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To cite this article: Snaefridur Gudmundsdottir Aspelund, Hjordis Lilja Lorange, Thorhildur Halldorsdottir, Birna Baldursdottir, Heiddis Valdimarsdottir, Unnur Valdimarsdottir & Harpa Lind Hjördísar Jónsdóttir (2025) Assessing neurocognitive outcomes in PTSD: a multilevel meta-analytical approach, European Journal of Psychotraumatology, 16:1, 2469978, DOI: [10.1080/20008066.2025.2469978](https://doi.org/10.1080/20008066.2025.2469978)

To link to this article: <https://doi.org/10.1080/20008066.2025.2469978>



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REVIEW ARTICLE



Assessing neurocognitive outcomes in PTSD: a multilevel meta-analytical approach

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ABSTRACT

Background: Evidence supporting the association between posttraumatic stress disorder (PTSD) and cognitive impairment is accumulating. However, less is known about which factors influence this association.

Objective: The aims of this meta-analysis were to (1) elucidate the association between PTSD and a broad spectrum of cognitive impairment, including the risk of developing neurocognitive disorder (NCD) later in life, using a multilevel meta-analytical approach, and (2) identify potential moderating factors of this association by examining the effects of age (20–39, 40–59, 60+), study design (cross-sectional or longitudinal), study population (war-exposed populations/veterans or the general population), neurocognitive outcome assessed (i.e. a diagnosis of NCD or type of cognitive domain as classified according to A Compendium of Neuropsychological tests), gender ($\geq 50\%$ women or $< 50\%$ women), study quality (high vs low), type of PTSD measure (self-report or clinical diagnosis), as well as the presence of comorbidities such as traumatic brain injury (TBI), depression, and substance use (all coded as either present or absent).

Method: Peer-reviewed studies on this topic were extracted from PubMed and Web of Science with predetermined keywords and criteria. In total, 53 articles met the criteria. Hedge's g effect sizes were calculated for each study and a three-level random effect meta-analysis conducted.

Results: After accounting for publication bias, the results suggested a significant association between PTSD and cognitive impairment, $g = 0.13$ (95% CI: 0.10–0.17), indicating a small effect. This association was consistent across all examined moderators, including various neurocognitive outcomes, age, gender, study design, study population, study quality, type of PTSD measure, and comorbidities such as depression, substance use, and TBI.

Conclusions: These findings collectively suggest that PTSD is associated with both cognitive impairment and NCD. This emphasizes the need for early intervention (including prevention strategies) of PTSD, alongside monitoring cognitive function in affected individuals.

International Prospective Register of Systematic Reviews (PROSPERO) registration number: CRD42021219189, date of registration: 02.01.2021.

Evaluación de los resultados neurocognitivos en TEPT: una aproximación meta-analítica multinivel

Antecedentes: Cada vez hay más evidencia que apoya la asociación entre el trastorno de estrés posttraumático (TEPT) y el deterioro cognitivo. Sin embargo, se conoce poco sobre qué factores que influyen en esta asociación.

Objetivo: Los objetivos de este metaanálisis fueron: (1) dilucidar la asociación entre el TEPT y un amplio espectro de deterioro cognitivo, incluido el riesgo de desarrollar un trastorno neurocognitivo (NCD por sus siglas en inglés) más adelante en la vida, utilizando una aproximación meta-analítica multinivel, y (2) identificar posibles factores moderadores de esta asociación examinando los efectos de la edad (20–39, 40–59, 60+), el diseño del estudio (transversal o longitudinal), la población del estudio (poblaciones expuestas a la guerra/veteranos o población general), consecuencias neurocognitivas evaluadas (ej., un diagnóstico de NCD o tipo de dominio cognitivo según la clasificación de Un Compendio de test Neuropsicológicos), género ($\geq 50\%$ mujeres o $< 50\%$ mujeres), calidad del estudio (alta

ARTICLE HISTORY

Received 24 October 2024

Revised 14 January 2025

Accepted 10 February 2025

KEYWORDS

Posttraumatic stress disorder; cognitive impairment; neurocognitive disorder; dementia; Alzheimer's disease; neuropsychological tests

PALABRAS CLAVE

Trastorno de estrés posttraumático; deterioro cognitivo; trastorno neurocognitivo; demencia; enfermedad de Alzheimer; pruebas neuropsicológicas

HIGHLIGHTS

- The current meta-analysis was the first to comprehensively investigate the association between posttraumatic disorder (PTSD) and a broad spectrum of cognitive impairment, including the risk of developing neurocognitive disorder (NCD), using a less biased multilevel meta-analytical approach.
- The results showed a significant association between PTSD and cognitive impairment, across all neurocognitive outcomes, age groups, genders, study designs, study populations, study qualities, types of PTSD measures, and the presence of comorbidities such as depression, traumatic brain injury or substance use, highlighting the broad effect of PTSD on cognitive

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Supplemental data for this article can be accessed online at <https://doi.org/10.1080/20008066.2025.2469978>.

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versus baja), tipo de medida del TEPT (auto informada o diagnóstico clínico), así como también la presencia de comorbilidades tales como lesión cerebral traumática (TBI por sus siglas en inglés), depresión y consumo de sustancias (todas codificadas como presente o ausente).

Método: Estudios de revisión por pares sobre este tópico se extrajeron de PubMed y Web of Science con palabras clave y criterios predeterminados. En total, 53 artículos reunieron los criterios. Se calcularon los tamaños del efecto g de Hedge para cada estudio y se condujo un metaanálisis de efectos aleatorios de tres niveles.

Resultados: Después de tener en cuenta el sesgo de publicación, los resultados sugirieron una asociación significativa entre TEPT y deterioro cognitivo, $g = 0.13$ (IC 95%: 0.10–0.17), indicando un efecto pequeño. Esta asociación fue consistente en todos los moderadores, incluidos varios resultados neurocognitivos, edad, género, diseño del estudio, población de estudio, calidad del estudio, tipo de medida para el TEPT y comorbilidades tales como depresión, consumo de sustancias y TBI.

Conclusiones: Estos hallazgos sugieren en conjunto que el TEPT se asocia tanto con deterioro cognitivo como con NCD. Esto enfatiza la necesidad de intervenciones tempranas (incluidas las estrategias de prevención) del TEPT, en conjunto con el seguimiento de la función cognitiva en las personas afectadas.

function.

- These findings emphasize the need for early intervention (including prevention strategies) of PTSD, alongside monitoring cognitive function in affected individuals.

Posttraumatic stress disorder (PTSD) is a mental disorder that occurs in a subset of individuals exposed to traumatic events where the individual's life or well-being or someone else's is threatened (American Psychiatric Association, 2013). PTSD is characterized by a severe reaction to trauma exposure, which impairs daily functioning (Iribarren et al., 2005). The diagnostic criteria for PTSD include a constellation of intrusion symptoms, persistent avoidance, marked changes in arousal and reactivity, and negative alterations in mood and cognition. The negative alterations in cognitive function, inherent in the diagnostic criteria, include memory and concentration problems (American Psychiatric Association, 2013). Previous meta-analyses have shown that individuals with PTSD have a broad cognitive impairment that extends beyond these diagnostic criteria, such as impaired attention, processing speed, working memory and executive functioning (Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015). These findings are concerning given that cognitive impairment may reduce the treatment response in psychosocial PTSD treatment since many treatments (e.g. cognitive behavioral therapy) require learning and acquisition of new skills (Falconer et al., 2013; Samuelson, 2011; Wild & Gur, 2008).

PTSD symptoms are thought to arise from stress-induced changes in the brain (Bremner, 2006). Neuroanatomical correlates of PTSD include the limbic system (i.e. the hippocampus, the amygdala and the cingulate cortex) and the prefrontal cortex (Liberzon & Sripada, 2007). In response to threats or emotionally arousing stimuli, PTSD is characterized by hyperactivation of the amygdala, i.e. 'the brain's threat alarm' can become overactivated. Concurrently, there is a hypoactivation of the ventromedial prefrontal cortex, impairing its ability to properly inhibit the amygdala, i.e. 'malfunctioning of the threat alarm's brake' (Liberzon & Sripada, 2007; Patel et al., 2012). This can lead to prolonged elevated levels of the stress hormone cortisol, which could potentially damage brain regions (e.g. hippocampus and prefrontal

cortex) critical for cognitive function (i.e. memory, processing speed and executive function) (Arnsten, 2009; McEwen et al., 2016). Supporting this notion, hippocampal atrophy (i.e. shrinkage of the hippocampus, a brain area essential for memory) has been observed among individuals with PTSD (Chao et al., 2014; Sapolsky, 2000).

