Trauma Stabilisation as a Sole Treatment Intervention for Post-Traumatic Stress Disorder in Southeast Asia

Research	Questions
- "The data set suggests trauma stabilisation, as a sole treatment intervention, was safe, effective, efficient and sufficient treatment intervention for PTSD. Furthermore, trauma stabilisation interventions have the advantage of being safe, flexible, and adaptable to the cultural and spiritual context in which they were applied" - Violence: "The intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, mal-development or deprivation" - "Our results strengthen earlier findings that trauma therapy doesn't necessarily require trauma exposure to be effective in reducing posttraumatic symptoms and increasing the level of functioning. Trauma Stabilisation doesn't focus on traumatic memories directly, but resource, stabilisation and skill development. Especially in post conflict areas, with a high risk for natural disasters, this treatment can prepare the clients for future traumatic events, strengthening coping skills and enhancing resilience and potentially post-traumatic growth	- "resource interventions, grounding techniques, comprehensive history taking, trauma preparation, trauma mapping, trauma case conceptualisation" how do counselors apply these? - What are some flaws of the phase approach? - What is therapeutic relationship?

General

- Factors that cause emotional trauma in Southeast Asia: Tsunami/Natural disasters & interpersonal violence (intimate partner violence or community violence).
- Eye Movement Desensitization and Reprocessing (EMDR) Therapy (For PTSD)
- TraumaFocused Cognitive Behavior Therapy (TF-CBT)
- ROTATE Approach
- Trauma-specific stabilisation

Attention focusing	Imaginative distancing techniques	Resource activating techniques
Away from traumatic internal experience and direct toward external neutral/positive stimuli = - distress	- trauma, +control and safety. Container technique and safe place exercise	+ positive emotions, - neg. Visualize positive outcome, success, role models, memories

Phase 1: Safety and Stabilisation	Phase 2: Remembering and Mourning	Phase 3: Reconnection
Making sure safety, reducing symptoms, + emotional, social, psychological competences, psychoeducation to explain symptoms and give hope and normalisation	Trauma confrontation: processing and resolving unresolved feelings/memories/experience	Consolidate changes and move forward

The role of executive function in posttraumatic stress disorder

Research	Questions
- "In posttraumatic stress disorder (PTSD) the memory of the traumatic event is thought to be fragmented, with storage in sensory fragments and retrieval occurring as sensory and emotional representations without the transcription into personal narratives" - PTSD impairs cognitive functioning, esp verbal memory, but inconsistently associated with impaired executive functioning (divided attention, cognitive flexibility, selective attention and inhibition, working memory and planning) - "This finding illustrates that impaired executive functioning in PTSD is not specifically nor merely related to exposure to trauma. Perhaps, people exposed to one or more traumatic events without having developed posttraumatic stress or depression might have more efficient coping strategies that do not interfere with the natural recovery and protect them from having PTSD"	- What are the appropriate actions to take for people who have exposure to trauma first time vs those who had continuous exposure? - How can counselors help clients with executive functioning?

General

- Declarative vs. Autobiographical memory
- "recall of emotional autobiographical events does not influence working memory function any more for PTSD patients than control"
- Different types of tests: Trail Making Test (TMT) measuring divided attention, Wisconsin Card Sorting Test (WCST) for cognitive flexibility or "'set shifting'", the WAIS-R Digit Span or WAIS III Digit Span measuring working memory, Stroop measuring selective attention and inhibition and the Rey-Osterrieth Complex Figure Test (CFT) measuring planning.
- Response Inhibition and Attention Regulation, PTSD
- Study examined different factors such as type of trauma, gender, culture etc and found that PTSD overall affected executive functioning which is crucial for daily functions. There were several differences among factors though.

Type of Trauma	Gender	Age	Comorbid Depression
+ profound for veterans maybe bc of repeated trauma exposure → dissociative symptoms, aggressive or socially avoidant behaviour (like seen in sexually abused patients). But there was not as profound - EF (executive function) in sexually abused. So symptom severity rather than frequency may be more predictive of - EF	Males did worse on EF but that may be bc of severity of PTSD and trauma rather than gender itself	Age was a major factor. Attention, executive and working memory are impacted by higher age. Maybe not higher age but total years of experiencing PTSD symptoms may impact EF	PTSD + Severe comorbid Depression = impairments in EF memory deficits, such as verbal memory, are related to (comorbid) depression too not just PTSD

Cognitive impairment and functioning in PTSD related to intimate partner violence

Research
- Study was done on women. Results can be generalized - Proven multiple throughout multiple studies that PTSD affects overall cognitive functioning, but which aspect is most affected is not researched thoroughly yet. - PTSD→ disruption in frontal subcortical circuits, - hippocampal size and functioning - "a consistent picture of the neuropsychological sequelae of PTSD has been elusive, partly due to the numerous confounds associated with trauma exposure" - + PTSD = worse speeded attention, +dissociation = - reasoning skills - Cognitive slowing → - attentional resources, which could be bc of brain resources being directed toward coping with psychological distress, unpleasant internal experiences or potential threats in the environment (hypervigilance) rather than the task at hand. "In effect, even simple tasks could become exercises in multitasking" - PTSD patients may experience micro-dissociations → -attention resources = slower performance. Also other factors: - sleep, +anxiety, +arousal, or recent triggers. Yes link bt +arousal and slower central processing.

Executive function and PTSD: Disengaging from trauma

Research	Questions
- Attention defined by William James : "the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. It implies withdrawal from some things in order to deal effectively with others" - "Lower IQ (often measured via military aptitude test performance) and educational achievement pre-trauma has been reported to relate to PTSD symptoms post-trauma" - "However, it is unclear whether PTSD is associated with primary problems in attention and working memory, or whether the inconsistent findings are due to difficulties coping with and inhibiting unintentional "distracters", such as internal (e.g., emotions, cognitions) or external stimuli" - "These results suggest that the acute emotional state may not have an overwhelming effect on objective cognitive function in PTSD. Instead, these studies support the existence of underlying deficits in working memory that in some situations can be worsened by the inclusion of emotional distractor stimuli." - "PTSD may be associated with hyperactivation of prefrontal areas in response to simple sustained attention tasks, but relative hypoactivation during tasks involving inhibition or "updating". The former could reflect the hypervigilance and enhanced attention towards "triggers" associated with PTSD, while the latter could relate to decreased ability to control or inhibit these attentional resources. Alternatively, hyperactivation during sustained attention could reflect compensatory activation to maintain attention during more simple tasks, which hits a ceiling or breaks down as working memory load increases, thus failing to compensate further for more complex inhibition or "updating" tasks."	- How do counselors help patients with attention based cognitive impairment? Takes lots of practice? - Revealing to a client that they have a cognitive impairment must be difficult, what's the best way to do it? Especially for South Asian clients? - How do counselors help clients disengage from perceived threat/triggers? And how do they help clients cope with hypervigilance toward threat? "it may be possible to experience hypervigilance towards threat but retain the ability to disengage or regulate that attention" - Is it beneficial to explain neurological/brain concepts to clients? - Is it helpful for counselors to identify what stage of PTSD client is in and help them accordingly/ or should always start from the beginning?

General

- Executive Function: "control of complex goal-directed behavior"

1. *Attention*: the voluntary allocation of processing resources or focusing of one's mind on a particular stimulus within the environment
    - Tests to measure attention: digit span, one trial word recall, spatial span.
      - performance on measures of auditory attention and working memory found in sexually abused & combat veterans.
    - There is a genetic component to PTSD because attention, executive function was lower for PTSD veterans and their twins→ lower pre-trauma cognitive functioning particularly in domains of attention, executive function, and memory may be as a risk factor for the development of PTSD.
    - But there is also evidence that says otherwise, that pre-trauma cognitive impair doesn't completely account for post-trauma cognitive impair in attention.

2. *Working Memory*: the active maintenance and manipulation of information in one's mind over a short period of time
    - PTSD→ - performance in auditory/visual sustained attention

3. *Sustained Attention*: the maintenance of attention on one set of stimuli or a task for long
4. *Inhibitory Function*: involving the inhibition of automatic responses to maintain goal-directed behavior
    - "Decreased inhibitory function has rather consistently been reported for PTSD" which could be because patients are re-experiencing symptoms and getting distracted. But also possible that primary inhibitory dysfunction results in not only difficulty in cognitive tasks but also inhibits emotional coping when triggers
    - Measure inhibitory function: Color word Stroop task examines response time to name the ink color of color related words. Red may be slower response time?
    - - performance = + detection of threat-relevant stimuli, or attentional interference involving difficulty disengaging from threat-related stimuli to focus attention on the task at hand. But inhibitory has most consistent findings on PTSD effects
    - Unclear whether impairment is specific to PTSD or most psychiatric disorders.

5. *Flexibility/Switching*: the ability to switch between two different tasks or strategies
    - Measures of flexibility: Trail Making Test: connection of "dots" while switching between letter and number and verbal fluency switching involving the production of words while switching between two categories.

6. *Planning*: the ability to develop and implement strategic behaviors to obtain a future goal
    - Measures: Wisconsin Card Sorting Test (WCST) and Tower of London Task
    - For the most part, no consistent deficits in this aspect.
    - **Neuropsychological research seems to provide inconsistent support for impairment in speed-reliant, attentional switching, but indicates that planning, rule-learning, and untimed strategy switching, may be mostly spared in PTSD"

- Decision making: PTSD slows down decision making
→ Could be bc of
1) lack of motivation or reward-seeking, (PTSD may be associated with dysfunction in reward system networks which could contribute to decreased motivation and reward-seeking)
2) impaired learning of response-outcome associations,
3) lack of disengagement from a non-optimal response strategy

Treatments

- CBT helped the attentional aspect. Verbal memory improved after paroxetine treatment (but more research needed to confirm this). Trauma-focused therapy (e.g., cognitive processing therapy, prolonged exposure therapy), for a small group of women resulted in + improvement on TMT number-letter switching and visual organization and overall executive function.
- Attentional training itself as a treatment for anxiety disorders. This research uses a modified dot-probe paradigm to "train" individuals to respond faster to probes presented away from negative stimuli → effective in reducing symptoms in social anxiety, generalized anxiety, and sub-clinical obsessive-compulsive disorder
- There have been no published studies investigating the effects of attention modification on PTSD symptoms or neural activation patterns. However, given the deficits in inhibitory and attentional functions observed in PTSD, this could be a promising area of research.

Other Concepts/Ideas

- "One basic ability we have as humans is to assess the value of environmental stimuli and quickly orient attention towards stimuli as needed (e.g., through "bottom-up" influences on attention). However, it is also important to be able to determine which stimuli are irrelevant or distracting to our current goals and disengage from those stimuli in order to orient towards those that are more goal-relevant (e.g., "top-down" regulation of attention)"

- Trauma → Trauma-associated stimuli = highly valued = demand greater attention → + difficult to disengage. Disengagement is amplified to extreme. Subtle deficits in inhibition = no influences until highly emotional trauma experience comes into the system. This development → PTSD = + re-experiencing and high arousal symptoms. Common coping - avoidance. Avoidance is ok for short term, but can be a problem when avoidance of such stimuli could be positive. (ex: PTSD patient avoids hospitals, or previously pleasurable activities like sports bc reminds them of injury and triggers. Continual avoidance = never learning how to stop re-experiencing. Also avoidance → socially isolated from close ones → emotional numbness/depression. Treatments can help with habituation of emotion and inhibition and regulation of automatic thoughts, feelings, and behaviors. There is evidence that training attention away from threats could help reduce symptoms and other anxiety disorders.

Trauma Narrative Goals and Processes

Things for Counselors to do	Questions
- Unpair thoughts, Desensitize, integrate thoughts and put trauma in meaningful experience, metacognitive abilities ability to think about and evaluate one's own thoughts and experiences → allows child integrate trauma to larger optimal self-concept - Intro child/parents theoretical basis and reassure - Tell children to tell more of what happened & thoughts/feelings. - Children are able to tell more before writing - Gradually expose to upsetting aspects of trauma - First ask a child to write perception of facts of trauma and then go back look and write thoughts/feelings. Don't interrupt let flow - Having a child read what is written helps desensitization. Also write down traumatic reminders/imaginations. Ask a child at some point to describe worst moment, worst memory. - If child becomes overwhelmed, remind that these are only feelings and/or memories, they are related to something that happened in the past and not something that is happening in the present - Use Subjective Units of Distress (SUDS) Scale to help children quantify their degree of distress within each (or some) sessions – fear thermometers, children's faces depicting different degrees of distress. Positive reinforcement - give reward after. - Children may elect to alter or modify parts of the TN during Cognitive Processing, write them down. - Finally reflect on how the child is now, what learned, advice to others.	- How much can parents be involved? - Ways to help children open up initially? - Should counselors tell children if they are imagining/making something up later? - What is something to avoid doing that discourages children?

Psychiatric Quarterly (2019) 90:63–88
https://doi.org/10.1007/s11126-018-9598-z

ORIGINAL PAPER

Trauma Stabilisation as a Sole Treatment Intervention for Post-Traumatic Stress Disorder in Southeast Asia

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Published online: 28 September 2018

Abstract

Southeast Asia contains high numbers of traumatised populations arising from either natural disasters or interpersonal violence. Consequently, the need for empirically based trauma treatments, compromised by insufficiency in appropriately trained clinicians and mental health workers, makes the situation more challenging in addressing traumatic sequelae in local populations. In response, the humanitarian/ trauma capacity building organisation, Trauma Aid Germany, trained 37 therapists in psycho-traumatology, based on EMDR Therapy, which included trauma stabilisation techniques. This research analyses the impact of Trauma Stabilisation as a sole treatment intervention for Post-Traumatic Stress Disorder (PTSD) in adults. Each client was screened for PTSD utilising the Harvard Trauma Questionnaire - pre- and post-treatment. Analysis of the data considered only those interventions focussed on trauma stabilisation, including psychoeducation. Participants receiving trauma confrontation interventions were excluded from the data. Trauma stabilisation - as a sole treatment intervention, was highly effective in alleviating PTSD diagnoses. Results demonstrate PTSD symptoms were reduced in both clinical and sub-clinical trauma groups. The data set suggests trauma stabilisation, as a sole treatment intervention, was safe, effective, efficient and sufficient treatment intervention for PTSD. Furthermore, trauma stabilisation interventions have the advantage of being safe, flexible, and adaptable to the cultural and spiritual context in which they were are applied. The research findings also have implications regarding teaching and learning and the potential utilisation of paraprofessionals, and other allied health professionals in addressing the global burden of psychological trauma.

Keywords Post-traumatic stress disorder · Trauma stabilisation · Cultural sensitivity · Southeast Asia · Therapeutic relationship · Trauma capacity building

Introduction

The Centre for Research on Epidemiology of Disasters (CRED) [1] reports, Asia as the continent most affected by natural disasters (44.4%), has the most disaster victims (69.5%)

Multiple use of data collected from the same sample
This is the first paper about the Mekong I data, which is a huge data set of 4799 clients with over 2000 variables, therefore the paper is only a partial analysis of the data. Several other analysis and publication will follow that can then refer to this paper for additional information. The sociographic information, several steps of inclusion and the high number of reported variables took a lot of space in the text as well as in forms of tables.

and suffers the most damage (64.4% of worldwide natural disasters reported costs). An example of this relates to the Indian Ocean Tsunami in Southeast Asia in December 2004. This natural disaster was responsible for the death of 225,841 people making it the sixth deadliest natural disaster in the world [2]. Post tsunami a survey by Souza et al. [3] carried out in Aceh Province, Indonesia, determined that 83.6% of survivors demonstrated signs of emotional distress and 77.1% depressive symptoms. Levels of emotional distress increased the more individuals were exposed to tsunami-related deaths among household members. Further post-tsunami studies highlight a prevalence of Posttraumatic Stress Disorder (PTSD) - 8.6 to 57.3% among Asian survivors of natural disasters. However, this was highly dependent upon methodologies and diagnostic instruments used [2, 4].

Apart from natural disasters Southeast Asian populations suffers from various kinds of interpersonal violence. The World Report on Violence and Health [5] defines violence as "The intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, mal-development or deprivation" (p. 5). It further distinguishes self-directed (inflicted upon person him−/herself), interpersonal (inflicted by another individual/small group of individuals) and collective violence (inflicted by larger groups such as organized political groups and terrorist organisation). Interpersonal violence is divided into family and intimate partner violence (usually taking place in the home, also called domestic violence) and community violence (violence between unrelated individuals, generally taking place outside the home) [5].

An example of violence being perpetrated on an industrial scale relates to the acts of genocide, perpetrated by the Khmer Rouge regime in Cambodia between 1975 and 1979. Kiernan [6] estimates that this brutal regime was responsible for the deaths of between 1.67 and 1.87 million people – some 20% of the entire Cambodian population. The Khmer Rouge were responsible for mass executions, persecution and perpetrating a regime of terror. As van Schaack et al. [7] purports, the impact on mental health continues to be in evidence with regard to PTSD, Depression and other severe mental health problems.

Availability of Therapists and Mental Health Services

At the same time, the education of the therapists in the region is quite poor and Mental Health services are very rare. In Cambodia there are 63 mental health outpatient facilities resulting in a rate of 0.42 per 100.000 people [8] and in Thailand 93 mental health outpatient facilities resulting in a rate of 0.14 per 100.000 people [9]. For comparison, in Germany there are 24.881 mental health outpatient facilities resulting in a rate of 30.32 per 100.000 people [10]. For Indonesia there is no information available [11].

Trauma Aid and the Mekong I Project

Trauma Aid (HAP Germany) is a Humanitarian Organisation, which aims to establish Trauma Capacity Building/ Psychotherapeutic services in crisis areas like Southeast Asia [12]. The primary objective of the organisation is to train local health workers and non-governmental organisations (NGOs), in psycho-traumatology, trauma interventions, and trauma self-care based upon empirical research and international treatment guidelines [13–15]. Within the

Trauma Aid Germany portfolio was the establishment of Mekong I [2010–2014] – a Trauma Capacity Building Project for Thailand, Cambodia and Indonesia - Mekong I was funded by the German Federal Ministry for Economic Cooperation and Development – also in co-operation with Terre des Hommes (Germany). One of the primary objectives is to increase the mental health – trauma capacity focussed on evidence-based treatment interventions. This involved teaching and learning, skills training, diagnostic screening, and trauma treatment interventions which included psychoeducation, stabilisation techniques and trauma processing (confrontation) mainly centred upon Eye Movement Desensitization and Reprocessing (EMDR) Therapy and empirically validated psychological treatment intervention for PTSD. There were 37 therapists trained during Mekong I of which their professional background was that they are either psychologists or psychiatrists.

Rationale for the Research

Herman [16] presented a model of trauma treatment that involves three critical phases:

Phase 1: Safety and Stabilisation
Phase 2: Remembering and Mourning
Phase 3: Reconnection

This three-phases approach is the recommended approach for treating PTSD, especially complex cases – as recommended by the current ISTSS guidelines for the treatment of complex PTSD [17]. Phase 1 – Safety & Stabilisation, focuses on the insurance of the individual's safety, reducing symptoms and increasing emotional, social and psychological competences. It also includes psychoeducation providing explanations to account for client's symptoms and experiences, giving the client hope for resolution and providing, when appropriate, a sense of normalisation when necessary. Phase 2 – Remembering & Mourning is ostensibly a 'Trauma Confrontation' phase, focuses on the processing of unresolved aspects of the individual's memory of the adverse life (traumatic) experience. WHO [18] empirically supports Trauma-Focused Cognitive Behavior Therapy (TF-CBT) and EMDR therapy as efficacious treatments for PTSD. Phase 3 – Re-connection, focuses on the consolidation of change and moving forward [16, 19, 20]. There is evidence that the therapeutic alliance and negative mood regulation achieved in Phase 1 predicts the success in reducing PTSD in Phase 2 emphasising the value of establishing a strong therapeutic relationship and emotion regulation skills before exposure work, especially among chronic PTSD populations [21–23]. This fits with Asay and Lambert [24], Lambert [25] who indicate that the therapeutic relationship accounts for 30% of client improvement (while specific techniques account for only 15%).

Stabilisation

The Mekong I project wanted to consider the impact of trauma stabilisation as a sole treatment intervention in PTSD. Research supports the activation of the patients' strength and resources as an important change mechanism in psychotherapy [26–28]. Especially with complex cases following childhood abuse, trauma-specific stabilisation

has a prominent role. It includes techniques of attention focussing, the focussed use of imaginative distancing techniques and the use of resource activating techniques [20, 29]. Techniques of attention refocusing direct the attention away from the traumatic internal experience toward stimuli of the external (neutral or positive) reality resulting in reduced distress. Imaginative distance techniques aim at reducing the traumatic affect and enhancing the feeling of control and safety. Imagination techniques well established are the "container" technique and the "safe place" exercise [20, 29, 30]. Resource activation is based on the principle that positive emotions can reduce the impact of negative emotions [31]. Resources can be activated through imagination evoking positive memories, personal successes, positive relationships and role models [29, 32, 33].

The content of trauma stabilisation taught in the Mekong I training is included in the treatment manual of Wöller and H. Mattheß [34] for Resource-oriented trauma therapy combined with EMDR resource installation - ROTATE. ROTATE itself is not a form of Trauma Stabilisation – but instead a manualisation of a multitude of different trauma stabilisation techniques, strategies and interventions. The approach aims at strengthening resilience and coping capacities by activating positive personal resources within a secure therapeutic relationship. A variety of imaginative resource-activating methods are included and based on a framework informed by affective neuroscience, resilience research, and attachment theory. Despite containing EMDR therapy elements and techniques, trauma stabilisation does not involve trauma confrontation or working with trauma memories. A RCT has shown that symptoms of PTSD and other co-morbid trauma-related symptoms (like depression and anxiety) can be effectively reduced using trauma stabilisation interventions as outlined in the ROTATE manual and subsequent research study [35, 36].

There are distinct advantages to trauma stabilisation as a specific intervention - it can be safely applied, is language-independent and flexible and thus culturally adaptable and especially suitable for clients in non-Western societies. One important aspect for its use in the Mekong I Project is the ease in which knowledge transfer – training, can take place. This enables broad dissemination in severely affected countries and the treatment of a vast number of trauma survivors in a safe and effective way.

Lack of Research about Stabilisation

Even though Phase Orientated approaches and stabilisation techniques are commonly used in therapeutic practise and recommended by the ISTSS guidelines for the treatment of complex PTSD, there is still a lack of evidence, especially compared to the numerous studies supporting evidence of TF-CBT and EMDR therapy. A group of researchers and therapists even published a letter to the editor criticizing the current ISTSS guideline that recommends the phase orientated approach [37]. They argue that with the lack of evidence of the effectiveness of a preparation/stabilisation phase and the effectiveness of stabilisation in general, only the evidence-based trauma focussed approaches like TF-CBT and EMDR therapy should be recommended in the guideline instead of a phase orientated approach.

The frequent use of trauma stabilisation techniques in the Mekong Project I, as this paper will demonstrate, offers the opportunity to better understand the impact of this approach as a distinct intervention, in an area where there is a scarcity of research data currently.

Method

The Data of the Mekong I Project

Therapists and Data Collection of the Mekong I Project

During the Mekong I Project 37 therapists were trained - 9 from Cambodia, 12 Indonesia, 16 Thailand. The professional background of the trainees – 32 Psychologists and 5 Psychiatrists, all of which had previous psychotherapy training. Of the 37 trainees – 21 Female, 16 Male with an age range of 29 to 62 years, and mean age of 39.48 years. All the therapists where trained on a pro-bono basis. However, a condition was that they were expected to offer pro-bono treatment back in their respective communities and collect clinical data about their clients. Data collection also included psychometrics and diagnostic measures. Ethical approval for the study was granted from the University of Worcester (UK) and was adopted in each the three countries.

The clients were service seekers that voluntarily participated in trauma treatment with each informed about the possibility to withdraw their participation at any point – in accordance with ethics approval guidelines. All in all, the demographic, diagnostic and treatment data of 4799 clients was documented and subsequently analysed. Data was first collected, via Microsoft Excel Spread Sheet, in each country for the period of the research study – 3 years, before being put together into a collective data set ready for retrospective analysis using post inclusion criteria.

