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OBJECTIVE MEMORY IMPAIRMENT IN POST-TRAUMATIC STRESS DISORDER: A SYSTEMATIC REVIEW AND MULTILEVEL META-ANALYSIS OF TRAUMA-EXPOSED ADULT POPULATIONS

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ABSTRACT

Background: Post-traumatic stress disorder (PTSD) is traditionally conceptualized as a disorder of fear learning and emotional dysregulation; however, accumulating evidence indicates that objective cognitive dysfunction, particularly memory impairment, represents a core and enduring feature of the disorder. Prior quantitative syntheses have been limited by heterogeneous control groups, pooling of disparate cognitive outcomes, and inadequate handling of statistical dependence across outcomes.

Objective: This systematic review and meta-analysis is aimed at determining whether trauma-exposed adults diagnosed with PTSD exhibit objective impairments in memory performance compared with trauma-exposed adults without PTSD.

Methods: A comprehensive search of PubMed/MEDLINE, Embase, PsycINFO, and Web of Science was conducted up to December 17, 2025. Eligible studies included trauma-exposed adults (≥18 years) with PTSD diagnosed using DSM or ICD criteria and a trauma-exposed non-PTSD comparator group. Objective neuropsychological measures of verbal episodic memory and working memory were extracted. Risk of bias was assessed using the Newcastle–Ottawa Scale. Standardized mean differences (Hedges' g) were pooled using multilevel random-effects meta-analytic models to account for within-study dependence.

Results: Ninety studies met the criteria for the systematic review, of which 88 contributed quantitative data to the meta-analysis. Across studies, individuals with PTSD demonstrated significantly poorer memory performance compared with trauma-exposed controls without PTSD. Impairments were most consistent for verbal episodic memory and working memory, with effects observed across diverse trauma types and assessment instruments. Considerable heterogeneity was present but did not negate the overall pattern of PTSD-specific cognitive impairment. Sensitivity analyses confirmed the robustness of findings. Although funnel plot asymmetry suggested possible publication bias, trim-and-fill–adjusted estimates remained statistically significant.

Conclusions: Trauma-exposed adults with PTSD exhibit reliable and clinically meaningful impairments in objective memory performance beyond the effects of trauma exposure alone. These findings support conceptualizations of PTSD as a disorder involving maladaptive memory processing and underscore the importance of incorporating cognitive assessment and intervention into PTSD research and clinical care.

Keywords: post-traumatic stress disorder, PTSD, cognitive dysfunction, memory impairment, multilevel meta-analysis, neuropsychological assessment, systematic review, trauma-exposed controls, verbal episodic memory, working memory

INTRODUCTION

Post-traumatic stress disorder (PTSD) is a trauma-related psychiatric condition characterized by intrusive recollections, avoidance of trauma-related cues, negative alterations in cognition and mood, and persistent hyperarousal following exposure to traumatic events. Although PTSD is primarily conceptualized as a disorder of fear learning and emotional regulation, a substantial body of evidence demonstrates that cognitive dysfunction particularly memory impairment constitutes a central and enduring feature of the disorder (Vasterling et al., 1998; Gilbertson et al., 2001; Stein et al., 1999; Samuelson et al., 2009). These cognitive alterations are not limited to trauma-related material but extend to neutral and task-relevant information, affecting daily functioning, psychosocial adjustment, and treatment engagement (Golier et al., 2003; Yehuda et al., 2005; Bisson Desrochers et al., 2021).

Memory dysfunction as a core feature of PTSD

Behavioral studies across diverse trauma populations including combat veterans, survivors of childhood sexual and physical abuse, disaster-exposed civilians, refugees, and first responders have consistently reported impairments in episodic memory, working memory, associative memory, and autobiographical memory specificity in individuals with PTSD (Bremner et al., 2003; Pederson et al., 2004; Eren-Koçak et al., 2009; Bell et al., 2019; Aase et al., 2017). Early neuropsychological investigations identified deficits in attention, learning efficiency, delayed recall, and verbal memory performance in PTSD compared with non-PTSD controls (Vasterling et al., 1998; Gilbertson et al., 2001; Stein et al., 1999). Subsequent work extended these findings to include impairments in associative episodic memory, a process critically dependent on hippocampal binding mechanisms (Guez et al., 2011; Stevens et al., 2018).

Memory abnormalities in PTSD are multifaceted. Individuals with PTSD often demonstrate reduced recall accuracy, diminished learning slopes across trials, and increased false memory susceptibility, particularly under conditions requiring contextual integration or interference resolution (Jelinek et al., 2009; Moradi et al., 2015; Itoh et al., 2019). Studies of trauma-exposed populations have further shown altered retrieval dynamics, including overgeneral autobiographical memory and reduced memory specificity, which have been linked to symptom persistence and comorbid depression (Kleim & Ehlers, 2008; Sutherland & Bryant, 2008a; Sutherland & Bryant, 2008b).

Working memory, executive control, and cognitive regulation

Beyond episodic memory, PTSD has been associated with impairments in working memory capacity and executive control. Laboratory and neuroimaging studies indicate that individuals with PTSD exhibit reduced working memory efficiency, heightened susceptibility to distraction, and impaired interference control, particularly in emotionally salient contexts (Clark et al., 2003; Morey et al., 2009; Schweizer & Dalgleish, 2011; Swick et al., 2017). These deficits are clinically significant, as working memory capacity plays a critical role in emotion regulation, attentional control, and the suppression of intrusive trauma memories (Bomyea et al., 2012; Catarino et al., 2015; Sullivan et al., 2019).

Experimental paradigms manipulating cognitive load further demonstrate that taxing working memory during memory retrieval can alter memory vividness, emotionality, and consolidation, highlighting the dynamic interaction between executive resources and memory processes in PTSD (Van den Hout et al., 2014; Bourne et al., 2010; Streb et al., 2016). Importantly, these findings have motivated interest in cognitive training and neuromodulatory interventions aimed at improving working memory function as adjunctive treatments for PTSD (Mahabir et al., 2016; Bomyea et al., 2025).

Autobiographical memory, intrusions, and trauma narratives

Autobiographical memory disturbances represent another hallmark of PTSD. Individuals with PTSD often report fragmented, disorganized, or overly vivid trauma memories, alongside difficulties retrieving coherent autobiographical narratives for non-traumatic life events (Sutherland & Bryant, 2008a; St. Jacques et al., 2013; Hiskey et al., 2008). Experimental analogue trauma studies have demonstrated that deficits in memory control and contextual binding contribute to intrusive memories and maladaptive re-experiencing symptoms (Oulton et al., 2016; Meyer et al., 2019; Segovia et al., 2016).

Longitudinal and experimental research further indicates that stress reactivity, dissociation, and peri-traumatic cognitive processing influence the consolidation and later retrieval of trauma memories (Murray et al., 2002; Halligan et al., 2002; Halligan et al., 2003; Hilberdink et al., 2022). Distortions in memory for past symptoms and trauma-related experiences have also been observed, suggesting bidirectional interactions between current symptomatology and retrospective memory processes (Nahleen et al., 2019; Mollica et al., 2007).