Recurrent stress, such as that experienced in PTSD, has been found to increase the risk of developing neurocognitive disorders (NCD) later in life. In line with this, PTSD has been identified as a risk factor for developing NCD (Greenberg et al., 2014; Günak et al., 2020). NCD is defined as cognitive impairment with substantial impairment in one or more cognitive domains which disrupt daily life (American Psychiatric Association, 2013) and includes conditions formerly referred to as dementia in the DSM 4th edition text revision (DSM-IVTR) (American Psychiatric Association, 2013; Escher et al., 2019; Nilaweera et al., 2020; American Psychiatric Association, 2000). Due to longer life expectancy and increased exposure to cardiovascular risk factors (e.g. diabetes, midlife hypertension and obesity, physical inactivity, and smoking), NCD rates are expected to triple by 2050, representing one of the greatest public health challenges of the century (Livingston et al., 2017; Prince et al., 2013). Considering that risk factors for NCD can be altered throughout life, and that approximately a third of NCD cases could have been prevented or delayed (Livingston et al., 2017; Salthouse, 2009), it is vital to investigate modifiable factors in the association between PTSD and cognitive impairment. This could potentially help reducing the impact of NCD on individuals and communities.

The current study aims to elucidate the relationship between PTSD and cognitive impairment. Previous meta-analyses explored the link between PTSD and cognitive impairment either without including an NCD diagnosis (Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015) or focused solely on individuals with an established NCD diagnosis (Günak et al., 2020). Furthermore, previous research (Günak

et al., 2020) has not examined the role of important moderators, such as depression and substance use. Expanding on the previous work, this study provides a more comprehensive understanding of the long-term effects of PTSD on cognitive function, including pre-clinical (cognitive impairment) to clinical (NCD) stages, and examines a broader range of clinically important moderators – namely age, study design, study population, neurocognitive outcomes, type of PTSD measurement, gender, study quality, and comorbidities such as traumatic brain injury (TBI), substance use and depression – that may influence the relationship between PTSD and cognitive impairment. Furthermore, the current study is the first to employ a multilevel design with maximum likelihood estimation to examine the association between PTSD and cognitive impairment, which reduces bias more effectively compared to traditional meta-analytic approaches (Cheung, 2019). Unlike conventional approaches that might select or average effect sizes from each study, our approach accounts for heterogeneity and complex relationships among multiple effect sizes within studies (Cheung, 2019).

1. Materials and methods

1.1. Protocol and registration

The protocol for the present study was registered with the International Prospective Register of Systematic Reviews (PROSPERO, registration number CRD42021219189). The Preferred Reporting Items for Systemic Reviews and Meta-Analyses (PRISMA) Statement (Moher et al., 2009) and the Cochrane Handbook for Systemic Reviews of Interventions reporting items for meta-analyses (Higgins & Green, 2011) were used to design, execute and report the findings.

1.2. Study eligibility

Inclusion criteria were that the studies (1) examined the association between a PTSD diagnosis and cognitive function in human adults aged 18 and older; (2) used validated and/or well-described methods to determine clinically impairing levels of PTSD (e.g. diagnosis or clinical cutoff on well-established measure); (3) employed neuropsychological assessment to measure at least two different cognitive domains; (4) used a clinical diagnosis of NCD (e.g. from medical records, clinical interviews and neuropsychological assessment); (5) included a comparison group consisting of individuals without a PTSD diagnosis or clinical levels of PTSD symptoms; (6) were published in a peer-reviewed journal in English; and (7) provided sufficient data to calculate effect sizes.

Both prospective and retrospective studies were included. When two manuscripts presented findings

from overlapping datasets, the one of most relevance to our research question or the one with the greater number of subjects was included. For studies in which specialized subgroups were examined (e.g. individuals diagnosed with HIV (Rubin et al., 2016) or schizophrenia (Fan et al., 2008)), analyses comparing groups most accurately addressing the research question (i.e. comparison of participants with and without PTSD in terms of cognitive impairment) were included in the meta-analysis. Case studies and studies that only examined self-reported cognitive function (without neuropsychological assessment) or trauma (i.e. studies focusing on PTSD symptoms without a clinical cutoff) were excluded.

1.3. Information sources and search strategy

In a comprehensive search initially conducted in December 2019 and updated in April 2024, all research articles on the association between PTSD and cognitive impairment in English conducted on humans indexed in PubMed/Medline and Web of Science meeting the aforementioned inclusion criteria were identified. No publication date limitations were set. The search was performed by investigators with research experience in the field of PTSD and cognitive impairment (S.G.A., H.L.L.). Titles, abstracts and articles were reviewed to identify relevant manuscripts. The search terms included combinations of: PTSD, posttraumatic stress disorder, cognitive function, cognitive impairment, cognitive decline, cognitive dysfunction, neurocognitive disorder, Lewy Body dementia, vascular dementia, frontotemporal dementia and Alzheimer's disease. The detailed search terms and filters are provided in the Supplementary material S1. Reference lists of the included studies and published reviews of the association between PTSD and cognitive impairment were also searched for relevant studies to increase the comprehensiveness of the search.

1.4. Data extraction

Data for this meta-analysis were extracted from each study independently (S.G.A., H.L.L.) according to protocol. Data extraction variables included bibliographic information, study design, demographic information on participants (i.e. age, gender, ethnicity, education, study population), PTSD status, cognitive impairment, primary findings, statistical information needed to calculate effect sizes, and covariates in the analyses. Any conflicts regarding data extraction were reviewed and resolved by consensus with two additional reviewers (T.H., H.L.H.J.). Data from fully adjusted models were used to calculate effect sizes.

The Cochrane Handbook for Systemic Reviews of Interventions reporting items for meta-analyses (Higgins & Green, 2011) and Agency for Healthcare

Research and Quality (Owens et al., 2010) guidelines were used to assess study quality. The Newcastle-Ottawa Scale (NOS) for case-control, cohort and cross-sectional design was coded (by S.G.A., H.L.L., H.L.H.J.) for all studies to assess the risk of bias within studies (Wells et al., 2012). Adequate reproducibility was found when replicating the NOS assessments (S.G.A., H.L.H.J.), with 76.7% agreement among the raters.

Since either high or low scores can be indicative of cognitive impairment, depending on the neuropsychological test, all analyses were adjusted such that positive effect sizes consistently indicate greater cognitive impairment for the PTSD group compared to controls.

1.5. Moderators

To gain a deeper understanding of how various factors may influence the relationship between PTSD and cognitive impairment, moderator analyses were conducted. In these analyses, moderator variables were treated as categorical.

1.5.1. Age

Given the increased risk for cognitive impairment with age (Morovic et al., 2019), we classified the studies into distinct chronological age groups based on the mean age of their samples: early adulthood (20–39-years-old), middle adulthood (40–59-years-old), and old age (60 years and older) (Lachman, 2001).

1.5.2. Gender

Since women may be likelier to develop both PTSD and NCD (Christiansen & Berke, 2020; Günak et al., 2020), gender was included as a moderator and coded based on the proportion of women in each study, $\geq 50\%$ or $< 50\%$ women. One reason for why women might be likelier to develop PTSD is that they are likelier than men to experience sexual assault (Tolin & Foa, 2006), which is strongly associated with PTSD (Resnick et al., 2007).

1.5.3. Study design

To capture both short-term and long-term effects of PTSD on cognitive function, including the development of NCD, the included studies were coded based on their study design as either longitudinal or cross-sectional. Longitudinal studies can provide insights into changes in cognitive function over time, i.e. whether cognitive function stays stable, worsens or improves over time (see Table 1 for the follow-up duration of each longitudinal study). Meanwhile,

cross-sectional studies offer a snapshot of cognitive function at a single point in time. Examining study design as a moderator helps determine if the duration and timing of data collection influences the strength or direction of the association between PTSD and cognitive impairment.

1.5.4. Study population

The study population was categorized into war-exposed individuals/veterans and the general population. Most research in this field focuses on veterans, who have higher prevalence rates of PTSD compared to many other subgroups. Therefore, it is important to determine whether the effects of PTSD on cognitive function are comparable in the general public (which includes different types of trauma exposures and clinical profiles), or if they are specific to characteristics associated with veterans (Scott et al., 2015).

1.5.5. Traumatic brain injury

TBI (also called concussion), including head injuries, is common among veterans and can also be a consequence of traffic-related accidents, falls, sports injuries, etc. Since TBI is a risk factor for developing both cognitive impairment and NCD (Barman et al., 2016; Shively et al., 2012), it was included as a moderator in this meta-analysis (coded as present or absent).

1.5.6. Depression

Depression (coded as present or absent) is a common comorbidity of PTSD and given its association with cognitive impairment and NCD (Cohn-Schwartz et al., 2024; Kuring et al., 2020), it was included as a moderator.

1.5.7. Substance use

Since substance use (coded as present or absent) is common among individuals with PTSD, as well as individually associated with cognitive impairment (Hua et al., 2022; Kutash et al., 2023), it was included as a moderator.

1.5.8. Study quality

To assess the impact of study quality on the association between PTSD and cognitive function, we conducted a moderator analysis, utilizing the NOS to rate the quality of the included studies. Initially, only studies with NOS scores of 7 or higher were considered high quality. Due to the limited number (11%) of studies meeting this criterion, we adjusted the threshold to include studies with scores of 6 or higher, thereby encompassing 28% of the total studies.