Data of Clients of the Mekong I Project

The centralised Microsoft Excel sheet was transferred into SPSS version 24. The recorded data of the 37 therapists had to be further structured and partly recorded to create a coherent data file. The result is that this large data set contained over 2000 variables of the 4799 clients who took part in the study. The profiles were as follows:

Indonesia ($n = 2363$, 49.2%), Cambodia ($n = 1483$, 30.9%), Thailand ($n = 953$, 19.9%)
Type of Client: Adults ($n = 2561$, 53.4%), Children/adolescents ($n = 2238$, 46.6%)
Female ($n = 2709$, 56.4%), Male clients ($n = 2057$, 42.9%), Transsexual ($n = 33$, 0.7%)

Preparation of the Data

Most of the data collected is mainly quantitative, however some qualitative material was also captured. About 30% of the qualitative data was documented using indigenous language of the respective therapists and then translated by local linguistic experts into English. The qualitative data was analysed using a program for semantic analysis by Braun and Clarke [38]. However only the quantitative date will be presented in this paper.

Analysis for this Article

The Included Data -Inclusion Criteria and Description of Clients and Therapies

This article will only present the research findings from the adult client group who received trauma stabilisation interventions only. For the study, Trauma Stabilisation is defined as

including resource interventions, grounding techniques, comprehensive history taking, trauma preparation, trauma mapping, trauma case conceptualisation and psychoeducation. For each client each therapist wrote a treatment outline containing the number of sessions, setting and duration of each session and indicated the specific stabilisation/confrontation interventions used each session. Only clients were included in the analysis who had received no trauma confrontation intervention at all and at least one intervention of trauma stabilisation. Treatment fidelity was ensured through active clinical supervision and through supervised self-experience teaching and learning sessions. Using this inclusion criteria data was analysed from $n = 1358$ clients. For detailed demographic information please view Table 1. The mean amount of sessions, for the included data, was 4.31 (SD 3.24, range 1 to 23). The mean number of Trauma Stabilisation interventions used was 7.23 (SD 5.50, range 1 to 64).

Diagnostic Measures

Two specific psychometrics were used. For assessing the PTSD diagnosis, the Harvard Trauma Questionnaire (HTQ - 40 items), and the Hopkins Symptoms Checklist Questionnaire (HSCL-25 items) to assess Anxiety and Depression – pre- and post-treatment. Both scales were

Table 1 Demographic information and number of sessions/trauma stabilisation techniques for the clients of the whole data set and respectively for all steps of inclusion

Characteristics		Inclusion criteria for analysis			
	Mekong data	only adults, stabilisation	Pre- & post-measures	HTQ diagnosis pre-treatment	
				DSM-V PTSD	ICD-11 PTSD
N	4799 (100%)	1358 (100%)	365 (100%)	197 (100%)	164 (100%)
Country					
Cambodia	1483 (30.9%)	654 (48.2%)	206 (56.4%)	128 (65.0%)	98 (59.8%)
Indonesia	2363 (49.2%)	414 (30.5%)	78 (21.4%)	46 (23.4%)	46 (28.0%)
Thailand	953 (19.9%)	290 (21.4%)	81 (22.2%)	23 (11.7%)	20 (12.2%)
Client group					
Adults	2561 (53.4%)	1358 (100%)	365 (100%)	197 (100%)	164 (100%)
Children/Adolescents	2238 (46.6%)	–	–	–	–
Gender					
Male	2057 (42.9%)	468 (34.5%)	103 (28.2%)	46 (23.4%)	37 (22.6%)
Female	2709 (56.4%)	885 (65.2%)	258 (70.7%)	147 (74.6%)	124 (75.6%)
Transgender	33 (0.7%)	5 (0.4%)	4 (1.1%)	4 (2.0%)	3 (1.8%)
Age (Mean (SD))	23.39 (12.27)	31.69 (12.02)	34.52 (11.81)	36.76 (11.01)	36.93 (11.07)
Therapies					
Mean N Sessions (SD)	5.4 (4.7)	4.3 (3.2)	6.7 (3.9)	7.7 (3.9)	7.6 (3.8)
Mean N stabilisation techniques (SD)	9.5 (10.1)	7.2 (5.5)	11.0 (6.6)	10.5 (7.1)	10.0 (6.9)

deliberately chosen as they have international application and validation and were subsequently available in language versions in Indonesian, Khmer and Thai. Therapists were trained and data evaluated strictly according to the manual "Measuring Trauma Measuring Torture" of the Harvard Program in Refugee Trauma [39]. Internal consistency (Cronbach's α) at baseline of the HTQ and HSCL-25 in our sample was 0.96 and 0.94, respectively.

Description of Reported Results

The publication focuses on PTSD diagnosis in adult populations in Indonesia, Cambodia and Thailand however, further outcomes will be reported in future publications.

For the clients, the following results can be reported:

Remission rates: the PTSD scale of the HTQ includes items reflecting the DSM-V and the forthcoming ICD-11 criteria for PTSD. A study investigating the impact of the changes to diagnostic criteria for PTSD in DSM-V and the proposed changes in ICD-11 found that while there is an overlap of the PTSD diagnoses, each identified a proportion of people with PTSD which the other system did not [40]. As different study populations are defined depending on the diagnostic classification system used, the comparison between studies using different diagnosis systems is hindered. To prevent this, for the analysis DSM-V as well as ICD-11 PTSD is reported. PTSD status was calculated using the item mapping for the DSM-V and ICD-11 models of PTSD as suggested by Hyland [41]. Criterion A (traumatic stressor) was established with a detailed list of Traumatic Events. PTSD is diagnosed via DSM-V criteria when a client scores 3 or 4 in (i) at least one of the four intrusion symptoms (criterion B), (ii) at least one of the two avoidance symptoms (criterion C), (iii) at least two of the seven symptoms regarding negative alternations in cognitions and mood (abbreviation Neg.Alt. Cogn&Mood, criterion D), and (iv) at least two of the five arousal symptoms (criterion E). PTSD is diagnosed via the purposed ICD-11 criteria when a client scores 3 or 4 in (i) at least one of the two re-experiencing symptoms, (ii) at least one of the two sense of threat symptoms and (iii) at least one of the two avoidance symptoms. Patients no longer fulling these criteria of DSM-V/ICD-11 PTSD after treatment are regarded as remitted.

Additionally to Remission rates, the Treatment Effect can also be reported. The treatment effect is calculated as the reduction of fulfilled PTSD diagnoses and the syndrome reduction due to Trauma Stabilisation interventions. Only clients were included in the analysis, where pre- and post-measures of the HTQ were available, 365 clients meet this criterion.

Inclusion Criterium Fulfilment of PTSD Diagnosis

Pre-treatment 197 (54.0%) of the included 365 clients fulfilled the diagnosis of PTSD via DSM-V criteria and 164 (44.9%) via ICD-11 criteria. For the following analysis, only clients fulfilling the respective diagnosis pre-treatment (DSM-V/ICD-11 PTSD) are included. In a later section, the results for all included clients regardless their PTSD diagnosis pre-treatment are reported.

Statistical Analysis

All data was analysed using SPSS version 24. The pairwise comparisons of the binary variables (remission rates of PTSD diagnoses and PTSD criteria) were calculated via

McNemar tests. The pairwise comparisons of the number of criteria fulfilled of the respective PTSD diagnoses and of the number of symptoms of the respective DSM-V/ICD-11 criteria were calculated via dependent t-tests. From the t-tests the effect sizes *r* and Hedges's g_{av} were calculated. As Cohen's d_z is argued to be an overestimation of the effect size of correlated samples and Cohen's d_{av} is positively biased, Hedges's corrected and recommended Hedges's g_{av} is reported [42, 43]. The *a*-level was Bonferroni adjusted for multiple comparisons ($a = 0.0028 = 0.05/18$; PTSD DSM-V and ICD-11 remission, Number of criteria of DSM-V and of ICD-11 PTSD, criteria of PTSD (DSM-V: Intrusions, Neg.Alt. Cogn&Mood, Arousal, Avoidance, ICD-11: Re-experiencing, Sense of threat, Avoidance), Number of symptoms of the five DSM-V and the four ICD-11 PTSD criteria).

Results

PTSD remission rates were 91.4% for DSM-V and 93.3% for ICD-11 diagnosis as highlighted in Fig. 1. McNemar tests determined that there was a significant difference of the proportion of clients with PTSD diagnosis pre- and post-treatment, ($p < 0.00001$ for both DSM-V and ICD-11 PTSD), for further details please view Table 2. On average, clients fulfilled less DSM-V PTSD criteria after the trauma stabilisation treatment ($M = 1.97$, $SD = 1.387$) than before treatment ($M = 5.0$, $SD = 0.00$). This difference, 3.025, BCa 95% CI [2.831, 3.220] was significant $t(196) = 30.623$, $p < 0.001$, and represented a large-sized effect, $r = 0.910$ and Hedges's $g_{av} = 3.078$, BCa 95% CI [2.875, 3.271]. The CL effect size indicates that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment is 98.6%. On average, clients fulfilled less ICD-11 PTSD criteria after treatment ($M = 1.54$, $SD = 0.92$) than before treatment ($M = 4.0$, $SE = 0.00$). This difference, 2.457, BCa 95% CI [2.316, 2.599] was significant $t(163) = 34.363$, $p < 0.001$, and represented a large-sized effect, $r = 0.937$ and Hedges's $g_{av} = 3.780$, BCa 95% CI [3.559, 3.994]. The CL effect size

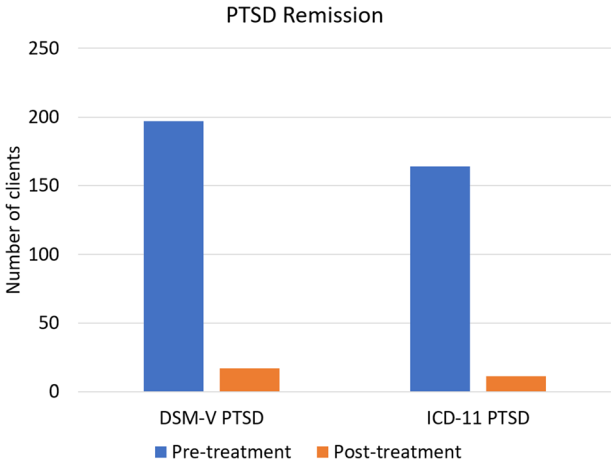


Fig. 1 PTSD Remission after trauma stabilisation treatment: Number of clients fulfilling DSM-V/ICD-11 PTSD prior treatment (blue/black bars) and Number of clients still fulfilling DSM-V/ICD-11 PTSD after treatment (orange/ grey bars)

Table 2 Remission of (respective DSM-V/ICD-11) PTSD diagnosis after trauma stabilisation treatment

HTQ	Number of participants		Remission rate	McNemar test	
	Pre-treatment	Post-treatment		Test statistic[a]	*p*
DSM-V PTSD	197 (100%)	17 (8.6%)	91.4%	178.006	< 0.00001
ICD-11 PTSD	164 (100%)	11 (6.7%)	93.3%	151.007	< 0.00001

[a] Chi-Square, continuity corrected

indicates that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment is 99.6%. These results are also highlighted in Table 3.

Remission rates for DSM-V PTSD and ICD-11 PTSD criteria ranged between 72.1 and 86.0%. McNemar tests determined that there were significant differences of the proportion of clients with PTSD criteria pre- and post-treatment, ($p < 0.00001$ for both DSM-V and ICD-11 PTSD criteria), for further details please view Table 4. On average, clients fulfilled less symptoms of the respective DSM-V PTSD criteria after treatment than before treatment. These differences were significant ($p < 0.001$ for all symptom criteria) and represented large-sized effects (r between 0.865 and 0.914 and Hedges's g_{av} between 2.50 and 3.09), for further details please view Table 5. The CL effect sizes indicate that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment in symptoms of DSM-V PTSD intrusions is 95.8%, in symptoms of DSM-V PTSD Neg. Alt. Cogn&Mood 97.8%, in symptoms of DSM-V PTSD Arousal 98.8%. and in symptoms of DSM-V PTSD Avoidance 97.9%. On average, clients fulfilled less symptoms of the respective ICD-11 PTSD criteria after treatment than before treatment. These differences were significant ($p < 0.001$ for all symptom criteria) and represented large-sized effects (r between 0.892 and 0.910 and Hedges's g_{av} between 2.88 and 3.25). The CL effect sizes indicate that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment in symptoms of ICD-11 PTSD re-experiencing is 98.5%, in symptoms of ICD-11 PTSD Sense of threat 97.6% and in symptoms of ICD-11 PTSD Avoidance 98.6%.

Results for the Entire Range of PTS Problems

Additionally to the treatment effect of clients with DSM-V/ICD-11 PTSD pre-treatment, the treatment effect for subclinical clients and 'normal' scoring clients can be reported. In this section, the analysis of all included clients (365 clients) regardless of their diagnosis before treatment is reported. The distribution of fulfilled PTSD criteria pre- and post-treatment is shown in Figs. 2 and 3.

Table 3 Remission of PTSD criteria after trauma stabilisation treatment: Pairwise comparisons of the mean number of fulfilled criteria for DSM-V/ICD-11 PTSD before and after the trauma stabilisation treatment for clients fulfilling DSM-V/ICD-11 PTSD pre-treatment

HTQ	Raw mean (standard deviation)		*t*	Bootstrap *p*	Hedges's g_{av}[a] [BCa 95% CI]	*r*
Nr. Of criteria	Pre-treatment	Post-treatment				
DSM-V PTSD[b]	5.00 (0.0)	1.97 (1.387)	30.623	< 0.001	3.08 [2.88, 3.27]	0.910
ICD-11 PTSD[c]	4.00 (0.0)	1.54 (0.916)	34.363	< 0.001	3.78 [3.56, 3.99]	0.937

[a] Hedges's g_{av} is used as a corrected effect size to the biased Cohen's d_{av}, [b] 0–5 criteria of DSM-V PTSD possible, [c] 0–4 criteria of ICD-11 PTSD possible

Table 4 Remission of DSM-V and of ICD-11 PTSD criteria after the trauma stabilisation treatment for clients fulfilling DSM-V/ICD-11 PTSD pre-treatment

HTQ	Number of participants		Remission rate	McNemar test	
	Pre-treatment	Post-treatment		Test statistic[a]	*p*
DSM-V PTSD[b]					
Intrusions	197 (100%)	50 (25.4%)	74.6%	145.007	< 0.00001
Neg.Alt. cogn.&mood	197 (100%)	46 (23.4%)	76.6%	149.007	< 0.00001
Arousal	197 (100%)	55 (27.9%)	72.1%	140.007	< 0.00001
Avoidance	197 (100%)	41 (20.8%)	79.2%	154.006	< 0.00001
ICD-11 PTSD[b]					
Re-experiencing	164 (100%)	23 (14.0%)	86.0%	139.007	< 0.00001
Sense of threat	164 (100%)	34 (20.7%)	79.3%	128.008	< 0.00001
Avoidance	164 (100%)	32 (19.5%)	80.5%	130.008	< 0.00001

[a] Chi-Square, continuity corrected

[b] except criterion A where no remission is possible

Six clients (1.6%) did not fulfil any criteria of DSM-V PTSD. For 83.3% this didn't change after treatment, 1 client was subclinical after treatment (fulfilling three criteria of DSM-V PTSD). 162 clients (44.4%) were subclinical prior treatment fulfilling one (28 clients), two (38 clients), three (38 clients) or four (58 clients) DSM-V PTSD criteria prior treatment. For this subclinical client group 12 clients remised (no criteria of DSM-V PTSD after treatment), 104 clients (64.2%) improved, but stayed subclinical, 7 clients (4.3%) deteriorated, but remained subclinical and 5 clients (3.1%) were diagnosed with DSM-V PTSD still after treatment.

6 clients (1.6%) did not fulfil any criteria of ICD-11 PTSD pre-treatment. For all 6 clients, this didn't change after treatment. 195 clients (53.2%) were subclinical prior treatment fulfilling one (59 clients), two (54 clients) or three (82 clients) ICD-11 PTSD criteria prior treatment. For

Table 5 Remission from PTSD symptoms after trauma stabilisation treatment: Pairwise comparisons of the mean number of fulfilled symptoms of the respective criterium for DSM-V/ICD-11 PTSD pre- and post-treatment for clients fulfilling DSM-V/ICD-11 PTSD pre-treatment

HTQ	Raw mean (standard deviation)		*t*	Bootstrap *p*	Hedges's g_{av}[a] [BCa 95% CI]	*r*
Nr. Of symptoms	Pre-treatment	Post-treatment				
DSM-V PTSD						
Intrusions[b]	3.06 (1.041)	0.50 (1.003)	24.171	< 0.001	2.50 [2.30, 2.68]	0.865
Neg.Alt. cogn.&mood[c]	5.17 (1.575)	0.86 (1.515)	28.285	< 0.001	2.78 [2.58, 3.00]	0.896
Arousal[d]	4.335 (0.886)	0.93 (1.272)	31.568	< 0.001	3.09 [2.91, 3.28]	0.914
Avoidance[e]	1.79 (0.407)	0.28 (0.598)	28.607	< 0.001	2.94 [2.75, 3.11]	0.900
ICD-11 PTSD						
Re-experiencing[f]	1.59 (0.493)	0.18 (0.469)	27.683	< 0.001	2.92 [2.72, 3.13]	0.908
Sense of threat[f]	1.77 (0.423)	0.28 (0.592)	25.228	< 0.001	2.88 [2.67, 3.09]	0.892
Avoidance[e]	1.84 (0.372)	0.26 (0.574)	28.044	< 0.001	3.25 [3.00, 3.48]	0.910

[a] Hedges's g_{av} is a corrected effect size to the biased Cohen's d_{av}

[b] 0–4 symptoms of DSM-V PTSD Intrusions possible

[c] 0–8 symptoms of DSM-V PTSD Neg.Alt. Cogn&Mood possible

[d] 0–5 symptoms of DSM-V PTSD Arousal possible

[e] 0–2 symptoms of DSM-V/ICD-11 PTSD Avoidance possible

[f] 0–2 symptoms of ICD-11 PTSD Re-experiencing/Sense of threat possible

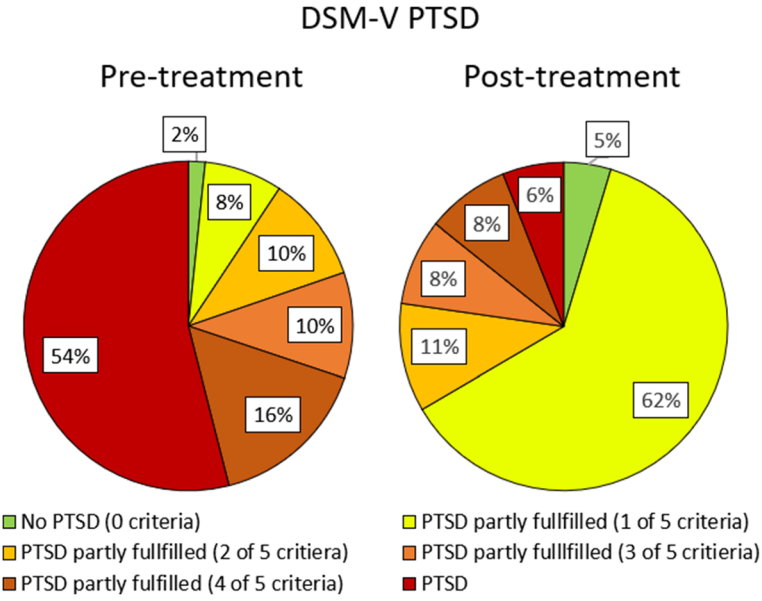


Fig. 2 Distribution of clients fulfilling DSM-V PTSD (black/red), partly fulfilling PTSD (1 up to 4 of 5 DSM-V PTSD criteria fulfilled, 4 ranges of grey/orange, lighter colours represent-ing less criteria), no PTSD (white/green) before and after trauma stabilisation treatment

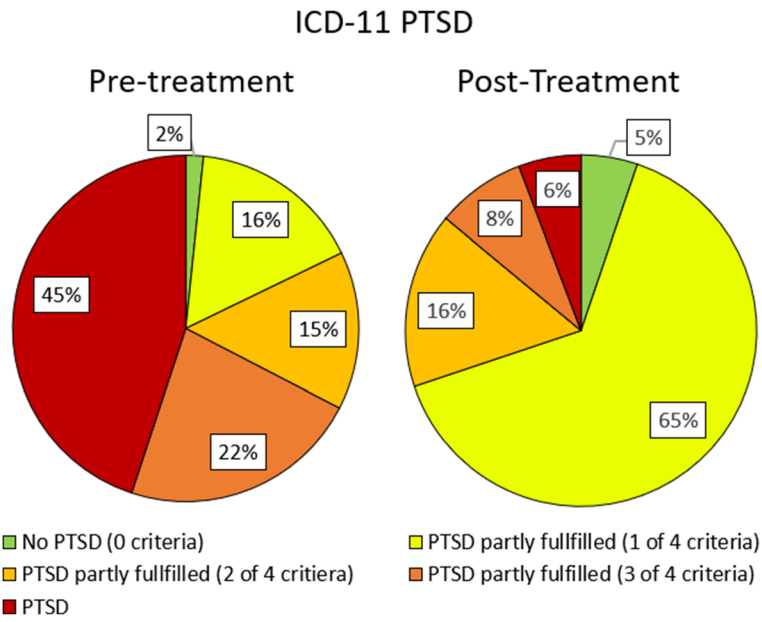


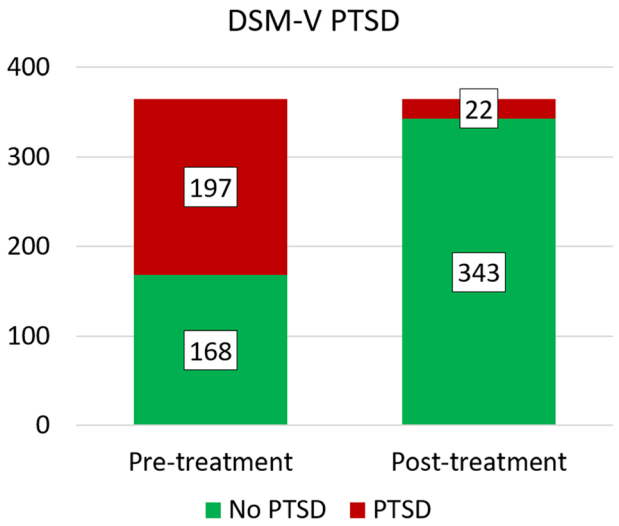
Fig. 3 Distribution of clients fulfilling ICD-11 PTSD (red/dark grey), partly fulfilling PTSD (1 (orange/grey) or 2 (yellow/light grey) of 3 ICD-11 PTSD criteria fulfilled), no PTSD (green/white) before and after trauma stabilisation treatment

this subclinical client group 13 clients remised (no criteria of ICD-11 PTSD after treatment), 93 clients (47.7%) improved, but stayed subclinical, 9 clients (4.6%) deteriorated, but remained subclinical and 10 clients (5.1%) were diagnosed with ICD-11 PTSD after treatment.

With 5 clients developing DSM-V PTSD and 10 clients developing ICD-11 PTSD during the time of the treatment, the analysed treatment effect might change, if the whole client group (no, subclinical, clinical PTSD) is included. Thus, all analyses were calculated once more with all clients regardless their diagnoses pre-treatment ($N = 365$ clients). The proportion of clients with and without PTSD before and after treatment is highlighted in Figs. 4 and 5.

McNemar tests determined that there was a significant difference of the proportion of clients with PTSD diagnosis pre- and post-treatment, ($p < 0.00001$ for both DSM-V and ICD-11 PTSD). The odd of remission of DSM-V PTSD is 36 times greater than the risk of getting the DSM-V PTSD diagnoses after treatment. The odd of remission of ICD-11 PTSD is 15.3 times greater than the risk of getting the ICD-11 PTSD diagnoses after treatment. For further details please view Table 6. On average, clients fulfilled less DSM-V PTSD criteria after treatment ($M = 1.72$, $SD = 1.299$) than before treatment ($M = 3.93$, $SD = 1.419$). This difference, 2.214, BCa 95% CI [2.046, 2.384] was significant $t(364) = 25.474$, $p < 0.001$, and represented a large-sized effect, $r = 0.800$ and Hedges's $g_{av} = 1.621$, BCa 95% CI [1.499, 1.750]. The CL effect size indicates that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment is 90.8%. On average, clients fulfilled less ICD-11 PTSD criteria after treatment ($M = 1.45$, $SD = 0.93$) than before treatment ($M = 2.93$, $SD = 1.18$). This difference, 1.48, BCa 95% CI [1.354, 1.611] was significant $t(365) = 20.948$, $p < 0.001$, and represented a large-sized effect, $r = 0.739$ and Hedges's $g_{av} = 1.391$, BCa 95% CI [1.273, 1.515]. The CL effect size indicates that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment is 86.3%. These results are also shown in Table 7.

