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REVIEW ARTICLE

The effects of psychological interventions on neurocognitive functioning in posttraumatic stress disorder: a systematic review

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ABSTRACT

Background: Posttraumatic stress disorder (PTSD) is a serious mental disorder, which is associated with emotional and cognitive functioning problems. Psychological interventions, such as trauma-focused cognitive behavioural therapy (tf-CBT) and eye movement desensitization and reprocessing (EMDR) are effective in reducing PTSD symptoms. Despite evidence showing that PTSD is associated with neurocognitive deficits, there is no systematic overview available on neurocognitive outcomes following treatment for PTSD. The current systematic review examined whether psychological treatments for PTSD improve neurocognitive functioning outcomes related to memory, attention, information processing, and executive functioning.

Method: A literature search in PubMed, PsycINFO, PTSDpubs, and Cochrane Library was performed up to March 7, 2022, in collaboration with a medical information specialist. Eligible PTSD treatment studies examining neurocognitive outcomes (memory, attention, information processing and executive function) in patients with a DSM-IV or ICD diagnosis of PTSD were included.

Results: Of the 3023 titles and abstracts identified, 9 articles met inclusion criteria, of which 5 randomized controlled trials (RCTs) and 4 non-randomized studies. Treatments included were cognitive behavioural therapy (CBT), cognitive processing therapy (CPT), brief eclectic psychotherapy (BEP), eye movement desensitization and reprocessing (EMDR), virtual reality graded exposure therapy (VR-GET), and resilience-oriented treatment (ROT).

Conclusions: This systematic review showed that psychological treatments for PTSD do not affect most neurocognitive functions, with exception of the memory outcomes. Future research, high-quality studies are needed to provide evidence of the effect of psychological treatment in improving neurocognitive functioning in PTSD.

Los efectos de intervenciones psicológicas en el funcionamiento neurocognitivo en Trastorno de Estrés Postraumático: Una revisión sistemática

Antecedentes: El trastorno de estrés postraumático (TEPT) es un trastorno mental serio, que está asociado con problemas emocionales y en el funcionamiento cognitivo. Las intervenciones psicológicas, tales como la terapia cognitivo conductual enfocada en el trauma (tf-CBT) y la terapia de Desensibilización y Reprocesamiento mediante Movimientos Oculares (EMDR) son efectivas en la reducción de los síntomas de TEPT. A pesar de que la evidencia ha mostrado que el TEPT está asociado con déficits neurocognitivos, no hay revisiones sistemáticas sobre los resultados neurocognitivos tras el tratamiento de TEPT. Esta revisión sistemática examinó si los tratamientos psicológicos para el TEPT mejoraron los resultados de funcionamiento neurocognitivo relacionados con la memoria, atención, procesamiento de información, y funcionamiento ejecutivo.

Método: Se realizó una búsqueda de la literatura en PubMed, PsycINFO, PTSDpubs, y Cochrane Library hasta el 7 de marzo de 2022, en colaboración con un especialista en información médica. Fueron incluidos los estudios elegibles sobre tratamiento de TEPT que examinaban resultados neurocognitivos (memoria, atención, procesamiento de información y función ejecutiva) en pacientes con un diagnóstico de TEPT según DSM-IV o CIE.

Resultados: De los 3023 títulos y resúmenes identificados, 9 artículos cumplieron los criterios de inclusión, de los cuales 5 eran ensayos clínicos aleatorizados (RCT) y 4 estudios no aleatorizados. Los tratamientos incluyeron terapia cognitivo conductual (CBT), terapia de procesamiento cognitivo (CPT), psicoterapia ecléctica breve (BEP), desensibilización y reprocesamiento mediante movimientos oculares (EMDR), terapia de exposición mediante realidad virtual graduada (VR-GET), y tratamiento orientado a la resiliencia (ROT).

HIGHLIGHTS
- This systematic review investigated the effects of psychological treatments on neurocognitive functioning in adults with PTSD.
- This review showed that most studies were very heterogeneous in design, method, and analysis.
- This review supports the evidence for psychological treatments for PTSD on improving memory outcomes.

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Conclusiones: Esta revisión sistemática mostró que los tratamientos psicológicos para el TEPT no afectan la mayoría de las funciones neurocognitivas, a excepción de los resultados en la memoria. Nuevas investigaciones, con estudios de alta calidad son necesarias para proveer de evidencia sobre el efecto del tratamiento psicológico en la mejoría del funcionamiento neurocognitivo en el TEPT.

心理干预对创伤后应激障碍神经认知功能的影响：一项系统综述

背景：创伤后应激障碍（PTSD）是一种严重的精神障碍，与情绪和认知功能问题有关。心理干预，如以聚焦创伤认知行为疗法 (tf-CBT) 和眼运脱敏和再加工 (EMDR) 可有效减轻 PTSD 症状。尽管有证据表明 PTSD 与神经认知缺陷有关，没有关于 PTSD 治疗后神经认知结果的系统概述。目前的系统综述考查了 PTSD 心理治疗是否能改善与记忆、注意力、信息加工和执行功能相关的神经认知功能结果。
方法：截至 2022 年 3 月 7 日，与医学信息专家合作，在 PubMed、PsycINFO、PTSDpubs 和 Cochrane Library 中进行了文献检索。纳入了符合 DSM-IV 或 ICD 诊断为 PTSD 的患者的神经认知结果（记忆、注意力、信息加工和执行功能）的合格 PTSD 治疗研究。
结果：在确定的 3023 篇标题和摘要中，9 篇文章符合纳入标准，其中 5 篇随机对照试验 (RCT) 和 4 篇非随机研究。治疗包括认知行为疗法（CBT）、认知加工疗法（CPT）、简短折衷心理疗法（BEP）、眼球运动脱敏和再加工（EMDR）、虚拟现实分级暴露疗法（VR-GET）和心理韧性导向治疗（腐烂）。
结论：本系统综述表明，PTSD 心理治疗不会影响大多数神经认知功能，但记忆结果除外。未来的研究，需要高质量的研究来提供心理治疗在改善 PTSD 神经认知功能方面作用的证据。

1. Introduction

Life can be seriously distorted by traumatic events, such as natural disasters, traffic accidents, sexual harassment, or domestic violence. After exposure to a traumatic event, people may develop posttraumatic stress disorder (PTSD). PTSD is a psychiatric disorder characterized by four symptom clusters: intrusion symptoms, persistent avoidance of stimuli associated with the trauma, negative alterations in cognitions and mood, and alterations in arousal and reactivity (American Psychiatric Association, 2013). PTSD impairs daily functioning, quality of life, and may be associated with psychological and somatic problems (Pacella, Hruska, & Delahanty, 2013).