Table 1. Overview of studies included in the meta-analysis.

Author, year	Participant recruitment	Study design	Sample size (PTSD)	Age in years, M (SD)	Male %	Continental region	Education (M/SD) in years	Study population	PTSD measure	Neurocognitive outcome	Follow-up duration (years)	Covariates in analyses	Primary finding	NOS
Barrett et al., 1996	Centers for Disease Control and Vietnam Experience Study	CS	2,071 (236)	≥30	100	N-America	NR	Military	NIMH-DIS	CVLT, ROCFT, WCST, Block Design	NA	Age, race and military-related covariates. For models examining immediate and delayed recall, scores on ROCFT copy were added as a covariate	PTSD was not associated with cognitive impairment	6/9
Bergman et al., 2023	Trends in Scottish Veterans' Health study and National Health Service (NHS) Scotland	Long	330,794 (NR)	NR	90.3	Europe	NR	Military or various	Medical chart: ICD-9 or ICD-10	Dementia – ICD-9 or ICD-10	36	Birth year, SES, Length of service	Increased risk of dementia was found among individuals with PTSD, regardless of veterans status	4/9
Bethausen et al., 2018	NR	CS	868 (54)	PTSD: 26.8 (5.9), HC: 26.7 (5.7)	100	N-America	Majority HS graduates	Military	PCL	ANAM-4	NA	Military rank, number of prior lifetime TBIs, and number of noncephalic pain sites	PTSD symptoms were associated with worse cognitive performance compared to HC	5/9
Bhattarai et al., 2019	US Department of VA	Long	4,800	64.6 (2.6)	50	N-America	NR	Military	ICD-9	Dementia, cognitive impairment – ICD-9	1	Age, marital status, ethnicity, gender, depression	History of PTSD increased the odds of developing NCD or cognitive impairment	6/9
Brown et al., 2022	Post-COVID-19 Rehabilitation Unit	CS	297	46.9 (14.1)	29.6	N-America	15.6 (1.3)	Post-Acute Sequelae of SARS-CoV-2 infection	IES-6	MoCA-Blind	NA	History of hospitalization for COVID-19, Sex, Ethnicity	Depressive symptom severity was associated with cognitive impairment severity, mainly in verbal fluency, attention and memory. However, PTSD was not associated with cognitive impairment.	4/9
Brownlow et al., 2014	The Warrior Strong Study	CS	44	22.1 (4.1)	45.3	N-America	Majority completed some college	Various	CAPS	ANAM	NA	Sleep duration, PTSD x Sleep duration	PTSD symptom severity was significantly associated with increased sustained attention omission errors	4/9
Burri et al., 2013	Swiss Child Laborers Database	Long	96	77.6 (6.3)	57	European	NR	CM	SSS-PTSD	SID-DAT	NA	Major depressive disorder	PTSD was associated with cognitive impairment, appearing independent of early childhood adversity.	3/9

(Continued)

Table 1. Continued.

Author, year	Participant recruitment	Study design	Sample size (PTSD)	Age in years, M (SD)	Male %	Continental region	Education (M(SD) in years)	Study population	PTSD measure	Neurocognitive outcome	Follow-up duration (years)	Covariates in analyses	Primary finding	NOS
Cimino et al., 2022	Columbia University/ New York State Psychiatric Institute, James J. Peters VA Medical Centre of the Icahn School of Medicine at Mount Sinai	CS	70 (44)	PTSD: 62.5 (9.1), HC: 66.8 (10.7)	47.7	N-America	PTSD: 15.6 (2.2), HC: 14.4 (3.1)	Military	SCID, PCL, CAPS	MMSE, NIHTB-CFB, TMT-A, DSST, SRT,	NA	NR	There were no differences between the group with PTSD compared to the HC. Additionally, findings did not support an association between PTSD and increased levels of Alzheimer's disease blood biomarkers.	5/9
Clouston et al., 2016	WTC Health Program	Long	813	53.7	93	N-America	< HS: 25%, Some college: 47.5%, University 21%, Graduate school 6.5%	World Trade Center responder trauma	PCL-5	MoCA	16	Head injury	Re-experiencing PTSD symptoms were predictive of cognitive impairment 14 years later	5/9
Cohen et al., 2013	Veterans Affair medical centers	CS	535 (196)	PTSD: 55.9 (10), HC: 54.2 (8.9)	93	N-America	PTSD: 14.5 (5.1), HC: 14.7 (5.1)	Military	CAPS	DSST, TMT-A, TMT-B, COWAT, HVLT	NA	Demographics, substance use, physical activity, sleep quality, health factors and depression	PTSD associated with worse executive function, working memory and learning compared to patients without PTSD	7/9
Duke et al., 2010	Community	CS	94 (21)	PTSD: 26.6 (13.2), HC: 36.3 (13.2)	32.6%	N-America	PTSD: 13.1 (1.0), HC: 13.2 (1.3)	Various	DSM – IV	TMT-A, TMT-B, WCST, COWAT, SCWT, DSF, DSDB, CVLT, WMS-III, BFLT, FTT, PTT, WAIS – III	NA	Age, education	Patients with PTSD performed worse in tests of attention, executive function and visual memory	7/9
Falconer et al., 2008	Brain Resource International Database (BRID)	CS	88 (44)	PTSD: 43.9 (12), HC: 42.2 (11)	50	Oceania	PTSD: 12.79 (3.1), HC: 13.91 (2.7)	NR	CAPS	VIT, SoA, SVM, STW, TE, EM, Working memory/vigilance task, DS	NA	Premorbid intelligence (SPTW)	PTSD was associated with impaired attention, cognitive flexibility, working memory, executive function and premorbid intelligence	5/9
Fan et al., 2008	Urban mental health outpatient center	CS	87 (15)	PTSD: 43.9 (9.6), HC: 43.7 (8.8)	74.7	N-America	12.1 (2.5)	NR	HTQ	DSST, DS, WCST, SCWT, CVLT	NA	NR	PTSD had significantly worse overall impairment, especially in attention, working memory and executive function, compared to HC	3/9
Flatt et al., 2018	Kaiser Permanente Northern California health system	Long	499,844 (1,147)	71.1 (7.9)	45.3	N-America	NR	Various	ICD-9	Dementia – ICD-9	8	Age, gender, ethnicity, TBI, depression, Vascular factors	PTSD increased the risk of developing NCD in both men and women	4/9
Folnegovic-Smalc et al., 1997	Refugee camps	Long	1,076 (538)	≥44	46.6	Europe	NR	Refugees	HTQ	Dementia – DSM-III-R, NINCDS-ADRDA	2.5	NR	PTSD was associated with increased risk of developing NCD (i.e. Alzheimer's Disease) in war-exposed individuals	2/9

Gilbertson et al., 2006	Vietnam Era Twin registry	CS	86 (38)	PTSD: 52.1 (3.2), HC: 51.8 (2.3)	100	NR	PTSD: 14.0 (2.9), HC: 14.6 (2.4)	Military	CAPS	DSF, DSB, CVLT, CVMT, WCST, Benton Facial Recognition Test, VOSP	NA	Amount of combat exposure	Those with PTSD performed worse in general intelligence, verbal memory and attention. No differences in nonverbal memory and visuospatial skills.	5/9
Gratz et al., 2019	Danish Psychiatric Central/National Research Patient Registry	Long	279,188 (2,189)	Mdn.: 51	41	Europe	NR	Various	ICD-10	Dementia – ICD-10	17	Age, Gender, Marital status, Substance misuse, depression, anxiety	Individuals with PTSD were twice as likely to have NCD compared to the HC. An interaction between sex and NCD were found.	5/9
Green et al., 2016	VA Palo Alto Health Care System Institutional Review Boards	CS	204 (55)	PTSD: 61.0 (4.3), HC: 66.1 (7.2)	100	N-America	PTSD: 14.4 (2.5), HC: 16.1 (2.2)	Military	CAPS	RAVLT, TMT, CWIT, DS, WCST, COWAT, SCWT	NA	Age and education	Individuals with PTSD did not perform worse in processing speed, verbal memory and executive function than HC	6/9
Hart et al., 2008	POW outreach program by the Central Arkansas Veteran's Health Care System	CS	25 (14)	PTSD: 80.9 (2.3), HC: 80.0 (1.2)	100	N-America	PTSD: 14.4 (2.0), HC: 15.9 (3.4)	POW	CAPS-2	MMSE, NAART, BNT, COWAT, DSF, DSB, RAVLT, WMS-R, WRMT, JLO, WRMT, SDMT, TMT	NA	Weight loss, time of imprisonment, and time as a POW	Individuals with PTSD performed worse in TMT-B and Symbol Digit compared to HC	5/9
Horner et al., 2013	VA Medical Center	CS	41 (19)	PTSD: 44 (12.9), HC: 51.3 (17.6)	PTSD: 84%, C: 91%	N-America	PTSD: 13.5 (3), HC: 13.1 (2.9)	Military	DSM-IV	DS, TMT-A, TMT-B, WMS-III	NA	NR	Patients with PTSD had significantly worse performance in DS compared to HC	5/9
Hudetz et al., 2010	VA Medical Center	CS	126 (30)	68	100	N-America	PTSD: 13 (2), HC: 12 (2)	Military	Medical chart	RBANS, SCWT, BVMT-R, DSB, COWAT	NA	NR	PTSD history may be associated with post-operative cognitive impairment	3/9
Islamaska et al., 2020	National registry	Long	103,484 (1,786)	Mdn.: PTSD: 53, HC: 51	48	Europe	NR	NR	ICD-10	ICD-8 and ICD-10 – Dementia	1–20	Sex, any other stress disorder, country of origin, marital status, education, morbidities	Severe stress in midlife is a risk factor for NCD. PTSD led to non-significantly increased NCD rates	4/9
Jak et al., 2020	VA	CS	32 (16)	34.8	90.7	N-America	15.4	Military	PCL-5	CVLT-II, BVMT-R, D-KEFS, WAIS-IV, WRAT, MSVT, CVLT-II	NA	Demographics, brain scanner update	No significant group differences in cognitive composite scores. PTSD was associated with altered myelin.	5/9
Koopowitz et al., 2021	Drakenstein Child Health Study (DCHS)	CS	175 (36)	28.9 (6.6)	0	Africa	10.8 (2.8)	Various	MINI, DSM-IV	RBANS	NA	Age and education	Participants with PTSD had more intrusion errors compared to HC. No group differences were found in attention and executive function	4/9