On average, clients fulfilled less symptoms of the respective DSM-V PTSD criteria after treatment than before treatment. These differences were significant ($p < 0.00001$ for all symptom criteria) and represented large-sized effects (r between 0.672 and 0.789 and Hedges's g_{av} between



Figs 4 and 5 Proportion of clients with PTSD (red/black) and without PTSD (green/grey) diagnoses before and after trauma stabilisation for DSM-V and ICD-11 PTSD respectively

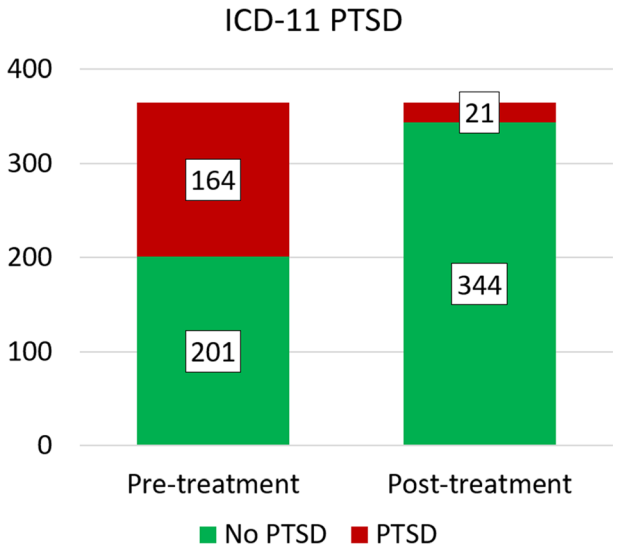


Fig. 4 and 5 (continued)

1.25 and 1.63). The CL effect sizes indicate that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment in symptoms of DSM-V PTSD intrusions is 87.1%, in symptoms of DSM-V PTSD Neg.Alt. Cogn&Mood 86.2%, in symptoms of DSM-V PTSD Arousal 90.0%. and in symptoms of DSM-V PTSD Avoidance 81.9%. On average, clients fulfilled less symptoms of the respective ICD-11 PTSD criteria after treatment than before treatment. These differences were significant ($p < 0.001$ for all symptom criteria) and represented large-sized effects (r between 0.672 and 0.789 and Hedges's g_{av} between 1.25 and 1.63). The CL effect sizes indicate that after controlling for individual differences, the likelihood that a person scores higher pre-treatment than after treatment in symptoms of ICD-11 PTSD re-experiencing is 78.4%, in symptoms of ICD-11 PTSD Sense of threat 83.8%. and in symptoms of DSM-V PTSD Avoidance 81.8%. For detailed results please see Tables 8 and 9. The remission of PTSD criteria after treatment is highlighted in Figs. 6 and 7.

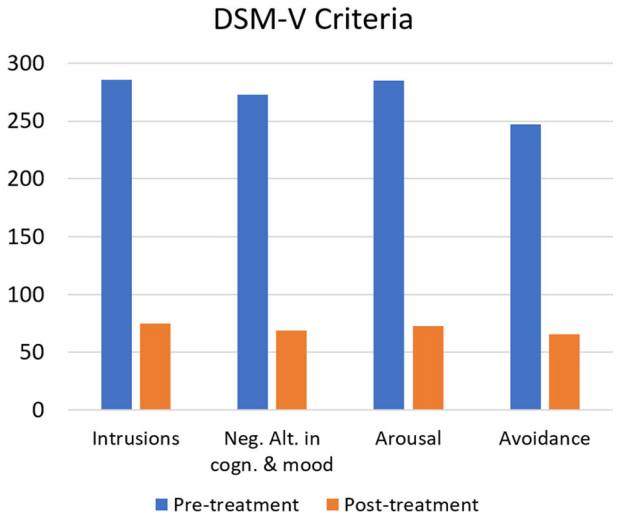
Comparison to Control Group

To put the extremely high effect sizes in relation, the treatment effect was compared to a control group from a previously published study [35, 36] and aggregated. This was possible

Table 6 Remission of PTSD diagnosis after trauma stabilisation treatment: Pairwise comparisons of PTSD diagnosis (via DSM-V and ICD-11 respectively) before and after trauma stabilisation treatment for all 365 clients

HTQ	Number of participants		McNemar		OR remission/ risk
	Pre-treatment	Post-treatment	Test statistic[a]	p	
DSM-V PTSD	197 (54.0%)	22 (6.0%)	163.654	< 0.00001	36
ICD-11 PTSD	164 (44.9%)	21 (5.8%)	123.706	< 0.00001	15.3

[a] Chi-Square, continuity corrected



Figs 6 and 7 Remission of PTSD criteria after trauma stabilisation treatment: Number of participants fulfilling DSM-V/ICD-11 PTSD criteria prior treatment (blue/black bars) and after treatment (orange/ grey bars)

because it involved the same therapists from the Mekong I Project and included the same diagnostic tools and psychometrics. This made for a suitable comparison. The control group consisted of 55 clients (38 female clients, mean age 25.64 (SD 9.425)) who were waiting 5 weeks between their first and second diagnostic assessment.

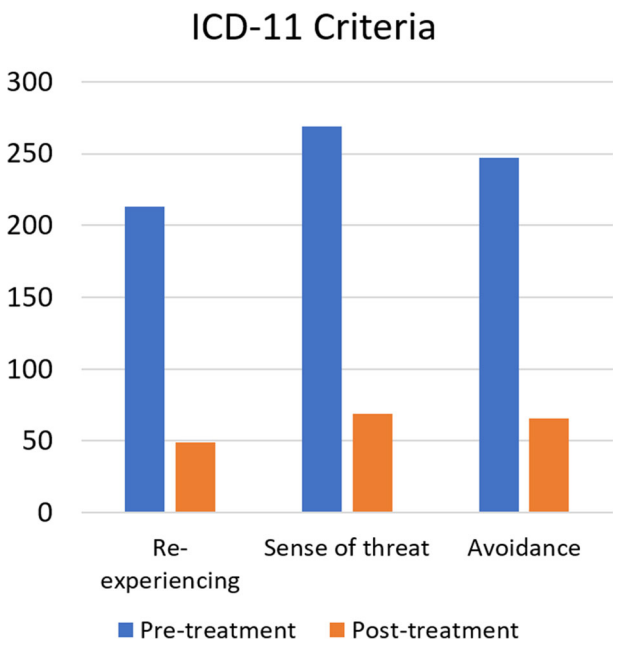


Fig. 6 and 7 (continued)

Table 7 Remission of PTSD criteria after trauma stabilisation treatment: Pairwise comparisons of the mean number of fulfilled DSM-V/ICD-11 PTSD criteria before and after trauma stabilisation treatment for all 365 clients

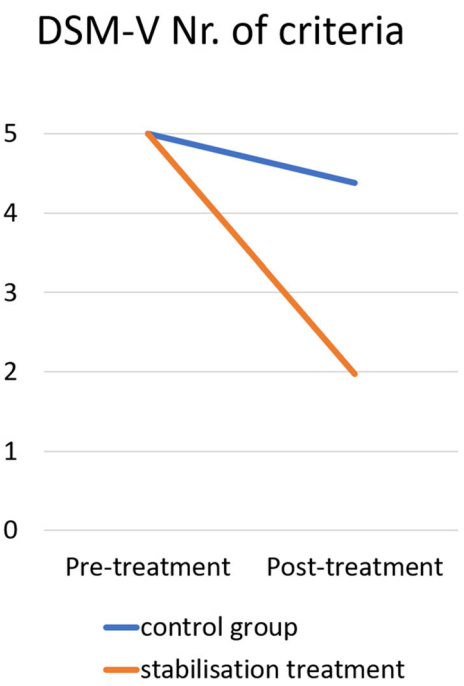
HTQ	Raw mean (standard deviation)		*t*	*p*	Hedges's g_{av}[a] [95% CI]	*r*
Nr. Of criteria	Pre-treatment	Post-treatment				
DSM-V PTSD[b]	3.93 (1.419)	1.72 (1.299)	25.474	< 0.001	1.62 [1.50, 1.75]	0.800
ICD-11 PTSD[c]	2.93 (1.179)	1.45 (0.929)	20.948	< 0.001	1.39 [1.27, 1.51]	0.739

[a] Hedges's g_{av} is used as a corrected effect size to the biased Cohen's d_{av}

[b] 0–5 criteria of DSM-V PTSD possible

[c] 0–3 criteria of ICD-11 PTSD possible

45 clients (81.8%) of the control group fulfilled the criteria for DSM-V PTSD before waiting. The remission rate was 24.4% (vs. 91.4% remission rate for stabilisation treatment group). On average, clients fulfilled less DSM-V PTSD criteria after waiting ($M = 4.38$, $SD = 1.451$) than before treatment ($M = 5.0$, $SE = 0.00$). This difference, 0.622, BCa 95% CI [0.282, 1.057] was significant $t(44) = 2.878$, $p = 0.025$, and represented a medium-sized effect, $r = 0.398$ and Hedges's $g_{av} = 0.594$, BCa 95% CI [0.270, 1.013]. The odd of remission of DSM-V PTSD is 2.2 times greater than the risk of getting the DSM-V PTSD diagnoses after waiting. 33 clients (60.0%) of the control group fulfilled the criteria for ICD-11 PTSD before waiting. The remission rate was 33.3% for the control group (vs. 93.3% remission rate for stabilisation treatment group). On average, clients fulfilled less ICD-11 PTSD criteria after waiting ($M = 3.33$, $SD =$



Figs 8 and 9 Remission of the five DSM-V PTSD criteria and of the four ICD-11 PTSD criteria for the trauma stabilisation treatment group (orange/grey line) and for the waiting-list control group (blue/black line)

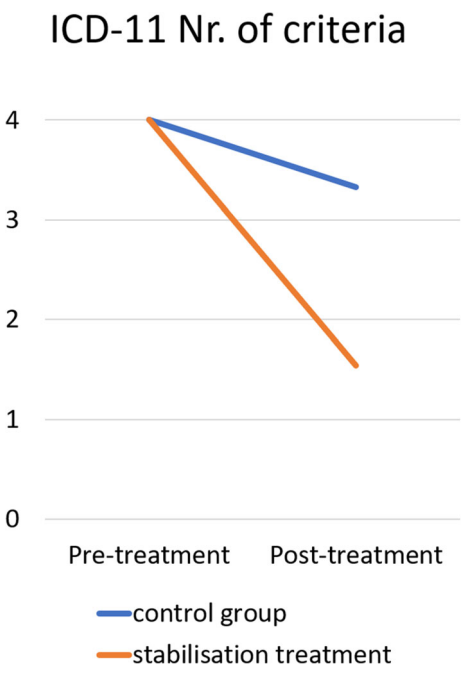


Fig. 8 and 9 (continued)

1.216) than before treatment ($M = 4.0$, $SE = 0.00$). This difference, 0.667, BCa 95% CI [0.321, 1.081] was significant $t(32) = 3.149$, $p = 0.025$, and represented a medium-sized effect, $r = 0.486$ and Hedges's $g_{av} = 0.761$, BCa 95% CI [0.365, 1.228]. The odd of remission of ICD-11 PTSD is 1.38 times greater than the risk of getting the ICD-11 PTSD diagnoses after waiting.

General linear model analyses were conducted to analyse differences in the PTSD remission between the two groups. There was a significant effect of the group, indicating that the remission of DSM-V PTSD criteria differs between stabilisation treatment and control group, $F(1, 240) = 108.156$, $p < 0.00001$, $r = 0.557$, $\eta_p^2 = 0.311$. This indicates that 31.03% of the

Table 8 Pairwise comparisons of DSM-V and ICD-11 PTSD criteria fulfilled before and after trauma stabilisation treatment for all 365 clients

HTQ	Number of participants		McNemar test		$OR_{remission/risk}$
	Pre-treatment	Post-treatment	Test statistic[a]	*P*	
DSM-V PTSD					
Intrusions	286 (78.4%)	75 (20.5%)	189.270	< 0.00001	20.18
Neg.Alt. cogn.&mood	273 (74.8%)	69 (18.9%)	189.032	< 0.00001	30.14
Arousal	285 (78.1%)	73 (20.0%)	202.368	< 0.00001	54
Avoidance	247 (67.7%)	66 (18.1%)	150.698	< 0.00001	11.65
ICD-11 PTSD					
Re-experiencing	213 (58.4%)	49 (13.4%)	130.240	< 0.00001	9.2
Sense of threat	265 (72.6%)	69 (18.9%)	176.042	< 0.00001	20.6
Avoidance	247 (67.7%)	66 (18.1%)	150.698	< 0.00001	11.65

[a] Chi-Square, continuity corrected

Table 9 Remission of PTSD symptoms after trauma stabilisation treatment: Pairwise comparisons of the mean number of fulfilled symptoms of the respective criterium for DSM-V/ICD-11 PTSD before and after trauma stabilisation treatment for all 365 clients

HTQ	Raw mean (standard deviation)		*t*	bootstrap *p*	Hedges's g_{av}[a] [BCa 95% CI]	*r*
Nr. Of symptoms	Pre-treatment	Post-treatment				
DSM-V PTSD						
Intrusions[b]	2.12 (1.512)	0.41 (0.932)	19.203	< 0.001	1.56 [1.40, 1.73]	0.709
Neg.Alt. cogn.&mood[c]	3.59 (2.411)	0.73 (1.414)	20.873	< 0.001	1.44 [1.30, 1.60]	0.738
Arousal[d]	3.145 (1.763)	0.72 (1.153)	24.489	< 0.001	1.63 [1.50, 1.76]	0.789
Avoidance[e]	1.16 (0.885)	0.24 (0.552)	17.356	< 0.001	1.25 [1.09, 1.39]	0.672
ICD-11 PTSD						
Re-experiencing[f]	0.89 (0.843)	0.16 (0.445)	14.900	< 0.001	1.08 [0.93, 1.20]	0.615
Sense of threat[f]	1.17 (0.831)	0.24 (0.543)	18.723	< 0.001	1.32 [1.18, 1.45]	0.700
Avoidance[e]	1.16 (0.885)	0.24 (0.552)	17.356	< 0.001	1.24 [1.10, 1.38]	0.672

[a] Hedges's g_{av} is a corrected effect size to the biased Cohen's d_{av}

[b] 0–4 symptoms of DSM-V PTSD Intrusions possible

[c] 0–8 symptoms of DSM-V PTSD Neg.Alt. Cogn&Mood possible

[d] 0–5 symptoms of DSM-V PTSD Arousal possible

[e] 0–2 symptoms of DSM-V/ICD-11 PTSD Avoidance possible

[f] 0–2 symptoms of ICD-11 PTSD Re-experiencing/Sense of threat possible

variance between DSM-V PTSD pre- and posttreatment is explained by the treatment. The number needed to treat is 1.49. There was a significant effect of the group, indicating that the remission of ICD-11 PTSD criteria differs between stabilisation treatment and control group, $F(1, 195) = 93.336$, $p < 0.00001$, $r = 0.569$, $\eta_p^2 = 0.324$. This indicates that 32.38% of the variance between ICD-11 PTSD pre- and posttreatment is explained by the treatment. The number needed to treat is 1.67. The differences in remission are highlighted in Figs. 8 and 9.

Discussion

Prevalence Rates

Prevalence of PTSD

To our knowledge, such a large group of clients with trauma-related disorders has never been scrutinised so thoroughly anywhere in one of the three countries or in Southeast-Asia as whole. There are very high levels of PTSD in this service-seeking group of the clients. In the client group fulfilling all inclusion criteria (only stabilisation/ psychoeducation, no confrontation, pre-and post-treatment measures) the prevalence rate of PTSD was 44.9% for ICD-11 PTSD (164 of 365 adults) and 54.0% for DSM-V PTSD (197 of 356 adults). In the client group fulfilling all inclusion criteria except post-diagnostic measures criteria (only stabilisation/ psychoeducation, no confrontation, pre-treatment measure) the prevalence rate was 42.4% for ICD-11 PTSD (382 of 901 adults) and 50.2% for DSM-V PTSD (452 of 901 adults). The prevalence of PTSD for all service-seeking adults with pre-treatment measurement is 47.5% for ICD-11 PTSD (947 of 1993 adults) and 54.8% for DSM-V PTSD (1092 of 1993 adults).

Prevalence of PTSD Syndrome Scales

The prevalence rates for the DSM-V and ICD-11 Syndrome Scales of the client group fulfilling all inclusion criteria (only stabilisation/psychoeducation, no confrontation, pre-post treatment measures) were high, ranging between 58.4 and 78.4%. The difference between the diagnosis via DSM-V and ICD-11 becomes apparent comparing the criteria, as 78.4% of the clients fulfilled the DSM-V criterium Intrusions whereas only 58.4% fulfilled the respective ICD-11 criterium Re-experiencing.

Subclinical and Non-clinical Group of Clients

The way the data aggregation was set up – providing therapy and collecting diagnostic and therapy data for all service-seeking clients without any screening for PTSD diagnosis before – gives the opportunity to explore the whole range of trauma populations in Indonesia, Cambodia and Thailand. Of the clients fulfilling all inclusion criteria, only 1.6% of the clients didn't fulfil any DSM-V PTSD criteria at all, 44.4% of the clients were subclinical for DSM-V PTSD. For ICD-11 PTSD the prevalence is similar, only 1.6% of the clients didn't fulfil any ICD-11 criteria, 53.2% of the clients were subclinical for ICD-11 PTSD.

Effectiveness of Trauma Stabilisation Treatment

Remission Rates

This study demonstrates a treatment effect of Trauma Stabilization in significantly reducing PTSD symptoms. Trauma Stabilisation treatment was associated with very high remission rates for PTSD (91.4% for DSM V, 93.3% for ICD-11). The remission rates were also high for all PTSD criteria (ranging between 72.1 and 86.0%). Thus, a symptom reduction was achieved for all criteria, showing Trauma Stabilisation treatment was effective in reducing core PTSD symptomatology - Intrusions/Re-experiencing, Arousal/Sense of threat, Neg.Alt.Cogn&mood and Avoidance Behaviour. This study demonstrates that Trauma Stabilisation is a therapeutic agent of change on the treatment of psychological trauma including PTSD.

Impact of Treatment on all Subgroups

The treatment effect impacted the whole range of post-traumatic stress symptoms. Subclinical clients also improved following Trauma Stabilisation Intervention in addition to those diagnosed with PTSD and severe PTSD.

Even if subclinical and non-clinical PTSD clients were included in the analysis, results suggest Trauma Stabilisation treatment still has a high treatment effect in reducing DSM-V PTSD (Hedges's $g_{av} = 1.62$) and ICD-11 PTSD (Hedges's $g_{av} = 1.39$) as well as in reducing all PTSD criteria (Hedges's g_{av} ranging between 1.08 and 1.63).

It is an advantage of the data aggregation of the Mekong I project that non-clinical and subclinical groups can be included in the analysis. In contrast to clients fulfilling the PTSD diagnoses pre-treatment, subclinical and non-clinical clients can get worse during the time of the treatment, fulfilling the diagnosis after treatment. This important information can't be given in studies that screen for the diagnosis pre-treatment and only include clinical clients in the

study. The rate of clients developing the diagnosis during treatment was low (1.4% of the clients developed DSM-V PTSD, 2.7% of the clients developed ICD-11 PTSD.

When looking at the tables of remission of PTSD criteria (Tables 8 and 9), it should be noted that a complete remission from all criteria is not possible for PTSD clients. There is no possibility to remise from criterion A, thus, the rate of complete remission was 0%.

High Effect Sizes Regardless of Diagnostic System

Regardless of the diagnoses of PTSD via DSM-IV, DSM-V or ICD-11, a very high rate of traumatized adults lost their diagnoses of PTSD after Trauma Stabilisation treatment. We can report very high effect sizes for the treatment effect on the PTSD diagnosis via DSM-V (Hedges's $g_{av} = 3.08$) or ICD-11 (Hedges's $g_{av} = 3.78$) as well as for the symptom reduction all criteria (DSM Hedges's g_{av} between 2.50 and 3.09, ICD Hedges's g_{av} between 2.88 and 3.258). They emphasize the great benefit for traumatised populations of Trauma Stabilisation Treatment. Nevertheless, the results from this study are extraordinarily high. Ferguson [44] concludes that effect size interpretation, as demonstrated by the group differences pre and post PTSD and different PTSD diagnostic criteria, indicate a strong effect (cut-off for strong effect: Hedges $g = 2.70$). By means of explanation to account for this one aspect could relate to the importance of the therapeutic relationship – which is also a potential agent of change. In fact the Second Task Force on Evidence-Based Therapy Relationships convened by the American Psychological Association consider this an integral factor [45]. They conclude - based on meta-analyses and reviews, that the therapy relationship accounts to the treatment success at least as much as the method/ paradigm used and thus should be explicitly addressed in practice and treatment guidelines. This recommendation is met by the Trauma Stabilisation Interventions as outlined in the ROTATE manual [34]. Norcross and Wampold [45] also describe that an adaption of the therapy relationship to specific client characteristics (in addition to diagnosis) enhanced the effectiveness of the treatment. One important aspect of adapting psychotherapy to the individual client relates to culture. Common themes regarding cultural adaptation include flexibility, adaptability, meaningfulness, empathy and traditional treatments being used alongside existing resources [46]. In their meta-analysis Smith, Rodríguez [46] show that culturally adapted mental health therapies are superior to therapies not incorporating cultural considerations. Especially Asian American clients profited from culturally adapted treatments compared to other American client groups. The Mekong Project I explicitly addressed cultural considerations in their trainings, adaptions of stabilisation techniques to fit the cultural background of the individual client were discussed and encouraged. Trauma Stabilisation Treatments have the advantage of being flexible in their use and thus, give the therapist the possibility to really adapt treatment interventions to the specific needs of their clients.

A further cultural consideration arose from the Mekong Project I therapists themselves regarding the high treatment effect sizes this research highlights. Traditionally, Cambodian, Thai and Indonesian clients don't get any psychotherapeutic treatment at all. Thus, for all clients the therapy connected to the Mekong I Project was their first psychological treatment. With initial treatment interventions clients receive attention, validation of their trauma and posttraumatic stress problems. This is also an agent of change. Both components may contribute to a desire of the client to please the therapist. The local therapists explained that this 'wanting to please' also has a strong cultural component. It can't be ruled out that at least some of the clients wanted to please their therapists and show gratitude for the subsequent treatment they received. Only further replication studies in other countries may either confirm or deny this hypothesis.

Putting the Effect Sizes into Context

Mekong Project I considered whether the use of a waiting list control-group was feasible, but this raised numerous ethical dilemmas and therefore was subsequently rejected. The Mekong I Project was always set up as a trauma capacity building project to meet the needs of treatment of a huge number of traumatised clients in South East Asia. Thus, the resources of the Project were used to train as many therapists as possible, in as thorough and sustainable manner as was possible. This included to offer trauma treatment to as many traumatized clients as possible – namely in the form of EMDR Therapy. This limits the interpretation of the results, as Durlak [47] emphasises effects for within-subject designs are usually much higher than for control group designs and can easily exceed 1.0. Nevertheless, Devilly and McFarlane [48] describe possibilities to still evaluate a study by comparison to existing data from wait-list controls. In their meta-analysis, the unweighted average effect size of wait-list conditions was 0.358 with a standard deviation of 0.276 and report clinical cut-offs to judge the relative efficacy of the treatment. With the effect sizes above the treatment meta-analysis mean effect size 2.42 (DSM-V Hedges's $g_{av} = 3.078$, BCa 95% CI [2.875, 3.271]; ICD-11 Hedges's $g_{av} = 3.780$, BCa 95% CI [3.559, 3.994]), the trauma stabilisation treatment appears to be better than best practices for PTSD treatment in the short term.