A large body of literature has shown robust evidence that PTSD is associated with neurocognitive deficits. The most commonly examined domains include attention, memory, executive functioning and information processing speed (Clausen et al., 2017; Schuitevoerder et al., 2013). Findings from these studies provide evidence for decreased performance in neurocognitive functions among PTSD patients (Hayes, Hayes, & Mikedis, 2012).

PTSD can effectively be treated with a number of psychological interventions. According to several meta-analyses, there is strong evidence for the effectiveness of trauma-focused psychotherapies for PTSD, including exposure therapy, cognitive behavioural therapy, and eye movement desensitization and reprocessing (EMDR; National Institute for Health and Care Excellence (NICE) guideline, 2018). Trauma-focused treatments essentially include all psychotherapies during which patients are exposed to the traumatic memory. They are assumed to lead to emotional processing of traumatic memories and integration of new corrective information, which is referred to as extinction learning (Bryant, 2019). Another explanation for the working mechanism of trauma-focused treatments is that during therapy, traumatic memories and their associated emotions are activated, while new emotional experiences during therapy are incorporated into the original memory trace via a process named reconsolidation (Lane, Ryan, Nadel, & Greenberg, 2015). Further, trauma-focused therapies are assumed to reduce PTSD symptoms by reducing avoidance, and changing trauma-related negative assumptions. Cognitive therapies, such as cognitive processing therapy (CPT), facilitate reprocessing thoughts and beliefs generated from a traumatic event, and modify dysfunctional beliefs (Cusack et al., 2016). In EMDR, the traumatic memories are retrieved while performing a dual task taxing the working memory (making saccadic eye movements). The working memory theory postulates that eye movements during traumatic memory retrieval and visual imagery interfere with reconsolidation of the traumatic memories by making them less vivid or emotional (van den Hout & Engelhard, 2012).

1.1. Neurocognitive deficits in PTSD

1.1.1. Memory and learning

Memory is the ability to learn new information and retain and recall the information after a delay (Jacob, Dodge, & Vasterling, 2019). Memory alterations are core features of PTSD, as shown in the symptoms of re-experiencing or poorly controlled recollections of the traumatic memory (Verfaellie & Vasterling, 2009). Consequently, several PTSD theories including Ehlers and Clark's cognitive theory (Ehlers & Clark, 2000) and Brewin's dual representation theory

(Brewin & Holmes, 2003), consider memory as a central component to explain neurocognitive functioning dysfunctions in PTSD. Both of these models are consistent with PTSD's clinical symptoms that involve involuntary recall of traumatic memories and enhanced trauma-related memory biases (Jacob et al., 2019). Ehlers and Clark's cognitive theory (Ehlers & Clark, 2000) proposes that negative appraisals of trauma and its consequences contribute to selective recall of information that is consistent with such negative appraisals. Conversely, disorganized and fragmented trauma memories that are poorly elaborated and inadequately integrated into other autobiographical memories contribute to negative appraisals of one's self (Ehlers & Clark, 2000).

In addition, the dual representation theory provides a detailed memory-based explanation for the etiology of PTSD. It assumes that two types of memories are encoded during the traumatic event: a sensory-bound representation (S-rep), reflecting sensory details and the affective/emotional state experienced during the traumatic event; and a contextual representation (C-rep), which is an abstract structural description of the event, along with the spatial and personal context of the person experiencing the event. In traumatized individuals, typical PTSD symptoms, such as re-experiencing (intrusions and flashbacks) are proposed to arise from an imbalance between the sensory-bound and contextual representations (Brewin & Burgess, 2014).

Several studies have shown that a higher level of PTSD symptoms is associated with worse neurocognitive performance. In particular, re-experiencing symptoms are related to poor performance on tasks measuring memory function and attention (Vasterling, Brailey, Constan, & Sutker, 1998). A study from Parslow and Jorm (2007) reported that patients who had more re-experiencing symptoms of PTSD had worse neurocognitive performance. A meta-analysis reported evidence for memory deficits in PTSD, with the most robust findings for reduced verbal learning and verbal memory (Scott et al., 2015). In addition, lower memory scores were found among older adults with PTSD, with evidence for an association between trauma exposure and poorer performance on verbal learning tests. The relationship between PTSD and memory deficit is assumed to be related to hippocampal volume reduction and involves the frontal area (Bremner et al., 1995; Olff, Polak, Witteveen, & Denys, 2014), which has implications for executive dysfunctions that interfere with strategic learning (Aupperle, Melrose, Stein, & Paulus, 2012).

1.2. Attention/working memory and executive functioning

Attention refers to the capacity of an individual to focus on a particular stimulus, while working memory is the active manipulation and maintenance of information in the short term (Aupperle et al., 2012). Attention and working memory are involved in maintaining successful executive function (McCabe et al., 2010). Adults with PTSD have shown poorer attention and working memory performance compared to non-trauma exposed control (Schuitevoerder et al., 2013; Scott et al. (2015).