(Continued)

Table 1. Continued.

Author, year	Participant recruitment	Study design	Sample size (PTSD)	Age in years, M (SD)	Male %	Continental region	Education (M(SD) in years)	Study population	PTSD measure	Neurocognitive outcome	Follow-up duration (years)	Covariates in analyses	Primary finding	NOS
Lawrence et al., 2023	Project SERVE, Central Texas Veterans Health Care System	CS	124 (51)	PTSD: 36.4 (9.0), HC: 38.8 (11.5)	85.5%	N-America	PTSD: 13.2 (1.6), HC: 14.4 (2.5)	WTC, Military	CAPS	CVLT-II, DSST, DSF, DSB, TMT-A, TMT-B, SCWT	NA	ADHD, probable TBI	PTSD symptom severity was associated with less health promoting behavior. Verbal learning and memory partially accounted for this association.	4/9
Logue et al., 2023	Million Veteran Program	CS	186552 (12555)	77.9 (6.7)	97.5	N-America	3.6 (1.6) out of a 7-point scale	Military	Medical chart	Alzheimer's disease and related dementias (ADRD) – ICD-9 or ICD-10	NA	Age, APOE e4 status	There was a strong association between ADRD and PTSD which was not due to confounding with alcohol use, smoking, or education.	4/9
Marcolini et al., 2023	Military and VA Compensation, Pension records, VA Health records, ADNI, the Department of Defense	Long	149 (78)	PTSD: 68.1 (3.3), HC: 71.4 (5.8)	100	N-America	PTSD: 14.5 (2.4), HC: 16.1 (2.1)	Military	SCID-1, CAPS	BNT, TMT-A, TMT-B, RAVLT	1	Age, education	PTSD affected cognitive function. TBI did not independently affect cognition. Veterans with PTSD had more progression of white matter abnormalities of the uncinate fasciculus compared to controls.	7/9
Mawanda et al., 2017	Veterans Health Administration National databases	Long	417,172 (22,674)	67.7 (8.1)	98	N-America	NR	Military	ICD-9	Dementia – ICD-9	9	Age, gender, marital status, substance misuse, depression, anxiety	Participants with PTSD had an increased risk of developing NCD that varied with psychotropics use.	4/9
Meziab et al., 2014	Veterans Health Administration National Patient Care Database	Long	182,879 (484)	71.6	100	NR	% with college degree in zip code: approx. 32.9%	Military	ICD-9	Dementia – ICD-9-CM	9	Age, gender, SES, vascular factors, chronic pulmonary, depression, substance use	After adjustments, PTSD and POW statuses were associated with an increased risk of developing NCD. Veterans who had been POW and had a PTSD status had the highest risk.	4/9
Moretta et al., 2022	Rehabilitation Unit of Telese Terme	CS	63 (28)	59.8 (10.8)	74.6	Europe	11.9 (3.5)	Acute illness due to COVID-19	IES-R	Corsi Span, Corsi supraspan, TMT, RAVLT, FAS	NA	Age, education, sex	Reduced cognitive efficiency was associated with longer and more severe illness, more clinically relevant PTSD symptoms, more frequent cognitive complaints, higher anxiety, lower quality of life and more.	5/9

Nakayama et al., 2020	NCNP, Women's Medical University Hospital and Nagoya City University Hospital	CS	144 (50)	38.7 (10.5)	0	Asia	Mdn: 3 (some college graduate/partial university)	CM or IPV	PDS	RBANS	NA	Age, education, current smoking status	Participants with PTSD performed worse on measures on immediate memory, language, attention and RBANS (total score)	7/9
Otsuka et al., 2021	NCNP, Women's Medical University and Nagoya City University	CS	130 (57)	PTSD: 39.7(9.3), HC: 35.1(13.8)	0	Asia	Majority completed some college	IPV, Accident, Disaster, Other	PDS	RBANS	NA	NR	Participants with PTSD performed worse on measures of memory, visuospatial ability, language, attention, and overall cognitive function compared to HC	5/9
Peleikis et al., 2013	Thematically Organized Psychosis Study in Norway	CS	292 (21)	NR	55	Europe	PTSD: 12, HC: 12.8	Various	SCID-I, MINI	DSF, DSB, DSST, SCWT, D-KEFS, COWAT, WASI	NA	NR	No association between PTSD and cognitive impairment in patients with schizophrenia	5/9
Van Praag et al., 2022	Collaborative European Neurotrauma Effectiveness Research (CENTER-TBI)	CS	1134 (153)	Mdn for PTSD: 43 and HC: 49	68.4	Europe	PTSD: 44.7%, HC: 58.6% post- high school training or college/university	NR	PCL-5	TMT, AVDEL30, CANTAB	NA	Age, sex, educational level, psychiatric history	Among individuals who had suffered from TBI, probable PTSD was associated with worse performance in task switching and delayed verbal recall	6/9
Qureshi et al., 2010	Veterans Integrated Service Network Data Warehouse	Long	10,481 (3,660)	73.6	99	N-America	NR	Military	ICD-9	Dementia – ICD-9, usage of dementia medication	9	Gender, race, vascular factors, TBI, substance misuse, primary care clinic visits	Veterans with PTSD had greater prevalence and incidence of NCD. Unclear whether due to a common risk factor for both conditions or whether PTSD is a risk factor for NCD.	4/9
Regier et al., 2019	The Boston VAMC and Houston VAMC tumor registries	Long	88	65.9 (9.4)	100	N-America	53.4% college education	Cancer and Military	PCL-5, PC-PTSD	MoCA	1.5	Comorbidity, Combat-related PTSD, Age ≥ 70, cancer treatment factors	Cancer-related PTSD was associated with worse cognitive function in older veterans	4/9
Rosen et al., 2022	Bellevue Hospital WTC Environmental Health Center	CS	480 (347)	Mdn: 56.4	46	N-America	>12 years: 72%, ≤ 12 years: 28%	WTC exposure	PCL-17	MoCA	NA	Age, education, income, WTC exposure and ethnicity	PTSD, trauma and WTC dust toxins adversely impacted cognitive function	5/9
Roughhead et al., 2017	Australian Department of Veterans's Affairs	Long	15,612	Mdn: 57	100	Australia	NR	Military	ICD-10	Dementia – ICD-10, any dementia record, usage of dementia medication	12	Age, SES, chronic health conditions, clinical depression, substance use, and benzodiazepine use	NCD risk was greater in veterans that received antipsychotic medication regardless of PTSD status	3/9
Rubin et al., 2016	WIHS	Long	496 (79)	PTSD: 46.7 (9), HC: 42.9 (10.1)	0	N-America	PTSD: 11.9 (2.9), HC: 12.7 (2.7)	Various	PCL-C	HVLT-R, Letter-Number span, TMT, SCWT, SDMT, VF, Grooved pegboard	NA	SES, substance use, positive Hepatitis C antibody, current depressive symptoms, recent antidepressant use and study location.	Association between PTSD and impaired verbal learning, memory and psychomotor speed in women with HIV	4/9

(Continued)

Table 1. Continued.