Nevertheless, the cultural context of the effect sizes isn't taken into account in this evaluation and Devilly and McFarlane [48] describe as an alternative approach to compare the treatment group with the waiting-list group of a similar previous study. By comparing the data to an intent to treat control group aggregated in the same time and region by the same therapists with the same diagnostic tools, the waiting-list group of the RCT ROTATE [Trauma Stabilisation] study [35, 36] is a very appropriate comparator. Additionally, the cultural influences of the effect sizes are also present in this control group. The clients of this group also didn't have any treatment at all previously and get attention and validation of their trauma for the first time. The only difference is that they get their free of charge treatments with a five-week delay. Treatment expectancy effect and an understanding of one's own presentation due to structured assessment as well as feeling understood and supported [48], results in some reduction in the severity pf PTSD symptoms in the short term. These aspects combined with the cultural desire to please can explain the remission rate of 24.4% of DSM-V PTSD and 33.3% of ICD-11 PTSD after waiting. The effect sizes of these significant remissions are medium (DSM-V PTSD Hedges's $g_{av} = 0.594$, ICD-11 PTSD Hedges's $g_{av} = 0.761$).

However, the effect sizes for trauma stabilisation are almost five times the size of being on a waiting list. A direct comparison between the stabilisation treatment group and the waiting-list group via general linear model analyses reveals a very large effect for the stabilisation treatment. The explained variance between the groups is 31.03% for DSM-V PTSD and 32.38% of ICD-11 PTSD. Looking at the whole sample, regardless of the PTSD diagnosis at first measurement, also a significant difference between the clients getting worse to the second measurement becomes apparent. For the waiting-list groups five to six times more clients get worse compared to the trauma stabilisation group (DSM-V PTSD 9.1% getting worse during waiting, 1.4% getting worse during treatment, ICD-11 PTSD 14.6% getting worse during waiting, 2.7% getting worse during treatment).

The results strengthen and corroborate the earlier findings of two RCTs that effective trauma therapy can also significantly reduce posttraumatic symptoms without the need of trauma exposure [35, 36, 49]. Nevertheless, with a comparison to a waiting list group non-specific aspects of the treatment aren't controlled. Further insights to the effectiveness of trauma

stabilisation could be gained by treatment studies comparing trauma stabilisation treatment to a control group receiving treatment as usual or comparing it to trauma confrontation interventions.

Criticism of Study

Nevertheless, the research and the results can be rightly criticized and viewed with a degree of caution. Replication studies are much needed. A compromise had to be made between conveying basic knowledge to the therapists and addressing the specific needs of the traumatised clients. With the challenge of responding to victims' mental health needs in a post-disaster area and the priority upon mental health capacity building through training, the ability to meet methodological quality of the studies as in Western samples is quite limited [14, 50]. Thus, limitations of our study are: (a) the lack of a follow-up period (b) no external, blind rater of the PTSD-symptoms. Without a follow-up measurement, no conclusions can be drawn to the sustainability of the treatment effect. But the Mekong I Project as a capacity building project with the primary aim to meet the needs of treatment of a vast number of traumatised clients, would have been compromised by focusing at high study standards as a follow-up measurement. A comparison to the remission rate of the ROTATE RCT study where the assessments were performed by a blind to treatment allocation investigator (95.9% PTSD remission, [36]) shows that the lack of a blind rater was of little consequence.

Advantages of the Study

Advantage of Local Therapists (Language, Culture)

A strength of our study is that the treatments taught in Mekong I were conducted by local therapists who had been extensively trained and supervised in Trauma Stabilisation Interventions. Consequently, Trauma Stabilisation has the benefit of being conducted using indigenous language and therefore no interpreters were necessary. Additionally, the therapists and the clients had similar cultural backgrounds, thus culture-specific interpretations of symptoms could be considered, and techniques could be culturally adapted if necessary.

Uniform Training of all Therapist

All therapists were trained in trauma stabilisation during the trainings of the Mekong I Project. They each had local clinical supervision which ensured treatment fidelity to the trauma stabilisation interventions used. These were deemed as being consistent with high levels of fidelity.

Generalizability to Real World Settings

Studies about the treatment of PTSD have been criticised for a vast number of exclusion criteria for entrance into study which decreases the generalizability of a lot of results. PTSD is associated with complex outcomes and multiple comorbid emotional, social and physical health difficulties, particularly among those who have experienced chronic traumatisation, but many published studies about Treatment Outcomes of PTSD exclude individuals with severe comorbid psychopathology [51–53]. With the data of the Mekong Project the generalizability to real world settings is extremely high, because for the clients in the Mekong Project I, there were no exclusion criteria at all. Additionally, all clients were service seekers, people

that were looking for therapy. Thus, the sample of the study is very generalizable to real world settings, it consisted of clients that were looking for a trauma specific treatment.

As in real world settings, all service seeking clients received treatment and weren't excluded if a previously screened diagnosis wasn't completely fulfilled. With the inclusion of all service seeking clients regardless of their diagnosis, the results are not only more generalisable to real world, but also important practical implications can be drawn. The analysis of how subclinical clients develop under treatment gives important additional information about the range of the treatment effect as well as potential risks of the specific treatment.

Trauma Stabilisation as Effective, Safe and Easy to Learn Therapy for Post Conflict Areas

Especially in areas were mental health facilities are limited and the education of therapists low, we see the need for an easy to learn and to culturally adapt treatment with no risks. Our results strengthen earlier findings that trauma therapy doesn't necessarily require trauma exposure to be effective in reducing posttraumatic symptoms and increasing the level of functioning. Trauma Stabilisation doesn't focus on traumatic memories directly, but resource, stabilisation and skill development. Especially in post conflict areas, with a high risk for natural disasters, this treatment can prepare the clients for future traumatic events, strengthening coping skills and enhancing resilience and potentially post-traumatic growth. A further advantage of Trauma Stabilisation Interventions is that it can be taught to paraprofessionals and allied health professionals in areas with a scarcity of mental health professionals. The results from this study are promising but more research is needed to further explore the wider impact of Trauma Stabilisation as a treatment effect in terms of clinical and economic benefits.

The study suggests that trauma stabilisation is safe, effective, efficient and sufficient in treating clinical and subclinical trauma populations with the data indicating high remission from PTSD and traumatic sequelae. Stabilisation techniques are adaptable, flexible, culturally contextualised, spiritually sensitive and individually tailored to specific needs.

Acknowledgements The data analysed in this study was collected as part of an ODA Project (Official Development Assistance, Project 2010.1572.6, Program for Trauma Treatment in Thailand, Burma, Cambodia and Indonesia, Mekong Projekt). This Project was carried out by Trauma Aid Germany and funded by the German Federal Ministry for Economic Cooperation and Development – also in co-operation with Terre des Hommes (Germany). The funders of this study had no role in study design, data collection, data analysis, data interpretation, or writing the report. The authors thank all involved therapists responsible for treating the patients and for the data collection and hence substantially contributing to this study: Ms. Rahmia Dewi, Mr. Mochtar Maulana, Mr. Desril Siregar, Ms. Yulia Direzkia, Mr. Tokok Widayanto, Mr. Bunna Phoeun, Mr. Thearom Ret, Mr. Fuye Ongko, Mr. Plaktin Om, Ms. Sotheary Yim, Ms. Serey Samchet, Ms. Pyaongsri Kanthikul, Ms. Thuon Nary, Ms. Fazlun Reza, Ms. Pairada Varnado, Ms. Mallika Supap, Ms. Boonnisa Boonprasop, Ms. Connie Kristanto, Mr. Tri Hadi, Ms. Supannee Kekina, Mr. Theerarat Boripuntakul, Ms. Nawanant Piyavhatkul, Mr. Sothearat Seoung, Mr. Prasongsak Jenkitjaphaiboon, Mr. Marid Kaewchinda, Ms. Amornrat Khongchub, Mr. Nil Ean, Mr. Sophearith Phul, Ms. Kanokwan Jirathongkhamchote, Ms. Dian Wisnuwardhani, Mr. Nathanael Sumampouw, Ms. Rattana Preechasuchat, Ms. Reni Kusumowardani, Ms. Thanyalak Wanliang. The authors thank Mr. Sombat Tapanya for translating Thai content in the data set into English.

Compliance with Ethical Standards

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Conflict of Interest The authors declare that they have no conflict of interest.

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Journal of Affective Disorders 141 (2012) 11–21

Contents lists available at SciVerse ScienceDirect

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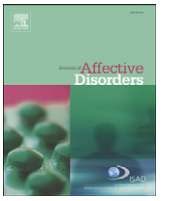

Review

The role of executive function in posttraumatic stress disorder: A systematic review

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ARTICLE INFO

Article history:
Received 8 November 2011
Received in revised form 31 December 2011
Accepted 1 January 2012
Available online 5 February 2012

Keywords:
PTSD
Neuropsychological impairments
Executive functioning
Comorbid depressive disorder

ABSTRACT

Background: Although posttraumatic stress disorder (PTSD) has been associated with disturbances in verbal memory, studies examining executive functioning in PTSD show mixed results.
Methods: A systematic review and meta-analysis were performed to compare executive functioning in patients with current PTSD and controls without any psychiatric disorder. Standard mean differences (SMD) in executive functioning scores were calculated using random-effects models. Covariates were added to examine whether differences exist between subgroups.
Results: Across 18 studies, 1080 subjects were included. In comparison with 431 exposed controls and 227 healthy controls, 422 people with PTSD showed significantly impaired executive functioning. Subgroup analyses revealed more pronounced differences between PTSD patients and exposed controls than healthy controls. Male gender, higher age, war trauma, and higher severity of co-morbid depressive symptoms were related to poorer executive functioning in PTSD patients compared to exposed controls.
Limitations: Due to insufficient data and heterogeneity, not all subgroup differences or characteristics could be taken into account.
Conclusions: Overall, PTSD patients were found to show impaired executive functioning. Future research should further elucidate the subgroup effects and focus on clinical implications with regard to daily functioning and treatment outcome.

Contents

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doi:10.1016/j.jad.2012.01.001

1. Introduction

In posttraumatic stress disorder (PTSD) the memory of the traumatic event is thought to be fragmented, with storage in sensory fragments and retrieval occurring as sensory and emotional representations without the transcription into personal narratives (Van der Kolk and Fisler, 1995). The clinical picture of PTSD with recurrent re-experiences and the failure of integrating the traumatic memories, indicates that autobiographical memory may be affected (Brewin, 2007).

Recent research has also shown that problems with memory concerning emotionally neutral, nonautobiographical information are apparent. This dysfunction may be associated with declarative rather than autobiographical memory. Several neuropsychological studies have now documented impairment in cognitive functioning in PTSD patients (Gilbertson et al., 2001; Stein et al., 2002), particularly in verbal memory (Brewin et al., 2007; Johnsen and Asbjørnsen, 2008). PTSD, however, has inconsistently been associated with impaired executive functioning like divided attention, cognitive flexibility, selective attention and inhibition, working memory and planning (Kanagaratman and Asbjørnsen, 2007; Koso and Hansen, 2006; Meewisse et al., 2005). No systematic review has been done focusing on impairments in these domains and thus far it is unclear whether impairment in executive functioning is present in PTSD. Adequate executive functioning is, apart from its vital role in daily functioning, crucial for participating in interventions like cognitive behavioural therapy (CBT) or even pharmacological treatment. For example, greater impairment in executive functioning predicted poorer response on treatment with fluoxetine in major depression (Dunkin et al., 2000). Likewise, executive impairment may also negatively affect treatment outcome in PTSD as was also found for impaired verbal memory in PTSD patients (Wild and Gur, 2008). To substantiate such assumptions, a further clarification of the association between executive functioning and PTSD is needed and prompted us to systematically review studies reporting on executive functioning in PTSD.

2. Methods

2.1. Identification of studies

Relevant studies were identified by systematic searches of databases PubMed and PsycINFO. Articles published between 1990 and July 2011 were included. A free-text search was performed with keywords 'Executive Function', 'Neuropsychological', AND 'Working Memory' in combination with 'Posttraumatic Stress Disorder' OR 'PTSD'. Reference lists of articles obtained were checked for relevant studies. Furthermore, researchers were contacted in order to include unpublished studies as well as to provide additional data when needed.

2.2. Inclusion and exclusion criteria

Studies were included if they reported on: (1) adults (aged 18–65) with current chronic PTSD diagnosed according to the criteria used in the DSM-III, DSM-III-R or DSM IV (American Psychiatric Association, 1980, 1987, 1994); (2) executive function of both a PTSD group and a comparison group (non-PTSD trauma-exposed or non-exposed controls) using one or more of the following neuropsychological instruments: Trail Making Test (TMT; Reitan, 1992) measuring divided attention, Wisconsin Card Sorting Test (WCST; Heaton, 1981) for cognitive flexibility or "'set-shifting'", the WAIS-R Digit Span (Wechsler, 1981) or WAIS-III Digit Span (Wechsler, 1997) measuring working memory, Stroop (Stroop, 1935) measuring selective attention and inhibition and the Rey-Osterrieth Complex Figure Test (CFT; Rey, 1941) measuring planning; (3) the mean, standard deviation and N for both groups. Other validated versions of neuropsychological tasks, such as sustained attention tasks (e.g. continuous performance tasks), other inhibition and planning tasks (e.g. stop-signal tasks and Tower of London tests, respectively) or the Delis-Kaplan Executive Function System (D-KEFS; Delis et al., 2001), measuring executive functioning (e.g. subtests Color-Word Interference Test or

Trail Making Test), were not included due to a limited number of studies reporting on these instruments.

Studies were excluded if they reported on: (1) PTSD groups consisting of comorbid disorders other than depression (e.g. personality disorders, dissociative disorders); (2) patient groups with lifetime or partial PTSD; (3) data in another language than English. The first and second author independently assessed each retrieved study and only included the study when final consensus was established.

2.3. Data analysis

To visualise the data from all available instruments, we calculated the standardised mean difference (SMD) in scores between the PTSD group and the control group together with its associated standard error (SE) and 95% confidence interval (CI). We used the SMD rather than the MD as our main outcome parameter because of the different scaling between the instruments. Furthermore, the direction of scores was also different between instruments (e.g. higher scores not uniformly indicate to better executive functioning) and therefore scores were reversed for the Trail Making Test (TMT B) and the Wisconsin Card Sorting Test perseverative errors (WCST-pe).

A forest plot was constructed to show these data grouped by type of instrument and type of control group. In general, two distinct control groups were used across and within studies: healthy controls that never experienced a previous traumatic event and control patients that at some point in their life experienced one or more traumatic events. In the first analysis, we examined the impact of this difference in control group.

In the subsequent analyses we excluded data coming from healthy control groups in order to be able to evaluate whether the difference was associated with PTSD rather than with having experienced a traumatic event. In this step, we examined whether specific characteristics of the PTSD group or the control group had an impact on the observed differences in scores between PTSD and exposed controls. The following characteristics were examined: type of trauma in the PTSD group, sex (men, women or both), mean age of the PTSD group, years of education and severity of comorbid depression symptoms in the PTSD group. The impact of these characteristics was examined by adding a covariate to the random effects regression model and subsequent testing for a difference between subgroups of studies. Covariates were added to the model one at the time. Given the explanatory nature of these subgroup analyses and the limited number of studies, we highlighted covariates where the test of interaction was 0.1 or below.

Subgroup analyses, as outlined above, were performed as follows: (1) type of trauma was investigated by grouping studies into the following categories: war veterans, victims of sexual or physical abuse, refugees, disaster and various traumas. To investigate (2) the influence of sex, studies were divided into those that included male participants, female participants and studies that used both male and female participants. To examine (3) the impact of age we added the mean age of the PTSD group to the model, knowing that all studies matched their control groups for age. Years of education (4) was examined by adding the mean number of years of education in the PTSD group as a covariate to the model, again because studies matched their control group for this factor. And finally, the severity of (5) depression was measured by constructing categories for minimal depression, mild depression, moderate depression and severe depression. These categories were based on existing cut-off scores of the depression questionnaires.

All pooled estimates together with 95% CI and meta-regression results were based on random-effects models using the SMD as the outcome parameter. The MIXED procedure in SAS version 9.2 was used to fit the various random effects models (Van Houwelingen et al., 2002). P values of less than .05 were considered statistically significant.

3. Results

3.1. Search and inclusion

Our initial search identified 23 articles that met our inclusion criteria. However, in five of these initially selected articles data was incomparable due to various reasons such as usage of different subtests or versions (e.g. Twamley et al., 2009) using different scoring techniques, or not reporting norm scores. For these reasons articles that reported on the Rey-Osterrieth task CFT (Barrett et al., 1996; Crowell et al., 2002; Dileo et al., 2008) had to be excluded. Consequently, 18 articles were included and were used in our data-analysis. Of the included articles, one study was unpublished (Koso et al., in prep) at the time of the search. In one study (Samuelson et al., 2006), we only included the patient and control groups without prior alcohol dependence, as this was in line with other PTSD groups and control groups as well as consistent with our own in- and exclusion criteria. The flow-chart is shown in Fig. 1.

Across all 18 studies, a total of 1080 people were included, with 422 PTSD patients and 431 exposed controls and 227 healthy controls. The median sample size was 19.5 for people with PTSD (range 10–45) and 22 for exposed controls (range 12–105) and 24.5 for healthy controls (range 16–87).

3.2. Study characteristics

All included studies and their characteristics are shown in Table 1. The instruments used were the Trail Making Test B (TMT B) (8 studies), the Wisconsin Card Sorting Test perseverative errors (WCST-pe) (3 studies), the Wisconsin Card Sorting Test categories completed (WCST-cc) (3 studies), Digit Span total (DS-tot) (11 studies), Digit Span backward (DS-bw) (5 studies) and Stroop interference (5 studies). The majority of the studies compared PTSD patients to exposed controls (12 studies), several studies used both exposed controls and healthy controls as comparison groups (3 studies), whereas others compared solely to healthy controls (3 studies). Twelve studies included patients that were exposed to war related traumata (war combat or refugees), others included victims of sexual abuse (3 studies), some included patients having PTSD due to various reasons (2 studies) and one study included patients exposed to a disaster. All studies applied matching for the factors age, gender and years of education, while only one study matched for intelligence (Koenen et al., 2001). All studies reported on factors that could influence executive functioning, and

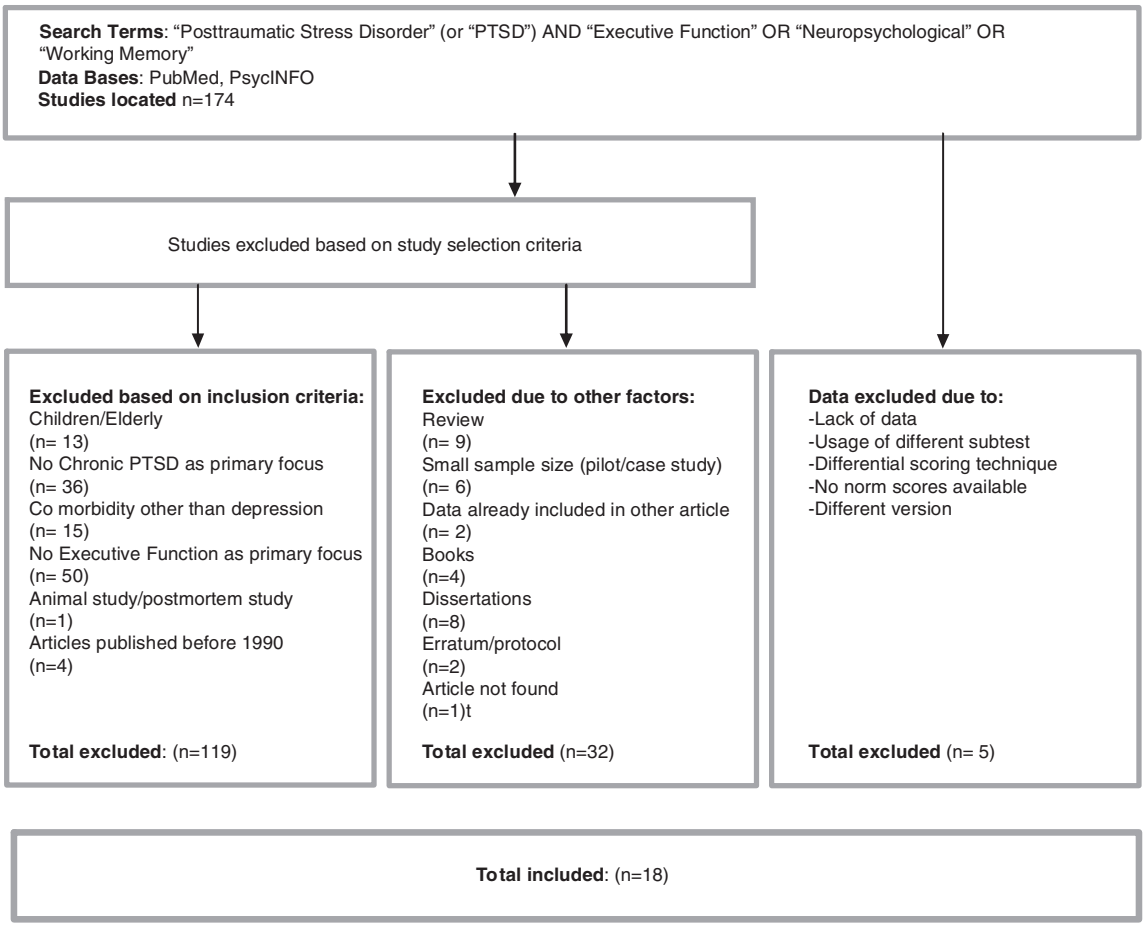

Fig. 1. Flowchart showing search results, and the number of included and excluded studies.

consequently excluded patients with traumatic brain injury or long periods of unconsciousness. Additionally, some studies excluded patients based on recent drug or alcohol usage, others did not but reported on alcohol screens (Gilbertson et al., 2001, 2006; Jenkins et al., 2000; Koso and Hansen, 2006; Samuelson et al., 2009; Twamley et al., 2004). Only one study excluded patients with current moderate to severe depressive disorders (Lindauer et al., 2006).

3.3. Overall comparison

Fig. 2 shows a forest plot of the SMD of executive functioning scores in patients with PTSD relative to controls in each of the 18 included studies. These results are stratified by type of instrument and type of control group. Because some studies reported on more than one instrument measuring executive functioning and others included both exposed and healthy controls, a total of 45 comparisons between a PTSD group and a control group are included in this forest plot.

3.4. Pooled estimates by type of instrument and type of control group

Table 2 shows the range of raw scores on all instruments in PTSD patients and controls. Fig. 3 presents the pooled SMD and its 95% CI for each instrument stratified by the type of control group. The pooled standard mean difference between PTSD patients compared to exposed controls were significantly different for the TMT B, WCST (perseverative errors and categories completed) and Digit Span total, with the PTSD groups scoring worse on these instruments of executive functioning than exposed controls. The pooled mean differences in PTSD patients versus healthy controls were only significantly different for the TMT B and the Stroop, showing that PTSD groups performed worse on these instruments.

3.5. Subgroup analyses in studies examining PTSD patients versus exposed controls

We excluded data coming from healthy control groups in order to be able to evaluate whether the difference was associated with PTSD rather than with having experienced a traumatic event. We examined whether specific study characteristics had an impact on the observed differences in scores between PTSD patients and exposed controls.

Table 3 shows the pooled standard mean differences for executive functioning measures between PTSD groups and exposed control groups on various factors, i.e. type of trauma, gender, age, years of education and comorbid depression.

Table 1
depicts all studies included based on in- and exclusion criteria. For each study several factors are shown; number of patients in the patient and control group, diagnostic criteria being used, trauma type used in the study and the instrument used for measuring executive functioning.