Executive functioning covers mental flexibility, inhibitory and cognitive control. Mental flexibility is the ability to switch between two different tasks or strategies, which is an essential aspect of executive control (Aupperle et al., 2012). A recent meta-analysis reported that PTSD patients had worse executive function compared to trauma-unexposed controls ($g = 0.46$, $p < .001$) or trauma-exposed controls without PTSD (Woon, Farrer, Braman, Mabey, & Hedges, 2017). These results are consistent with the findings of previous meta-analyses (Schuitevoerder et al., 2013; Scott et al., 2015).

1.3. Information processing speed

Information processing speed is the speed with which a cognitive operation is performed (Higgins, Martin, Baker, Vasterling, & Risbrough, 2018), and can be reduced in PTSD (Scott et al., 2015; Twamley et al., 2009). Information processing speed is significantly associated with PTSD symptoms (Scott et al., 2015). A reduction in cognitive function in general can reduce attention to a related stimulus, and complete the task at hand (Morey et al., 2009), thus affecting the processing speed (Scott et al., 2015). However, only a few studies on this topic are available and the mechanism for a potential processing speed deficit in PTSD remains unclear (Jak, Crocker, Aupperle, Clausen, & Bomyea, 2018).

1.4. Aims of this review

Building on the evidence for neurocognitive deficits associated with PTSD, studies have been carried out that examined whether psychological treatments for PTSD directly improve neurocognitive functioning and/or interact with treatment effects (Higgins et al., 2018; Jacob et al., 2019). Currently, there are no systematic reviews available of neurocognitive outcomes following treatment for PTSD. The aim of this systematic review was to systematically summarize the evidence regarding the effects of psychological treatments on neurocognitive functioning outcomes in PTSD. Based on the existing literature, we hypothesize that psychological treatments will result in improvements in neurocognitive functioning outcomes, such as memory, attention, information processing, and executive functioning in PTSD patients.

2. Method

2.1. Search strategy

We followed the recommendations in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)-statement (www.prisma-statement.org). We searched for studies on the effect of psychological intervention on neurocognitive functioning in PTSD patients. To identify all relevant publications, we conducted systematic searches in the following bibliographic databases: PubMed, APA PsycInfo (Ebsco), PTSDpubs (ProQuest) and Wiley/Cochrane Library from inception to March 7, 2022, in collaboration with a medical information specialist. A protocol for the review was published by PROSPERO (registration number CRD42020148444).

The following terms were used (including synonyms and closely related words) as index terms or free-text words: 'Posttraumatic', 'PTSD', 'PTSS', 'Desensitization', 'Cognitive Behavioural Therapy', 'Memory', 'Executive Function', 'Attention'. The references of the identified articles were searched for relevant publications. Duplicate articles were excluded. See Appendix A Online supplementary material) for the full search strategies.

2.2. Eligibility criteria

Expecting the number of RCTs examining the effect of psychological interventions in neurocognitive functioning in PTSD to be limited, we also screened for all published papers with a non-randomized design from 1982 to 2022. Studies were eligible for analysis if: (1) participants were at least 18 years of age; (2) participants were diagnosed with PTSD according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2013) or the International Classification of Diseases (ICD; Reed et al., 2019); (3) participants received psychological treatment for PTSD (i.e. trauma-focused CBT, prolonged exposure, resilience oriented treatment, EMDR); (4) control groups were either waiting list control, treatment as usual, or an active treatment group; (5) at least one of the following neurocognitive functioning outcomes before and after treatment were reported: attention, memory, working memory, executive function or information processing; and (6) the study was reported in English.

2.3. Data collection

After deduplication, all titles and abstracts were screened by two independent reviewers (ES, WD) (cf. Lefebvre et al., 2021). Differences were resolved by discussion, with consultation of the other team members. The same procedure was carried out for screening the full text.

2.4. Data extraction and quality assessment

Data (study methodology, participant characteristics, interventions, and outcomes) of included papers were extracted by two independent researchers (ES, WD). The same researchers assessed all included studies for risk of bias, using the Cochrane Risk of Bias tool (Higgins, Altman, Higgins, & Green, 2008, 2019). This included (1) sequence allocation for randomization; (2) allocation concealment; (3) incomplete outcome data; (4) selective reporting; (5) blinding of participants and personnel and (6) blinding of outcome assessment. The studies were classified as having a low, high or unclear risk of bias across each of these six domains. Any discrepancies between the researchers were discussed with a third researcher to reach a unanimous decision.

2.5. Analysis and data synthesis

We provided a synthesis of the studies by summarizing the characteristics and outcomes of the included in tables. Further, we generated forest plots in Comprehensive Meta-Analysis Version 3.0 (Borenstein, Hedges, Higgins, & Rothstein, 2004) to present the effect sizes of all RCT's per outcome type (attention, memory, information processing or executive function). We analyzed continuous scores for neurocognitive functioning outcomes at post-treatment to calculate effect sizes for attention/working memory, memory/learning, executive function, and information processing speed. When studies included both intention-to-treat (ITT) and completers analyses, we presented ITT results in our analyses. The current review used the Comprehensive Meta-Analysis Version 3.0 program for the statistical analysis. Hedges' g was calculated to determine the effect size for each individual study, since it is assumed to be more accurate than other effect size measures when the sample size of the study is small (Cuijpers, 2016). Hedges' g was computed as a between group effect size, comparing psychological treatment to control (waiting list or treatment as usual or active control). The effect size is considered small when $g = .20$, medium when $g = .50$, and large when $g = .80$. We pooled the data if we could include at least three studies using the same type of neurocognitive outcome that compared a psychological treatment to an inactive (waitlist control) group. For non-randomized studies, we reported the effect sizes as reported by the authors in original study.

3. Results

3.1. Study selection

We found a total of 4632 references: 1187 in PubMed, 1553 in APA Psyinfo, 1252 in PTSDpubs and 640 in

Cochrane Library. Appendix B (Online supplementary material) shows the flow chart of the search and selection process. After the exclusion of 2993 studies, we retrieved 30 studies for full-text consideration. Finally, nine studies (421 participants) met inclusion criteria. We excluded twenty-one studies for the following reasons: five studies did not meet the criterion for inclusion of PTSD diagnosed participants, five studies did not assess neurocognitive outcomes, two studies did not include a post-treatment assessment, seven studies did not evaluate psychological treatment, one study did not include criterion inclusion of adult participants, and one study was a secondary analysis of one of the already included studies.