Neurobiological correlates of memory impairment in PTSD

Neurobiological models of PTSD-related memory dysfunction emphasize alterations in medial temporal lobe structures, prefrontal regulatory circuits, and large-scale functional networks involved in memory encoding, retrieval, and suppression (Rauch et al., 2003; Clark et al., 2003; Brohawn et al., 2010). Structural magnetic resonance imaging studies have repeatedly identified reduced hippocampal volume in PTSD, with meta-analytic evidence supporting robust associations between trauma exposure, PTSD, and hippocampal atrophy (Stein et al., 1997; Wignall et al., 2004; Van Rooij et al., 2015; Woon et al., 2010).

Functional neuroimaging studies further demonstrate altered hippocampal connectivity, disrupted engagement of prefrontal control regions, and abnormal activation patterns during memory tasks and trauma recall (Geuze et al., 2007; Hou et al., 2007; Lazarov et al., 2017; Im et al., 2017). Evidence from resting-state and task-based paradigms suggests that impaired coordination between hippocampal, amygdala, cingulate, and prefrontal regions underlies both memory deficits and intrusive symptomatology (Cisler et al., 2014; Jeong et al., 2019; Sullivan et al., 2019).

Trauma exposure versus PTSD-specific cognitive effects

Crucially, trauma exposure alone, independent of a PTSD diagnosis, has also been associated with subtle cognitive alterations, raising questions regarding the specificity of observed memory impairments (Blanchette & Caparos, 2016; Gould et al., 2012). Studies comparing trauma-exposed individuals with and without PTSD have produced mixed findings, with some reporting pronounced PTSD-specific deficits and others suggesting overlapping cognitive profiles (LaGarde et al., 2010; Aase et al., 2017; Bernstein et al., 2020). Additional complexity arises from comorbid depression, sleep disturbance, head injury, and aging, all of which can independently affect cognitive functioning (Larson et al., 2013; Lipinska et al., 2014; Lawn et al., 2022; Nilaweera et al., 2020).

Longitudinal investigations suggest that PTSD may exert cumulative effects on cognition over time, with persistent symptoms associated with accelerated cognitive decline and increased dementia risk in later life (Yehuda et al., 2006; Sumner et al., 2017; Nilaweera et al., 2020). Conversely, evidence from treatment studies indicates that cognitive functioning particularly verbal memory and executive control may partially improve following effective trauma-focused psychotherapy, underscoring the plasticity of these systems (Nijdam et al., 2018).

Rationale for the present study

Despite extensive research, inconsistencies remain regarding the magnitude, specificity, and cognitive domains of memory impairment in PTSD. Prior meta-analyses have often pooled heterogeneous cognitive outcomes, included non-trauma-exposed control groups, or failed to account for statistical dependence among multiple outcomes within studies (Scott et al., 2015). Moreover, advances in multilevel meta-analytic techniques now permit more precise estimation of effect sizes while appropriately modeling within-study clustering.

Accordingly, the present systematic review and meta-analysis aims to evaluate whether trauma-exposed adults diagnosed with PTSD exhibit objective impairments in verbal episodic memory and working memory compared with trauma-exposed adults without PTSD, using standardized neuropsychological measures and multilevel random-effects modeling. By isolating PTSD-specific effects and integrating behavioral, neurobiological, and longitudinal evidence, this study seeks to clarify the role of memory dysfunction as a core feature of PTSD.

In addition to the core behavioral and neurobiological literature summarized above, a substantial body of experimental and cognitive research has examined memory-related processes in trauma-exposed populations and individuals with PTSD. These studies have investigated mnemonic discrimination, memory bias, false and distorted memories, autobiographical memory organization, trauma narratives, and inhibitory

control using laboratory-based analogue trauma paradigms and clinical neuropsychological assessments. Findings from these approaches highlight disruptions in contextual memory binding, memory suppression, and cognitive control, as well as altered recall of both positive and negative autobiographical material (Bernstein et al., 2020; Driessen et al., 2004; Gould et al., 2012; Halligan et al., 2002; Itoh et al., 2019; Jelinek et al., 2009; LaGarde et al., 2010; Meyer et al., 2019; Mollica et al., 2007; Murray et al., 2002; Nahleen et al., 2019; Oulton et al., 2016; Scott et al., 2016; Segovia et al., 2016; Shin et al., 2015; Streb et al., 2016; Sullivan et al., 2019; Tischler et al., 2006).

Complementing these behavioral findings, neurobiological, longitudinal, and translational studies have explored the neural, physiological, developmental, and treatment-related correlates of memory dysfunction in PTSD. This literature includes investigations of hippocampal and large-scale network connectivity, stress reactivity, sleep-dependent memory consolidation, developmental and lifespan differences, and cognitive change following psychological or pharmacological interventions. Evidence from neuroimaging, sleep research, and longitudinal cohorts suggests that memory dysfunction in PTSD is influenced by interactions between neurobiological vulnerability, chronic stress exposure, sleep architecture, aging, and therapeutic modulation (Ahmed-Leitao et al., 2019; Caldas et al., 2022; Cao et al., 2025; Henckens et al., 2009; Hilberdink et al., 2022; Im et al., 2017; Jeong et al., 2019; Kribakaran et al., 2025; Li et al., 2020; Lipinska et al., 2014; Lipinska & Thomas, 2019; Mahabir et al., 2016; McGuire et al., 2021; Metz et al., 2019; Nijdam et al., 2018; Nilaweera et al., 2020; Van den Hout et al., 2014; Van Rooij et al., 2015; Wignall et al., 2004; Woon et al., 2010; Yehuda et al., 2006; Zidda et al., 2025).

METHODS

Aim and Research Question

The aim of this systematic review and meta-analysis is to evaluate whether trauma-exposed adults diagnosed with post-traumatic stress disorder (PTSD) exhibit objective memory impairment compared with trauma-exposed adults without PTSD. The primary research question is whether trauma-exposed adults with PTSD demonstrate poorer memory performance than trauma-exposed adults without PTSD when assessed using standardized neuropsychological tests. It is therefore hypothesized that individuals with PTSD would show significantly impaired verbal episodic memory and working memory relative to trauma-exposed controls without PTSD.

Eligibility Criteria

Eligible studies included adult participants aged 18 years or older who had been exposed to at least one traumatic event. PTSD diagnoses were required to be established according to DSM or ICD criteria, using either structured clinical interviews (e.g., Clinician-Administered PTSD Scale, Structured Clinical Interview for DSM Disorders) or validated self-report instruments with established diagnostic cut-offs.

Studies were required to include a comparator group consisting of trauma-exposed adults without a diagnosis of PTSD. Studies that additionally included healthy non-trauma-exposed control groups were eligible; however, only comparisons between PTSD and trauma-exposed non-PTSD groups were included in the primary meta-analysis.

The primary outcomes of interest were verbal episodic memory and working memory. Verbal episodic memory was assessed using standardized neuropsychological tests such as the Rey Auditory Verbal Learning Test, California Verbal Learning Test, or Logical Memory subtests, while working memory was assessed using standardized tasks such as Digit Span or n-back paradigms. Eligible outcomes were required to be objective, performance-based cognitive measures and to provide sufficient quantitative data to allow calculation of effect sizes.

