

# Using network models to explore the associations between posttraumatic stress disorder symptoms and subjective cognitive functioning

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## ARTICLE INFO

### Keywords:

Trauma  
Posttraumatic stress disorder  
Symptoms  
Cognitive ability  
Cognitive impairment

## ABSTRACT

Several studies have identified relationships between posttraumatic stress disorder (PTSD) and cognitive functioning. Here, we aimed to elucidate the nature of this relationship by investigating cross-sectional associations between subjective cognitive functioning (SCF) and 1) the PTSD sum score, 2) symptom domains, and 3) individual symptoms. We also investigated temporal stability by testing whether results replicated over a 3-year period. We estimated partial correlation networks of DSM-5 PTSD symptoms (at baseline) and SCF (at baseline and follow-up, respectively), using data from the National Health and Resilience in Veterans Study (NHRVS;  $N = 1484$ ;  $Mdn = 65$  years). The PTSD sum score was negatively associated with SCF. SCF was consistently negatively associated with the PTSD symptom domains ‘marked alterations in arousal and reactivity’ and ‘negative alterations in cognitions and mood’, and showed robust relations with the specific symptoms ‘having difficulty concentrating’ and ‘trouble experiencing positive feelings’. Results largely replicated at the 3-year follow-up, suggesting that some PTSD symptoms both temporally precede and are statistically associated with the development or maintenance of reduced SCF. We discuss the importance of examining links between specific PTSD domains and symptoms with SCF—relations obfuscated by focusing on PTSD diagnoses or sum scores—as well as investigating mechanisms underlying these relations.

Registration Number: 37069 (<https://aspredicted.org/n5sw7.pdf>)

## 1. Introduction

Posttraumatic stress disorder (PTSD) may arise in response to a traumatic event such as life-threatening violence, combat, abuse, or injury (American Psychiatric Association, 2013). According to the DSM-5, symptoms are clustered into four domains: intrusions, avoidance of reminders and distressing memories of the trauma, negative alterations in cognitions and mood, and alterations in arousal and reactivity (American Psychiatric Association, 2013). Varying greatly across trauma types, the conditional risk for developing PTSD after any trauma

exposure is estimated to be 4.0 %, and 3.5 % after any lifetime war-related trauma exposure (Kessler et al., 2017). Delay in treatment for PTSD is common (Wang et al., 2005), often resulting in a chronic condition accompanied by impairments across a range of areas, including cognitive functioning, daily living, and mental health-related quality of life (Hunnicut-Ferguson et al., 2018; Pittman, Goldsmith, Lemmer, Kilmer, & Baker, 2012; Qureshi et al., 2011; Ross, Murphy, & Armour, 2018).

Cognitive impairment in PTSD has attracted attention in recent years. Several studies have found impairment across cognitive domains

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in both veteran and non-military populations with PTSD compared to those without, including impairments in (working) memory, attention, learning, executive function, and processing speed, assessed using neuropsychological tests (Clouston et al., 2016; Cohen et al., 2013; Koso & Hansen, 2006; Samuelson et al., 2006; Schuitevoerder et al., 2013; Vasterling et al., 2012, 2018; Vasterling, Brailey, Constans, & Sutker, 1998; Yehuda, Golier, Tischler, Stavitsky, & Harvey, 2005), traditionally assessed using behavioral and computerized tasks (Schuitevoerder et al., 2013; Scott et al., 2015), which have been considered the gold standard to assess specific cognitive functions (Savard & Ganz, 2016). PTSD has also been associated with *subjective* cognitive difficulties (Boals & Banks, 2012; Singh et al., 2020; Vasterling et al., 2012), which, in turn, have been shown to predict future objective cognitive decline and dementia (Jessen et al., 2010; Koppa et al., 2015; Mitchell, Beaumont, Ferguson, Yadegarfar, & Stubbs, 2014). While less often investigated, everyday subjective cognitive concerns likely represent different—though also valid and relevant—facets of cognition relative to those assessed in the lab (Carrigan & Barkus, 2016). In fact, some of the subjective difficulties may be too subtle to be detected by objective neuropsychological assessment (Geerlings, Jonker, Bouter, Adèr, & Schmand, 1999) and only noted by the individual (Molineuvo et al., 2017); such difficulties can indicate early-stage cognitive impairment (Singh et al., 2020). Another advantage of subjective cognitive assessment is that this is much more feasible in clinical practice (Silverberg et al., 2017). Studies increasingly indicate that both objective and subjective cognitive measures have their benefits and limitations, and are not interchangeable (Hess et al., 2020; Lau, Connor, & Baum, 2021; Savard & Ganz, 2016).

Most prior studies have examined the association between a diagnosis of PTSD and cognitive functioning. The few studies that have decomposed PTSD into symptom domains have found that intrusive symptoms in particular are strongly linked to cognitive difficulties (Boals, 2008; Bomyea, Amir, & Lang, 2012; Clouston et al., 2016, 2019; Johnsen, Kanagaratnam, & Asbjørnsen, 2008; Kivling-Bodén & Sundbom, 2003; Parslow & Jorm, 2007; Saltzman, Weems, & Carrion, 2006; Vasterling et al., 1998). Both intrusive and hyperarousal symptoms have been suggested to compete for attentional resources with ongoing cognitive processes (Boals, 2008; Boals & Banks, 2012; Kolb, 1987). This may be associated with a reduced ability to inhibit reactions to irrelevant information (Vasterling et al., 1998) and regulate the content of cognition (Bomyea, Amir, & Lang, 2012). Yet, evidence is inconsistent whether hyperarousal symptoms are related to impaired cognitive functioning (Bomyea, Amir, & Lang, 2012; Clouston et al., 2016; Judah, Renfro, Wangelin, Turner, & Tuerk, 2018; Kivling-Bodén & Sundbom, 2003; Parslow & Jorm, 2007; Vasterling et al., 1998; Wrocklage et al., 2016). Vasterling et al. (1998) found that in Persian Gulf War veterans, such disinhibition was negatively associated with avoidance-numbing symptoms, which may reflect the tendency to avoid (i.e., inhibit), at least superficially, intense trauma-related experiences and thereby, preserve cognitive functioning. Other studies found no link between avoidance and cognitive functioning (Boals, 2008; Bomyea, Amir, & Lang, 2012; Clouston et al., 2016; Wrocklage et al., 2016).

In addition to these mixed findings, research has generally focused on the dichotomy between individuals with a diagnosis of a mental disorder and those without (Armour, Fried, & Olf, 2017). Most mental health studies are based on case-control or randomized controlled trial study designs. This is suboptimal, however, as not all treatment-seeking individuals meet diagnostic criteria for mental disorders. Additionally, there are 636,120 possible symptom combinations that qualify for a DSM-5 PTSD diagnosis alone (Galatzer-Levy & Bryant, 2013), calling into question the usefulness of categorical diagnoses for research purposes. The heterogeneity of symptom presentations has led to ongoing debates about the validity and reliability of DSM diagnostic criteria, both in general (Insel, 2013) and specific to PTSD (Armour, Müllerová, & Elhai, 2016a; Armour, Contractor, Shea, Elhai, & Pietrzak, 2016b). Taken together, this work suggests that there may be value in trying to understand the relation between PTSD symptoms with cognitive

functioning by examining the symptoms people experience, both within their domains and individually, rather than as a more diffuse, homogeneous syndrome.