Author, year	Participant recruitment	Study design	Sample size (PTSD)	Age in years, M (SD)	Male %	Continental region	Education (M(SD) in years)	Study population	PTSD measure	Neurocognitive outcome	Follow-up duration (years)	Covariates in analyses	Primary finding	NOS
Scheiner et al., 2014	Conte Center for Study of the Neuroscience of Mental Disorders and New York State Psychiatric Institute	CS	267 (25)	PTSD: 35.3 (10.3), HC: 31.4 (10.1)	47	N-America	PTSD: 14.7 (2.4), HC: 15.9 (2.2)	Various	SCID	The Buschke Selective Reminding Test, CPT, A not B reasoning task, WCST	NR	Age, gender and education	PTSD was associated with a verbal learning impairment	5/9
Shandera-Ochsner et al., 2013	Partners Human Research Committee, Boston, MA	CS	81 (40)	PTSD: 32.5 (7), HC: 34 (8.9)	PTSD: 89.5, HC: 90.5	N-America	PTSD: 13.6 (1.3), HC: 14.95 (1.6)	Military	DSM-IV	CULT-II, BVMT-R, D-KEFS, WAIS-IV, Wechsler Test of Adult Reading, CPT-II	NA	Age	PTSD was associated with worse performance on executive tasks, verbal fluency, and verbal memory	6/9
Song et al., 2020	Nationwide health register	Long	61,748 (3,743)	NA	39.4	Europe	NA	NR	ICD-9	Alzheimer's Disease – ICD-9 or ICD-10	5	SES, demographics, medical background	Stress disorders, including PTSD were associated with an increased the risk of developing NCD	4/9
Stein et al., 2002	Domestic violence community service	CS	39 (17)	PTSD: 33.8 (9.8), HC: 34.5 (9.0)	0	N-America	PTSD: 12.6 (1.7), HC: 13.3 (2.3)	IPV	CAPS, SCID, IES-R	WMS, CVLT, VPA, CVMT, ROCF, COWAT, WAIS, DSF, DSB, ACT, DVT, PASAT, TMT-A, TMT-B, SCWT	NA	NA	Participants with PTSD performed worse on TMT-B. PTSD was not uniformly associated with worse cognitive performance	5/9
Stricker et al., 2015	Operation Enduring Freedom and Operation Iraqi Freedom	CS	109 (56)	PTSD: 36.8 (11.5), HC: 37.1 (11.7)	0	N-America	PTSD: 14.7 (1.8), HC: 15.2 (1.9)	Military	CAPS, SCID	CVLT-II, The Processing Speed Index (WAIS), DSB, DSST, WAIS-IV, D-KEFS	NA	Age	Female veterans with PTSD performed worse than HC in verbal learning, processing speed, and executive functioning	5/9
Stricker et al., 2017	VA Boston TBI National Research Center, the Translational Research Center for TBI and Stress Disorders	CS	171 (92)	PTSD: 31.4 (7.8), HC: 32.3 (8.8)	83.6	N-America	PTSD: 14.0 (1.9), HC: 14.5 (2.0)	Military	CAPS	DSF, DSB, ACT, Test of Variables of Attention, DSST, D-KEFS, CVLT-II, BVMT-R, MCI – DSMS	NA	NR	PTSD status was associated with more frequent memory impairments in veterans. No group differences in attention, processing speed or executive function.	5/9
Twamley et al., 2009	NR	CS	75 (55)	PTSD: 36.7 (8.8), HC: 35.0 (8.6)	0	N-America	PTSD: 14.0 (1.9), HC: 14.6 (1.5)	IPV	PCL-C, SCID-I, CAPS	ROCF, D-KEFS, WCST	NA	Age, education, ethnic minority status, premorbid intellectual functioning	More severe PTSD symptoms were associated with slower processing speed. Participants with PTSD scored within average range on all cognitive tests	7/9
Vasterling et al., 2002	VA Medical Canter and Veterans Resource Center	CS	47 (26)	PTSD: 50.2 (3.3), HC: 51.6 (4.9)	100	Asia	PTSD: 12.6 (2.2), HC: 14.9 (2.1)	Military	M-PTSD	CPT, WCST, WAIS – R, RAVLT CVMT	NA	Level of combat exposure	Participants with PTSD showed deficits in sustained attention, working memory, initial learning, and premorbid intelligence, indicating cognitive impairment regardless of general intelligence	5/9

Walczewska et al., 2014	Clinic for Former Victims of Political Persecution	CS	195 (80)	PTSD: 69.3 (5.9), HC: 70.8 (4.9)	50	European	NR	Deportation	DSM-IV-TR	MMSE	NA	Age, education	Participants with PTSD had more cognitive impairment compared to HC. More severe PTSD was associated with significant cognitive deterioration	6/9
Wang et al., 2016	Taiwan National Health Insurance Research Database	Long	8,750 (1,750)	55.4 (9.2)	23.4	Asia	NR	Various	ICD-9-CM	Dementia – CM	2	Gender, depression, substance use, head injury, vascular factors, urbanicity	PTSD and its severity was associated with a greater risk of developing NCDs	3/9
Weiner et al., 2022	Military and VA Compensation, Health records, the Department of Defense, ADNI	Long	289 (81)	PTSD: 68.2 (3.3), HC: 71.4 (5.8)	99.3	N-America	PTSD: 14.5 (2.3), HC: 16.1 (2.1)	Military	CAPS	Alzheimer's Disease Assessment Scale-cognitive, MMSE, Clinical Dementia rating, RAVLT	5.2	Age, education, APOE e4 status	Participants with PTSD had higher prevalence of mild cognitive impairment and worse MMSE scores compared to controls.	7/9
Wisdom et al., 2014	VA hospitals	CS	134	PTSD: 32.6, HC: 30.6	94	N-America	PTSD: 13.0, HC: 13.2	Military	PCL	CULT-II, WAIS-IV, PASAT, TMT-A, TMT-B, SCWT, WCST, WRAT-4, COWAT	NA	Symptom validity	No significant differences between participants with and without PTSD after separating veterans who failed on performance validity testing	6/9
Yaffe et al., 2010	Department of VA National Patient Care Database	Long	181,093 (53,155)	PTSD: 66.2 (9.0), HC: 69.9 (8.2)	96.5	N-America	NR	Military	ICD-9	Dementia- ICD-9-CM	7.2	Age, gender, race, education, vascular factors, cancer, substance use, depression, head injury	Veterans with PTSD were more than twice as likely to develop NCD	6/9
Yaffe et al., 2019	Veterans health Administration Medical Center	Long	109,140 (1,363)	PTSD: 65.1 (6.7), HC: 69.2 (9.8)	0	N-America	NR	Military	ICD-9	Dementia – ICD-9	7	Age, race, education, income, vascular factors, TBI, depression, substance use	Female veterans with military-related risk factors had 50–80% risk of developing NCD	6/9

Note. NA = Not applicable; NR = Not reported; PTSD = Posttraumatic Stress Disorder; Long = Longitudinal; CS = Cross-sectional; VA = Veterans Affairs; NIMH-DIS = National Institute of Mental Health Diagnostic Interview Schedule; HC = Healthy controls; Mdn = Median; NCNP = National Center of Neurology and Psychiatry Tokyo; HS = High school; TBI = traumatic brain injury; SES = Socioeconomic status; IPV = interpersonal violence; CM = childhood maltreatment; POW = Prisoner of War; SCWT = Stroop Color and Word test; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status; MMSE = Mini-Mental State Examination; RAVLT = Rey's Auditory Verbal Learning Test; TMT = Trail Making Test (A and B); HVLRT = Hopkins Verbal Learning Test-revised; SDMT = Symbol Digit Modalities Test; WCST = Wisconsin Card Sorting Test; COWAT = Controlled Oral Word Association Test; CANTAB = The Cambridge Automated Neuropsychological Test Battery; DSF = Digit Span forward; DSB = Digit Span backward; ROCF = Rey-Osterrieth complex figure test; CVMT = Continuous visual memory test; D-KEFS = Delis-Kaplan Executive Function System; MoCA = the Montreal Cognitive Assessment; WRAT = The Wide Range Achievement Test; BVMT-R = The Brief Visual Memory Test Revised; CPT = Continuous Performance task; WMS = Wechsler Memory Scale; WAIS = Wechsler Adult Intelligence Scale; CVLT = The California Verbal Learning Test; DSST = Digit Span Sequencing; PASAT = Paced Auditory Serial Addition Task; PSI = The Processing Speed Index of the Wechsler Adult Intelligence Scale; ANAM = The Automated Neuropsychological Assessment Metrics; DSST = Digit Symbol Substitution Test/Digit-Symbol Coding/Coding; FTT = Finger Tapping Test; PPT = Purdue Pegboard Test; ACT = Auditory Consonant Triangles; DVT = Digit Vigilance Test; BFLT = Biber Figure Learning Test extended; NAART = North American Adult Reading Test; BNT = Boston Naming Test; WRMT = Warrington Recognition Memory Test; JLO = Judgement of Line Orientation; VIT = Verbal Interference Test; SoA = Switching of Attention; SVM = Span of Visual memory; STM = Spot the word; TE = Time Estimation; EM = Executive Maze; WASI = The Wechsler Abbreviated Scale of Intelligence; VPA = Verbal Paired Associates; CAPS = The Clinician-Administered PTSD Scale; PDS = Posttraumatic Diagnostic Scale; ES-R = Impact of Event Scale-Revised; PCL-C = PTSD Checklist-Civilian version; PCL-M = PTSD Checklist-Military version; PCL-S = PTSD Checklist-Stressor Specific version; HTQ = Harvard Trauma Questionnaire; SSS-PTSD = Short Screening Scale for DSM-IV PTSD; SCID = Structured Clinical Interview for the DSM-IV Axis I and Axis II disorders; ICD-9-CM = International Classification of Diseases, Ninth Revision, Clinical Modification; PC-PTSD = Primary Care PTSD Screen; M-PTSD = Mississippi Scale for Combat-Related Post Traumatic Stress Disorder; SID-DAT = Structured Interview for Diagnosis of Dementia of Alzheimer Type; MINI = The Mini-International Neuropsychiatric Interview; NIHTB-CFB = NIH Toolbox Cognitive Function Battery; SRT = The Selective Reminding Test; AVDEL30 = 30-minutes delay Auditory Verbal Learning Test; ADHD = Attention deficit hyperactivity disorder; WTC = The World Trade Center; SERVE = Studies Evaluating Returning Veterans' Experiences; ADNI = Alzheimer's Disease Neuroimaging Initiative; NOS = The Newcastle-Ottawa Scale.