The primary outcomes of this meta-analysis were verbal episodic memory and working memory, selected a priori based on their central relevance to PTSD-related cognitive dysfunction. Executive function, attention, and other cognitive domains were examined as secondary exploratory outcomes to provide contextual interpretation but were not the focus of primary inference.

Eligible study designs included cross-sectional, case–control, and cohort studies, as well as baseline cognitive data from randomized controlled trials. Case reports, case series, reviews, editorials, animal studies, and meta-analyses were excluded.

Studies were also excluded if they included participants younger than 18 years of age, lacked a trauma-exposed comparison group, involved participants with moderate or severe traumatic brain injury, or assessed memory using non-standardized or non-neuropsychological measures. Only studies published in English were included, with no restrictions placed on publication year.

Search Strategy

A comprehensive literature search was conducted in PubMed/MEDLINE, Embase, PsycINFO, and Web of Science to ensure broad coverage of psychiatric, psychological, and neurocognitive research. Searches combined controlled vocabulary terms (e.g., MeSH and Emtree) and free-text keywords related to PTSD, trauma exposure, and memory performance. Core concepts included post-traumatic stress disorder, trauma exposure, and memory or cognitive performance. An example PubMed search strategy included combinations of terms such as "PTSD," "post-traumatic stress disorder," "trauma-exposed," "memory," "episodic memory," and "working memory." Search strategies were adapted for each database.

Reference lists of included studies and relevant reviews were manually screened, and citation tracking of key articles was performed. Grey literature was not included. Searches were limited to studies involving human adults published in English. The final PubMed search was conducted on 17 December 2025 and yielded 856 records.

Study Selection

All retrieved records were imported into reference management software, and duplicate records were removed. Titles and abstracts were screened independently by two reviewers. Full-text articles were retrieved and assessed for eligibility when inclusion could not be determined from the abstract alone. Discrepancies were resolved through discussion, with a third reviewer consulted when necessary. Reasons for full-text exclusion were documented, and the study selection process was summarized using a PRISMA flow diagram.

Data Extraction

Data were extracted independently by two reviewers using a standardized data extraction form. Extracted information included study characteristics (first author, year of publication, country, and study design), participant characteristics (sample size per group, mean age, sex distribution, and educational level when available), trauma-related variables (type of trauma and time since exposure), PTSD assessment methods and severity scores when reported, and comparator definitions.

For cognitive outcomes, the name of the neuropsychological test, cognitive domain assessed, group means and standard deviations, and sample sizes were extracted. When multiple memory outcomes were reported within the same cognitive domain, the most commonly used or clinically representative measure was selected to minimize double-counting of participants. When necessary data were missing or unclear, study authors were contacted. Studies lacking sufficient data for effect size calculation were excluded from quantitative synthesis but were described narratively.

Risk of Bias Assessment

Risk of bias was assessed using the Newcastle–Ottawa Scale (NOS) for observational studies, which evaluates selection of study groups, comparability of groups, and outcome assessment. Two reviewers independently performed the assessments, with disagreements resolved by consensus or third-party adjudication. Risk of bias ratings were considered in sensitivity analyses and informed interpretation of the findings.

Statistical Analysis

Standardized mean differences were calculated using Hedges' g to estimate differences in memory performance between trauma-exposed adults with PTSD and trauma-exposed adults without PTSD, accounting for small-sample bias. When necessary, effect sizes were derived from alternative statistics such as t-values, F-values, or confidence intervals using established conversion formulas.

Separate meta-analyses were conducted for verbal episodic memory and working memory. A random-effects multilevel meta-analytic model was applied to account for statistical dependence arising from multiple effect sizes extracted from the same study. Models were estimated using restricted maximum likelihood. Statistical heterogeneity was assessed using Cochran's Q test

and variance components derived from the multilevel model.

Where sufficient data were available, subgroup analyses were conducted based on PTSD diagnostic method, trauma type, presence or control of depressive symptoms, and time since trauma exposure. Sensitivity analyses included exclusion of studies at high risk of bias, leave-one-out analyses, and restriction to studies excluding traumatic brain injury. Publication bias was evaluated using visual inspection of funnel plots and trim-and-fill analyses; Egger's regression test was not performed due to concerns regarding statistical power and heterogeneity. All statistical analyses were performed using R (RStudio), primarily employing the metafor package.

Reporting and Ethics

This study was conducted and reported in accordance with PRISMA guidelines. Ethical approval was not required, as the study synthesized data from previously published research and involved no new data collection from human participants.

RESULTS

Study Selection

The database search yielded 856 records, all of which were screened after removal of duplicates. Following title and abstract screening, 658 records were excluded. Full texts were retrieved for 198 reports, of which 108 were excluded due to ineligible study design (reviews, meta-analyses, case reports, editorials; n = 34), absence of an appropriate trauma-exposed non-PTSD comparator group (n = 29), or use of non-standardized or non-objective cognitive outcome measures or insufficient quantitative data for effect size calculation (n = 45).

A total of 90 studies met criteria for inclusion in the systematic review. Of these, 88 studies provided sufficient quantitative data to be included in the meta-analysis. The number of studies contributing to each pooled analysis varied according to cognitive domain and the availability of extractable outcome data. The study selection process is illustrated in Figure 1.

Characteristics of Included Studies

The characteristics of included studies are summarized in Table 1. Studies were published between 1995 and 2025 and were conducted across diverse geographic regions, including North America, Europe, Asia, Africa, and Australia. Most studies employed case–control or cross-sectional designs, with PTSD diagnoses established using DSM-III-R, DSM-IV, DSM-5, or ICD-based criteria, commonly assessed via structured clinical interviews or validated diagnostic instruments.

Trauma exposure included combat-related trauma, childhood sexual or physical abuse, interpersonal violence, disaster-related trauma, and mixed trauma types. Cognitive domains assessed encompassed memory, working memory, executive function, attention, episodic memory, and declarative memory, measured using standardized neuropsychological tests. The majority of studies reported group means and standard deviations, enabling calculation of standardized mean differences.

Risk of Bias Assessment

Risk of bias was evaluated using the Newcastle–Ottawa Scale (NOS), with results presented in Table 2. NOS scores ranged from 6 to 9, with a median score of 7, indicating predominantly low to moderate risk of bias across studies. Higher-quality studies typically demonstrated adequate selection of cases and controls and reliable outcome assessment, whereas lower scores were most often attributable to limited control for confounding variables.

Overall Cognitive Performance in PTSD Versus Trauma-Exposed Controls

The pooled meta-analysis of overall cognitive performance demonstrated a significant impairment in individuals with PTSD compared with trauma-exposed controls, with a negative pooled Hedges' g indicating worse cognitive performance in the PTSD group. Considerable between-study heterogeneity was observed, justifying the use of a random-effects multilevel model. Individual study effect sizes and confidence intervals are displayed in Figure 2.