3.2. Study characteristics

Study characteristics of the included nine studies are presented in Table 1. Appendix C (Online supplementary material) provides an overview of all measurements and outcomes. Included studies were conducted in the United States ($n = 6$), the Netherlands ($n = 1$), Germany ($n = 1$) or Iran ($n = 1$). Four studies were carried out in samples of war veterans in military affairs (Haaland, Sadek, Keller, & Castillo, 2016; Jak et al., 2019; Kent, Davis, Stark, & Stewart, 2011; McLay et al., 2014), whereas five studies were performed in outpatient clinics of hospitals or mental health units (Akbarian et al., 2015; Maxwell, 2016; Nijdam, Martens, Reitsma, Gersons, & Olff, 2018; Schindler et al., 2020; Walter, Palmieri, & Gunstad, 2010). Five studies were RCTs (Kent et al., 2011; Akbarian et al., 2015; Maxwell, 2016; Nijdam et al., 2018; Jak et al., 2015), whereas four were non-randomized intervention studies (Haaland et al., 2016; McLay et al., 2014; Schindler et al., 2020; Walter et al., 2010). Seven studies measured memory (Akbarian et al., 2015; Haaland et al., 2016; Jak et al., 2019; Kent et al., 2011; Maxwell, 2016; Nijdam et al., 2018; Schindler et al., 2020), six attention (Haaland et al., 2016; Jak et al., 2019; Kent et al., 2011; McLay et al., 2014; Nijdam et al., 2018; Schindler et al., 2020), seven executive function (Haaland et al., 2016; Jak et al., 2019; Kent et al., 2011; Maxwell, 2016; McLay et al., 2014; Nijdam et al., 2018; Walter et al., 2010), and two studies measured information processing speed (Jak et al., 2019; Nijdam et al., 2018) and seven studies measured several outcomes (Haaland et al., 2016; Jak et al., 2019; Kent et al., 2011; Maxwell, 2016; Nijdam et al., 2018; Schindler et al., 2020; Walter et al., 2010). Although we found three to five RCTs for memory, working memory and executive function, only one study for memory, one for working memory and one for executive function included an inactive control group, which was not reaching the threshold for performing a meta-analysis. Beforehand, we decided that we would only pool data with at least three studies using the same type of neuropsychological outcome, and this criterion was not met. Therefore, the data were not pooled.

3.3. Participant characteristics

The total number of participants was 285 (147 in the treatment groups and 138 in the control groups) for the RCTs and 180 for the non-randomized intervention studies. The sample sizes ranged between 18 and 100 participants ($M = 57$, SD = 35.16) for the RCTs and between 15 and 103 participants ($M = 45$, SD = 40.41) for the non-randomized intervention studies. Two studies included only females, two studies included a majority of male participants, while four studies included both male and female participants.

3.4. Diagnosis and characteristics of PTSD

In five studies PTSD was diagnosed with the Clinician-Administered PTSD Scale for DSM-IV (CAPS-IV; Weathers, Ruscio, & Keane, 1999, 2001) or a standard structured interview for PTSD, such as the Structured Clinical Interview for DSM-IV (SCID-IV; Glasofer, Brown, & Riegel, 2015). Four studies included war trauma-related PTSD, while five studies included mixed trauma types, such as physical or sexual assaults, accident-related injuries, cancer, or domestic violence.

3.5. Change in neurocognitive functioning

3.5.1. Memory

Five RCTs included a memory outcome. We computed Hedges' g to determine the effect size across the subdomains of memory (Table 2). The details of measurements and tasks were attached as Appendix C. (Online supplementary material) First, the study by Kent et al. (2011) used a resilience-oriented treatment to improve memory function among 20 PTSD patients compared to 19 waiting list control PTSD patients exposed to war trauma. Resilience-oriented treatment aimed to build the patient's resilience by helping to find protective, supporting and strengthening factors in order to survive (Laban, 2015). For episodic memory, medium-sized effects of resilience-oriented treatment versus waiting list were found (RBANS; Hedges' $g = 0.39$, 95% CI: 0.02 to 0.75).

Akbarian et al. (2015) compared the effect of CBT combined with pharmacotherapy (Selective Serotonin Reuptake Inhibitors, neuroleptics, or benzodiazepines; $n = 20$) to pharmacotherapy only ($n = 20$) among PTSD patients with accident-related injuries, cancer, and domestic violence. The study reported a significant improvement in autobiographical memory performance for the PTSD patients in the combined CBT-pharmacotherapy group compared to pharmacotherapy only, in terms of semantic-adult memory

Table 1. Characteristic of included studies.

