1. Results best subset regression to predict CSA score



Appendix Figure 2. Plot of final CSA model containing 13 predictors



Appendix Figure 3. Results best subset regression to predict CPC scores



Appendix Figure 4. Plot of final CPC model containing 11 predictors

Appendix

Eidesstattliche Erklärung

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