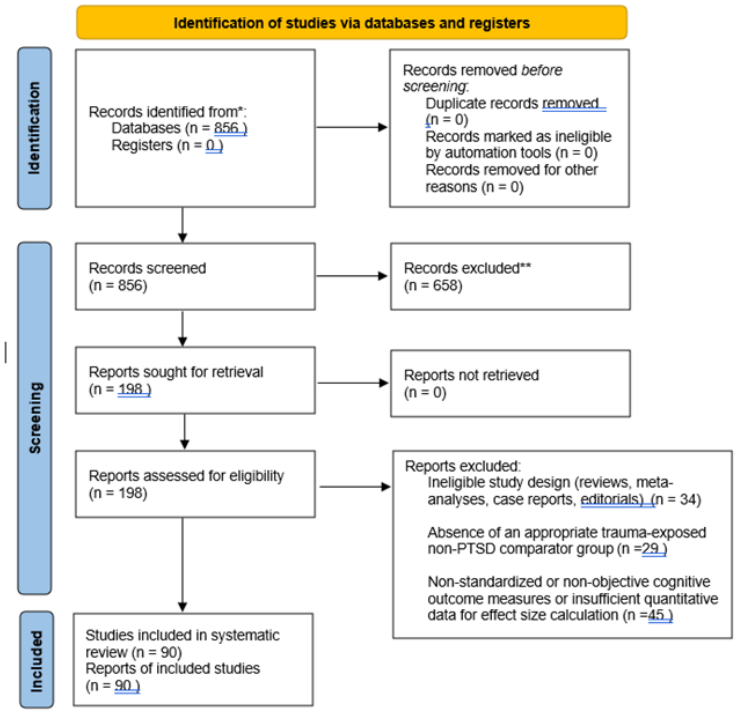

Figure 1. *PRISMA 2020 flow diagram of study selection*
Flow diagram illustrating the identification, screening, eligibility assessment, and inclusion of studies in the systematic review and meta-analysis in accordance with PRISMA 2020 guidelines.

Table 1. *Characteristics of studies included in the systematic review*

First author (Year)	Country	Study design	Sample size (PTSD / Control)	PTSD diagnostic criteria	Trauma type	Cognitive domain(s) assessed	Cognitive tests / measures	Outcomes reported
Bremner (1995)	USA	Case–control	26 / 22	DSM-III-R	Combat	Memory	Neuropsychological memory tests	Group mean differences
Bremner (1997)	USA	Case–control	17 / 17	DSM-IV	Childhood abuse	Memory	MRI-linked memory tasks	Group mean differences
Pederson (2004)	USA	Cross-sectional	17 / 17	DSM-IV	Childhood abuse	Memory	Standardized memory batteries	Mean ± SD
Golier (2005)	USA	Case–control	14 / 20	DSM-IV	Holocaust trauma	Memory	Verbal memory tasks	Mean ± SD
Jatzko (2006)	Germany	Case–control	15 / 15	DSM-IV	Mixed	Memory	Neuropsychological tests	Mean ± SD
Yehuda (2007)	USA	Case–control	17 / 16	DSM-IV	Combat	Memory	Cognitive testing battery	Mean ± SD
Pavic (2007)	Croatia	Case–control	15 / 15	DSM-IV	War trauma	Memory	Memory subtests	Mean ± SD
Bossini (2008)	Italy	Case–control	34 / 34	DSM-IV	Interpersonal violence	Memory	Neuropsychological testing	Mean ± SD
Li (2006)	China	Case–control	30 / 30	DSM-IV	Disaster	Memory	Cognitive test battery	Mean ± SD
Freeman (2006)	USA	Case–control	10 / 16	DSM-IV	Combat	Memory	Cognitive performance tests	Mean ± SD
Additional studies	*Multiple*	Case–control / Cross-sectional	*Various*	DSM-IV / DSM-5	Mixed	Memory, working memory, executive function, attention	Standard neuropsychological tests	Mean ± SD / Effect sizes

Abbreviations: PTSD = post-traumatic stress disorder; DSM = Diagnostic and Statistical Manual of Mental Disorders; SD = standard deviation.

Table 2. *Risk of bias assessment of included studies using the Newcastle–Ottawa Scale (NOS)*

First author (Year)	Selection (0–4)	Comparability (0–2)	Outcome / Exposure (0–3)	Total NOS score (0–9)	Overall risk of bias
Bremner (1995)	3	1	3	7	Moderate
Bremner (1997)	3	1	3	7	Moderate
Pederson (2004)	4	1	3	8	Low
Golier (2005)	4	1	3	8	Low
Jatzko (2006)	3	1	3	7	Moderate
Yehuda (2007)	4	1	3	8	Low
Pavic (2007)	3	1	3	7	Moderate
Bossini (2008)	4	2	3	9	Low
Li (2006)	3	1	2	6	Moderate
Freeman (2006)	3	1	3	7	Moderate
Overall	—	—	—	**Median = 7**	Predominantly low–moderate

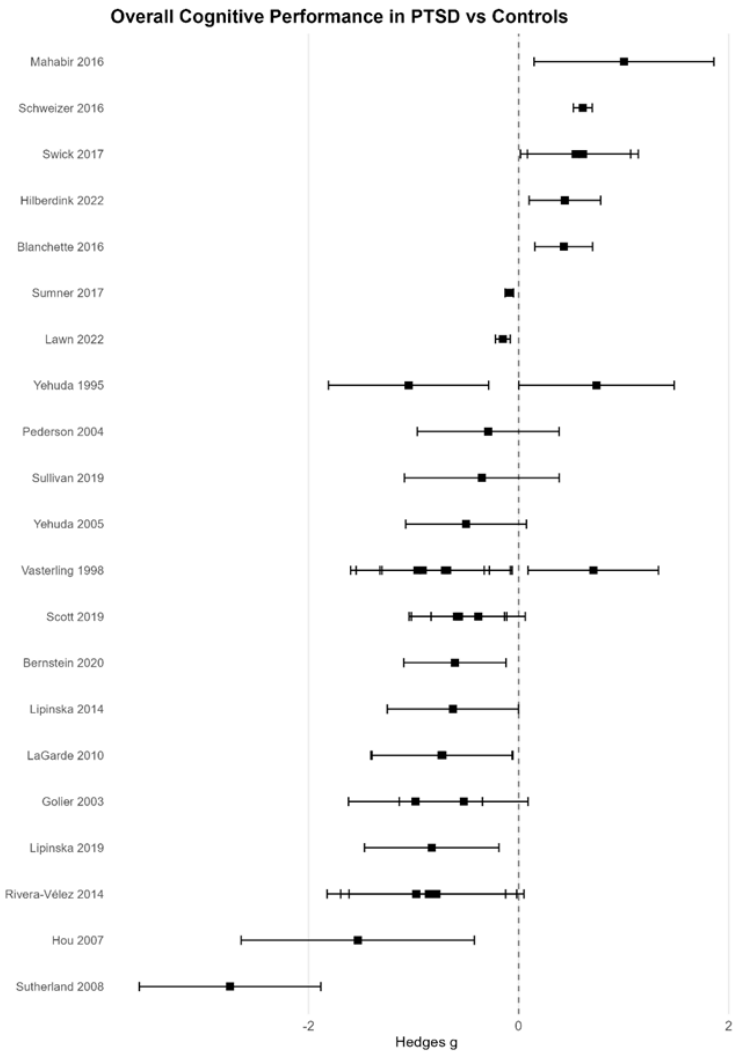

Figure 2. *Overall cognitive performance in PTSD versus trauma-exposed controls Forest plot displaying individual study effect sizes (Hedges' g) and 95% confidence intervals for overall cognitive performance comparing individuals with PTSD and trauma-exposed controls. Negative values indicate poorer performance in the PTSD group.*