First author, Country, setting	Intervention (Experiment versus (vs.) Control)	N	Gender M/F	PTSD diagnosis	Number of sessions/duration per session	Time point	Type of trauma	Measures	Outcome
Randomized controlled trials									
Akbarian et al. (2015), Iran, Hospital and Psychiatric centres	Psychopharmacological-CBT (n = 20) vs. Pharmachological (n = 20)	40	12/28	DSM-V criteria	10 sessions, 60–90 min, weekly	Pre, post	Mixed	AMI	Memory
Jak et al. (2019), US, Healtcare System	SMART-CPT intervention (n = 51) vs. CPT intervention (n = 49)	100	89/11	DSM-IV criteria, CAPS-IV	12 sessions, 60–75 min, weekly	Pre, post, 3 months follow up	War trauma. Mild-moderate TBI	CVLT, WAIS-IV, WCST-64, D-KEFS	Memory, Attention, Executive Function, Psychomotor processing speed
Kent et al. (2011), US, veterans	ROT (n = 20) vs. Waiting List (n = 19)	39	20/19	CAPS	12 sessions, 90 min, weekly	Pre, post (5 weeks)	War trauma	NAB, D-KEFS	Executive Function, Memory
Maxwell (2016), US, mental health units and hospital	MeST (n = 9) vs. CPT (n = 9)	18	3/13	CAPS	MeST = 6 sessions, 90 min CPT = 12 sessions, 90 min, weekly	Pre, post, 3-months follow up	Mixed	AMT	Memory
Nijdam et al. (2018), the Netherlands, Psychological trauma centre	BEP (n = 41) vs. EMDR (n = 47)	88	39/49	SI-PTSD, DSM-IV criteria	BEP (14.7 average), 90 min EMDR (6.4 average), 45 min, weekly	pre, post	Mixed	CVLT, RBMT, TMT, Stroop Colour Word Test	Memory, Attention, Executive Function, Information processing speed
Non-randomized studies									
Haaland et al. (2016), US, veterans	TFP (n = 42)	42	0/42	CAPS	5 sessions of PE, 5 sessions of CPT and 4 behaviour skills training, weekly	Pre, post	War trauma	WTAR, CVLT-II, D-KEFS, Digit Span (WAIS-IV)	Memory, Executive function, Working memory
McLay et al. (2014), US, veterans	VR-GER (n = 15)	15	14/1	CAPS	10 sessions, 90 min, weekly	Pre, post	War trauma	ARES	Executive function, Attention
Schindler et al. (2020), Germany, the Technische Universität Dresden	CBT (n = 58) vs. non-traumatized control (n = 45)	103	11/92	DSM-IV criteria, M-CIDI	25 sessions	Pre, post	Mixed	Digit Span (WAIS-IV), AMT	Memory, Working memory
Walter et al. (2010), US, trauma centre	TFP (n = 15) vs. TFP non-completer (n = 5)	20	0/20	SCID-IV	10 sessions of PE, 12 sessions of CPT or 12 sessions of non-manualized TFT	Pre, 3-months follow up	Mixed	D-KEFS, BQSS, Stroop colour-word Test	Executive Function

Note: AMI = Autobiographical Memory Interview, AMT = Autobiographical Memory Test, ARES = ANAM Readiness Evaluation System, ANAM = Automated Neuropsychological Assessment Metric, BEP = Brief Eclectic Therapy, BIS = the Barratt Impulsiveness Scale (BIS), BQSS = The Boston Qualifying Scoring System, CAPS = The Clinician-Administered PTSD Scale, CBT = Cognitive Behavioural Therapy, CPT = Cognitive Process Therapy, CVLT = California Verbal Learning Test-II, C-Stroop = Congruent Stroop, D-KEFS = Delis-Kaplan Executive Function System, DSM-V = Diagnostic and Statistical Manual of Mental Disorders, 5th Edition, E-Stroop = Emotional Stroop, EMDR = Eye Movement Desensitization and Processing, I-Stroop = Incongruent Stroop, M-CIDI = Munich Composite International Diagnostic Interview, MeST = Memory Specificity Training, NAB = Neuropsychological Assessment Battery, N-Stroop = Neutral Stroop, PE = Prolonged Exposure, PRTT = Procedural Reaction Time Test, RBMT = the Rivermead Behavioural Memory Test, ROT = Resilience Oriented Treatment, SCID = the Structured Clinical Interview for DSM-5 disorders, SMART-CPT = Cognitive Symptom Management and Rehabilitation Therapy (CogSMART)-Cognitive Processing Therapy, SRTT = Simple Reaction Time Test, TBI = Traumatic Brain Injury, TFP = Trauma Focus Psychotherapy, TMT = the Trail Making Test, VR-GER = Virtual Reality Graded Exposure Therapy, WAIS-IV = Wechsler Adult Intelligence Scale-IV, WCST-64 = Wisconsin Card Sorting Test-64 card version, WL = Waiting list, WMC = Working Memory Capacity, WMC = Working Memory Capacity, WTAR = Wechsler Test of Adult Reading.

Table 2. Effect size calculation of the control trial studies for neurocognitive functioning outcomes.

Study	Outcomes	Psychological intervention			Control			Effect size Hedges' *g* (95% CI)	Standard difference in means and 95% CI
		Mean	SD	N	Mean	SD	N		
Effect on memory									
Akbarian et al. (2015)	AMI-Childhood episodic	3.85	0.63	20	4.18	1.14	20	0.36 (−0.27, 1.00)	
	AMI-Adulthood episodic	4.71	0.85	20	3.89	1.15	20	0.81 (0.17, 1.46)	
	AMI-Recent episodic	4.57	0.99	20	3.32	1.34	20	1.06 (0.40, 1.72)	
	AMI-Childhood semantic	16.21	1.49	20	15.79	2.58	20	0.20 (−0.42, 0.82)	
	AMI-Adult semantic	17.04	1.22	20	15.05	1.37	20	1.53 (0.83, 2.24)	
	AMI-Recent semantic	17.86	2.21	20	16.75	2.87	20	0.43 (−0.19, 1.06)	
	Combined subscale							0.71 (0.32, 1.11)	
Jak et al. (2019)	CVLT trial 1-5	62.10	11.30	51	49.00	10.56	49	1.20 (0.77, 1.62)	
	CVLT SDFR	0.78	0.94	51	−0.27	1.31	49	0.92 (0.51, 1.34)	
	CVLT LDFR	0.70	0.97	51	0.00	0.83	49	0.77 (0.37, 1.18)	
	Combined subscale							0.96 (0.72, 1.20)	
Kent et al. (2011)	RBANS-Story memory	17.35	4.16	20	15.58	3.44	19	0.45 (−0.19, 1.09)	
	RBANS-List recall	4.95	2.24	20	4.37	1.98	19	0.27 (−0.36, 0.91)	
	RBANS-Story recall	9.35	1.84	20	8.42	2.34	19	0.44 (−0.19, 1.08)	
	Combined subscale							0.39 (0.02, 0.75)	
Maxwell (2016)	AMT	8.63	2.00	8	9.13	0.83	8	−0.33 (−1.31, 0.66)	
Nijdam et al. (2018)	CVLT Sum trial 1-5	55.8	10.8	47	50.4	11.7	41	0.48 (0.06, 0.91)	
	CVLT ST Free recall	12.3	3.2	47	11.7	3.0	41	0.19 (−0.23, 0.61)	
	CVLT LT Free recall	13.0	3.0	47	12.0	3.7	41	0.30 (−0.12, 0.72)	
	CVLT LT recognition	42.7	1.9	47	41.7	2.8	41	0.42 (0.00, 0.85)	
	RBMT-Immediate recall	21.4	7.1	47	21.4	7.1	41	0.00 (−0.42, 0.42)	
	RBMT-Delayed recall	17.6	7.6	47	17.3	7.4	41	0.04 (−0.38, 0.46)	
	Combined subscale							0.24 (0.07, 0.41)	
Effect on attention/working memory									
Jak et al. (2019)	WAIS-IV Digit span	10.55	2.98	51	9.00	1.96	49	0.61 (0.21, 1.01)	
Kent et al. (2011)	RBANS-List learning	27.95	5.40	20	24.74	5.39	19	0.59 (−0.05, 1.24)	
Nijdam et al. (2018)	Stroop Card 3	94.8	44.6	47	102.6	50.0	41	0.17 (−0.25, 0.59)	