Statistical network models lend themselves well as a tool to examine the link between individual PTSD symptoms and cognitive functioning. First, they are well suited to model a larger number of variables simultaneously. Second, commonly used network models are conditional dependence models, i.e., they estimate the link between two variables A and B after controlling for all other variables in the network, helping to identify potential mechanisms. Finally, network models can visualize statistical relations, which can guide interpretation of highly multivariate dependency structures (Borsboom, 2017; Fried et al., 2017). Non-technical introductions to network analyses can be found in Isvoranu et al. (Isvoranu, Epskamp, Waldorp, & Borsboom, 2022).

To date, no known published network analysis study has investigated the link between PTSD symptoms and (subjective) cognitive functioning. Moreover, the majority of previous (non-network) studies have used cross-sectional designs to examine this association (Schuitevoerder et al., 2013). Although existing cohort studies indicate longitudinal associations between the two constructs (Gould et al., 2018; Vasterling et al., 2018), little is known whether PTSD-related (subjective) cognitive impairment is stable over time. Clarifying the associations of PTSD symptom domains and specific symptoms with subjective cognitive functioning (SCF), and their temporal relations, may facilitate future work to identify individuals with PTSD who may be at risk of cognitive decline, and guide individualized treatment planning (Fried et al., 2017; Kivling-Bodén & Sundbom, 2003).

The aim of the present study is to identify specific PTSD symptoms and symptom domains that are associated with SCF, and to investigate temporal stability of the relations by analyzing a second wave of data three years later. We investigated four specific research questions (RQs): (1) Is overall severity of PTSD symptoms associated with SCF in U.S. veterans; (2) which PTSD symptom domains are most strongly related to SCF; (3) which individual PTSD symptoms are most robustly associated with SCF; and (4) do the findings of questions 1–3 hold over a three-year follow-up? We predicted that the overall severity of PTSD symptoms would be negatively associated with SCF; that the symptom domain of intrusion shows the strongest overall link to reduced SCF compared to other symptom domains; and that the associations of the estimated network models at baseline (i.e., Wave 1) will hold at a three-year follow-up (i.e., Wave 2). The main analyses of the present study were pre-registered (<https://aspredicted.org/n5sw7.pdf>). All data, code, measures, and **Supplementary materials** are freely accessible online (<https://osf.io/5w6k4/>).

## 2. Methods

### 2.1. Participants and procedure

We analyzed data drawn from the second cohort of the National Health and Resilience in Veterans Study (NHRVS), a survey of a nationally representative sample of U.S. military veterans (Wisco et al., 2016). This prospective cohort was recruited in September and October 2013 (i.e., baseline; Wave 1) from a research panel of U.S. households that has been developed and maintained by Growth for Knowledge (GfK) Incorporated (now Ipsos), a survey research company based in Menlo Park, California (GfK Knowledge, 2020). Panel members were employed through a sampling procedure that includes listed and unlisted phone numbers; telephone, non-telephone, and cell-phone only households; and households with or without Internet access, allowing coverage of approximately 98 % of U.S. households. Of 1602 veterans who were in the survey panel when the NHRVS cohort was recruited, 1484 (92.6 %) took part in the NHRVS and completed a confidential, 60-min Web-based survey that assessed a range of sociodemographic, psychiatric and health variables. The cohort was re-assessed in September and October 2016 (i.e., follow-up; Wave 2). A total of 713 (48.0 %) veterans

completed both assessments at baseline and follow-up. All participants provided informed consent. The Human Subjects Subcommittee of the Veterans Affairs (VA) Connecticut Healthcare System and VA Office of Research & Development approved the study.

## 2.2. Measures

### 2.2.1. Lifetime exposure to trauma

The 14-item self-report measure Trauma History Screen (THS) (Carlson et al., 2011) assesses lifetime exposure to 14 DSM-5 Criterion A-qualifying trauma events for PTSD (Yes/No) (American Psychiatric Association, 2013). It includes traumatic experiences across the lifespan such as physical or sexual assault, accidents, traumatic incidences during military service, and unexpected loss of a close person. “Life-threatening illness or injury” was added as a potentially traumatic event before data collection, given a sample of older military veterans. Participants who endorsed multiple traumatic experiences were asked, “Which of these experiences was the worst for you?”.

### 2.2.2. PTSD symptoms

The PTSD Checklist for DSM-5 (PCL-5) is a self-report measure that assesses the presence and severity of PTSD symptoms (Weathers et al., 2013). It comprises 20 items, which are rated on a 5-point Likert scale ranging from 0 (*Not at all*) to 4 (*Extremely*). The items on the PCL-5 assess individual DSM-5 symptoms of PTSD and represent clusters B-E (i.e., ‘intrusion’, ‘persistent avoidance’, ‘negative alterations in cognitions and mood’, ‘marked alterations in arousal and reactivity’) (American Psychiatric Association, 2013; Weathers et al., 2013). In the NHRVS cohort, the PCL-5 was modified to include both lifetime (at baseline) and past-month (at baseline and follow-up) ratings of PTSD symptoms with regards to respondents’ self-selected “worst” stressful experience identified on the THS. Higher sum scores indicate greater severity of PTSD symptoms. Internal consistency was excellent for baseline past-month and lifetime PCL-5 (Cronbach’s  $\alpha = .95$ , respectively). Probable PTSD was determined as a past-month PCL-5 sum score of  $\geq 31$ , as recommended by previous evidence (Bovin et al., 2016). While this cut-off score served to identify participants with probable PTSD to describe the sample, no cut-off score was applied for the analyses to examine the relationship between PTSD on a dimensional continuum rather than categorically. Thus, all participants in the sample who were exposed to trauma and consequently filled out the PCL-5 were included in the study. This also mitigates the impact of Berkson’s bias, which threatens inferences when including participants based on a specific threshold of symptoms (De Ron, Fried, & Epskamp, 2021).

### 2.2.3. Cognitive functioning

One subscale of the Medical Outcomes Study scale assesses past-month cognitive functioning (MOS-CF) and is a self-report measure encompassing six Likert-type items on difficulties in the following cognitive domains: reasoning, memory, attention, concentration and thinking, confusion, psychomotor speed (Averill et al., 2019; Stewart and Ware, 1992). Sample item: “During the past month, how much of the time did you forget (e.g., things that happened recently, where you put things, appointments)?” The responses to the individual items were standardized to a scale ranging from 0 (*All of the time*) – 100 (*None of the time*), and then averaged (Hays, Sherborne, & Mazel, 1995). The MOS-CF has been shown to be a reliable and valid measure (Revicki, Chan, & Gevirtz, 1998; Yaras, White, & Bjorner, 2013). Internal consistency in our data was excellent at baseline and follow-up (Cronbach’s  $\alpha = .93$ , respectively).