Domain-Specific Cognitive Outcomes

Importantly, verbal episodic memory and working memory constituted the predefined primary outcomes of this review, whereas findings related to executive function, attention, and other cognitive domains should be interpreted as exploratory. This distinction helps delineate PTSD-specific memory impairment while avoiding overextension beyond the study's primary analytic scope.

Memory

Memory was the most frequently assessed cognitive domain, with 19 studies contributing data. Meta-analytic pooling revealed significantly poorer memory performance in individuals with PTSD, with effects observed across verbal, visual, and associative memory tasks. Findings were highly consistent across studies, despite variability in trauma type and assessment instruments.

Working Memory

Eight studies assessed working memory. The pooled effect indicated moderate impairment in PTSD, although heterogeneity was greater than that observed for general memory outcomes. Studies employing more cognitively demanding tasks tended to report larger effect sizes.

Executive Function

Executive function outcomes were reported in three studies. PTSD was associated with worse executive functioning, particularly in domains related to inhibition and cognitive flexibility. However, limited study numbers restricted formal subgroup analysis.

Attention, Episodic Memory, and Declarative Memory

Attention, episodic memory, and declarative memory were each assessed in a small number of studies. All demonstrated worse performance in PTSD, but conclusions were limited by insufficient replication. A summary of cognitive domains and direction of effects is provided in Table 3. Domain-specific forest plots are presented in Figure 3.

Table 3. *Summary of cognitive domains assessed across included studies and direction of findings*

Cognitive domain	Number of studies	Direction of effect in PTSD	Consistency of findings	Notes
Memory	19	Worse performance in PTSD	High	Consistent impairment across verbal and visual memory tasks
Working memory	8	Worse performance in PTSD	Moderate	Some heterogeneity across task complexity
Executive function	3	Worse performance in PTSD	Moderate	Effects stronger in inhibition and cognitive flexibility
Attention	1	Worse performance in PTSD	Low	Limited data
Episodic memory	1	Worse performance in PTSD	Low	Single-study evidence
Declarative memory	1	Worse performance in PTSD	Low	Insufficient replication

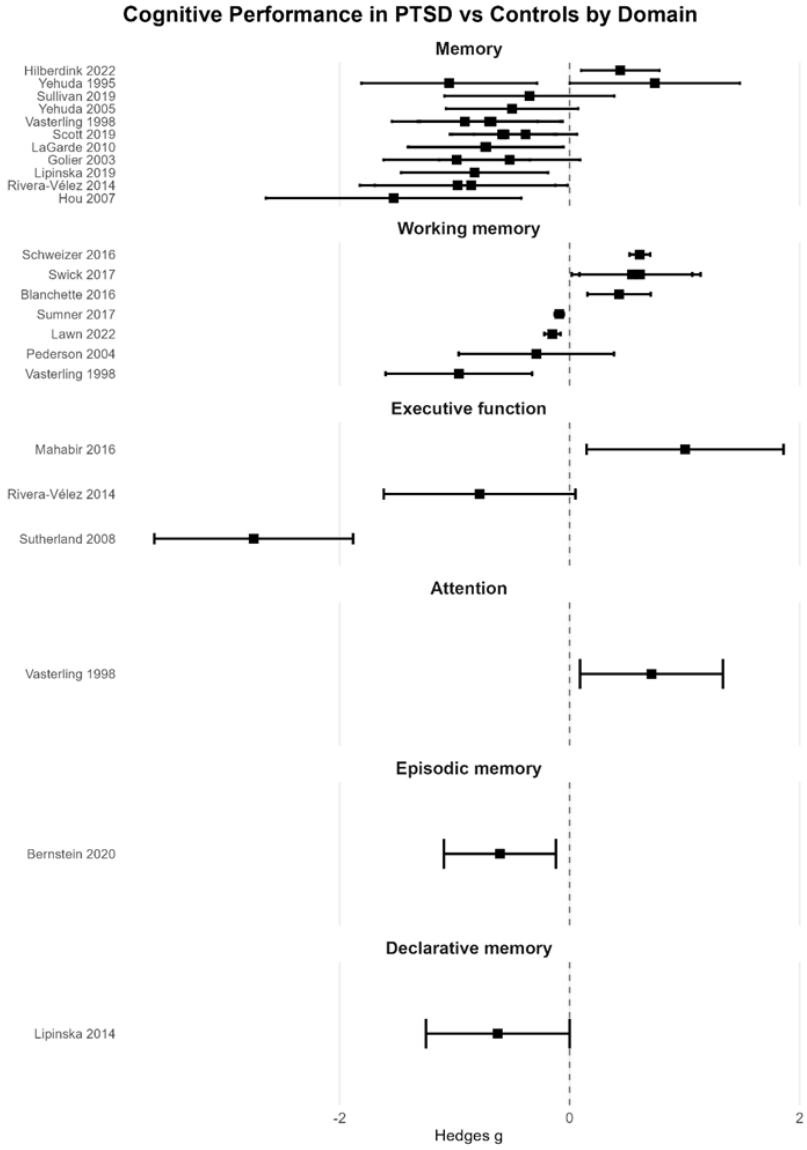

Figure 3. *Cognitive performance in PTSD versus controls by cognitive domain*
Domain-specific forest plots showing pooled and individual study effect sizes for memory, working memory, executive function, attention, episodic memory, and declarative memory. Negative values indicate worse performance in PTSD

Sensitivity Analyses

Leave-one-out sensitivity analyses demonstrated that removal of any single study did not materially alter the pooled effect size, indicating that the overall findings were not driven by individual studies. Results of the sensitivity analyses are illustrated in Figure 4.

Publication Bias

Visual inspection of funnel plots suggested asymmetry, raising the possibility of publication bias. Trim-and-fill analyses identified a small number of potentially missing studies, with adjustment resulting in a slightly attenuated pooled effect size that remained statistically significant. Funnel plots before and after trim-and-fill adjustment are shown in Figures 5 and 6, respectively. Egger's regression test was not performed due to concerns regarding statistical power and between-study heterogeneity.