Study	*n* (patients/exp controls/ healthy controls)	Diagnostic criteria	Trauma type	Instrument
Beckham et al. (1998)	45/45/-	SCID [a] (DSM-III-R), Mississippi PTSD Scale	War combat	TMT B
Eren-Koçak et al. (2009)	16/22/-	CIDI [b] (DSM-IV), CAPS [c] (DSM-IV)	Disaster	Stroop
Gilbertson et al. (2001)	19/13/-	SCID (DSMIII-R), CAPS (DSM-IV)	War combat	TMT B Digit Span (total) WCST (categories completed) WCST (perseverative errors)
Gilbertson et al. (2006)	19/-/24	SCID (DSM-IV), CAPS (DSM-IV)	War combat	WCST (perseverative errors)
Jenkins et al. (2000)	15/16/16	SCID (DSM-III-R), PTSD Interview	Sexual abuse	Digit Span (backward) Digit Span (total) TMT B
Johnsen et al. (2008)	21/21/-	M.I.N.I. [d] (DSM-IV), CAPS (DSM-IV)	Refugees	Digit Span (backward)
Kanagaratman and Asbjørnsen (2007)	22/23/-	M.I.N.I. (DSM-IV), CAPS (DSM-IV)	Refugees	WCST (categories completed) WCST (perseverative errors)
Koenen et al. (2001)	16/-/53	C-DIS [e] (DSM-III-R), CAPS (DSM-III-R)	War combat	TMT B
Koso, and Hansen (2006)	20/20/-	Diagnosed by psychiatrists (DSM-IV)	War civilians	TMT B Digit Span (total)
Koso et al. (in preparation)	45/34/-	Diagnosed by psychiatrists (DSM-IV)	War civilians	TMT B Digit Span (total)
Lindauer et al. (2006)	12/12/-	SCID (DSM-IV), SI-PTSD [f] (DSM-IV)	Mixed	Stroop
Samuelson et al. (2006)	37/31/-	SCID (DSM-IV), CAPS (DSM-IV)	War combat	Digit Span (total)
Samuelson et al. (2009)	25/22/-	SCID (DSM-IV), CAPS (DSM-IV)	War combat	Digit Span (total)
Stein et al. (2002)	17/22/22	SCID (DSM-IV), CAPS (DSM-IV)	Sexual abuse	Digit Span (backward) Digit Span (total) TMT B Stroop
Twamley et al. (2004)	38/105/87	PDS [g]	Mixed	Digit Span (backward) Digit Span (total) TMT B
Vasterling et al. (1998)	19/24/-	SCID (DSM-III-R and DSM-IV)	War veterans	Digit Span (total) WCST (categories completed) Stroop
Vasterling et al. (2002)	26/21/-	SCID (DSM-IV)	War combat	Digit Span (total) Stroop
Weniger et al. (2008)	10/-/25	SCID (DSM-IV), IES-R [h]	Sexual abuse (type II)	Digit Span (backward) Digit Span (total)

[a]Structured Clinical Interview for DSM-Diagnosis.
[b]Composite International Diagnostic Interview.
[c]Clinician Administered PTSD Scale.
[d]Mini-International Neuropsychiatric Interview.
[e]Computerized Diagnostic Interview Scale-Revised.
[f]Structured Interview for Posttraumatic Stress Disorder.
[g]Post-Traumatic Stress Diagnostic Scale.
[h]Impact of Events Scale-Revised.

3.5.1. Type of trauma

Concerning type of trauma, a significant interaction was found on the TMT B ($p=0.0027$), the Digit Span total ($p=0.10$) and the Digit Span backward ($p=0.069$). Trauma type differences on the TMT B seemed to be related to worse scores on groups with war related trauma (SMD = −1.17, CI −1.5 to −0.8; $p<0.0001$) and to a lesser extent to the sexually abused group (SMD = −0.79, CI −1.4 to −0.2; $p=0.0082$). On the Digit Span total, also groups with war related trauma seemed to score significantly worse than exposed controls (SMD = −0.78, CI −1.2 to −0.4; $p=0.0001$). On the Digit Span backward, subgroup differences seemed to be related to worse scores on the refugee patient group (SMD = −1.08, CI −2.1 to −0.1; $p=0.038$).

3.5.2. Gender

Concerning gender, a significant interaction was found on the TMT B ($p=0.0027$) and on the Digit Span total ($p=0.065$). On the TMT B interaction effects seemed to be mostly related to male patient groups (SMD = −1.17, CI −1.5 to −0.8; $p<0.0001$), but also to female groups (SMD = −0.79, CI −1.4 to −0.2; $p=0.0082$). On the Digit Span total, interaction effects seemed to be related to worse scores of male groups (SMD = −0.97, CI −1.4 to −0.5; $p<0.0001$).

3.5.3. Age

Concerning age, on both the TMT B ($p=0.026$) and the Digit Span total ($p=0.026$), a significant interaction was

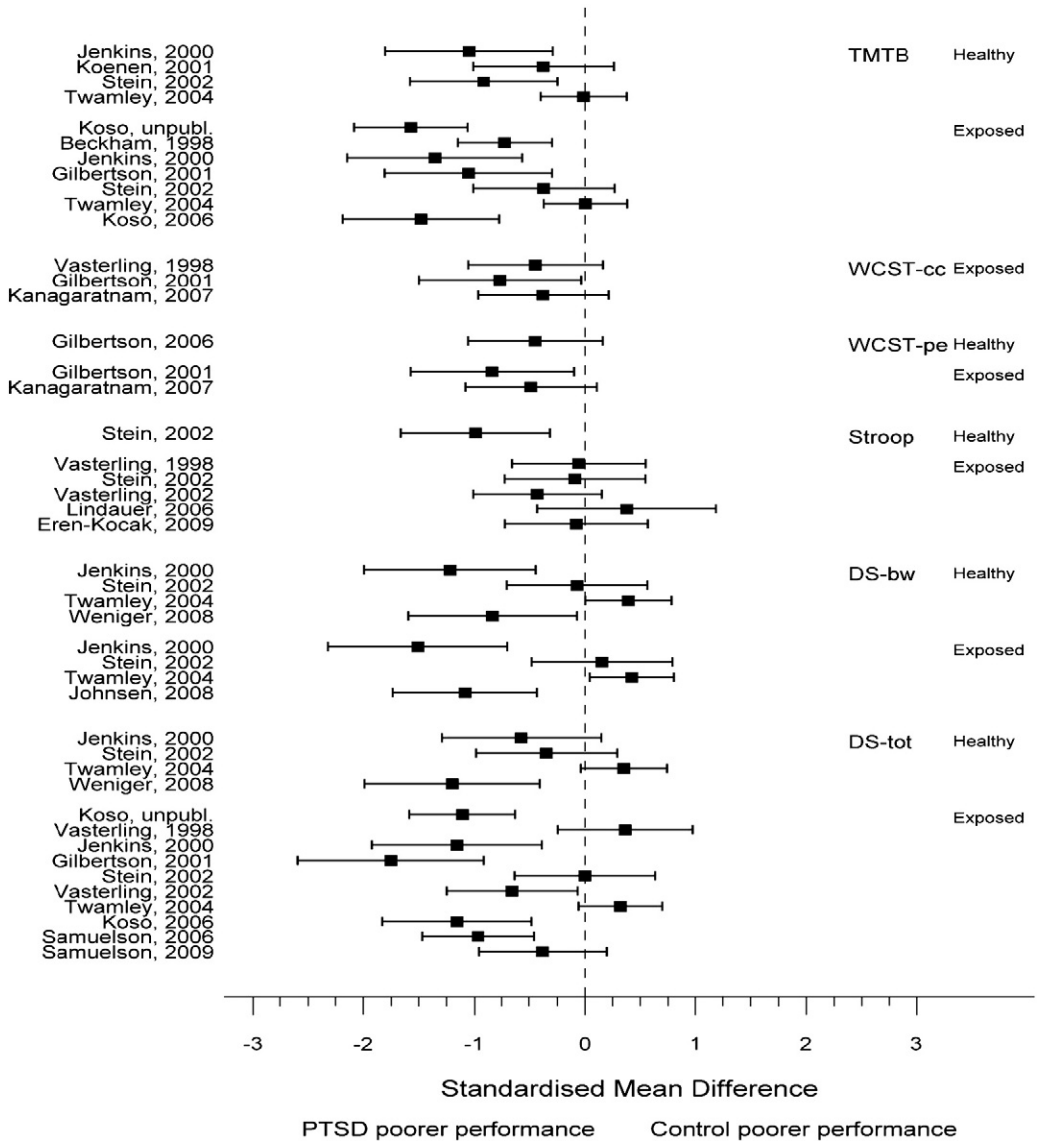

Fig. 2. Forest plot, standardized mean differences (with 95% CI) showing scores on instruments measuring executive functioning in PTSD groups compared to control groups. Studies are ordered according to instrument and type of control group. The direction of scores across instruments has been uniformly recoded so that higher scores indicate better executive functioning.

found. Scores worsened with higher age, with significant differences in patient groups and controls on 40 years (SMD = −0.94, CI −1.3 to −0.6; $p<0.0001$) and 50 years (SMD = −1.27, CI −1.8 to −0.8; $p<0.0001$). Also on the Digit Span total, on age 40 (SMD = −0.59, CI −0.9 to −0.3; $p=0.0003$) and on age 50 (SMD = −0.94, CI −1.4 to −0.5; $p<0.0001$) significant differences were found with controls.

Table 2
range of raw scores on different instruments in PTSD patients, exposed controls and healthy controls.

Group	TMT B	WCST-pe	WCST-cc	DS-tot	DS-bw	Stroop
PTSD patients	53.4–173.3	28.9–31.3	2.7–4.5	8.7–18.3	4.8–7.3	33.8–49.2
Exposed controls	45.2–90.2	12.2–20.4	3.6–5.5	9.5–19.6	6.5–9.1	29.4–49.8
Healthy controls	52.1–64.4	21.9	–	15.0–18.2	6.5–8.6	50.9

3.5.4. Comorbid depression

Concerning comorbid depression, a significant interaction was found on the TMT B ($p=0.0029$), Digit Span total ($p=0.053$) and Digit Span backward ($p=0.052$). For WCST categories completed and WCST perseverative errors, no studies with mild depressive symptoms were included and could therefore not be compared. On the TMT B, interaction effects seemed to be related to severe depressive symptoms (SMD = −0.85, CI −1.3 to −0.4; $p<0.0001$). This was also the case for the Digit Span total (SMD = −0.90, CI −1.6 to −

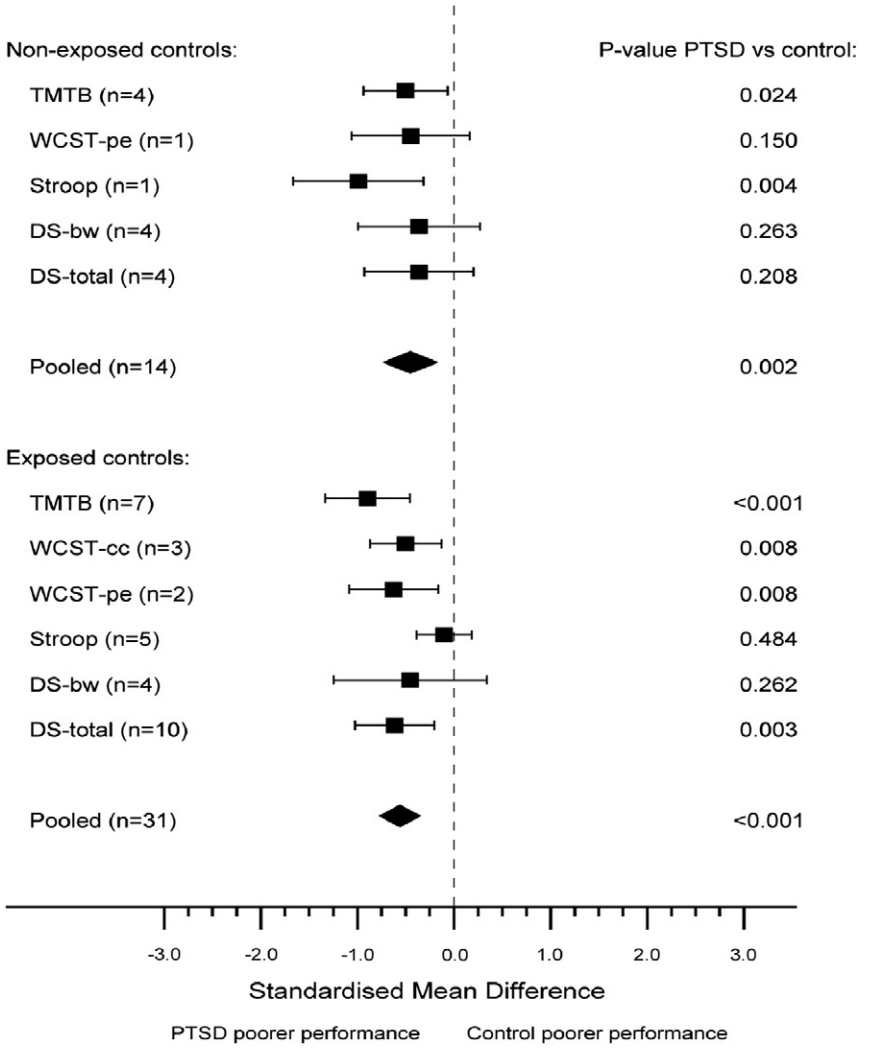

Fig. 3. Forest plot showing pooled standardized mean differences (with 95% CI) of executive functioning instruments between PTSD patients versus control groups. Results ordered by type of control group and instrument. The direction of scores across instruments has been uniformly recoded so that higher scores indicate better executive functioning.

0.2; $p = 0.011$) and the Digit Span backward (SMD = −0.77, CI −1.4 to −0.1; $p = 0.024$).

4. Discussion

In this systematic review, 18 studies were included in which executive functioning was examined in adult groups with current PTSD in comparison with exposed or healthy control groups. In general, PTSD patients performed significantly worse than controls on most instruments of executive functioning. Results were, however, quite heterogeneous, indicating that several subgroup differences effected executive functioning such as type of trauma and comorbid depression.

4.1. Control group

Our general finding of poorer executive functioning in PTSD patients compared to controls is consistent with earlier studies of PTSD patients (Beckham et al., 1998; Koso and Hansen, 2006) as well as with previous findings in depression (Fossati et al., 2002). Subgroup analyses revealed, however, that people with PTSD scored significantly worse when compared to exposed controls while less profound differences were found when compared to non-exposed controls. This finding is contrary to our expectations, as we expected controls exposed to trauma to have a greater number of posttraumatic stress symptoms than non-trauma exposed controls and in turn to have more neuropsychological deficits associated with these symptoms than non-exposed controls (Buodo et al., 2011). This finding illustrates that impaired executive functioning in PTSD is not specifically nor merely related to exposure to trauma. Perhaps, people exposed to one or more traumatic events without having developed posttraumatic stress or depression might have more efficient coping strategies that do not interfere with the natural recovery and protect them from having PTSD (previously reviewed by Olff et al., 2005). More

Table 3
Executive functioning differences between PTSD and exposed controls by various factors.

Subgroup	Pooled SMD TMT B	Pooled SMD WCST-pe	Pooled SMD WCST-cc	Pooled SMD DS-tot	Pooled SMD DS-bw	Pooled SMD Stroop
Type of trauma						
War	−1.17	−0.84	−0.58	−0.78	N.A.	−0.25
Sexual abuse	−0.79	N.A.	N.A.	−0.53	−0.58	−0.089
Mixed	0.0052	N.A.	N.A.	0.32	0.42	0.38
Refugees	N.A.	−0.49	−0.38	N.A.	−1.08	N.A.
Disaster	N.A.	N.A.	N.A.	N.A.	N.A.	−0.077
P-value	0.0027*	0.47	0.60	0.10*	0.069*	0.61
Gender						
Males	−1.17	−0.84	−0.77	−0.97	N.A.	−0.43
Women	−0.79	N.A.	N.A.	−0.53	−0.64	−0.089
Mixed	0.0052	−0.49	−0.41	−0.091	−0.28	0.037
P-value	0.0027*	0.47	0.41	0.065*	0.66	0.43
Age						
Coefficient for 10 year increase	−0.33	−0.35	−0.36	−0.35	−0.58	−0.29
P-value	0.026*	0.47	0.41	0.026*	0.22	0.22
Years of Education						
Coefficient for 1 year longer education	0.29	−0.32	−0.24	0.07	−0.58	−0.20
P-value	0.28	0.47	0.54	0.79	0.44	0.52
Comorbid depression						
Not severe	0.0052	N.A.	N.A.	0.32	0.42	0.38
Severe	−0.85	N.A.	N.A.	−0.90	−0.77	−0.083
P-value	0.0029*	N.A.	N.A.	0.053*	0.052*	0.33

* The PTSD group scored worse than control groups with a p-value = 0.1.
N.A.: no contrast between studies on this factor or no data reported on this factor.

specifically, less avoidant coping strategies and less reactivity to trauma reminders (Pineles et al., 2011) might in turn be associated with less neuropsychological deficits (Buodo et al., 2011). This also relates to the concept of posttraumatic growth (Bostock et al., 2009) which has been associated with lower posttraumatic stress symptoms (Hall et al., 2008).

4.2. Type of trauma

Impaired executive functioning was most profound in people that developed PTSD following war combat while people with PTSD following various events showed to be least impaired when compared to other subgroups. Although more prevalent personality traits within groups of soldiers like risk taking behavior such as hazardous drinking (Foran et al., 2011) or drug abuse (Skodol et al., 1996) may account for this greater impairment, it is unlikely that these factors obscure our findings as almost all studies either excluded patients with prior or current abuse or controlled for these factors. Repeated trauma exposure in this group is another likely contributing factor to impaired executive functioning. Exposure to multiple traumas may result in a complex symptom presentation, including dissociative symptoms, and aggressive or socially avoidant behaviors, as previously seen in chronic sexually abused patients (Cloitre et al., 2009). We, however, did not find an overall significant impairment in executive functioning in sexually abused PTSD patients, even though this type of trauma may have a chronic character as well. This may suggest that symptom severity rather than the character and frequency of trauma exposure may be associated with impaired executive functioning, as was also found for emotion regulation difficulties (Ehring and Quack, 2010). There is some indication that in our study severity of PTSD symptoms is the main factor instead of type of trauma since most of the included studies with war combat patients (Koso et al., in preparation; Gilbertson et al., 2001, 2006) reported symptom levels higher than those in studies of sexually abused patients (Stein et al., 2002) or mixed patients (Twamley et al., 2004).

4.3. Gender

Male PTSD patients performed worse on executive functioning than exposed-controls of the same gender while female PTSD patients were not significantly different compared to female exposed-controls. Previous findings suggest differences between males and females in functional organization of the brain for working memory (Speck et al., 2000). The latter, however, seems not related to a different cognitive performance between males and females (Bell et al., 2006). Only one study in depressed patients showed poorer performance in females on cognitive interference (Sárosi et al., 2008) while no studies have described gender differences in executive functioning in PTSD. From our meta-analysis we may conclude that these differences can only be detected by studying large numbers of individuals. Another explanation is

that poor executive functioning performance in males is associated with trauma type and symptom severity of the male patient groups included in this study rather than with gender per se.

4.4. Age

Higher age was significantly associated with poorer executive functioning in PTSD patients compared to exposed controls, in particular (divided) attention and working memory. Previous research suggests that domains like attention, executive and working memory are impacted by higher age. Furthermore, these domains seem to be specifically sensitive and show to be impaired in mild cognitive impairment, such as in the prodromal phase of Alzheimer's disease (Saunders and Summers, 2011). In PTSD patients, impact of aging was previously found in some domains of memory like paired associate learning, but not in others such as verbal memory (Yehuda et al., 2006). The latter was only and particularly associated with severity of PTSD symptoms. Another possibility is that not higher age per se but total years of experiencing PTSD symptoms may impact executive functioning (Gilbertson et al., 2001; Kanagaratman and Asbjørnsen, 2007).

4.5. Comorbid depression

On almost all instruments an effect of comorbid depressive symptoms was found, implicating that in particular PTSD with severe comorbid depression is associated with impairments in executive functioning. This is consistent with previous studies showing that the memory deficits, such as verbal memory, are related to (co-morbid) depression, rather than to PTSD alone (Johnsen et al., 2008). These authors state, however, that the verbal memory deficits may be related to problems in executive control. Possibly our findings concerning the effects of depressive symptoms on executive functioning as well as the previous findings of deficits in verbal memory, may be explained by the same mediating factor, i.e. comorbid depression. Future research should include PTSD patients with and without a comorbid depression, in order to determine the role of both disorders in executive functioning.

4.6. Limitations

Several limitations need to be pointed out. Firstly, some subgroup differences that may have had an impact on executive functioning could not be taken into account. For example, severity of PTSD symptoms may have had an impact on executive functioning, however, due to usage of differential (semi)structured interviews for diagnosing PTSD (i.e. IES-R; CAPS; SI-PTSD), it was difficult to differentiate between all studies on this factor. Furthermore, in some studies, severity of symptoms was not reported (Jenkins et al., 2000; Koenen et al., 2001). Another factor that may have influenced executive functioning is years since trauma instead of age. No subgroup analysis could be performed on this factor as this information was lacking in the majority of included studies. Furthermore, we did not take into account processing speed due to a lack of reported data in the included studies on this measure.

Secondly, although we aimed for the inclusion of homogeneous studies, heterogeneity between studies was inevitable due to the diversity of measuring executive functioning. As not all instruments measure the same aspect of executive functioning (i.e. divided attention or cognitive flexibility), studies using different constructs were analyzed separately. Although this improves the accuracy of our results, this led to fewer studies within each subgroup and consequently some lack of power. In order to be able to compare studies on executive functioning in PTSD, more consensus on instruments measuring executive functioning is needed. The use of similar instruments over studies makes it easier to differentiate between impairments in several aspects of executive functioning in PTSD and other factors that may influence executive functioning. Furthermore, subgroup analyses based on characteristics of PTSD groups (i.e. trauma type, gender) or control groups (exposed-controls, healthy controls) also led to fewer studies within each group reducing the statistical power to detect differences.

Finally, publication bias may have influenced our results. Despite our efforts to include unpublished data, studies with significant findings on executive functioning may have been published more often than studies in which no difference between groups was found and we may not have succeeded in finding all unpublished studies meeting our inclusion criteria.

In conclusion, across 18 studies, people with PTSD performed worse on executive functioning than people without PTSD. With the relatively high lifetime prevalence of PTSD around 7% (De Vries and Olff, 2009; Olff et al., 2007) and the great individual distress as well as societal burden associated with PTSD, our study adds important findings particularly because executive function is crucial for effective occupational functioning (Kalechstein et al., 2003). Differences were more profound between PTSD patients and exposed controls than between PTSD patients and non-exposed controls. Subgroup analyses revealed that older and male patients performed worse as well as people with PTSD following war. Also, significantly worse executive functioning was seen in groups with more severe comorbid depressed symptoms than in groups with less depressive symptoms. Although PTSD may influence executive functioning directly, we must not rule out the possibility of pre-trauma impairments in executive functioning. These impairments may contribute negatively to the ability to cope with traumatic stress (i.e., response inhibition and attention regulation) (Aupperle et al., 2011). As this meta-analysis illustrates impaired executive functioning in PTSD patients, it may be particularly relevant to find out whether impaired executive functioning has clinical implications. Executive functioning is crucial for processing complex information as for participating in interventions like cognitive behavioural therapy. It has already been shown that impaired verbal memory in PTSD patients (Wild and Gur, 2008; Nijdam et al., in prep) predicts treatment outcome. Also in depression, poor executive functioning has been found to be associated with treatment outcome and prognosis (Dunkin et al., 2000). Further research should elucidate whether there is an association between response rates on various treatments (i.e. pharmacotherapy and psychotherapy) and executive functioning. If confirmed, scores on executive functioning

may be used as a predictor in the course of treatment and may help clinicians in identifying patients with delayed response or higher risk to relapse.

Role of funding source
None.

Conflict of interest
We have no conflicts of interests to disclosure.

Acknowledgements
None.

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Journal of the International Neuropsychological Society (2009), **15**, 879–887.

doi:10.1017/S135561770999049X

SYMPOSIUM

Cognitive impairment and functioning in PTSD related to intimate partner violence

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(RECEIVED December 23, 2008; FINAL REVISION June 16, 2009; ACCEPTED June 16, 2009)

Abstract

Posttraumatic stress disorder (PTSD) has been associated with neuropsychological impairments across multiple domains, but consensus regarding the cognitive profile of PTSD has not been reached. In this study of women with PTSD related to intimate partner violence ($n = 55$) and healthy, demographically similar comparison participants (NCs; $n = 20$), we attempted to control for many potential confounds in PTSD samples. All participants were assessed with a comprehensive neuropsychological battery emphasizing executive functioning, including inhibition, switching, and abstraction. NCs outperformed PTSD participants on most neuropsychological measures, but the differences were significant only on speeded tasks (with and without executive functioning components). The PTSD group's mean performance was within the average range on all neuropsychological tests. Within the PTSD group, more severe PTSD symptoms were associated with slower processing speed, and more severe dissociative symptoms were associated with poorer reasoning performance. These results suggest that women with PTSD related to intimate partner violence demonstrate slower than normal processing speed, which is associated with the severity of psychiatric symptoms. We speculate that the cognitive slowing seen in PTSD may be attributable to reduced attention due to a need to allocate resources to cope with psychological distress or unpleasant internal experiences. (*JINS*, 2009, *15*, 879–887.)