### 2.2.4. Covariates

Age, sex, level of education, depression and alcohol misuse were included as pre-registered covariates. A demographic questionnaire assessed, amongst others, the first three covariates. Lifetime history of major depressive disorder and alcohol abuse/dependence were

measured with the Mini International Neuropsychiatric Interview for DSM-IV (Sheehan et al., 1998).

## 2.3. Statistical analysis

We compared baseline and follow-up sample characteristics using the Wilcoxon Signed-Rank Test, the McNemar, and the McNemar’s-Bowker test. Additionally, to test for systematic dropout, we compared baseline characteristics (i.e., age, sex, race/ethnicity, level of education, employment, number of lifetime traumatic events, combat exposure, lifetime major depressive episode and alcohol abuse/dependence, past-month and lifetime PCL-5 sum scores, probable PTSD, and MOS-CF average scores) of veterans who completed the follow-up assessment relative to those who did not, using the Mann-Whitney U test and Chi-squared test.

To test whether severity of overall PTSD symptoms was associated with SCF cross-sectionally and longitudinally, three years later (RQ1 and RQ4), we computed Spearman correlations between PTSD (past-month and lifetime PCL-5 sum scores) at baseline and SCF (MOS-CF mean scores) at baseline and follow-up, respectively; we used Spearman correlations because distributions of PCL-5 and MOS-CF items were skewed and were measured on an ordinal scale.

### 2.3.1. Network estimation

For RQ2, RQ3, and RQ4—which symptom domains/individual PTSD symptoms are most strongly associated with SCF and whether these associations persist at follow-up—we estimated two types of networks (see Table 1 for an overview). Network 1 included baseline past-month PCL-5 items and SCF scores. Network 2 included baseline past-month PCL-5 items and follow-up SCF scores. We use the term “cross-sectional network models” if all included variables were measured at Wave 1 (i.e., baseline) and “longitudinal network models” if associations between the variables of interest were assessed across two waves (i.e., baseline and three-year follow-up). We estimated network models based on Spearman correlations (Epskamp & Fried, 2018) and controlled for all preregistered covariates in each network. In network models, ‘nodes’ represent variables and ‘edges’ between these nodes conditional dependence relations (akin to partial correlations), which are associations between nodes after controlling for the influence of all other nodes (i.e., variables) (Epskamp, Borsboom, & Fried, 2018; Epskamp & Fried, 2018). As the data involves mostly ordinal variables, we estimated the networks by means of the Gaussian Graphical Model (GGMs) with the R-package *bootnet* (Epskamp, Borsboom, & Fried, 2018). Sex, level of education, lifetime depression, and lifetime alcohol abuse/dependence were treated as ordinal. To avoid false positive findings and reduce the risk of overfitting, we estimated GGMs by using the ‘least absolute shrinkage and selection operator’ (LASSO) (Tibshirani, 1996). LASSO shrinks all coefficients towards zero and sets small weights exactly to

**Table 1**  
Overview of the six network models.

Network	PTSD Symptoms	Subjective Cognitive Functioning
1	PCL-5 <sup>a</sup> past-month	MOS-CF <sup>a</sup>
2	PCL-5 <sup>a</sup> past-month	MOS-CF <sup>b</sup>
2 <sub>adj</sub>	PCL-5 <sup>a</sup> past-month	MOS-CF <sup>b,c</sup>
3	PCL-5 <sup>a</sup> lifetime	MOS-CF <sup>a</sup>
4	PCL-5 <sup>a</sup> lifetime	MOS-CF <sup>b</sup>
4 <sub>adj</sub>	PCL-5 <sup>a</sup> lifetime	MOS-CF <sup>b,c</sup>

*Note.* PTSD = Posttraumatic stress disorder; PCL-5 = PTSD Checklist for DSM-5; MOS-CF = Medical Outcomes Study – Cognitive Functioning scale. Each estimated network model is adjusted for age, sex, level of education, lifetime depression, and lifetime alcohol abuse/dependence. We use the term “cross-sectional network model” for models 1 and 3, and “longitudinal network model” for models 2, 2<sub>adj</sub>, 4 and 4<sub>adj</sub>.

<sup>a</sup>Wave 1, baseline; <sup>b</sup>Wave 2, three-year follow-up; <sup>c</sup>additionally adjusted for subjective cognitive functioning at baseline.



zero. The strength of the shrinkage is controlled via the tuning parameter  $\lambda$ , which is selected by minimizing the Extended Bayesian Information Criterion (EBIC) (Chen & Chen, 2008; Epskamp, Borsboom, & Fried, 2018; Epskamp & Fried, 2018; Foygel & Drton, 2010). The EBIC itself involves  $\gamma$ , a hyperparameter that controls to what extent the EBIC favors simpler models with fewer edges, which was set to 0.5 (the default setting) for all network analyses.

### 2.3.2. Network inference

To test which PTSD symptom domain was most strongly associated with SCF scores (RQ2), we computed average connectivity of each symptom domain with SCF scores. That is, signed values of edge weights between all PTSD symptoms of a domain and SCF scores were summed and then divided by the total number of potential edges within that domain (that is, domains with more variables are penalized, otherwise they are more likely to relate to SCF simply because they have more nodes). Differences in average connectivity between PTSD symptom domains and SCF scores were bootstrapped with 1000 iterations using the R-package *bootnet* (Epskamp, Borsboom, & Fried, 2018). As a minor deviation from the pre-registration, we used signed rather than absolute edge weight values in these calculations, given that negative and positive edges are meaningfully different here.

We estimated node predictability using the *mgm* R-package which can be interpreted akin to  $R^2$ , quantifying how well a node can be predicted by other nodes (Haslbeck & Fried, 2017; Haslbeck & Waldorp, 2018).

We quantified the accuracy of estimated edge weights using bootstrapping routines from the *bootnet* R-package (Epskamp, Borsboom, & Fried, 2018), see [Supplementary materials](https://osf.io/5w6k4/) for details (<https://osf.io/5w6k4/>).

### 2.3.3. Network visualization

We visualized all resulting associations as network graphs using the R-package *qgraph*. The layout was constrained across all figures, and we set the same maximum value as the strongest edge in all networks, to allow for comparisons between the network structures.