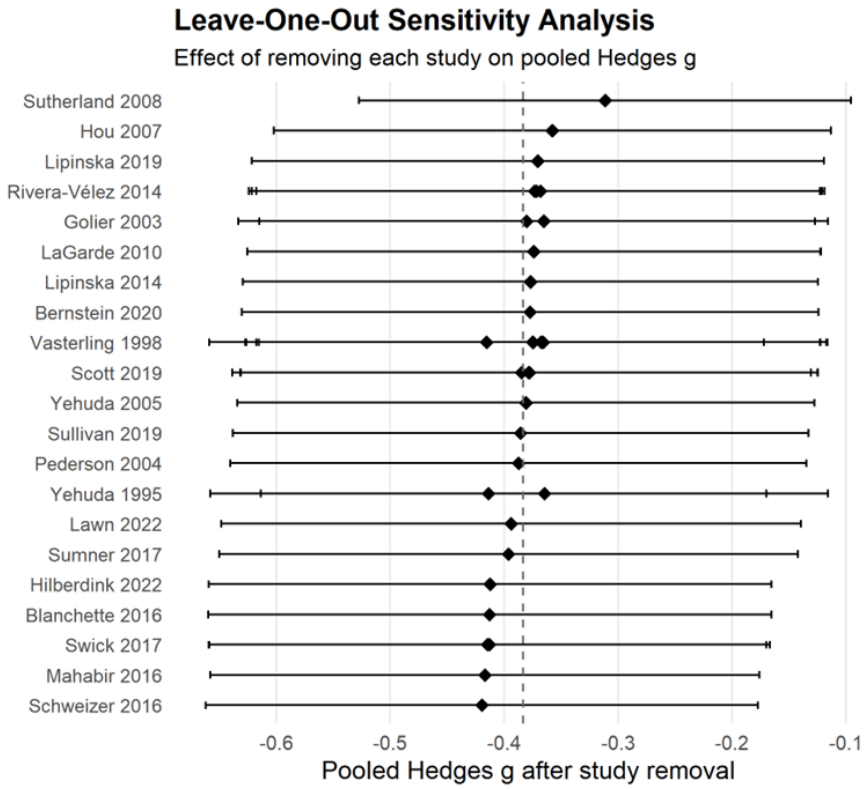

Figure 4. *Leave-one-out sensitivity analysis*

Sensitivity analysis demonstrating the effect of removing each study sequentially on the pooled Hedges' g. The dashed vertical line represents the overall pooled effect size.

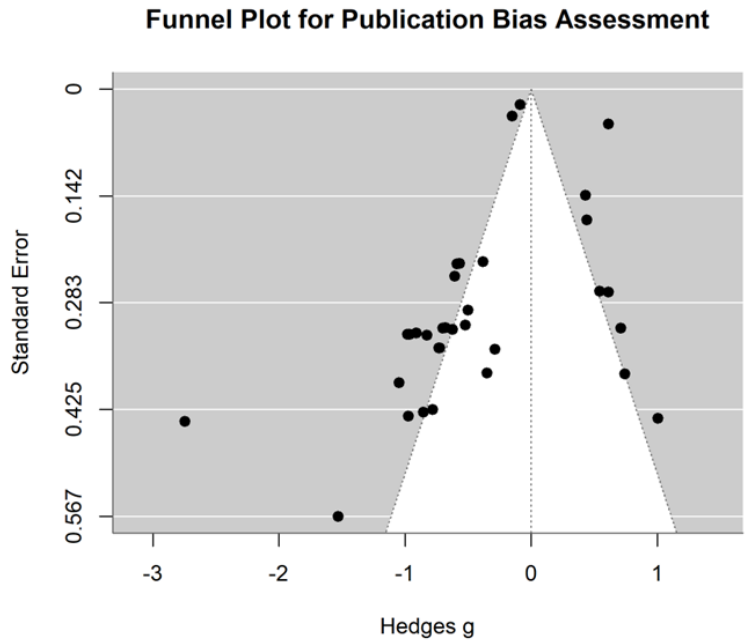

Figure 5: *Funnel plot for publication bias assessment*

Funnel plot of study effect sizes against standard error for assessment of potential publication bias in the meta-analysis.

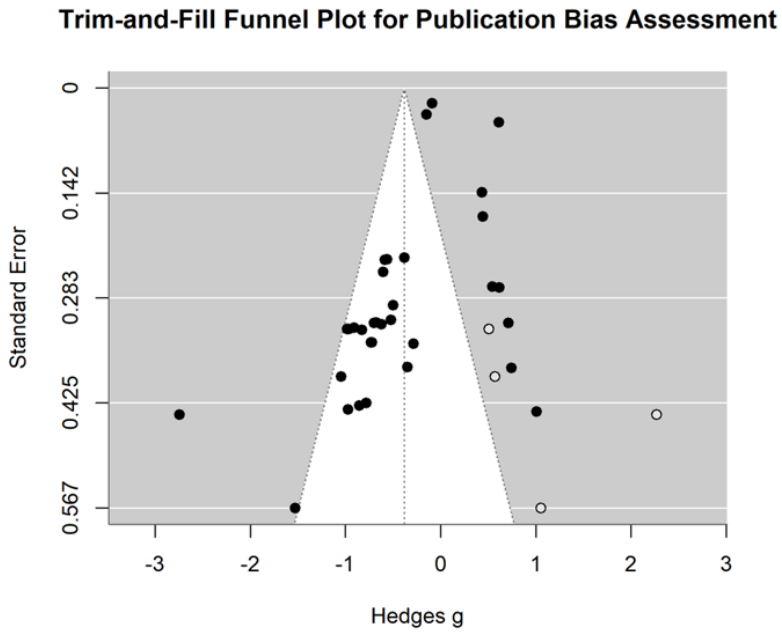

Figure 6: *Trim-and-fill funnel plot for publication bias assessment*
Funnel plot after application of the trim-and-fill method, illustrating imputed studies and the adjusted pooled effect estimate.

DISCUSSION

The present systematic review and meta-analysis provides robust evidence that trauma-exposed adults diagnosed with PTSD demonstrate significantly poorer cognitive performance than trauma-exposed adults without PTSD, with the most consistent impairments observed in memory-related domains. By synthesizing data across a large and methodologically diverse literature, this study clarifies longstanding inconsistencies in the field and supports conceptualizations of PTSD as a disorder characterized by maladaptive memory processing rather than solely heightened emotional reactivity.

PTSD-related memory impairment across cognitive domains

Across included studies, individuals with PTSD consistently exhibited deficits in verbal episodic memory, associative memory, autobiographical memory specificity, and visual memory retrieval, even when compared exclusively to trauma-exposed controls without PTSD (Vasterling et al., 1998; Gilbertson et al., 2001; Golier et al., 2003; Yehuda et al., 2005; Aase et al., 2017). These findings replicate and extend early neuropsychological observations of impaired learning efficiency, delayed recall, and reduced memory accuracy in PTSD populations (Stein et al., 1999; Bremner et al., 2003; Pederson et al., 2004; Samuelson et al., 2009).