Keywords: Posttraumatic stress disorder, Trauma, Stress, Neuropsychological, Cognition, Domestic violence

COGNITIVE IMPAIRMENT AND FUNCTIONING IN PTSD

Posttraumatic Stress Disorder (PTSD) was included in the third edition of the Diagnostic and Statistical Manual (DSM-III) in 1980 (American Psychiatric Association, 1980) and began to be studied from a neuropsychological perspective soon afterward (e.g., Everly & Horton, 1989). Interest in the neuropsychological effects of stress and trauma has risen during the past decade, particularly in light of the current conflicts in Iraq and Afghanistan, along with research showing that deployment alone is a risk factor for neurocognitive dysfunction (Vasterling et al., 2006). Research on the cognitive impairments of trauma and PTSD over the past three decades has yet to yield a consensus regarding which cognitive domains are most affected, however. Impairments have been identified in almost every cognitive domain, including premorbid intellectual functioning (Vasterling et al., 2002), attention/working memory (Horner & Hamner, 2002; Samuelson et al., 2006; Stein, Kennedy, & Twamley, 2002; Vasterling, Brailey, Constans, & Sutker, 1998; Vasterling et al., 2002), processing speed (Samuelson et al., 2006; Stein et al., 2002), learning (Horner & Hamner, 2002; Samuelson et al., 2006; Vasterling et al.,

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1998, 2002), and executive functioning (Bremner et al., 2004; Leskin & White, 2007). Most studies have found no association between PTSD and impairment in memory retention or recognition (see Golier & Yehuda, 2002, for review). The pattern of neuropsychological impairments found across studies suggests disruption in frontal-subcortical circuits (Stein et al., 2002; Vasterling et al., 1998, 2002), although decreased hippocampal size and functioning have also been associated with PTSD (see Bremner, 2006, for review). Importantly, not all studies have found differences in cognitive functioning between PTSD and non-PTSD samples (Gurvits et al., 1993; Stein, Hanna, Vaerum, & Koverola, 1999; Twamley, Hami, & Stein, 2004).

Certain aspects of trauma exposure and PTSD have been associated with high likelihood of neurocognitive dysfunction and real-world functional impairments. For example, exposure to childhood trauma is associated with worse academic performance and numerous areas of cognitive deficits (Carrey, Butter, Persinger, & Bialik, 1995; Perez & Widom, 1994; Saigh, Mroueh, & Bremner, 1997). Among Axis I psychiatric disorders including PTSD, greater cognitive deficits are associated with greater psychiatric symptoms and functional impairment (Geuze, Vermetten, de Kloet, Hijman, & Westenberg, 2009; Green, Kern, Braff, & Mintz, 2000; Kalechstein, Newton, & van Gorp, 2003; Marvel & Paradiso, 2004; Twamley et al., 2002). Therefore, cognitive impairments are an important area of focus in PTSD research. However, a consistent picture of the neuropsychological sequelae of PTSD has been elusive, partly due to the numerous confounds associated with trauma exposure. Potential factors that result in inconsistencies across samples include: variation in the history of PTSD (e.g., number, timing, spacing, and severity of traumatic events; age of onset, severity, and chronicity of PTSD symptoms); variation in types of traumatic events (e.g., betrayal-related trauma such as rape or incest, nonbetrayal events such as accidents or acts of nature, and other events such as killing or being injured in combat or terrorist attacks); and variation in comorbid conditions (e.g., current or historical depression, substance use disorders, dissociation, brain injury, or other medical illnesses known to affect cognition).

Our program of research on PTSD-associated neuropsychological impairment has attempted to reduce the influence of some of these factors by focusing on one type of trauma—domestic or intimate partner violence (IPV). We have limited our trauma sample to women survivors of IPV. Thus, each of the women experienced betrayal trauma, which has been shown to result in a greater likelihood and severity of posttraumatic symptoms (Freyd, 1996; Freyd, Klest, & Allard, 2005). All of the women in our sample experienced trauma during adulthood (although some also experienced childhood trauma), and none had comorbid alcohol or substance use disorders when they participated in our research. Furthermore, we do not include women with comorbid medical conditions or medications that could affect brain functioning. In previous studies of women who meet these criteria, we have found impairments primarily on timed tasks of attention/working memory, inhibition, and switching (Stein et al., 2002). To further examine these executive functioning domains, the current study included an expanded neuropsychological battery with finer-grained assessments of executive functions in a new sample of women survivors of IPV.

We hypothesized that compared with non-traumatized comparison participants (NCs), PTSD participants would perform worse on neuropsychological tests of executive functions, particularly timed tasks. Furthermore, we expected that within the PTSD group, worse neuropsychological performance would be associated with greater childhood history of trauma, more severe current psychiatric symptoms, and greater levels of functional disability.

METHOD

Participants

From a larger initial sample, 3 PTSD participants with low levels of education (≤10 years) and 11 NC participants with high levels of education (≥21 years) were excluded from the current study to create groups that did not differ statistically on education. The final sample of participants included 55 women with full or partial PTSD related to IPV experienced within the past 5 years and 20 women who never experienced IPV and never met full or partial criteria for PTSD (current or lifetime) related to any trauma (NCs). Participants were recruited through ads in a local events and entertainment magazine, on a community volunteer Web site, and flyers distributed to community agencies (including agencies that provide IPV-related services). Of the 55 subjects with IPV exposure, 48 met full DSM-IV criteria for PTSD, while seven partially met criteria for PTSD (i.e., fulfilled Criterion A and the impairment/distress criterion, and had one less symptom than necessary to fulfill C or D criteria). Excluding the seven subjects with partial PTSD did not change the results in any meaningful way. Therefore, they were included in the PTSD group. Partial PTSD has been associated with the same level of distress and impairment as full PTSD (Stein, Walker, Hazen, & Forde, 1997), although this has not been observed uniformly across studies (Breslau, Lucia, & Davis, 2004).

Participants were excluded for factors known to affect neurocognitive functioning because these would interfere with understanding the relationship between cognitive functioning and PTSD. These exclusion criteria were: (1) current abuse of alcohol or other substances; (2) abuse of alcohol or other substances for a period of 5 years or more; (3) use of psychotropic medication within the last 4 weeks (fluoxetine within the last 6 weeks) or steroids in the past 4 months; (4) history of bipolar disorder, schizophrenia, attention deficit disorder, learning disability, loss of consciousness greater than 10 min or requiring hospitalization for 24 hr or more, or any neurological illness; and (5) English reading ability below the 8th grade level.

PTSD participants were not excluded if they had other mood disorders, such as major depressive disorder, because comorbid mood disorders are common among patients with PTSD and excluding them would have yielded a non-representative sample. NCs were excluded if they had any DSM-IV Axis I disorder. All participants gave informed written consent to participate in this study, which was approved by the University of California San Diego Human Research Protection Program and the VA San Diego Healthcare System Research and Development Review Committee.

Procedure

After obtaining verbal informed consent, a telephone interview was used to screen potential participants for the inclusion and exclusion criteria above and to gather demographic data. Callers were asked first about IPV history. If present, the Posttraumatic Stress Disorder Checklist - Civilian (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993) was administered with regard to the IPV history. This brief, widely used self-report instrument was used to quantify PTSD symptoms and assess the likelihood of PTSD. If the caller met all criteria, she was invited for an in-person interview (see measures below). If the caller denied IPV history, she was asked about other trauma history and screened for PTSD using stem questions from the PTSD section of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002).

To screen for other disorders, the telephone interview included stem questions from other diagnostic categories in the SCID-I. Screeners administered stem questions from the psychotic and bipolar disorder sections to all callers and additionally the major depression, panic, generalized anxiety, and PTSD stem questions to potential NCs. When a caller answered any of the stem questions in the positive, the screener administered the full section of the SCID for that disorder. Screeners were doctoral level clinicians.

All participants gave written informed consent at their initial in-person visit. IPV participants completed a set of diagnostic interviews in person, took home a set of questionnaires to complete, and were administered a neuropsychological assessment battery at a subsequent visit within 6 weeks. NCs underwent neuropsychological testing and completed the take-home questionnaires, but were not administered further diagnostic interviews.

Measures

IPV participants were administered the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), a standard semi-structured interview to assess PTSD status and severity. Respondents were asked to describe the most traumatic IPV event(s) they experienced in their most recent abusive relationship, to be used as the basis of assessing PTSD. The CAPS assesses the frequency and intensity of each of the 17 items from the DSM-IV criteria B, C, and D during the past month. The F1/I2 method of scoring was used in the current study to determine diagnostic status (Weathers, Keane, & Davidson, 2001), and a severity score was computed by summing the frequency and intensity scores over all 17 items. Ten percent of the CAPS outcome assessments were randomly selected throughout the study for audiotape review to establish inter-rater reliability. The intraclass correlation coefficient obtained on seven CAPS independently rated by two raters was .99 for the total score.

A take-home battery of self-report questionnaires was completed by all participants, including the following measures of child abuse history, current psychiatric symptom severity, and level of functioning. The Childhood Trauma Questionnaire (CTQ; Bernstein, Fink, Handelsman, & Foote, 1994) measures childhood maltreatment across five domains: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. The total score, ranging from 5 to 125, was used in the current study, with higher scores reflecting higher levels of childhood trauma. The Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996) was used to determine the presence and severity of symptoms of depression during the past week. Higher scores indicate greater severity of depressive symptoms. The Dissociative Experiences Scale Taxon (DES-T; Waller, Putnam, & Carlson, 1996) is a modified version of the Dissociative Experiences Scale that relies on a subset of eight categorical items that reflect abnormal or severe dissociative experiences. Scores on the DES-T range from 0 to 100, with higher scores reflecting higher levels of dissociative experiences. The Sheehan Disability Scale (SDS; Sheehan, 1983) assessed level of disablement using visual 0 to 10 rating scales and verbal descriptive anchors in three domains: work, social life, and family/home life. Higher scores on these three subscales reflect greater levels of impairment.

All participants underwent neuropsychological testing. Premorbid intellectual functioning was measured with the American National Adult Reading Test (ANART; Grober & Sliwinski, 1991). The neuropsychological battery emphasized executive functioning, using multiple subtests of the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001). The Wisconsin Card Sorting Test (Kongs, Thompson, Iverson, & Heaton, 2000) was used as a general measure of reasoning. Verbal learning and memory tests were not included because in our previous work, we found no difference between women with PTSD due to IPV and healthy comparison subjects on verbal learning and memory tests using story, list, or paired associate stimuli. The battery included assessments of multiple domains, including the following: (1) *Processing Speed* (scaled scores from the D-KEFS Trail Making Test Visual Scanning, Number Sequencing, Letter Sequencing, and Motor Speed conditions); (2) *Visuoconstruction* (Rey-Osterrieth Complex Figure Test copy *t* score; Rey, 1941; norms from Spreen & Strauss, 1998); (3) *Visual Memory* (Rey-Osterrieth Complex Figure Test 30-min delayed recall *t* score; Rey, 1941; norms from Spreen & Strauss, 1998); (4) *Speeded Fluency* (scaled scores from the D-KEFS Design Fluency Test [Combined

Filled Dots and Empty Dots], Letter Fluency, and Category Fluency); (5) *Inhibition* (D-KEFS Color-Word Interference Test Inhibition *vs.* Color Naming scaled score); (6) *Switching* (scaled scores from the D-KEFS Trail Making Test Number-Letter Switching *vs.* Combined Number Sequencing and Letter Sequencing Contrast Score; D-KEFS Design Fluency Test Switching *vs.* Combined Filled Dots and Empty Dots Contrast Score; D-KEFS Category Fluency Switching *vs.* Category Fluency Contrast Score; D-KEFS Color-Word Interference Test Inhibition/Switching *vs.* Inhibition Contrast Score); (7) *Reasoning* (Wisconsin Card Sorting Test-64 total errors *t* score [WCST]; Kongs et al., 2000).

The *t* scores (characterized in a normal population by a mean of 50 and standard deviation of 10) were used for the Rey-Osterrieth Complex Figure Test and for the WCST; scaled scores (characterized in a normal population by a mean of 10 and standard deviation of 3) were used for all other tests.

Analyses

All variables were normally distributed. To assess the extent to which our groups were demographically matched, we conducted *t* tests to compare groups on age and education, and χ^2 analyses to measure distribution differences in minority status. To reduce the possibility of Type I error, hypothesized differences between the two groups were tested with MANOVA (multivariate analysis of variance; when there was more than one test per neuropsychological domain) and *t* tests (when there was only one test per neuropsychological domain or when the MANOVA was significant). Associations between neuropsychological performance and symptom and disability severity were tested with Pearson correlations. Alpha for significance was set at .05 for the MANOVAs and .01 for all other tests, to reduce the chance of Type I error due to multiple comparisons.

RESULTS

The groups did not differ statistically on age, education, ethnic minority status, or premorbid intellectual functioning as measured by the ANART (see Table 1). Within the PTSD group, the mean CAPS score was 73.3 (*SD* = 21.9); the mean PCL-C score was 62.3 [*SD* = 11.7; CAPS scores above 60 and PCL-C scores above 50 are each suggestive of PTSD (Weathers et al., 1993, 2001)]; the mean BDI-II score was 17.9 (*SD* = 8.9), in the range of moderate depressive symptoms (Beck et al., 1996); the mean score on the DES-T was 11.0 (*SD* = 14.3), indicative of high average levels of dissociation (Seedat, Stein, & Forde, 2003). PTSD participants' mean CTQ score was 52.4 (*SD* = 20.4), suggesting that many participants had experienced childhood abuse. On the SDS, PTSD participants reported moderate levels of disablement in work, social, and home/family domains [means (*SDs*) were 5.7 (2.8), 6.7 (2.7), and 5.4 (2.9), respectively].

On the neuropsychological measures, within the PTSD group, the percentage of the sample that scored within the impaired range (<1.5 *SD* below the mean) was 13% on the Rey-Osterrieth copy, 17% on the Rey-Osterrieth delay, 17% on the WCST, and less than 10% on all other tests. By comparison, rates of impairment among the NC group were 14% on Rey-Osterrieth delay and less than 10% on all other tests.

In partial support of our first hypothesis, NCs outperformed participants in the PTSD group on most neuropsychological measures. However, these differences were only significant in the domains of processing speed and speeded fluency, with a trend toward significance in reasoning (see Table 1). Within the processing speed domain, the PTSD group performed worse than did the NC group on D-KEFS Trail Making Letter Sequencing and Motor Speed. Within the speeded fluency domain, the PTSD group performed worse than did NCs on D-KEFS Design Fluency and Letter Fluency. It is important to note that on all tests, the PTSD group's mean performance was within the average range.

Supporting our second hypothesis (see Table 2), we found that, within the PTSD group, more severe current PTSD symptoms (measured with the CAPS interview) were associated with lower processing speed (Trail Making Visual Scan; $r = -.36$; $p = .009$). More severe dissociative symptoms (DES-T) were associated with worse reasoning (WCST; $r = -.54$; $p < .001$). More severe childhood maltreatment (CTQ) was associated with worse performance on D-KEFS Category Fluency Switching *vs.* Category Fluency ($r = -.42$; $p = .005$). On the SDS, unexpectedly, self-reported family/home impairment was associated with better visuoconstruction performance (Rey-Osterrieth copy; $r = .51$; $p < .001$). Current level of depressive symptomatology (BDI-II) was not associated with performance on any neuropsychological measure.

DISCUSSION

Although we expected PTSD participants to perform significantly worse than would NCs on tasks of executive functioning, the pattern of results showed that they differed primarily on speeded tasks (some of which involved the executive functioning domain of fluency). Thus, our results are similar to those finding PTSD-associated deficits in processing speed and executive functioning in recent U.S. veterans of the Iraq and Afghanistan conflicts (Nelson, Yoash-Gantz, Pickett, & Campbell, 2009). Notably, the differences we found between groups do not appear to be attributable to depressive symptom severity, as BDI-II scores were not associated with any performance on neuropsychological measure. Our PTSD sample did not differ significantly from the NC sample on any of the switching tests in the D-KEFS. It is important to note that most group differences were in the direction of NCs outperforming PTSD participants; larger sample sizes may have resulted in more statistically significant differences. However, such differences between groups may have little clinical significance, considering that the mean performance of the PTSD group on all tests was in the average range. Importantly, as a group, they were not

Table 1. Comparison of demographic characteristics and cognitive function in the PTSD and NC groups (*p* values <.05 for MANOVAs and <.01 for other comparisons are highlighted in bold)

	PTSD		NC		Test statistic	df	p
	Mean	SD	Mean	SD			
Age, years	36.7	8.8	35.0	8.6	t = 0.74	73	.460
Education, years	14.0	1.9	14.6	1.5	t = 1.32	73	.191
	%		%				
% ethnic minority status	51		50		χ^2 = 0.005	1	.944
	Mean	SD	Mean	SD			
ANART	30.2	9.7	31.1	10.4	t = 0.25	44	.808
Processing Speed MANOVA			Hotelling's Trace F = 4.69			4,64	**.002**
Trail Making Visual Scanning	10.6	1.8	11.9	2.1	t = 2.61	68	.011
Trail Making Number Sequencing	10.3	2.6	10.7	2.3	t = 0.57	68	.572
Trail Making Letter Sequencing	9.5	2.8	11.7	1.1	t = 4.63	65.9*	**<.001**
Trail Making Motor Speed	10.6	1.7	12.0	1.7	t = 2.95	67	**.004**
Visuoconstruction							
Rey-Osterrieth Copy	49.0	13.4	52.5	11.8	t = 0.87	65	.388
Visual Memory							
Rey-Osterrieth Delay	45.5	9.0	44.6	7.6	t = 0.34	65	.737
Speeded Fluency MANOVA			Hotelling's Trace F = 4.71			3,66	**.005**
Design Fluency (Filled + Empty)	9.1	2.3	11.1	2.0	t = 3.20	68	**.002**
Letter Fluency	10.1	3.5	12.6	2.7	t = 2.69	68	**.009**
Category Fluency	11.0	3.3	11.8	3.5	t = 0.93	68	.354
Inhibition							
Color-Word Interference Inhibition vs. Color Naming	11.1	2.3	11.3	1.8	t = 0.34	68	.739
Switching MANOVA			Hotelling's Trace F = 0.47			4,65	.758
Trail Making Number-Letter Switching vs. Combined Number Sequencing + Letter Sequencing	9.0	2.7	9.2	1.9	—	—	—
Design Fluency Switching vs. Combined Filled + Empty	10.9	3.0	10.7	2.1	—	—	—
Category Fluency Switching vs. Category Fluency	11.1	3.1	10.6	2.9	—	—	—
Color-Word Interference Inhibition/ Switching vs. Inhibition	9.3	2.9	10.2	2.3	—	—	—
Reasoning							
Wisconsin Card Sorting Test total errors *t* score	45.1	9.0	51.3	5.9	t = 2.40	59	.020

Note. ANART = American National Adult Reading Test; MANOVA = multivariate analysis of variance; PTSD = posttraumatic stress disorder group; NC = normal comparison group; SD = standard deviation.
*degrees of freedom adjusted for unequal variances

impaired on any test. Although individuals with PTSD frequently present with subjective cognitive difficulty, previous research has found that cognitive complaints tend to be related more to psychiatric symptom severity than to actual cognitive performance (Binder, Storzbach, Anger, Campbell, & Rohlman, 1999; Garcia et al., 2004). Indeed, some well-controlled research on combat veterans with PTSD found no neuropsychological differences between patients with current PTSD, past PTSD, psychiatric comparison subjects, and healthy comparison subjects, despite large sample sizes (80 per group; Crowell et al., 2002). We found that higher levels of PTSD symptoms were associated with worse speeded attention, and higher levels of dissociation were associated with poorer reasoning performance. These results are similar to previous work finding associations between dissociation severity and greater Stroop interference (Freyd, Martorello, Alvarado, Hayes, & Christman, 1998).

Why would PTSD adversely affect processing speed? Cognitive slowing may be attributable to reduced attentional resources, which could be a consequence of brain resources being directed toward coping with psychological distress, unpleasant internal experiences (see DePrince & Freyd, 1999, 2004), or potential threats in the environment (i.e., hypervigilance) rather than the task at hand. In effect, even simple tasks could become exercises in multitasking. If people with PTSD experience micro-dissociations when confronted with intrusive memories, for example, reduced attentional resources could yield slower performance on processing speed measures. Other factors that could influence speeded tasks include sleep deficits, heightened anxiety or

Table 2. Correlations between cognitive performance and clinical variables within the PTSD group (correlation, *p* value, n; p-values <.01 are highlighted in **bold**)

	CTQ	CAPS	BDI-II	DES-T	SDS-Work	SDS-Social	SDS-Home/Family
Processing Speed							
Trail Making Visual Scanning	−.033	**−.363**	−.018	.053	−.172	−.166	.018
	.831	**.009**	.905	.733	.269	.282	.906
	44	**51**	46	44	43	44	44
Trail Making Number Sequencing	.145	−.048	.017	−.139	−.022	−.042	.047
	.348	.739	.913	.367	.889	.788	.763
	44	51	46	44	43	44	44
Trail Making Letter Sequencing	.217	−.079	.069	−.384	−.166	−.160	−.117
	.158	.581	.647	.010	.286	.300	.449
	44	51	46	44	43	44	44
Trail Making Motor Speed	.126	.039	−.115	−.087	.015	−.084	−.091
	.421	.790	.451	.581	.924	.592	.560
	43	50	45	43	42	43	43
Visuoconstruction							
Rey-Osterrieth Copy	−.180	.064	−.037	.177	.036	.301	**.505**
	.237	.653	.804	.245	.815	.044	**<.001**
	45	52	47	45	44	45	**45**
Visual Memory							
Rey-Osterrieth Delay	.096	.149	.017	.165	−.031	.048	.227
	.532	.291	.912	.280	.841	.757	.134
	45	52	47	45	44	45	45
Speeded Fluency							
Design Fluency (Filled + Empty)	.191	−.039	.241	.021	.174	.100	−.133
	.215	.785	.107	.893	.265	.519	.389
	44	51	46	44	43	44	44
Letter Fluency	.377	−.151	−.015	−.120	.093	−.071	−.301
	.012	.289	.920	.437	.555	.645	.047
	44	51	46	44	43	44	44
Category Fluency	.225	−.133	.210	−.056	.151	.178	−.047
	.142	.352	.162	.719	.335	.249	.760
	44	51	46	44	43	44	44
Inhibition							
Color-Word Interference	−.088	.103	.160	−.009	.077	.117	.133
Inhibition vs. Color Naming	.569	.471	.287	.955	.624	.451	.389
	44	51	46	44	43	44	44
Switching							
Trail Making Switching vs.	−.037	.079	.257	.079	.189	.061	.036
Combined Number Sequencing +	.811	.580	.085	.609	.225	.693	.819
Letter Sequencing	44	51	46	44	43	44	44
Design Fluency Switching vs.	−.079	.074	−.264	.031	−.307	−.269	.105
Combined Filled + Empty	.612	.604	.077	.843	.046	.078	.497
	44	51	46	44	43	44	44
Category Fluency Switching vs.	**−.417**	.012	−.122	.121	−.150	−.070	.027
Category Fluency	**.005**	.931	.418	.433	.338	.651	.862
	44	51	46	44	43	44	44
Color-Word Interference Inhibition/	.088	.126	.078	−.103	.016	.042	−.100
Switching vs. Inhibition	.570	.377	.607	.507	.918	.788	.519
	44	51	46	44	43	44	44
Reasoning							
Wisconsin Card Sorting Test	−.095	−.039	.212	**−.544**	−.047	−.004	−.130
total errors t-score	.564	.795	.190	**<.001**	.778	.979	.429
	39	46	40	**39**	38	39	39

Note. BDI-II = Beck Depression Inventory-II; CAPS = Clinician-Administered PTSD Scale; CTQ = Childhood Trauma Questionnaire; DES-T = Dissociative Experiences Scale-Taxon; SDS = Sheehan Disability Scale.

arousal due to the timed nature of the task, or exposure to recent PTSD-related triggers. Indeed, a recent event-related potential study linked heightened arousal to slowed central processing and attention problems in PTSD (Shucard, McCabe, & Szymanski, 2008). Importantly, we did not find that poor performance on any test was correlated with increased level of self-reported disability (on the Sheehan Disability Scale). While this finding did not support our prediction, it is in keeping with our inference that the cognitive dysfunction seen in our PTSD sample is, at most, subtle, and unlikely to be clinically meaningful at a group level. At minimum, our failure to find a relationship between cognitive test performance and functional disability raises questions about the clinical relevance of neuropsychological dysfunction in PTSD.