(Continued)

Table 2. Continued.

Study	Outcomes	Psychological intervention			Control			Effect size Hedges' g (95% CI)	Standard difference in means and 95% CI
		Mean	SD	N	Mean	SD	N		
Effect on executive function									
Jak et al. (2019)	WSCT-64	54.85	8.11	51	50.47	9,99	49	0.48 (0.09, 0.88)	
	D-KEFS Colour	9.70	3.50	51	10,67	3,29	49	0.29 (−0.11, 0.68)	
	D-KEFS Trail making switching	11.20	1.54	51	11.29	1.68	49	0.06 (−0.34, 0.45)	
Kent et al. (2011)	NAB-Word generation	11.75	4.82	20	8.63	4.45	19	0.67 (0.03, 1.32)	
	D-KEFS-Category fluency	10.55	3.47	20	9.32	2.77	19	0.39 (−0.24, 1,02)	
	D-KEFS-Category switching	11.70	3.60	20	9.84	4.02	19	0.49 (−0.15, 1,13)	
	D-KEFS-Colour word switching	9.80	2.98	20	8.74	3.11	19	0.35 (−0.28, 0.98)	
	Combining							0.47 (0.15, 0.79)	
Maxwell (2016)	Stroop task	967.64	243.10	8	817.43	177.50	8	0.70 (−0.31, 1.71)	
Nijdam et al. (2018)	TMT part B	70.4	32.1	47	75.6	28.1	41	0.17 (−0.25, 0.59)	
	Stroop interference	35.2	34.1	47	38.3	37.9	41	0.09 (−0.33, 0.51)	
Information processing speed									
Jak et al. (2019)	WAIS-IV Processing Speed Index	100.15	13.53	51	101.60	24.96	49	0.07 (−0.32, 0.47)	
Nijdam et al. (2018)	Stroop Card 2	60.0	13.1	47	64.6	21.8	41	0.26 (−0.16, 0.68)	
	TMT part A	27.9	10.4	47	32.0	15.7	41	0.31 (−0.11, 0.73)	

Notes: AMI = Autobiographical Measure Interview, AMT = Autobiographical Measure Test, CI = Confidence Interval, D-KEFS = Delis-Kaplan Executive Function System Trail Making Test, CVLT = California Verbal Learning Test-II, LDFR = Long delay free recall, LT = Long Term, NAB = Neuropsychological Assessment Battery, N = Number of participants, N-Stroop = Neutral Stroop, PRTT = Procedural Reaction Time Test, RBANS = Repeatable Battery for the Assessment of Neuropsychological Status, RBMT = the Rivermead Behavioural Memory Test, SD = Standard Deviation, SDFR = Short delay free recall, ST = Short Term, TMT = the Trail Making Test, WAIS-IV = Wechsler Adult Intelligence Scale-IV, WCST-64 = Wisconsin Card Sorting Test-64 card version.

and episodic-recent memory at 10 weeks after treatment. Our analysis of combining autobiographical memory outcomes showed a large effect size of CBT-pharmacotherapy versus pharmacotherapy (AMI; $g = 0.71$, 95% CI: 0.32 to 1.11).

A small study of Maxwell (2016) investigated the effectiveness of a 6-sessions group MeST intervention ($n = 9$) as compared to 12-sessions group CPT ($n = 9$) in PTSD patients who had experienced a motor vehicle accident, assault, and/or other traumatic event. The participant was given one minute to provide a personal memory in response to each cue word. The effect size of MeST versus CPT for recall specific memories was not significant (AMT, $g = -0.33$, 95% CI: -1.31 to 0.66).

Nijdam et al. (2018) randomized participants into brief eclectic psychotherapy (BEP) ($n = 41$) or EMDR ($n = 47$). BEP combines a cognitive–behavioural and a psychodynamic approach. It consists of psychoeducation, imaginal exposure, writing assignments and cognitive restructuring. Verbal memory improved significantly after both BEP or EMDR, with EMDR being slightly more effective than BEP at post-treatment (CVLT and RBMT; $g = 0.24$, 95% CI: 0.07 to 0.41).

Jak et al. (2019) examined the effects of SMART-CPT ($n = 51$) compared with regular CPT ($n = 49$) in the treatment of veterans with PTSD for 12 weeks. SMART-CPT integrated compensatory cognitive training from the rehabilitation therapy (CogSMART) into CPT for PTSD. The control group received CPT only. The SMART-CPT group showed significantly higher verbal learning scores than the CPT only group at three months follow up, with a large effect size (CVLT; $g = 0.96$, 95% CI: 0.72 to 1.2).