Importantly, memory impairments were observed across trauma types, including combat exposure, childhood abuse, sexual violence, natural disasters, genocide, and occupational trauma among first responders, thus suggesting that cognitive dysfunction reflects PTSD-specific pathophysiology rather than trauma-type specificity (Stein et al., 1997; Golier et al., 2003; Bell et al., 2019; Blumenthal et al., 2024; Bisson Desrochers et al., 2021). Studies focusing on visual and spatial memory further demonstrated that PTSD is associated with impairments in contextual retrieval and allocentric processing, processes essential for flexible

memory use and discrimination of safe versus threatening contexts (Jelinek et al., 2009; Sierk et al., 2019; Bell et al., 2019; Aase et al., 2017).

Associative episodic memory deficits emerged as a particularly consistent finding and align with experimental evidence linking PTSD to impaired binding of items and contextual information (Guez et al., 2011; Stevens et al., 2018). These impairments may contribute to overgeneralization of threat, memory fragmentation, and intrusive re-experiencing, all hallmark symptoms of PTSD (Halligan et al., 2003; St. Jacques et al., 2013; Hiskey et al., 2008).

Working memory, executive dysfunction, and inhibitory control

Beyond long-term memory, working memory impairments were consistently observed across laboratory, neuropsychological, and neuroimaging studies (Clark et al., 2003; Morey et al., 2009; Schweizer & Dalgleish, 2011; Swick et al., 2017). Individuals with PTSD showed reduced working memory capacity, impaired updating, and diminished resistance to interference, particularly in emotionally salient or trauma-related contexts (Bomyea et al., 2012; Schweizer & Dalgleish, 2016; Swick et al., 2017).

Deficits in executive control and inhibitory processes appear central to PTSD-related memory dysfunction. Experimental paradigms demonstrate that individuals with PTSD exhibit impaired suppression of unwanted memories and reduced ability to disengage from trauma-related information (Catarino et al., 2015; Sullivan et al., 2019; Streb et al., 2016). These inhibitory-control deficits have been linked to intrusive memory frequency and symptom severity, providing a mechanistic bridge between cognitive dysfunction and clinical phenomenology (Bomyea et al., 2012; Oulton et al., 2016; Meyer et al., 2019).

Studies manipulating working memory load further suggest that taxing executive resources during memory retrieval can modulate memory vividness and emotional intensity, supporting models in which limited cognitive control capacity exacerbates intrusive symptomatology (Van den Hout et al., 2014; Bourne et al., 2010). These findings have direct translational relevance, as they inform cognitive and behavioral interventions aimed at modifying memory reconsolidation processes (Mahabir et al., 2016; Bomyea et al., 2025).

Autobiographical memory, intrusions, and narrative coherence

Disturbances in autobiographical memory represent another prominent cognitive feature of PTSD. Multiple studies indicate that individuals with PTSD demonstrate reduced autobiographical memory specificity, fragmented trauma narratives, and altered temporal organization of personal memories (Sutherland & Bryant, 2008a; Sutherland & Bryant, 2008b; St. Jacques et al., 2013; Hiskey et al., 2008). Overgeneral autobiographical memory has been associated with both PTSD and comorbid depression and is predictive of poorer clinical outcomes (Kleim & Ehlers, 2008; Halligan et al., 2003) (see also transdiagnostic perspectives on emotional awareness, coping, and self-regulation; Sulejmani & Pop-Jordanova, 2025a–c).

Experimental analogue trauma paradigms further demonstrate that deficits in memory control and contextual binding contribute to the development and persistence of intrusive memories (Halligan et al., 2002; Oulton et al., 2016; Segovia et al., 2016; Meyer et al., 2019). Longitudinal studies suggest that trauma memory characteristics evolve over time and may be influenced by stress reactivity, dissociation, and ongoing symptomatology (Murray et al., 2002; Mollica et al., 2007; Nahleen et al., 2019).

Notably, autobiographical memory alterations have also been documented in trauma-exposed individuals without formal PTSD diagnoses, although deficits are generally more pronounced among those meeting diagnostic criteria, supporting a dose–response relationship between symptom severity and memory dysfunction (Contractor et al., 2019; McIntosh et al., 2022).

Neurobiological mechanisms underlying memory dysfunction

Neuroimaging findings across structural, functional, and connectivity studies converge on disrupted medial temporal lobe and prefrontal circuitry as key substrates of memory impairment in PTSD. Reduced hippocampal volume has been one of the most replicated neurobiological findings in PTSD, with evidence from both cross-sectional and longitudinal studies

suggesting that smaller hippocampal volume may represent both a vulnerability factor and a consequence of chronic stress exposure (Stein et al., 1997; Wignall et al., 2004; Van Rooij et al., 2015; Woon et al., 2010).

Functional imaging studies further reveal altered hippocampal activation and connectivity during memory encoding, retrieval, and suppression tasks (Bremner et al., 2003; Brohawn et al., 2010; Lazarov et al., 2017; Sullivan et al., 2019). Disrupted coordination between hippocampal, amygdala, cingulate, and prefrontal regions has been implicated in impaired contextual processing, exaggerated threat responses, and intrusive re-experiencing (Cisler et al., 2014; Hou et al., 2007; Jeong et al., 2019; Im et al., 2017).

Importantly, neurobiological abnormalities are not restricted to trauma recall but are also evident during neutral cognitive tasks, supporting the interpretation that PTSD involves generalized alterations in memory-related neural systems (Clark et al., 2003; Geuze et al., 2007; Morey et al., 2009).

Trauma exposure versus PTSD-specific cognitive effects

By focusing on trauma-exposed control groups, the present meta-analysis addresses a critical methodological limitation of prior research directly. Although trauma exposure alone has been associated with subtle cognitive alterations, including working memory inefficiencies and stress-related memory modulation (Blanchette & Caparos, 2016; Grégoire et al., 2020; Henckens et al., 2009), the magnitude and consistency of impairments were greater among individuals with PTSD.

Several studies included in this review demonstrate that trauma-exposed individuals without PTSD often exhibit preserved or only mildly reduced cognitive performance relative to PTSD groups, underscoring the importance of diagnostic specificity (LaGarde et al., 2010; Aase et al., 2017; Bernstein et al., 2020). Comorbid factors such as depression, sleep disturbance, head injury, and medication use further modulate cognitive outcomes and contribute to between-study heterogeneity (Larson et al., 2013; Lipinska et al., 2014; Lipinska & Thomas, 2019; Lawn et al., 2022).

Longitudinal outcomes, aging, and treatment effects

Longitudinal evidence suggests that PTSD-related cognitive impairments may persist over time and, in some cases, worsen with aging. Studies of older trauma survivors and large epidemiological cohorts indicate associations between chronic PTSD symptoms, accelerated cognitive decline, and increased dementia risk (Yehuda et al., 2006; Sumner et al., 2017; Nilaweera et al., 2020). Conversely, treatment studies suggest partial reversibility of cognitive dysfunction following effective trauma-focused psychotherapy, particularly in verbal memory and executive domains (Nijdam et al., 2018).

Emerging intervention studies targeting cognitive processes directly, including pharmacological modulation of memory reconsolidation and computerized working memory training, offer promising avenues for adjunctive treatment approaches (Mahabir et al., 2016; Bomyea et al., 2025). However, further research is needed to determine whether cognitive improvements translate into sustained symptom reduction and functional recovery.