Our study has several limitations. Although we restricted our sample to female victims of IPV to better understand the nature of neuropsychological dysfunction from this type of trauma, narrowing our sample also limits the generalizability of our results. However, our sample also allowed us to control for some of the common confounds (e.g., substance abuse, multiple trauma types within the same sample) in PTSD research. Although all of the women in this study met PTSD criteria based on exposure to IPV, some women had also experienced additional traumatic events. Future studies should include resilient groups of individuals with trauma exposure but without PTSD to clarify whether neuropsychological differences are associated with trauma exposure, PTSD, or both. Although we excluded participants with probable learning disability and those with moderate to severe head injury, individuals with mild traumatic brain injury from repeated, brief losses of consciousness of <10 min may have been included. Given data from the recent U.S. Iraq and Afghanistan military conflicts suggesting that mild traumatic brain injury may be associated with mental disorders such as PTSD (Hoge et al., 2008; Schneiderman, Braver, & Kang, 2008), and given the rates of mild TBI in survivors of IPV (Jackson, Philip, Nuttall, & Diller, 2004), it will be especially important to account for such potential associations in future research. Smoking status was not measured in our study, but given the links between acute smoking and cognitive performance (Evans & Drobes, 2009), smoking status should be measured in future studies. We included multiple measures of cognitive function across several domains. We attempted to mitigate the likelihood of false-positive results by limiting our initial between-groups comparisons to the functional domains, and then constraining attribution of statistical significance to *post hoc* and correlational analyses with p values < .01. Nonetheless, it is possible that some of our findings represent Type I errors. By the same token, it is also possible that, given our modest sample sizes, that some negative findings represent Type II errors. Larger studies, focused on the domains of cognitive function most consistently found to be abnormal in PTSD, will be needed. Finally, with our cross-sectional sample, we cannot make any inferences regarding the temporal precedence of our results. It is possible that the poorer neuropsychological performance of the PTSD group is a consequence of PTSD or trauma exposure, but it is also possible that it reflects pre-existing differences in cognitive reserve that might increase risk for PTSD (Koenen et al., 2009).

In summary, our results suggest that women with PTSD due to IPV perform slower than do healthy women on speeded graphomotor tasks and speeded fluency tasks. Although the mean performance of the PTSD group was solidly within the normal range, rates of impairment on various tests were higher in the PTSD group. Thus, a subset of women in the PTSD group may experience cognitive impairment, which can have implications for real-world functioning situations that require speeded processing (e.g., responding quickly when driving, solving problems quickly at work, or working quickly with one's hands). Because most measures of symptoms and functioning were not associated with neuropsychological performance, more detailed modeling will be needed to identify risk factors for cognitive impairment in these women. It will also be important to assess whether appropriate treatment of PTSD symptoms results in cognitive improvement. Future research will examine the trajectory of neuropsychological performance and subjective cognitive difficulty over the treatment course of PTSD.

ACKNOWLEDGMENT

This research was supported by a grant from the Veterans Affairs (VA) Merit Review Program to Murray B. Stein. The authors have no conflicts of interest.

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Neuropharmacology 62 (2012) 686–694

Contents lists available at SciVerse ScienceDirect

Neuropharmacology

journal homepage: www.elsevier.com/locate/neuropharm

Invited review

Executive function and PTSD: Disengaging from trauma

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ARTICLE INFO

Article history:
Received 2 December 2010
Accepted 7 February 2011

Keywords:
Neuropsychology
Neuroimaging
Posttraumatic stress disorder
Executive function
Attention
Inhibition

ABSTRACT

Neuropsychological approaches represent an important avenue for identifying susceptibility and resiliency factors relating to the development and maintenance of posttraumatic stress disorder (PTSD) symptoms post-trauma. This review will summarize results from prospective longitudinal and retrospective cross-sectional studies investigating executive function associated with PTSD. This research points specifically towards subtle impairments in response inhibition and attention regulation that may predate trauma exposure, serve as risk factors for the development of PTSD, and relate to the severity of symptoms. These impairments may be exacerbated within emotional or trauma-related contexts, and may relate to dysfunction within dorsal prefrontal networks. A model is presented concerning how such impairments may contribute to the clinical profile of PTSD and lead to the use of alternative coping styles such as avoidance. Further neuropsychological research is needed to identify the effects of treatment on cognitive function and to potentially characterize mechanisms of current PTSD treatments. Knowledge gained from cognitive and neuroscientific research may prove valuable for informing the future development of novel, more effective, treatments for PTSD.

This article is part of a Special Issue entitled 'Post-Traumatic Stress Disorder'.

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"*The significant problems we face in life cannot be solved at the level of thinking that created them.*"
– *Albert Einstein*

1. Introduction

An estimated 50–60% of people will experience a serious trauma—as a result of combat, sexual assault, major accidents, or other real-life horrors—at some point in their lives (Kessler et al., 1995). However, only 5–10% of people are estimated to develop symptoms qualifying them for diagnosis of posttraumatic stress disorder (PTSD). This observation has led researchers to consider what factors other than the trauma itself may contribute to, or protect against, the development and maintenance of PTSD symptoms. Neuropsychological approaches may provide an important insight into susceptibility and resiliency factors by identifying pre-trauma cognitive functions that relate to subsequent development of PTSD as well as posttraumatic cognitive processes that may influence development or maintenance of the disorder. Finally, understanding these cognitive processes may provide new approaches for treatment to improve long-term outcomes of individuals with PTSD.

Although much of neuropsychological research in PTSD has focused on learning and memory, there has also been an accumulation of research examining potential "frontal lobe" or executive dysfunction. William James, in *The Principles of Psychology*, defined attention as "the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought" (James, 1890). He went on to say that "…It implies withdrawal from some things in order to deal effectively with others." In the modern world of neuropsychology and cognitive neuroscience, there are many disagreements about distinct and common definitions of attention, working memory, and executive function. For the purposes of this manuscript, we will focus on concepts that have been considered throughout the literature to be involved in maintaining successful "executive function", or the control of complex goal-directed behavior (Royall et al., 2002; Alvarez and Emory, 2006; McCabe et al., 2010). This includes 1) *attention*, or the voluntary allocation of processing resources or focusing of one's mind on a particular stimulus within the environment, 2) *working memory*, or the active maintenance and manipulation of information in one's mind over a short period of time, 3) *sustained attention*, or the maintenance of attention

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0028-3908/$ – see front matter Published by Elsevier Ltd.
doi:10.1016/j.neuropharm.2011.02.008

on one set of stimuli or a task for a prolonged period, 4) *inhibitory function*, involving the inhibition of automatic responses to maintain goal-directed behavior, 5) *flexibility/switching*, or the ability to switch between two different tasks or strategies, and 6) *planning*, or the ability to develop and implement strategic behaviors to obtain a future goal (Smith and Jonides 1999; McCabe et al., 2010; Carlson et al., 2005; Salthouse et al., 2003; Miyake and Shah 1999; Repovs and Baddeley, 2006).

We chose to focus on attentional and executive functions for the current review, rather than learning and memory, for two primary reasons. First, there have been several recent reviews summarizing findings related to learning and memory in PTSD—both in regards to neutral information as well as emotional information, such as with fear conditioning and extinction (Rubin et al., 2008; Johnsen and Asbjornsen, 2008; Moore, 2009). The second reason we chose to focus on executive and attentional functions is because recent research indicates that attentional modification programs may be beneficial in the treatment of anxiety disorders (Amir et al., 2009a; Li et al., 2008; Schmidt et al., 2009; Amir et al., 2009b; Najmi and Amir, 2010; Amir et al., 2008). This suggests that research related to attention and working memory function may not only increase our understanding of PTSD, but may also lead to more effective treatments for these patients.

The majority of neuropsychological research in PTSD uses cross-sectional designs from which it is impossible to determine whether any observed cognitive dysfunctions represent pre-trauma risk and resiliency factors or if they represent responses to the experience of trauma or PTSD. We therefore begin our review by discussing results from longitudinal and twin studies that may shed some light on this issue. We then synthesize results from cross-sectional studies concerning "frontal lobe" dysfunction associated with PTSD, focusing on simple attention and working memory, "higher-order" executive functions, flexibility, and inhibition, and the effects of emotional context on attention and executive function. The focus of this discussion will be on how difficulties regulating attention and inhibiting responses to stimuli (particularly emotional or trauma-related stimuli) could contribute to the clinical profile of PTSD—for example, leading to the development of alternative, potentially maladaptive, coping mechanisms. We will also discuss research concerning potential neural substrates of executive dysfunction, and the relationship between treatment and cognitive dysfunction, in PTSD. This review will not discuss the issue of comorbid disorders (e.g., traumatic brain injury, substance use disorders) and their potential influence on neurocognitive function in PTSD. We recognize the significant influence comorbid conditions can have on neurocognitive function and refer to recent articles focusing on this important and complex issue (Stein and McAllister, 2009; Samuelson et al., 2006).

2. Cognitive risk factors versus sequelae of PTSD

Although historically considered a controversial issue, research examining cognitive risk and resilience factors could be invaluable in understanding mechanisms for PTSD and in developing better preventive and treatment interventions. Lower IQ (often measured via military aptitude test performance) and educational achievement pre-trauma has been reported to relate to PTSD symptoms post-trauma (Gale et al., 2008; Macklin et al., 1998; Green et al., 1990; Pitman et al., 1991; Thompson and Gottesman, 2008; Vasterling et al., 2002). In most studies, the IQ range for individuals later developing PTSD, though lower than controls, is within the normal range. It has therefore been suggested that premorbid cognitive risk factors of PTSD may be relatively subtle, or relate to specific deficits in circumscribed areas measures that are difficult to ascertain retrospectively.

Recently, prospective longitudinal studies have been initiated, involving more fine-tuned assessment pre- and post- trauma (e.g., combat deployment). Parslow and Jorm (Parslow and Jorm, 2007) reported that pre-trauma performances on immediate and delayed verbal recall (California Verbal Learning Test [CVLT]) (Delis et al., 1988), working memory (digit span backward), visuomotor speed (Symbol Digit Modalities Test [SDMT]) (Smith, 1982), and verbal intelligence (National Adult Reading Test [NART]) (Nelson, 1982; Nelson and Willison, 1991) was negatively related to post-trauma PTSD re-experiencing and arousal symptoms. Marx et al. (Marx et al., 2009) conducted a similar study with veterans, and reported that pre-deployment visual immediate recall performance (Wechsler Memory Scale) (Wechsler, 1997b) was negatively related to PTSD symptoms post-deployment.

Twin studies also have the ability to highlight potential pre-trauma risk and resilience factors in PTSD (Kremen et al., 2007; Gilbertson et al., 2006). Gilbertson et al. examined neuropsychological functioning in combat-exposed individuals with and without PTSD and their high-risk and low-risk monozygotic twins. Measures of overall IQ, verbal memory (immediate and delayed recall), attention (digit span) (Wechsler, 1987), and executive function (Wisconsin Card-Sorting Test [WCST]) (Heaton, 1981) performance was decreased not only for the PTSD group, but also their twins, compared to the non-PTSD group and their twins. These results provide further support that lower pre-trauma cognitive functioning—particularly in domains of attention, executive function, and memory—may serve as a risk factor for the development of PTSD.

However, there is also evidence that pre-trauma cognitive function does not completely account for post-trauma cognitive deficits. Studies examining neuropsychological differences between individuals with and without PTSD have reported cognitive function (e.g., learning and memory) to correlate with PTSD severity above and beyond that accounted for by premorbid IQ (Vasterling et al., 2002; Gilbertson et al., 2001). It is likely there are both pre-trauma and acquired differences in cognitive function associated with PTSD. A decrease in specific cognitive functions pre-trauma may not only influence the development of PTSD, but may itself be exacerbated by the experience of trauma. The experience of trauma could cause subtle pre-trauma cognitive deficits to morph into more significant symptoms detectable not only during sensitive neuropsychological assessment, but also to patients as they try to function in their daily lives. Although cross-sectional neuropsychological studies do not provide insight into etiology, they provide important information concerning the resulting deficits associated with PTSD—which may relate not only to individuals' daily functioning and clinical symptoms, but also potentially to treatment outcome.

3. Attention, working memory, and executive function in PTSD

3.1. Attention and working memory

Attention and *working memory* are often measured using digit span, one-trial word recall (e.g., CVLT Trial 1), and spatial span (e.g., Crosi blocks) (Milner, 1971) tasks. These tasks require individuals to attend to a series of presented digits, words, or spatial locations and immediately recreate sequentially what was presented. Tasks such as digit span backward and letter-number sequencing (Wechsler, 1997a; Wechsler, 2008) require increased *working memory* load, as they involve greater manipulation of information held in one's mind. Decreased performance on measures of auditory attention and working memory have been found in combat- and sexual assault- related PTSD when compared to victims without PTSD and

non-trauma controls (Samuelson et al., 2006; Brandes et al., 2002; Gilbertson et al., 2001; Vasterling et al., 1998, 2002; Marmar et al., 2006; Lagarde et al., 2010; Gilbertson et al., 2001; Jenkins et al., 2000), and these deficits have been reported to correlate with PTSD symptom severity (Burriss et al., 2008). However, it should be noted that several studies have failed to identify auditory attention deficits in PTSD (Vasterling et al., 1998; Neylan et al., 2004; Samuelson et al., 2006; Leskin and White, 2007). Research has also failed to identify impairments in *visual* attention and working memory associated with PTSD (Samuelson et al., 2006; Jenkins et al., 2000). Therefore, it seems there is evidence, albeit inconsistent, that mild deficits in simple auditory attention and working memory may be one aspect of the cognitive profile of PTSD. However, it is unclear whether PTSD is associated with primary problems in attention and working memory, or whether the inconsistent findings are due to difficulties coping with and inhibiting unintentional "distracters", such as internal (e.g., emotions, cognitions) or external stimuli (e.g., environmental sounds and sights; stimuli presented in previous tasks).

3.2. Sustained attention and inhibitory functions

Sustained or selective attention is often measured via continuous performance tasks (CPT) (Loong, 1988; Conners, 1992), requiring individuals to attend to a long series of auditory or visual stimuli and respond (via a button press) when a target stimulus is presented. Studies have repeatedly found PTSD patients to exhibit impaired performance (e.g., increased omissions, commissions, or reaction time) in auditory and visual sustained attention (Vasterling et al., 1998; McFarlane et al., 1993; Wu et al., 2010; Shucard et al., 2008; Jenkins et al., 2000; Vasterling et al., 2002); except see (Golier et al., 1997), and the number of correct hits has been reported to negatively correlate with PTSD symptom severity (Vasterling et al., 2002). It should be noted that many studies using CPT tasks to assess performance in PTSD have reported increased errors of commission to distracter stimuli (Wu et al., 2010; Vasterling et al., 1998), suggesting difficulty with *inhibition* of automatic responses. Other measures requiring *inhibition* of responses include the go-nogo, stop-signal, and attention network (ANT) tasks (Shucard et al., 2008; Jenkins et al., 2000). Decreased inhibitory function has rather consistently been reported for PTSD (Falconer et al., 2008; Wu et al., 2010; Koso and Hansen 2006; Casada and Roache 2005; Shucard et al., 2008; Bressan et al., 2009; Jenkins et al., 2000; Leskin and White, 2007), and performance has been reported to relate to PTSD symptom severity (Falconer et al., 2008; Leskin and White, 2007). The color-word Stroop task examines response time to name the ink color of a color-related word (e.g., "red" printed in blue ink) and is also thought to be a measure of inhibitory function. Impaired performance on the color-word Stroop has been reported for various PTSD populations, though it is unclear whether such a deficit is specific to PTSD or a more general impairment across psychiatric disorders (Lagarde et al., 2010; Litz et al., 1996). Interestingly, several studies have also reported PTSD to be associated with increased intrusions during memory recall (Vasterling et al., 1998; Lindauer et al., 2006), which may reflect difficulty inhibiting related, but non-relevant internally-generated stimuli. Interestingly, Vasterling et al. (Vasterling et al., 1998) found the tendency to intrude information across various cognitive tasks (commissions on sustained attention and intrusions on memory measures) related to severity of re-experiencing and hyperarousal symptoms.

There seems to be growing evidence to support PTSD being associated with inhibitory dysfunction—through comparisons with control groups and correlations with symptom severity measures. Such inhibitory dysfunction may specifically relate to re-experiencing and hyperarousal symptoms. However, it is difficult to determine the directionality of this effect, given these studies rely primarily on cross-sectional designs. Heightened arousal and re-experiencing symptoms could create more distracters when an individual is attempting to concentrate on the task at hand, thereby interrupting working memory, sustained attention, and inhibitory functions. However, it is also possible that primary inhibitory dysfunction could result not only in decreased performance on cognitive tasks, but also impaired ability to inhibit emotional memories and physiological arousal in response to triggers.

3.3. Flexibility/switching and planning

The ability to shift between different tasks is an essential aspect of executive control. Classical measures of flexibility and switching include the Trail-Making Test (TMT, Partington and Leiter, 1949; Reynolds, 2002; Delis, et al 2001; Reitan, 1958), involving connection of "dots" while switching between letter and number (i.e., 1-A-2-B-3-C), and verbal fluency switching (as in the Delis-Kaplan Executive Function Scale [D-KEFS]) (Delis et al., 2001), involving the production of words while switching between two categories. Some studies with PTSD have reported impairment (e.g., increased time on TMT; decreased total words on fluency) on such tasks (Stein et al., 2002; Beckham et al., 1998; Jenkins et al., 2000), while others have not (Zalewski et al., 1994; Twamley et al., 2004, 2009; Lagarde et al., 2010; Barrett et al., 1996; Crowell et al., 2002; Gurvits et al., 1993; Leskin and White, 2007). Executive function measures involving the added dimensions of *planning* and strategy use include, among others, the Wisconsin Card-Sorting Test (WCST) (Heaton, 1981) and Tower of London Task (Simon, 1975; Shallice, 1982). For the most part, no consistent deficits on these measures have been reported (Vasterling et al., 1998; Lagarde et al., 2010). Although Kanagaratnam and Asbjornsen (Kanagaratnam and Asbjornsen, 2007) reported PTSD to be associated with increased number of trials to complete the first category of the WCST (indicating deficits in initial problem solving), they found no impairment on overall performance. A similar finding was reported by Twamley et al. (Twamley et al., 2009), who found PTSD to be associated with increased trials to complete the first category, but with increased overall learning efficiency. It can be argued that the WCST involves switching and flexibility similar to the Trail Making Test. However, tests such as TMT are timed and require quick attentional switching between pre-defined tasks. The WCST on the other hand is untimed and requires initial production of a strategy and subsequent switching of *strategies*—rather than flexibility and quick switching of *attention* (as with the TMT). Neuropsychological research therefore seems to provide inconsistent support for impairment in speed-reliant, attentional switching, but indicates that planning, rule-learning, and untimed strategy switching, may be mostly spared in PTSD.

3.4. Influence of emotional factors on executive function in PTSD

Although research examining "cognitive" and "emotional" aspects of learning in PTSD have traditionally been kept separate, it is clear these interact with one another and are intrinsically intertwined. Additionally, neural systems responsible for executive control within affective or neutral situations are most likely non-distinct and overlapping. Reflective of this, research has begun to examine cognitive function in PTSD when trauma-relevant, affective but trauma-irrelevant, or other highly-valued stimuli are involved.

PTSD has repeatedly been associated with attentional biases towards threat and negative emotional stimuli, as exhibited by

performance on the modified Stroop (Williams et al., 1996), which involves timed verbalization regarding the ink color of emotional and neutral words (Mathews and MacLeod, 1985), and dot-probe tasks (MacLeod et al., 1986), in which targets are displayed in locations closer to previously-presented trauma-relevant, generally negative, or neutral stimuli (Dalgleish et al., 2003; Foa et al., 1991; McNally et al., 1990; Chemtob et al., 1999; Mueller-Pfeiffer et al., 2010; Kimble et al., 2010); except see (Bremner et al., 2004; Kimble et al., 2009). Observed performance differences on these tasks could be due to either attentional facilitation involving *enhanced detection* of threat-relevant stimuli, or attentional interference involving *difficulty disengaging* from threat-related stimuli to focus attention on the task at hand. Recent studies suggest the bias may be most associated with attentional interference, indicating potential underlying dysfunction in disengagement and inhibition (Pineles et al., 2007, 2009).

Studies on PTSD have also examined the effects of symptom provocation prior to completion of a cognitive task (Jelinek et al., 2006, 2008). These studies corroborated previous neuropsychological findings of general working memory dysfunction in PTSD. However, results indicate that recall of emotional autobiographical events does not influence working memory function any more for PTSD patients than controls. Other studies have examined the effect of inserting emotional images into the working memory task as distractors (e.g., directly prior to number Stroop decisions or in between encoding and recall for working memory tasks). Results from these studies have been mixed, with one study reporting PTSD to be associated with worse detectability scores across working memory trials regardless of distractor type (neutral vs. trauma-related) (Morey et al., 2009), while another study reported PTSD patients to exhibit greater response latency for number Stroop after negative (versus positive or neutral) images as compared to trauma-exposed and non-trauma-exposed control groups (Mueller-Pfeiffer et al., 2010). These results suggest that the acute emotional state may not have an overwhelming effect on objective cognitive function in PTSD. Instead, these studies support the existence of underlying deficits in working memory that in some situations can be worsened by the inclusion of emotional distractor stimuli.

Decision making involves comparative valuation of potential choices and presented stimuli. Many decision making tasks (e.g., Iowa Gambling Task) have been proposed to involve not only rational input regarding the objective value of choices, but also an "emotional" or "somatic" input directing an individual towards one choice over another (Bechara et al., 2000, 2003). Decision making paradigms therefore offer a unique way of examining the influence of PTSD on functions involving affective input, valuation, and cognitive resources. Two studies have been conducted thus far to examine decision making in PTSD patients, both of which found PTSD to be associated with an increase in the number of trials needed to learn optimal patterns of responding (Sailer et al., 2008; Koenen et al., 2001). However, these tasks involve various components that, if disrupted, could account for the impairment observed in PTSD: 1) lack of motivation or reward-seeking, 2) impaired learning of response-outcome associations, or 3) lack of disengagement from a non-optimal response strategy. Further research is therefore needed to clarify these findings.

The most consistent finding in the emotional-cognition PTSD literature is on inhibitory tasks requiring quick, in-the-moment disengagement from emotional stimuli, as is involved in the emotional Stroop and dot-probe tasks. Increased hypervigilance towards threat-related materials may serve to enhance attention and reaction time to stimuli presented in the same locale. Recent evidence suggests that difficulty with subsequent *disengagement* from threat-related material may be a primary culprit influencing attention and executive dysfunction in PTSD. Obviously, if there was no initial hypervigilance towards threat, there would be no need to disengage. However, it may be possible to experience hypervigilance towards threat but retain the ability to disengage or regulate that attention. Potentially, the ability to disengage from even highly-valued stimuli could serve as a resiliency factor for preventing the development and maintenance of PTSD.