1.5.9. Neurocognitive outcomes

Informed by previous research on how PTSD can affect various cognitive domains and the risk of developing NCD (Günak et al., 2020; Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015), neurocognitive outcomes were included as a moderator in the current study. PTSD may influence these neurocognitive outcomes differently, leading to varying degrees of cognitive impairment.

The neurocognitive outcomes in the present study included a diagnosis of NCD (i.e. either a clinical diagnosis of major NCD or mild cognitive impairment, see the supplementary material for more details), as well as the cognitive domains assessed in the retrieved studies. Neuropsychological tests were classified into cognitive domains based on the classification system of the Compendium of Neuropsychological Tests (Sherman & Spreen, 2006): general cognitive function, which included broad-scale measures of cognitive functioning (e.g. Mini Mental State Examination, Montreal Cognitive Assessment); attention (e.g. Trail Making Test); working memory (e.g. WAIS-Digit Span); processing speed (e.g. Digit Symbol); memory (e.g. Rey Auditory Verbal Learning Test); executive function (e.g. Controlled Oral Word Association Test, Stroop test); visuospatial perception (e.g. Judgement of Line Orientation, Block Design). Cognitive domains assessed by neuropsychological tests, which were underrepresented in the analyses, were added to the general cognitive function domain (i.e. language tests, motor function and time perception). See Supplementary Table S1 for the complete classification of the neuropsychological tests.

1.6. Statistical analysis

Effect sizes were calculated for each reported analysis in the eligible studies. Given that Cohen's d tends to overestimate effect sizes in small samples, Hedge's g correction formula was used to decrease this bias (Borenstein et al., 2009; Hedges, 1981). Data were converted into Hedge's g effect sizes using the *esc* package in R (Lüdtke, 2019). As is customary, effect sizes of 0.2, 0.5 and 0.8 were considered small, medium and large, respectively (Cohen, 1992).

All retrieved studies used multiple neuropsychological tests to assess various cognitive domains. To include these multiple findings within each study, we conducted a three-level meta-analysis using the maximum likelihood method to estimate heterogeneity and account for dependency between the effect sizes. This statistical approach is preferred over traditional meta-analytic procedures, such as averaging effect sizes across studies or selecting one effect size per study, as it is less biased (Cheung, 2019).

In the three-level meta-analytic model, we assumed that individual effect sizes (random effect at level 2) were nested within studies (random effect at level 3). Heterogeneity was calculated using the I^2 statistic, a measure of the variance attributed to differences between the studies (0% = none, 25% = low, 50% = moderate, and 75% = high heterogeneity) (Thorlund et al., 2012). The fixed effects model analyses revealed high inter-study heterogeneity ($I^2 = 86.42\%$). Subsequently, a random effects model was used to account for this heterogeneity. Confidence intervals (CI; 95%) and p -values (significance threshold set at $\alpha = .05$) were calculated. The *metafor* package in R (Viechtbauer, 2010) was used to perform the three-level random effect meta-analysis (the *rma.mv* function).

The Egger's test was utilized to assess publication bias (Egger et al., 1997), and Duval and Tweedie's trim and fill analysis was employed to estimate the adjusted effect size (Duval & Tweedie, 2000). Additionally, Rosenthal's fail-safe N was calculated to estimate the number of missing studies with non-significant results needed to nullify the findings of the current meta-analysis (Rosenthal, 1979), ensuring the robustness of the results. The fail-safe N threshold was set to $5 \times k + 10$, where k represents the number of effect sizes ($k = 377$) included in the current study (Rosenthal, 1991; Shi et al., 2021).

2. Results

2.1. Study selection and characteristics

Figure 1 is a PRISMA flow diagram of the systematic search for applicable studies. A total of 1,525 abstracts were identified after duplicates were removed. Following the review of abstracts, 156 studies were selected for full-text review. Five studies were excluded as the sample overlapped with another study (Hori et al., 2020; Narita-Ohtaki et al., 2018; Prieto et al., 2023; Rubin et al., 2017; Weiner et al., 2017). Overall, 53 original studies met the inclusion criteria (Rubin et al., 2016; Fan et al., 2008; Barrett et al., 1996; Betthausen et al., 2018; Bhattarai et al., 2019; Brownlow et al., 2014; Burri et al., 2013; Clouston et al., 2016; Cohen et al., 2013; Duke et al., 2010; Falconer et al., 2008; Flatt et al., 2018; Folnegovic-Smalc et al., 1997; Gilbertson et al., 2006; Gradus et al., 2019; Green et al., 2016; Hart et al., 2008; Horner et al., 2013; Hudetz et al., 2010; Islamoska et al., 2020; Jak et al., 2020; Koopowitz et al., 2021; Mawanda et al., 2017; Meziab et al., 2014; Nakayama et al., 2020; Otsuka et al., 2021; Peleikis et al., 2013; Qureshi et al., 2010; Regier et al., 2019; Roughead et al., 2017; Scheiner et al., 2014; Shandera-Ochsner et al., 2013; Song et al., 2020; Stein et al., 2002; Stricker et al., 2015; Stricker et al., 2017; Twamley et al., 2009; Vasterling et al., 2002; Walczewska et al., 2014; Wang et al.,

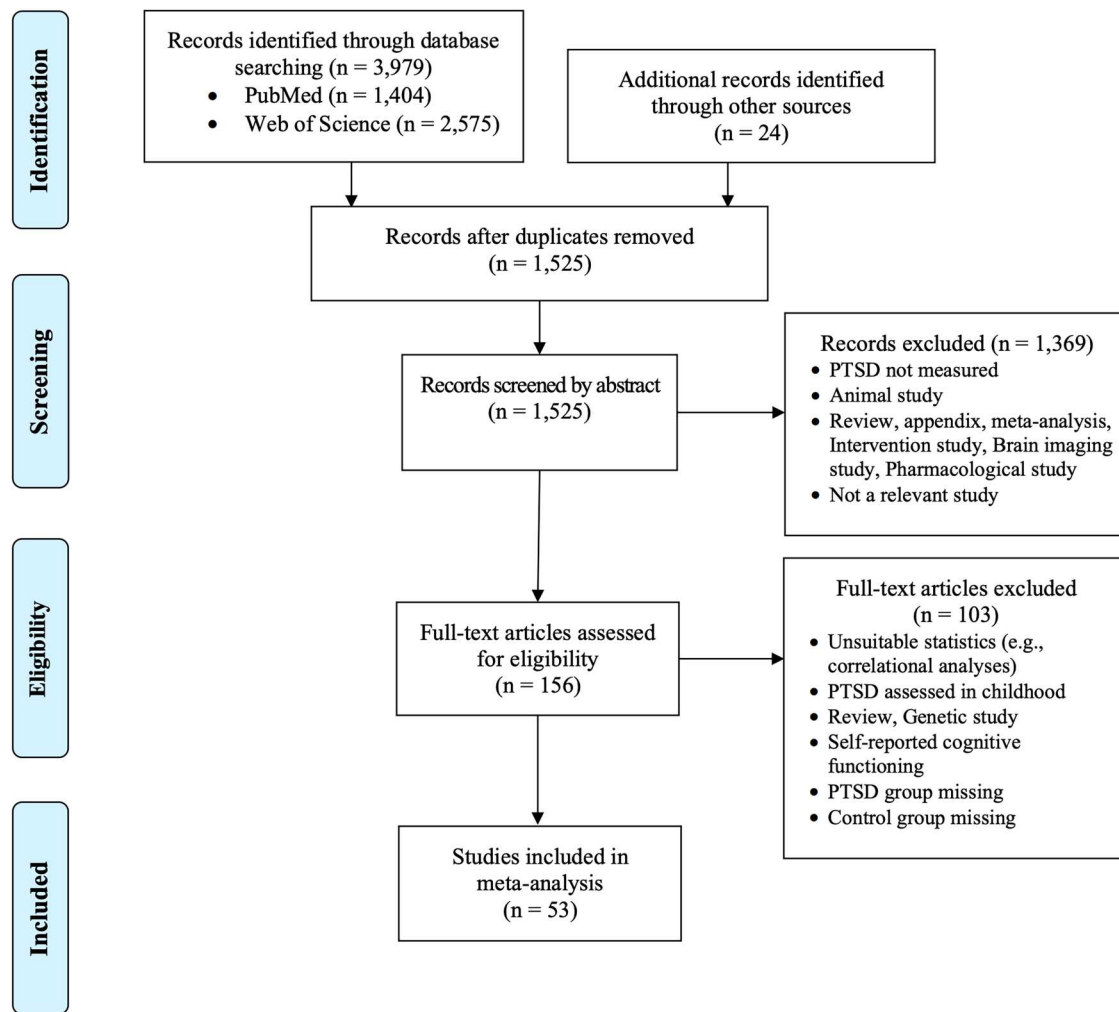


Figure 1. PRISMA flow diagram of the review process and study selection.