### 2.3.4. Network comparison test

To investigate temporal stability (RQ4), we statistically compared Network 1 with Network 2. First, to obtain a coefficient of similarity for the networks, we computed Spearman correlations of the adjacency matrices. Second, we tested whether network models 1 and 2 differed from one another, using the R-package *NetworkComparisonTest* (NCT) (van Borkulo et al., 2022); NCT is a permutation test, and we used 1000 iterations. By the time of the preregistration, samples for Networks 1 and 2 needed to have equal size. We deviate from the preregistration and include a larger sample for Network 1, because the NCT-package no longer requires this restriction. We tested whether the two network models had equal global strength (i.e., sum of signed edge weight values) and edge weight distributions (i.e., network structure). If the network structures differed statistically significantly, we specifically investigated individual edges.

### 2.3.5. Missing data

Our pre-registration protocol did not specify how missing data would be handled. We used multiple imputation by chained equations to impute missing past-month and lifetime PCL-5 item values prior to analysis for participants who were missing less than 5 % of data. For further details and an overview of sample sizes for each analysis, see [Supplementary Materials](https://osf.io/5w6k4/) (<https://osf.io/5w6k4/>).

### 2.3.6. Robustness analyses

We performed several analyses to assess the robustness of the results. Our main models were estimated regularized network models without thresholding, which are the default in the literature. However, since recent research identified potential problems with regularization under

specific scenarios (Williams, Rhemtulla, Wysocki, & Rast, 2019), we also used alternative approaches to estimate network models with 1) thresholding (Epskamp and Fried, 2018; Muthén, 1984; Epskamp, 2018) and 2) using *ggmModSelect* (Epskamp, 2018), see [Supplementary Materials](https://osf.io/5w6k4/) for more information (<https://osf.io/5w6k4/>). Second, we computed Spearman correlations between the adjacency matrices of PTSD symptoms at baseline and three-year follow-up to estimate similarity between the two, followed by repeating all the above analyses with PTSD symptoms assessed during lifetime rather than last month (i.e., Network 3: lifetime PCL-5 with SCF scores at baseline; Network 4: lifetime PCL-5 at baseline with SCF at follow-up, see [Table 1](#)). Third, we correlated SCF at baseline and at follow-up; this was followed by repeating the analyses of the two longitudinal networks of past-month and lifetime PCL-5 at baseline with SCF at follow-up (i.e., Networks 2 and 4)—but this time additionally adjusting for SCF at baseline. In total, we estimated six network models, see [Table 1](#) for an overview. Fourth, we compared cross-sectional (i.e., Network 1 with Network 3), and longitudinal networks (i.e., Network 2 with Network 4, both with and without adjusting for SCF at baseline) using NCT. The R-package NCT currently cannot compare network models that do not contain an equal number of variables. Hence, the re-estimated longitudinal networks taking SCF at baseline into account cannot be compared to the cross-sectional models (which had one variable less), and therefore, were included as robustness analyses. Finally, we repeated the above analyses on the subsample of 91 % (or more depending on the subsample) individuals with complete (i.e., non-imputed) data.

The analyses were conducted in June 2022, using R (version 4.2.0) for all statistical analyses except for the Wilcoxon Signed-Rank Test, the McNemar, and the McNemar-Bowker test, which were performed using SPSS (version 28.0.1.1). All R-packages and versions can be found online (<https://osf.io/5w6k4/>).

## 3. Results

### 3.1. Sample characteristics

Respondents were predominantly non-combat veterans (61.7 %), male (89.4 %), non-Hispanic White (81.1 %), and older adults, with a median age of 65 years ( $IQR = 54\text{--}73$  years); see [Table 2](#) for baseline characteristics. Of the 1484 veterans, 1268 (85.4 %) had been exposed to at least one traumatic event at baseline and on average, experienced approximately three such events. Types of trauma experienced are listed online (<https://osf.io/5w6k4/>). Participants of the 3-year follow-up significantly differed from those at baseline on three variables: race/ethnicity ( $p = .046$ ), with fewer non-Hispanic veterans at follow-up; reduced SCF ( $p = .002$ ), likely explained by aging; and employment ( $p < .001$ ) with more veterans being retired and fewer currently looking for work.

Veterans who did not complete the follow-up assessment did not differ significantly from those who did with respect to most socio-demographic or clinical variables, except for employment ( $p = .017$ ), past-month PCL-5 sum scores ( $p = .024$ ), and MOS-CF average scores ( $p = .043$ ). Details can be found in [Supplementary Materials](https://osf.io/5w6k4/) (<https://osf.io/5w6k4/>).

The main results will be presented in the following order: the overall association between SCF and the sum score of PTSD symptoms (RQ1); results of the network analyses, including the associations between SCF and both individual PTSD symptoms and PTSD symptom domains (RQ3, RQ2); followed by testing the temporal stability of the associations over three years (RQ4).

### 3.2. Overall association between PTSD and SCF

Associations between PTSD and SCF were of similar magnitude for past-month and lifetime PTSD scores. At baseline, past-month and lifetime PCL-5 sum score were negatively associated with MOS-CF scores ( $r$

**Table 2**  
Baseline characteristics.

	Entire Sample (N = 1484)	Participants Exposed to Trauma (n = 1268)
Age		
Median (IQR)	65 (54–73)	65 (54–73)
Mean (SD)	62.8 (14.7)	62.8 (14.6)
Female, n (%)	158 (10.6)	132 (10.4)
Race/Ethnicity, n (%)		
Non-Hispanic White	1204 (81.1)	1028 (81.1)
Non-Hispanic Black	112 (7.5)	95 (7.5)
Hispanic	99 (6.7)	85 (6.7)
Other, Non-Hispanic	23 (1.5)	20 (1.6)
2 + Races, Non-Hispanic	46 (3.1)	40 (3.2)
Education, n (%)		
Less than high school	26 (1.8)	23 (1.8)
High school	211 (14.2)	174 (13.7)
Some college	629 (42.4)	548 (43.2)
Bachelor's degree or higher	618 (41.6)	523 (41.2)
Employment, n (%)		
Working	476 (32.1)	403 (31.8)
Retired	718 (48.4)	604 (47.6)
Not working	290 (19.5)	261 (20.6)
Number of lifetime traumatic events		
Median (IQR)	3.0 (1.0–5.0)	3.0 (2.0–5.0)
Mean (SD)	3.3 (2.8)	3.9 (2.7)
Combat exposure, n (%)	564 (38.0)	508 (40.1)
Major depressive episode (lifetime), n (%)	137 (9.2)	131 (10.3)
Alcohol abuse/dependence (lifetime), n (%)	542 (36.5)	490 (38.6)
PCL-5 (past month)		
Median (IQR)	4.5 (1.0–13.0)	4.5 (1.0–13.0)
Mean (SD)	9.7 (13.0)	9.7 (13.0)
Probable PTSD, n (%)	93 (6.3)	93 (7.3)
PCL-5 (lifetime)		
Median (IQR)	9.0 (4.0–19.0)	9.0 (4.0–19.0)
Mean (SD)	14.1 (14.6)	14.1 (14.6)
MOS-CF		
Median (IQR)	96.7 (86.7–100.0)	96.7 (83.3–100.0)
Mean (SD)	90.0 (15.0)	89.1 (15.5)
MOS-CF – Wave 2		
Median (IQR)	96.7 (86.7–100.0)	95 (85.8–100.0)
Mean (SD)	89.5 (15.3)	89.2 (15.5)

Note. N = sample size; IQR = Interquartile range; SD = standard deviation; PCL-5 = PTSD Checklist for DSM-5; MOS-CF = Medical Outcomes Study – Cognitive Functioning scale.