Strengths, limitations, and future directions

The key strengths of this study include the exclusive use of trauma-exposed control groups, restriction to objective neuropsychological outcomes, and application of multilevel meta-analytic models that appropriately account for within-study dependence. These methodological features enhance confidence that observed effects reflect PTSD-specific cognitive impairment rather than trauma exposure alone.

The limitations include residual heterogeneity related to study design, trauma type, comorbidity profiles, and outcome selection. Additionally, although publication bias analyses suggested possible small-study effects, adjusted estimates remained statistically significant, supporting the robustness of findings.

Future research should prioritize longitudinal designs, standardized cognitive batteries, and multimodal neuroimaging approaches to further elucidate mechanisms underlying memory dysfunction in PTSD. Integrating developmental, lifespan, and treatment-response

perspectives will be critical for advancing precision interventions.

Taken together, the collective evidence synthesized in this review which include experimental analogue trauma studies, clinical neuropsychological investigations, neuroimaging research, longitudinal cohort studies, and intervention trials supports the conclusion that memory dysfunction in PTSD is multidimensional, mechanistically complex, and clinically meaningful. Across methodologies and populations, converging findings indicate disruptions in episodic memory, working memory, autobiographical memory organization, inhibitory control, and memory–emotion interactions, influenced by trauma exposure, symptom chronicity, neurobiological vulnerability, sleep and stress physiology, and treatment effects (Bernstein et al., 2020; Bomyea et al., 2012, 2025; Caldas et al., 2022; Contractor et al., 2019; Driessen et al., 2004; Gould et al., 2012; Halligan et al., 2003; Im et al., 2017; Kribakaran et al., 2025; Li et al., 2020; McGuire et al., 2021; Metz et al., 2019; Mollica et al., 2007; Nilaweera et al., 2020; Scott et al., 2016; Shin et al., 2015; Streb et al., 2016; Sullivan et al., 2019; Van den Hout et al., 2014; Yehuda et al., 2006; Zidda et al., 2025).

CONCLUSION

This systematic review and multilevel meta-analysis provides converging evidence that post-traumatic stress disorder is associated with objective and clinically meaningful memory impairment that cannot be explained by trauma exposure alone. Across a large and methodologically diverse body of literature, trauma-exposed adults with PTSD consistently demonstrated poorer performance in verbal episodic memory, working memory, and related cognitive domains compared with trauma-exposed adults without PTSD.

By restricting comparisons to trauma-exposed control groups and applying multilevel meta-analytic models, the present study addresses key methodological limitations of prior research and clarifies the specificity of cognitive dysfunction to PTSD. The findings indicate that memory impairment in PTSD is not confined to trauma-related material but extends to neutral and task-relevant information, with implications for daily functioning, symptom maintenance, and treatment engagement.

Neurobiological, experimental, and longitudinal evidence reviewed alongside the meta-analytic findings suggests that disruptions in hippocampal prefrontal circuitry, impaired inhibitory control, and altered memory consolidation processes contribute to the observed cognitive deficits. Importantly, evidence of partial cognitive improvement following effective trauma-focused treatment highlights the plasticity of memory systems and the potential utility of cognitive-targeted interventions.

Future research should prioritize longitudinal designs, standardized neuropsychological batteries, and multimodal approaches integrating behavioral, neuroimaging, and sleep-related measures to further elucidate mechanisms underlying memory dysfunction in PTSD. Clinically, the present findings support the integration of cognitive assessment and remediation strategies into comprehensive PTSD care. Taken together, this review reinforces the view of PTSD as a disorder of memory as much as of fear, with important implications for theory, research, and intervention.

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Резиме

ОБЈЕКТИВНО ОШТЕТУВАЊЕ НА ПАМЕТЕЊЕТО КАЈ ПОСТТРАУМАТСКОТО СТРЕСНО НАРУШУВАЊЕ: СИСТЕМАТСКИ ПРЕГЛЕД И ПОВЕЌЕСЛОЈНА МЕТААНАЛИЗА НА ВОЗРАСНАТА ПОПУЛАЦИЈА ИЗЛОЖЕНА НА ТРАУМА

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Вовед: Посттрауматското стресно растројство (ПТСН) традиционално се концептуализира како нарушување на учењето на страв и емоционалната дисрегулација; меѓутоа, сè поголем број докази укажуваат дека објективната когнитивна дисфункција, особено нарушувањето на меморијата, претставува централна и трајна карактеристика на растројството. Претходните квантитативни синтези се ограничени поради хетерогените контролни групи, групирање на различни когнитивни исходи и недоволно соодветно справување со статистичката зависност меѓу исходите.

Цел: Оваа систематска ревија и метаанализа имаше цел да утврди дали возрасни лица изложени на траума со дијагностицирано ПТСН покажуваат објективни нарушувања во мемориското функционирање во споредба со возрасни лица изложени на траума без ПТСН.

Методи: Спроведено е сеопфатно пребарување на базите PubMed/MEDLINE, Embase, PsycINFO и Web of Science до 17 декември 2025 година. Вклучени беа студии со возрасни испитаници (≥18 години) изложени на траума, со ПТСН, дијагностицирано според критериумите DSM или ICD, и со споредбена група лица изложени на траума без ПТСН. Беа екстрахирани објективни невропсихолошки мерки на вербална епизодна меморија и работна меморија. Ризикот од пристрасност беше оценет со скалата Newcastle–Ottawa. Стандардизираните средни разлики (Hedges' g) беа сумирани со повеќеслојни метааналитички модели за да се земе предвид зависноста на исходите во рамките на студиите.

Резултати: Деведесет студии ги исполнија критериумите за систематската ревија, од кои 88 придонесоа со квантитативни податоци за метаанализата. Во споредба со контролните групи изложени на траума без ПТСН, лицата со ПТСН покажаа значително полошо мемориско функционирање. Нарушувањата беа најконзистентни за вербалната епизодна меморија и работната меморија, при што ефектите беа забележани низ различни типови траума и инструменти за процена. Присутна беше значителна хетерогеност, но таа не го поништи општиот образец на когнитивно оштетување специфично за ПТСН. Анализите на сензитивност ја потврдија робусноста на наодите. Иако асиметријата на дијаграмите funnel сугерираше можен публицистичка пристрасност, коригираните процени со trim-and-fill методот останаа статистички значајни.

Заклучоци: Возрасните лица изложени на траума со ПТСН покажуваат сигурни и клинички значајни нарушувања во објективното мемориско функционирање, надвор од ефектите на самата трауматска изложеност. Овие наоди ја поддржуваат концептуализацијата на ПТСН како растројство што вклучува маладаптивна обработка на меморијата и ја нагласуваат потребата од вклучување на когнитивна процена и интервенции во истражувањето и во клиничката грижа за ПТСН.

Клучни зборови: посттрауматско стресно растројство, ПТСН, когнитивна дисфункција, нарушување на меморијата, повеќеслојна метаанализа, невропсихолошка процена, систематска ревија, контроли изложени на траума, вербална епизодна меморија, работна меморија