Note. PTSD = Posttraumatic Stress Disorder. Correlational studies were excluded based on our inclusion criteria requiring studies to compare groups with and without PTSD.

2016; Wisdom et al., 2014; Yaffe et al., 2010; Yaffe et al., 2019; Logue et al., 2023; Bergman et al., 2023; Rosen et al., 2022; Weiner et al., 2022; Cimino et al., 2022; Van Praag et al., 2022; Brown et al., 2022; Marcolini et al., 2023; Lawrence et al., 2023; Moretta et al., 2022) (see Figure 1 for the PRISMA flow diagram).

Table 1 provides detailed information on the included studies. The majority of the studies were cross-sectional (67%) and conducted on veterans (55%). The studies were relatively equal regarding the age groups: 30% were performed on 20–39-year-old participants, 36% on 40–59-years-old, and 34% aged 60 years and older (three studies did not report the age in their sample). When broken down by cognitive domain, the majority of studies examined a diagnosis of NCD (30%) and general cognitive function (23%).

2.2. Primary meta-analysis of PTSD and cognitive impairment

Fifty-three studies were included in the meta-analyses with a pooled sample size of 5,709,547 participants across all the studies and an overall of 376 analyses

examining the association between PTSD and cognitive impairment. The three-level model provided a significantly better fit than a two-level model, as indicated by lower Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) values, and a significant likelihood ratio test ($\chi^2 = 108.66, p < .0001$). In the three-level model, the pooled effect size across the studies was $g = 0.30, p < .0001, 95\% \text{ CI } (0.23–0.37)$, indicating a small to medium effect (Table 2). The finding suggests greater cognitive impairment among individuals with PTSD compared to controls.

2.3. Moderator analyses

All coefficients for the moderator analyses are displayed in Table 2. None of the moderators (i.e. study design, study population, age group, gender, neuro-cognitive outcome, depression, TBI, type of PTSD assessment, or substance use) significantly moderated the association between PTSD and cognitive impairment. However, significant effects were found within all subgroups, with effect sizes of small to medium magnitudes (see Table 2).

Table 2. Meta-analysis and moderator analyses of the association between PTSD and cognitive impairment.

Analysis	Number of studies (analyses included)	Effect sizes (95% CI)	Heterogeneity Q, I^2
Main analysis	53 (376)	30 (0.23–0.37)	1998.24***, 86.6%
Study design			1491.50***, 86.8%
$F(df1 = 1, df2 = 375) = 0.46, p = .50$			
Cross-sectional [index]	36 (346)	0.28 (0.21–0.36)	
Longitudinal	17 (30)	0.35 (0.27–0.43)	
Neurocognitive outcomes			1258.12***, 87.1%
$F(df1 = 6, df2 = 369) = 0.72, p = .63$			
Diagnosis [index]	16 (26)	0.34 (0.21–0.47)	
Attention & processing speed	6 (63)	0.29 (0.17–0.42)	
Executive function	5 (84)	0.26 (0.13–0.38)	
General cognitive function	12 (42)	0.29 (0.17–0.42)	
Memory	7 (116)	0.31 (0.19–0.44)	
Visuospatial & perceptual ability	2 (9)	0.30 (0.17–0.42)	
Working memory	5 (36)	0.24 (0.11–0.37)	
Study population			1360.23***, 86.9%
$F(df1 = 1, df2 = 374) = 0.00, p = .99$			
General population [index]	24 (186)	0.30 (0.20–0.40)	
War exposure (including veterans)	29 (190)	0.30 (0.06–0.54)	
Age group			1043.19***, 86.6%
$F(df1 = 2, df2 = 343) = 0.31, p = .74$			
20–39 [index]	15 (191)	0.32 (0.20–0.44)	
40–59	18 (83)	0.27 (–0.03 to 0.56)	
60+	17 (72)	0.33 (0.04–0.62)	
Traumatic Brain Injury			1698.88***, 86.7%
$F(df1 = 1, df2 = 374) = 1.17, p = .28$			
Without TBI [index]	45 (349)	0.32 (0.24–0.39)	
With TBI	8 (27)	0.22 (0.02–0.41)	
Depression			1760.61***, 98.1%
$F(df1 = 1, df2 = 374) = 0.06, p = .81$			
Depression absent [index]	42 (352)	0.30 (0.22–0.37)	
Depression present	11 (24)	0.32 (0.07–0.56)	
Proportion of women			1457.63***, 86.9%
$F(df1 = 1, df2 = 374) = 0.01, p = .93$			
<50% women [index]	30 (202)	0.30 (0.21–0.39)	
≥50% women	23 (174)	0.30 (0.14–0.47)	
Type of PTSD measure			1297.28***, 86.8%
$F(df1 = 1, df2 = 374) = 0.19, p = .66$			
Self-report diagnosis [index]	15 (75)	0.27 (0.14–0.41)	
Clinical diagnosis	38 (301)	0.31 (0.02–0.59)	
Substance use			1712.29***, 86.9%
$F(df1 = 1, df2 = 374) = 0.03, p = .87$			
Absent [index]	43 (345)	0.30 (0.22–0.37)	
Present	10 (31)	0.31 (0.06–0.56)	
Study quality			1697.25***, 86.9%
$F(df1 = 1, df2 = 374) = 0.00, p = .98$			
Low quality (<6) [index]	38 (243)	0.30 (0.22–0.38)	
High quality (≥6)	15 (133)	0.30 (0.22–0.38)	

Note. PTSD = Posttraumatic Stress Disorder; TBI = Traumatic Brain Injury.

*** $p < .0001$.

2.4. Publication bias

The Egger's test, which was used to assess publication bias in the main analysis for the association between PTSD and cognitive impairment, was significant, indicating a publication bias ($t[374] = 3.08, p = .002$). Therefore, Duval and Tweedie's trim-and-fill procedure was employed to estimate the actual effect size. The initial pooled effect across the studies was $g = 0.30$, but after accounting for publication bias, it was $g = 0.13, p < .0001, 95\% \text{ CI } (0.10–0.17)$, indicating a small effect size. The robustness of the association between PTSD and cognitive impairment was supported by Rosenthal's fail-safe N, which indicated that 9,531,366 null findings would be needed to nullify the effect, suggesting negligible risk of bias (Rosenthal, 1991; Shi et al., 2021).

3. Discussion

This is the first meta-analysis, to our knowledge, to comprehensively investigate the association between PTSD and a broad spectrum of cognitive impairment, including the risk of developing NCD later in life. Additionally, this is the first meta-analysis to explore this topic employing a multilevel meta-analytic approach, which is less susceptible to bias than traditional meta-analytic methods (Cheung, 2019). The objectives of the current meta-analysis were firstly, to elucidate the relationship between PTSD and cognitive impairment, and secondly, to identify moderating factors for cognitive impairment among individuals with PTSD.

In line with previous findings (Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015), we

found that PTSD was associated with a higher risk of cognitive impairment. This finding remained robust regardless of the moderators explored, i.e. individuals with PTSD were at an increased risk of cognitive impairment compared to controls across all neurocognitive outcomes, ages, genders, study designs, study populations, study qualities, types of PTSD measurements, and presence of comorbidities such as depression, substance use, and TBI. This means we did not achieve our aim of identifying moderating factors for cognitive impairment among individuals with PTSD. However, we were able to elucidate the relationship between PTSD and cognitive function further, demonstrating a consistent link to cognitive impairment that persisted across all explored moderators. The findings suggest that PTSD is associated with a higher risk of developing NCD later in life, which is consistent with a previous meta-analysis on the topic (Günak et al., 2020). There is also a possibility that individuals with pre-existing neurocognitive vulnerabilities – such as limited cognitive ability, low educational levels, and hippocampal atrophy – may be likelier to develop PTSD. These factors, which also predispose individuals to NCD, might heighten the risk of subsequent cognitive impairment (Elias et al., 2021; Günak et al., 2020).