= −0.58 and −0.54, respectively, both  $p < .001$ ). Similarly, baseline past-month and lifetime PTSD symptoms were each negatively associated with MOS-CF scores at three-year follow-up ( $r = -0.32$  and  $-0.33$ , respectively, both  $p < .001$ ). Our hypothesis of a negative relationship between the total PTSD symptom score and SCF was supported (RQ1), and they remained negatively associated at follow-up (RQ4).

### 3.3. Individual PTSD symptoms and SCF

We report edge weights and predictability values that were most relevant to our research questions. Unless stated otherwise, edge weights represent negative relationships. Fig. 1 shows Networks 1 and 2. Edges between SCF and PTSD mostly emerged for symptoms of ‘alterations in arousal and reactivity’, and ‘negative cognitions and mood’ (RQ2, RQ3), with similar findings at follow-up (RQ4). At baseline, 182 (56.0 %) of 325 possible edges were estimated to be non-zero, with an overall mean edge weight of the respective network model of 0.025. At follow-up, these values were 178 (54.8 %) and 0.027, implying a similar level of sparsity. With the aim to identify consistent, robust edges across network models, we defined robustly estimated (thereafter: “robust”) edges as above the overall average edge weight of the respective network model; we consider these edges robust in the sense that they are

reliably estimated above zero. Table 3 provides an overview of such robust edges between individual PTSD symptoms and SCF for each network model, and all edge weights of each network model can be found in [Supplementary Materials](https://osf.io/5w6k4/) (<https://osf.io/5w6k4/>).

In both network models 1 and 2, robust edges emerged between SCF and the two PTSD symptoms ‘having difficulty concentrating’ (E5) and ‘trouble experiencing positive feelings’ (D7). In Network 1, robust edges were found between SCF and ‘irritable behavior, angry outbursts, or acting aggressively’ (E1), ‘avoiding memories, thoughts, or feelings related to the stressful experience’ (C1), ‘trouble falling or staying asleep’ (E6), ‘feeling jumpy or easily startled’ (E4), ‘trouble remembering important parts of the stressful experience’ (D1), and ‘loss of interest in activities that you used to enjoy’ (D5). In Network 2, robust edges were found between SCF at follow-up and ‘having strong negative beliefs about yourself, other people, or the world’ (D2) and ‘blaming yourself or someone else for the stressful experience or what happened after it’ (D3).

Similarity between the adjacency matrices of PCL-5 in the past month and during lifetime (i.e., network models estimated based on PCL-5 past month and lifetime, respectively, excluding SCF and covariates) was high ( $r = 0.79$ ). Fig. 2 shows the estimated networks of lifetime PTSD symptoms and SCF at baseline (Network 3; panel A) vs. SCF at follow-up (Network 4; panel B). In both networks, robust edges appeared between SCF and the three PTSD symptoms E5, D1, and D7. Thus, across network models 1–4, consistent, robust edges have been found between SCF and the two PTSD symptoms E5 and D7 (RQ3). Further information is provided in the [Supplementary materials](https://osf.io/5w6k4/) (<https://osf.io/5w6k4/>).

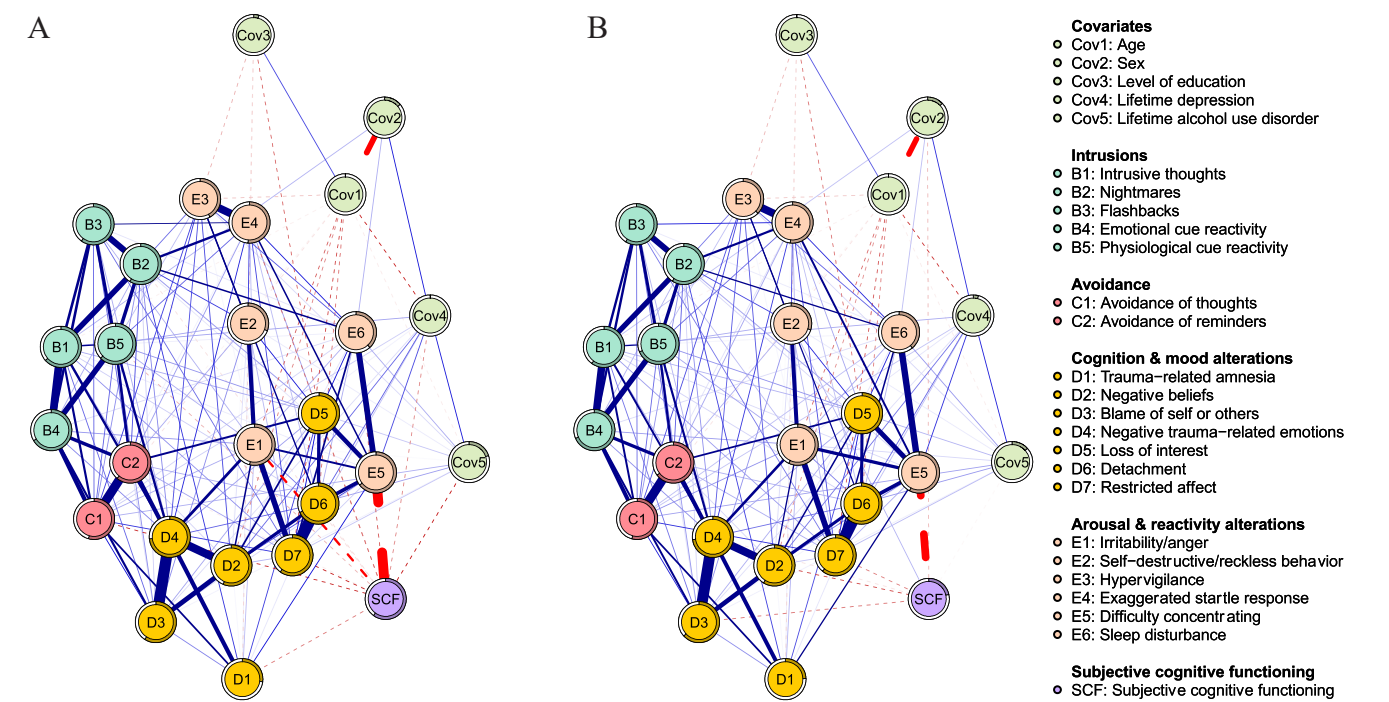
There was a positive association between SCF at baseline and SCF three years later ( $r = 0.53$ ,  $p < .001$ ). We re-estimated the longitudinal networks of past-month (Network 2<sub>adj</sub>) and lifetime (Network 4<sub>adj</sub>) PTSD symptoms at baseline and SCF at follow-up, with additional adjustment for SCF at baseline. The magnitude of edge weights generally was attenuated in the adjusted network models 2 and 4. Robust edges emerged between SCF at follow-up and E5, D3, and D2 in Network 2<sub>adj</sub> and 4<sub>adj</sub>, and a robust edge between D7 and SCF at follow-up was found in Network 4<sub>adj</sub>.