4. Neural correlates of attention and executive function in PTSD

Neuroimaging studies (using positron emission tomography [PET] or functional magnetic resonance imaging [fMRI]) in PTSD have primarily focused on symptom provocation or responses to trauma-related or emotional stimuli. These results have been discussed in recent reviews (Shin and Liberzon 2010; Liberzon and Sripada, 2008; Francati et al., 2007) and meta-analyses (Etkin and Wager, 2007) and suggest hyperactivation within limbic regions (particularly amygdala and insula) and hypoactivation of prefrontal regions, including anterior cingulate (ACC; including both rostral and dorsal) and ventromedial prefrontal cortex (vMPFC). A small collection of studies have been conducted to examine neural substrates of executive functions in PTSD. These studies have focused on 1) sustained attention using oddball tasks, 2) inhibitory functions using go-nogo, n-back, or continuous performance tasks, or 3) inhibitory functions during tasks involving emotional stimuli.

fMRI studies with healthy adults have shown that tasks requiring sustained attention activate medial PFC and ACC as well as parietal cortex (Kirino et al., 2000; Yamasaki et al., 2002; Fichtenholtz et al., 2004; Morey et al., 2008; Bledowski et al., 2010; Clark et al., 2000; McCarthy et al., 1997; Menon et al., 1997; Yoshiura et al., 1999), while those involving inhibitory functions activate areas of the inferior frontal cortex (IFC), lateral PFC (including ventrolateral [vlPFC] and dorsolateral [dlPFC]), and ventromedial or orbitofrontal cortex (OFC) (Aron et al., 2003; Garavan et al., 1999; Menon et al., 2001; Kiehl et al., 2000; Konishi et al., 1998; Liddle et al., 2001; Rubia et al., 1998; Bledowski et al., 2010). The lateral PFC specifically has been implicated in response inhibition—whether it be emotional or non-emotional contexts (Compton et al., 2003; Bledowski et al., 2010). However, the ACC may have some specialization in this regard, as more ventral regions are thought to be primarily involved in inhibition of responses to emotional stimuli, while more dorsal regions are thought to be involved in the inhibition of neutral information (Whalen et al., 1998; Bush et al., 1998; Mohanty et al., 2007; Yamasaki et al., 2002; Fichtenholtz et al., 2004).

PTSD has been associated with *increased* activation in dorsal ACC and other PFC regions during an auditory oddball task (Bryant et al., 2005). However, during the go-nogo task, PTSD patients exhibited *reduced* activation in the inferior frontal and ventral and dorsal lateral PFC, as well the medial OFC (Falconer et al., 2008). Activation in these areas was negatively correlated with PTSD symptom severity and rate of commission errors, suggesting that attenuated activations were not related to compensatory mechanisms but instead to the observed impairments in performance. Discrepancies between these two studies may be due to the varying degrees of working memory and inhibition required by the tasks. Along these lines, Moores et al. (Moores et al., 2008) used a task with conditions of working memory "maintenance" requiring subjects to maintain attention and respond to pre-specified target stimuli ("fixed target") and "updating" requiring subjects to respond to stimuli that matched that presented directly before it (i.e., 1-back task, "variable target"). Thus, the "updating" condition involved greater working memory load and inhibition of responses. For the updating condition, PTSD subjects had *decreased* activation in several PFC regions, including dlPFC, ACC, and inferior frontal cortex, as well

as the insula. During maintenance, there were no significant differences between groups, but there was a trend noted towards *increased* activation in lateral PFC, inferior frontal cortex, and insula. It therefore seems that PTSD may be associated with hyperactivation of prefrontal areas in response to simple sustained attention tasks, but relative hypoactivation during tasks involving inhibition or "updating". The former could reflect the hypervigilance and enhanced attention towards "triggers" associated with PTSD, while the latter could relate to decreased ability to control or inhibit these attentional resources. Alternatively, hyperactivation during sustained attention could reflect compensatory activation to maintain attention during more simple tasks, which hits a ceiling or breaks down as working memory load increases, thus failing to compensate further for more complex inhibition or "updating" tasks.

Studies involving the presentation of emotional "distracters" during working memory tasks have reported PTSD to be associated with increased activation in ventral PFC regions (e.g., ventromedial PFC) during processing of emotional distracters, but decreased activation in dorsal PFC regions (e.g., dorsal ACC, dlPFC) and parietal cortex (Morey et al., 2008, 2009; Pannu Hayes et al., 2009) during the working memory task itself. Similarly, PTSD has been associated with reduced medial PFC and rostral ACC activation during the emotional Stroop (Bremner, 2001; Shin et al., 2001). These results could be taken as support for an overactive ventral/limbic "emotional" processing stream that interferes with more dorsal prefrontal "cognitive" processing streams. However, a somewhat different interpretation could be that PTSD is associated with difficulties recruiting those regions necessary to disengage from highly-valued stimuli (e.g., dorsolateral PFC, dorsal ACC). PTSD may therefore relate to the combination of enhanced "emotional" processing networks that serve to enhance attention towards specific stimuli and decreased "inhibitory" networks meant to disengage attention and redirect it to the task at hand.

As discussed, decision making paradigms require not only attention and working memory, but also the integration of outcome valuations, and the inhibition of automatic responses (Hare et al., 2010; Kim and Lee, 2010). There is a rather extensive neuroimaging literature attempting to tease apart the various aspects of decision making and this literature primarily implicates regions of the PFC, including OFC, ACC, and dlPFC, as well as striatal systems (Rangel et al., 2008; Hare et al., 2010; Kim and Lee, 2010)—regions which have exhibited dysfunction in PTSD. Decision making paradigms therefore offer a useful framework for teasing apart PFC dysfunction in PTSD and other anxiety disorders (Aupperle and Paulus, 2010). However, there has only been one study that has used fMRI to investigate neural substrates of decision making in PTSD. Sailer et al. (Sailer et al., 2008) reported that PTSD patients were slower to learn an optimal response pattern during decision making, and also showed attenuated activation in the nucleus accumbens in response to reward. This suggests PTSD may be associated with dysfunction in reward system networks—which could contribute to decreased motivation and reward-seeking.

5. Treatment and cognitive function in PTSD

Cognitive processes may relate to treatment in a number of different ways. First, cognitive function may be used as a predictor of treatment outcome, or as a treatment outcome measure in and of itself. Second, cognitive factors may themselves be treatment targets to improve clinical symptoms. Thus far, there has been one study published to examine the predictive utility of cognitive function in the treatment of PTSD. Wild et al. (Wild and Gur, 2008) reported that performance on immediate recall for stories, above and beyond initial treatment severity and even attentional measures, significantly predicted improvement in symptoms after cognitive behavior therapy. Surprisingly, there have also been very few studies reporting the effects of PTSD treatment on neuropsychological function. Vermetten et al. (Vermetten et al., 2003) reported significant improvement on verbal memory after paroxetine treatment, but this study did not include a control group for comparison. Fani et al. (Fani et al., 2009) used a double-blind, placebo-controlled design and reported paroxetine treatment to be associated with a non-significant trend toward improved verbal declarative memory. Walter et al. (Walter et al., 2010) reported that trauma-focused therapy (e.g., cognitive processing therapy, prolonged exposure therapy), for a small group of women ($N = 10$), resulted in significant improvement on TMT number-letter switching and visual organization (Rey-Osterrieth Complex Figure task) (Osterrieth, 1944; Rey, 1941), with a trend towards overall improvement in executive function. Another study investigated the effects of psychotherapeutic treatment on emotional Stroop performance in PTSD and found no significant effects (Devineni et al., 2004). Due to the small number of studies using varied methodologies (e.g., medication vs. therapy; varying neuropsychological measures), it is difficult to reach any firm conclusions regarding PTSD treatment effects on neuropsychological function. Further research in this regard could be important in determining potential mechanisms of current treatments and whether such treatments are effective for both emotional and cognitive symptoms of PTSD.

A novel and interesting field of research has evolved concerning the use of attentional training itself as a treatment for anxiety disorders. This research uses a modified dot-probe paradigm to "train" individuals to respond faster to probes presented away from negative stimuli (MacLeod et al., 2002). Attention modification has been effective in reducing symptoms in social anxiety (Li et al., 2008; Schmidt et al., 2009; Amir et al., 2008), generalized anxiety (Amir et al., 2009b), and sub-clinical obsessive-compulsive disorder (Najmi and Amir, 2010). Such training is meant to contradict the automatic attentional bias towards threat observed in anxiety disorders and to facilitate attentional disengagement from threatening stimuli (Amir et al., 2008, 2009b). A recent fMRI study suggests that attention modification training may modulate lateral PFC and striatal activations during emotional working memory (Browning et al., 2010). A recent EEG study reported that attentional training in anxious individuals resulted in increased N2 amplitudes, thought to play a role in attentional control processes, and decreased P2 amplitudes, thought to be associated with emotional processing (Eldar and Bar-Haim, 2010). Thus far, there have been no published studies investigating effects of attention modification on PTSD symptoms or neural activation patterns. However, given the deficits in inhibitory and attentional functions observed in PTSD, this could be a promising area of research.

6. Discussion

Although PTSD does not have a substantial effect on general cognition, neuropsychological research provides evidence for subtle deficits concerning inhibition of automatic responses and the regulation of attention—in both emotional and non-emotional contexts (Falconer et al., 2008; Pineles et al., 2007, 2009; McNally et al., 1990; Kimble et al., 2010; Leskin and White, 2007). In particular, PTSD has been associated with difficulty disengaging attention from one stimulus to focus on more task-relevant stimuli (Pineles et al., 2007, 2009). Such impairment may be most evident when the "distractor" stimuli are of high value—either negative or even potentially, positive. One basic ability we have as humans is to assess the value of environmental stimuli and quickly orient attention towards stimuli as needed (e.g., through "bottom-up"

influences on attention). However, it is also important to be able to determine which stimuli are irrelevant or distracting to our current goals and disengage from those stimuli in order to orient towards those that are more goal-relevant (e.g., "top-down" regulation of attention)(Bishop, 2008). There is evidence that PTSD may be associated with enhanced activation in prefrontal networks during tasks involving non-flexible, sustained attention to a stimulus (e.g., as with the continuous performance task), but with attenuated activation of prefrontal networks on tasks requiring inhibition or flexibility in attention (Bryant et al., 2005; Falconer et al., 2008; Moores et al., 2008). This combination of neural response patterns in PTSD may relate to observed difficulties disengaging and reorienting attention to perform optimally on cognitive tasks, and may underlie at least part of the symptom profile in PTSD.

Additional prospective, longitudinal research is needed to determine whether or not inhibitory dysfunction is a pre-trauma risk and resiliency factor. However, given the pervasiveness of such dysfunction across both trauma-related (e.g., dot-probe or modified Stroop tasks) and neutral (e.g., Go-NoGo tasks) tasks, it is likely that subtle deficits in executive function pre-date the trauma and influence the development of PTSD. In particular, there may be a subtle pre-trauma deficit concerning disengagement of attention from stimuli or behaviors that are no longer relevant. This subtle deficit could be amplified when a task or situation involves highly-valued stimuli that increase the pull on attentional resources. When a trauma occurs, trauma-associated stimuli become very highly "valued"—thereby demanding greater attention and making it more difficult to disengage. In such situations, any disengagement or "switching" dysfunctions could be amplified to their extreme. Subtle deficits in inhibition and disengagement may not significantly influence daily functioning until a highly emotional or traumatic experience serves to perturbate the system. It is easy to imagine how such difficulties, when combined with a significant traumatic experience, could spiral in a way that influences the development of symptoms we label as PTSD. A chart representing how these difficulties could relate to the progression of PTSD symptoms is displayed in Fig. 1.

As documented in the literature, a high percentage of people experience symptoms of posttraumatic stress within the few weeks following trauma (Rothbaum et al., 1992; Shalev et al., 1998). Fortunately, these symptoms decrease over time for most, while for those with PTSD, symptoms remain and may even worsen with time (Orcutt et al., 2004). We therefore suggest that trauma exposure may be associated with increased attention towards trauma- or threat- related stimuli for most, if not all, individuals (Fig. 1b). However, only a subset of these individuals would have underlying deficits related to inhibition of responses (Fig. 1c) and disengagement of attention (Fig. 1e). Such deficits could contribute to development of pervasive re-experiencing and hyperarousal symptoms. Furthermore, when individuals have difficulty

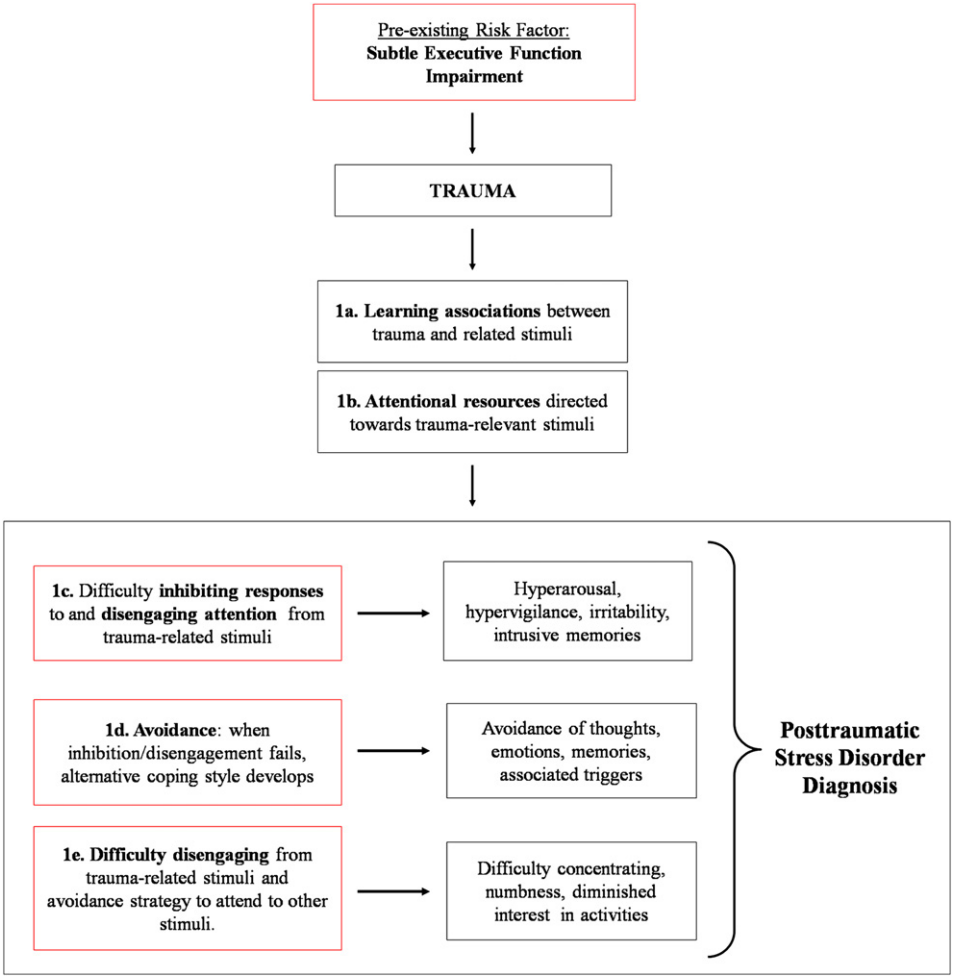

Fig. 1. Potential role of executive dysfunction in the development of PTSD. Most individuals experiencing a trauma may learn associations and direct attentional resources towards trauma-relevant stimuli (1a and 1b). However, subtle impairments in executive dysfunctions may relate to difficulties inhibiting responses and disengaging attention from trauma-related stimuli (1c and 1e) and lead to a reliance on avoidant coping strategies (1d), which contribute to the development of PTSD symptoms.

inhibiting responses to triggering stimuli, they may rely on other coping mechanisms—namely avoidance of arousing stimuli. In other words: if you can't inhibit it—avoid it (Fig. 1d).

Adoption of an avoidant coping strategy may be adaptive in the short term, as it decreases reliance on dysfunctional inhibitory and attentional networks. This coping strategy becomes a problem when avoiding emotional triggers also requires sacrifice of rewarding and positive aspects of a person's life. For example, a PTSD patient may avoid hospitals despite needing medical care in order to avoid smells or sights for which they have difficulty inhibiting emotional responses. Another PTSD patient may avoid previously-pleasurable activities (e.g., sports activities, family functions) due to crowds or other triggering stimuli. Persistent use of avoidance strategies keeps individuals from situations in which they could potentially learn to inhibit re-experiencing and hyper-arousal symptoms—thus helping maintain the disorder (Foa and Kozak, 1986). Difficulty disengaging from trauma-related stimuli and from adopted avoidant coping strategies, may also prevent individuals from attending to other aspects of their life—such as their family, friends, pleasurable activities, and positive emotions and cognitions in general. This in turn could contribute to the emotional numbness and depressive symptoms often experienced by PTSD patients.

One basic assumption of the most effective treatments for PTSD (e.g., Cognitive Processing Therapy and Prolonged Exposure) is that patients must decrease avoidant behaviors, habituate to triggering stimuli, learn that they can cope with strong emotions, and alter their cognitions and perceptions of the trauma, themselves, and the world in general (Foa and Kozak 1986; Foa et al., 2007; Resick and Schnicke, 1996). Such treatments disrupt avoidant coping styles and lead patients through experiences in which they can practice both habituation of emotion as well as inhibition and regulation of their automatic thoughts, feelings, and behaviors. Could treatments that more directly target inhibition, disengagement, and control of attention serve to benefit PTSD patients and decrease clinical symptoms? As discussed, there is evidence that training attention away from threat may help to reduce symptoms in other anxiety disorders. Given that between 20 and 50% of patients do not respond to current, first-line treatments for PTSD (Cukor et al., 2010; Schottenbauer et al., 2008), it is imperative that we direct *our* attentional resources towards the development and examination of such novel treatment strategies.

7. Conclusion

There is evidence for subtle deficits in attentional and inhibitory functions in PTSD that may predate trauma exposure, serve as risk factors for the development of PTSD, and relate to the severity of symptoms. We propose that such dysfunction could contribute to hypervigilance and arousal symptoms and the reliance on avoidant coping strategies, which are considered hallmark symptoms of PTSD. Further neuropsychological and neuroimaging research is needed to determine the exact nature of these deficits and the specific role they play in the etiology of the disorder. The use of attentional and inhibitory tasks within prospective, longitudinal studies could help in determining whether or not observed deficits are pre-trauma risk and resiliency factors. Additionally, the effect of current PTSD treatments on executive functions, as well as the effect of training in attention and inhibitory functions on PTSD symptoms, is of utmost importance. Neuropsychological, neuroimaging, and clinical research conducted thus far has led us to have specific, objective targets in sight on which treatments could potentially be aimed. It is hoped that by incorporating knowledge from cognitive and neuroscientific research, we can develop novel treatments that will allow us to more successfully treat those suffering from PTSD.

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TRAUMA NARRATIVE

- **GOALS:**
  - Unpair thoughts, reminders, or discussions of the traumatic event from overwhelming negative emotions (terror, horror, extreme helplessness, rage, shame, etc)
  - Desensitization procedure (repeated sharing and elaboration) decreases physical and psychological hyper-arousal upon exposure to reminders = decreases PTSD symptoms
  - Integration of thoughts and feelings into the creation of the trauma narrative allows child to put traumatic event into consistent and meaningful experience
  - Child will be able to integrate the traumatic experience into the totality of the rest of his/her life
  - Metacognitive abilities = ability to think about and evaluate one's own thoughts and experiences --- co-occurs to some degree and while creating the trauma narrative --- allows child to successfully integrate the trauma and its meaning into a larger optimal self-concept

- **PROCESS:**
  - Over course of several sessions, child is encouraged to tell more and more details of what happened before, during, and after traumatic event as well as thoughts and feelings during these times
  - Gradually expose child to increasingly upsetting aspects of the traumatic event
  - Many children will be able to tolerate "put yourself back there in your mind" or remember all of the details "just like it was happening now" until they have spent one or more sessions describing the events, thoughts, and feelings from their present perspective
  - Prior to initiating – therapist should introduce the child and parent(s) to the theoretical basis of this intervention and reassure any concerns/fears (PTSD-based avoidance or discomfort about discussing upsetting events)
  - Often child is better able to verbally describe things before getting them down in some written form (Every child is different)
  - Generally best to have child first describe his/her perception of **the facts about the traumatic event,** and after these have been written, to return to the beginning and ask about thoughts and feelings
  - Interrupting the child in the flow of his/her narrative may make it harder for them to focus on the experience and may also encourage avoidance of describing further details of what happened
  - Capturing the entire narrative make take more than one session, depending on how difficult it is for the child to recall, how much detail is provided, and how long a time period is covered

- Having the child read what he/she has written thus far is helpful in both desensitizing the child to verbalizing the details of the trauma, and in re-focusing the child for the next segment
- Over **several repetitions**, the child will typically experience progressively less extreme emotional reactions and physiological reactivity
- In some cases, the child may not know all of the exact details and may be imagining horrifying scenes of other loved ones suffering, in these cases it is important for the child to verbalize and write these imagined traumatic reminders
- Once the child has completed his/her description of what happened, the therapist should ask the child to read it from beginning, and ask the child to add thoughts and feelings he/she was having at the time of the events described (ex: what was weather like outside and on the inside) – record the thoughts and feelings but **do not challenge at this time**
- Not unusual for child to recall more details when adding thoughts and feelings – add them to the narrative
- At some point during creation of TN – therapist should ask child to describe the **worst moment, worst memory, and/or worst part of the traumatic event** – encourage to describe in as much detail as possible and draw a picture of the memory – encourage the child to write his/her feelings and to describe physical sensations that accompany
- If child becomes overwhelmed, remind him/her that these are only feelings and/or memories, they are related to something that happened in the past and not something that is occurring in the present
- Can use the Subjective Units of Distress (SUDS) Scale to help child quantify their degree of distress within each (or some) sessions – fear thermometers, children's faces depicting different degrees of distress
- At the end of each TN session, PRAISE the child and give a small reward (food, stickers, playing a game, etc)
- Once child has written the full narrative of his/her memories, thoughts, and feelings, cognitive processing techniques are employed to explore and correct cognitive distortions and errors
- Children may elect to alter or modify parts of the TN during Cognitive Processing – they should be encouraged to do so, incorporating new cognitions and metacognitions into the trauma narrative
- Therapist should encourage child to include at the end of the TN ways in which the child is different now from when events happened and when therapy began; what they have learned; and advice they might give to other children who have experienced similar types of trauma

MULTIPLE TRAUMAS OR COMPLEX TRAUMA: When considering how to structure trauma narratives for children who have experienced more than one trauma, or for children whose entire lives have been characterized by trauma – allow the child to guide you in which traumatic experiences to include and in what order. Goal is to help the child contextualize different traumatic events.

May want to consider creating a "life narrative" rather than a trauma narrative. Some children may make a "timeline" of their life, others may prefer to create a picture album starting with their birth describing significant memories up until the present time. Helpful to have child also recognize happy events and fun memories. Also allows you to point out how strong they must be to have gotten through so many difficult and challenging times.

SUGGESTED BOOKS (Cohen and Mannarino)_:

- Please Tell (Jessie, 1991, for sexual abuse)
- All Kinds of Separation (Cunningham, 1992, parental separation d/t child abuse, parental substance abuse, or hospitalization)
- Creative Healing Books series – fill in the blanks books (Alexander, 1993)
    - It's My Life
    - All My Dreams (exposure to crime or violence)
    - It Happened in Autumn (exposure to homicide)
    - When I Remember (exposure to traumatic death)
    - It Happens To Boys Too (Satullo, Russell, and Bradway, sexual abuse)

EXAMPLE OF INTRODUCTORY SCRIPT (Cohen and Mannarino):

"It is very hard to talk about painful things, and often children and parents try to avoid doing this. In fact, they say things like "let sleeping dogs lie," and wonder if it is a good thing to bring back memories of sad things. We tell kids and parents that if they had been able to put those memories behind them, children would not be having any problems, and they would not be coming here to therapy in the first place. It's like when you fall off a bicycle and skin your knee on the sidewalk, and all that dirt and germs get into the wound. You have two choices about what to do with that wound. You can leave it alone, not wash it off or put any medicine on it, and hope that it gets better all by itself. Sometimes that works fine. But other times, if you do that it will get infected. Infections don't usually get better by leaving them alone; they get worse and worse. Your other choice is to wash the wound out real carefully, getting all the dirt and germs out of there. Than stings, it hurts at first, but then the pain goes away, and it doesn't get infected, and can heal quickly. In the end, once an infection starts, it hurts a lot less to clean it out than to let it get worse and worse. Creating the trauma narrative, or telling the story of what happened, is like cleaning out the wound. It might be a little painful at first, but it hurts less and less as we go on, and then the wound can heal. Just like when you clean out a wound, if you rub it too hard or too fast, it will hurt a lot more than if you go more carefully. We try to go at just the right pace in telling your story so that it never hurts more than a little bit. You can let us know at any point if we are going too fast for you, and we will slow down."