Consistently, earlier meta-analyses have also found that PTSD is associated with a higher risk of impairments in specific cognitive domains such as attention, processing speed, working memory and executive functioning (Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015). The inclusion of NCD in our study (not explored in previous studies (Qureshi et al., 2011; Schuitevoerder et al., 2013; Scott et al., 2015)) may have contributed to the smaller effect sizes observed in our meta-analysis compared to those reported by e.g. Scott and colleagues, whose meta-analysis is the most similar to ours in terms of study diversity and total number of included studies (Scott et al., 2015). This inclusion could potentially have diluted the specific neurocognitive effects of PTSD, as cognitive impairment may arise from both conditions. Another reason for the smaller effect sizes in our study compared to theirs might be differences in search criteria. Scott et al. searched for cognitive domain-specific keywords (e.g. memory, attention) and traumatic stress in addition to PTSD. Meanwhile, we excluded studies examining traumatic stress and did not search for cognitive domain-specific keywords. Our keywords were more targeted toward PTSD and more broadly defined cognitive function, cognitive impairment and NCD.

Consistent with the findings of Scott et al. (2015), neither age, type of PTSD measurement, depression, TBI, or substance use, significantly moderated the association between PTSD and cognitive impairment. The observation that PTSD was associated with

cognitive impairment in both veterans and among the general population was also in line with previous results (Günak et al., 2020; Scott et al., 2015). Regarding gender differences, our findings diverge from those of Scott et al. (2015), who found that studies with a larger portion of men exhibited greater cognitive impairment. Conversely, Günak et al. (2020) found an increased risk of developing NCD among women with PTSD. The inclusion of both cognitive impairment and NCD in our study may have contributed to the absence of significant gender differences in our results.

While previous meta-analyses (that we know of) have not examined study design as a potential moderator of the relationship between PTSD and cognitive impairment, our observation that this association remains robust regardless of study design, provides new insights. This indicates that individuals with PTSD consistently exhibit cognitive impairments, regardless of whether a study collects data at a single timepoint or over a long period of time. This observation highlights the importance of early intervention and ongoing support for individuals with PTSD to mitigate potential development of cognitive impairment and NCD over time. To minimize patient burden, the cognitive function of individuals with PTSD could be monitored via self-report, since studies have found that self-reported memory complaints may predict NCD in later-life (Mitchell et al., 2014; Rönnlund et al., 2015). However, to our knowledge, this has not been studied yet among individuals with PTSD. Clinically, these findings underscore the importance of addressing the risk of cognitive impairment in individuals with PTSD. To mitigate this risk, it is essential to implement regular monitoring of cognitive function as part of standard care for PTSD patients. Additionally, targeted interventions should focus on promoting healthy sleep hygiene and encouraging engagement in regular physical and social activities (Livingston et al., 2017; Miller, 2015). These approaches address common challenges faced by individuals with PTSD, such as disrupted sleep and social isolation, which are also recognized risk factors for cognitive impairment (Beidel et al., 2019; Han et al., 2021). By integrating these strategies into clinical practice, healthcare providers can better support the long-term cognitive and mental health of individuals with PTSD. Our findings also highlight the importance of future research focusing on identifying modifiable risk factors for cognitive impairment and NCD among individuals with PTSD, as well as potential treatment targets.

The strengths of the current study lie in its use of the three-level meta-analytic approach, and its inclusion of a large number of analyses and a broad range of studies examining a wide spectrum of cognitive impairment. Additionally, we only included

studies that employed validated methods to clinically diagnose PTSD or established cutoffs shown to be indicative of a probable diagnosis. Furthermore, the current research encompasses all age groups and maintains a balanced representation of veterans and the general population in its analyses. This inclusive approach enhances the generalizability of the findings to the broader population compared to some previous meta-analyses (Qureshi et al., 2011; Schuitevoerder et al., 2013). When meta-analyses in this field consist primarily of veterans, they tend to overrepresent men (with comorbidities individually increasing the risk of cognitive impairment and NCD, such as TBI) and represent a different type of trauma occurring under special circumstances, which could potentially affect the study results (Scott et al., 2015).

Limitations of this study include that studies assessing cognitive function may exclude participants with TBI, potentially leading to a selection bias that could affect the generalizability of our findings regarding the moderating role of TBI. Furthermore, we could not control for anxiety, a common comorbidity of PTSD that may also elevate the risk of cognitive impairment, since only two of the included studies controlled for anxiety. One meta-analysis found an increased risk of NCD among individuals with depression and anxiety but not among individuals with PTSD (Kuring et al., 2020). In the current study, we found that even when controlling for depression, PTSD was associated with cognitive impairment. This contrasts the previous study (Kuring et al., 2020), which only included four studies on the association between PTSD and NCD, possibly limiting its ability to fully capture the impact of PTSD on cognitive function due to its narrow scope and small sample size. Moving forward, it is important that future studies control for both anxiety and depression and explore their potential roles in the association between PTSD and cognitive function. This approach would provide a clearer understanding of the mechanisms at play, which could inform therapeutic targets, and enhance the generalizability and applicability of the findings. Another limitation is that only two studies controlled for pre-morbid intelligence and one for attention-deficit hyperactivity disorder, which Scott et al. (2015) found affected their effect sizes. Additionally, we did not control for antipsychotic use. One study (Roughead et al., 2017) found that veterans who were administered antipsychotics had a higher NCD diagnosis rate irrespective of PTSD status, but this could not be explored further due to lack of information on medication status in the studies.

Moreover, our findings exhibited considerable heterogeneity, the origins of which remain unclear. This heterogeneity persisted despite conducting sensitivity analyses, an issue also noted in previous meta-analyses

in the field (Günak et al., 2020; Scott et al., 2015). To address this, the development of consensus guidelines on key methodological aspects – such as diagnostic criteria, control of confounders, and the inclusion of large, non-biased samples – is recommended to enhance the quality and consistency of findings across studies. As previously mentioned, only 11% of the included studies in this meta-analysis were categorized as having a low risk of bias (NOS score ≥ 7) (Lo et al., 2014). Additionally, the presence of a publication bias in this meta-analysis underscores the importance of pre-registering studies and publishing all results, including those with negative or null findings, to provide a more balanced view of the evidence.

Lastly, like many neuropsychological studies, ours contends with the limitation that neuropsychological tests often assess multiple cognitive domains simultaneously, complicating precise categorization into single domains. For example, impairments in executive function can adversely affect performance across various other domains (Harvey, 2019). To apply a structured approach to classifying the wide array of tests, we used the Compendium of Neuropsychological Tests (Sherman & Spreen, 2006) as a guide.

Our findings indicate that future meta-analyses on the association between PTSD and cognitive impairment might consider including NCD diagnoses, and a diverse range of studies in terms of populations, age groups and types of trauma. Such inclusions could expand upon our findings and provide deeper insights into the long-term effects of PTSD on cognitive function. The field would benefit from recruiting more women to enhance gender diversity and implementing performance validity tests to ensure that neuropsychological assessments accurately reflect participants' true cognitive function, rather than their effort (Tierney et al., 2023; Wisdom et al., 2014). Future meta-analyses should stratify the results by gender, and account for the potential confounding variables mentioned above. It is also important to match the control group to the participants with PTSD on age, given the increased risk of cognitive impairment with age (Morovic et al., 2019). Additionally, they could include the age at which participants were exposed to the traumatic experience as well as their subsequent PTSD duration since trauma could have more profound and lasting effects on the developing brain (Nilaweera et al., 2020; Scott et al., 2015). Furthermore, future longitudinal research should explore whether self-reported cognitive complaints can predict NCD later in life among individuals with PTSD. Lastly, conducting longitudinal intervention studies is essential to determine whether the risk of cognitive impairment or NCD later in life can be mitigated among individuals with PTSD.

4. Conclusions

The results indicate that individuals with PTSD are at an increased risk of cognitive impairment, including the development of NCD later in life, compared to controls. This association remained robust regardless of the explored moderators, as it was consistently observed across all neurocognitive outcomes, age groups, genders, study designs, study populations, study qualities, types of PTSD measurements, and the presence of comorbidities such as TBI, depression and substance use.

Acknowledgments

We thank Dr. Brynja Björk Magnúsdóttir for her guidance on classifying neuropsychological tests into cognitive domains. In the preparation of this manuscript, we utilized OpenAI's ChatGPT (version 4) to occasionally assist in language refinement and advising on statistical analysis techniques.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was funded by the Reykjavik University Research Fund.

Authors' contributions

SGA, HLL, TH, and HLHJ designed the study. SGA and HLL extracted the data. SGA, HLL, and HLHJ assessed the data quality. SGA, TH and HLHJ did the data analysis. SGA, TH and HLHJ drafted the manuscript. TH, HLL, UAV, HBV, BB, and HLHJ provided feedback on the manuscript. SGA incorporated feedback and finalized the manuscript. All authors read and approved the final manuscript.

Data availability statement

The data supporting the findings of this study are openly available in the Figshare repository at <https://doi.org/10.6084/m9.figshare.27285219.v2>.

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