A non-randomized study by Haaland et al. (2016) showed an improvement in verbal learning/memory (Cohen's $d = 0.55$) after treatment when evaluating trauma-focused individual CBT among 15 female PTSD patients exposed to war-related trauma. In addition, a recent non-randomized study by Schindler et al. (2020) showed no significant effect of CBT in PTSD patients ($n = 25$) as compared to non-traumatized healthy control ($n = 34$) in improving autobiographical memory ($\alpha = .05$; AMT, $p = .76$).

3.5.2. Attention/working memory and executive function

Other important domains of neurocognitive functions are attention and executive functioning. Kent et al. (2011) compared resilience-oriented treatment to waiting-list control on working memory and executive functioning in PTSD patients with war-related trauma. A non-significant effect size for working memory was found (RBANS-list learning; $g = 0.59$, 95% CI: -0.05 to 1.24), and a medium effect for executive function (NAB and D-KEFS; $g = 0.47$ 95% CI: 0.15 to 0.79). Maxwell (2016) compared MeST and CPT in

terms of controlled thinking in PTSD patients. The results showed no significant difference both groups in reducing response time with regard to Stroop interference (Stroop task; $g = 0.70$, 95% CI: -0.31 to 1.71).

A study by Nijdam et al. (2018) compared neurocognitive functioning between BEP and EMDR, and found that EMDR showed non-significant improvements in shift attention (TMT B; $g = 0.17$ 95% CI: -0.25 to 0.59), and planning and cognitive flexibility (Stroop interference; $g = 0.09$ 95% CI: -0.33 to 0.51).

Furthermore, Jak et al. (2019) evaluated the effects of SMART-CPT treatment versus CPT among PTSD patients exposed to war trauma. The effect sizes of SMART-CPT versus CPT were significant for attention/working memory (WAIS-IV digit span; $g = 0.61$, 95% CI: 0.21 to 1.01), and problem solving (WSCT-64; $g = 0.48$ 95% CI: 0.09 to 0.88). In contrast, there was no significant effect size for inhibition and cognitive flexibility (D-KEFS Colour; $g = 0.29$ 95% CI: -0.11 to 0.68; D-KEFS Trail making switching; $g = 0.06$ 95% CI: -0.34 to 0.45).

In addition, two non-randomized studies indicated improvements in executive function after treatment. First, the study by Walter et al. (2010) reported that trauma-focused psychotherapy had a medium effect on cognitive flexibility/set-shifting (D-KEFS; Cohen's $d = 0.59$) and organization/planning (BQSS; Cohen's $d = 0.48$) in 15 completers with PTSD compare to 5 non-completers after three months follow up. Second, the study by Haaland et al. (2016) reported that trauma focused psychotherapy had a small effect in inhibition/switching (D-KEFS; Cohen's $d = 0.19$) and working memory (WAIS-IV Digit span and Arithmetic; Cohen's $d = 0.21$) among female PTSD patients with war related trauma.

Another non-randomized study used virtual reality graded exposure therapy (VR-GET) treatment to improve neurocognitive performance in 15 patients with war-related PTSD (McLay et al., 2014). VR-GET combined graded virtual reality exposure with physiological monitoring and skills training. This treatment provided a participant to confront and tolerate simulated memories and fears more fully within the VR environment (McLay et al., 2014). The results showed no significant improvements in attention and executive function (ARES; $\alpha = .05$; C-Stroop, $p = .55$; I-Stroop, $p = .09$). The results of Schindler et al. (2020) also showed no significant improvement in working memory following CBT in PTSD patients as compared to non-traumatized healthy control (Holm–Bonferroni adjusted $p = \geq .41$).

3.5.3. Information processing speed

We found only two RCTs that reported information processing speed outcomes following psychological treatment. First, the study by Nijdam et al. (2018) reported that information processing speed improved

over the course of two trauma-focused psychotherapies (either BEP or EMDR). The effect sizes of EMDR versus BEP for information processing speed were non-significant (Stroop card 2; $g = 0.26$ 95% CI: -0.16 to 0.68; TMT-A, $g = 0.31$ 95% CI: -0.11 to 0.73). Second, Jak et al. (2019) reported that both SMART-CPT and CPT groups showed improvements in processing speed index over time (at posttreatment and at three months follow up). The effect size of SMART-CPT versus CPT for processing speed was not significant (WAIS-IV processing speed index; $g = -0.07$, 95% CI: -0.32 to 0.47). In addition, a non-randomized study by McLay et al. (2014) reported no significant improvements in information processing speed after treatment with VR-GER.

3.6. Quality assessment

Overall, the quality of the included studies in this review varied (Appendix D, online supplementary material). The methods of sequence generation were judged to pose 'low' risk of bias for four studies, one study was rated 'high' risk and another study was rated 'unclear'. Allocation concealment was judged 'low' risk for three studies and the remainder rated 'unclear'. Incomplete outcome data was judged as 'low' risk for four studies, and two other studies reported completers only analysis. Selective reporting was judged 'low' risk across six studies. The blinding of participants and personnel was judged as 'high' risk across six studies. The blinding of the outcome assessments was judged as 'low' risk for five studies, and as 'high risk' for one study. Four studies appeared to be free of other bias sources, one study had insufficient information to assess whether an important risk of bias exist and one study high risk for other source of bias. In sum, our review included one study that met all six risks of bias criteria, three studies met five criteria, and two studies met three criteria (Appendix E, online supplementary material).