Node predictability of SCF for Networks 1 and 2 dropped from 60.1 % (baseline) to 21.6 % (follow-up). That is, at baseline, a large proportion of the variability in SCF was predominantly explained by PTSD symptoms and covariates, whereas over time this was reduced. Similar results were found for network models 3 and 4, with predictability of SCF changing from 52.6 % (baseline) to 17.7 % (follow-up). When added, SCF at baseline explained an additional eight to nine percent of the variance of SCF at follow-up (i.e., predictability was increased to 29.7 % and 26.5 % in Network 2<sub>adj</sub> and 4<sub>adj</sub>, respectively). We found similar results in the complete case analyses.

Across all network models, accuracy analyses revealed that the edge between E5 and SCF was stronger than all other edges between PTSD symptoms and SCF (see Figures in the [Supplementary Materials](https://osf.io/5w6k4/) for Network 1, <https://osf.io/5w6k4/>).

### 3.4. PTSD symptom domains and SCF

Although mean differences in average connectivity (i.e., average edge weight) between PTSD symptom domains with SCF were small, we observed robust and consistent patterns based on bootstrapped confidence intervals. Cross-sectionally (Networks 1 and 3), the domain of ‘alterations in arousal and reactivity’ was most strongly associated with SCF (RQ2). Over the three-year follow-up (Networks 2, 2<sub>adj</sub>, 4, and 4<sub>adj</sub>), both symptom domains of ‘alterations in arousal and reactivity’, as well as ‘negative cognitions and mood’, were most strongly associated with SCF (RQ2, RQ4). Our hypothesis that ‘intrusion’ symptoms would be most strongly linked to SCF is therefore not supported (more detailed results are available in the [Supplementary Materials](https://osf.io/5w6k4/); <https://osf.io/5w6k4/>). Results remained the same following complete case analyses.



**Fig. 1.** Networks displaying the relationship between baseline past-month posttraumatic stress disorder (PTSD) symptoms and subjective cognitive functioning (SCF) at baseline (Network 1; panel A) and SCF at follow-up (Network 2; panel B), after controlling for covariates. Blue lines indicate positive associations, dashed red lines negative associations, and thickness and brightness of an edge represent the association strength. Rings around nodes convey predictability, with shadowed parts depicting variance explained by connected nodes. For comparison, the maximum edge weight was set to the strongest edge across all estimated networks (0.36). For color, see online version.

**Table 3**  
Overview of robust edges between individual PTSD symptoms and SCF.

PTSD Symptoms	Robust Edges with SCF in Network Models
B1–Intrusive memories	
B2–Nightmares	
B3–Flashbacks	N3
B4–Emotional cue reactivity	N2 <sub>adj</sub> (positive), N3
B5–Physiological cue reactivity	N3
C1–Avoidance of thoughts	N1, N3
C2–Avoidance of reminders	
D1–Trauma-related amnesia	N1, N3, N4
D2–Negative beliefs	N2, N2 <sub>adj</sub> , N4, N4 <sub>adj</sub>
D3–Blame of self or others	N2, N2 <sub>adj</sub> , N4, N4 <sub>adj</sub>
D4–Negative trauma-related emotions	
D5–Loss of interest	N1, N3
D6–Detachment	
D7–Restricted affect	N1, N2, N3, N4, N4 <sub>adj</sub>
E1–Irritability/anger	N1, N3
E2–Self-destructive/reckless behavior	N2 <sub>adj</sub> (positive)
E3–Hypervigilance	
E4–Exaggerated startle response	N1, N3
E5–Difficulty concentrating	N1, N2, N2 <sub>adj</sub> , N3, N4, N4 <sub>adj</sub>
E6–Sleep disturbance	N1

*Note.* PTSD = Posttraumatic stress disorder; SCF = Subjective cognitive functioning; N1 = Network 1 (past-month PTSD symptoms and SCF at baseline); N2 = Network 2 (past-month PTSD symptoms at baseline and SCF at follow-up); N3 = Network 3 (lifetime PTSD symptoms and SCF at baseline); N4 = Network 4 (lifetime PTSD symptoms at baseline and SCF at follow-up); adj = additionally adjusted for SCF at baseline.

List of symptoms based on the PTSD Checklist for DSM-5 (PCL-5). Edges are identified as “robust” if their weight is above the overall mean edge weight of the respective network model. Unless stated otherwise, edge weights are negative.

3.5. Network comparison test

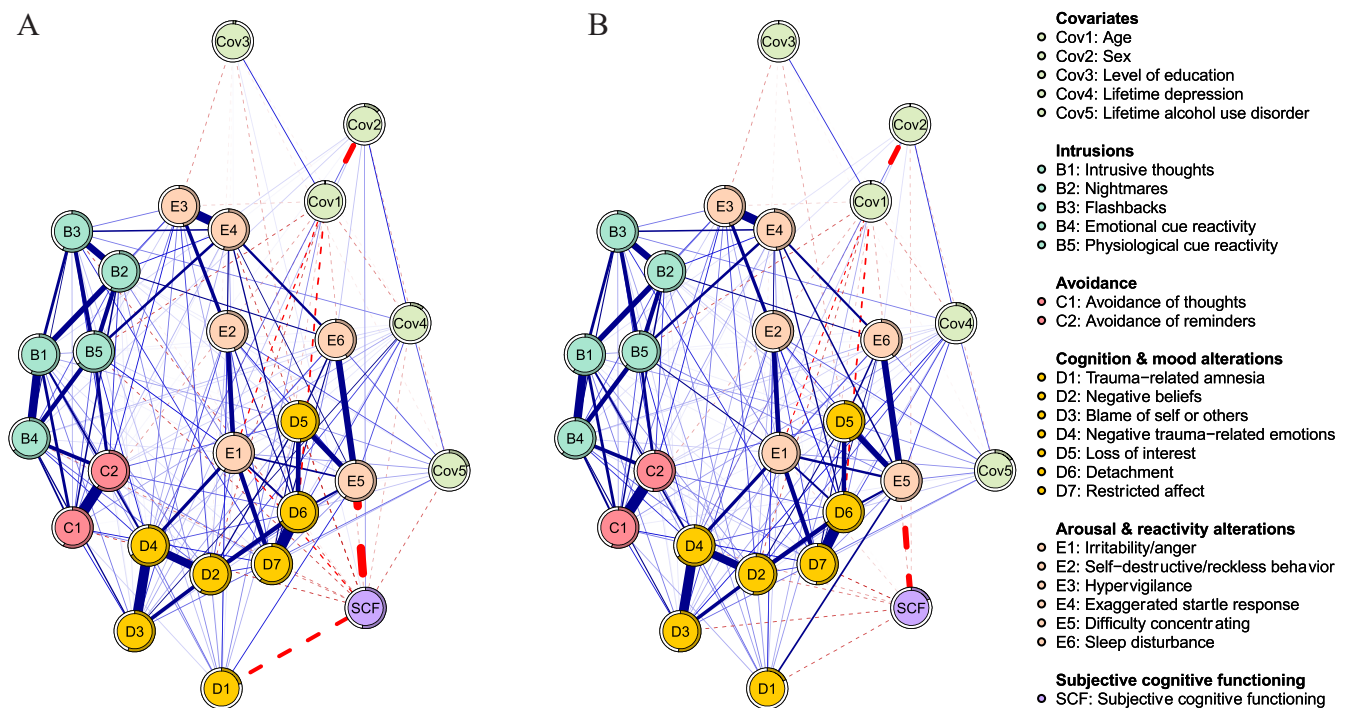
How stable were the relations between PTSD symptoms and SCF at baseline compared to 3 years later (RQ4)? Overall, results indicate

considerable temporal stability. First, the two corresponding Networks 1 and 2 were nearly identical ( $r = 0.97$ ), indicating temporal stability of the association between PTSD symptoms and SCF. However, according to the NCT, the two networks did significantly differ from each other regarding global strength ( $p < .001$ ) and network structure ( $p = .03$ ). Individual edges between SCF and PTSD symptoms that significantly differed between Networks 1 and 2 included B4, E1 and E2, of which E1 is a robust edge in Network 1. Comparing Networks 3 and 4 revealed differences regarding global strength ( $p < .001$ ) but no differences in network structure ( $p = .077$ ), with a strong correlation between the two ( $r = 0.97$ ). Additionally, we formally compared the cross-sectional (Networks 1 and 3) and longitudinal network models (Networks 2 and 4; both with and without adjustment for SCF at baseline). Similarity was high within each pair of networks, with  $r \sim 0.8$  between the respective adjacency matrices. The permutation tests of NCT revealed that global strength and network structure did not differ across networks within each pair ( $p > .05$ ). The above results did not meaningfully change following the complete case analyses.

4. Discussion

Four core findings are worth noting. First, as hypothesized, having had PTSD symptoms both in the past month or during lifetime was significantly and negatively associated with SCF, with a correlation of  $\sim 0.6$  at baseline and  $\sim 0.3$  at follow-up. Second, we did not find support for the hypothesis that intrusive symptoms of PTSD are most strongly associated with SCF relative to other domains. Instead, the two symptom domains of ‘alterations in arousal and reactivity’ (in cross-sectional and longitudinal network models), as well as ‘negative cognitions and mood’ (in longitudinal network models), were most strongly related to reduced SCF. Third, various individual PTSD symptoms were negatively associated with SCF. Across estimated networks, the PTSD symptoms of ‘difficulty concentrating’ and ‘trouble experiencing positive feelings’ were





**Fig. 2.** Networks displaying the relationship between baseline lifetime posttraumatic stress disorder (PTSD) symptoms and subjective cognitive functioning (SCF) at baseline (Network 3; panel A) and SCF at follow-up (Network 4; panel B), after controlling for covariates. Blue lines indicate positive associations, dashed red lines negative associations, and thickness and brightness of an edge represent the association strength. Rings around nodes convey predictability, with shadowed parts depicting variance explained by connected nodes. For comparison, the maximum edge weight was set to the strongest edge across all estimated networks (0.36). For color, see online version.

consistently and robustly linked to reduced SCF. Cross-sectionally, additional PTSD symptoms associated with reduced SCF included ‘irritable behavior, angry outbursts or acting aggressively’, ‘trouble remembering important parts of the stressful experience’, ‘avoiding memories, thoughts, or feelings related to the stressful experience’, ‘feeling jumpy or easily startled’, and ‘loss of interest in activities’. At the three-year follow-up, the additional PTSD symptoms ‘blaming yourself or someone else’ and ‘negative beliefs about yourself, other people, or the world’ were linked to reduced SCF, with and without adjusting for baseline SCF. Fourth, the association between PTSD symptoms and reduced SCF held over a three-year follow-up. Across all models, node predictability of SCF remained substantial and findings largely replicated at follow-up, despite some differences in global strength and network structures.

Some of our results are consistent with prior literature. PTSD symptoms are associated with impaired cognitive functioning (Brewin, Kleiner, Vasterling, & Field, 2007; Schuitevoerder et al., 2013; Scott et al., 2015) and the association is stable over time (Gould et al., 2018; Vasterling et al., 2018), with the former predicting the latter in the present study. Our results are also in line with prior findings that the symptom domain of ‘avoidance’ is not (strongly) associated with reduced cognitive functioning (Boals, 2008; Bomyea, Amir, & Lang, 2012; Clouston et al., 2016; Kivling-Bodén & Sundbom, 2003). Some of our findings are inconsistent, however, with previous evidence. Namely, that symptoms of ‘intrusion’ are most strongly linked to reduced cognitive functioning (Boals, 2008; Bomyea, Amir, & Lang, 2012; Clouston et al., 2016, 2019; Johnsen, Kanagaratnam, & Asbjørnsen, 2008; Kivling-Bodén & Sundbom, 2003; Parslow & Jorm, 2007; Saltzman, Weems, & Carrion, 2006; Vasterling et al., 1998).

A potential mechanism for the relationship between SCF and ‘alterations in arousal and reactivity’ as well as ‘negative cognitions and mood’ symptoms is that the latter two may preoccupy cognitive capacities, which are then less available for other actions (Kolb, 1987;

Schweizer & Dalglish, 2016). With regard to the PTSD symptom ‘trouble experiencing positive feelings’, evidence suggests that positive feelings are associated with less memory decline over time (Hittner et al., 2020), and enhance cognitive performance including working memory, decision making (Carpenter, Peters, Västfjäll, & Isen, 2013), and the ability to think flexibly (Isen, 2004). The broaden-and-build theory is one example of how positive emotions may improve cognitive function: by broadening a person’s mindset and momentary thought-action repertoire, the scopes of attention, cognition and action are expanded and various long-term personal resources (e.g., intellectual complexity) built (Fredrickson, 2004, 2001). Conversely, restricted positive affect may result in impaired cognitive performance.

Lower cognitive abilities may also serve as a pre-existing risk factor for PTSD (Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Marx, Doron-Lamarca, Proctor, & Vasterling, 2009; McNally & Shin, 1995; Moore, 2009; Parslow & Jorm, 2007; Vasterling et al., 2002, 2018; Vasterling, Brailey, Constans, Borges, & Sutker, 1997). Indeed, pathways between PTSD symptoms and cognitive functioning likely are bidirectional and complex. PTSD previously also has been identified as a risk factor for dementia (Günak et al., 2020), indicating longitudinal processes. Collectively, current evidence on mechanisms underlying the relationship of PTSD and cognitive functioning is preliminary. Future research is needed to provide a better understanding of the observed associations.