4. Discussion

This systematic review examined the existing evidence for the effects of psychological interventions on improvements in neurocognitive functioning in PTSD patients. The number of included studies was relatively small, namely five RCTs and four non-randomized intervention studies including a total of 285 patients (147 in the experiment groups and 138 in the control groups) for RCT and 180 for the non-randomized intervention studies that examined at least one of the following outcomes: attention/working memory, memory, executive function and information processing. Treatment samples varied in size, ranging from 15 to 100 participants. The study participants varied from veterans, the victims of assault, road

traffic accidents, domestic violence, abuse rape, cancer, the witness of death, and robbery victims. Treatments examined in the RCTs were trauma-focused CBT, BEP, PE, EMDR, CPT, and resilience oriented treatment, whereas the non-randomized studies used trauma-focused CBT and VR-GER.

We did not pool the data because none of these studies met the criterion of at least 3 studies using the same type of neuropsychological outcome that compared a psychological treatment to an inactive control group. The studies showed that the effect sizes of treatment on memory were medium to high, although some of the effect sizes were non-significant. Meanwhile, the effect sizes of the treatment on other neurocognitive functions were mostly non-significant. These results support the beneficial effect of PTSD treatments on memory rather than on any of the other measures of neurocognitive functioning.

Several mechanisms may be assumed to explain the effects of psychological treatments on neurocognitive functions, specifically memory. Some have argued that PTSD can be considered a memory function disorder (e.g. van Marle, 2015), as patients suffer from too vivid and emotional memories that are poorly integrated into long-term memory. It has been suggested that in PTSD, traces of the traumatic memory stay primarily in the perceptual and subcortical areas, and may not be not properly integrated within the autobiographical memory system, which resides in the cortical memory network (van Marle, 2015). We found that memory functions improve too as a result of successful PTSD treatment. It has been suggested that effective treatment such as CBT may normalize hippocampal volumes in PTSD patients (Jak et al., 2018). Lindauer et al. (2008) reported that normalized activity in the dorsolateral prefrontal cortex after successful treatment with BEP was associated with increased control of unwanted memories. In addition, psychological treatments seem to normalize the fear network in PTSD patients through improved executive function in the prefrontal cortex, which inhibits emotional responses in the limbic system (Quidé, Witteveen, El-Hage, Veltman, & Olff, 2012). Likewise, a decrease in neurocognitive function is assumed to increase the risk of persistent PTSD symptoms by reducing such cognitive control mechanisms and/or impede recovery through its potential effect on coping mechanisms and ability to benefit maximally from treatment effect (Jacob et al., 2019).

The current review complements previous studies that reported that PTSD severity is associated with decreased cognitive performance (Clausen et al., 2017; Schuitevoerder et al., 2013) and that the effects of effective PTSD treatments extend beyond reducing PTSD symptoms by improving associated neurocognitive functions as well. Improved cognitive performance of patients following treatment is likely to be

noticed by patients in their daily lives. For example, they may experience a better recall of information presented in therapy or improvement in their day-to-day social or occupational functioning (Geuze, Vermetten, De Kloet, Hijman, & Westenberg, 2009).

We found that most psychological interventions did not significantly improve neurocognitive function with the exception of memory functions. Interestingly, the studies included in this review were trauma-focused treatments that were not designed to directly address neuropsychological functioning. These studies should be distinguished from studies that examined the effects of cognitive training (e.g. computerized cognitive training, attention control training) that focused directly on neurocognitive function (e.g. interference control-sub-function of executive function, attention bias) (Badura-Brack et al., 2015; Bomyea, Stein, & Lang, 2015). However, cognitive training studies also showed unclear improvements in terms of neurocognitive outcomes (Badura-Brack et al., 2015; Bomyea et al., 2015; Schoorl, Putman, & Van Der Does, 2013).

Finally, the current study tentatively suggested that combined treatment strategies (e.g. CBT with pharmacotherapy; SMART-CPT) had a more positive impact on improving memory function in PTSD patients than single treatments. Interestingly, previous studies reported that pharmacotherapy treatment with paroxetine was associated with improvements in verbal declarative memory function in PTSD (Fani et al., 2009; Vermetten, Vythilingan, Southwick, Charney, & Bremner, 2003). This finding may merit further investigation.

4.1. Limitations

This review has several limitations. First, the small number of studies limited the generalizability of the conclusions and prevented from pooling the data. Second, several studies examined multimodal interventions, consisting of multiple effective components (e.g. combined pharmacotherapy with CBT or SMART-CPT), making it difficult to conclude which specific component was effective. In these studies (Akbarian et al., 2015; Jak et al., 2019), it was not clear whether these treatments would have been effective without such adjuncts. Third, this review showed that most studies were very heterogeneous in terms of how neurocognitive outcomes were measured and instruments and test batteries used. Due to the limited number of studies using the same types of outcomes, we could not compare the differential effects of psychological treatments on different aspects of memory function, such as anterograde memory and autobiographical memory. In addition, we also included non-evidence-based interventions of which the effectiveness has not been confirmed (e.g. resilience

oriented treatment). Furthermore, since we also included studies with active control arms, it is difficult to draw firm conclusions regarding the effectiveness of PTSD interventions in general.

5. Conclusions

This systematic review examined the effects of psychological treatments on neurocognitive functioning in adults with PTSD. We found no evidence for improvements as a result of PTSD treatment for most neurocognitive domains examined, including attention, executive function and information processing speed. However, the effect sizes were predominantly significant for memory. This review was a first step to gather existing research and provide a trigger for future research on the effect of psychological treatments on neurocognitive functioning in PTSD.

For clinicians treating patients with PTSD, it relatively safe to expect that when treatment is effective, memory functioning is also likely to improve. Since impaired memory affects daily functioning of patients to a great extent, this is an important benefit that will add to patients' quality of life.

For future research, there is a need for consensus among researchers concerning the most appropriate instruments and norm standards for neurocognitive measurements in PTSD patients. Furthermore, future studies should be informed by a sample size calculation to ensure they are adequately powered to detect significant effects. Finally, longitudinal designs with follow-up assessments are needed to examine longer-term effects of psychological treatments on neurocognitive functioning.

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