#### 4.1. Implications

The findings of the present study, if replicated in other samples and populations, may have several implications for the clinical management of individuals with PTSD symptoms. The results highlight the importance for clinicians’ awareness of potentially impaired cognitive functioning among patients, specifically, in older-aged individuals, and to monitor cognitive functioning when treating them (Clouston et al.,

2016). Based on our findings, this may be even more relevant when there are elevations in symptoms of ‘alterations in arousal and reactivity’ and ‘negative cognitions and mood’. The findings that these associations persisted over a three-year follow-up suggest that they may reflect stable processes.

Previous findings of a meta-analysis indicate that samples of individuals seeking or undergoing treatment for PTSD (compared to samples who do not) are more likely to show objectively measured cognitive difficulties (Scott et al., 2015). This may suggest that treatment-seeking individuals have more severe PTSD symptoms, greater comorbidity, and/or a chronic duration of the symptoms (Scott et al., 2015); that treatment does not prevent or protect from a decline in cognitive functioning; and/or that patients with impaired cognitive functioning are particularly likely to seek professional help. Cognitive impairment may also impede effective treatment as it might entail reduced ability to comply with therapeutic regimes and self-management during the treatment (Clouston et al., 2016). Indeed, poorer performance on certain objective cognitive measures such as verbal memory and neural activity underlying inhibitory control have been linked to a poorer treatment outcome in cognitive-behavioral therapy in people with PTSD (Falconer, Allen, Felmingham, Williams, & Bryant, 2013; Wild & Gur, 2008). Additionally, objective memory performance has been shown to predict occupational and social functioning (Geuze, Vermetten, de Kloet, Hijman, & Westenberg, 2009), and perceived cognitive problems to mediate the association between PTSD diagnosis, and perceived physical, emotional and social functioning and reintegration in veterans (Samuelson et al., 2017). The extent to which implementation of and response to PTSD treatment is affected by subjective cognitive impairment, and how this may relate to the previously found impact of objective cognitive impairment on treatment, should be further examined in future.

#### 4.2. Strengths and Limitations

The present study extends current knowledge by providing evidence regarding the potential link between PTSD and the regularly overlooked, yet relevant, subjectively experienced cognitive functioning (Hess et al., 2020; Lau et al., 2021; Savard & Ganz, 2016; Schuitevoerder et al., 2013; Scott et al., 2015) by exploring unique mutual associations between PTSD symptoms and SCF. The prospective cohort study design allowed us to examine temporal stability and to determine precedence of PTSD symptoms to cognitive functioning. We controlled for important covariates to minimize potential confounding, including SCF at baseline in the longitudinal network models.

Our study has several limitations. First, given the observational design and correlational results, we consider our findings to be hypothesis-generating for future studies on the link between PTSD and cognitive impairment. Second, although veterans represent an important subpopulation of individuals at heightened risk of developing PTSD symptoms (Wisco et al., 2016, 2022), the homogeneity of the sample (i. e., predominantly older-aged White males) may reduce the generalizability of the results to the general population and more sociodemographically diverse veteran populations. As post-stratification weights based on demographic distributions, to date, cannot be incorporated into network analyses, generalization might be further compromised. Replication studies are needed, in non-veteran and other ethnic populations. Third, we used self-report measures to assess PTSD symptoms and cognitive functioning instead of structured interviews and objective cognitive testing, respectively. While PTSD has repeatedly been related to neurocognitive deficits, presence, extent, and nature of change in cognitive functioning are not invariant (Scott et al., 2015). Moreover, despite the previously mentioned relevance of measuring SCF and one study suggesting that the MOS-CF correlates moderately with objective measures of corresponding cognitive domains (i.e., memory, attention, psychomotor speed) (Klein et al., 2002), results of the present study necessitate validation using comprehensive neuropsychological

assessment. Fourth, we could have disaggregated SCF to investigate the relations between PTSD symptoms and individual SCF items. Likewise, we could have included covariates as moderators to control for their impact on existing associations between nodes of interest (Haslbeck, 2022; Haslbeck, Borsboom, & Waldorp, 2021). However, both were not feasible in the current sample due to power constraints. Fifth, we defined “robust” edges as edges with weights above the mean edge weight of the respective network, and no, or other operationalizations could plausibly be chosen. Finally, we analyzed data from the entire sample in order to prevent Berkson’s bias leading to spurious correlations when analyzing a subset of the sample only (De Ron, Fried, & Epskamp, 2021). The minority of the sample screened positive for PTSD and results may not generalize to clinical populations of individuals with PTSD but rather exhibit the average network structure of the broader population of trauma-exposed veterans (von Stockert, Fried, Armour, & Pietrzak, 2018). Thus, our results may describe more normative developmental patterns.

#### 4.3. Conclusion

Notwithstanding the aforementioned limitations, our results indicate that not all PTSD symptoms are equally important in the relationship between PTSD and self-perceived cognitive functioning, both cross-sectionally and longitudinally. Certain individual PTSD symptoms as well as the symptom domains of ‘alterations in arousal and reactivity’, and ‘negative cognitions and mood’ are more strongly related to reduced self-reported cognitive functioning than symptoms of ‘intrusion’ or ‘avoidance’. The results of the present study aim at stimulating new research as much remains unknown regarding this striking relationship, which may have important implications for effective clinical care of people with PTSD symptoms.

#### CrediT authorship contribution statement

Conceptualization: M.M. Günak and E.I. Fried; Methodology: M.M. Günak, E.I. Fried, and R.H. Pietrzak; Resources: R.H. Pietrzak; Formal Analysis: M.M. Günak; Validation: O.V. Ebrahimi and E.I. Fried; Supervision: E.I. Fried; Writing – Original Draft: M.M. Günak; Writing – Review & Editing: M.M. Günak, E.I. Fried, O.V. Ebrahimi, and R.H. Pietrzak.

#### Supplementary material

Supplementary Material including data, code and measures can be found at <https://osf.io/5w6k4/>.

#### Prior versions

The present study is based on the first author’s master thesis uploaded on the Leiden University’s page at this link: <https://studenttheses.universiteitleiden.nl/handle/1887/3180705>.

#### Funding

Preparation of this report was supported in part by the U.S. Department of Veterans Affairs National Center for Posttraumatic Stress Disorder. The sponsors had no role in the design or conduct of the study; collection, management, analysis, or interpretation of the data; preparation, review, or approval of the manuscript; or in the decision to submit the article for publication.

#### Declaration of Competing Interest

None.



## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.janxdis.2023.102768](https://doi.org/10.1016/j.janxdis.2023.102768).

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